Center for Biological Diversity

Please accept the attached comments filed on behalf of the Center for Biological Diversity. The Center's comments will be provided through three submissions to relay all relevant attachments. This is submission 3 of 3.

Submitted Via State Public Comment Portal

May 7, 2024

Casey Sixkiller
Regional Administrator
Environmental Protection Agency, Region 10
1200 6th Avenue
Seattle, WA 98101

Marla Koberstein
Department of Ecology
Water Quality Program
P.O. Box 47696
Olympia, WA 98504

Re: Comments on Washington's Proposed Updates to Aquatic Life Toxics Criteria, WAC 173-201A-240 (CR-102)

Dear Ms. Koberstein and Regional Administrator Sixkiller,

Please accepted the following public comments submitted on behalf of the Center for Biological Diversity (Center) and its 1.7 million members and supporters to the Washington Department of Ecology's (Ecology) proposal to revise Washington's aquatic life toxics criteria, WAC 173-201A-240.

The Center is concerned that the proposed criteria provide insufficient protections for federally listed endangered and threatened species and, in consideration of prior national, Oregon, and Idaho Section 7 consultation findings, likely violates the Endangered Species Act's prohibition on the take of listed species. The Center, therefore, urges Ecology to revisit its proposed criteria for the benefit of endangered and threatened species and revise downward those criteria to levels that meet the obligations of the Clean Water Act to support the most sensitive aquatic life uses[1] and the Endangered Species Act's requirement that "endangered species [] be afforded the highest of priorities." *Tennessee Valley Authority v. Hill*, 437 U.S. 153, 174 (1978).

I. The Methodologies Used by Ecology and EPA for Deriving Water Quality Criteria Are Legally Deficient and Under-Protective of Endangered Species and Critical Habitats

The presence of toxic pollutants in waterways has a significant impact on aquatic and aquatic-dependent species' survival. According to the National Marine Fisheries Service (NMFS), "degraded water quality has been one of the contributing factors for the decline of almost all of

[1] *See* 40 C.F.R. § 131.11(a) (criteria must support the most sensitive use).

the anadromous fish species NMFS has listed since the mid-1980s."[2] Cyanide, cadmium, and mercury are three toxic pollutants that present significant threats to endangered and threatened aquatic species and their critical habitats.[3]

Over the last two decades, a series of lawsuits and consultations regarding EPA's national criteria and its approval of state standards and criteria for various pollutants—including cyanide, cadmium, and mercury—have raised profound concerns regarding the overall approaches that EPA utilizes in reviewing and approving water quality criteria; these cases also raise concerns about the inadequate and antiquated methodologies EPA used to establish national water quality criteria. *See, e.g., Center for Biological Diversity v. EPA*, Case No. 22-138, 2023 U.S. Dist. LEXIS 145674 (D. Ariz. Aug. 18, 2023) (finding that EPA acted unlawfully when it failed to engage in Endangered Species Act Section 7 consultation prior to issuing nationwide water quality criteria for cadmium and vacating EPA's 2016 chronic freshwater cadmium criterion); *Northwest Environmental Advocates v. National Marine Fisheries Service et al.*, Case No. 10-907-BR (2010) (dealing with the Oregon's Endangered Species Act consultation history and failures); *Northwest Environmental Advocates v. The National Marine Fisheries Service et al*., Case No. 13-00263-DCN (2013) (dealing with the Idaho's Endangered Species Act consultation history and failures).

The Center hereby attaches and incorporates into these comments past biological opinions and draft biological opinions and request they be made part of the record for this rulemaking as well as incorporated into EPA's review of Ecology's ultimate submission. The biological opinions describe severe methodological flaws and inadequate approaches that have inevitably yielded legally insufficient and under protective criteria. Each document included provides information that can guide Ecology's development of its criteria. More recent science, however, suggests the need for even more protective standards to fully comply with the Endangered Species Act.

Even further, because Washington is downstream of a number of states with known aquatic toxic pollution issues, including Idaho, Oregon, and even small portions of Wyoming and Montana, some of its waters are already receiving significant pollutants from upstream states, which raises concerns about cumulative impacts, and suggests even more stringent criteria are required to address pollution in a legally sufficient manner.[4] While in theory, Clean Water Act section 303(d)

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[2] NATIONAL MARINE FISHERIES SERVICE, DRAFT ENDANGERED SPECIES ACT SECTION 7 CONSULTATION BIOLOGICAL OPINION & CONFERENCE OPINION ON THE U.S. ENVIRONMENTAL PROTECTION AGENCY'S APPROVAL OF STATE OR TRIBAL, OR FEDERAL NUMERIC WATER QUALITY STANDARDS FOR CYANIDE BASED ON EPA'S RECOMMENDED 304(A) AQUATIC LIFE CRITERIA, 270 (2010) [hereinafter NMFS National Cyanide Draft BiOp].

[3] While these comments focus on the cyanide, cadmium, and mercury pollution and Washington's associated criteria, several additional pollutants are of concern to the Center. We request that Washington finalize toxics criteria across the board that are adequately protective of endangered and threatened species and their critical habitats.

[4] *See* EPA, Downstream Protection Guidance, Goal: Illustrate Considerations and Procedures Associated with Incorporating Downstream Protection into Development of Numeric Criteria, at 7 (2014) (describing that to develop downstream protections, the state should "establish numeric criteria in the receiving waterbody and build upstream")*; see also* 40 C.F.R. § 131.10(b) (a state "shall ensure that its water quality standards provide for the attainment and maintenance of water quality standards of downstream waters").

total maximum daily loads (TMDLs) are the mechanism to address total pollutant loading, Washington's TMDL program is largely moribund, it issues very few TMDLs for toxic pollutants, and its TMDLs do not take into consideration the cumulative effects of multiple toxic pollutants. For these reasons, Washington's water quality criteria for toxic pollutants must address the need to provide full protection of these downstream waters.

While the Center is generally supportive of Ecology's proposal to establish more stringent criteria, the proposed criteria still raise concerns regarding their effects on Washington's threatened and endangered species, including salmonids, southern resident orcas, and amphibians. Illustratively, for example, Washington's proposed chronic cyanide criteria is significantly higher than the level recommended in Fish and Wildlife Service's (FWS) biological opinion on EPA's national 304(a) cyanide criteria for bull trout. The proposal also does not appear to account for or address amphibian sensitivity to these toxics—another issue identified in FWS's biological opinion on EPA's national 304(a) criteria for cyanide.

II. Washington's Proposed Cyanide Water Quality Criteria are Not Adequately Protective of Listed Species or Critical Habitats

Cyanide, Freshwater	Proposed Acute (µg/L)	Proposed Chronic (µg/L)	ESA Consultation History, if Applicable
Idaho	22	5.2	Both received a jeopardy determination[5]
EPA	22	5.2	Both received a draft jeopardy determination[6]
FWS Draft BiOp	13.77	0.68	Recommended level for bull trout[7]
NMFS Draft BiOp	None Provided	None Provided	
WA Ecology	12	2.7	Yet to be fulfilled.

a. Salmonids

Past consultations by FWS and NMFS on toxics criteria nationally and standards in several Pacific Northwest states indicate that the presence of cyanide threatens a number of federally listed salmonids species found in Washington, including bull trout, Chinook salmon, chum salmon, coho salmon, sockeye salmon, and steelhead.[8]

[5] NATIONAL MARINE FISHERIES SERVICE, ENDANGERED SPECIES ACT SECTION 7(A)(2) BIOLOGICAL OPINION AND MAGNUSON-STEVENS FISHERY CONSERVATION AND MANAGEMENT ACT ESSENTIAL FISH HABITAT (EFH) CONSULTATION, 299 (2014) [hereinafter NMFS Idaho Toxics BiOp].
[6] FISH AND WILDLIFE SERVICE, DRAFT BIOLOGICAL OPINION ON EPA'S PROPOSED PROGRAM OF CONTINUING APPROVAL OR PROMULGATION OF NEW CYANIDE CRITERIA IN STATE AND TRIBAL WATER QUALITY STANDARDS, 298 (2010) [hereinafter FWS National Cyanide Draft BiOp].
[7] *Id.* at 304.
[8] NMFS National Cyanide Draft BiOp at 270.

On the basis of these past actions, the bull trout appears to be the most sensitive of Washington's federally endangered and threatened species that is threatened by presence of cyanide. As detailed in the above chart, Ecology's proposed criteria for cyanide are higher than levels established through past biological opinions as necessary to adequately protect bull trout as required by the Endangered Species Act.[9]

Cyanide has been shown to cause reduced growth rates, reproductive performance, and survival in bull trout.[10] High chronic levels of cyanide can reduce the number of eggs spawned by females, reduce the number of eggs that hatch, and drastically reduce the survivorship of young fish. In the biological opinion for EPA's national 304(a) cyanide criteria, FWS found that exposure to bull trout at the chronic criterion proposed by EPA would likely "substantially reduce their reproduction" and that exposure at the proposed acute criterion would likely cause "substantial reductions in survival."[11] Based on this "magnitude of adverse effects," FWS found that the species was likely to be extirpated from the waters where they are exposed to cyanide toxicity at either criterion amount and suggested a chronic freshwater criterion of 0.68 µg/L—significantly lower than the chronic freshwater criterion of 2.7µg/L for cyanide the Ecology proposes here.

Washington should, therefore, revisit its proposed criteria and revise downward to a proposed chronic freshwater criterion for cyanide of no more than 0.68 µg/L, more so if updated science shows that a more stringent standard is necessary to protect bull trout and other salmonid populations; the Center does not take immediate issue with Washington's proposed acute freshwater criteria but request that it be revised as necessary subject to the outcome of further Washington-specific Endangered Species Act consultation activities.

b. Oregon Spotted Frog

In its 2010 consultation with EPA regarding national 304(a) water quality criteria for cyanide, FWS noted a lack of data for effects of cyanide on amphibian species but concluded that because amphibians are among the most sensitive species for a significant number of the pollutants examined, it is likely that amphibian species are highly sensitive to cyanide.[12] There, FWS used data for relative sensitivity of amphibians to rainbow trout, since rainbow trout is a species often used for criteria development.[13] Based on this analysis, FWS concluded that amphibian species are estimated to be as or more sensitive to cyanide than rainbow trout and thus likely to be adversely affected by exposure to cyanide at EPA's suggested chronic criterion of 5.2µg/L.

Since that consultation was completed, the Oregon spotted frog was listed as a threatened species in 2014 and has two critically imperiled populations in Washington.[14] The Oregon spotted frog is considered "the most aquatic native frog species in the Pacific Northwest (PNW)."[15] In making

[9] FWS National Cyanide Draft BiOp at 304.
[10] FWS National Cyanide Draft BiOp at 221.
[11] *Id.*
[12] *Id.* at 250.
[13] *Id.*
[14] 79 Fed. Reg. 51,658 (Aug. 29, 2014).
[15] *Id*. at 51,661.

its listing determination, the FWS determined that toxic chemicals pose a hazard to the Oregon spotted frog.[16] Yet, Ecology does not even appear to have included the Oregon spotted frog on its list of relevant Endangered Species Act listed species.[17] Cyanide criteria must therefore be adjusted accordingly following Endangered Species Act consultation.

c. Orcas

Southern Resident Orcas could also be indirectly affected by Ecology's proposed cyanide criteria due to the possible reduction in salmonid populations.[18] Salmon, particularly Chinook salmon, are a key food source for the southern resident orcas and if proposed criteria harm salmonids, it is likely that the orcas will suffer as well. In NMFS consultation for EPA's national 304(a) cyanide criteria, the agency found that EPA's criteria would "reduce freshwater production of all listed salmon species, as well as non-listed salmon species where cyanide concentrations are allowed to reach EPA's recommended aquatic life criteria concentrations."[19]

III. Washington's Cadmium Water Quality Criteria are Not Adequately Protective of Listed Species and Critical Habitats

Cadmium is one of the most toxic metals to fish and can have various effects on aquatic organisms, including spinal deformities, inhibited respiration, immobility, and population alterations.[20] It can also cause neurotoxic effects in fish, manifesting as altered behavior, reduced growth, reproductive failure, and death.[21] Salmonids are particularly sensitive to cadmium pollution.[22] The principal acute effect of cadmium is gill toxicity, which causes an inability to breathe in aquatic organisms. Cadmium toxicity increases with water temperature.[23]

a. Freshwater Cadmium

Cadmium, Freshwater	**Proposed Acute (µg/L)**	**Proposed Chronic (µg/L)**	**ESA Consultation History, if Applicable**
Oregon	2.0	0.25	Acute standard received jeopardy determination.[24] Both standards likely to adversely affect listed species.

[16] *Id*. at 51,689-90.
[17] *See* Washington Dep't. of Ecology, Proposed Updates to Aquatic Life Toxics Criteria, WAC 173-201A-240 Technical Support Document, 31-32 (2024) [hereinafter Ecology Technical Support Doc].
[18] NMFS National Cyanide Draft BiOp at 271.
[19] *Id.* at 256.
[20] NATIONAL MARINE FISHERIES SERVICE, JEOPARDY AND DESTRUCTION OR ADVERSE MODIFICATION OF CRITICAL HABITAT ENDANGERED SPECIES ACT BIOLOGICAL OPINION FOR ENVIRONMENTAL PROTECTION AGENCY'S PROPOSED APPROVAL OF CERTAIN OREGON ADMINISTRATIVE RULES RELATED TO REVISED WATER QUALITY CRITERIA FOR TOXIC POLLUTANTS, 270 (2012) [hereinafter NMFS OR Toxics BiOp].
[21] *Id.* at 271.
[22] *Id.* at 270.
[23] *Id.* at 271.
[24] *Id.* at 547

Idaho	1.3	0.6	NMFS independent analysis: standards not likely to adversely affect ESA listed Chinook salmon, sockeye salmon, or steelhead in the state, but noted that determination was location specific[25]
EPA 2016	1.8	[0.72]	No consultation.[26] Chronic criterion vacated to 2001 value; acute criterion levels remain in place but have been remanded back to EPA by court order[27]
EPA 2001	[2.0]	0.25	No consultation.
WA Ecology	1.3	0.41	Yet to be fulfilled.

For cadmium, Ecology proposes a freshwater acute criterion of 1.3µg/L and a chronic freshwater criterion of 0.41 µg/L. Since EPA's nationwide 304(a) freshwater cadmium criterion was vacated by court order, the maximum concentration reverted back to the 2001 criterion of 0.25 µg/L; at a minimum, Washington must do the same.

However, based on the outcome of Endangered Species Act consultation, these criteria must be set at a level that is protective of federally listed species in Washington. Comparatively, the FWS biological opinion for Oregon toxics stated that "chronic exposure to cadmium at the proposed chronic level [of 0.25µg/L] is considered to have adverse effects to all bull trout potentially exposed by reducing their fitness through a reduction in growth."[28] The NMFS biological opinion for Oregon similarly found that "listed species exposed to waters equal to the acute or chronic [cadmium] criteria concentrations will suffer acute and chronic toxic effects."[29]

a. Saltwater Cadmium

Cadmium, Saltwater	**Proposed Acute (µg/L)**	**Proposed Chronic (µg/L)**	**ESA Consultation History, if Applicable**
Oregon	40	8.8	Listed species will suffer acute or chronic toxic effects including mortality (moderate intensity) and sublethal effects (moderate intensity)[30]
EPA 2016[31]	33	7.9	
WA Ecology 2024	33	7.9	Yet to be fulfilled.

[25] National Marine Fisheries Service, Comments on Environmental Protection Agency's Draft Aquatic Life Ambient Water Quality Criteria for Cadmium, 2 (Jan. 26, 2016).
[26] Center for Biological Diversity, EPA Approves Dangerous Water Quality Standards for Cadmium (April 1, 2016), https://www.biologicaldiversity.org/news/press_releases/2016/cadmium-04-01-2016.html.
[27] *Ctr. For Biological Diversity v. United States Env't Prot. Admin*, No. CV-22-00138-TUC-JCH, 2023 U.S. Dist. LEXIS 145674, at *44 (D. Ariz. Aug. 18, 2023).
[28] NMFS Oregon Toxics BiOp at 193.
[29] *Id.* at 270.
[30] *Id.* at 367.
[31] ENVIRONMENTAL PROTECTION AGENCY, AQUATIC LIFE AMBIENT WATER QUALITY CRITERIA CADMIUM – 2016, XV (2016).

Ecology's proposed change to saltwater cadmium criteria is also likely to put threatened and endangered species at risk. Ecology proposes to set saltwater cadmium criteria at EPA's 304(a) chronic criterion of 33µg/L and acute criterion of 7.9µg/L. During the peer review of EPA's 304(a) criteria, it was pointed out that the development of these criteria was based on insufficient toxicity data for effects on anadromous salmon and that "only one study evaluated Cd toxicity in coho salmon smolts in saltwater conditions, and this was at nearly full seawater strength."[32] This was a concern because anadromous salmonids encounter cadmium at lower salinities. It is important to better understand the impact of varying levels of salinity on cadmium toxicity of anadromous fish species and incorporate those findings into Washington's criteria.

The same peer review also noted that sea level rise associated with climate change is likely to cause saltwater intrusion into salmonid spawning habitat making it particularly important to understand how salinity affects cadmium toxicity.[33] Comparatively, in NMFS's biological opinion for Oregon's cadmium criteria, the agency pointed out various issues with EPA's criteria derivation methods, including for saltwater cadmium.[34] Therefore, relying on the EPA's 304(a) will not necessarily result in adequate protection for threatened and endangered species and their critical habitats in Washington waters.

IV. Washington's Existing Mercury Water Quality Criteria are Not Adequately Protective of Listed Species or Critical Habitats and Must be Updated

Washington should learn from Idaho's mistakes and move forward with updating its water quality criteria for mercury.[35] In Idaho, which Ecology cites as a reason for not proceeding with amended mercury criteria at this time, EPA recently issued a proposed rule providing for both tissue and water column criteria for mercury.[36] The proposed chronic total mercury criteria are 0.225 µg/kg wet weight for muscle fish tissue, 0.162 µg/kg wet weight for whole body fish tissue, and 0.0021 µg/L for water column values.[37] In so doing, EPA asserted that these results were consistent with reasonable and prudent alternatives in the Services' biological opinions, and explained that it is important to include both a tissue and water column value in mercury and methylmercury criteria.[38]

In contrast, Washington is not only proposing to neglect updating its mercury criteria through this rulemaking but, in doing so, it is continuing to rely on an outdated freshwater chronic criterion which measures the proposed water column value at 0.012 µg/L. That is insufficient. First, "[b]ecause tissue measurements provide a more direct measure of toxicity for bioaccumulative pollutants such as mercury, . . . it appropriate to establish tissue criteria for these pollutants. However, criteria expressed as organism tissue concentrations can prove challenging

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[32] ENVIRONMENTAL PROTECTION AGENCY, EPA RESPONSE TO EXTERNAL PEER REVIEW COMMENTS ON THE DRAFT AQUATIC LIFE AMBIENT WATER QUALITY CRITERIA FOR CADMIUM, 39 (2015).
[33] *Id.*
[34] NMFS OR Toxics BiOp at 366-367.
[35] *See, e.g., Northwest Environmental Advocates et al. v. United States Environmental Protection Agency*, Case No. 13-00263-DCN (Memorandum Decision and Order, ECF No. 103, July 19, 2021).
[36] *See* EPA, *Mercury Criterion to Protect Aquatic Life in Idaho*, 89 Fed. Reg. 24,758 (April 9, 2024).
[37] *Id*. at 24,774.
[38] *Id*. at 24,762, 24,768.

to implement in CWA programs such as NPDES permitting and Total Maximum Daily Loads (TMDLs) because these programs typically demonstrate that water quality standards are met by using a water column concentration to calculate a load-based effluent limit or daily load, respectively."[39] Both are needed.

Second, per Idaho's earlier FWS biological opinion, which Ecology quotes in its TSD at 82, "[b]ased on the above information, implementation of the proposed chronic criterion for mercury is likely to adversely affect growth, reproduction, and behavior in the bull trout throughout its distribution in Idaho." Idaho's proposed freshwater chronic criterion was 0.012 µg/L or the same as Washington's current criterion. This means that Washingtons mercury criteria are, a minimum, likely not to be sufficiently protective of bull trout.

V. EPA Methodologies for Derivation of Water Quality Criteria Do Not Prevent Adverse Effects to Listed Species and Critical Habitats

To the extent that Ecology based its proposed criteria on EPA's methodology, its analysis will suffer from the same issues as EPA's methodology—issues that are detailed in the NMFS biological opinions for EPA's national 304(a) cyanide criteria and Oregon's toxics criteria. The Center appreciates Ecology's attempts to account for some shortcomings in EPA's methodology by utilizing alternative derivation methods for some toxics and by using the 1st percentile of the genus toxicity data distribution rather than the 5th percentile. However, considering the extensive flaws underlying the toxicity data developed by EPA, using the 1st percentile of that data is not sufficient to protect endangered and threatened species.

For the freshwater acute cadmium criterion, for example, Ecology appears to be using the same derivation methods as EPA's recommendation;[40] for its chronic cadmium criterion, it used an EPA dataset and the 1st percentile of the toxicity distribution.[41] Although using the 1st percentile is more protective of species than the 5th, it is possible that issues in the underlying data still would not allow for a sufficiently protective calculation. Additionally, as discussed above, the proposed chronic cadmium criterion is in excess of the EPA criteria of 0.25µg/L, which is the current nationwide criteria following vacatur of EPA's 2016 criteria.

For cyanide, Ecology used new science in developing its proposed acute criterion, and an "acute to chronic" (ACR) ratio to develop its proposed chronic criterion because it lacked the toxicity data needed to calculate a chronic criterion using other methods.[42] The ACR is the ratio of the mean LC_{50} (concentration causing 50% lethality following acute exposure) for the species to the concentration following chronic exposure that causes a level of adverse effect that is the threshold of unacceptability.[43] Since the ACR was calculated by EPA and is based on underlying values that could suffer from the flaws in EPA's methodology highlighted by NMFS in its national 304(a) cyanide and Oregon toxics biological opinions, it is possible that the values proposed by Ecology reflect some of those issues as well.

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[39] *Id*. at 24,762.
[40] Ecology Technical Support Doc. at 60.
[41] *Id.* at 62.
[42] *Id.* at 127–128.
[43] NMFS National Cyanide Draft BiOp at 245.

Importantly, EPA's methodology for calculating toxicity values at which adverse effects occur *does not* adequately account for compounding stressors such as temperature, dissolved oxygen, and others on the responses of aquatic life to toxics.[44] In its biological opinion for Idaho's toxics standards, FWS recommended that any new standards be calculated "using a temperature/toxicity correlation"[45] to account for the inverse relationship between cyanide toxicity and temperature.[46] Dissolved oxygen is also important to account for because in environments with less than optimal dissolved oxygen, fish compensate by increasing gill movement and ventilation volume to maintain adequate oxygen volumes. Since cyanide is a powerful asphyxiant, additional cyanide in waters with low dissolved oxygen further stresses fish and reduces the lethal concentration at which survival is expected.[47] In the NMFS biological opinion for the national 304(a) cyanide criteria, the agency pointed out that EPA's attempts to "avoid confounding factors" in their analysis that prevents them from replicating realistic conditions in the wild.[48]

It is not clear whether or to what extent Ecology accounted for the increased toxicity of cyanide at low temperatures. This is an important consideration, particularly for salmonids that spawn in cold waters and could face serious consequences from increased toxicity of cyanide at these low temperatures. It is also unclear whether the proposed criteria accounted for the impact of low dissolved oxygen or concurrent exposures with other contaminants and stressors.

VI. Conclusion

Cyanide, cadmium, and mercury pollution threatens Washington's many endangered and threatened aquatic species. The Center urges Ecology to propose criteria that are sufficiently protective of Washington's federally protected endangered and threatened species, including by taking into consideration toxic pollution from upstream states and accounting for EPA's methodological limitations.

Please contact Hannah Connor at hconnor@biologicaldiversity.org with any questions.

Sincerely,

Hannah Connor
Environmental Health Deputy Director
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hconnor@biologicaldiversity.org

Trisha Sharma

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[44] *Id.* at 266.
[45] FISH AND WILDLIFE SERVICE, BIOLOGICAL OPINION FOR THE WATER QUALITY STANDARDS FOR NUMERIC WATER QUALITY CRITERIA FOR TOXIC POLLUTANTS (2015) at 277 [hereinafter FWS Idaho Toxics BiOp].
[46] *Id*. at 143.
[47] NMFS National Cyanide Draft BiOp at 221.
[48] *Id.* at 266.

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DRAFT

Endangered Species Act Section 7 Consultation Biological Opinion & Conference Opinion On the U.S. Environmental Protection Agency's Approval of State or Tribal, or Federal Numeric Water Quality Standards for Cyanide Based on EPA's Recommended 304(a) Aquatic Life Criteria

National Marine Fisheries Service
Office of Protected Resources
Silver Spring, MD 20910

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National Marine Fisheries Service
Endangered Species Act Section 7 Consultation
Biological Opinion & Conference Opinion

Agency: U.S. Environmental Protection Agency

Activities Considered: Approval of State or Tribal, or Federal Numeric Water Quality Standards for Cyanide Based on EPA's Recommended 304(a) Aquatic Life Criteria

Consultation Conducted by: Endangered Species Division of the Office of Protected Resources, National Marine Fisheries Service

Approved by: _____

Date: _____

Section 7(a)(2) of the Endangered Species Act of 1973, as amended (ESA; 16 U.S.C. 1539(a)(2)) requires each federal agency to insure that any action they authorize, fund, or carry out is not likely to jeopardize the continued existence of any endangered or threatened species or result in the destruction or adverse modification of critical habitat of such species. When a federal agency's action "may affect" a protected species, that agency is required to consult formally with the National Marine Fisheries Service (NMFS) or the U.S. Fish and Wildlife Service (FWS; together, the Services), depending upon the endangered species, threatened species, or designated critical habitat that may be affected by the action (50 CFR 402.14(a)). Federal agencies are exempt from this general requirement if they have concluded that an action "may affect, but is not likely to adversely affect" endangered species, threatened species, or designated critical habitat and NMFS or the FWS concur with that conclusion (50 CFR 402.14(b)).

The U.S. Environmental Protection Agency (EPA) initiated formal consultation with NMFS and the FWS on their recommended 304(a) criteria and the approval of state and tribal water quality standards, or federal water quality standards promulgated by EPA that are identical to or more stringent than the section 304(a) aquatic life criteria published pursuant to the Clean Water Act (CWA; 33 U.S.C. 1251 *et seq*.), for the protection of aquatic life from harmful effects of cyanide (CN). This document represents NMFS' biological and conference opinion (Opinion) on EPA's approval of numeric standards for cyanide in fresh and salt waters of the U.S and its effects on threatened and endangered species, their designated critical habitat, and species proposed for listing as threatened or endangered, and critical habitat proposed for designation. This consultation does not address the effects of specific modifications of these criteria that are undertaken by states and tribes or the permits issued by particular states or tribes. This Opinion contains a detailed explanation of the particular circumstances warranting subsequent consultation (tiered consultations) with NMFS' Regional Offices in the section titled *Application*

of this Consultation to Other EPA Actions.

This Opinion is based on our review of the EPA's *Biological Evaluation of Aquatic Life Criteria- Cyanide*, status reviews, listing documents, and recovery plans for the threatened and endangered species under NMFS' jurisdiction, reports on the status and trends of water quality in the United States that have been prepared by the U.S. Geological Survey, EPA, and others, past and current research and population dynamics modeling efforts, and published and unpublished scientific information on the biology and ecology of threatened and endangered sea turtles, marine mammals, salmon, sturgeon, sawfish, abalone, and seagrasses in the action area, and other sources of information which are discussed in greater detail in the *Approach to the Assessment* section of this Opinion.. This Opinion has been prepared in accordance with section 7 of the ESA and associated implementing regulations.

Consultation History

On January 18, 2001, the Services and EPA signed a Final Memorandum of Agreement (MOA) on the enhanced coordination under the ESA and the CWA. The final MOA published in the *Federal Register* on February 22, 2001 (66 FR 36) and described, among other things, a plan for assisting EPA in meeting it's section 7 responsibilities on two CWA programs: water quality standards and the National Pollutant Discharge Elimination System (NPDES) permits program.

In January 2004, the Services and EPA decided to proceed with a data call for the first batch of pollutants that would be reviewed in consultation, while continuing to work on the *Draft Methodology for Conducting Biological Evaluations of Aquatic Life Criteria--Methods Manual.*

On May 14, 2004, the Services and EPA issued data calls to the regional staff and science center staff requesting information and data on cyanide, ammonia, chromium III and chromium VI. The data call requested regions and science centers send relevant studies to Headquarters by June 30, 2004.

On November 12, 2004, EPA provided the Services a revised *Draft Methodology for Conducting Biological Evaluations of Aquatic Life Criteria--Methods Manual* (dated October 29, 2004, on the document). This version represented a methodology developed collaboratively, and which had been peer reviewed by subject experts outside of the Federal government.

In December 2004, NMFS and EPA exchanged comments on recommended revisions to the November draft methodology. EPA also informed the Services that they had received a draft BE for cyanide from their contractor and were reviewing the document to ensure the contractor had followed the BE methodology accurately.

On January 24, 2005, EPA emailed NMFS a partial draft of their CN BE.

On May 3, 2005, the Services jointly provided comments to EPA on their January 19, 2005, draft biological evaluation for cyanide criteria.

On January 26, 2006, EPA provided NMFS with a draft CN BE and requested a review of the

BE's "completeness" in fulfilling the information requirements for section 7 consultation. On April 21, 2006, NMFS provided comments to EPA on the "completeness" of the draft BE.

In a June 29, 2006, letter, EPA requested NMFS' concurrence with their determination that proposed approval of cyanide criteria was not likely to adversely affect all listed species and critical habitat under NMFS' jurisdiction.

On November 11, 2006, the FWS sent NMFS a copy of EPA's revised *Draft Framework for Conducting Biological Evaluations of Aquatic Life Criteria: Methods Manual*, which EPA revised and submitted to FWS in July 31, 2006 and which EPA used to support their effects determinations.

On November 15, 2006, NMFS sent EPA a letter with a detailed explanation as to why NMFS could not concur with EPA's determinations that the recommended water quality standards for cyanide "may affect, but are not likely to adversely affect" threatened and endangered species and designated critical habitat.

On March 23, 2007, EPA requested formal consultation supported by their March 23, 2007, *Biological Evaluation of Aquatic Life Criteria—Cyanide*, which concluded their action was "not likely to jeopardize the continued existence of any federally listed species or result in the destruction or adverse modification of designated critical habitat [*sic*]."

On June 21, 2007, NMFS sent a letter to EPA acknowledging the initiation of formal consultation. NMFS' letter acknowledged that the scope and complexity of the national consultation on the aquatic life criteria for cyanide may require more time than usual to complete the biological opinion.

On May 5-9, 2008, the Services met with EPA to conduct a "Kaizen" "lean event." The purpose of the meetings was to analyze the cyanide consultation process from the development of a biological assessment through the anticipated completion of formal consultation in an effort to find efficiencies in the process. The Services and EPA also discussed coordination and communication with respect to the national consultation on cyanide and local consultation on EPA promulgation of Oregon water quality standards.

On June 12, 2008, the Services and EPA met to follow up on the Kaizen lean event. Subsequent follow up meetings were cancelled until the Services completed draft biological opinions.

BIOLOGICAL & CONFERENCE OPINION

Description of the Proposed Action

The action considered in this Opinion, and beginning a series of national water quality consultations with EPA, is EPA's continuing approval of state or tribal water quality standards, or federal water quality standards promulgated by EPA, that are identical to or more stringent than EPA's recommended 304(a) aquatic life criteria for cyanide. These water quality standards define water column concentrations of cyanide that should protect against adverse ecological effects to aquatic life in fresh and salt water. The 304(a) aquatic life criteria recommendations, which are the foundation for many approved 303(c) standards, are designed to protect aquatic organisms from unacceptable toxicity during acute (short) and chronic (long) exposures in the water column. The intent is to define a level in the waterbody of a pollutant that will be fully protective of the designated use and which a regulatory authority may use in adopting regulatory water quality standards and thereby control, reduce, or eliminate discharges of that pollutant (BE page 11).

Section 304(a)(1) of the CWA directs EPA to publish criteria for water quality accurately reflecting the latest scientific knowledge on a number of factors including "… the kind and extent of all identifiable effects on health and welfare including, but not limited to, plankton, fish, shellfish, wildlife, plant life, shorelines, beaches, esthetics, and recreation which may be expected from the presence of pollutants in any body of water, including ground water; on the concentration and dispersal of pollutants, or their byproducts, through biological, physical and chemical processes; and on the effects of pollutants on biological community diversity, productivity, and stability including information on the factors affecting rates of eutrophication and rates of organic and inorganic sedimentation for varying types of receiving waters." The water quality standards program is authorized under section 303(c) of the CWA (33 U.S.C. 1313(c)) and directs states to adopt numeric criteria for specific toxic pollutants that appear on a priority pollutant list[1] and for which EPA published 304(a) criteria recommendations. States can, pursuant to section 303(c) of the CWA, adopt water quality standards that differ from EPA's 304(a) criteria values whenever adequately justified, but states and tribes generally choose to adopt EPA's 304(a) criteria verbatim. Once adopted into state water quality standards, criteria form the legal basis for implementing the CWA programs to control pollution and achieve the goals and requirements of the CWA.

The purpose of these national consultations is to assess the effect of the EPA's 304(a) criteria recommendation and the subsequent approval of state and tribal water quality standards, or federal water quality standards promulgated by EPA that are identical to or more stringent than the section 304(a) aquatic life criteria on threatened and endangered species and their designated critical habitat (together, listed resources), and species and critical habitat that are proposed for

[1] Section 307(a) of the CWA, which defines priority pollutants as compounds and families that are among the most persistent, prevalent and toxic chemicals.

listing or designation (together, proposed resources). In particular, this Opinion analyzes whether EPA's approval of state standards that rely on the national criteria for cyanide are not likely to jeopardize the continued existence of threatened and endangered species (including species proposed for listing as threatened or endangered), or result in the destruction or adverse modification of designated critical habitat (see the BE, page 1).

In 1985 EPA published two values for cyanide pollution in each fresh and salt "waters of the United States," the criterion maximum concentration (CMC) and the criterion continuous concentration (CCC). EPA's ambient water quality criteria for cyanide are expressed as free cyanide (Table 1). The CMC represents EPA's estimate of the highest concentration of cyanide in fresh or salt water to which an aquatic community's brief exposure (acute limit) would not result in an unacceptable effect. The CMC is derived from a set of LC50 values for a variety of aquatic species. The LC50 value is the lethal concentration of a chemical that causes 50% mortality, immobilization, or loss of equilibrium in the test organism in 48 to 96-hour laboratory tests. The CMC is then set to one-half of the fifth percentile of the genus mean acute value (GMAV) for the various species tested to provide a level of protection that is better than 50% mortality. EPA recommends that the one-hour average exposure concentrations should not exceed the CMC more than once every three years on the average, making such exceedances a relatively rare event (EPA 1991).

Table 1. Cyanide 304(a) Aquatic Life Criteria (in μg/L of free cyanide [EPA 1985])

Medium	Criterion Maximum Concentration	Criterion Continuous Concentration
Fresh water	22.36	5.221
Saltwater	1.015	1.015

The CCC represents EPA's estimate of the highest concentration of cyanide in either fresh or salt water, to which an aquatic community's prolonged exposure (chronic limit) would not result in an unacceptable effect. The CCC is derived from a set of chronic values, which are the geometric mean of the highest no observed effect concentrations (NOECs) and lowest observed effect concentrations (LOECs) for survival, growth, or reproduction in tests that range from seven days to several months. EPA sets the CCC to the estimated fifth percentile of the chronic values either by direct calculation or by using the acute-to-chronic ratios (ACRs). For the CCC, EPA recommends that the four-day average exposure concentrations should not exceed the CCC more frequently than once every three years on average (EPA 1991).

Approach to the Assessment

Section 7(a)(2) of the ESA of 1973, as amended (16 U.S.C. 1536(a)(2)), requires federal agencies, in consultation with and with the assistance of the Services, to ensure that any action they authorize, fund, or carry out is not likely to jeopardize the continued existence of endangered species or threatened species or result in the destruction or adverse modification of designated critical habitat. When NMFS consults with federal agencies to help them comply with this requirement, we first assess the direct and indirect effects of the proposed federal action

to determine whether the proposal is likely to (a) appreciably increase a species' extinction probability (or reduce their probability of being conserved or recovered) or (b) appreciably reduce the conservation value of critical habitat that has been designated for one or more of those species. If we conclude that one of these outcomes is likely, we work with the federal agency, applicant, or both, to develop alternatives that avoid this likelihood.

NMFS approaches its section 7 analyses through a series of steps. The first step identifies those aspects of proposed actions that are likely to have individual, interactive, or cumulative direct and indirect effects on the environment (the potential stressors of an action). As part of this step, we identify the spatial extent of these stressors, including changes in their spatial extent over time. The spatial extent of these stressors represents the *Action Area* for consultation.

To begin the second step of our analyses, we determine whether endangered species, threatened species or designated critical habitat are likely to occur in the same space and the same time as the potential stressors. These species then become the focus of our *Exposure Analysis*. As our point of reference for evaluating the risk posed by their exposure, we rely on our understanding of the condition of the species and the conservation value of critical habitat, and any biological and ecological information on the species and their critical habitat that is relevant to our effects analysis (this information is represented in the *Status of the Species and Critical Habitat*). In the status of the species section of our Opinion, we review the species' legal status, trends, and the threats that led to this status as well as those that may be impeding the species' chances of recovery. Our assessment is also informed by the effects of past and ongoing human and natural factors leading to the current status of the species, its habitat, and ecosystem. This information is presented in the *Environmental Baseline*. By regulation, the environmental baseline for an action includes the past and present impacts of all federal, state, or private actions and other human activities in an action area, and the anticipated impacts of all proposed federal projects in the action area that have already undergone formal or early section 7 consultation, and the impact of state or private actions that are contemporaneous with the consultation in process. The environmental baseline is designed to assess the condition of the habitat and the species within the action area.

Often, NMFS will combine the status of the species and the environmental baseline where the status encompasses the entire range of a species. In this Opinion, we address the two separately, focusing the environmental baseline on the current condition of the nation's fresh water and marine aquatic habitats. In some cases we address watersheds that may not contain listed species under NMFS' jurisdiction because the watershed influences coastal conditions where listed marine and anadromous species occur. Our summary of the environmental baseline complements the information provided in the status of the species section of this Opinion, and provides information on the past and present ecological conditions of the action area that is necessary to further understand the species' current risk of extinction.

Our effects analyses, summarized in the *Effects of the Action* section of this Opinion, identify the nature of the listed species and critical habitat co-occurrence with the effects of the action over space and time (their exposure). Our exposure analyses identify the physical or biological features of critical habitat that would be exposed to the action, including any listed primary constituent elements that require special management consideration or protection such as sites for

breeding and rearing, food, water, space for growth and normal behavior, and cover and shelter; and we identify the number, age or life stage, and gender of the individuals that are likely to be exposed to an action's effects and the populations or subpopulations those individuals represent. Once we identify the individuals and populations, or constituent laments that are likely to be exposed to an action's effects and the nature of that exposure, we examine the scientific and commercial data available to determine whether and how those listed species and their critical habitat (collectively termed listed resources) are likely to respond given their exposure (these represent our *response analyses*). The final steps of our analyses—establishing the risks those responses pose to listed resources—are different for listed species and designated critical habitat (these represent our *risk analyses*).

Our jeopardy determinations must be based on an action's effects on the continued existence of threatened or endangered species as those "species" have been listed, which can include the biological species, subspecies, or distinct population segments of vertebrate species. Because the continued existence of listed species depends on the fate of the populations that comprise them, the viability (probability of extinction or probability of persistence) of listed species depends on the viability of the populations that comprise them. Similarly, the continued existence of populations are determined by the fate of the individuals that comprise them; populations grow or decline as the individuals that comprise the population live, die, grow, mature, migrate, and reproduce (or fail to do so). Our risk analyses reflect the relationships between the listed species and the populations that comprise them, and the individuals that comprise those populations. Our risk analyses begin by identifying the probable risks actions pose to listed individuals that are likely to be exposed to an action's effects. Our analyses then integrate those individuals' risks to identify consequences to the populations they represent and next we determine the consequences of population-level effects on the species as listed.

We measure risks to listed individuals using the individual's "fitness," which are changes in an individual's growth, survival, annual reproductive success, or lifetime reproductive success. In particular, we examine the scientific and commercial data available to determine if an individual's probable responses to an action's effect on the environment (which we identify during our response analyses) are likely to have consequences for the individual's fitness. When individual listed plants or animals are expected to experience reductions in fitness, we would expect those reductions to also reduce the abundance, reproduction rates, or growth rates (or increase variance in one or more of these rates) of the populations those individuals represent (see Stearns 1992). A reduction in one or more of these variables (or one of the variables we derive from them) is a *necessary* condition for reductions in a population's viability, which itself is a *necessary* condition for reductions in a species' viability. On the other hand, when listed plants or animals exposed to an action's effects are not expected to experience reductions in fitness, we would not expect the action to have adverse consequences on the viability of the populations those individuals represent or the species those populations comprise (for example, see Anderson 2000, Mills and Beatty 1979, Stearns 1992). If we conclude that listed plants or animals are not likely to experience reductions in their fitness we would conclude our assessment.

If, however, we conclude that listed plants or animals are likely to experience reductions in their fitness, our assessment examines if those reductions are likely to be sufficient to reduce the

viability of the populations those individuals represent (measured using changes in the population's abundance, reproduction, spatial structure and connectivity, growth rates, genetic health, or variance in these measures to make inferences about the population's extinction risks). In this step of our analyses, we use the population's base condition (established in the *Environmental Baseline* and *Status of Listed Resources* sections of this Opinion) as our point of reference.

Our assessment framework assumes—an assumption that is supported by published evidence—that the health and fitness of individual plants or animals will integrate the effects of the physical, chemical, and biological phenomena they are exposed to during their lifetimes and at specific developmental stages of their lives. That is, our assessment framework assumes that the total effects of exposing an animal to a suite of stressors, for example, coho salmon to a combination of toxic chemicals and an altered hydrograph from various flow controls will appear as a reduction in the fitness (reductions in annual or lifetime reproductive success) of individual coho salmon thus exposed. If exposing endangered or threatened marine and anadromous animals to chemical pollutants interacts with their exposure to other anthropogenic stressors, such as construction noise or disturbance or other toxic chemicals, and produces consequences that would not occur without that interaction, the consequence would appear as a reduction in performance of the individual animals.

Thus our assessment of the impact of the proposed action begins by considering the impact of the environmental baseline on the fitness of the individuals in the action area. As part of this assessment, we must consider how listed individuals are likely to respond to any interactions and synergisms between the proposed action and its stressors, pre-existing stressors and experience (represented by the *Status of the Species* and *Environmental Baseline*, as well as those stressors that are reasonably certain to occur in the action area for the future life of the action (represented by *Cumulative Effects*). If we conclude that listed individuals are likely to experience reductions in their annual or lifetime reproductive success, we then ask if those reductions are likely to be sufficient to reduce the viability of the populations those individuals represent (measured using changes in the population's abundance, reproduction, spatial structure and connectivity, genetic health, growth rates, or variance in these measures to make inferences about the population's probability of becoming extinct). Finally, if we conclude that the viability of one or more populations of a listed species is likely to be reduced, we determine whether that reduction is likely to be sufficient to reduce the viability of the species those populations comprise (here, a species' "viability" is its probability of becoming extinct or of being "recovered" to the point at which the protections of the ESA are no longer necessary or warranted). In this step of our analyses, we use the species' status as our point of reference.

For designated critical habitat, our destruction or adverse modification determinations must be based on an action's effects on the conservation value of habitat that has been designated as critical.[2] If an area encompassed in a critical habitat designation is likely to be exposed to the direct or indirect consequences of the proposed action on the natural environment, we ask if

[2] Several courts have ruled the definition of destruction or adverse modification that appears in the section 7 regulations at 50 CFR 402.02 as invalid. Consequently, we do not rely on that definition for the determinations we make in this Opinion. Instead, we use the conservation value of critical habitat for our determinations which focuses on the designated area's ability to contribute to the conservation of the species for which the area was designated.

constituent elements included in the designation (if there are any) or physical, chemical, or biotic phenomena that give the designated area value for the conservation of the species, are likely to respond to that exposure. If those constituent elements (or phenomena) are likely to respond, we ask if those responses are likely to be sufficient to reduce the quantity, quality, or availability of those constituent elements or physical, chemical, or biotic phenomena. If the conservation value is reduced, we then ask if those reductions are likely to be sufficient to reduce the conservation value of the entire critical habitat designation.

National Programmatic Consultations

Our national programmatic consultations typically analyze the general environmental consequences of a broad scope of actions or policy alternatives under consideration by a federal agency. In these types of consultations we focus on the general patterns associated with an agency's decision to authorize a particular national or programmatic action. Subsequent consultations that "tier" off of these national consultations, when warranted, would analyze the project and site specific effects typical of most consultations. Any subsequent section 7 consultations conducted by NMFS personnel would be designed to determine whether and to what degree the specific action under review fits within the general pattern identified in the national consultations, and would determine whether the specific action, is or is not likely to jeopardize the continued existence of endangered and threatened species or result in the destruction or adverse modification of designated critical habitat.

Thus, our national programmatic consultations focus on the evidence available to determine whether and to what degree the agency's action is likely to prevent exposure, or mitigate the responses or risks any responses would pose to listed species or their designated critical habitat. An agency can generally satisfy this requirement when the action contains features that: (1) prevent listed resources from being exposed to subsequent actions or their direct or indirect effects; (2) mitigate how listed resources respond to that exposure, when listed resources are exposed to the actions and their effect; or (3) mitigate the risks any responses pose to listed individuals, populations, species, or designated critical habitat when listed resources are likely to be exposed and respond to that exposure.

In examining an agency's program, we would examine the general activities the agency would authorize, fund or carry out. The steps of the national-level assessment remain much the same as described for our site-specific consultation, as outlined earlier in this section. National broad scale assessments and programmatic assessments, however, are necessarily focused on whether and to what degree a federal action can ensure that actions taken under the program are not likely to individually or cumulatively, jeopardize the continued existence of endangered or threatened species and are not likely to result in the destruction or adverse modification of designated critical habitat. Our description of the probable responses of the listed resources to the national action and the risks the national action poses to those listed resources is at the core of our evaluation, and is informed by the general patterns we observed through prior experience with an agency's actions or classes of activities.

The conceptual model NMFS uses for national consultations focuses on four main elements of action agency's national action: (1) the decision-making process an action agency uses to

authorize, fund, or carry out national actions; (2) the national action, and any subsequent actions or activities the agency would authorize, fund or carry out in accordance with the national action; (3) the intended and unintended consequences that are likely to result from authorized activities; and (4) the mechanisms that improve the agency's action(s) over time. We begin our national consultations by recognizing that an agency's program normally represents the agency's decision to authorize fund, or carry out a suite or class of activities (or recommend actions) that may (or may not) require specific actions undergo subsequent review and decision-making.

An agency's decision-making process will normally identify certain standards that an action must satisfy before an agency would authorize, fund or carry them out. Generally, decision-making involves hard or formal procedures (such as agency regulatory procedures and public noticing requirements), soft or flexible information standards (e.g., agency "guidelines", and the best professional judgments personnel make when considering conflicting information and making recommendation in the face of uncertainty). These procedures outline how the agency would decide whether or not to authorize, fund or carry out specific actions. Typically an agency's decision making process is shaped to respond to:

- the statutory and regulatory standards an action must satisfy before the agency would authorize, fund, or carry them out;
- any data and other information the agency must gather and evaluate to satisfy their statutory and regulatory requirements, as well as requirements of the Administrative Procedure Act, Information Quality and related administrative statutes, like the Paperwork Reduction Act, Regulatory Flexibility Act, and so on.
- the agency's obligation to review and analyze the relevant information within the context of applicable standards to ensure that specific actions satisfy all applicable statutory and regulatory requirements;
- the results of the agency's efforts to monitor specific actions the agency has authorized, funded or carried out, and the consequences of those decisions;
- and any feedback mechanism an agency has created to ensure that a program satisfies its statutory mandates, regulatory requirements, and applicable goals, and minimizes unintended consequences from the agency action.

If an agency proposes to satisfy its section 7(a)(2) obligations using a decision-making process that insures that listed resources are not exposed to specific actions without undergoing a tiered section 7 consultation on a specific action, we examine the evidence available to determine whether and to what degree the agency's decision-making process is likely to produce that outcome. If the agency's decision-making process is designed to mitigate the consequences of exposing listed resources to specific actions, we examine the evidence available to determine whether and to what degree the agency's decision-making process produces that outcome. When we consult on a pre-existing program, the program's general pattern of performance over its history becomes our primary evidence.

After we examine an agency's decision-making process, we then examine the classes of actions the program would authorize, fund, or carry out. This step of our assessment is designed to determine whether and to what degree listed resources are likely to be exposed to different classes of activities that would be authorized, funded, or carried out under a program. During

this step of our assessment, we consider the geographic distribution, timing, and constraints of the different classes of activities that would be authorized, funded, or carried out by a program (the geographic distribution of the activities' effects defines the action area of programmatic consultations). These analyses represent the "exposure analyses" of our programmatic consultations in which we try to identify the populations or subpopulations, ages (or life stages), and gender of the individuals that are likely to be exposed to an action's effects.

Then we use the best scientific and commercial data available to identify the classes of intended and unintended consequences that are likely to result from the different classes of activities. These analyses identify the probable direct and indirect consequences of exposing listed resources to those classes of activities for listed individuals, populations, and species, and designated critical habitat; these analyses represent the "response analyses" and "risk analyses" of our programmatic consultations. Our "response analyses" review the scientific and commercial data available to determine whether, how, and to what degree listed resources are likely to respond given their exposure to the intended and unintended consequences of classes of activities. Our "risk analyses" begin by identifying the probable consequences of those responses for the "performance" of listed individuals, and then they identify the consequences of changes in individual performance on the viability of the populations those individual represent. Our "risk analyses" conclude by determining the consequences of changing the viability of the populations, and the species those populations comprise. As stated earlier, our assessment is based on the general patterns that we observe through our prior experiences with a program or class of activities.

Evidence Available for the Consultation

To conduct our analyses, we considered lines of evidence available through published and unpublished sources that represent evidence of adverse consequence or the absence of such consequences. In particular, we considered information contained in *EPA's Biological Evaluation for Cyanide*, and published information used in deriving the 304(a) aquatic life criteria for cyanide. We supplemented this information by conducting electronic searches of literature published in English or with English abstracts using research platforms in the *Online Computer Library Center's* (OCLC) *First Search*, *CSA Illumina*, *Toxline*, *Science Direct*, *Water Resources Abstracts, Oceanic Abstracts, BioOne Abstracts and Indexes, Conference Papers Index*, *Lexis-Nexis, Google Scholar,* and *ISI Web of Science*. These platforms allowed us to cross search multiple databases for journals, open access resources, books, proceedings, web sites, doctoral dissertations and master's theses for literature on the biological, ecological, and medical sciences. Particular databases we searched for this consultation included *Basic Biosis*, *Dissertations*, *ArticleFirst*, *Proceedings*, *Aquatic Sciences and Fisheries Abstracts* and ECO databases, which index the major journals dealing with ecological risk, biology and ecology of particular species, and the toxicology of cyanide in freshwater, estuarine, and marine ecosystems (e.g., journals such as *Environmental Toxicology and Chemistry*, *Human and Ecological Risk Assessment*, *Journal of Mammalogy*, *Canadian Journal of Zoology*, *Transactions of the American Fisheries Society*, *Conservation Biology*, and others).

For our literature searches, we used paired combinations of the keywords cyanide, salmon,

marine mammals, sea turtles, sturgeon, coral, sawfish, seagrass, and many others to search these electronic databases. Electronic searches have important limitations, however. First, often they only contain articles from a limited time span (e.g., First Search only provides access to master's theses and doctoral dissertations completed since 1980 and Aquatic Sciences and Fisheries Abstracts only provide access to articles published since 1964). Second, electronic databases commonly do not include articles published in small or obscure journals or magazines. Third electronic databases do not include unpublished reports from government agencies, consulting firms, and non-governmental organizations. To overcome these limitations, we supplemented our electronic searches by searching the literature cited sections and bibliographies of references we retrieved to identify additional papers that had not been captured in our electronic searches. We acquired references that, based on a reading of their titles and abstracts, appeared to comply with our keywords. If a references' title did not allow us to eliminate it as irrelevant to this inquiry, we acquired the reference.

Additionally, we separately searched the websites of the U.S. Geological Survey, EPA, states, U.S. Department of Health and Human Services, and the International Union for the Conservation of Nature (IUCN) for documents and data that identified potential effects of cyanide on marine, estuarine, and freshwater ecosystems and the individuals that inhabit these ecosystems. We conducted searches of EPA's Toxics Release Inventory (TRI) and Storage and Retrieval (STORET) databases for water quality data to identify areas where discharges are monitored for cyanide, and to characterize the general patterns of known occurrence and reported values over time and space.

From these documents we extracted the following: when the information for the study or report was collected, the study design, which species the study gathered information on, the sample size, the form of cyanide associated with the study, whether the study was conducted in a controlled laboratory environment or *in situ* (in the field or natural environment), whether other stressors were associated with study, study objectives, and study results. There is some concern that the exposure concentration and response observed in some studies on cyanide may not be accurate or reliable given differences between the analytical methods used, and forms of cyanide studied. Therefore, we followed a similar classification scheme as developed by Gensemer et al. (2007) to make comparisons among the type of cyanide exposure measurements performed in the studies. We classified studies according to whether they measured: (1) free cyanide using a reliable test method (e.g., ASTM 4282-95); (2) measured free cyanide but the test method accuracy is unknown; (3) measured weak acid dissociable cyanide; (4) measured total cyanide, and provided an estimate of free cyanide; (5) measured total cyanide, but did not estimate free cyanide; (6) did not provide an analytical verification of the cyanide concentration. Within each class of studies, we ranked each of the studies based on the quality of their study design, sample sizes, level of scrutiny before and during publication, and study results. We ranked carefully designed experiments (for example, experiments that control potentially confounding variables) higher than experiments that were not designed to control potentially confounding variables. We ranked carefully designed experiments higher than computer simulations, and we ranked studies on the response of listed species higher than studies on other, non-listed species. We also ranked studies that produced large sample sizes with small variances higher than studies with small sample sizes or large variances. Articles that did not rely on evidence produced by controlled experiment, uncontrolled field experiments, opportunistic observations of animal behavior or

computer simulation received the lowest rating, but we considered the arguments and conclusions within these articles within our analyses.

Application of this Approach in this Consultation

The EPA proposes to continue approving state and tribal water quality standards for cyanide, which are based on their recommended 304(a) aquatic life criteria that were developed and published in the 1980s under the authority of the CWA. Section 304(a) of the CWA, the goals and purposes of the CWA, the implementing regulations for water quality standards (40 CFR 130-131), and the *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (later referred to as "the *Guidelines*"; Stephan et al. 1985) form the foundation, or the standards that the cyanide criteria were designed to meet. This Opinion represents NMFS' evaluation of whether EPA's approval of state or tribal water quality standards, or federal water quality standards promulgated by EPA, that are identical to or more stringent than the section 304(a) aquatic life criteria for cyanide satisfies EPA's obligations pursuant to section 7(a)(2) of the ESA of 1973, as amended.

NMFS' evaluation proceeds by asking if the approval of cyanide consistent with (or more stringent than) the 304(a) aquatic life criteria for cyanide proposed by EPA is likely to prevent the exposure of endangered species, threatened species, and designated critical habitat from aqueous cyanide concentrations that are toxic, given the approach it uses to approve water quality standards? If listed resources are not likely to be exposed to the direct and indirect effects of cyanide from activities the water quality standards would authorize, both individually and cumulatively, given the approach EPA uses to approve a water quality standards, we would conclude that EPA's proposal to continue recommending the 304(a) aquatic life criteria for cyanide is not likely to jeopardize the continued existence of endangered species, threatened species, or result in the destruction or adverse modification of designated critical habitat under NMFS' jurisdiction. If, however, listed resource are likely to be exposed to the direct and indirect effects of cyanide from activities the water quality standards would authorize, both individually and cumulatively, we would ask whether and to what degree listed species are likely to respond to their exposure, given the approach EPA uses to approve a water quality standards. As part of this analysis, we would examine whether and to what degree EPA has identified chemical, physical and biological scenarios that influence cyanide toxicity and presence in the environment inhabited by listed species and their critical habitat, the nature of any in situ effects, and the consequences of those effects for listed resources under NMFS' jurisdiction, to determine if EPA can insure that the approval of state and tribal water quality standards that they are proposing is not likely to jeopardize the continued existence of endangered species or threatened species, or result in the destruction or adverse modification of critical habitat that has been designated for these species.

Understanding the Water Quality Program

EPA has asked that the Services consult on their approval of water quality standards where states and tribes adopt the standards that are consistent with or more stringent than the nationally recommended 304(a) aquatic life criteria. Since our analysis must consider the direct and indirect effects of the action together with the direct and indirect effects of any interdependent

and interrelated actions[3], a critical first step to any consultation is determining whether and to what extent there are actions interrelated and interdependent with the action under consultation.

While EPA's BE does not examine interrelated and interdependent actions, it did provide us partial insight into the issue of what EPA considers interrelated and interdependent actions, inasmuch as EPA highlighted the general protective measures that states may adopt as part of their water quality programs as further evidence that listed resources would rarely, if ever, be exposed to cyanide at the recommended criteria values. Since the action as EPA has described it in its BE and subsequent documents, is the approval of water quality standards that states and tribes implement as enforceable standards for cyanide then it follows that the direct and indirect effects of any actions that are interrelated or interdependent with that approval must be considered in this consultation.

We developed a simple conceptual model to illustrate our understanding of the overall water quality program, and to assist us in determining whether there are actions that are interrelated or interdependent to the EPA's recommended 304(a) aquatic life criteria and subsequent approval of cyanide standards when states and tribes adopt the recommended numeric values. In part, we were interested in exploring the relationships among program components and EPA's decision to approve a particular standard and, specifically, whether the protective measures described in the BE and imposed by states and tribes should be considered in this consultation as interrelated and interdendent with the action to approve.

Our model depicts the relationship between EPA's 304(a) aquatic life criteria and other components of EPA's water quality-based approach to pollution control (Figure 1). Figure 1 also illustrates those relationships between "any action authorized, funded or carried out by" EPA under the composite program and section 7(a)(2) of the ESA. The model is based on the discussion of the water quality-based approach to pollution control, and the interrelated parts of executing the CWA as it was described by EPA in the *Water Quality Standards Handbook* (EPA 1994), information on the program characteristics that were provided by EPA in the cyanide BE, and is also based on our prior experiences with state water quality standards and NPDES permits issued by states and EPA. Our model, as with any descriptive model, represents a simplified map of the characteristics of the larger water-quality based pollution control program.

The goals and policies of the CWA establish the foundation for EPA's pollution control program. Pollution control begins, in part, with the identification of a target or priority pollutant and EPA's decision to "develop and publish" … (and from time to time thereafter revise) 304(a) criteria for water quality for that particular pollutant. EPA derives 304(a) aquatic life criteria through an established decision-making process outlined by the *Guidelines* (Stephan et al. 1985), which we depict at the top of Figure 1. Upon deriving a numeric value for a pollutant, EPA recommends (publishes) the numeric value for adoption and implementation. Publication typically involves a draft stage and a final stage in between which EPA solicits public comments.

The national aquatic life criteria provide the foundation for a wide variety of programs aimed at

[3] Interdependent actions are those actions that have no independent utility apart from the action under consideration. Interrelated actions are those actions that are part of a larger action and depend upon the larger action for their justification (50 CFR 402.02).

addressing pollution control under the CWA. EPA's 304(a) aquatic life criteria serve as guidelines or recommendations to states and tribes for defining water column concentrations of cyanide that EPA expects would protect against adverse ecological effects to aquatic life in fresh and salt water. The 304(a) aquatic life criteria recommendations are calculated to protect aquatic organisms from unacceptable toxicity during acute (short) and chronic (long) exposures in the water column. The intent is to define a level in the waterbody of a pollutant that will be fully protective of the designated uses of a water body and that a state or tribe may use in adopting its regulatory water quality standards and achieve the goals of their waterbodies (BE page 11, 40 CFR 131.2). States and tribes may use the 304(a) aquatic life criteria as a basis for developing enforceable water quality standards. The CWA requires all states to adopt water quality standards to restore and maintain the physical, chemical, and biological integrity of the Nation's waters. The CWA allows that states with an approved water quality program may adopt the 304(a) criteria as an enforceable standard (in combination with other relevant program elements), or they may modify the recommended criteria to reflect site-specific conditions, or create unique water quality standards (40 CFR 131.11(b)).

The focus of our national consultation with EPA, are those instances where a state or tribe "adopts" a water quality standard that is consistent with the recommended aquatic life criteria. In Figure 1, the consultation on this national approval is depicted by the yellow box, "National Section 7 Consultation".

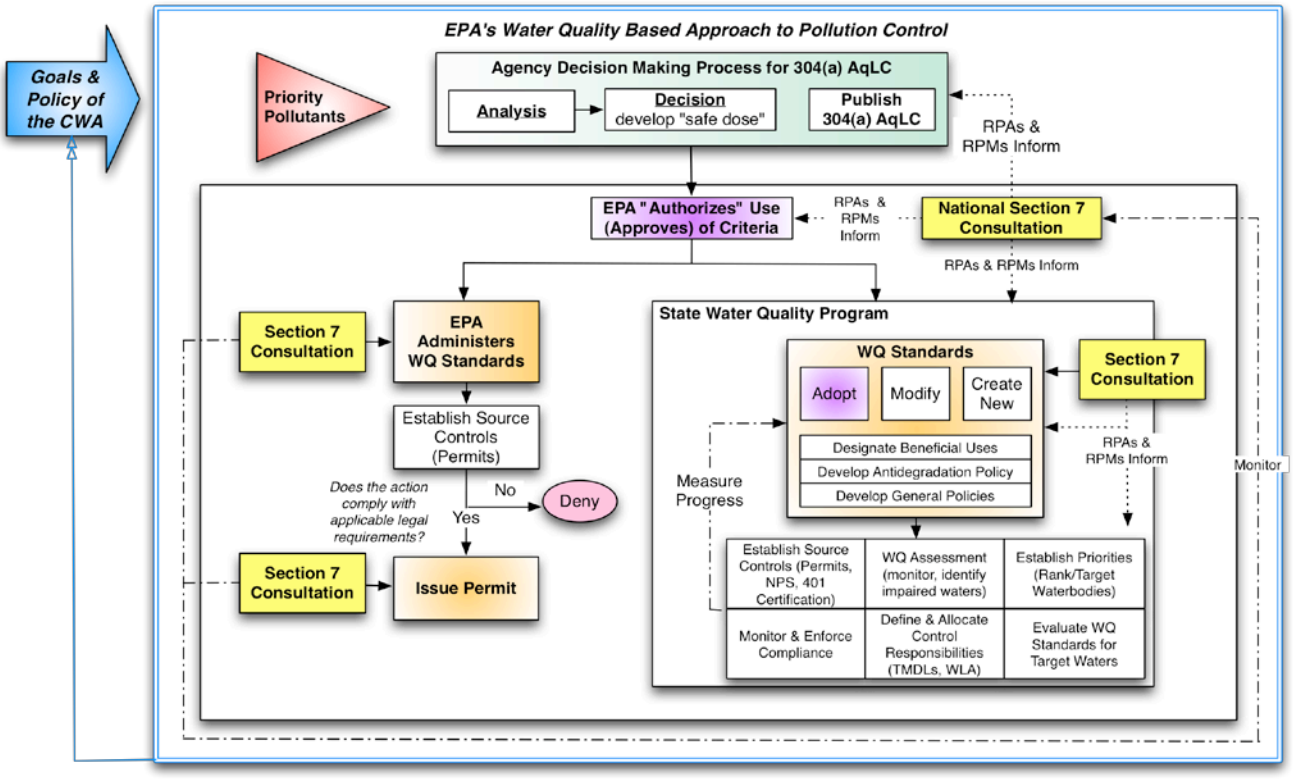

Figure 1. EPA's 304(a) aquatic life criteria and its relationship to the water quality-based pollution control program and section 7.

An approved standard, however, is more than just a numeric value for pollutants. Rather "a water quality standard defines the water quality goals of a water body, or portion thereof, by designating the use or uses to be made of the water and by setting the criteria necessary to protect the uses. States adopt water quality standards to protect public health or welfare, enhance the quality of water and serve the purposes of the Clean Water Act….. Such standards serve the dual purposes of establishing the water quality goals for a specific water body and serve as the regulatory basis for the establishment of water-quality based treatment controls and strategies….. (40 CFR 131.2)." A state's water quality program contains eight general parts with specific regulatory requirements and guidance. We included the eight general parts of a state's water quality program on the right side of Figure 1. The eight parts are described by EPA (1994) as follows:

Establish protection levels. EPA's approach to pollution control begins with the identification of problem water bodies, and the water quality standards establish the assessment goals, and the water body uses intended for protection. Standards are not simply a numeric pollutant threshold level, but standards consist of three main elements (1) designated beneficial uses of a waterbody or segment of a waterbody (e.g., protection of aquatic life, recreation), (2) water quality criteria necessary to protect the use or uses of that particular waterbody (expressed in either numeric or narrative form[4]), and (3) an antidegradation policy. Additionally, states, at their discretion, may adopt general policies in their standards affecting the application and implementation of standards (e.g., mixing zone policies, variance policies, critical flow policies for permit based-limits).

Water quality assessments. Once water quality standards are adopted, states conduct water quality monitoring to identify those waters that are "water quality limited" or not meeting standards. Monitoring is important to evaluating whether designated uses are attained, determining whether Total Maximum Daily Limits (TMDL) are needed, and assessing compliance with permits and so on. Under section 305(b) of the CWA states are required to prepare a water quality inventory every two years to document the status of assessed water bodies. At this point the state may make a determination that the water body is not impaired but that the condition is due to natural conditions.

Establish priority waterbodies. When waters are identified that don't meet standards or are water quality limited, a state is expected to prioritize (rank) waterbodies for TMDL development.

Evaluate water quality standards for target waters. At this point in the water quality management process, States have targeted priority water quality-limited water bodies. EPA recommends that States re-evaluate the appropriateness of the water quality standards for the targeted waters if: 1) States have not conducted in-depth analyses of appropriate uses and criteria; 2) changes in the uses of the water body may require changes in the standard; 3) more recent water quality monitoring show the standard is being met; and, 4) site-specific criteria may be appropriate because of specific local environmental conditions or the presence of species more or less sensitive than those included in the national criteria data set.

[4] States must adopt numeric standards for toxic pollutants listed pursuant to section 307(a)(1) of the CWA and for which criteria have been published under 304(a).

1. *Define and allocate control responsibilities.* For water quality limited waters, States must establish a total maximum daily load (TMDL) that quantifies pollutant sources, and a margin of safety, and allocates allowable loads to the contributing point and non-point source discharges so that the water quality standards are attained. EPA recommends States develop TMDLs on a watershed basis.

2. *Establish source controls.* Source loads of pollutants are controlled through the TMDL, waste load allocations (WLA), best management practices (BMPs), and through the technology-based and water quality-based controls implemented through the NPDES permitting process. Although, many states and territories have authority to implement at least a portion of the NPDES program in their jurisdiction, EPA retains full or partial authority in many states and territories. In the case of nonpoint sources, both State and local laws may authorize the implementation of nonpoint source controls, such as best management practices (BMPs) or other management measures.

Monitor and enforce compliance. Monitoring is critical to the water quality-based decision making, and includes assessing compliance with TMDLs, permits, as well as in water loading (necessary to also capture nonpoint source pollution loads) and attainment of water quality goals. Point source dischargers are required to provide reports on compliance with NPDES permit limits. A monitoring requirement can be put into the permit as a special condition as long as the information is collected for the purposes of writing a permit limit. Effective monitoring programs are also required for evaluating nonpoint source control measures and EPA provides guidance in implementing and evaluating nonpoint source control measures. EPA and States are authorized to bring civil or criminal action against facilities that violate their NPDES permits. State nonpoint source programs are enforced under State law and to the extent provided by State law.

Measure progress. Arguably, one of the most important elements of the overall program are the efforts by the states (and EPA) to assess the effectiveness of the controls and standards, to determining water quality standards need to be revised, or more stringent controls are necessary (e.g., through permits or WLA and TMDLs). This is particularly important in determining whether a water body on the 303(d) list of impaired waters achieves water quality standards and can be removed from the state's 303(d) list, or to determine if WLA must be modified. This element is depicted as a feedback arrow between the general program elements and the foundation of state programs, the numeric standards and the policies that govern the program execution.

The left side of Figure 1 depicts those aspects of the water quality-based approach to pollution control that are approved and carried out directly by EPA. Criteria developed, published and approved by EPA are the foundation for many actions administered by EPA, including the promulgation of national water quality standards, and the issuance of NPDES permits.

Figure 1 also illustrates a general need by EPA to consult on actions that EPA "approves, funds, and carries out" under the program, which includes nationally approved criteria, as well as the approval of new state standards and the triennial review of those standards, and EPA's issuance of NPDES permits. The scope and details of such consultations depend upon EPA's

discretionary control or authority to insure that its decisions on these actions comply with the CWA, its implementing regulations and policies. The yellow boxes in Figure 1 generally depict those areas where EPA would consult with the Services on their actions.

The consultation boxes in Figure 1 are linked to the national consultation to illustrate that NMFS will use the evidence obtained in regional and site-specific consultations to determine whether a particular consultation produced the expected results or produced results that were not consistent with the assumptions and conditions of the national consultation. That is, this first national consultation establishes a feedback framework to assist NMFS in assessing (a) the reliability, validity, or relevance of any evidence it relied upon in its national consultation; (b) whether the national consultation produced the anticipated results or produced results that were not consistent with subsequent consultations, (c) assessing the current status of any reasonable and prudent alternatives, reasonable and prudent measures, terms and conditions, and reporting requirements that EPA must comply with under the national consultation; and (e) the current and projected trends of listed resources, and the altered environmental baseline. The arrows in connecting these consultations in Figure 1 are broken because this is a newly developed feedback framework and has not previously been implemented by NMFS in its water quality consultations with EPA.

Interrelated and Interdependent Actions

The effects of EPA's 304(a) criteria recommendation must be understood in the larger context of the CWA. This larger context is framed by Congress' stated objective, goals, and policies of the CWA, and the programs and activities authorized by the CWA and implemented by EPA, and states and tribes to achieve these objectives, goals and policies. It is the CWA requirement that all states adopt water quality standards to restore and maintain the physical, chemical, and biological integrity of the Nation's waters that places the standards at the core of the overall strategy for water-quality based pollution control. As described previously, standards serve as the regulatory basis for the water quality-based approach to pollution control and are used to identify water quality problems caused by various land uses, such as improperly treated wastewater discharges, runoff or discharges from active or abandoned mining sites, sediment, and so on.

As a practical matter most states and tribes adopt EPA's recommended 304(a) criteria for most pollutants as part of their water quality standards even though they can develop unique criteria for their waters (EPA 1999). According to a review of state water quality criteria for cyanide, we found that more than 80% of the states and territories adopted EPA's acute and chronic freshwater criteria for cyanide or criteria that were more stringent[5] (Appendix A). Eleven states (Arizona, Arkansas, California, Iowa, Louisiana, Nebraska, Ohio, Oklahoma, Texas, Washington, and Wisconsin) adopted higher values in their standards, some significantly so. Some of these states adopted different values for cold waters versus warm waters (e.g., Arizona) or specified particular areas subject to these different values (e.g., Washington, California). States that set significantly higher standards than EPA's nationally recommended 304(a) criteria included Iowa, Louisiana, Ohio and Texas. No states set lower salt water values than EPA

[5] We interpreted "more stringent" to be a lower value that would lead to less cyanide in the water. Most states and territories that had set lower standards for cyanide were only a few tenths to hundredths lower than the value recommended by EPA.

recommended, but a few established higher values. California established levels as high as 10.0 µg/L CN for the saltwater instantaneous maximum and Texas set their chronic and acute saltwater criteria at 5.6 µg/L CN. Local exemptions in some state waters are much higher than these broader state limits. For instance, Illinois allows for 100 µg/L for acute exposure and 1,000,000 µg/L in some waterways in Cook County (home to Chicago). Although several states adopted new standards that differ from EPA's recommended values, the fact that most states follow EPA recommendations for cyanide verbatim illustrates the influence that EPA's guidance has on state standards. We suspect that EPA's action to develop and publish (recommend) 304(a) aquatic life criteria likely is sufficient to dissuade many states from investing the resources to develop unique water quality standards, particularly in times of economic hardship and reduced state budgets.

That the CWA creates an independent statutory requirement that states adopt enforceable water quality standards is sufficient reasoning to support the argument that state standards have "independent utility" and would not generally be considered interdependent with EPA's 304(a) criteria. However, since the vast majority of states adopt the 304(a) criteria as developed and published by EPA, and EPA has requested that this consultation, programmatically, address their need to consult on their approval of the water quality standards that are consistent with, or more stringent than the 304(a) recommended criteria the argument of independent utility is moot. That is, it is EPA's expectation that this national consultation address their general action to approve any state or tribal water quality standards for cyanide that are consistent with, or more stringent than the numeric value they recommend, and by doing so EPA hopes to eliminate subsequent regional consultations on water quality standards. .

As we described earlier, the level of protection afforded to a water body under the CWA is defined by the sum of the designated uses, criteria, antidegradation policy[6], and general policies. While all are required in a state submission, the designated uses and criteria are particularly inseparable components of a water quality standard as evidenced by EPA's language on approving a submission. That is, to approve a proposed water quality standard EPA must find that a state has adopted uses that are consistent with the requirements of the CWA and that adopted criteria protect those designated uses. EPA cannot approve a numeric value for a particular pollutant, like cyanide, if that numeric value does not support the uses the state has designated for a particular water body. The designated uses are integral to the approval and have no independent utility apart from the approval of a water quality standard, but are one of the most important parts of a water quality standard. More so, a water quality standard, by definition, is not complete without a finding that a particular criterion meets the designated uses. Therefore, designated uses are also interrelated with a particular criterion value because they are integral parts of the standard (part of the larger action), and depend upon the larger action for their justification.

[6] In a January 27, 2005, memorandum to it Regional Offices, EPA concluded that ESA section 7 consultation does not apply to EPA's approvals of state antidegradation policies because EPA's approval action does not meet the "Applicability" standard defined in the regulations implementing section 7 of the ESA (EPA 2005; 50 CFR 402.03). Section 402.03 of the consultation regulations (50 CFR part 402) states that section 7 and the requirements of 50 CFR part 402 apply to all actions in which there is discretionary Federal involvement or control. EPA concluded that they are compelled to approve State antidegradation policies if State submissions meet all applicable requirements of the *Water Quality Standards Regulation* (40 CFR part 131) and lack discretion to implement measures that would benefit listed species. As a result, EPA determined that consultation is not warranted on antidegradation policies because the Agency does not possess the regulatory authority to require more than the minimum required elements of the regulations.

When we embarked on this evaluation, however, we noted we were particularly interested in determining whether the protective measures described in the BE and imposed by states and tribes should be considered in this evaluation. EPA stated that states and tribes may, in addition to adopting numeric criteria, adopt: narrative criteria, biological criteria, or site-specific criteria for cyanide. EPA also noted that during implementation of their water quality standards, several other assumptions are made when allocating pollutants, for permitting purposes, among point source discharges to protection of species. As part of the TMDL and NPDES permit development, according to EPA most states and tribes use the following protective assumptions in the development of their TMDLs and water quality based effluent limitations: (1) assume that all dischargers are discharging the contaminant at the maximum permitted levels, (2) provide for an unallocated "margin of safety" when developing TMDLs, (3) assume the maximum permitted discharge volume, (4) assume the maximum concentration of loading of pollutants, (5) assume no environmental degradation of pollutants, (6) assume all discharged pollutants remain biologically available, (7) assume receiving stream flows are very low, (8) assume that acute toxicity limits apply at the "end of the pipe", (9) assume that only a portion of the design flow is available for mixing for controls on chronic toxicity, (10) assume that aquatic species live continuously at the "edge of the mixing zone", (11) assume no internal dilution of process wastewater, (12) assume conservative values for upstream concentrations of pollutants, (13) permit conditions should not be relaxed in subsequent permit reissuance (antibacksliding), (14) antidegradation requirements protect existing uses, (15) assume low threshold for "reasonable potential" if few data are available. While we cannot disagree that these components of a state's water quality program warrant further examination, and may even qualify as interrelated and interdependent actions that demonstrate the success (or failures) of various specific programs and the success of the overall water quality program, the BE provided no evidence of the general patterns of the implementation of these measures, nor an evaluation of the success or failures of these protective mechanisms across the national landscape. We further acknowledge that each TMDL, WLA, and NPDES could in fact be considered actions interdependent to EPA's approval of a state's water quality standards because the standards and goals for a water body "serve as the regulatory basis for the establishment of water-quality based treatment controls and strategies (40 CFR 131.2)."

Perhaps the most compelling reason that the above mentioned actions and other general program operations have independent utility, however, is the fact Congress intentionally divided many of these state and tribal actions into different sections of the CWA. In fact much of the statute directs the actions of state and tribes, not EPA's, supporting state autonomy for the protection of their waters. That the sections were designed to work together to achieve the goals set forth by Congress should not be a surprise, and in of itself should not be reason to consider all programs that rely on the water quality standards as interrelated or interdependent to the approval of water quality standards. Thus we default to the statutory construct, and the distinctive sections of the Act that instruct states and tribes on the execution and operation of their overall water quality program, as providing the strongest argument for independent utility.

Moveover, we note that the inclusion into this consultation of the myriad of such actions as dictated by the different programs that rely on water quality standards would easily make this national consultation untenable in short order. Thus, unless we can establish evidence of the general pattern in which the protective measures EPA noted in their BE are implemented across

states and tribes (information which was not contained within the BE) then these assertions served little relevance to our analysis on the national scale. We further submit that individual NPDES permits, TMDLs, WLA, and other management aspects of a state's water quality program, while emanating from EPA's approved water quality standards, merit evaluation in subsequent consultations, where appropriate. Where EPA does not retain discretion, and such actions may affect listed resources, then states and tribes ought to seek a permit from the Services pursuant to section 10(a)(1)(b) of the ESA. We therefore propose that while each of the actions that are part of the overall approach to protecting aquatic life in waters of the United States are targeted to assessing compliance with standards and instituting change to achieve compliance through modification to allowable discharges or to the standards themselves, they have independent and significant roles in achieving the goals of the CWA. Consequently, they merit separate reviews as appropriate under the ESA. Such separate reviews can be linked through our conceptual model feedback links (see Figure 1), to assist NMFS and EPA in conducting holistic review of the effectiveness of the programs for protecting listed resources.

What we cannot separate on the basis of independent utility, however, as they are intimately a part of the action as EPA has proposed it, are the elements of a state or tribal water standard that must be included in each submittal to EPA for review and in order for EPA to approve said standard (see EPA 1994). As established in the foregoing discussion, these include: designated uses, criteria, antidegradation policy, and general policies. Hence, we address these other components as they are an essential part of any standard EPA approves, as interrelated and interdependent actions to EPA's approval of a numeric pollutant value in a state or tribal standard. These interrelated and interdependent actions are discussed in the *Effects of the Action* section of this Opinion.

Water Quality Standards

Water quality standards, as mentioned previously, are the mechanism by which protection levels for a water body are established. The water quality standards establish the assessment goals (e.g., numeric or narrative criteria), and the water body uses intended for protection. Whenever a state revises or adopts a new water quality standard such revised or new standard must be submitted to EPA for review. The water quality standard must include designated uses consistent with the provisions of section 101(a)(20 and 303(c)(2) of the CWA, the methods used and analyses conducted to support water quality standards revisions, water quality criteria sufficient to protect the designated uses, an antidegradation policy, certification that the water quality standards were duly adopted pursuant to state law, and general information that will aid the EPA in determining the adequacy of the scientific basis of the standards (40 CFR 131.6).

According to the CWA, the standards shall protect the public health or welfare, enhance the quality of water and serve the purposes of the Act, and shall be established taking into consideration their use and value for public water supplies, propagation of fish and wildlife, recreational purposes, and agricultural, industrial, and other purposes, and also taking into consideration their use and value for navigation. The phrase to "serve the purposes of the Act" as defined in 303(c) of the CWA, means that the water quality standards should meet the objectives of the Act "to restore and maintain the chemical, physical, and biological integrity of the Nation's waters." In order to achieve this objective Congress declared that---

(1) It is the national goal that the discharge of pollutants into the navigable waters be eliminated by 1985;

(2) It is the national goal that where ever attainable, an interim goal of water quality which provides for the protection and propagation of fish, shellfish, and wildlife and provides for recreation in and on the water be achieved by July 1, 1983;

(3) It is the national policy that the discharge of toxic pollutants in toxics amounts be prohibited…."

These three goals, which are commonly referred to as the "zero discharge" goal, "the fishable/swimmable" goal, and the "no toxics in toxic amounts" goal, are accompanied in the statute by a number of subsidiary goals and policies (Adler et al. 1993). Water quality standards for aquatic life are primarily designed to meet the fishable/swimmable goal of the CWA.

Water quality standards (in particular, the numeric criteria coupled with a water body's designated uses) are the core mechanism for meeting the goal of the CWA, and "getting water quality standards right starts with getting designated uses right (EPA 2008a)." When a state submits a water quality standard, EPA must review and approve (or disapprove) a standard based upon whether a state has: (a) adopted uses that are consistent with the requirements of the CWA, (b) adopted criteria that protect the designated uses, (c) followed legal procedures for adopting standards, (d) whether the submission meets the regulatory requirements (40 CFR 131.5). In specifying appropriate water uses, each state must take into consideration the protection and propagation of fish, shellfish and wildlife, and recreation in and on the water (the "fishable/swimmable" goal among other things; 40 CFR 131.10(a)), whether or not a use is currently being attained.

Designated Uses

Designated uses are statements of management objectives and expectations for water bodies under state or tribal jurisdiction. As defined in 40 CFR 131.3, designated uses are specified in the water quality standards for each water body or water body segment regardless of whether or not they are being attained. Designated uses include, but are not limited to: water supply (domestic, industrial and agricultural); stock watering; fish and shellfish uses (salmonid migration, rearing, spawning, and harvesting; other fish migration, rearing, spawning, and harvesting); wildlife habitat; ceremonial and religious water use; recreation (primary contact recreation; sport fishing; boating and aesthetic enjoyment); and commerce and navigation.

The water quality standards regulation requires that states and tribes specify which water uses are to be achieved and protected. These uses are determined by considering the value and suitability of water bodies based on their physical, chemical, and biological characteristics as well as their geographical settings, aesthetic qualities and economic attributes. Each water body does not necessarily require a unique set of uses. Rather, water bodies sharing characteristics necessary to support a use can be grouped together. If water quality standards specify designated uses of a lower standard than those that are actually being attained, the State or Tribe is required to revise its standards to reflect these uses.

Antidegradation

Antidegradation implementation procedures identify the steps and questions that must be addressed when proposed activities may affect water quality. The water quality standards regulation requires that states and tribes establish a three-tiered antidegradation program. The specific steps to be followed depend upon which tier or tiers apply. These tiers are listed below:

- Tier 1: These requirements are applicable to all surface waters. They protect existing uses and water quality conditions necessary to support such uses. These uses can be established if they can be demonstrated to have actually occurred since November 28, 1975, or if water quality can be demonstrated to be suitable for such uses. If an existing use is established, it must be protected even if it is not listed in the water quality standards as a designated use.

- Tier 2: These requirements maintain and protect "high quality" water bodies where existing conditions are better than those necessary to support CWA § 101(a)(2) "fishable/swimmable" uses. Although the water quality in these water bodies can be lowered, states and tribes must identify procedures that must be followed and questions that must be answered before a reduction in water quality can be allowed. The water quality of these water bodies cannot be lowered to a level that would interfere with existing or designated uses.

- Tier 3: These requirements maintain and protect water quality in outstanding national resource waters (ONRWs) and generally include the highest quality waters of the United States. ONRW classification also offers special protection for waters of exceptional ecological significance. Except for certain temporary changes, water quality cannot be lowered in these waters. states and tribes decide which water bodies qualify as ONRWs.

In a January 27, 2005, memorandum to it Regional Offices, EPA concluded that ESA section 7 consultation does not apply to EPA's approvals of state antidegradation policies because EPA's approval action does not meet the "Applicability" standard defined in the regulations implementing section 7 of the ESA (EPA 2005; 50 CFR 402.03). Section 402.03 of the consultation regulations (50 CFR Part 402) states that section 7 and the requirements of 50 CFR part 402 apply to all actions in which there is discretionary Federal involvement or control.

EPA concluded that they are compelled to approve State antidegradation policies if State submissions meet all applicable requirements of the *Water Quality Standards Regulation* (40 CFR part 131) and lack discretion to implement measures that would benefit listed species. As a result, EPA determined that consultation is not warranted on antidegradation policies because the Agency does not possess the regulatory authority to require more than the minimum required elements of the regulations. For these reasons, antidegradation will not be a part of this consultation.

General Policies

States and tribes may adopt general policies and provisions regarding the implementation of water quality standards. These policies and provisions are subject to EPA review and approval. General policies must relate to designated use criteria or antidegradation. These policies and provisions include:

1. Mixing Zones: A mixing zone is a defined area surrounding or downstream from a point source discharge where the effluent is diluted by the receiving water and criteria otherwise applicable to the water body may be exceeded. At their discretion, states and tribes may allow mixing zones for point source discharges. Mixing zone procedures describe the methodology for determining the location, size, shape, and quality of mixing zones.

2. Variances: Variances temporarily relax a water quality standard. They are subject to public review every three years and may be extended. A variance may specify interim water quality criteria applicable for the duration of the variance. States or tribes may wish to include a variance as part of a water quality standard as an alternative to removing a designated use. Variances are intended to help assure that further progress toward improving water quality is achieved.

3. Low Flows: State and tribal water quality standards may identify policies and procedures to determine critical low flow conditions. For example, such procedures are applied when calculating discharge requirements to be included in National Pollutant Discharge Elimination System (NPDES) permits.

Evaluating Exposure at the National Level

The next step in our analysis involved evaluating the contaminant, cyanide (the stressor), in the environment in which the listed resources occur. Although we searched, we simply could not find sufficient data to conduct a quantitative assessment of the likelihood of exposure, or the likelihood of exposure at a particular numeric value. Therefore, our analysis focuses largely on the consequences of an exposure at criterion value. However, to examine a species' (and their critical habitat's) risk of exposure, we searched for evidence that would help us describe the (1) the transport, fate, and persistence of cyanide in the environment, (2) the distribution of uses and occurrences of cyanide across the U.S., and (3) temporal and spatial changes, where we could find evidence of these changes, across the U.S.

We began by constructing a simple conceptual model for evaluating the effects of contaminants on listed species and critical habitat. This model depicts the release of a contaminant into the environment, its transport through the environment and its contact with the listed species (Figure 2). The fate of pollutant, and whether it reaches habitats containing listed species, depends upon a wide number of variables including chemical form and structure, volume dispersed and the manner in which it is dispersed, distance of travel, and processes of sorption, degradation, and dilution, to name a few.

In describing the basic properties of cyanide, we also looked at chemical, biological and physical attributes in the environment that might act as "filters" or "magnifiers" that influence the relationship between cyanide and the induction of effects on listed species. For instance, Cloern (2001) used tidal energy to illustrate the importance of filters in eliciting certain responses within an ecosystem—tidal energy influences turbulent kinetic energy and mixing in shallow waters, and ultimately the expression of eutrophication. Differences in tidal amplitude are one mechanism by which different estuaries will respond dissimilarly to equally high loads of

nutrients, and in turn the filters acting within different ecosystems would dictate potentially very different pollutant concentrations to which listed species would be exposed.

Some of the particular features of an ecosystem or site that can act as filters, influencing the nature, magnitude, and spatial and temporal distribution of pollutants to which listed species may be exposed include: water hardness, pH, precipitation, wind, light, bathymetry, stratigraphy, topography, trace gas absorption, mineral weathering, elemental storage ability, soil chemical processes, microbial transformation, and so on. For site-specific assessments, as much as possible, the site's features should be described and used to evaluate associations between the listed species and their critical habitat, and the particular pollutant under evaluation. At the national scale, however, we look for evidence of the types of filters that generally would be expected to interact with cyanide along its general transport pathway, and that would influence its availability, toxicity and severity.

Our simple transport model, illustrated in Figure 2, serves as a map for our analysis. That is, it illustrates the main pathways— the physical course cyanide generally takes from the source to the receptor organism or communities of interest (Suter et al. 2002). For section 7 evaluations of pollutants, the receptor organism is the listed species or designated critical habitat. An exposure pathway is complete when the chemical(s) under evaluation reach the receptor organism. A pathway is incomplete when the stressor does not reach the organism under evaluation. Simply, in the latter case when the pathway is incomplete, the chemical does not co-occur with the listed species or its designated critical habitat.

Our conceptual transport model emphasizes the exposure route through surface waters because the primary route of exposure to chemical contaminants for most of NOAA's trust resources will often be through water-borne exposures. As with any conceptual model, this visual depiction of exposure pathways is a simplified representation of what can be expected in the natural environment. For instance, not only would some species be exposed to surface water contaminants, animals that live portions of their life cycle out of water like many marine mammals (aquatic-dependent species) may be exposed to contaminants on land. Even wholly aquatic species, like salmon may be exposed to contaminants in terrestrial vegetation—through leaf litter and insects (allochthonous stream input)—and contaminated soils that enter the aquatic environment.

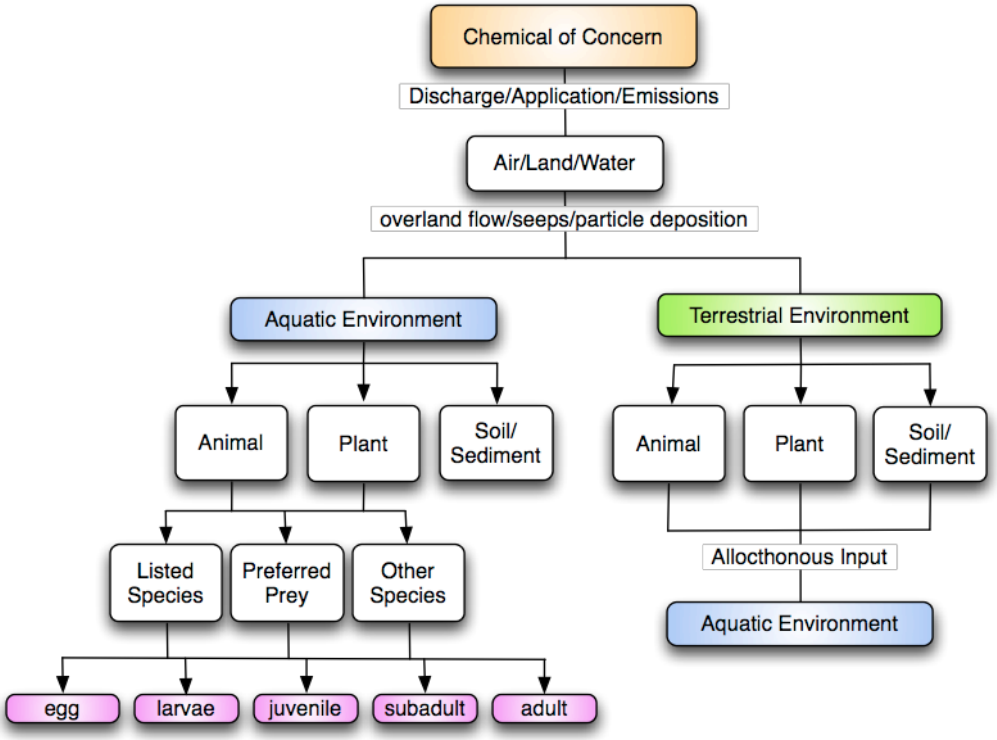

Figure 2. Simple transport model depicting pollutant pathways to aquatic habitats and aquatic species.

We would consider an exposure pathway complete when the chemical under evaluation would generally be expected to reach the listed species and incomplete when the stressor does not reach the listed species. Often the more difficult aspect of a section 7 evaluation is identifying the indirect pathways by which a listed species or their critical habitat is affected by a chemical stressor, which requires an examination of relationship of the listed species to the communities of which it is a part, and the environment in which it resides, depends upon, and is adapted. To capture indirect exposure pathways we look at the relationship of the listed species to the community and environment in which it lives. This means, that not only do we look for effects of cyanide directly acting on the listed species, we examine the effect that cyanide has on the biological community and environment in which the listed species lives (Figure 3). We do this to determine if cyanide would induce community and environmental changes that would likely affect the listed species, such as changes in prey availability or health.

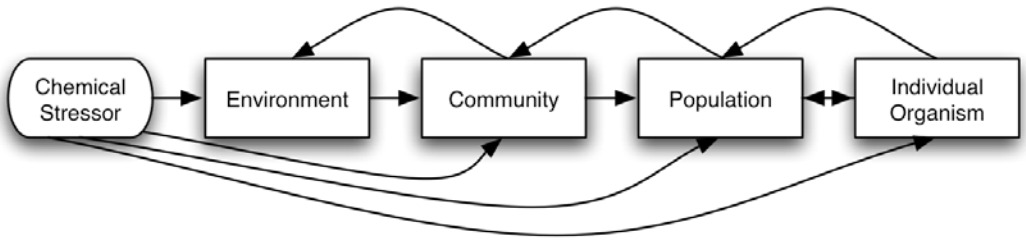

Figure 3. A chemical stressor and its potential relationships with organisms in the wild

Our challenge in this step is to identify: what populations, life history forms or stages are exposed to the proposed action; the number of individuals that are exposed; the pathways of their exposure; the timing and duration of their exposure; the frequency and magnitude of the concentrations of the exposure; and how exposure might vary depending upon the characteristic of the environment and individual behavior. Typically, in this step of our analysis we would identify how many individuals are likely to be exposed, which populations the individuals represent, where and when the exposure would occur, how long the exposure would occur, the frequency of the exposure, and any other particular details that help characterize the exposure. To do this we require knowledge of a species' population structure and distribution, migratory behaviors, life history strategy, and abundance.

All of the species under NMFS' jurisdiction are "aquatic" or "aquatic-dependent", meaning that at least one or more life stages are aquatic and could potentially be exposed to aqueous pollutants. Therefore, since EPA has asked that this consultation cover their national approval of standards that are consistent with their recommended aquatic life criteria, we began our assessment with the basic assumption that all of the listed species and critical habitat under NMFS' jurisdiction, as well as any species proposed for listing and critical habitat proposed for listing, would potentially be exposed to cyanide at the recommended criteria values at some time during their life cycle. NMFS assumes the recommended criteria value is an appropriate starting assumption for exposure in particular because the recommended value is assumed to represent a "safe dose" of cyanide.

Using this assumption, we asked whether and to what degree would animals that are exposed at the recommended level be protected if exposed at that value (this is part of our response analysis). Next, we asked whether and to what degree the proposed action and any interrelated and interdependent actions would mitigate, minimize or avoid allowing cyanide discharges to reach (or exceed) the recommended criteria. Because this examination is done at the national level, we looked for general patterns of cyanide where that information was available to us. We used such information as general patterns of the distribution of uses, manufacturing, and incidental occurrences of cyanide in the environment, and we looked for temporal and spatial changes in these uses to characterize the past 20 years of cyanide in the environment, and as a basis for predicting the future of cyanide in the environment across our action area. Our evaluation is explained in detail in our effects analysis.

Action Area

EPA has defined the action area for the cyanide consultation, and for the 304(a) aquatic life criteria consultations in general as all "waters of the United States" including "territorial seas" (see the BE, pages 8 and 9, and the *Methods Manual* page 6). The CWA (33 USC 1362) defines territorial seas as "the belt of the seas measured from the line of ordinary low water along that portion of the coast which is in direct contact with the open sea and the line marking the seaward limit of inland waters, and extending seaward a distance of three miles." This action area includes such waters within and surrounding Indian Country, the 50 States, and all United States territories. The terms "waters of the United States" is defined under 40 CFR Section 122.2 and reiterated in EPA's cyanide BE.

As early as 1789, the United States territorial sea was established at three nautical miles. On 27 December 1988, however, President Regan issued a proclamation that extended the United States territorial sea to 12 nautical miles from the baselines of the United States. Although, nothing in the proclamation extended or otherwise altered existing federal or State law subsequent to the 1988 proclamation, several federal laws adopted the terms of the Proclamation to define the United States territorial sea for purposes of that particular statute (e.g., the Nonindigenous Aquatic Nuisance Prevention and Control Act of 1990, the Antiterrorism and Effective Death Penalty Act of 1996). However, others, including the Federal Water Pollution Control Act (aka. the CWA) continue to use the three mile limit in its definition of the United States territorial sea.

The action area for the purposes of consultation, however, is not limited to the area of an agency's jurisdiction. Rather, in consultation the action area is defined as all areas to be affected directly or indirectly by the federal action and not merely the immediate area involved in the action (50 CFR 402.02). Many federal actions that NMFS consults on occur in the United States territorial sea, the contiguous zone, exclusive economic zone, and on the high seas. The issue of jurisdiction is relegated to the point in the Opinion at which NMFS prescribes management actions (Reasonable and Prudent Alternatives and Reasonable and Prudent Measures) for the purpose of exempting the taking of threatened and endangered species from the prohibitions of section 4(d) and 9 of the ESA. (See the section of this Opinion titled *Reasonable and Prudent Alternatives*). Consequently, the action area for EPA's 304(a) aquatic life criteria consultations includes the minimal area, as defined by the freshwater, estuarine and ocean water bodies of the United States and its territories (delineated by the CWA) and any areas the particular pollutant under consultation (in this case cyanide) is transported beyond these limits by such biotic and abiotic factors as river runoff, tidal energy, topography, stratigraphy, biota [trapping/assimilation), that may influence chemical transport processes beyond original areas of dispersion. We expect, based on the chemical processes (sources, transport, and fate) of cyanide, which are described later in this Opinion, that most of the action area for this consultation on cyanide is contained by the jurisdictional waters as outlined by the CWA. However, in certain localities we expect that conveyance systems may extend to the outer edge of this action area, or that the discharge plume may extend beyond three nautical miles. Unfortunately, we cannot identify the specific areas or conveyance systems where this may occur, and thus recognize that our action area is generally delineated according to three nautical miles extending from the United States coastline.

Since NMFS has jurisdiction over marine and anadromous threatened and endangered species, and their critical habitat, this Opinion addresses the potential effects of EPA's aquatic life criteria in a portion of the action area defined for 304(a) aquatic life criteria. Specifically, this Opinion focuses on the direct and indirect effects of the recommended criteria along the coastal regions of the United States and its territories, where listed resources under NMFS' jurisdiction occur. As such, although interior fresh waters (e.g., landlocked lakes and ponds of the midwest United States) constitute a portion of the action area for this consultation, listed resources under NMFS' jurisdiction do not occur in these areas and these portions of the action area are not considered further in this Opinion.

Status of the Species and Critical Habitat

In this section of this Opinion we describe the threatened and endangered species and their designated critical habitat that occur in the action area and may be exposed to EPA's approved aquatic life criteria for cyanide. All listed species within NMFS' jurisdiction are "aquatic" or "aquatic dependent" and may occur within portions of the action area for the recommended aquatic life criteria. NMFS has determined that the following species and critical habitat may occur within the action area for EPA's 304(a) aquatic life criteria for cyanide (Table 2).

Table 2. Species Listed as Threatened and Endangered and Proposed for listing, and their designated Critical Habitat (denoted by asterisk) in the Action Area. Double asterisks denote Proposed Critical Habitat.

Common Name (Distinct Population Segment or Evolutionarily Significant Unit)	Scientific Name	Status
Cetaceans		
Beluga whale** (Cook Inlet)	*Delphinapterus leucas*	Endangered
Blue whale	*Balaenoptera musculus*	Endangered
Bowhead whale	*Balaena mysticetus*	Endangered
Fin whale	*Balaenoptera physalus*	Endangered
Humpback whale	*Megaptera novaeangliae*	Endangered
Killer Whale (Southern Resident*)	*Orcinus orca*	Endangered
North Atlantic right whale*	*Eubalaena glacialis*	Endangered
North Pacific right whale*	*Eubalaena japonica*	Endangered
Sei whale	*Balaenoptera borealis*	Endangered
Sperm whale	*Physeter macrocephalus*	Endangered
Pinnipeds		
Hawaiian monk seal*	*Monachus schauinslandi*	Endangered
Steller sea lion (Eastern*)	*Eumetopias jubatus*	Threatened
Steller sea lion (Western*)		Endangered
Marine Turtles		
Green sea turtle (Florida & Mexico's Pacific coast colonies)*	*Chelonia mydas*	Endangered
Green sea turtle (All other areas)*		Threatened
Hawksbill sea turtle*	*Eretmochelys imbricate*	Endangered
Kemp's ridley sea turtle	*Lepidochelys kempii*	Endangered
Leatherback sea turtle* (also **)	*Dermochelys coriacea*	Endangered
Loggerhead sea turtle	*Caretta caretta*	Threatened
Olive ridley sea turtle (Mexico's Pacific coast breeding colonies)	*Lepidochelys olivacea*	Endangered
Olive ridley sea turtle (All other areas)		Threatened
Anadromous Fish		
Atlantic salmon*	*Salmo salar*	Endangered
Chinook salmon (California coastal*)	*Oncorhynchus tschawytscha*	Threatened
Chinook salmon (Central Valley spring-run*)		Threatened
Chinook salmon (Lower Columbia River*)		Threatened
Chinook salmon (Upper Columbia River spring-run*)		Endangered
Chinook salmon (Puget Sound*)		Threatened
Chinook salmon (Sacramento River winter-run*)		Endangered
Chinook salmon (Snake River fall-run*)		Threatened
Chinook salmon (Snake River spring/summer-run*)		Threatened

Chinook salmon (Upper Willamette River*)		Threatened
Chum salmon (Columbia River*)	*Oncorhynchus keta*	Threatened
Chum salmon (Hood Canal summer-run*)		Threatened
Coho salmon (Central California coast*)	*Oncorhynchus kisutch*	Endangered
Coho salmon (Lower Columbia River)		Threatened
Coho salmon (Southern Oregon & Northern California coast*)		Threatened
Coho salmon (Oregon coast*)		Threatened
Green sturgeon (Southern*)	*Acipenser medirostris*	Threatened
Gulf sturgeon*	*Acipenser oxyrinchus desotoi*	Threatened
Shortnose sturgeon	*Acipenser brevirostrum*	Endangered
Smalltooth sawfish*	*Pristis pectinata*	Endangered
Sockeye salmon (Ozette Lake*)	*Oncorhynchus nerka*	Threatened
Sockeye salmon (Snake River*)		Endangered
Steelhead (Central California coast*)	*Oncorhynchus mykiss*	Threatened
Steelhead (California Central Valley*)		Threatened
Steelhead (Lower Columbia River*)		Threatened
Steelhead (Middle Columbia River*)		Threatened
Steelhead (Northern California*)		Threatened
Steelhead (Puget Sound)		Threatened
Steelhead (Snake River*)		Threatened
Steelhead (South-Central California Coast*)		Threatened
Steelhead (Southern California*)		Endangered
Steelhead (Upper Columbia River*)		Threatened
Steelhead (Upper Willamette River*)		Threatened
Marine Invertebrates		
Black abalone	*Haliotis cracherodii*	Endangered
Elkhorn coral*	*Acropora palmata*	Threatened
Staghorn coral*	*Acropora cervicornis*	Threatened
White abalone	*Haliotis sorenseni*	Endangered
Marine Plants		
Johnson's seagrass*	*Halophilia johnsonii*	Threatened
Proposed for Listing		
Bocaccio	*Sebastes paucispinis*	Proposed Endangered
Canary rockfish	*Sebastes pinniger*	Proposed Threatened
Pacific eulachon/smelt	*Thaleichthys Pacificus*	Proposed Threatened
Spotted seal	*Phoca largha*	Proposed Threatened
Yelloweye rockfish	*Sebastes ruberrimus*	Proposed Threatened

The species' narratives that follow focus on attributes of a species' life history and distribution that influence the manner and likelihood that a particular species may be exposed to the proposed action, as well as the species potential response and risk when exposure occurs. Consequent narratives summarize a larger body of information on worldwide distribution, as well as localized movements within fresh water, estuarine, intertidal, and ocean waters, population structure, feeding, diving, and social behaviors. We also provide a brief summary of the species status and trends as a point of reference for our jeopardy determinations, which we make later in this Opinion. That is, we rely on a species' status and trend to determine whether or not an action's direct or indirect effects are likely to increase the species' probability of becoming extinct. Similarly, each species narrative is followed by a description of its critical habitat with particular emphasis on any essential features of the habitat that may be exposed to the proposed action and

may warrant special attention. Because this is a national consultation that does not consider site-specific data, we only summarize information on the geographic distribution of the species, their ecological relationship with waters of the United States, their status, and the principal threats to their survival and recovery.

Species Not Considered Further in This Opinion

Species and Critical Habitat under Joint Jurisdiction

The Services share joint jurisdiction for the management of sea turtles, gulf sturgeon, Atlantic salmon. For sea turtles, NMFS is responsible for their in-water conservation while FWS is responsible for their conservation on land. This Opinion discusses the effects of the proposed action on listed marine sea turtles and their designated critical habitat in the following section.

The Services have divided the consultation responsibilities for Atlantic salmon according to whether the federal action occurs in fresh water or estuarine or marine waters (74 FR 29344). When a federal action traverses marine and fresh waters, then the Services decide which agency will assume the lead role for consultation. For the purposes of this consultation, the FWS' Opinion addresses the effects of the action on Atlantic salmon pursuant to section 7. However, because Atlantic salmon are one of the few species for which direct exposure data are available on the effects of cyanide, this Opinion contains numerous references to this data and its utility in evaluating the effects of cyanide on other species. The full evaluation as to how the federal action affects Atlantic salmon, and whether the action is likely to jeopardize the continued existence of Atlantic salmon is addressed in the FWS' Biological Opinion on cyanide. Similarly, NMFS and FWS share jurisdiction over Gulf sturgeon and generally divide consultations according to whether the federal activity occurs within marine or fresh water. The critical habitat listing for gulf sturgeon clarifies, however, that the FWS will consult with EPA on water quality issues (68 FR 13370). Therefore, the FWS' Biological Opinion on cyanide addresses whether the federal action is likely to jeopardize the continued existence of gulf sturgeon, and the likelihood that the designated critical habitat would be destroyed or adversely modified.

Species and Critical Habitat Not Likely to be Adversely Affected by the Proposed Action

Based upon our analysis, we established that we can concur with EPA's effect determination that a number species are not likely to be adversely affected when exposed to cyanide at criterion values. Specifically, we would not expect the following threatened or endangered species to respond physically, physiologically, or behaviorally to cyanide at the CMC or the CCC: Blue whale, bowhead whale, fin whale, humpback whale, North Atlantic right whale, North Pacific right whale, sei whale, sperm whale, Hawaiian monk seal, Western Steller sea lion, Eastern Steller sea lion, green sea turtle, hawksbill sea turtle, Kemp's ridley sea turtle, leatherback sea turtle, olive ridley sea turtle, smalltooth sawfish, elkhorn coral, staghorn coral, white abalone, black abalone, and Johnson's seagrass. Similarly, we expect the designated critical habitat for the following species is not likely to be adversely affected by cyanide at the CMC or the CCC: North Pacific right whale, Hawaiian monk seal, Western Steller sea lion, Eastern Steller sea lion, green sea turtle, hawksbill sea turtle, leatherback sea turtle, smalltooth sawfish, elkhorn coral, staghorn coral, and Johnson's seagrass. Based upon our analysis, the following proposed

species[7] are not likely to be adversely affected when exposed to cyanide at the salt water CMC or the CCC: bocaccio, canary rockfish, spotted seal and yelloweye rockfish. The effects of the proposed action on the Pacific eulachon have not been evaluated.

Listed cetaceans, pinnipeds, sea turtles, marine invertebrates and plants, and marine fishes are distributed in coastal areas that may be exposed to aquatic cyanide. Certain species, like the blue whale and sei whale, are likely to have limited exposure to cyanide sources as their migratory patterns are circumglobal with definite seasonal movements to offshore areas outside the likely extent of cyanide discharges. Nonetheless, we could not conclude that exposures would not occasionally occur, and thus evaluated the potential responses of these species when exposed to cyanide levels equivalent to the salt water CCC and CMC.

Unfortunately, data to evaluate the potential responses of listed marine species or for suitable surrogate species when exposed to cyanide at the recommended aquatic life values is severely lacking. It is for these reasons that Gensemer et al. (2007) declined to evaluate the protectiveness of the saltwater cyanide criteria for marine threatened and endangered species. Pursuant to Section 7 of the ESA, however, we are not proffered the opportunity to withhold judgment. To evaluate the effects of cyanide, particularly on marine species, the lack of data is disconcerting and warrants studies to evaluate response thresholds for more marine species.

In the interim, until further investigations that establish threshold responses are available, current information suggests that the effects of cyanide at the salt water CMC and CCC values of 1.015 µg CN/L on listed marine species and their designated critical habitat, and proposed marine species are extremely unlikely to occur and thus discountable. Our conclusion is based on available data on the responses of marine species relative to the saltwater aquatic life criteria thresholds. The recommended saltwater CMC and CCC are set at very low levels, 1.015 µg CN/L. The CMC value for cyanide was driven by data on the eastern rock crab, *Cancer irroratus.* The species mean acute value for eastern rock crab is 4.893 µg CN/L making the crab six times more sensitive than the next most sensitive marine species, the calanoid copepod, *Acartia tonsa* (EPA 1985). Data were available on the chronic effects of cyanide to only two marine species when EPA established the recommended aquatic life criteria, the mysid, *Mysidopsis bahia*, and the sheepshead minnow, *Cyprinodon variegatus*. Recognizing that these species are relatively resistant to cyanide, EPA set the CCC equal to the CMC because doing so was probably more indicative of the chronic sensitivity of the rock crab than obtained using chronic response data from other species and using other derivation methods (ACR). We found no data to suggest that listed marine species would respond to cyanide exposures at or below 1.015 µg CN/L.

Marine Mammals & Turtles

According to the *Methods Manual*, marine mammals and sea turtles are part of a broad category of "aquatic-dependent" species that whose respiratory oxygen is gained from surface air, not from oxygen dissolved in the water column (like "aquatic species"). For these species, the

7 Proposed species were listed after the completion of EPA's BE. Little data exists to discern adverse effects at levels below the saltwater CCC or CMC. Unlike the other proposed species, the Pacific Eulachon has a freshwater and saltwater life stage. Salt water exposure to cyanide at the CCC and CMC is not likely to result in adverse effects; however, Pacific eulachon still to be evaluated consistent with the approach used to evaluate the effects of the action on other freshwater fishes.

analysis would focus primarily on dietary exposure because this route is generally considered the important route of exposure. The *Methods Manual* expressly discounts dermal or other routes of exposure as areas that are "not explicitly sought in the literature search" when EPA develops the biological evaluations for pollutants but notes that in the event information is uncovered during a literature search that would suggest otherwise, it would be considered in EPA's effects analysis. Otherwise, the assessment of toxicity on aquatic-dependent listed species, which accounts for all listed marine mammals, sea turtles, and pinnipeds, is based on the estimated dietary effects concentration (dietary EC). The dietary effect would be evaluated by producing estimates of bioconcentration factors (BCFs) and bioaccumulation factors (BAFs). However, there is no published evidence to suggest that cyanide bioaccumulates in fresh- or saltwater aquatic animals. As such exposure to cyanide via the dietary or sediment pathways may not be particularly important.

High doses of cyanide that are ingested can be rapidly lethal (doses exceeding the saltwater CCC), and low doses of cyanide are rapidly metabolized and excreted. Eisler (1991) suggested that repeated sublethal dietary doses may be tolerated by many species for extended periods. The acute oral toxicity of cyanide was calculated on a small set of surrogate species and based on the wet weight of the oral dose. Species used for this analysis ranged from a variety of birds to small and large mammals such as rats, and cows. The minimum acute dietary LD_{50} for birds is 1.4 mg/kg body mass and for mammals is 2.2 mg/kg body mass. Based on these values, marine mammals, sea turtles, and pinnipeds would have to consume cyanide well in excess of the saltwater CMC to experience a lethal response. The saltwater CMC is also likely set below any potential chronic dietary threshold for marine mammals and turtles.

EPA also evaluated toxicity values for a wide range of food items, grouping them into common categories (e.g., insects, invertebrates, fish, etc). Calculated response values were above the CMC and the CCC for both saltwater and freshwater environments. Although the central tendency of the response value was used for the assessment, and not the 5^{th} percentile conservative estimate as was used for listed species, we expect this approach provides a reasonable estimate of adverse effects to prey species particularly given that most of NMFS' species are generalist feeders and a minor reduction in a particular food item should generally result in discountable and insignificant effects to listed species. For instance, the fin whale is a baleen whale and eats krill, a tiny crustacean. As mentioned previously, the species most sensitive to cyanide is the eastern rock crab. The threshold values from the eastern rock crab were used to determine the effect that cyanide may have on krill. Similarly, the loggerhead sea turtle feeds on mollusks, sponges and crabs. The food item analysis conducted by EPA for this species, was driven by the EC for mollusks (4.7) but should have been reviewed against the invertebrate EC (2.2), because it eats invertebrates and mollusks the dietary analysis should have been reviewed against the lowest EC possible. Nonetheless, the outcome remains the same in this instance—that is, marine food items should not be adversely affected by cyanide at the saltwater criteria.

Based on the best scientific and commercial data available, as discussed previously, we do not expect that the proposed action would adversely affect the quantity, quality or availability any of the constituent elements of critical habitat, or the physical, chemical, or biotic phenomena that give the designated area value for the conservation of the species when no constituent elements

were identified in the designation. Although through the proposed action, we would expect critical habitat for North Pacific right whale, Hawaiian monk seal, Western Steller sea lion, Eastern Steller sea lion, green sea turtle, hawksbill sea turtle, the leatherback sea turtle, and proposed critical habitat for the leatherback sea turtle would be exposed to cyanide, the concentration of cyanide would be sufficiently low that we expect the effects would be discountable. As reviewed in the above summary, there is little evidence to discern the effects of cyanide at levels as low as recommended by EPA in the saltwater aquatic life criteria. That said, the data that is available suggests that 1.015 µg CN/L is not likely to adversely alter water quality that supports growth and development, feeding and food resources, reproduction, areas for nesting and reproduction, or other physical, chemical or biological attributes of critical habitat for these species.

Marine Invertebrates and Plants

No dose-response data is available to derive a lethal threshold for *Acropora* species. Much of the data on corals is largely from studies that have examined the effects of the very destructive practice of cyanide fishing, which tends to employ cyanide concentrations well in excess of the saltwater criteria. At high doses, cyanide kills coral, causes loss of zooxanthellae, impaired photosynthesis, disruption of protein synthesis and altered rates of mitosis (Jones and Steven 1997; Jones and Hoegh-Guldberg 1999; Cato and Brown 2003; Cervino 2003). A few studies have been conducted on the short-term exposure of coral species to sublethal concentrations, but the concentrations have been well above the saltwater criteria. According to Dzombak et al. (2006) some studies have observed no response of coral to cyanide exposures at concentrations as low as 26 µg CN/L. More research is needed to discern the response threshold for listed species. However, given the limited data available at this time, it appears that exposure to cyanide at the low concentrations recommended by the aquatic life criteria that that any effects would likely be discountable and insignificant.

We also have very little data to suggest what the threshold response concentrations would be for marine plants. Evidence suggests that some plants are capable of transforming cyanide through enzymatic activity and can avoid cyanide intoxication by directly degrading the cyanogenic compounds or assimilating them into their metabolism. The effectiveness of this response would depend upon the plant, the balance of activity and the exposure concentration. EPA's best estimate of response thresholds is based on the freshwater blue-green algae, *Microcystis aeruginos*, and the marine red algae. The latter has a NOEC of 11 µg CN/L, well above the saltwater CMC or CCC. Using red algae as a surrogate to predict the response of Johnson's sea grass, we expect the effects of cyanide at the aquatic life criteria would be discountable and insignificant.

There were too few data available to generate a species sensitivity distribution for white or black abalone through the class level. We found only one study on the effect of cyanide on an abalone species, the *Haliotis varia,* the varied ear shell or variable abalone. Given that the varied ear shell abalone is within the same genus, the reported LC_{50} of 1012 µg CN/L is the best estimate of a lethal response for both black abalone and white abalone. Lasut (1999) studied the effects of cyanide and salinity on the mortality of abalone and found that mortality increased within decreased salinity. Abalone subjected to lethal concentrations of potassium cyanide and sodium

cyanide experienced a 19% increase risk of mortality when exposed to 25‰ salinity over that observed in 34‰ salinities. Even so, the response occurs well above the saltwater CMC. Therefore, we would not expect the species would be adversely affected when exposed to cyanide at the CMC saltwater value of 1.015 µg CN/L.

Based on the best scientific and commercial data available, as discussed previously, we do not expect that the proposed action would adversely affect the quantity, quality or availability any of the constituent elements of critical habitat, or the physical, chemical, or biotic phenomena that give the designated area value for the conservation of the species when no constituent elements were identified in the designation. Although through the proposed action, we would expect critical habitat for elkhorn coral, staghorn coral, and Johnson's seagrass would be exposed to cyanide, the concentration of cyanide would be sufficiently low that we expect the effects would be discountable. As reviewed in the above summary, there is little evidence to discern the effects of cyanide at levels as low as recommended by EPA in the saltwater aquatic life criteria. That said, the data that is available suggests that 1.015 µg CN/L is not likely to adversely alter water quality that supports growth and development, feeding and food resources, reproduction, areas for nesting and reproduction, or other physical, chemical or biological attributes of critical habitat for these species.

Marine Fishes

Too few data exist to generate a species sensitivity distribution estimate for this smalltooth sawfish, or the recently proposed rockfish species, bocaccio, yelloweye, and canary rockfish, through the class level. In comparison of the mean LC50 and NOEC values for the most closely related marine fishes range from 59.3 to 372 and 5.608 to 35.18 µg CN/L, respectively. Data on most acutely sensitive marine fish, the Atlantic silverside, results in acute and chronic EC_As of 26.12 and 5.608 µg CN/L, in the range of the most acutely sensitive freshwater fish species. Since insufficient data are available to model species sensitivity distributions for marine species, we relied on the calculated EC_As of the most sensitive marine fish for which data was available in making our effects determination. Although not included in EPA's biological evaluation, the three proposed rockfish would be evaluated using the same EC_A values, as not enough data exists to employ other evaluation methods. As such, data on the Atlantic silverside suggests that the saltwater cyanide criteria would likely result in discountable and insignificant effects on bocaccio, yelloweye, and canary rockfish, and smalltooth sawfish.

Based on the best scientific and commercial data available, as discussed previously, we do not expect that the proposed action would adversely affect the quantity, quality or availability any of the essential features of critical habitat. Although through the proposed action, we would expect critical habitat for smalltooth sawfish would be exposed to cyanide, the concentration of cyanide would be sufficiently low that we expect the effects would be discountable. As reviewed in the above summary, there is little evidence to discern the effects of cyanide at levels as low as recommended by EPA in the saltwater aquatic life criteria. That said, the data that is available suggests that 1.015 µg CN/L is not likely to adversely alter water quality that supports growth and development, feeding and food resources, reproduction, areas for nesting and reproduction, or other physical, chemical or biological attributes of critical habitat for these species.

Species and Critical Habitat Likely to be Adversely Affected by the Proposed Action

Anadromous Fishes

Chinook Salmon

Description of the Species

Chinook salmon are the largest of the Pacific salmon and historically ranged from the Ventura River in California to Point Hope, Alaska in North America, and in northeastern Asia from Hokkaido, Japan to the Anadyr River in Russia (Healey 1991). In this section, we discuss the distribution, status, and critical habitats of the nine species[8] of endangered and threatened Chinook salmon separately, and summarize their common dependence on waters of the United States. However, because Chinook salmon in the wild are virtually indistinguishable between listed species, and are the same biological species we begin this section describing those characteristics common across ESUs (the listed species).

Of the Pacific salmon species considered herein, Chinook salmon exhibit arguably one of the most diverse and complex life history strategies with multiple races within which there is substantial variation. One form, the "stream-type", resides in freshwater for a year or more following emergence and the "ocean-type" migrates to the ocean within their first year. The ocean-type typifies populations north of 56ºN (Healy 1991). Within each race, there is often variation in age at seaward migration, age of maturity, timing of spawning migrations, male precocity, and female fecundity.

The general Chinook salmon life cycle spans fresh and marine waters, with one reproductive event per adult (that is, Chinook salmon are semelparous and die after spawning). Spawning migrations generally occur in the spring and fall, although the precise timing of spawning migrations and spawning varies across populations and can vary within populations. Temperature and stream flow can significantly influence the timing of upstream migrations and spawning, and the selection of spawning habitat (Geist et al. 2009; Hatten and Tiffan 2009). However, a general latitudinal cline is apparent across the species' range with spawning typically occurring earlier in the spring/summer at northern latitudes and later in southern latitudes (Healy 1991).

On the spawning grounds, mate competition is intense with males competing to fertilize eggs and females competing for optimal nest site selection. Once fertilization occurs, female Chinook salmon bury the eggs in nests –termed "redds"- and they guard the nests until their death, which generally occurs a couple days later to a couple weeks after spawning. A female generally deposits eggs in more than one depression within a redd, excavating stream rock as she moves upstream, increasing the size of her redd until all eggs are deposited.

Size and age at maturity is partially under genetic control, but can be influenced by environment

[8] We use the word "species" as it has been defined in section 3 of the ESA, which include "species, subspecies, and any distinct population segment of any species of vertebrate fish or wildlife which interbreeds when mature (16 U.S.C 1533)." Pacific salmon that have been listed as endangered or threatened were listed as "evolutionarily significant units (ESU)" which NMFS uses to identify distinct population segments (DPS) of Pacific salmon. Any ESU or DPS is a "species" for the purposes of the ESA.

and migration behavior (Roni and Quinn 1995). Generally, ocean-type salmon are at sea longer than their stream-type counterparts and tend to be larger in size at spawning. Body size can be important in determining reproductive success in terms of nest selection and mating competition (Foote 1990). Chinook salmon age at maturity ranges from 1 to 7 years with most returning to spawn between 3 and 4 years of age.

The time necessary for egg incubation until emergence of alevins in fresh water varies among basins and among years within a basin, and is closely correlated to water temperatures such that low temperatures can prolong incubation. Incubation generally takes a couple of months or more. Alevin (also called "yolk-sac" fry) remain buried until their yolk-sac is absorbed, at which time they become free swimming fry. Egg to fry survival can also vary widely across basins, years, and habitat conditions within a basin. In general, the survival of eggs and alevin, and the fitness of emerging fry are affected by sediment loading, intergravel water flow and dissolved oxygen levels, gravel composition, spawn timing, floods, redd and spawner density, and water temperatures.

Once emerged, fry behavior varies among populations and among individuals within races. Some juvenile Chinook salmon rear in fresh water for a few weeks to a few years, others move immediately downstream coastal waters where they rear in estuaries for a few weeks to months, while others migrate directly to ocean waters. Stream-type Chinook salmon do not migrate to sea until the spring following emergence, and ocean-type Chinook salmon migrate to the ocean within their first year. Generally, most fry move at night probably to reduce detection by predators, although some fish will move downstream during daylight. Not all movement is volitional as stream flows often displace fry to downstream areas after emergence. Density-dependent factors such as space, prey, or stream flows may influence the outmigration behavior of individual juvenile Chinook salmon.

While in fresh water, juvenile Chinook salmon are often found in the lower reaches of a river near its estuary, where they inhabit river margins in areas of shallow water, near woody debris, or other areas of low water velocity. As juveniles grow in size, they tend to move away from the shoreline to deeper waters where the velocity is higher (Healey 1991). Generally, Chinook salmon outmigrants (termed smolts) are about 2 to 5 inches long when they enter saline (often brackish) waters. The process of smoltification is a physiologically demanding process that enables salmon to adapt to sea water and maintain the appropriate osmotic pressure necessary to maintain body fluid concentration and composition, and homeostasis as the fish enters waters of increased salinity. The transformation from the fresh water fry/parr juvenile stage to smolt involves multiple physiological changes including an increase in: body silvering, hypoosmotic regulatory capability, salinity tolerance and preference, growth rate, oxygen consumption, ammonia production, endocrine activity (e.g., activation of thyroid, interregnal and pituitary growth hormone cells), and gill Na^+, K^+-ATPase activity. At the same time, the ratio of weight standardized to length (condition factor) declines and total body lipid content declines (Wedemeyer et al. 1980). Several factors can affect smoltification process, not only at the interface between fresh water and salt water, but higher in the watershed as the process of transformation begins long before fish enter salt waters including: exposure to chemicals such as heavy metals, and elevated water temperatures (Wedemeyer et al. 1980).

Life at sea varies according to population, race, and age-class. Chinook salmon tend to remain at sea between 1 and 6 years, with most fish returning to fresh water after 2 to 4 years at sea. Fishery catches indicate that ocean- and stream-type fish exhibit divergent migratory pathways while in the ocean (Healey 1983, 1991). Ocean-type Chinook salmon tend to be found along the coastline, whereas stream-type Chinook salmon are found in the open ocean far from the coast (Healey 1983, 1991).

Chinook salmon feed on a variety of prey organisms depending upon life stage. Adult oceanic Chinook salmon eat small fish, amphipods, and crab megalops (Healey 1991). Fish, in particular herring, make up the largest portion of an adult Chinook salmon's diet. In estuaries, Chinook salmon smolts tend to feed on chironomid larvae and pupae, *Daphnia*, *Eogammarus*, *Corphium* and *Neomysis*, as well as juvenile herring, sticklebacks and other small fish. In fresh water, Chinook salmon juveniles feed on adult and larval insects including terrestrial and aquatic insects such as dipterans, beetles, stoneflies, chironomids, and plecopterans (Healey 1991).

Threats

Natural Threats. Chinook salmon are exposed to high rates of natural predation during freshwater rearing and migration stages, as well as during ocean migration. In general, Chinook salmon are prey for pelagic fishes, birds, and marine mammals, including harbor seals, sea lions, and killer whales. There have been recent concerns that the increasing size of tern, seal, and sea lion populations in the Pacific Northwest may have reduced the survival of some salmon species.

Anthropogenic Threats. Salmon survive only in aquatic ecosystems and, therefore, depend on the quantity and quality of those ecosystems. Chinook salmon have declined under the combined effects of fishery over-harvest; competition from fish raised in hatcheries and native and non-native exotic species; dams that block their migrations and alter river hydrology; gravel mining that impedes their migration and alters the dynamics (hydrogeomorphology) of the rivers and streams that support juveniles; water diversions that deplete water levels in rivers and streams; destruction or degradation of riparian habitat that increase water temperatures in rivers and streams sufficient to reduce the survival of juvenile Chinook salmon; and land use practices (logging, agriculture, urbanization) that destroy wetland and riparian ecosystems while introducing sediment, nutrients, biocides, metals, and other pollutants into surface and ground water and degrade water quality in the freshwater, estuarine, and coastal ecosystems throughout the Pacific Northwest (Buhle et al. 2009).

Salmon along the west coast of the United States share many of the same threats. Therefore, anthropogenic threats for all species and populations are summarized here. Population declines have resulted from several human-mediated causes, but the greatest negative influence has likely been the establishment of waterway obstructions such as dams, power plants, and sluiceways for hydropower, agriculture, flood control, and water storage. These structures have blocked salmon migration to spawning habitat or resulted in direct mortality and have eliminated entire salmon runs as a result. While some of these barriers remain, others have been reengineered, renovated, or removed to allow for surviving runs to access former habitat, but success has been limited. These types of barriers alter the natural hydrograph of basins, both upstream and downstream of the structure, and significantly reduce the availability and quality of spawning and rearing habitat (Hatten and Tiffan 2009). Many streams and rivers, particularly in urban or suburban areas,

suffer from streamside development, which contributes sediment, chemical pollutants from pesticide applications and automobile or industrial activities, altered stream flows, loss of streamside vegetation and allochthonous materials to name a few. These factors can directly cause mortality, reduce reproductive success, or affect the health and fitness of all salmon life stages.

Artificial propagation of hatchery fish has had profound consequences on the viability of some natural salmon populations, but there are potential benefits to the artificial production of salmon as well. Adverse effects of artificial propagation include: a decline in the natural population from the taking of wild broodstock for artificial propagation, the genetic erosion of populations (introgression, hybridization), an increased incidence of disease in the wild and increased rates of competition with and predation on naturally spawned salmon populations. Potential benefits to artificial propagation include the bolstering of the numbers of naturally spawning fish in the short-term, the conservation of genetic resources, and guarding against the catastrophic loss of naturally spawned populations at critically low abundance levels.

Fishing for salmon has also negatively impacted salmon populations. Fishing reduces the number of individuals within a population and can lead to uneven exploitation of certain populations and size classes (Reinsenbichler 1997; Mundy 1997). Targeted fishing of larger individuals results in excluding the most fecund individuals from spawning (Reinsenbichler 1997). Genetic changes that promote smaller body sizes have occurred in heavily exploited populations in response to size-selective harvest pressures (Reinsenbichler 1997; Mundy 1997; Swain et al. 2007). Fishing pressure can reduce age at maturity in fished populations as the fished populations compensate for the reductions in the numbers of spawning adults (Reinsenbichler 1997).

Pacific salmon species are exposed to a number of contaminants throughout their range and life history cycle. Exposure to pollution is also of significant concern for all life stages, but is likely particularly significant for freshwater life stages. Organic pollutants, particularly PCBs, DDT and its congeners, pesticides, and endocrine disruptors are of particular concern. These chemicals can inhibit smell, disrupt reproductive behavior and physiology, impair immune function, and lead to mortality through impairment of water balance when traveling between fresh and salt water systems (Varanasi et al. 1993). Diffuse and extensive population centers contribute increase contaminant volumes and variety from such sources as wastewater treatment plants and sprawling development. Urban runoff from impervious surfaces and roadways often contains oil, copper, pesticides, PAHs, and other chemical pollutants and flow into surface waters. Point and nonpoint pollution sources entering rivers and their tributaries affect water quality in available spawning and rearing habitat for salmon. Juvenile salmonids that inhabit urban watersheds often carry high contaminant burdens, which is partly attributable to the biological transfer of contaminants through the food web (Brown et al. 1985; Stein et al. 1992; Varanasi et al. 1993).

Climate change poses significant hazards to the survival and recovery of salmonids along the west coast. Paleoecological data (which exclude anthropogenic influences) suggest regional and global climate factors on decadal, centennial, and millennial time scales are tied to abundance patterns of Pacific salmonids (Finney et al. 2009). Increases in global temperatures are likely to

have profound effects on salmonids directly and indirectly through altered hydrological regimes. Increases in instream temperatures may decrease habitat available for refugia, increase species interactions and competition, accelerate incubation timing and premature emergence, increase susceptibility to parasites and disease, reduce fry survival, delay migration and spawning, and accelerate loss of energy reserves. Using emission scenarios from the Intergovernmental Panel on Climate Change (IPCC), O'Neal (2002) estimates that direct thermal changes in freshwater temperatures could cause the loss of between 4-20% of existing salmon and trout habitat by the year 2030, 7-34% by 2060, and 14-42% by 2090, depending on the trout or salmon species, IPCC emission scenario considered, and the model used. Projected salmon habitat loss would be most severe in Oregon and Idaho, at losses of 40% or greater of 2007 habitat estimates. While the predicted losses are substantial, the estimates may underestimate the overall effect global climate change will have on salmon and trout abundance since these models do not consider the related effects from changes in seasonal hydrological patterns and water volumes that result from altered weather patterns and precipitation (O'Neal 2002).

Changes in hydrological regimes are closely linked to salmon abundance (Hicks et al. 1991; Clark et al. 2001). From studies that have examined the effects of timber harvest and other changes in land use patterns, we know that changes in hydrology (i.e., increased peak flows, decreased low flows, altered timing discharge events, and rapid fluctuations in flows) can profoundly affect salmon abundance and the amount and availability of quality habitat. Hydrology is strongly correlated to in-redd and young of the year survival, can lead to the displacement of young fish, alter immigration and emigration timing, alter the volume of available habitat by affecting channel structure (e.g., pool to riffle ratios, debris loading, substrate composition, erosion and sediment loading) and the relative abundance of salmon and trout species within a watershed, as well as the relative abundance of age-classes (see Hicks et al. 1991; Gregory and Bisson 1997). Such ecosystem changes are also likely to alter macroinvertebrate communities and habitats, affecting important forage for salmon and trout (McCarthy et al. 2009; Williams et al. 2009).

Upstream changes in riverine habitat can affect downstream estuarine ecosystems through alterations in sediment delivery (timing and volume), and changes in freshwater volumes and timing can influence the volume of the spring/summer salt-wedge (O'Neal 2002). In turn, changes in the trophic dynamics of the estuary may occur. At the same time, physical changes in the ocean associated with warming include increases in temperature, increased water column stratification, and changes in the intensity and timing of coastal upwelling. These changes will alter primary and secondary productivity, the structure of marine communities, and, in turn, the growth, productivity, survival, and migrations of salmonids. Changing ocean temperatures may alter salmon behavior, distribution, and migrations, increasing the distance from home streams to ocean feeding areas. Energetic demands increase at warmer temperatures, requiring increased feeding to maintain growth. This could lead to intensified competition for food and reduction in growth rates, further exacerbating the prey/predator relationship. Increasing concentrations of carbon dioxide in the oceans lowers pH, which reduces the availability of carbonate for shell-forming marine animals. Pteropods are expected to be negatively affected, and they can comprise more than 40% of some salmon diets. If salmon migrate farther to the north and/or food is less available, longer times may be required to reach maturity, delaying return of adult migrations into coastal water and rivers.

California Coastal Chinook Salmon

Distribution and Description of the Listed Species

The California Coastal Chinook salmon ESU includes all naturally spawned populations of Chinook salmon from rivers and streams south of the Klamath River to the Russian River, California. Seven artificial propagation programs are part of this ESU: The Humboldt Fish Action Council (Freshwater Creek), Yager Creek, Redwood Creek, Hollow Tree, Van Arsdale Fish Station, Mattole Salmon Group, and Mad River Hatchery fall-run Chinook hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU.

California Coastal Chinook salmon are a fall-run, ocean-type fish. A spring-run (river-type) component existed historically, but is now considered extinct (Bjorkstedt et al. 2005). Table 3 identifies populations within the California Coastal Chinook salmon ESU, their abundances, and the relative contribution of artificially propagated fish to the population.

Table 3. California coastal Chinook populations and selected measures of population viability

Population	Historical Abundance[a]	Mean Number of Spawners (Range)[b]	Percent Hatchery Contribution[c]	Long-term Trend[d]
Freshwater Creek		22 (13-22)	30-70	0.137 (-0.405, 0.678)
Eel River	17,000-55,000		~30	
Mainstem Eel River	13,000			
Sprowl Creek		43 (43-497)		-0.096 (-0.157, -0.034)
Tomki Creek		61 (13-2233)		-0.199 (-0.351, -0.046)
Van Duzen River	2,500			
Middle Fork Eel River	13,000			
South Fork Eel River	27,000			
North Fork Eel River				
Upper Eel River				
Redwood Creek	1,000-5,000			
Mad River	1,000-5,000			
Canyon Creek		73 (19-103)		0.0102 (-0.106, 0.127)
Bear River	100			
Mattole River	1,000-5,000		~17	
Russian River	50-500		~0	
Humbolt Bay tributaries	40			
Tenmile to Gualala			0	
Small Humboldt County rivers	1,500		0	
Rivers north of Mattole River	600		0	
Noyo River	50		0	

[a]Historical abundance estimates based on professional opinion and evaluation of habitat conditions (reported in Good et al. 2005).
[b]5-year (1997-2001) geometric mean number of counts of adults (quasi-systematic surveys of spawners – Canyon, Tomki, and Sprowl creeks; returning spawners at Freshwater Creek weir).
[c]Hatchery production in this ESU is at low levels, aimed at supplementing depressed runs. Operational procedures and low production suggest that the ESU may not be at substantial risk of degraded genetic integrity (Good et al. 2005).

[d]Long-term trends were calculated using the entire available data set (see Good et al. 2005). The 90% confidence intervals are noted in parentheses.

Status and Trends

NMFS listed California Coastal Chinook salmon as threatened on September 16, 1999 (64 FR 50393), and they retained their threatened status on June 28, 2005 (70 FR 37160). California Coastal Chinook salmon were listed due to the combined effect of dams that prevent them from reaching spawning habitat, logging, agricultural activities, urbanization, and water withdrawals in the river drainages that support them. Historical estimates of escapement, based on professional opinion and evaluation of habitat conditions, suggest abundance was roughly 73,000 in the early 1960s with the majority of fish spawning in the Eel River (CDFG 1965 *in* Good et al. 2005). The species exists as small populations with highly variable cohort sizes. The Russian River probably contains some natural production, but the origin of those fish is not clear because of a number of introductions of hatchery fish over the last century. The Eel River contains a substantial fraction of the remaining Chinook salmon spawning habitat for this species. Since its original listing and status review, little new data are available or suitable for analyzing trends or estimating changes in this population's growth rate (Good et al. 2005).

Long-term trends in Freshwater Creek are positive, and in Canyon Creek, although only slightly different than zero, the trend is positive (Table 3). Long-term trends in Sprowl and Tomki creeks (tributaries of the Eel River), however, are negative. Good et al. (2005) caution making inferences on the basin-wide status of these populations as they may be weak because the data likely include unquantified variability due to flow-related changes in spawners' use of mainstem and tributary habitats. Unfortunately, none of the available data is suitable for analyzing the long-term trends of the ESU or estimating the population growth rate.

Critical Habitat

NMFS designated critical habitat for California Coastal Chinook salmon on September 2, 2005 (70 FR 52488). Specific geographic areas designated include the following CALWATER hydrological units: Redwood Creek, Trinidad, Mad River, Eureka Plain, Eel River, Cape Mendocino, Mendocino Coast, and the Russian River. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this ESU identifies primary constituent elements that include sites necessary to support one or more Chinook salmon life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The critical habitat designation (70 FR 52488) contains additional details on the sub-areas that are included as part of this designation, and the areas that were excluded from designation.

In total, California Coastal Chinook salmon occupy 45 watersheds (freshwater and estuarine). The total area of habitat designated as critical includes about 1,500 miles of stream habitat and about 25 square miles of estuarine habitat, mostly within Humboldt Bay. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not

defined the lateral extent is defined as the bankfull elevation. In estuarine areas the lateral extent is defined by the extreme high water because extreme high tide areas encompass those areas typically inundated by water and regularly occupied by juvenile salmon during the spring and summer, when they are migrating in the nearshore zone and relying on cover and refuge qualities provided by these habitats, and while they are foraging. Of the 45 watershed reviewed in NMFS' assessment of critical habitat for California Coastal Chinook salmon, eight watersheds received a low rating of conservation value, 10 received a medium rating, and 27 received a high rating of conservation value for the species.

Critical habitat in this ESU consists of limited quantity and quality summer and winter rearing habitat, as well as marginal spawning habitat. Compared to historical conditions, there are fewer pools, limited cover, and reduced habitat complexity. The limited instream cover that does exist is provided mainly by large cobble and overhanging vegetation. Instream large woody debris, needed for foraging sites, cover, and velocity refuges is especially lacking in most of the streams throughout the basin. NMFS has determined that these degraded habitat conditions are, in part, the result of many human-induced factors affecting critical habitat including dam construction, agricultural and mining activities, urbanization, stream channelization, water diversion, and logging, among others.

Central Valley Spring-Run Chinook Salmon

Distribution and Description of the Listed Species

The Central Valley spring-run Chinook salmon ESU includes all naturally spawned populations of spring-run Chinook salmon in the Sacramento River and its tributaries in California. This ESU includes one artificial propagation program, the Feather River Hatchery spring-run Chinook salmon program. This artificially propagated population is no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU.

Central Valley spring-run Chinook salmon ESU includes Chinook salmon entering the Sacramento River from March to July and spawning from late August through early October, with a peak in September. Spring-run fish in the Sacramento River exhibit an ocean-type life history, emigrating as fry, sub-yearlings, and yearlings. Central Valley spring-run Chinook salmon require cool freshwater while they mature over the summer.

Status and Trends

NMFS originally listed Central Valley spring-run Chinook salmon as threatened on September 16, 1999 (64 FR 50393), a classification this species retained on June 28, 2005 (70 FR 37160). This species was listed because dams isolate them from most of their historic spawning habitat and the habitat remaining to them is degraded. Historically, spring-run Chinook salmon were predominant throughout the Central Valley occupying the upper and middle reaches (1,000 to 6,000 feet) of the San Joaquin, American, Yuba, Feather, Sacramento, McCloud and Pit Rivers, with smaller populations in most tributaries with sufficient habitat for over-summering adults (Stone 1874; Rutter 1904; Clark 1929).

Table 4. Central Valley spring-run Chinook salmon populations and selected measures of population viability

Population	Historical Abundance[a]	Mean Number of Spawners (Range)[b]	Percent Hatchery Contribution[c]	Mean Annual Population Growth Rate (λ)[d]
Butte Creek spring-run		4,513 (67-4,513)		1.30 (1.09-1.60)
Deer Creek spring-run		1,076 (243-1,076)		1.17 (1.04-1.35)
Mill Creek spring-run		491 (203-491)		1.19 (1.00-1.47)

[a]Historical abundance for the total ESU, based on gillnet fishery catches, is estimated at about 700,000 (Fisher 1994). Individual river estimates of historical abundance not provided.
[b]Recent geometric mean number of spawners as reported by Good et al. 2005. Note the current geometric mean for Butte, Deer and Mill creeks are also the maximum means.
[c]Between 1967 and 1999 the Feather River Hatchery released between less than 1 million to as much as 5.5 million spring-run Chinook salmon in any given year. Returns ranged from less than 1,000 spawners to about 7,000 in the late 1980s (see Good et al. 2005). No other hatchery data reported.
[d]The λ calculation, provided by Good et al. 2005, is an estimate of the population growth rate. The 90% confidence intervals are noted in parentheses.

The Central Valley drainage as a whole is estimated to have supported spring-run Chinook salmon runs as large as 700,000 fish between the late 1880s and the 1940s (Fisher 1994), although these estimates may reflect an already declining population, in part from the commercial gillnet fishery that occurred in this ESU (Good et al. 2005). Before construction of Friant Dam, nearly 50,000 adults were counted in the San Joaquin River alone (Fry 1961). Following the completion of Friant Dam, the native population from the San Joaquin River and its tributaries (i.e., the Stanislaus and Mokelumne Rivers) was extirpated. Spring-run Chinook salmon no longer exist in the American River due to the operation of Folsom Dam. Naturally spawning populations of Central Valley spring-run Chinook salmon currently are restricted to accessible reaches of the upper Sacramento River, Antelope Creek, Battle Creek, Beegum Creek, Big Chico Creek, Butte Creek, Clear Creek, Deer Creek, Feather River, Mill Creek, and Yuba River (CDFG 1998). Since 1969, the Central Valley spring-run Chinook salmon ESU (excluding Feather River fish) has displayed broad fluctuations in abundance ranging from 25,890 in 1982 to 1,403 in 1993 (CDFG unpublished data *in* Good et al. 2005).

The average abundance for the ESU was 12,499 for the period of 1969 to 1979, 12,981 for the period of 1980 to 1990, and 6,542 for the period of 1991 to 2001. In 2003 and 2004, total run size for the ESU was 8,775 and 9,872 adults respectively, well above the 1991 to 2001 average. Evaluating the ESU as a whole, however, masks significant changes that are occurring among populations that comprise the ESU (metapopulation). For example, the mainstem Sacramento River population has undergone a significant decline while the abundance of many tributary populations increased. Average abundance of Sacramento River mainstem spring-run Chinook salmon recently declined from a high of 12,107 for the period 1980 to 1990, to a low of 609 for the period 1991 to 2001, while the average abundance of Sacramento River tributary populations increased from a low of 1,227 to a high of 5,925 over the same periods.

Abundance time series data for Mill, Deer, Butte, and Big Chico creeks spring-run Chinook salmon confirm that population increases seen in the 1990s have continued through 2001 (Good et al. 2005). Habitat improvements, including the removal of several small dams and increases in summer flows in the watersheds, reduced ocean fisheries, and a favorable terrestrial and marine climate, have likely contributed to this. All three spring-run Chinook salmon populations in the

Central Valley have long-and short-term positive population growth. Although the populations are small, Central Valley spring-run Chinook salmon have some of the highest population growth rates in the Central Valley.

Critical Habitat

NMFS designated critical habitat for Central Valley spring-run Chinook salmon on September 2, 2005 (70 FR 52488). Specific geographic areas designated include the following CALWATER hydrological units: Tehama, Whitmore, Redding, Eastern Tehama, Sacramento Delta, Valley-Putah-Cache, Marysville, Yuba, Valley-American, Colusa Basin, Butte Creek, and Shasta Bally hydrological units. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this ESU identifies primary constituent elements that include sites necessary to support one or more Chinook salmon life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The critical habitat designation (70 FR 52488) contains additional details on the sub-areas that are included as part of this designation, and the areas that were excluded from designation.

In total, Central Valley spring-run Chinook salmon occupy 37 watersheds (freshwater and estuarine). The total area of habitat designated as critical includes about 1,100 miles of stream habitat and about 250 square miles of estuarine habitat in the San Francisco-San Pablo-Suisun Bay complex. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. In estuarine areas the lateral extent is defined by the extreme high water because extreme high tide areas encompass those areas typically inundated by water and regularly occupied by juvenile salmon during the spring and summer, when they are migrating in the nearshore zone and relying on cover and refuge qualities provided by these habitats, and while they are foraging. Of the 37 watersheds reviewed in NMFS' assessment of critical habitat for Central Valley spring-run Chinook salmon, seven watersheds received a low rating of conservation value, three received a medium rating, and 27 received a high rating of conservation value for the species.

Factors contributing to the downward trends in this ESU include: reduced access to spawning/rearing habitat behind impassable dams, climatic variation, water management activities, hybridization with fall-run Chinook salmon, predation, and harvest (CDFG 1998). Several actions have been taken to improve and increase the primary constituent elements of critical habitat for spring-run Chinook salmon, including improved management of Central Valley water (e.g., through use of CALFED Environmental Water Account and Central Valley Project Improvement Act (b)(2) water accounts), implementing new and improved screen and ladder designs at major water diversions along the mainstem Sacramento River and tributaries, removal of several small dams on important spring-run Chinook salmon spawning streams, and changes in ocean and inland fishing regulations to minimize harvest. Although protective measures and critical habitat restoration likely have contributed to recent increases in spring-run Chinook salmon abundance, the ESU is still below levels observed from the 1960s through 1990.

Threats from hatchery production (i.e., competition for food between naturally spawned and hatchery fish, and run hybridization and homogenization), climatic variation, reduced stream flow, high water temperatures, predation, and large scale water diversions persist.

Lower Columbia River Chinook Salmon

Distribution and Description of the Listed Species

The Lower Columbia River Chinook salmon ESU includes all naturally spawned populations of Chinook salmon from the Columbia River and its tributaries from its mouth at the Pacific Ocean upstream to a transitional point between Washington and Oregon, east of the Hood River and the White Salmon River, and includes the Willamette River to Willamette Falls, Oregon, exclusive of spring-run Chinook salmon in the Clackamas River. Seventeen artificial propagation programs are part of this ESU: The Sea Resources Tule, Big Creek Tule, Astoria High School (STEP) Tule, Warrenton High School (STEP) Tule, Elochoman River Tule, Cowlitz Tule, North Fork Toutle Tule, Kalama Tule, Washougal River Tule, Spring Creek National Fish Hatchery Tule, Cowlitz spring (Upper Cowlitz River and Cispus River), Friends of the Cowlitz spring, Kalama River spring, Lewis River spring, Fish First spring, and the Sandy River Hatchery Chinook salmon programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU.

Lower Columbia River Chinook salmon have three life history types, including early fall runs (tules), late fall runs (brights), and spring-runs. Spring and fall runs have been designated as part of a Lower Columbia River Chinook salmon ESU. The Cowlitz, Kalama, Lewis, White Salmon, and Klickitat Rivers are the major river systems on the Washington side, and the lower Willamette and Sandy Rivers are foremost on the Oregon side. The eastern boundary for this species occurs at Celilo Falls, which corresponds to the edge of the drier Columbia Basin Ecosystem and historically may have been a barrier to salmon migration at certain times of the year. The predominant life history type for this species is the fall-run. Fall Chinook salmon typically enter the Columbia River in August through October to spawn in the mainstem of the large rivers (Kostow 1995). Spring Chinook salmon enter freshwater in March through June to spawn in upstream tributaries and generally emigrate from fresh water as yearlings.

Status and Trends

NMFS originally listed Lower Columbia River Chinook salmon as threatened on March 24, 1999 (64 FR 14308); NMFS reaffirmed the threatened status of Lower Columbia River Chinook salmon on June 28, 2005 (70 FR 37160). Historical records of Chinook salmon abundance are sparse, but cannery records suggest a peak run of 4.6 million fish (43 million pounds) in 1883 (Lichatowich 1999). Although fall-run Chinook salmon are still present throughout much of their historical range, they are still subject to large-scale hatchery production, relatively high harvest, and extensive habitat degradation. The Lewis River late-fall-run Chinook salmon population is the healthiest and has a reasonable probability of being self-sustaining. Abundances largely declined during 1998 to 2000 and trend indicators for most populations are negative, especially if hatchery fish are assumed to have a reproductive success equivalent to that of natural-origin fish (see Table 5).

Most populations for which data are available have a long-term declining population trend (Table 5). Currently, the spatial extent of populations in the Coastal and Cascade fall runs are similar to their respective historical conditions. New data include spawner abundance estimates through 2001, new estimates of the fraction of hatchery spawners, and harvest estimates. In addition, estimates of historical abundance have been provided by the Washington Department of Fish and Wildlife. The Willamette/Lower Columbia River Technical Review Team estimated that 8 to 10 historic populations have been extirpated, most of them spring-run populations. Near loss of that important life history type remains an important concern. Although some natural production currently occurs in 20 or so populations, only one exceeds 1,000 spawners. Almost all spring-run Chinook salmon are at very high risk of extinction. High hatchery production continues to pose genetic and ecological risks to natural populations and to mask their performance for Coastal, Cascade, and Gorge fall run populations. Most Lower Columbia River Chinook salmon populations have not seen increases in recent years as pronounced as those that have occurred in many other geographic areas.

Table 5. Lower Columbia River Chinook salmon life histories, populations and selected measures of population viability

Life History	Population	Historical Abundance[a]	Mean Number of Spawners (range)[b]	Percent Hatchery Contribution[c]	Long-term Median Growth Rate (λ)[d]
Fall run	Youngs Bay				
	Grays River	2,477	99	38	0.944, 0.844
	Big Creek				
	Elochoman River		676	68	1.037, 0.800
	Clatskanie River[e]		50 (34-74)		0.99
	Mill, Abernathy, and Germany Creeks		734	47	0.981, 0.829
	Scappoose Creek				
	Coweeman River	4,971	274	0	1.092, 1.091
	Lower Cowlitz River	53,956	1,562	62	0.998, 0.682
	Upper Cowlitz River		5,682		
	Toutle River	25,.392			
	Kalama River	22,455	2,931	67	0.973, 0.818
	Salmon Creek and Lewis River	47,591[f]	256	0	0.984, 0.979
	Clackamas River		40		
	Washougal River	7,518	3,254	58	1.025, 0.815
	Sandy River		183		
	Columbia Gorge-lower tributaries				
	Columbia Gorge-upper tributaries	2,363	136 (Wind River only)	13 (Wind River only	0.959, 0.955
	Hood River		18		
	Big White Salmon River		334	21	0.963, 0.945
Late fall (bright)	Sandy River[e]		3085 (2337-4074)	3	0.997

	North Fork Lewis River		7,841	13	0.968, 0.948
Spring run	Upper Cowlitz River				
	Cispus River		1,787		
	Tilton River				
	Toutle River	2,901			
	Kalama River	4,178	98		
	Lewis River		347		
	Sandy River[e]		297 (202-438)		0.961
	Big White Salmon River				
	Hood River		51		

[a]Historical abundance for various rivers was calculated using the Ecosystem and Diagnosis Treatment (EDT) model, which attempts to predict population performance based on reach-specific habitat attributes. Estimates are provided as a means of comparing the historical abundance of populations relative to current abundance. See Good et al. (2005) for a discussion about the uncertainty associated with these estimates.
[b]Recent geometric mean number of spawners as reported in Good et al. 2005
[c]Recent average hatchery-origin spawners (%) as reported by Good et al. 2005. Natural-origin spawners are those that had parents that spawned in the wild, as opposed to hatchery-origin fish, whose parents were spawned in a hatchery.
[d]The long-term median growth rate (λ) is an estimate of the natural growth rate after accounting for hatchery-origin spawners. The two values are estimates under two hypotheses about the reproductive success of hatchery origin spawners. Hatchery fish are assumed to have zero reproductive success in the first estimate. In the second estimate hatchery fish are assumed to have the same relative reproductive success as natural-origin fish. Growth rates were not calculated for all populations, as adequate data were not available (see Good et al. 2005 for 95% confidence intervals on growth estimates).
[d]Values for these populations are reported in McElhany et al. 2007, and represent estimates based on the total available data series, which varies by population.
[fc]Combined estimate of Lewis River fall run (East Fork only) and Lewis River brights (Good et al. 2005)

Critical Habitat

NMFS designated critical habitat for Lower Columbia River Chinook salmon on September 2, 2005 (70 FR 52630). Designated critical habitat includes all Columbia River estuarine areas and river reaches proceeding upstream to the confluence with the Hood Rivers as well as specific stream reaches in a number of tributary subbasins. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this ESU identifies primary constituent elements that include sites necessary to support one or more Chinook salmon life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. Of 52 subbasins reviewed in NMFS' assessment of critical habitat for the Lower Columbia River Chinook salmon ESU, 13 subbasins were rated as having a medium conservation value, four were rated as low, and the remaining subbasins (35), were rated as having a high conservation value to Lower Columbia River Chinook salmon. Factors contributing to the downward trends in this ESU are hydromorphological changes resulting from hydropower development, loss of tidal marsh and swamp habitat, and degraded freshwater and marine habitat from industrial harbor and port development, and urban development. Limiting factors identified for this species include reduced access to spawning/rearing habitat in tributaries, hatchery impacts, loss of habitat diversity and channel stability in tributaries, excessive fine sediment in spawning gravels, elevated water temperature in tributaries, and harvest impacts.

Upper Columbia River Spring-run Chinook Salmon

Distribution and Description of the Listed Species

The Upper Columbia River spring-run Chinook salmon ESU includes all naturally spawned populations of Chinook salmon in all river reaches accessible to Chinook salmon in Columbia River tributaries upstream of Rock Island Dam and downstream of Chief Joseph Dam in Washington, excluding the Okanogan River. Six artificial propagation programs are part of this ESU: the Twisp River, Chewuch River, Methow Composite, Winthrop National Fish Hatchery, Chiwawa River, and White River spring-run Chinook salmon hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU. Spring-run Chinook salmon currently spawn in only three river basins above Rock Island Dam: the Wenatchee, Entiat, and Methow Rivers. Table 6 identifies the Upper Columbia River Chinook salmon ESU populations, their abundances, and estimates of the proportion of hatchery fish that contribute to the run size.

Upper Columbia River spring-run Chinook salmon begin returning to the Columbia in early spring and enter upper Columbia tributaries from April through July, with a peak in mid-May. After migration, Upper Columbia River spring-run Chinook salmon hold in freshwater tributaries until spawning in late summer, peaking in mid- to late August. Juvenile spring-run Chinook salmon remain in fresh water for a full year before emigrating to salt water in the spring of their second year.

Table 6. Upper Columbia River Chinook salmon populations and selected measures of population viability

Population	Mean Number of Spawners (Range)[a]	Percent Hatchery Contribution[b]	Current Short-term trend (Previous)[c]
Methow River	680 (79-9,904)	59	+2.0 (-15.3)
Methow mainstem	161 redds (17-2,864)	59	+6.5
Twisp River	58 redds (10-369)	54	-9.8 (-27.4)
Chewuch River	58 redds (6-1,105)	41	-2.9 (-28.1)
Lost/Early Winter creeks	12 (3-164)	54	-14.1 (-23.2[d])
Entiat River	111 (53-444)	42	-1.2 (-19.4)
Wenatchee River	470 (119-4,446)	42	-1.5 (-37.4)
Chiwawa River	109 redds (34-1,046)	47	-0.7 (-29.3)
Nason Creek	54 redds (8-374)	39	-1.5 (-26.0)
Upper Wenatchee River	8 redds (0-215)	66	-8.9
White River	9 redds (1-104)	8	-6.6 (-35.9)
Little Wenatchee River	11 redds (3-74)	21	-25.8 (-25.8)

[a]5-year geometric mean number of spawners unless otherwise noted; Includes hatchery fish. Range denoted in parentheses. Means calculated from years 1997 to 2001, except Lost/Early Winter creeks did not include 1998 as no data was available. Data reported in Good et al. 2005.
[b]Percent hatchery-origin from 1987-1996, and reported in Good et al. 2005.
[c]Current trend – percent/year – from years 1997 to 2001. Previous trend, noted in parentheses, from 1987-1996. From Good et al. 2005.
[d]Lost River data only.

Status and Trends

NMFS listed Upper Columbia River spring-run Chinook salmon as endangered on March 24, 1999 (64 FR 14308), and reaffirmed their status as endangered on June 28, 2005 (70 FR 37160), because they had been reduced to small populations in three watersheds. Based on redd count

data series, spawning escapements for the Wenatchee, Entiat, and Methow rivers have declined an average of 5.6%, 4.8%, and 6.3% per year, respectively, since 1958. In the most recent 5-year geometric mean (1997 to 2001), spawning escapement for naturally produced fish was 273 for the Wenatchee population, 65 for the Entiat population, and 282 for the Methow population, only 8% to 15% of the minimum abundance thresholds, although escapement increased substantially in 2000 and 2001 in all three river systems. Based on 1980-2004 returns, the average annual growth rate for this ESU is estimated as 0.93 (meaning the population is not replacing itself; Fisher and Hinrichsen 2006). Assuming that population growth rates were to continue at 1980 to 2004 levels, Upper Columbia River spring-run Chinook salmon populations are projected to have very high probabilities of decline within 50 years. Population viability analyses for this species (using the Dennis Model) suggest that these Chinook salmon face a significant risk of extinction: a 75 to 100% probability of extinction within 100 years (given return rates for 1980 to present).

Hatchery influence and genetic diversity are significant issues for the continued survival of Upper Columbia River Chinook salmon. This is a result of reduced genetic diversity from homogenization of populations that occurred under the Grand Coulee Fish Maintenance Project from 1939 to 1943. Stray hatchery fish and a high proportion of hatchery fish during spawning have contributed to the high genetic diversity risk.

Critical Habitat

NMFS designated critical habitat for Upper Columbia River spring-run Chinook salmon on September 2, 2005 (70 FR 52630). The designation includes all Columbia River estuaries and river reaches upstream to Chief Joseph Dam and several tributary subbasins. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this ESU identifies primary constituent elements that include sites necessary to support one or more Chinook salmon life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat, and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The Upper Columbia River spring-run Chinook salmon ESU has 31 watersheds within its range. Five watersheds received a medium rating and 26 received a high rating of conservation value to the ESU. The Columbia River rearing/migration corridor downstream of the spawning range was rated as a high conservation value. Factors contributing to the downward trends in this ESU include mainstem Columbia River hydropower system mortality, tributary riparian degradation and loss of in-river wood, altered tributary floodplain and channel morphology, reduced tributary stream flow and impaired passage, and harvest impacts.

Puget Sound Chinook Salmon

Distribution and Description of the Listed Species

The Puget Sound Chinook salmon ESU includes all naturally spawned populations of Chinook salmon from rivers and streams flowing into Puget Sound including the Straits of Juan De Fuca

from the Elwha River, eastward, including rivers and streams flowing into Hood Canal, South Sound, North Sound and the Strait of Georgia in Washington. Twenty-six artificial propagation programs are part of the ESU: the Kendal Creek Hatchery, Marblemount Hatchery (fall, spring yearlings, spring sub-yearlings, and summer run), Harvey Creek Hatchery, Whitehorse Springs Pond, Wallace River Hatchery (yearlings and sub-yearlings), Tulalip Bay, Issaquah Hatchery, Soos Creek Hatchery, Icy Creek Hatchery, Keta Creek Hatchery, White River Hatchery, White Acclimation Pond, Hupp Springs hatchery, Voights Creek Hatchery, Diru Creek, Clear Creek, Kalama Creek, George Adams Hatchery, Rick's Pond Hatchery, Hamma Hamma Hatchery, Dungeness/Hurd Creek Hatchery, and Elwha Channel Hatchery Chinook salmon hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU.

The Puget Sound ESU is comprised of 31 historical populations, of which 22 or more are believed to be extant and nine are considered extinct. Table 7 identifies the current populations within the Puget Sound Chinook salmon ESU for which there are data, and their recent abundance and long-term trends.

Chinook salmon in this area generally have an "ocean-type" life history. Puget Sound populations include both early-returning and late-returning Chinook salmon spawners described by Healey (1991). However, within these generalized behavioral forms, significant variation occurs in residence time in fresh water and estuarine environments. For example, Hayman et al. (1996) described three juvenile Chinook salmon life histories with varying residency times in the Skagit River system in northern Puget Sound. Chinook salmon utilize nearshore Puget Sound habitats year-round, although they can be far from their natal river systems (Brennan et al. 2004).

Table 7. Puget Sound Chinook salmon populations and selected measures of population viability

Population	Historical Abundance[a]	Mean Number of Spawners (Natural-origin)[b]	Percent Hatchery Contribution (Range)[c]	λ (+/- SE)[d]
Nooksack-North Fork	26,000	1,538 (125)	91 (88-95)	0.75 (0.07)
Nooksack-South Fork	13,000	338 (197)	40 (24-55)	0.94 (0.05)
Lower Skagit	22,000	2,527 (2,519)	0.2 (0-0.7)	1.05 (0.09)
Upper Skagit	35,000	9,489 (9,281)	2 (2-3)	1.05 (0.06)
Upper Cascade	1,700	274 (274)	0.3	1.06 (0.05)
Lower Sauk	7,800	601 (601)	0	1.01 (0.12)
Upper Sauk	4,200	324 (324)	0	0.96 (0.06)
Suiattle	830	365 (365)	0	0.99 (0.06)
Stillaguamish-North Fork	24,000	1,154 (671)	40 (13-52)	0.92 (0.04)
Stillaguamish-South Fork	20,000	270		0.99 (0.02)*
Skykomish	51,000	4,262 (2,392)	40 (11-66)	0.87 (0.03)
Snoqualmie	33,000	2,067(1,700)	16 (5-72)	1.00 (0.04)
North Lake Washington		331		1.07 (0.07)*
Cedar		327		0.99 (0.07)*
Green		8,884 (1,099)	83 (35-100)	0.67 (0.06)*
White		844		1.16 (0.06)*
Puyallup	33,000	1,653		0.95 (0.06)*
Nisqually	18,000	1,195		1.04 (0.07)*
Skokomish		1,392		1.04 (0.04)*
Dosewallips	4,700	48		1.17 (0.10)*

Duckabush		43	
Hamma Hamma		196	
Mid Hood Canal		311	
Dungeness	8,100	222	1.09 (0.11)*
Elwha		688	0.95 (0.11)*

[a]Estimated total historical abundance for this ESU was about 700,000 fish, but is not meant to reflect a summation of individual river historic estimates. Individual river estimates of historical abundance are based on an EDT analysis as reported in Good et al. 2005.
[b]5-year geometric mean number of spawners (hatchery plus natural) for years 1998-2002. Geometric mean of natural origin spawners noted in parentheses. From Good et al. 2005.
[c]Percent hatchery-origin from 1997-2001. Estimates are from the TRT database and reported in Good et al. 2005.
[d]Short-term median population growth rate estimates assume that the reproductive success of naturally spawning hatchery fish is equivalent to that of natural origin fish. Except estimates noted * where an estimate of the fraction of hatchery fish is not available then λ represents hatchery fish + natural-origin spawners. Data years used for calculation 1990-2002 (Good et al. 2005).

Status and Trends

NMFS listed Puget Sound Chinook salmon as threatened in 1999 (64 FR 14308); that status was reaffirmed on June 28, 2005 (70 FR 37160). This ESU has lost 15 spawning aggregations (nine from the early-run type) that were either independent historical populations or major components of the remaining 22 existing independent historical populations identified (Good et al. 2005). The disproportionate loss of early-run life history diversity represents a significant loss of the evolutionary legacy of the historical ESU.

Data reported by Good et al. (2005) indicate that long term trends in abundance for this ESU are split with about half of the populations declining, and the other half increasing. In contrast, the short-term trend for four populations is declining. The overall long-term trend in abundance indicates that, on average, populations are just replacing themselves. Estimates of the short-term median population growth rate (λ) (data years 1990-2002) indicate an even split between populations that are growing and those that are declining, although estimates would be lower for several populations if the fraction of naturally spawning hatchery fish were available for all populations within the ESU. For available data, when λ is calculated assuming that hatchery fish have the equivalent success of natural spawners then the largest estimated decline occurs in the Green River. Populations with the largest positive short and long-term trends include the White River and the North Fork Nooksack River (Good et al. 2005). Lambda for the Skagit River, which produces the most Chinook salmon in this ESU, has increased slightly. Overall, the recent analysis by Good et al. (2005) illustrated that there has not be much change in this ESU since NMFS' first status review (Busby et al. 1996). Individual populations have improved, while others have declined. However, the lack of information on the fraction of naturally spawning, hatchery-origin fish for 10 of the 22 populations within this ESU limits our understanding of the trends in naturally spawning fish for a large portion of the ESU.

The estimated total run size of Chinook salmon in Puget Sound in the early 1990s was 240,000 fish, representing a loss of nearly 450,000 fish from historic numbers. During a recent 5-year period, the geometric mean of natural spawners in populations of Puget Sound Chinook salmon ranged from 222 to just over 9,489 fish. Most populations had natural spawners numbering in the hundreds (median recent natural escapement is 766), and of the six populations with greater than 1,000 natural spawners, only two have a low fraction of hatchery fish. The populations with the greatest estimated component of hatchery fish tend to be in mid- to southern Puget Sound, Hood Canal, and the Strait of Juan de Fuca regions. Estimates of the historical equilibrium abundance, based on pre-European settlement habitat conditions, range from 1,700 to 51,000

potential Puget Sound Chinook salmon spawners per population. The historical estimates of spawner capacity are several orders of magnitude higher than spawner abundances currently observed throughout the ESU (Good et al. 2005).

Critical Habitat

NMFS designated critical habitat for Puget Sound Chinook salmon on September 2, 2005 (70 FR 52630). The specific geographic area includes portions of the Nooksack River, Skagit River, Sauk River, Stillaguamish River, Skykomish River, Snoqualmie River, Lake Washington, Green River, Puyallup River, White River, Nisqually River, Hamma Hamma River and other Hood Canal watersheds, the Dungeness/Elwha Watersheds, and nearshore marine areas of the Strait of Georgia, Puget Sound, Hood Canal and the Strait of Juan de Fuca. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high water line is not defined the lateral extent is defined as the bankfull elevation.

The designation for this ESU includes sites necessary to support one or more Chinook salmon life stages. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. Specific primary constituent elements include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat, and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. Of 49 subbasins (5th field Hydrological Units) reviewed in NMFS' assessment of critical habitat for the Puget Sound ESUs, nine subbasins were rated as having a medium conservation value, 12 were rated as low, and the remaining subbasins (40), where the bulk of Federal lands occur for this ESU, were rated as having a high conservation value to Puget Sound Chinook salmon. Factors contributing to the downward trends in this ESU are hydromorphological changes (such as diking, revetments, loss of secondary channels in floodplains, widespread blockages of streams, and changes in peak flows), degraded freshwater and marine habitat affected by agricultural activities and urbanization, and upper river tributaries widely affected by poor forest practices. Changes in habitat quantity, availability, diversity, flow, temperature, sediment load, and channel stability are common limiting factors in areas of critical habitat.

Sacramento River Winter-Run Chinook Salmon

Distribution and Description of the Listed Species

The Sacramento River winter-run Chinook salmon ESU includes all naturally spawned populations of winter-run Chinook salmon in the Sacramento River and its tributaries in California. Two artificial propagation programs are included in this ESU: winter-run Chinook salmon from the Livingston Stone National Fish Hatchery, and winter-run Chinook salmon in a captive broodstock program maintained at the Livingston Stone National Fish Hatchery and the University of California Bodega Marine Laboratory. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU.

This ESU consists of a single spawning population that enters the Sacramento River and its tributaries in California from November to June and spawns from late April to mid-August, with a peak from May to June (Table 8). Sacramento River winter-run Chinook salmon historically occupied cold, headwater streams, such as the upper reaches of the Little Sacramento, McCloud, and lower Pit Rivers. Young winter-run Chinook salmon venture to sea in November and December, after only four to seven months in fresh water (Groot et al. 1991).

Table 8. Sacramento River winter-run Chinook salmon abundance and selected measures of population viability

Population	Historical Abundance[a]	Mean number of Spawners (Range)[b]	Percent Hatchery Contribution	Population growth rate (λ)[c]
Sacramento River winter-run	200,000	2,191 (364-65,683)	<10	0.97 (0.87, 1.09)

[a]Historical abundance for the total ESU based on commercial fishery landings in the 1870s (Fisher 1994). Individual river estimates of historical abundance not provided.
[b]Recent geometric mean number of spawners from Good et al. 2005.
[c]Lambda value reported by Good et al. 2005. The 90% confidence intervals are noted in parentheses.

Status and Trends

NMFS listed Sacramento River winter-run Chinook salmon as endangered on January 4, 1994 (59 FR 440), and reaffirmed their status as endangered on June 28, 2005 (70 FR 37160), because dams restrict access to a small fraction of their historic spawning habitat and the habitat remaining to them is degraded. Sacramento River winter-run Chinook salmon consist of a single self-sustaining population which is entirely dependent upon the provision of suitably cool water from Shasta Reservoir during periods of spawning, incubation and rearing.

Construction of Shasta Dams in the 1940s eliminated access to historic spawning habitat for winter-run Chinook salmon in the basin. Winter-run Chinook salmon were not expected to survive this habitat alteration (Moffett 1949). However, cold water releases from Shasta Dam have created conditions suitable for winter Chinook salmon for roughly 60 miles downstream from the dam. As a result the ESU has been reduced to a single spawning population confined to the mainstem Sacramento River below Keswick Dam, although some adult winter-run Chinook salmon were recently observed in Battle Creek, a tributary to the upper Sacramento River.

Quantitative estimates of run-size are not available for the period before 1996, the completion of Red Bluff Diversion Dam. However, winter-runs may have been as large as 200,000 fish based upon commercial fishery records from the 1870s (Fisher 1994). The California Department of Fish and Game estimated spawning escapement of Sacramento River winter-run Chinook salmon at 61,300 (60,000 in the mainstem, 1,000 in Battle Creek, and 300 in Mill Creek) in the early 1960s. During the first 3 years of operation of the county facility at the Red Bluff Diversion Dam (1967 to 1969), the spawning run of winter-run Chinook salmon averaged 86,500 fish. From 1967 through the mid-1990s, the population declined at an average rate of 18% per year, or roughly 50% per generation. The population reached critically low levels during the drought of 1987 to 1992; the 3-year average run size for the period of 1989 to 1991 was 388 fish. Based on the Red Bluff Diversion Dam counts, the population has been growing rapidly since the 1990s. Mean run size from 1995-2000 has been 2,191, but have ranged from 364 to 65,683 (Good et al.

2005). Most recent estimates indicate that the short term trend is 0.26, while the population growth rate is still less than 1 (Table 8). The draft recovery goal for the ESU is an average of 10,000 female spawners per year and a population growth rate >1.0, calculated over 13 years of data (Good et al. 2005).

Critical Habitat

NMFS designated critical habitat for Sacramento River winter-run Chinook salmon on June 16, 1993 (58 FR 33212). The following areas consisting of the water, waterway bottom, and adjacent riparian zones: the Sacramento River from Keswick Dam, Shasta County (river mile 302) to Chipps Island (river mile 0) at the westward margin of the Sacramento-San Joaquin Delta, and other specified estuarine waters. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. Factors contributing to the downward trends in this ESU include reduced access to spawning/rearing habitat, possible loss of genetic integrity through population bottlenecks, inadequately screened diversions, predation at artificial structures and by nonnative species, pollution from Iron Mountain Mine and other sources, adverse flow conditions, high summer water temperatures, unsustainable harvest rates, passage problems at various structures, and vulnerability to drought (Good et al. 2005).

Snake River Fall-Run Chinook Salmon

Distribution and Description of the Listed Species

The Snake River fall-run Chinook salmon ESU includes all naturally spawned populations of fall-run Chinook salmon in the mainstem Snake River below Hells Canyon Dam, and in the Tucannon River, Grande Ronde River, Imnaha River, Salmon River, and Clearwater River subbasins. Four artificial propagation programs are part of this ESU: The Lyons Ferry Hatchery, Fall Chinook salmon Acclimation Ponds Program, Nez Perce Tribal Hatchery, and Oxbow Hatchery fall-run hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU.

Historically, the primary fall-run Chinook salmon spawning areas occurred on the upper mainstem Snake River (Connor et al. 2005). A series of Snake River dams blocked access to the upper reaches, which significantly reduced spawning and rearing habitat. Consequently, salmon now reside in waters that are generally cooler than pre-dam habitats. Currently, natural spawning occurs at the upper end of Lower Granite Reservoir to Hells Canyon Dam, the lower reaches of the Imnaha, Grande Ronde, Clearwater, and Tucannon rivers, and small mainstem sections in the tailraces of the lower Snake River hydroelectric dams.

Adult Snake River fall-run Chinook salmon enter the Columbia River in July and August, and spawning occurs from October through November. Juveniles emerge from the gravels in March and April of the following year, moving downstream from natal spawning and early rearing areas from June through early fall. Prior to dam construction, fall Chinook salmon were primarily ocean-type (migrated downstream and reared in the mainstem Snake River during their first year). However, today both an ocean-type and reservoir-type occur (Connor et al. 2005). The reservoir-type juveniles overwinter in pools created by dams before migrating to sea; this

response is likely due to early development in cooler temperatures which prevents rapid growth. Phenotypic characteristics have shifted in apparent response to environmental changes from hydroelectric dams (Connor et al. 2005). Migration downstream appears to be influenced by flow velocity within both river and reservoir systems (Tiffan et al. 2009).

Status and Trends

NMFS originally listed Snake River fall-run Chinook salmon as endangered in 1992 (57 FR 14653) but reclassified their status as threatened on June 28, 2005 (70 FR 37160). Estimated annual returns for the period 1938 to 1949 was 72,000 fish, and by the 1950s, numbers had declined to an annual average of 29,000 fish (Bjornn and Horner 1980). Numbers of Snake River fall-run Chinook salmon continued to decline during the 1960s and 1970s as approximately 80% of their historic habitat was eliminated or severely degraded by the construction of the Hells Canyon complex (1958 to 1967) and the lower Snake River dams (1961 to 1975). Counts of natural-origin adult Snake River fall-run Chinook salmon at Lower Granite Dam were 1,000 fish in 1975, and ranged from 78 to 905 fish (with an average of 489 fish) over the ensuing 25-year period (Good et al. 2005). Numbers of natural-origin Snake River fall-run Chinook salmon have increased over the last few years, with estimates at Lower Granite Dam of 2,652 fish in 2001, 2,095 fish in 2002, and 3,895 fish in 2003.

Snake River fall-run Chinook salmon have exhibited an upward trend in returns over Lower Granite Dam since the mid 1990s. Returns classified as natural-origin spawners exceeded 2,600 fish in 2001, compared to a 1997 to 2001 geometric mean natural-origin count of 871 (35% of the proposed delisting abundance criteria of 2,500 natural spawners averaged over 8 years). Both the long- and short-term trends in natural returns are positive. Harvest impacts on Snake River fall Chinook salmon declined after listing and have remained relatively constant in recent years. Mainstem conditions for subyearling Chinook migrants from the Snake River have generally improved since the early 1990s. The hatchery component, derived from outside the basin, has decreased as a percentage of the run at Lower Granite Dam from the 1998/99 status reviews (5-year average of 26.2%) to 2001 (8%). This reflects an increase in the Lyons Ferry component, systematic removal of marked hatchery fish at the Lower Granite trap, and modifications to the Umatilla supplementation program to increase homing of fall Chinook salmon release groups. Hatcheries stocking fish to the Snake River fall run produce genetic affects in the population due to three major components: natural-origin fish (which may be progeny of hatchery fish), returns of Snake River fish from the Lyons Ferry Hatchery program, and strays from hatchery programs outside the Snake River.

Critical Habitat

NMFS designated critical habitat for Snake River fall-run Chinook salmon on December 28, 1993 (58 FR 68543). This critical habitat encompasses the waters, waterway bottoms, and adjacent riparian zones of specified lakes and river reaches in the Columbia River that are or were accessible to listed Snake River salmon (except reaches above impassable natural falls, and Dworshak and Hells Canyon Dams). These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. Adjacent riparian zones are defined as those areas within a horizontal distance of 300 feet from the normal line of high water of a stream channel or from the shoreline of a standing body of water. Designated critical habitat includes the Columbia River from a straight line connecting the west end of the Clatsop

jetty (Oregon side) and the west end of the Peacock jetty (Washington side) and including all river reaches from the estuary upstream to the confluence of the Snake River, and all Snake River reaches upstream to Hells Canyon Dam. Critical habitat also includes several river reaches presently or historically accessible to Snake River fall-run Chinook salmon. Limiting factors identified for Snake River fall-run Chinook salmon include: mainstem lower Snake and Columbia hydrosystem mortality, degraded water quality, reduced spawning and rearing habitat due to mainstem lower Snake River hydropower system, harvest impacts, impaired stream flows, barriers to fish passage in tributaries, excessive sediment, and altered floodplain and channel morphology (NMFS 2005a).

Snake River Spring/Summer-Run Chinook Salmon

Distribution and Description of the Listed Species

The Snake River spring/summer-run Chinook salmon ESU includes all naturally spawned populations of spring/summer-run Chinook salmon in the mainstem Snake River and the Tucannon River, Grande Ronde River, Imnaha River, and Salmon River subbasins. Fifteen artificial propagation programs are part of the ESU: The Tucannon River conventional Hatchery, Tucannon River Captive Broodstock Program, Lostine River, Catherine Creek, Lookingglass Hatchery Reintroduction Program (Catherine Creek), Upper Grande Ronde, Imnaha River, Big Sheep Creek, McCall Hatchery, Johnson Creek Artificial Propagation Enhancement, Lemhi River Captive Rearing Experiment, Pahsimeroi Hatchery, East Fork Captive Rearing Experiment, West Fork Yankee Fork Captive Rearing Experiment, and the Sawtooth Hatchery spring/summer-run Chinook salmon hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU. The Interior Columbia Basin Technical Recovery Team has identified 32 populations in five major population groups (Upper Salmon River, South Fork Salmon River, Middle Fork Salmon River, Grande Ronde/Imnaha, Lower Snake Mainstem Tributaries) for this species. Historic populations above Hells Canyon Dam are considered extinct (ICBTRT 2003). Table 9 identifies extant populations within the Snake River spring/summer Chinook salmon ESU, their abundances, and the relative contribution of hatchery fish.

Snake River spring/summer-run Chinook salmon have a stream-type life history. Spawning occurs in late summer and early fall and eggs incubate over the following winter and hatch in late winter and early spring of the following year. Juveniles mature in the river for one year before migrating to the ocean in the spring of their second year. Larger outmigrants have a higher survival rate during outmigration (Zabel and Williams 2002; Zabel and Achord 2004). Depending on tributary and the specific habitat conditions, juveniles may migrate widely from natal reaches into alternative summer-rearing or overwintering areas. Spawners return to spawn primarily as 4- and 5-year-olds after 2 to 3 years in the ocean. A small fraction return as 3-year-old “jacks” (although sexually mature upon return, these fish are smaller in body and 1-2 years younger than most males on the spawning ground).

Table 9. Snake River spring/summer Chinook salmon populations and selected measures of population viability

Current Populations[c]	Mean Number of Spawners (Range)[a]	Percent Hatchery Contribution[b]	Short-term Trend (Previous)[c]
Tucannon River	303 (128-1,012)	76	-4.1 (-11.0)
Wenaha River	225 (67-586)	64	-9.4 (-23.6)
Wallowa River	0.57 redds (0-29)	5	11.5
Lostine River	34 redds (9-131)	5	12.7
Minam River	180 (96-573)	5	3.3 (-14.5)
Catherine Creek	50 (13-262)	56	-25.1 (-22.5)
Upper Grande Ronde River	46 (3-336)	58	-9.4
South Fork Salmon River	496 redds (277-679)	9	1.1 (-13.6)
Secesh River	144 redds (38-444)	4	9.8
Johnson Creek	131 redds (49-444)		-1.5
Big Creek spring run	53 (21-296)		5.4 (-34.2)
Big Creek summer run	5 redds (2-58)		1.7 (-27.9)
Loon Creek	27 redds (6-255)		12.2
Marsh Creek	53 (0-164)		-4.0
Bear Valley/Elk Creek	266 (72-712)		6.2
North Fork Salmon River	5.6 redds (2-19)		
Lemhi River	72 redds (35-216)		12.8 (-27.4)
Pahsimeroi River	161 (72-1,097)		12.8
East Fork Salmon spring run	0.27 rpm (0.2-1.41)		-5.7
East Fork Salmon summer run	1.22 rpm 0.35-5.32)		0.9 (-32.9)
Yankee Fork spring run	0 rpm		-6.3
Yankee Fork summer run	2.9 redds (1-18)		4.1
Valley Creek spring run	7.4 redds (2-28)		14.9 (-25.9)
Valley Creek summer run	2.14 rpm(0.71-9.29)		5.8 (-29.3
Upper Salmon spring run	69 redds (25-357)		5.3
Upper Salmon summer run	0.24 rpm (0.07-0.58)		-3.3
Alturas Lake Creek	2.7 redds (0-18)		10.2
Imnaha River	564 redds (194-3,041)	62	12.8(-24.1)
Big Sheep Creek	0.25 redds (0-1)	97	0.8
Lick Creek	1.4 redds (0-29)	59	11.7

[a]All data reported in Good et al. 2005. Except where noted values represent the recent geometric mean number of spawners. RPM =redds per mile.
[bc]Reported in Good et al. 2005.
[c]For details on data series used in calculating the population's short term trend see Good et al. 2005.

Status and Trends

NMFS originally listed Snake River spring/summer-run Chinook salmon as threatened on April 22, 1992 (57 FR 14653), and reaffirmed their status as threatened on June 28, 2005 (70 FR 37160). Although direct estimates of historical annual Snake River spring/summer Chinook salmon returns are not available, returns may have declined by as much as 97% between the late 1800s and 2000. According to Matthews and Waples (1991), total annual Snake River spring/summer Chinook salmon production may have exceeded 1.5 million adult fish in the late 1800s. Total (natural plus hatchery origin) returns fell to roughly 100,000 spawners by the late 1960s and were below 10,000 by 1980 (Fulton 1968). Between 1981 and 2000, total returns fluctuated between extremes of 1,800 and 44,000 fish. The 2001 and 2002 total returns increased to over 185,000 and 97,184 adults, respectively. The 1997 to 2001 geometric mean total return for the summer run component at Lower Granite Dam was slightly more than 6,000 fish,

compared to the geometric mean of 3,076 fish for the years 1987 to 1996. The 2002 to 2006 geometric mean of the combined Chinook salmon runs at Lower Granite Dam was over 18,000 fish. However, it is important to note that over 80% of the 2001 return and over 60% of the 2002 return originated in hatcheries (Good et al. 2005). Good et al. (2005) reported that risks to individual populations within the ESU may be greater than the extinction risk for the entire ESU due to low levels of annual abundance and the extensive production areas within the Snake River basin. Although the average abundance in the most recent decade is more abundant than the previous decade, there is no obvious long-term trend.

Critical Habitat

NMFS designated critical habitat for Snake River spring/summer-run Chinook salmon on October 25, 1999 (64 FR 57399). This critical habitat encompasses the waters, waterway bottoms, and adjacent riparian zones of specified lakes and river reaches in the Columbia River that are or were accessible to listed Snake River salmon (except reaches above impassable natural falls, and Dworshak and Hells Canyon Dams). Adjacent riparian zones are defined as those areas within a horizontal distance of 300 feet from the normal line of high water of a stream channel or from the shoreline of a standing body of water. Designated critical habitat includes the Columbia River from a straight line connecting the west end of the Clatsop jetty (Oregon side) and the west end of the Peacock jetty (Washington side) and including all river reaches from the estuary upstream to the confluence of the Snake River, and all Snake River reaches upstream to Hells Canyon Dam; the Palouse River from its confluence with the Snake River upstream to Palouse Falls, the Clearwater River from its confluence with the Snake River upstream to its confluence with Lolo Creek; the North Fork Clearwater River from its confluence with the Clearwater river upstream to Dworshak Dam. Critical habitat also includes several river reaches presently or historically accessible to Snake River spring/summer Chinook salmon. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. Limiting factors identified for this species include hydrosystem mortality, reduced stream flow, altered channel morphology and floodplain, excessive fine sediment, and degraded water quality (NMFS 2006c).

Upper Willamette River Chinook Salmon

Distribution and Description of the Listed Species

The Upper Willamette River Chinook salmon ESU includes all naturally spawned populations of spring-run Chinook salmon in the Clackamas River and in the Willamette River, and its tributaries, above Willamette Falls, Oregon. Seven artificial propagation programs are part of the ESU: The McKenzie River Hatchery, Marion Forks/North Fork Santiam River, South Santiam Hatchery in the South Fork Santiam River, South Santiam Hatchery in the Calapooia River, South Santiam Hatchery in the Mollala River, Willamette Hatchery, and Clackamas hatchery spring-run Chinook salmon hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU.

Upper Willamette River Chinook salmon occupy the Willamette River and its tributaries. All spring-run Chinook salmon in the ESU, except those entering the Clackamas River, must pass

Willamette Falls. In the past, this ESU included sizable numbers of spawning salmon in the Santiam River, the middle fork of the Willamette River, and the McKenzie River, as well as smaller numbers in the Molalla River, Calapooia River, and Albiqua Creek. Historically, access above Willamette Falls was restricted to the spring when flows were high. In autumn, low flows prevented fish from ascending past the falls. The Upper Willamette spring-run Chinook salmon are one of the most genetically distinct Chinook salmon groups in the Columbia River Basin. Upper Willamette River Chinook salmon enter the Columbia River and estuary earlier than other spring Chinook salmon ESUs (Meyers et al. 1998). Fall-run Chinook salmon spawn in the Upper Willamette but are not considered part of the ESU because they are not native.

Status and Trends

NMFS originally listed Upper Willamette River Chinook salmon as threatened on March 24, 1999 (64 FR 14308), and reaffirmed their status as threatened on June 28, 2005 (70 FR 37160). The total abundance of adult spring-run Chinook salmon (hatchery-origin plus natural-origin fish) passing Willamette Falls has remained relatively steady over the past 50 years (ranging from approximately 20,000 to 70,000 fish), but it is an order of magnitude below the peak abundance levels observed in the 1920s (approximately 300,000 adults). Until recent years, interpretation of abundance levels has been confounded by a high but uncertain fraction of hatchery-produced fish. Although the number of adult spring-run Chinook salmon crossing Willamette Falls is in the same range (about 20,000 to 70,000 adults) it has been for the last 50 years, a large fraction of these are hatchery produced. Estimates of the percentage of hatchery fish range according to tributary, several of which exceed 70 percent (Good et al. 2005). The Calapooia River is estimated to contain 100 percent hatchery fish. Insufficient information on hatchery production in the past prevents a meaningful analysis of the population trend; therefore no formal trend analysis is available.

Most natural spring Chinook salmon populations of the Upper Willamette River are likely extirpated or nearly so, with only one remaining naturally reproducing population identified in this ESU: the spring Chinook salmon in the McKenzie River. Unfortunately, recently short-term declines in abundance suggest that this population may not be self-sustaining (Myers et al. 1998; Good et al. 2005). Abundance in this population has been relatively low (low thousands) with a substantial number of these fish being of hatchery origin. The population increased substantially from 2000 to 2003, probably due to increased survival in the ocean. Future survival rates in the ocean are unpredictable, and the likelihood of long-term sustainability for this population has not been determined. Of concern is that a majority of the spawning habitat and approximately 30 to 40% of total historical habitat are no longer accessible because of dams (Good et al. 2005). Individuals from the ESU migrate far north and are caught incidentally in ocean fisheries, particularly off southeast Alaska and northern Canada, and in the mainstem Columbia and Willamette rivers during spring.

Critical Habitat

NMFS designated critical habitat for Upper Willamette River Chinook salmon on September 2, 2005 (70 FR 52630). Critical habitat for upper Willamette River Chinook salmon includes defined areas within subbasins of the middle fork Willamette River, upper Willamette River, McKenzie River, Santiam River, Crabtree Creek, Molalla River, and Clackamas River. This

designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. The critical habitat designation for this ESU identifies primary constituent elements that include sites necessary to support one or more Chinook salmon life stages. Specific sites include freshwater spawning and rearing sites, freshwater migration corridors. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. Of 65 subbasins reviewed in NMFS' assessment of critical habitat for the Upper Willamette River Chinook salmon ESU, 19 subbasins were rated as having a medium conservation value, 19 were rated as low, and the 27 remaining subbasins were rated as having a high conservation value to Upper Willamette River Chinook salmon. Federal lands were generally rated as having high conservation value to the species' spawning and rearing. Factors contributing to the downward trends in this ESU include reduced access to spawning/rearing habitat in tributaries, hatchery impacts, altered water quality and temperature in tributaries, altered stream flow in tributaries, and lost or degraded floodplain connectivity and lowland stream habitat.

Chum Salmon

Description of the Species

Chum salmon are more widely distributed than other salmon, and may have at one time made up nearly 50% of the Pacific salmon biomass in the Pacific Ocean (Salo 1991). Historically, chum salmon were distributed throughout the coastal regions of western Canada and the United States, as far south as Monterey Bay, California, to the Arctic coast and east to the Mackenzie River, in the Beaufort Sea. They also ranged in Asia from Korea to the Arctic coast of the Soviet Union and west to the Lena River. Presently, major spawning populations on the west coast of the United States are found only as far south as Tillamook Bay on the northern Oregon coast. In this section of our Opinion, we discuss the distribution, status, and critical habitats of the two listed species of threatened chum salmon separately; however, because chum salmon in the wild are virtually indistinguishable between listed ESUs, and are the same biological species sharing the same generalized life history, we begin this section describing those characteristics common across ESUs.

Chum salmon exhibit obligatory anadromy (there are no recorded landlocked or naturalized freshwater populations), and like Chinook salmon, chum salmon are semelparous so they die after one spawning event. Their general life cycle spans fresh and marine waters, although chum salmon are more marine oriented than the other Pacific salmon, in that they spend very little time rearing in fresh water. Chum salmon spend 2 to 5 years in feeding areas in the northeast Pacific Ocean, which is a greater proportion of their life history than other Pacific salmonids. Chum salmon distribute throughout the North Pacific Ocean and Bering Sea, although North American chum salmon (as opposed to chum salmon originating in Asia), rarely occur west of 175° E longitude. North American chum salmon migrate north along the coast in a narrow coastal band that broadens in southeastern Alaska, although some data suggest that Puget Sound chum, including Hood Canal summer run chum, may not make extended migrations into northern British Columbian and Alaskan waters, but instead may travel directly offshore into the north

Pacific Ocean.

Spawning migrations generally occur in the summer and fall; the precise spawn timing and migration varies across populations. Stream flows and water temperatures can influence stream entry. Sexual differences in the timing of returns to spawning grounds are apparent with males generally arriving early and females later in the run. Once on the spawning grounds mate competition is intense with males competing to fertilize eggs and females competing for optimal nest site selection. Size and age at maturity is partially under genetic control, but can be influenced by environment and migration behavior. Generally, spawning runs consist of fish between 2 and 5 years of age, and like Chinook salmon, chum females will build large redds that consist of four or five egg pockets laid in succession. Chum salmon fecundity is highly variable, and is correlated with body size and region (latitudinal trends are evident with northern population having lower absolute and relative fecundities; Salo 1991).

The time necessary for egg incubation until emergence of alevins in fresh water varies among basins and among years within a basin, and is closely correlated to water temperatures such that low temperatures prolong incubation. Egg and alevin survival, and the fitness of emerging fry are affected by sediment loading, intergravel water flow and dissolved oxygen levels, gravel composition, spawning time and density, and water temperatures. Once they emerge from their gravel nests, chum salmon fry outmigrate to seawater almost immediately (Salo 1991). This ocean-type migratory behavior contrasts with the stream-type behavior of other species in the genus Oncorhynchus (e.g., coastal cutthroat trout, steelhead, coho salmon, and most types of Chinook and sockeye salmon, exception pink salmon), which usually migrate to sea at a larger size, after months or years of freshwater rearing. Because of their small size chum salmon will form loosely aggregated schools, presumably to reduce predation by swamping predators (Miller and Brannon 1982; Pitcher 1986).

Generally, chum fry emigrate to estuaries between March through May where they forage on epibenthic and neritic food resources. The timing of juvenile entry into sea water is commonly correlated with nearshore warming and associated plankton blooms (Groot et al. 1991). As food resources decline and the fish grow, they move further out to forage on pelagic and nektonic organisms (Simenstad and Salo 1982; Salo 1991). Migratory studies indicate that chum salmon in their first year of life will typically maintain a coastal migratory pattern although the pattern is variable as they mature at sea. At sea chum salmon feed on pteropods, euphausiids, amphipods, fish and squid larvae (Salo 1991).

Threats

Natural Threats. Chum salmon are exposed to high rates of natural predation each stage of their life stage, and in particular during migration. Mortality at emergence or prior to emergence is significant because eggs develop in the interstitial spaces in the stream gravel, and storm surges that redeposit gravels and wash out eggs or introduce silt to the interstitial spaces can reduce egg survival. Other factors that reduce egg survival and larvae development include low dissolved oxygen, poor percolation, and extreme cold or warm temperatures.

Anthropogenic Threats. Chum salmon, like the other listed salmon, have declined under the combined effects of overharvests in fisheries; competition from fish raised in hatcheries and

native and non-native exotic species; dams that block their migrations and alter river hydrology; gravel mining that impedes their migration and alters the dynamics (hydrogeomorphology) of the rivers and streams that support juveniles; water diversions that deplete water levels in rivers and streams; destruction or degradation of riparian habitat that increase water temperatures in rivers and streams sufficient to reduce the survival of juvenile chum salmon; and land use practices (logging, agriculture, urbanization) that destroy wetland and riparian ecosystems while introducing sediment, nutrients, biocides, metals, and other pollutants into surface and ground water and degrade water quality in the fresh water, estuarine, and coastal ecosystems throughout the Pacific Northwest. These threats for are summarized in detail under Chinook salmon.

Columbia River Chum Salmon

Distribution and Description of the Listed Species

The Columbia River chum ESU includes all naturally spawned populations of chum salmon in the Columbia River and its tributaries in Washington and Oregon. Three artificial propagation programs are part of the ESU: The Chinook River (Sea Resources Hatchery), Grays River, and Washougal River/Duncan Creek chum hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU.

Most of the chum within this ESU return to northern tributaries of the Columbia River (in Washington State), primarily the Grays River, in areas immediately below Bonneville Dam, and in smaller numbers under the I-205 bridge near Vancouver. Chum populations that formerly occupied tributaries on the south bank of the Columbia (in Oregon) are considered extirpated or nearly so. Observers have documented spawning over multiple years in the mainstem Columbia River, near McCord Creek and Multnomah Falls in Oregon, although the number of spawners in these areas are generally quite low (McElhany et al. 2007).

Chum salmon return to the Columbia River in late fall (mid-October to December).

Table 10. Columbia River chum salmon populations and selected measures of population viability

Current Populations	Historical Abundance[a]	Recent Spawner Abundance	Short-Term Median Growth Rate (λ)[c]
Youngs Bay			
Gray's River	7,511	331/704[b]	1.043 (0.957-1.137)
Big Creek			
Elochoman River			
Clatskanie River			
Mill, Abernathy, and Germany Creeks			
Scappoose Creek			
Cowlitz River	141,582		
Kalama River	9,953		
Lewis River	89,671		
Salmon Creek			
Clackamus River			
Sandy River			
Washougal River	15,140		

Lower gorge tributaries	>3,141	425[b]	0.984 (0.883-1.096)
Upper gorge tributaries	>8,912		

[a]Estimated total historical abundance for this ESU was about 283,421 fish, but is not meant to reflect a summation of individual river historic estimates. Individual river estimates of historical abundance are based on an EDT analysis using equilibrium abundance under historical conditions. All data are reported in Good et al. 2005.

[b]Two different time series estimate are available but based on overlapping years. The first estimate is based on 1996-2000 data, while the second is based on 1996-2000 data.

[c]The λ calculation is an estimate of what the natural growth rate would have been after accounting for hatchery-origin spawners. Two different estimates are available for the Grays River population; the Rawlings estimate (depicted in the table above) is believed to be more accurate. Other estimates, long- and short-term trends, suggest the population is declining (see Good et al. 2005).

Status and Trends

NMFS listed Columbia River chum salmon as threatened on March 25, 1999, and reaffirmed their threatened status on June 28, 2005 (71 FR 37160). Chum salmon in the Columbia River once numbered in the hundreds of thousands of adults and were reported in almost every river in the Lower Columbia River basin, but by the 1950s most runs disappeared (Rich 1942; Marr 1943; Fulton 1970). The total number of chum salmon returning to the Columbia River in the last 50 years has averaged a few thousand per year, with returns limited to a very restricted portion of the historical range. Significant spawning occurs in only two of the 16 historical populations, meaning that 88% of the historical populations are extirpated, or nearly so. The two remaining populations are the Grays River and the lower Columbia Gorge tributaries (Good et al. 2005). Both long- and short-term trends for Grays River abundance are negative, but the current trend in abundance for the lower Columbia Gorge tributaries is slightly positive. Chum salmon appear to be extirpated from the Oregon portion of this ESU. In 2000, ODFW conducted surveys to determine the abundance and distribution of chum salmon in the Columbia River, and out of 30 sites surveyed, only one chum salmon was observed.

Few Columbia River chum salmon have been observed in tributaries between The Dalles and Bonneville dams. Surveys of the White Salmon River in 2002 found one male and one female carcass, with no evidence of spawning (Ehlke and Keller 2003). Chum salmon were not observed in any upper Columbia Gorge tributaries during the 2003 and 2004 spawning ground surveys. Finally, most Columbia River chum populations have been functionally extirpated or are presently at very low abundance levels.

Historically, the Columbia River chum salmon supported a large commercial fishery in the first half of this century which landed more than 500,000 fish per year as recently as 1942. Commercial catches declined beginning in the mid-1950s, and in later years rarely exceeded 2,000 per year. During the 1980s and 1990s, the combined abundance of natural spawners for the lower Columbia Gorge, Washougal, and Grays River populations was below 4,000 adults. In 2002, however, the abundance of natural spawners exhibited a substantial increase at several locations (estimate of natural spawners is approximately 20,000 adults). The cause of this dramatic increase in abundance is unknown. However, long- and short-term productivity trends for populations are at or below replacement. The loss of off-channel habitat and the extirpation of approximately 17 historical populations increase this species' vulnerability to environmental variability and catastrophic events. Overall, the populations that remain have low abundance, limited distribution, and poor connectivity (Good et al. 2005).

Critical Habitat

NMFS designated critical habitat for Columbia River chum salmon on September 2, 2005 (70 FR 52630). The designated includes defined areas in the following subbasins: Middle Columbia/Hood, Lower Columbia/Sandy, Lewis, Lower Columbia/Clatskanie, Lower Cowlitz, Lower Columbia subbasin and river corridor. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation.

The critical habitat designation for this ESU identifies primary constituent elements that include sites necessary to support one or more chum salmon life stages. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding and are rated as having high conservation value to the species. Columbia River chum salmon have primary constituent elements of freshwater spawning, freshwater rearing, freshwater migration, estuarine areas free of obstruction, nearshore marine areas free of obstructions, and offshore marine areas with good water quality. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. Of 21 subbasins reviewed in NMFS' assessment of critical habitat for the Columbia River chum salmon ESU, three subbasins were rated as having a medium conservation value, no subbasins were rated as low, and the majority of subbasins (18), were rated as having a high conservation value to Columbia River chum salmon. The major factors limiting recovery for Columbia River chum salmon are altered channel form and stability in tributaries, excessive sediment in tributary spawning gravels, altered stream flow in tributaries and the mainstem Columbia River, loss of some tributary habitat types, and harassment of spawners in the tributaries and mainstem.

Hood Canal Summer-Run Chum Salmon

Distribution and Description of the Listed Species

The Hood Canal summer-run chum salmon ESU includes all naturally spawned populations of summer-run chum salmon in Hood Canal and its tributaries as well as populations in Olympic Peninsula rivers between Hood Canal and Dungeness Bay, Washington (64 FR 14508) from mid-September to mid-October (WDF (Washington Department of Fisheries) 1993), but may enter natal rivers in late August. Eight artificial propagation programs are considered to be part of the ESU: the Quilcene National Fish Hatchery, Hamma Hamma Fish Hatchery, Lilliwaup Creek Fish Hatchery, Union River/Tahuya, Big Beef Creek Fish Hatchery, Salmon Creek Fish Hatchery, Chimacum Creek Fish Hatchery, and the Jimmycomelately Creek Fish Hatchery summer-run chum hatchery programs. NMFS determined that these artificially propagated populations are no more divergent relative to the local natural population(s) than what would be expected between closely related natural populations within the species. Table 11 identifies populations within the Hood Canal summer-run chum salmon ESU, their abundances, and hatchery input.

On average Hood Canal chum salmon reside in estuaries for 23 days; daily tidal migrations have not been observed, but prey availability does influence movement patterns (Bax 1983). Upon leaving their natal estuaries summer-run chum salmon generally migrate through Hood Canal and

into the main body of Puget Sound.

Status and Trends

NMFS listed Hood Canal summer-run chum salmon as threatened on March 25, 1999 (64 FR 14508), and reaffirmed as threatened on June 28, 2005 (70 FR 37160). Historically, Hood Canal summer-run chum salmon comprised an estimated 16 populations. Only eight extant populations remain within this ESU (Good et al. 2005). Most of the extirpated populations historically occurred on the eastern side of Hood Canal, which is cause for concern over the current spatial structure of this ESU. The widespread loss of estuary and lower floodplain habitat is a continuing threat to ESU spatial structure and connectivity.

Although many population remain adult returns for some populations showed modest improvements in 2000, with upward trends continuing in 2001 and 2002. The recent 5-year mean abundance is variable among populations in the species, ranging from one fish to nearly 4,500 fish in the Big/Little Quilcene rivers. Hood Canal summer-run chum are the focus of an extensive rebuilding program developed and implemented since 1992 by the state and tribal comanagers. Two populations (the combined Quilcene and Union River populations) are above the conservation thresholds established by the rebuilding plan. However, most populations remain depressed. Estimates of the fraction of naturally spawning hatchery fish exceed 60% for some populations, indicating that reintroduction programs are supplementing the numbers of total fish spawning naturally in streams. Long-term trends in productivity are above replacement for only the Quilcene and Union River populations. Buoyed by recent increases, seven populations are exhibiting short-term productivity trends above replacement.

Table 11. Hood Canal summer-run chum populations and selected measures of population viability

Populations [a]	1999-2002 Mean Escapement (range)	Percent Hatchery Contributions (1995-2001)	λ (+/- SE)
Jimmycomelately Creek	10 (1-192)		0.85 (0.16)
Salmon/Snow creeks	1,521 (463-5,921)	0-69	1.23 (0.10)
Big/Little Quilcene rivers	4,512 (3,065-6,067)	5-51	1.39(0.22)
Lilliwaup Creek	13 (1-775)		1.19 (0.44)
Hamma Hamma River	558 (173-2,260)		1.3 (0.19)
Duckabush River	382 (92-942)		1.1 (0.17)
Dosewallips River	919 (351-1,627)		1.17 (0.24)
Union River			1.15 (0.10))
Chimacum Creek*	198 (0-903)[c]	100	
Big Beef Creek*	17 (0-826)[c]	100	
Dewatto Creek*	9 (2-32)[d]		

[a]All data is reported in Good et al. 2005. * Denotes extinct populations that have recently had some natural recolonization or have been seeded with hatchery fish.

Of the eight programs releasing summer-run chum salmon that are considered to be part of the Hood Canal summer chum ESU, six of the programs are supplementation programs implemented to preserve and increase the abundance of native populations in their natal watersheds. NMFS' assessment of the effects of artificial propagation on ESU extinction risk concluded that these hatchery programs collectively do not substantially reduce the extinction risk of the ESU. The hatchery programs are reducing risks to ESU abundance by increasing total ESU abundance as

well as the number of naturally spawning summer-run chum salmon.

Critical Habitat

NMFS designated critical habitat for Hood Canal summer-run chum salmon on September 2, 2005 (70 FR 52630). The specific geographic area includes the Skokomish River, Hood Canal subbasin, which includes the Hamma Hamma and Dosewallips rivers and others, the Puget Sound subbasin, Dungeness/Elwha subbasin, and nearshore marine areas of Hood Canal and the Strait of Juan de Fuca from the line of extreme high tide to a depth of 30 meters. This includes a narrow nearshore zone from the extreme high-tide to mean lower low tide within several Navy security/restricted zones. This also includes about 8 miles of habitat that was unoccupied at the time of the designation Finch, Anderson and Chimacum creeks (69 FR 74572; 70 FR 52630), but has recently been re-seeded. Chimacum Creek, however, has been naturally recolonized since at least 2007 (T. Johnson, pers. comm., Jan. 2010). The designation for Hood Canal summer-run chum, like others made at this time, includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation.

The specific primary constituent elements identified for Hood Canal summer-run chum salmon are areas for spawning, freshwater rearing and migration, estuarine areas free of obstruction, nearshore marine areas free of obstructions, and offshore marine areas with good water quality. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. Of 17 subbasins reviewed in NMFS' assessment of critical habitat for the Hood Canal chum salmon ESU, 14 subbasins were rated as having a high conservation value, while only three were rated as having a medium value to conservation. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. Limiting factors identified for this species include degraded floodplain and mainstem river channel structure, degraded estuarine conditions and loss of estuarine habitat, riparian area degradation and loss of in-river wood in mainstem, excessive sediment in spawning gravels, and reduced stream flow in migration areas.

Coho Salmon

Description of the Species

Coho salmon occur naturally in most major river basins around the North Pacific Ocean from central California to northern Japan (Laufle et al. 1986). The typical life history of coho salmon is similar to most of the other large bodied Pacific salmonids, in as much as adult fish spawn in the fall and winter, young emerge in the spring, rear in fresh water and saltwater and return to spawn as adults. Sympatric in many river basins with Chinook, chum, sockeye, and pink salmon, partitioning occurs through the species use of different areas of a river for reproduction and rearing, and the length of time they spend in these ecosystems. For instance, Chinook salmon spawn in fast flowing mainstem riverine reaches with large substrate; sockeye salmon spawn in rivers and lakes, and chum salmon spawn in mid- to lower reaches of rivers and have been observed spawning in areas of tidal influence. Coho salmon characteristically spawn in tributaries and slow-flowing shallow creeks in tributaries with gradients of three percent or less,

which may be fed by cool groundwater sources, and are often widely dispersed within watershed. Adult coho salmon may remain in fresh water three or more months before spawning, with early migrants often moving farther upstream than later migrants (Sandercock 1991).

Most coho salmon enter rivers between September and February, but entry is influenced by discharge and other factors. In many river systems, coho salmon and other Pacific salmon are unable to enter the rivers until sufficiently strong flows open passages and provide sufficient depth. First fall freshets combined with high tides triggers the upstream migration of coho salmon in many basins. Until then, if river flows are low or warm summer temperatures persist, fish may congregate in pools near the mouth of the river or natal stream until conditions change. Typically coho salmon spawn from November to January, although there are many exceptions throughout their range. Spawning duration usually spans about three months in most basins, with individual fish actively spawning for several days to weeks. Spawning occurs in a few third-order streams, but most spawning activity occurs in fourth- and fifth-order streams. As with other Pacific salmon, coho salmon fecundity varies with the size of the fish and latitudinally with coho salmon in northern climes generally exhibiting higher rates of fecundity (Sandercock 1991). Most coho salmon mature and spawn at age 3, although there are exceptions; in many basins in the northern portion of the species' range coho salmon spawn at age 2.

Rates of incubation are largely temperature dependent: colder water temperatures will slow the rate of development. Generally, in optimal temperatures eggs incubate for about 35 to 50 days, and fry start emerging from the gravel two to three weeks after hatching. Incubation and emergence success are also influenced by dissolved oxygen levels, sediment loading, and scouring high flows. Following emergence, fry aggregate and move to shallow areas near the stream banks. Most coho salmon rear in fresh water for about 15 to 18 months. As fry grow, they disperse upstream and downstream to establish and defend territories. Juvenile rearing usually occurs in tributaries with gradients of three percent or less, although they may move to streams with gradients of four to five percent. Juvenile coho salmon are often found in small streams less than five feet wide, and may migrate considerable distances to rear in lakes and off-channel ponds. During the summer, fry prefer pools featuring adequate cover such as large woody debris, undercut banks, and overhanging vegetation. Overwintering tends to occur in larger pools, backwater areas and off stream channels and ponds (e.g., wall-based channels that are groundwater fed).

At not quite 2 years of age, coho salmon will migrate downstream where they, like other anadromous fish, undergo the physiological transition to salt water. The outmigration of coho smolts begins as early as February and may continue through the summer and fall, with peak outmigration often between March and June, although this varies among basins and environmental conditions (Sandercock 1991). Once in the ocean, coho salmon generally migrate north along the coast in a narrow coastal band that broadens in southeastern Alaska. During this migration, juvenile coho salmon tend to occur in both coastal and offshore waters. During spring and summer, coho salmon will forage in waters between 46º N, the Gulf of Alaska, and along Alaska's Aleutian Islands.

Coho salmon, like many other salmon, are opportunistic feeders. While at sea, coho salmon tend to eat fish including herring, sand lance, sticklebacks, sardines, shrimp and surf smelt. While in

estuaries and in fresh water coho salmon are significant predators of Chinook, pink, and chum salmon, as well as aquatic and terrestrial insects. Smaller fish, such as fry, eat chironomids, plecoptera, and other larval insects, and typically use visual cues to find their prey.

Threats

Natural Threats. Coho salmon, like other salmon, are exposed to high rates of natural predation at each life stage. Most mortality, however, occurs in the freshwater life stages. Winter mortality may be significant for coho salmon because they overwinter in fresh water, where they can be swept downstream from freshets or eaten by raccoon, cutthroat trout, or other small animals. Once coho reach the ocean, survival is high (Sandercock 1991).

Anthropogenic Threats. Coho salmon have declined under the combined effects of overharvests in fisheries; competition from fish raised in hatcheries and native and non-native exotic species; dams that block their migrations and alter river hydrology; gravel mining that impedes their migration and alters the dynamics (hydrogeomorphology) of the rivers and streams that support juveniles; water diversions that deplete water levels in rivers and streams; destruction or degradation of riparian habitat that increase water temperatures in rivers and streams sufficient to reduce the survival of juvenile coho salmon; and land use practices (logging, agriculture, urbanization) that destroy wetland and riparian ecosystems while introducing sediment, nutrients, biocides, metals, and other pollutants into surface and ground water and degrade water quality in the fresh water, estuarine, and coastal ecosystems throughout the species' range. These threats for are summarized in detail under Chinook salmon.

Central California Coast Coho Salmon

Distribution and Description of the Listed Species

The Central California Coast coho salmon ESU extends from Punta Gorda in northern California south to and including the San Lorenzo River in central California (Weitkamp et al. 1995). The ESU includes all naturally spawned populations of coho salmon from Punta Gorda in northern California south to and including the San Lorenzo River in central California, as well as populations in tributaries to San Francisco Bay, excluding the Sacramento-San Joaquin River system. Four artificial propagation programs are part of the Central California Coast coho salmon ESU: the Don Clausen Fish Hatchery Captive Broodstock Program, Scott Creek/King Fisher Flats Conservation Program, Scott Creek Captive Broodstock Program, and the Noyo River Fish Station egg-take Program coho hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this ESU.

Coho salmon in this ESU enter rivers to spawn very late (peaking in January), with little time spent in fresh water between river entry and spawning. This compressed adult freshwater residency appears to coincide with the single, brief peak of river flow characteristic of this region.

Status and Trends

NMFS originally listed the central California coast coho salmon ESU as threatened on October

31, 1996 (61 FR 56138) and later reclassified their status to endangered June 28, 2005 (70 FR 37160). Information on the abundance and productivity trends for the naturally spawning component of the central California coast coho ESU is extremely limited. There are no long-term time series of spawner abundance for individual river systems. Historical estimated escapement for this ESU is 56,100 for 1963, and more recent estimates suggest the ESU dropped to about one-fourth that size by the late 1980s and early 1990s (Good et al. 2005).

Where data are available, analyses of juvenile coho presence-absence information, juvenile density surveys, and irregular adult counts for the South Fork Noyo River indicate low abundance and long-term downward trends for the naturally spawning populations throughout the ESU. Improved ocean conditions coupled with favorable stream flows and harvest restrictions have contributed to increased returns in 2001 in streams in the northern portion of the ESU, as indicated by an increase in the observed presence of fish in historically occupied streams. Data are particularly lacking for many river basins in the southern two-thirds of the ESU where naturally spawning populations are considered to be at the greatest risk. The extirpation or near extirpation of natural coho salmon populations in several major river basins, and across most of the southern historical range of the ESU, represents a significant risk to ESU spatial structure and diversity (Good et al. 2005).

Artificial propagation of coho salmon within the Central California Coast ESU has declined since the ESU was listed in 1996 though it continues at the Noyo River and Scott Creek facilities, and two captive broodstock populations have recently been established. Genetic diversity risk associated with out-of-basin transfers appears to be minimal, but diversity risk from domestication selection and low effective population sizes in the remaining hatchery programs remains a concern. An out-of-ESU artificial propagation program for coho was operated at the Don Clausen hatchery on the Russian River through the mid 1990s, but was terminated in 1996. Termination of this program was considered by the biological review team as a positive development for naturally produced coho in this ESU.

For the naturally spawning component of the ESU, the biological review team found very high risk of extinction for the abundance, productivity, and spatial structure of the Viable Salmon Population (VSP) parameters and comparatively moderate risk with respect to the diversity VSP parameter. The lack of direct estimates of the performance of the naturally spawned populations in this ESU, and the associated uncertainty this generates, was of specific concern to the biological review team. Informed by the VSP risk assessment and the associated uncertainty, the strong majority opinion of the biological review team was that the naturally spawned component of the Central California Coast coho ESU was “in danger of extinction.” The minority opinion was that this ESU is “likely to become endangered within the foreseeable future.” (70 FR 37160) Accordingly, NMFS upgraded the status of central California coast coho ESU to endangered on June 28, 2005 (70 FR 37160).

Central California Coast coho salmon populations continue to be depressed relative to historical numbers. Strong indications show that breeding groups have been lost from a significant percentage of historical stream range. A number of coho populations in the southern portion of the range appear to be either extinct or nearly so, including those in Gualala, Garcia, and Russian rivers, as well as smaller coastal streams in and south of San Francisco Bay (Good et al. 2005).

Critical Habitat

NMFS designated critical habitat for central California coast coho salmon on May 5, 1999 (64 FR 24049). The designation encompasses accessible reaches of all rivers (including estuarine areas and riverine reaches) between Punta Gorda and the San Lorenzo River (inclusive) in California, including two streams entering San Francisco Bay: Arroyo Corte Madera Del Presidio and Corte Madera Creek. This critical habitat designation includes all waterways, substrate, and adjacent riparian zones of estuarine and riverine reaches (including off-channel habitats) below longstanding naturally impassable barriers (i.e. natural waterfalls in existence for at least several hundred years). These areas are important for the species' overall conservation by protecting growth, reproduction, and feeding.

Lower Columbia River Coho Salmon

Distribution and Description of the Listed Species

The lower Columbia River coho salmon ESU includes all naturally spawned populations of coho salmon in the Columbia River and its tributaries in Washington and Oregon, from the mouth of the Columbia up to and including the Big White Salmon and Hood Rivers, and includes the Willamette River to Willamette Falls, Oregon, Twenty-five artificial propagation programs are part of this ESU: Grays River, Sea Resources Hatchery, Peterson Coho Project, Big Creek Hatchery, Astoria High School (STEP) Coho Program, Warrenton High School (STEP) Coho Program, Elochoman Type-S Coho Program, Elochoman Type-N Coho Program, Cathlamet High School FFA Type-N Coho Program, Cowlitz Type-N Coho Program in the Upper and Lower Cowlitz Rivers, Cowlitz Game and Anglers Coho Program, Friends of the Cowlitz Coho Program, North Fork Toutle River Hatchery, Kalama River Type-N Coho Program, Kalama River Type-S Coho Program, Lewis River Type-N Coho Program, Lewis River Type-S Coho Program, Fish First Wild Coho Program, Fish First Type-N Coho Program, Syverson Project Type-N Coho Program, Washougal River Type-N Coho Program, Eagle Creek NFH, Sandy Hatchery, and the Bonneville/Cascade/ Oxbow complex coho hatchery programs.

Two distinct runs distinguished by the timing of adult returns to fresh water (early returners and later returners) occur within the ESU. Early returning adults generally migrate south of the Columbia River once they reach the ocean, returning to fresh water in mid-August and to spawning tributaries in early September. Peak spawning of early returning adults occurs from mid-October to early November. Late returning adult coho salmon exhibit a northern oceanic distribution, return to the Columbia River from late September through December, and enter tributaries from October through January. Most late return adults spawn between November through January, although some spawn in February and as late as March (LCFRB 2004). Almost all Lower Columbia River ESU coho salmon females and most males spawn at 3 years of age.

Status and Trends

NMFS listed Lower Columbia River coho salmon as endangered on June 28, 2005 (70 FR 37160). The vast majority (over 90%) of the historic population in the Lower Columbia River coho salmon ESU appear to be either extirpated or nearly so. Recent counts of natural-origin spawners and the recent fraction of hatchery-origin spawners are noted in Table 12 , where available.

Only two populations of coho salmon within this ESU produce a sizeable number of naturally spawned fish, the upper Sandy River population above Marmot Dam and the Clackamas River population above the North Fork Dam. Most of the other populations are believed to have very little, if any, natural production. The long-term and short-term trends for Marmot Dam counts are both negative. The long-term median growth rate is slightly positive for both the Sandy and Clackamas rivers, but the confidence intervals for each are very wide indicating there is a large amount of uncertainty. Both populations within the Sandy and Clackamas rivers have suffered from recruitment failure a number of times over the past 15 years, despite the reductions in harvest.

Table 12. Lower Columbia River coho salmon populations and selected measures of population viability

River	2002 Spawner Count[a]	Geometric Mean Abundance 2000-2002[b]	Percent Hatchery Contributions[c]	Long-term Median Growth Rate (λ)[d]
Youngs Bay and Big Creek	4,473		91	
Grays River				
Elochoman River				
Clatskanie River	229		60	
Mill, Germany, and Abernathy creeks				
Scappoose Rivers	458		0	
Cispus River				
Tilton River				
Upper Cowlitz River				
Lower Cowlitz River				
North Fork Toutle River				
South Fork Toutle River				
Coweeman River				
Kalama River				
North Fork Lewis River				
East Fork Lewis River				
Upper Clackamas River	1,001	2,122	12	1.009 (0.898-1.177)
Lower Clackamas River	2,402		78	
Salmon Creek				
Upper Sandy River	310	643	0	1.012 (0.874-1.172)
Lower Sandy River	271		97	
Washougal River				
Columbia River Gorge – lower tributaries				
White Salmon				
Columbia River Gorge – upper tributaries	1,317		>65	
Hood River				

[a]All data are reported in Good et al. 2005. Spawner data from 2002 only.
[b]Geometric mean number of coho salmon above the dams. * is a combined totoal for the upper and lower Clackamas River. Reported in Good et al. 2005
[c]Hatchery production likely dominates yearly returns for the ESU as a whole.
[d]The λ calculated estimates the natural growth rate after accounting for hatchery-origin spawners. The estimate provided above assumes that hatchery-origin spawners make no reproductive contribution. The λ for the Clackamas River is calculated with data spanning 1973-2002, and for the Sandy River covers 1977-2002. The Clackamas River value includes both early-run and late-run coho salmon.

The most serious threat facing this ESU is the scarcity of naturally-produced spawners, with attendant risks associated with small populations, loss of diversity, and fragmentation and isolation of the remaining naturally-produced fish. Spatial structure has been substantially reduced by the loss of access to upper basins from tributary hydro development (i.e., Condit Dam on the Big White Salmon River and Powerdale Dam on the Hood River). The diversity of populations in all three areas has been eroded by large hatchery influences and periodically, low effective population sizes.

Critical Habitat

NMFS has not designated critical habitat for Lower Columbia River coho salmon.

Southern Oregon/Northern California Coast Coho Salmon

Distribution and Description of the Listed Species

Southern Oregon/Northern California coast coho salmon consists of all naturally spawning populations of coho salmon that reside below long-term, naturally impassible barriers in streams between Punta Gorda, California and Cape Blanco, Oregon, as well as three artificial propagation programs: the Cole Rivers Hatchery, Trinity River Hatchery, and Iron Gate Hatchery coho hatchery programs. The three major river systems supporting Southern Oregon – Northern Coastal California coast coho are the Rogue, Klamath (including the Trinity), and Eel rivers.

Southern Oregon and Northern California coast coho immigrate to natal rivers in September or October. River entry is much later south of the Klamath River Basin, occurring in November and December, as well as in basins south of the Klamath River to the Mattole River, California. River entry occurs from mid-December to mid-February in rivers farther south. Because individuals enter rivers late, they spend much less time in the river. Coho salmon adults spawn at age 3, spending just over 1 year in fresh water and a year and a half in the ocean.

Status and Trends

Southern Oregon/Northern California coast coho salmon were listed as threatened on May 7, 1997 (62 FR 24588); they retained that classification when their status was reviewed on June 28, 2005 (70 FR 37160). Southern Oregon/Northern California Coast coho salmon extend from Cape Blanco in southern Oregon to Punta Gorda in northern California (Weitkamp et al. 1995). The status of coho salmon coast-wide, including the Southern Oregon/Northern California Coast coho salmon ESU, was formally assessed in 1995 (Weitkamp et al. 1995). Two subsequent status review updates have been published by NMFS, one addressing all West Coast coho salmon ESUs and a second specifically addressing the Oregon Coast Southern Oregon/Northern California Coast coho salmon ESUs (NMFS 1996, 1997). In the 1997 status update, estimates of natural population abundance were based on very limited information. New data on presence/absence in northern California streams that historically supported coho salmon were even more disturbing than earlier results, indicating that a smaller percentage of streams contained coho salmon compared to the percentage presence in an earlier study. However, it was unclear whether these new data represented actual trends in local extinctions or were biased by

sampling effort.

Data on population abundance and trends are limited for the California portion of this ESU. No regular estimates of natural spawner escapement are available. Historical point estimates of coho salmon abundance for the early 1960s and mid-1980s suggest that statewide coho spawning escapement in the 1940s ranged between 200,000 and 500,000 fish. Numbers declined to about 100,000 fish by the mid-1960s with about 43% originating from this ESU. Brown et al. (1994) estimated that the California portion of this ESU was represented by about 7,000 wild and naturalized coho salmon (Good et al. 2005). In the Klamath River, the estimated escapement has dropped from approximately 15,400 in the mid-1960s to about 3,000 in the mid 1980s, and more recently to about 2,000 (Good et al. 2005). The second largest producing river in this ESU, the Eel River, dropped from 14,000, to 4,000 to about 2,000 during the same period. Historical estimates are considered "best guesses" made using a combination of limited catch statistics, hatchery records, and the personal observations of biologists and managers.

Most recently, Williams et al. (2006) described the structure of historic populations of Southern Oregon/Northern California Coast coho salmon. They described three categories of populations: functionally independent populations, potentially independent populations and dependent populations. Functionally independent populations are populations capable of existing in isolation with a minimal risk of extinction. Potentially independent populations are similar but rely on some interchange with adjacent populations to maintain a low probability of extinction. Dependent populations have a high risk of extinction in isolation over a 100-year timeframe and rely on exchange of individuals from adjacent populations to maintain themselves.

Critical Habitat

NMFS designated critical habitat for Southern Oregon/Northern California Coast coho salmon on May 5, 1999 (64 FR 24049). Critical habitat for this species encompasses all accessible river reaches between Cape Blanco, Oregon, and Punta Gorda, California. Critical habitat consists of the water, substrate, and river reaches (including off-channel habitats) in specified areas. Accessible reaches are those within the historical range of the ESU that can still be occupied by any life stage of coho salmon. Of 155 historical streams for which data are available, 63% likely still support coho salmon. These river habitats are important for a variety of reasons, such as supporting the feeding and growth of juveniles and serving as spawning habitat for adults. Limiting factors identified for this species include: loss of channel complexity, connectivity and sinuosity, loss of floodplain and estuarine habitats, loss of riparian habitats and large in-river wood, reduced stream flow, poor water quality, temperature and excessive sedimentation, and unscreened diversions and fish passage structures.

Oregon Coast Coho Salmon

Distribution and Description of the Listed Species

The Oregon Coast coho salmon ESU includes all naturally spawned populations of coho salmon in Oregon coastal streams south of the Columbia River and north of Cape Blanco (63 FR 42587; August 1998). One hatchery population, the Cow Creek hatchery coho salmon, is considered part of the ESU. Table 13 identifies populations within the Oregon Coast coho salmon ESU,

their abundances, and hatchery input.

Table 13. Oregon Coast coho populations and selected measures of population viability

Basin[a]	Mean Spawner Abundance[b]	13-Year Spawner Trend (SE) [c]	Percent Hatchery Contribution[d]
Necanicum	1,889	1.169 (0.860)	2.9-6.4
Nehalem	18,741	1.206 (0.889)	0.5-26.0
Tillamook Bay	3,949	1.191 (1.084)	0-5.6
Nestucca	3,846	1.230 (1.015)	0-10.4
Siletz	2,295	1.070 (0.760)	1.8-100
Yaquima	3,665	1.204 (1.205)	0-37.5
Alsea	3,621	1.042 (0.960)	0-87.5
Siuslaw	16,213	1.120 (1.037)	0.3-11.1
Umpqua	24,351	1.182 (0.662)	2.1-8.3
Coos	20,136	1.088 (1.066)	0-1.9
Coquille	8,847	1.070 (0.649)	0-6.0

[a]Population structure is unclear. The above data reflects the assumption that spawners from major river basins are largely isolated, and each basin comprises a population. All data are reported in Good et al. 2005.
[b]Recent 3-year geometric mean of natural-origin spawners.
[c]Data years 1990-2002.
[d]Data represents the range of percent hatchery contributions from 1998 through 2002 (from Jacobs et al. 2002, 2001, and 2002 as cited in Good et al. 2005).

Status and Trends

The Oregon coast coho salmon ESU was listed as a threatened species under the ESA on February 11, 2008 (73 FR 7816), the conclusion to a 13-year history of court cases. The most recent NMFS status review for the Oregon Coast coho ESU was conducted by the biological review team in 2003, which assessed data through 2002. The abundance and productivity of Oregon Coast coho since the previous status review represented some of the best and worst years on record (NMFS 1997a). Yearly adult returns for the Oregon Coast coho ESU were over 160,000 natural spawners in 2001 and over 260,000 in 2002, far exceeding the abundance observed for the past several decades. These increases in spawner abundance in 2000 to 2002 followed three consecutive brood years (the 1994 to 1996 brood years returning in 1997 to 1999, respectively) exhibiting recruitment failure (recruitment failure is when a given year class of natural spawners fails to replace itself when its offspring return to the spawning grounds 3 years later). These 3 years of recruitment failure were the only such instances observed thus far in the entire 55-year abundance time series for Oregon Coast coho salmon (although comprehensive population-level survey data have only been available since 1980). The 2000 to 2002 increases in natural spawner abundance occurred in many populations in the northern portion of the ESU, which were the most depressed at the time of the last review (NMFS 1997a). Although encouraged by the increase in spawner abundance in 2000 to 2002, the biological review team noted that the long-term trends in ESU productivity were still negative due to the low abundances observed during the 1990s.

Since the biological review team convened, the total abundance of natural spawners in the Oregon Coast coho ESU has declined each year (i.e., 2003 to 2006). The abundance of total natural spawners in 2006 (111,025 spawners) was approximately 43 % of the recent peak abundance in 2002 (255,372 spawners). In 2003, ESU-level productivity (evaluated in terms of

the number of spawning recruits resulting from spawners 3 years earlier) was above replacement, and in 2004, productivity was approximately at replacement level. However, productivity was below replacement in 2005 and 2006, and dropped to the lowest level since 1991 in 2006 (73 FR 7816).

Preliminary spawner survey data for 2007 (the average peak number of spawners per mile observed during random coho spawning surveys in 41 streams) suggest that the 2007 to 2008 return of Oregon Coast coho is either (1) much reduced from abundance levels in 2006, or (2) exhibiting delayed run timing from previous years. As of December 13, 2007, the average peak number of spawners per mile was below 2006 levels in 38 of 41 surveyed streams (ODFW 2007 *in* 73 FR 7816). It is possible that the timing of peak spawner abundance is delayed relative to previous years, and that increased spawner abundance in late December and January 2008 will compensate for the low levels observed thus far.

The recent 5-year geometric mean abundance (2002 to 2006) of approximately 152,960 total natural spawners remains well above that of a decade ago (approximately 52,845 from 1992 to 1996). However, the decline in productivity from 2003 to 2006, despite generally favorable marine survival conditions and low harvest rates, is of concern (73 FR 7816).

Critical Habitat

NMFS designated critical habitat for Oregon Coast coho on February 11, 2008 (73 FR 7816). The designation includes 72 of 80 watersheds occupied by Oregon Coast coho salmon, and totals about 6,600 stream miles including all or portions of the Nehalem, Nestucca/Trask, Yaguina, Alsea, Umpqua and Coquille basins. These areas are essential for feeding, migration, spawning, and rearing. The specific primary constituent elements include: spawning sites with water and substrate quantity to support spawning, incubation, and larval development; freshwater rearing sites with water quantity and floodplain connectivity to form and maintain physical habitat conditions and support juvenile growth, foraging, behavioral development (e.g., predator avoidance, competition), and mobility; freshwater migratory corridors free of obstruction with adequate water quantity and quality conditions; and estuarine, nearshore and offshore areas free of obstruction with adequate water quantity, quality and salinity conditions that support physiological transitions between fresh- and saltwater, predator avoidance, foraging and other life history behaviors.

Sturgeon

Description of the Genus

Sturgeon (*Acipenseridae*) are one of the oldest *Osteichthyes* (bony fish) in existence, and are native to rivers and coastal areas of North America. The two listed sturgeon, discussed below, are part of the genus *Acipenser*, and share some common characteristics. Sturgeon, in general, have a characteristic external morphology distinguished by the inferior mouth typical of bottom-feeders. Most species are anadromous, although a few species are entirely fresh water and many species can survive if they become land-locked. Both listed species (discussed below) are anadromous and tend to remain in coastal waters. As an anadromous fish, sturgeon spawn in fresh water and feed and rear in marine or estuarine waters. Sturgeon are also iteroparous spawners and tend to be very long-lived.

Threats

Natural Threats. Freshwater predation of eggs and larvae from birds and larger fish, and marine predation of adult and subadult fish by sharks, pinnipeds and other large body predators.

Anthropogenic Threats. In general sturgeon have declined from the combined effects from the construction of dam and water diversion projects, dredging and blasting, water pollution, and fisheries. As a result of their longevity, slow rate of growth and delayed maturation, and bottom-feeding habits, in general sturgeon have a life history that makes them susceptible to over-harvest and exposure to (and the accumulation of) contaminants. Many sturgeon also do not spawn on an annual basis, but may spawn every other year or even more infrequently. Thus even small increases in mortality can affect population productivity (Heppell 2007). The body form and feeding habits of sturgeon may expose them to a different suite of contaminants or contaminant properties than pelagic fish due to their affinity with bottom sediments. Exposure pathways would include a dissolved or water borne exposure, but for sediment-associated contaminants the sediment exposure pathway may be more significant. Benthic dwelling fish may be exposed through the direct contact with sediment, exposed to the boundary layer over the sediment, and commonly have a higher rate of incidental ingestion and exposure through direct consumption of sediments.

Southern Green Sturgeon

Distribution and Description of the Listed Species

Green sturgeon occur along the west coast of North America from Mexico to the Bering Sea (Adams et al. 2002; Moyle 2002; Colway and Stevenson 2007). Distinguished primarily according genetic differences and spawning locations, NMFS recognizes two distinct population segments (DPS) of green sturgeon: a northern DPS whose populations are relatively healthy, and a Southern DPS that has undergone significant decline (Adams et al. 2007). NMFS listed the Southern DPS of green sturgeon as threatened in 2006 (71 FR 17757).

Green sturgeon are considered one of the most marine-oriented sturgeon species, spending much of their lives in coastal marine waters, estuaries and bays. Early life stages rear in fresh water, and adults return to fresh water when they are 15 years old or older to spawn. Across the

species' range only three rivers contain documented spawning and only one of the rivers is part of the southern green sturgeon DPS, the Sacramento River (Moyle et al. 1992; CDFG 2002). Outside of natal rivers, the distribution of southern green sturgeon and northern green sturgeon overlap. Both northern DPS and southern DPS green sturgeon occupy coastal estuaries and coastal marine waters from southern California to Alaska, including Humbolt Bay, the lower Columbia River estuary, Willapa Bay, Grays Harbor and southeast Alaska. In general, green sturgeon are more common north of Point Conception, California.

Green sturgeon are spring spawners and initiate spawning migrations as early as March, spawn late spring to early summer, hold in deep pools and return to salt water in the fall early, often with the first increases in fall flows. There may a be a latitudinal cline in the timing of upstream spawning migrations, as fish in the Klamath River have been observed initiating migrations between April and June, Rogue River fish between May and July, whereas Heubein et al. (2009) observed Sacramento River fish making their upstream migrations between March and April. Spawning generally occurs in deep pools of large rivers or off-channel coves (Moyle et al. 1992, 1995; Erickson and Webb 2007; Erickson et al. 2001; Heublein et al. 2009; Rien et al. 2001). Fish then tend to aggregate in deep pools, where they will over-summer before outmigrating in the fall, although some fish have been observed outmigrating relatively soon after presumed spawning events (Heubein et al. 2009). In the Sacramento River adult green sturgeon spawn in late spring and early summer above Hamilton City, above Red Bluff Diversion Dam, and possibly as far upstream as Keswick Dam (CDFG 2002; Heubein et al. 2009). It appears that specific habitat for spawning includes large cobblestones (where eggs can settle between), although spawning is known to occur over clean sand or bedrock.

Green sturgeon are a long-lived fish, and likely live for 60 to 70 years (Moyle 2002). Age at first maturation for green sturgeon is at least 15 years old, after which adults likely return every 2 to 5 years to spawn (Adams et al. 2002; Moyle 2002; Van Eenennaam et al. 2006). Most male spawners are young (17 to 18 years) while females on the spawning grounds are often older (27 to 28 years). Females produce roughly 60,000 to 140,000 eggs per spawning event (Scott and Crossman 1973; Moyle et al. 1992). Temperature may trigger spawning behavior, with ranges of 48º to 62º F being influential (Moyle et al. 1995). Water temperature is also critical for egg survival with temperatures above 68º F being fatal to developing embryos (Cech et al. 2000).

Green sturgeon spend their first 1 to 4 years in their natal streams and rivers (Nakamoto et al. 1995; Beamsesderfer and Webb 2002), although they are believed to be physiologically adapted to sea water survival at 6 months of age (Allen and Cech 2007; Allen et al. 2009a, b). Larvae are active at night, a behavior that likely reduces predation and avoids being moved downstream more than necessary (Cech et al. 2000). Green sturgeon larvae grow very rapidly, reaching about 300 mm by age one (Deng 2000). Temperature is strongly correlated with growth rates, with optimal growth rates occurring at about 59º F (Cech et al 2000). While in fresh water, juveniles feed on a variety of fishes and invertebrates (Moyle et al. 1992). One juvenile from the Sacramento-San Joaquin estuary was found to have preyed most commonly upon opisthobranch mollusks (*Philline* sp.), with bay shrimp (*Crangon* sp.) and overbite clams (*Potamocorbula amurensis*) as secondary prey. Other juveniles in the Sacramento River delta feed on opossum shrimp (*Neomysis mercedis*) and *Corophium* amphipods (Radtke 1966).

Upon outmigration from fresh water, subadult green sturgeon disperse widely along through continental shelf waters of the west coast within the 110 meter contour (Moyle et al. 1992; NMFS 2005b; Erikson and Hightower 2007). Biologists have recaptured fish tagged in the Sacramento River, in coastal and estuarine waters to the north. It appears that green sturgeon generally distribute north of the river mouth from whence they emerge as juveniles during fall and move into bays and estuaries during summer and fall (Israel et al. 2009; Moser and Lindley 2007). The limited feeding data available for subadult and adult green sturgeon show that they consume benthic invertebrates including shrimp, clams, chironomids, copepods, mollusks, amphipods, and small fish (Houston 1988; Moyle et al. 1992; Wilson and McKinley 2004; Dumbauld et al. 2008). Starting as larvae, sturgeon use electroreception to identify prey. Olfaction and taste may also be important to foraging, while vision is thought play a minor role in prey capture (Miller 2004).

Status and Trends

NMFS listed the southern population of the North American green sturgeon as threatened on April 7, 2006 (71 FR 17757). Trend data for green sturgeon is severely limited. Available information comes from two predominant sources, fisheries and tagging. Only three data sets were considered useful for the population time series analyses by NMFS' biological review team: the Klamath Yurok Tribal fishery catch, a San Pablo sport fishery tag returns, and Columbia River commercial landings (NMFS 2005b). Using San Pablo sport fishery tag recovery data, the California Department of Fish and Game produced a population time series estimate for the southern DPS. San Pablo data suggest that green sturgeon abundance may be increasing, but the data showed no significant trend. The data set is not particularly convincing, however, as it suffers from inconsistent effort and since it is unclear whether summer concentrations of green sturgeon provide a strong indicator of population performance (NMFS 2005b). Although there is not sufficient information available to estimate the current population size of southern green sturgeon, catch of juveniles during state and federal salvage operations in the Sacramento delta are low in comparison to catch levels before the mid-1980s.

Threats

Natural Threats. Green sturgeon eggs and larvae are likely preyed upon by a variety of larger fish and animals, while sub-adult and adult sturgeon may occasionally be preyed upon by shark sea lions, or other large body predators. Physical barriers, changes in water flow and temperatures may also affect fresh water survival.

Anthropogenic Threats. The principle threat to southern green sturgeon comes from a drastic reduction in available spawning area from impassible barriers (e.g., Oroville, Shasta and Keswick dams). Other threatens include potentially lethal temperature limits, harvest, entrainment by water projects and toxins and invasive species (Adams et al. 2007; Erickson and Webb 2007; Lackey 2009). Since this DPS is composed of a single spawning population within the Sacramento River, stochastic variation in environmental conditions and significant fluctuations in demographic rates increases the risk of extinction for this DPS.

Climate change has the potential to affect sturgeon in similar, if not more significant ways it affects salmonids. Elevated air temperatures could lead to precipitation falling as rain instead of

snow. Additionally, snow would likely melt sooner and more rapidly, potentially leading to greater flooding during melting and lower water levels at other times, as well as warmer river temperatures. Although sturgeon can spawn over varied benthic habitat, they prefer localized depressions in riverbeds (Erickson et al. 2001; Moyle et al. 1992; Moyle et al. 1995; Rien et al. 2001). Increased extremes in river flow (i.e., periods of flooding and low flow) can alternatively disrupt and fill in spawning habitat that sturgeon rely upon (ISAB 2007). If water flow is low during migration events, it is likely that new obstacles can impede or block sturgeon movement. As with other anadromous fishes, sturgeon are uniquely evolved to the environments that they live in. Because of this specificity, broad scale changes in environment can be difficult to adapt to, including changes in water temperature (Cech et al. 2000). Sturgeon are also sensitive to elevated water temperatures. Temperature triggers spawning behavior. Warmer water temperatures can initial spawning earlier in a season for salmon and the same can be true for sturgeon (ISAB 2007). If river and lake temperatures become anomalously warm, juvenile sturgeon may experience elevated mortality due to lack of cooler water refuges in freshwater habitats. Apart from direct changes to sturgeon survival, altered water temperatures may disrupt habitat, including the availability of prey (ISAB 2007). Warmer temperatures may also have the effect of increasing water use in agriculture, both for existing fields and the establishment of new ones in once unprofitable areas (ISAB 2007). This means that streams, rivers, and lakes will experience additional withdrawal of water for irrigation and increasing contaminant loads from returning effluent. Overall, it is likely that global warming will increase pressures on sturgeon survival and recovery.

Studies from other sturgeon species indicate that sturgeon readily bioaccumulate contaminants. White sturgeon from the Kootenai River have been found to contain aluminum, arsenic, cadmium, chromium, cobalt, copper, iron, lead, manganese, mercury, nickel, selenium, zinc, DDE, DDT, PCBs, and other organochlorines (Kruse and Scarnecchia 2001). Mercury has also been identified from white sturgeon of the lower Columbia River (Webb et al. 2006). Numerous organochlorines, including DDT, DDD, DDE, chlordane, and dieldrin have also been identified in these fish (Foster et al. 2001). Observed concentrations are likely sufficient to influence reproductive physiology.

Critical Habitat

On October 9, 2009, NMFS designated critical habitat for southern green sturgeon (74 FR 52300). The geographical area identified as critical habitat is based upon the overlapping distribution of the southern and northern DPS, and encompasses all areas where the presence of southern green sturgeon have been confirmed or where their presence is likely. Therefore the geographical area defined as critical habitat is the entire range of the biological species, green sturgeon, from the Bering Sea, AK, to Ensenada, Mexico. Specific fresh water areas include the Sacramento River, Feather River, Yuba River, and the Sacramento-San Joaquin Delta. Specific coastal bays and estuaries include estuaries from Elkhorn Slough, California, to Puget Sound, Washington. Coastal marine areas include waters along the entire biological species' range within a depth of 60 fathoms. The principle biological or physical constituent elements essential for the conservation of southern green sturgeon in fresh water include: food resources; substrate of sufficient type and size to support viable egg and larval development; water flow, water quality such that the chemical characteristics support normal behavior, growth and viability;

migratory corridors; water depth; and sediment quality. Primary constituent elements of estuarine habitat include food resources, water flow, water quality, migratory corridors, water depth, and sediment quality. The specific primary constituent elements of marine habitat include food resources, water quality, and migratory corridors.

Critical habitat of the Southern DPS of green sturgeon is threatened by several anthropogenic factors. Four dams and several other structures currently are impassible for green sturgeon to pass on the Sacramento, Feather, and San Joaquin rivers, preventing movement into spawning habitat. Threats to these riverine habitats also include increasing temperature, insufficient flow that may impair recruitment, the introduction of striped bass that may eat young sturgeon and compete for prey, and the presence of heavy metals and contaminants in the river.

Shortnose Sturgeon

Distribution and Description of the Listed Species

Shortnose sturgeon occur along the Atlantic Coast of North America, from the St. John River in Canada, south to the St. John's River in Florida. NMFS' recovery plan (1998a) recognized 19 wild populations based on their strong fidelity to their natal streams, and several captive populations (from a Savannah River broodstock) that are maintained for educational and research purposes (NMFS 1998a; Table 14).

Shortnose sturgeon are generally anadromous (they migrate between sea and fresh water for reproductive purposes) or amphidromous (some fish migrate between fresh and salt water for reasons other than spawning, such as feeding), but such migratory behavior may not be obligatory for the species as they can also maintain land-locked (freshwater resident) populations. In general, shortnose sturgeon are benthic fish that occupy the deep channel sections of large rivers or estuarine waters of their natal rivers, and will migrate considerable distances. Dadswell (1979 in Dadswell et al. 1984) observed shortnose sturgeon traveling up 160 km between tagging and recapture in the St. John estuary, and it is not uncommon for adults to migrate 200 km or more to reach spawning areas (Kynard 1997).

The general migratory strategy of shortnose sturgeon is similar to many fresh water and diadromous fishes, which probably optimizes feeding opportunities, minimizes losses due to unfavorable conditions (winter refuge migrations), and optimizes spawning success (Northcote 1978; Harden-Jones 1968 in Dadswell 1984). Water temperatures, flow regimes, and barriers influence their movement patterns (Kynard 1997; Kynard et al. 2000). Adult shortnose sturgeon will migrate upstream to spawning areas in the spring or in the fall. Fish that migrate upstream in the fall generally overwinter in areas just downstream of spawning sites, while others including non-spawners will overwinter in estuarine waters. After spawning in the spring, spent (post-spawned) adults tend to migrate rapidly downstream to feeding areas in the estuary or to tidally influence fresh water (see Dadswell et al. 1984 for a review).

Young-of-the year shortnose sturgeon move downstream after hatching, remaining in fresh water for about 1 year (Kynard 1997). Initially, young shortnose sturgeon will reside short distances from spawning areas, and as they grow will tend to move further downstream (Dadswell et al.

1984). By age 3 or older juvenile sturgeon will spend a large portion of their year at the salt- and fresh water interface of coastal rivers (NMFS 1998a).

Habitat use in fresh water during summer and winter months overlaps between adult and age-1 shortnose sturgeon (O'Herron et al. 1993; Rogers and Weber 1995 in Moser et al. 2000; Kynard et al. 2000). Kynard et al. (2000) found that both age classes preferred deep-water curves with sand and cobble to higher velocity runs, particularly during winter months, and shifted to channel habitat as water temperatures rose in summer months. Many fish also exhibited diel movement patterns between deeper waters during the day and shallower waters at night (Kynard et al. 2000). During the summer, at the southern end of their range, shortnose sturgeon congregate in cool, deep, areas of rivers where adult and juvenile sturgeon can take refuge from high temperatures (Flournoy et al. 1992, Rogers and Weber 1995, and Weber 1996 cited in Moser et al. 2000). In the Connecticut River and the Merrimack, Kynard et al. (2000) found shortnose generally used water about 3 meters deep, ranging from less than a meter to about 15 meters deep.

Sturgeon are iteroparous, and based on limited data it appears that females sturgeon spawn every three to five years while males spawn every other year, although some may spawn in consecutive years (Dovel et al. 1992; Collins and Smith 1993; Kieffer and Kynard 1993; NMFS 1998a). Spawning typically occurs during the spring, between mid-March and late May. Spawning areas are often located just below the fall line at the farthest accessible upstream reach of the river (NMFS 1998a). The onset of spawning may be cued to decreasing river discharge following the peak spring freshet, when water temperatures range from 8 to 12 °C and bottom water velocities range between 25-130 cm/s, although photoperiod appears to control spawning readiness (Dadswell et al. 1984; NMFS 1998a; Kynard et al., in draft).

Length at maturity is about 45-55 cm fork length for shortnose sturgeon and age at first spawning appears to vary along a latitudinal cline. According to spawning checks, it appears that male shortnose sturgeon in southern rivers will first spawn between ages 2 and 5, while fish as far north as the St. Johns River, Canada first spawn at about 10 to 11 years of age (Dadswell et al. 1984; NMFS 1998a). Age at first spawning for female shortnose sturgeon varies from about age 6 to 18 years, like males, varying on a latitudinal cline (Dadswell et al. 1984; NMFS 1998a). In general, fish in the northern portion of the species' range live longer than individuals in the southern portion of the species' range (Gilbert 1989). The maximum age reported for a shortnose sturgeon in the St. John River in New Brunswick is 67 years (for a female), 40 years for the Kennebec River, 37 years for the Hudson River, 34 years in the Connecticut River, 20 years in the Pee Dee River, and 10 years in the Altamaha River (Gilbert 1989 using data presented in Dadswell et al. 1984). Male shortnose sturgeon appear to have shorter life spans than females (Gilbert 1989).

Like all sturgeon, shortnose have ventrally located, sucker-like mouths, structured for feeding on benthos. Foraging generally occurs in areas with abundant macrophytes, where juvenile and adult shortnose sturgeon feed on amphipods, polychaetes, and gasteropods (Dadswell et al. 1984; Moser and Ross 1995; NMFS 1998a). Starting as larvae sturgeon use electroreception to identify prey. Olfaction and taste are also likely important to foraging, while vision is thought to play a minor role (Miller 2004). As adults, a significant portion of a shortnose sturgeon's diet may consist of freshwater mollusks (Dadswell et al. 1984). Based on observations by Kynard et al.

(2000), shortnose sturgeon will consume the entire mollusk, excreting the shell after ingestion.

Table 14. Shortnose sturgeon populations and their estimated abundances

Population (Location)[a]	Data Series	Abundance Estimate (C.I.)[b]	Population Segment	Reference
Saint John River (Canada)	1973-1977	18,000 (+/-30%)	Adults	Dadswell 1979
Kennebecasis River (Canada)	1998-2005	2,068 (801-11,277)		COSEWIC 2005
Kennebecasis River	2005	4,836 (+/-69)		Li et al. 2007, NMFS unpubl.
Penobscot River (ME)	2006-2007	1,049 (673-6,939)		UME 2008
	2008	1739 (846-3653)	Summer	P. Dionne, pers. comm..
		667 (451-1013)	Fall	P. Dionne, pers. comm..
Kennebec River (ME)	1977-1981	7,222 (5,046-10,765)	Adult	Squiers et al. 1982
	2003	9,488 (6,942-13,358)	Adults	Squiers 2003
Merrimack River (MA)	1987-1991	32 (20-79)	Adults	Kynard & Kieffer, unpubl.; NMFS unpubl.
Connecticut River (MA, CT)	1989-2002	1,042-1,580 [c]	Adults	Savoy 2004
Upper Connecticut River[d]	1976-1977	516 (317–898)	Total	Taubert 1980; NMFS 1998a
	1977-1978	370 (235–623)	Total	Taubert 1980; NMFS 1998a
	1976-1978	714 (280-2,856)	Total	Taubert 1980; NMFS 1998a
	1976-1978	297 (267–618)	Total	Taubert 1980; NMFS 1998a
	1994	328 (188-1,264)	Adults	Kynard & Kieffer, unpubl.; NMFS unpubl.
	1994-2001	143 (14-360)	Spawning Adults	Kynard & Kieffer, unpubl.; NMFS unpubl.
Lower Connecticut River[e]	1988-1993	895 (799-1,018)	Adult	Savoy and Shake 1992; NMFS 1998a
Hudson River (NY)	1980	30,311	Total	Dovel 1979; NMFS 1998a
	1994-1997	61,057 (52,898-72,191)	Total	Bain et al. 2007
Delaware River (NJ, DE, PA)	1981-1984	12,796 (10,288-16,267)	Partial	Hastings et al. 1987
	1981-1984	14,080 (10,079-20,378)	Partial	Hastings et al. 1987
	1999-2003	12,047 (10,757-13,589)		Brundage and O'Herron 2003
Chesapeake Bay (MD, VA) Cape Fear River (NC) Winyah Bay (NC, SC) Santee River (SC) Cooper River (SC) ACE Basin (SC)	1996-1998	300	Adults	Cooke et al. 2004
Savannah River (SC, GA)		1,000 - 3,000	Adults	B Post, SCDNR 2003; NMFS unpubl.
Ogeechee River (GA)	1993	266 (236 – 300)		Weber 1996, 1998
	1993	361 (326 – 400)	Total	Rogers and Weber 1994, NMFS 1998a
	1999-2004	147 (104-249)		Fleming et al. 2003; NMFS unpubl.
Altamaha River (GA)	1988	2,862 (1,069 - 4,226)	Total	NMFS 1998a
	1990	798 (645 - 1,045)	Total	NMFS 1998a

	1993	468 (316 – 903)	Total	NMFS 1998a
		6,320 (4,387-9,249)	Total	DeVries 2006
Satilla River (GA)				
Saint Mary's River (FL)				
Saint Johns River (FL)				FFWCC 2007c

[a]The original 19 populations identified by NMFS in the 1998 recovery plan are left aligned in this column. Estimates for a tributary or river segment are indented.
[b]Population estimates are established using different techniques and should be viewed with caution. In some cases, sampling biases may have violated the assumptions of the procedures used or resulted in inadequate representation of a population segment. Some estimates (e.g., those without confidence intervals or are depicted by ranges only) are the "best professional judgment" of researchers based on their sampling effort and success.
[c]Range represents total population estimates using four different techniques. All techniques suggest the population increased during the sampling period (see Savoy 2004 for more details).
[d]Above Holyoke Dam.
[e]Below Holyoke Dam.

Status and Trends

Shortnose sturgeon were listed as endangered on March 11, 1967, under the Endangered Species Preservation Act (32 FR 4001) and remained on the endangered species list with enactment of the ESA of 1973, as amended. Although the original listing notice did not cite reasons for listing the species, a 1973 Resource Publication issued by the U.S. Department of Interior (USDOI), stated that shortnose sturgeon were "in peril ... gone in most of the rivers of its former range [but] probably not as yet extinct" (USDOI 1973). Pollution and overfishing, including bycatch in the shad fishery, were listed as principal reasons for the species' decline. Shortnose sturgeon are listed as an endangered species throughout all of its range

Northern shortnose sturgeon population abundances are generally larger than southern populations (Kynard 1997). Updated population estimates also suggest that three of the largest populations (Kennebec, Hudson, and Delaware River) may be increasing or stable, although data is limited. The New York (Hudson River) shortnose sturgeon population is the largest extant population of this species and based on available data exhibits appears to have increased (NMFS 1998a; Bain *et al.* 2000). The most recent population estimate indicates this population consists of about 61,000-shortnose sturgeon (95% confidence interval [CI] was between 52,898 and 72,191 fish [Bain et al. 2000]). A comparison of the Bain estimate to the 1979/1980 population estimate of spawning adults by Dovel et al. (1992; about 13,000 fish) led Bain et al. (2000) to conclude that the population had made a dramatic increase (about 400 % increase) between 1979 and 1997. While still evidence of an increasing population, a comparison of total population estimates (30,000:60,000) would suggest the population has only doubled in size during the study years. Similarly, the Kennebec River population appears to be increasing. Early estimates suggest that the Kennebec River contained an estimated 7,200 adult shortnose sturgeon in 1977-81 (Squiers et al. 1982), while the most recent estimate for this population is about 9,500 fish (Squiers 2003), suggesting the population has increased by about 30 % in about a twenty year period.

Data from the Delaware River, suggests that the population may be stable. Brundage and O'Herron (2003) estimate that the current population for the Delaware River is 12,047 adult fish (1999-2003; 95% CI: 10,757-13,589), which is similar to the 1981/84 estimate by Hastings *et al.* (1987) of 12,796 fish (95% CI: 10,288-16367). The recent capture of several fish that were tagged as adults by Hastings et al. (1987) suggests that older fish may comprise a substantial

portion of the Delaware River population. Based on studies from other sturgeon species we know of no evidence of senescence in sturgeon, and we would expect that these fish are reproductively active (Paramian et al. 2005). Despite their longevity, the viability of sturgeon populations is sensitive to variability in juvenile recruitment and survival (Anders et al. 2002; Gross et al. 2002; Secor et al. 2002). Although interannual variation in juvenile recruitment would be expected as a result of stochastic factors that influence spawning and egg/larval survival, if the mean population size does not change over the long-term it then it would appear there is sufficient juvenile survival to provide at least periodic recruitment into the adult age classes. Data on juvenile recruitment or age-1+ survival would, however, establish whether this population is at a stable equilibrium.

South of Chesapeake Bay, populations are relatively small compared to their northern counterparts. The largest of the southern populations of shortnose sturgeon is the Altamaha River population. Population estimates have been calculated several times for sturgeon in the Altamaha since 1993, and s. Total population estimates shown pretty sizeable interannual variation is occurring; estimates have ranged from as low as 468 fish in 1993 to over 6,300 fish in 2006 (NMFS 1998a; DeVries 2006). The Ogeechee River is the next most studied river south of Chesapeake Bay, and abundance estimates indicate that the shortnose sturgeon population in this river is considerably smaller than that in the Altamaha River. The highest point estimate in 1993 using a modified Schnabel technique resulted in a total population estimate of 361 shortnose sturgeon (95% CI: 326-400). In contrast the most recent survey resulted in an estimate of 147 shortnose sturgeon (95% CI: 104-249), suggesting that the population may be declining.

Annual variation in population estimates in many basins is due to changes in yearly capture rates, which are strongly correlated with weather conditions (river flow and water temperatures). In "dry years" fish move into deep holes upriver of the saltwater/freshwater interface, which can make them more susceptible to gillnet sampling. Consequently, rivers with limited data sets among years and limited sampling periods within a year may not offer a realistic representation of the size or trend of the shortnose sturgeon population in the basin. As a whole, the data on shortnose sturgeon populations is rather limited and some of the differences observed between years may be an artifact of the models and assumptions used by the various studies. Long-term data sets and an open population model would likely provide for more accurate population estimates across the species' range, and could provide the opportunity to more closely link strong-year classes to habitat conditions.

Throughout the species' range there are other extant populations, or at least evidence that several other basins are used periodically. That is, shortnose sturgeon have been documented in the St. John's River (FL), the St. Mary's River, Chesapeake Bay, Potomac River, Piscataqua River, the Housatonic River, and others. Some basins probably previously contained shortnose populations, but recent sampling has been largely unsuccessful. Despite the occasional observations of shortnose sturgeon, populations may be extinct in several basins (e.g., St. John's (FL), St. Mary's, Potomac, Housatonic, and Neuse rivers). Those few fish that have been observed in these basins are generally presumed to be immigrants from neighboring basins. In some cases, (e.g. Chesapeake Bay) migratory information collected from tagged fish and genetic evidence confirms that fish captured in Chesapeake Bay were part of the Delaware River population (Grunwald et al. 2002; Wirgin et al. 2005; and T. King, in progress)..

Threats

Natural Threats. Yellow perch, sharks, and seals are predators of shortnose sturgeon juveniles (NMFS 1998a). The effects of disease and parasites are generally unknown.

Anthropogenic Threats. Shortnose sturgeon have declined from the combined effects from the construction of hydropower and water diversion projects, dredging and blasting, water pollution, fisheries, and hatcheries. The construction of dams has resulted in substantial loss of shortnose sturgeon habitat along the Atlantic seaboard. In many cases dams divide shortnose sturgeon spawning habitat (e.g., Connecticut River, Penobscot River) and impede passage or block it completely. Where it has occurred, remediation measures, such as obstruction removal or modification to allow for fish passage have improved shortnose sturgeon habitat and likely improved productivity and more such modifications are planned in certain basins. For instance, with the breaching of the Bangor Dam in the Penobscot River in 1977 five river kilometers were opened to sturgeon and other anadromous fishes. With the recent signing of the Penobscot River Restoration Trust, access may be restored to another 29 km of habitat.

Historic fishery harvests, as well as the incidental harvest in current fisheries, have had lasting effects on shortnose sturgeon. In the late nineteenth and early twentieth centuries shortnose sturgeon commonly were harvested incidental to Atlantic sturgeon, the larger and more commercially valuable of these two sympatric sturgeon species (NMFS 1998a). The effects of these harvests may have latent and long-lasting impacts on some populations. At present there is no legal directed fishing effort for shortnose sturgeon in the United States, although some illegal poaching is suspected. Additionally, shortnose sturgeon are often caught incidental to other fisheries. For instance, shortnose are caught incidentally by bass anglers, and incidentally to alewife/gaspereau and shad fisheries in the St. John's River in Canada, shad fisheries in the Altamaha River, Hudson River, and others (COSEWIC 2005; Bahn & Peterson 2009).

Habitat alterations from discharges, dredging or disposal of material into waterways, and other developmental activities along riverine and estuarine systems threaten shortnose sturgeon habitat. Periodic maintenance of harbors and rivers likely results in the direct take of some sturgeon, but perhaps of greater impact is the manner in which dredging alters benthic topography and community structure, and water quality (increase in suspended sediments). Shoreline development of liquefied natural gas facilities and alternative power sources also alters coastal habitats through changes in benthic communities by dredging, changes in water quality and water temperatures, and may increase the potential of ship strikes. In the Bay of Fundy, a tidal turbine killed at least three Atlantic salmon in the 1980s, and may be a threat to shortnose sturgeon as well (Dadswell and Rulifson 1994). Although currently the only example of this type of turbine in North America, increasing interests in finding alternative energy sources is expected to result in an increase in the number of marine turbines along the coast.

Fish kills have also been observed where estuaries are affected by urban and agricultural discharges that cause vegetative blooms and eutrophic conditions. Extreme declines in dissolved oxygen levels have occurred periodically throughout the species' range. In the late 1960s and early 1970s, dissolved oxygen levels reached zero ppm in the Penobscot, Kennebec, and Androscoggin rivers and estuaries during the summer. Extreme low dissolved oxygen levels have also plagued Chesapeake Bay. In most cases, dissolved oxygen levels have improved

through improved treatment and control of waste discharges in the past twenty years, but degraded conditions of benthos are still common in many estuaries throughout the species' range as a result of this historic loading of organic materials, waste, and legacy toxins such as dioxin. As a result, shortnose sturgeon and other benthic organisms are regularly in direct contact with legacy pollutants, as well as a suite of common contaminants added from more current industrial and agricultural practices. Studies demonstrate that shortnose sturgeon carry a wide number of potentially hazardous contaminants. Individuals from the Delaware River contain numerous metals (mercury, aluminum, antimony, barium, cadmium, calcium, chromium, copper, iron, magnesium, manganese, nickel, potassium, sodium, vanadium, and zinc), PCDDs, PCDFs, PCBs, DDE, DDD, bis (2-ethylhexyl) phthalate, di-n-butylphthalate, and chlordane (ERC 2002). Most of these metals, PCDDs, PCDFs, and PCBs were also found in shortnose sturgeon in the Kennebec River (ERC 2003).

Climate change has the potential to affect sturgeon in similar, if not more significant, ways than it affects salmonids. Elevated air temperatures could lead to precipitation falling as rain instead of snow. Additionally, snow would likely melt sooner and more rapidly, potentially leading to greater flooding during melting and lower water levels at other times, as well as warmer river temperatures (ISAB 2007). Although sturgeon can spawn over varied benthic habitat, they prefer localized depressions in riverbeds (Erickson et al. 2001; Moyle et al. 1992; Moyle et al. 1995; Rien et al. 2001). Increased extremes in river flow (i.e., periods of flooding and low flow) can alternatively disrupt and fill in spawning habitat that sturgeon rely upon (ISAB 2007). If water flow is low during migration events, it is likely that new obstacles can impede or block sturgeon movement. As with other anadromous fishes, sturgeon are uniquely evolved to the environments that they live in. Because of this specificity, broad scale changes in environment can be difficult to adapt to, including changes in water temperature (Cech et al. 2000). Sturgeon are also directly sensitive to elevated water temperatures. Temperature triggers spawning behavior. Warmer water temperatures can initiate spawning earlier in a season for salmon and the same can be true for sturgeon (ISAB 2007). If river and lake temperatures become anomalously warm, juvenile sturgeon may experience elevated mortality due to lack of cooler water refuges in freshwater habitats. Apart from direct changes to sturgeon survival, altered water temperatures may disrupt habitat, including the availability of prey (ISAB 2007). Warmer temperatures may also have the effect of increasing water use in agriculture, both for existing fields and the establishment of new ones in once unprofitable areas (ISAB 2007). This means that streams, rivers, and lakes will experience additional withdrawal of water for irrigation and increasing contaminant loads from returning effluent. Overall, it is likely that global warming will increase pressures on sturgeon survival and recovery.

Critical Habitat

NMFS has not designated critical habitat for shortnose sturgeon.

Sockeye Salmon

Description of the Species

Sockeye salmon are the second most abundant of the seven Pacific salmon species, and occur in the North Pacific and Arctic oceans and associated freshwater systems. This species' ranges

south as far as the Sacramento River in California and northern Hokkaido in Japan, to as far north as far as Bathurst Inlet in the Canadian Arctic and the Anadyr River in Siberia (Burgner 1991). The largest populations, and hence the most important commercial populations, occur north of the Columbia River

Sockeye salmon exhibit a very diverse life history, characteristically using both riverine and lake habitat throughout their range, exhibiting both freshwater resident and anadromous forms. The vast majority of sockeye salmon are anadromous fish that make use of lacustrine habitat for juvenile rearing. These "lake-type" fish typically spawn in the outlet streams of lakes and occasionally in the lakes themselves. Juvenile sockeye salmon will then use the lake environment for rearing for up to 3 years before migrating to sea. After 1 to 4 years at sea, sockeye salmon will return to their natal lake to spawn. Some sockeye, however, spawn in rivers without lake habitat for juvenile rearing. Offspring of these riverine spawners tend to use the lower velocity sections of rivers as the juvenile rearing environment for 1 to 2 years, or may migrate to sea in their first year.

Sockeye salmon also have a wholly freshwater life history form, called kokanee (Burgner 1991). In some cases a single population will give rise to both the anadromous and freshwater life history form. While in fresh water juveniles of both life history types prey primarily upon insects. The presence of both life history types may be related to the energetic costs of outmigrating to sea, and the productivity of the lacustrine system they inhabit. In coastal lakes, where the migration to sea is relatively short and energetic costs are minimal, kokanee populations are rare.

Once smolts enter the Pacific Ocean, they distribute widely across the North Pacific, generally above 40ºN where a current boundary is located. Season, temperature, salinity, life stage, age, size, availability of prey and population-of-origin are all factors that influence offshore movements (Burgner 1991). Sockeye tend to stay within several dozen feet of the surface, although they tend to be closer to the surface at night versus daytime (Manzer 1964; French et al. 1976). However, they may migrate several thousand miles in search of prey and are considered to travel continuously (Royce et al. 1968). While at sea, sockeye prey upon a variety of organisms, including small fish (capelin, lantern fish, cod, sand lance, herring, and pollock), squid, crustacean larvae, krill, and other invertebrates (Foerster 1968; French et al. 1976; Wing 1977). Thermoclines may also influence vertical distribution, with fish only mingling between surface and deeper waters when the boundary temperature difference is weak. Sockeye appear to prefer cooler waters relative to other salmon species, but younger salmon may prefer warmer sea surface temperatures (39 to 50º F) than larger, older fish (37 to 41º F), possibly an artifact of older fish being distributed further north. Adult upstream migration may be blocked by temperatures above 70º F (McCullough 1999). However, temperatures below 70º F can stress fish by increasing their susceptibility to disease and elevating their metabolism (Brett 1979; Berman 1990). Maturation and timing of return to spawn by sockeye appears to be linked to water temperature, with gonad development increasing in late May through early July (Nishiyama 1984).

Spawning generally occurs in late summer and autumn, but the precise time can vary greatly among populations. Age at maturity varies by region from 2 to 5 years, but is generally 2 to 4

years in Washington State (Burgner 1991). Males often arrive earlier than females on the spawning grounds, and will persist longer during the spawning period. Average fecundity ranges from about 2,000 to 2,400 eggs per female to 5,000 eggs, depending upon the population and average age of the female. Fecundity in kokanee is much lower and may range from about 300 to less than 2,000 eggs.

Incubation is a function of water temperatures, but generally lasts between 100 and roughly 200 days (Burgner 1991). After emergence, fry move rapidly downstream or upstream along the banks to the lake rearing area. Fry emerging from lakeshore or island spawning grounds may simply move along the shoreline of the lake (Burgner 1991).

Ozette Lake Sockeye Salmon

Distribution and Description of the Listed Species

This ESU includes all naturally spawned sockeye salmon in Ozette Lake, Ozette River, Coal Creek, and other tributaries flowing into Ozette Lake, Washington. Composed of only one population, the Ozette Lake sockeye salmon ESU consists of five spawning aggregations or subpopulations which are grouped according to their spawning locations. The five spawning locations are Umbrella and Crooked creeks, Big River, and Olsen's and Allen's beaches (NMFS 2009).

Adult Ozette Lake sockeye salmon enter Ozette Lake through the Ozette River from mid-April to mid-August, holding three to nine months in Ozette Lake prior to spawning in late October through January. Sockeye salmon spawn primarily in lakeshore upwelling areas in Ozette Lake (particularly at Allen's Bay and Olsen's Beach), and in two tributaries Umbrella Creek and Big River. Minor spawning may occur below Ozette Lake in the Ozette River or in Coal Creek, a tributary of the Ozette River. Beach spawners are almost all age-4 adults, while tributary spawners are ages 3 and 5 (Haggerty et al. 2009 in NMFS 2009). Spawning occurs in the fall through early winter, with peak spawning in tributaries in November and December. Eggs and alevins remain in the gravel until the fish emerge as fry in spring. Fry then migrate immediately to the limnetic zone in Ozette Lake, where the fish rear. After one year of rearing, in late spring, Ozette Lake sockeye salmon emigrate seaward as age-1+ smolts, where they spend between 1 and 3 years in ocean before returning to fresh water.

Status and Trends

NMFS originally listed Ozette Lake sockeye salmon ESU as a threatened species in 1999 (64 FR 14528). This classification was retained on June 28, 2005 (70 FR 37160). This ESU includes all naturally spawned populations of sockeye salmon in Ozette Lake, Ozette River, Coal Creek, and other tributaries flowing into Ozette Lake, Washington. Two artificial propagation programs are considered part of this ESU: The Umbrella Creek and Big River sockeye salmon hatchery programs. NMFS considers these artificially propagated populations no more divergent relative to the local natural population than would be expected between closely related natural populations (70 FR 37160).

The historical abundance of Ozette Lake sockeye salmon is poorly documented, but may have

been as high as 50,000 individuals (Blum 1988). The overall abundance of naturally–produced Ozette Lake sockeye salmon is believed to have declined substantially from historical levels. In the first study of lake escapement of Ozette Lake sockeye salmon (Kemmerich 1945), the run size entering the lake was estimated at a level of several thousand fish. These counts appear to be roughly double the current mean lake abundance, considering that they were likely conducted upstream from fisheries in or near to the Ozette River. Makah Fisheries Management (MFM 2000 *in* Good et al. 2005) concluded that there appears to be a substantial decline in the Tribal catch of Ozette Lake sockeye salmon beginning in the 1950s and a similar decline in the run size since the 1920s weir counts reported by Kemmerich (1945).

An analysis of total annual Ozette Lake sockeye salmon abundance (based on adult run size data presented in Jacobs et al. 1996) indicates a trend in abundance averaging -2% per year over the period 1977 through 1998 (NMFS 1998b). The current tributary-based hatchery program was planned and initiated in response to the declining population trend identified for the Ozette Lake sockeye salmon population. The most recent (1996 to 2003) run-size estimates range from a low of 1,609 in 1997 to a high of 5,075 in 2003, averaging approximately 3,600 sockeye per year (NMFS 2009). For return years 2000 to 2003, the 4-year average abundance estimate was slightly over 4,600 sockeye. Because run-size estimates before 1998 are likely to be even more unreliable than recent counts, and new counting technology has resulted in an increase in estimated run sizes, no statistical estimation of trends is reported. The current trends in abundance are unknown for the beach spawning aggregations. Although overall abundance appears to have declined from historical levels, whether this resulted in fewer spawning aggregations, lower abundances at each aggregation, or both, is not known (Good et al. 2005). Based on estimates of habitat carrying capacity, a viable sockeye salmon population in Lake Ozette watershed would range between 35,500 to 121,000 spawners (Rawson et al. 2008 in NMFS 2009).

There has been no harvest of Ozette Lake sockeye salmon for the past four brood-cycle years (since 1982). Prior to that time, ceremonial and subsistence harvests by the Makah Tribe were low, ranging from 0 to 84 fish per year. Harvest has not been an important mortality factor for the population in over 35 years. In addition, due to the early river entry timing of returning Ozette Lake sockeye salmon (beginning in late April, with the peak returns prior to late-May to mid-June), the fish are not intercepted in Canadian and United States marine area fisheries directed at Fraser River sockeye salmon. There are currently no known marine area harvest impacts on Ozette Lake sockeye salmon.

Overall abundance is substantially below historical levels (Good et al. 2005). Declines in abundance have been attributed to a combination of introduced species, predation, loss of tributary populations, a loss of quality of beach spawning habitat, temporarily unfavorable ocean conditions, habitat degradation, and excessive historical harvests (Jacobs et al. 1996). In the last few years the number of returning adults has increased, although some of these individuals are of hatchery origin. This produces uncertainty regarding natural growth rate and productivity of the ESU's natural component. In addition, genetic integrity has perhaps been compromised due to the artificial supplementation that has occurred in this population, since approximately one million sockeye have been released into the Ozette watershed from the late 1930s to present (Kemmerich 1945; Boomer 1995).

Critical Habitat

On September 2, 2005, NMFS designated critical habitat for the Ozette Lake sockeye salmon ESU (70 FR 52630). The specific geographic areas designated as critical are the Hoh/Quillayute Subbasin, Ozette Lake and the Ozette Lake watershed, and include: the Ozette River upstream to endpoints in Big River, Coal Creek, East Branch Umbrella Creek, the North and South Fork of Crooked Creek and several other tributaries. The specific primary constituent elements identified for Lake Ozette sockeye salmon are areas for spawning, freshwater rearing and migration, estuarine areas free of obstruction, nearshore marine areas free of obstructions, and offshore marine areas with good water quality. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, and adequate passage conditions. Only one watershed supports this ESU, and it is rated as having a high conservation value. This watershed is essential to the species' overall conservation by protecting quality growth, reproduction, and feeding.

Snake River Sockeye Salmon

Distribution and Description of the Listed Species

Snake River sockeye salmon are unique compared to other sockeye salmon populations: it spawns at a higher elevation (6,500 feet) and a longer freshwater migration (approximately 900 miles) than any other sockeye salmon population in the world. Sockeye salmon in this ESU spawn in Redfish Lake in Idaho's Stanley Basin (Bjornn et al. 1968; Foerster 1968). Stanley Basin sockeye salmon are separated by 700 or more river miles from two other extant upper Columbia River populations in the Wenatchee River and Okanogan River drainages. These latter populations return to lakes at substantially lower elevations (Wenatchee at 1,870 feet and Okanagon at 912 feet) and occupy different ecoregions. The Snake River sockeye salmon ESU includes all anadromous and residual sockeye salmon from the Snake River basin of Idaho, as well as hatchery individuals from the Redfish Lake Captive Broodstock Program.

Status and Trends

Snake River sockeye salmon were originally listed as endangered in 1991 and retained that classification when their status was reviewed on June 28, 2005 (70 FR 37160). The only extant sockeye salmon population in the Snake River basin at the time of listing was that in Redfish Lake, in the Stanley Basin (upper Salmon River drainage) of Idaho. Other lakes in the Snake River basin historically supported sockeye salmon populations, including Wallowa Lake (Grande Ronde River drainage, Oregon), Payette Lake (Payette River drainage, Idaho) and Warm Lake (South Fork Salmon River drainage, Idaho; Waples et al. 1997). These populations are now considered extinct. Although kokanee, a resident form of *O. nerka*, occur in numerous lakes in the Snake River basin, other lakes in the Stanley Basin, and sympatrically with sockeye in Redfish Lake, resident *O. nerka* were not considered part of the species at the time of listing (1991). Subsequent to the 1991 listing, a residual form of sockeye residing in Redfish Lake was identified. The residuals are non-anadromous, completing their entire life cycle in fresh water, but spawn at the same time and in the same location as anadromous sockeye salmon. In 1993, NMFS determined that residual sockeye salmon in Redfish Lake were part of the Snake River sockeye salmon. Also, artificially propagated sockeye salmon from the Redfish Lake Captive

Propagation program are considered part of this species (70 FR 37160; June 28, 2005).

NMFS has determined that this artificially propagated population is genetically no more than moderately divergent from the natural population (NMFS 2005a). Five lakes in the Stanley Basin historically contained sockeye salmon: Alturas, Pettit, Redfish, Stanley and Yellowbelly (Bjornn et al. 1968). It is generally believed that adults were prevented from returning to the Sawtooth Valley from 1910 to 1934 by Sunbeam Dam. Sunbeam Dam was constructed on the Salmon River approximately 20 miles downstream of Redfish Lake. Whether Sunbeam Dam was a complete barrier to adult migration remains unknown. It has been hypothesized that some passage occurred while the dam was in place, allowing the Stanley Basin population or populations to persist (Bjornn et al. 1968; Waples et al. 1991).

Adult returns to Redfish Lake during the period 1954 through 1966 ranged from 11 to 4,361 fish (Bjornn et al. 1968). Sockeye salmon in Alturas Lake were extirpated in the early 1900s as a result of irrigation diversions, although residual sockeye may still exist in the lake (Chapman and Witty 1993). From 1955 to 1965, the Idaho Department of Fish and Game eradicated sockeye salmon from Pettit, Stanley, and Yellowbelly lakes, and built permanent structures on each of the lake outlets that prevented re-entry of anadromous sockeye salmon (Chapman and Witty 1993). In 1985, 1986, and 1987, 11, 29, and 16 sockeye, respectively, were counted at the Redfish Lake weir (Good et al. 2005). Only 18 natural origin sockeye salmon have returned to the Stanley Basin since 1987. During the fall of 1990, during the course of NMFS' first status review on the species, no fish were observed at Lower Granit Dam or entering the lake and only one fish was observed in each of the two previous years. The first adult returns from the captive broodstock program returned to the Stanley Basin in 1999. From 1999 through 2005, a total of 345 captive brood program adults that had migrated to the ocean returned to the Stanley Basin.

Recent annual abundances of natural origin sockeye salmon in the Stanley Basin have been extremely low. No natural origin anadromous adults have returned since 1998 and the abundance of residual sockeye salmon in Redfish Lake is unknown. This species is entirely supported by adults produced through the captive propagation program at the present time. Current smolt-to-adult survival of sockeye originating from the Stanley Basin lakes is rarely greater than 0.3% (Hebdon et al. 2004). The status of this ESU is extremely precarious, such that there was unanimous consent among the biological review team members that the species remains in danger of extinction (Good et al. 2005).

Critical Habitat

Critical habitat for these salmon was designated on December 28, 1993 (58 FR 68543), and encompasses the waters, waterway bottoms, and adjacent riparian zones of specified lakes and river reaches in the Columbia River that are or were accessible to listed Snake River salmon (except reaches above impassable natural falls, and Dworshak and Hells Canyon Dams). Adjacent riparian zones are defined as those areas within a horizontal distance of 300 feet from the normal line of high water of a stream channel or from the shoreline of a standing body of water. Designated critical habitat includes the Columbia River from a straight line connecting the west end of the Clatsop jetty (Oregon side) and the west end of the Peacock jetty (Washington side) and including all river reaches from the estuary upstream to the confluence of the Snake River, and all Snake River reaches upstream to the confluence of the Salmon River; all

Salmon River reaches to Alturas Lake Creek; Stanley, Redfish, yellow Belly, Pettit, and Alturas Lakes (including their inlet and outlet creeks); Alturas Lake Creek and that portion of Valley Creek between Stanley Lake Creek and the Salmon River. Critical habitat also includes all river lakes and reaches presently or historically accessible to Snake River sockeye salmon. These habitats are critical for the conservation of the species because it provides spawning and juvenile rearing habitat, areas for juvenile growth and development, and migration corridors for smolts to the ocean and adults to spawning habitat from the Pacific Ocean. Limiting factors identified for Snake River sockeye include: reduced tributary stream flow, impaired tributary passage and blocks to migration, and mainstem Columbia River hydropower system mortality.

Steelhead

Description of the Species

Steelhead, the common name of the anadromous form of *O. mykiss*, are native to Pacific Coast streams extending from Alaska south to northwestern Mexico (Moyle 1976; Stolz & Schnell 1991; NMFS 1997b). The life history of this species varies considerably throughout its range. Generally, steelhead can into two races: the stream-maturing type, summer steelhead, enters fresh water in a sexually immature condition and requires several months in fresh water to mature and spawn; and the ocean-maturing type, winter steelhead, enters fresh water with well-developed gonads and spawns shortly after river entry. Variations in migration timing exist between populations, and some river basins have both summer and winter steelhead, while others only have race.

Summer steelhead enter fresh water between May and October in the Pacific Northwest (Nickelson et al. 1992; Busby et al. 1996). They require cool, deep holding pools during summer and fall, prior to spawning (Nickelson et al. 1992). Summer steelhead migrate inland toward spawning areas, overwinter in the larger rivers, resume migration in early spring to natal streams, and then spawn in January and February (Barnhart 1986; Meehan and Bjornn 1991; Nickelson et al. 1992). Winter steelhead enter fresh water between November and April in the Pacific Northwest (Nickelson et al. 1992; Busby et al. 1996), migrate to spawning areas, and then spawn, generally in April and May (Barnhart 1986). Some adults, however, do not enter some coastal streams until spring, just before spawning (Meehan and Bjornn 1991).

There is a high degree of overlap in spawn timing between populations regardless of run type (Busby et al. 1996). Difficult field conditions at that time of year and the remoteness of spawning grounds contribute to the relative lack of specific information on steelhead spawning. Unlike Pacific salmon, steelhead are iteroparous, or capable of spawning more than once before death, although steelhead rarely spawn more than twice before dying; most that do spawn more than twice tend to be female (Nickelson et al. 1992; Busby et al. 1996). Second time spawners often make up about 70 to 85 % of repeat spawners, with third time spawners make up between 10 to 25 % of repeats (Stolz & Schnell 1991). Iteroparity is more common among southern steelhead populations than northern populations (Busby et al. 1996).

As with other salmonids, the larger the fish the more eggs produced. Egg and hatching success are related to the conditions within the redd, and time to hatching is temperature dependent.

Fertilization to hatching is generally less than a month, after which newly hatched fish will remain in the redd for another 2-3 weeks. In late spring, and following yolk sac absorption, alevins emerge from the gravel and begin actively feeding. After emerging from the gravel, fry usually inhabit shallow water along banks of perennial streams. Fry occupy stream margins (Nickelson et al. 1992). Summer rearing takes place primarily in the faster parts of pools, although young-of-the-year are abundant in glides and riffles. Winter rearing occurs more uniformly at lower densities across a wide range of fast and slow habitat types. Some older juveniles move downstream to rear in larger tributaries and mainstem rivers (Nickelson et al. 1992).

Juvenile steelhead migrate little during their first summer and occupy a range of habitats featuring moderate to high water velocity and variable depths (Bisson et al. 1988). Steelhead hold territories close to the substratum where flows are lower and sometimes counter to the main stream; from these, they can make forays up into surface currents to take drifting food (Kalleberg 1958). Juveniles rear in fresh water from 1 to 4 years, then smolt and migrate to the ocean in March and April (Barnhart 1986). Winter steelhead juveniles generally smolt after 2 years in fresh water (Busby et al. 1996). Juvenile steelhead tend to migrate directly offshore during their first summer from whatever point they enter the ocean rather than migrating along the coastal belt as salmon do. Steelhead typically reside in marine waters for 2 or 3 years prior to returning to their natal stream to spawn as 4- or 5-year olds; fish in the northern portion of the range may spend more time rearing in marine waters (Stolz & Schnell 1991). Juveniles feed primarily on insects (chironomids, baetid mayflies, and hydropsychid caddisflies; Merz 1994). Adults feed on aquatic and terrestrial insects, mollusks, crustaceans, fish eggs, minnows, and other small fishes (including greenling and other trout; Chapman and Bjornn 1969; Stolz & Schnell 1991).

Threats

Natural Threats. Steelhead, like other salmon, are exposed to high rates of natural predation each stage of their life stage. The highest mortality occurs between the egg stage and smolt outmigration, and is highest in the first few months following emergence from the redd (Stolz & Schnell 1991). In fresh water, fry fall prey to older steelhead and other trout, as well as birds, sculpin, and various mammals. In the ocean, marine mammals, and other fish prey on steelhead but the extent of such predation is not well known.

Anthropogenic Threats. Steelhead have declined under the combined effects of overharvests in fisheries; competition from fish raised in hatcheries and native and non-native exotic species; dams that block their migrations and alter river hydrology; gravel mining that impedes their migration and alters the dynamics (hydrogeomorphology) of the rivers and streams that support juveniles; water diversions that deplete water levels in rivers and streams; destruction or degradation of riparian habitat that increase water temperatures in rivers and streams sufficient to reduce the survival of juvenile steelhead; and land use practices (logging, agriculture, urbanization) that destroy wetland and riparian ecosystems while introducing sediment, nutrients, biocides, metals, and other pollutants into surface and ground water and degrade water quality in the fresh water, estuarine, and coastal ecosystems throughout the species' range. These threats for are summarized in detail under Chinook salmon.

Central California Coast Steelhead

Distribution and Description of the Listed Species

The Central California Coast steelhead DPS includes all naturally spawned anadromous steelhead populations below natural and manmade impassable barriers in California streams from the Russian River (inclusive) to Aptos Creek (inclusive), and the drainages of San Francisco, San Pablo, and Suisun Bays eastward to Chipps Island at the confluence of the Sacramento and San Joaquin Rivers. Tributary streams to Suisun Marsh including Suisun Creek, Green Valley Creek, and an unnamed tributary to Cordelia Slough (commonly referred to as Red Top Creek), excluding the Sacramento-San Joaquin River Basin, as well as two artificial propagation programs: the Don Clausen Fish Hatchery, and Kingfisher Flat Hatchery/ Scott Creek (Monterey Bay Salmon and Trout Project) steelhead hatchery programs.

The DPS is entirely composed of winter run fish, as are those DPSs to the south. As winter-run fish adults migrating upstream from December-April, and smolts emigrating between March-May (Shapovalov and Taft 1954; Hayes et al. 2008). At the time of the 1996 status review and 1997 listing, little information was available on the specific demographics and life history characteristics of steelhead in this DPS. While age at smoltification typically ranges from 1 to 4 years, recent studies by Sogard et al. (2009) that growth rates in Soquel Creek likely prevent juveniles from undergoing smoltification until age 2. Survival in freshwater reaches tends to be higher in summer and lower from winter through spring for year classes 0 and 1 (Sogard et al. 2009). Larger individuals also survive more readily than do smaller fish within year classes (Sogard et al. 2009). Greater movement of juveniles in fresh water has been observed in winter and spring versus summer and fall time periods, with smaller individuals more likely to move between stream areas (Sogard et al. 2009). Growth rates during this time have rarely been observed to exceed 0.3 mm per day and are highest in winter through spring, potentially due to higher water flow rates and greater food availability (Boughton et al. 2007; Hayes et al. 2008; Sogard et al. 2009).

Status and Trends

The Central California Coast steelhead DPS was listed as a threatened species on August 18, 1997 (62 FR 43937); threatened status was reaffirmed on January 5, 2006 (71 FR 834). Table 15 identifies runs within the Central California Coast steelhead DPS and their estimated run sizes.

Table 15. Central California coast steelhead populations and their estimated abundances

Basin	Estimated Abundance[a]	Year
Russian River	65,000	1970
	1,750-7,000	1994
Lagunitas	500	1994
	400-500	1990s
San Gregorio	1,000	1973
Waddell Creek	481	1933-1942
	250*	1982
	150*	1994
Scott Creek	400	1991
	<100	1991

	300	1994
San Vicente Creek	150*	1982
	50*	1994
San Lorenzo River	20,000	Pre 1965
	1,614	1977
	>3,000*	1978
	600	1979
	3,000	1982
	"few"	1991
	<150*	1994
Soquel Creek	500-800*	1982
	<100	1991
	50-100*	1994
Aptos Creek	200*	1982
	<100	1991
	50-75*	1994

[a]A complete list of data sources is available in Good et al. 2005. According to Good et al. the basis for certain estimates is questionable (noted with an asterisk above).

Estimates of historical abundance are provided here only for background, as the accuracy of the estimates is unclear. An estimate of historical abundance for the total DPS is provided by CDFG at 94,000 fish. This estimate is based on a partial data set and "best professional judgment" (see Good et al. 2005 for a discussion). Other estimates of historical abundance are on a per river basis: According to Busby et al. (1996), Shapovalov and Taft (1954) described an average of about 500 adults in Waddell Creek (Santa Cruz County) for the 1930s and early 1940s, whereas Johnson (1964) estimated a run size of 20,000 steelhead in the San Lorenzo River before 1965. Most of the estimates for run sizes within the DPS are more recent (see Table 15). Two rivers thought to have contained the largest populations within the DPS were the Russian River, and the San Lorenzo River. Based on run size estimates from the 1990s, the Russian River is still likely the largest run within the DPS, albeit estimates suggest the population has declined between 90-96 % from 1970 levels.

No current estimates of total population size are available for this DPS, and consequently there is no time series data available to evaluate the central California coast steelhead population trends. Rather, a general dearth of data on adult steelhead within the DPS, led the biological review team to examine data collected on juvenile steelhead (see Good et al. 2005). In general, juvenile data is considered a poor indicator of the reproductive portion of the population as juvenile age classes exhibit greater mortality rates, which are closely tied to stochastic events, and may move widely within a basin (which may include intermixing with other populations). There is no simple relationship between juvenile and adult numbers (Shea and Mangel 2001). Nonetheless, there was not enough adult data upon which the biological review team could base an assessment of the population trends within the DPS. Therefore, the biological review team log-transformed and normalized juvenile survey data from a number of watersheds (presumed populations). As a result, the team derived trend estimates for five populations: the San Lorenzo River, Scott Creek, Waddell Creek, Gazos Creek, and Redwood Creek in Marin County (see Good et al. 2005 for a detailed discussion of the approach). All populations exhibited downward trends in abundance. Accordingly, provided the juvenile data is representative of the true trend, this data suggests that there is an overall downward trend in abundance in the DPS.

In the most recent review of the status of this DPS, most members of the biological review team (69 %) considered this DPS "likely to become endangered" thus supporting the renewal of the threatened status for central California coast steelhead. Notably, 25 % of the team voted that the DPS be upgraded to endangered status (voted the DPS as" in danger of extinction"; Good et al. 2005). Abundance and productivity were of relatively high concern (as a contributing factor to risk of extinction), and spatial structure was also of concern.

Since the original status review, fishing regulations have changed in a way that probably reduces extinction risk for Central California Coast steelhead. Ocean sport harvest is prohibited, and ocean harvest is considered rare. Although freshwater streams are closed to fishing year round, CDFG has identified certain streams as exceptions where they allow catch-and-release angling or summer trout fishing. In catch-and-release streams, all wild steelhead must be released unharmed.

Critical Habitat

Critical habitat was designated for the Central California Coast steelhead DPS on September 2, 2005 (70 FR 52488), and includes areas within the following hydrologic units: Russian River, Bodega, Marin Coastal, San Mateo, Bay Bridge, Santa Clara, San Pablo, and Big Basin. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this ESU identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The critical habitat designation (70 FR 52488) contains additional details on the sub-areas that are included as part of this designation, and the areas that were excluded from designation.

In total, Central California Coast steelhead occupy 46 watersheds (fresh water and estuarine). The total area of habitat designated as critical includes about 1,500 miles of stream habitat and about 400 square miles of estuarine habitat (principally Humboldt Bay). This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. In estuarine areas the lateral extent is defined by the extreme high water because extreme high tide areas encompass those areas typically inundated by water and regularly occupied by juvenile salmon during the spring and summer, when they are migrating in the nearshore zone and relying on cover and refuge qualities provided by these habitats, and while they are foraging. Of the 46 occupied watersheds reviewed in NMFS' assessment of critical habitat for Central California Coast steelhead, 14 watersheds received a low rating of conservation value, 13 received a medium rating, and 19 received a high rating of conservation value for the species.

California Central Valley Steelhead

Distribution and Description of the Listed Species

California Central Valley steelhead occupy the Sacramento and San Joaquin Rivers and their tributaries, although they were once widespread throughout the Central Valley (Busby et al. 1996; Zimmerman et al. 2009). Steelhead were found from the upper Sacramento and Pit River systems (now inaccessible due to Shasta and Keswick Dams), south to the Kings and possibly the Kern River systems (now inaccessible due to extensive alteration from water diversion projects), and in both east- and west-side Sacramento River tributaries (Yoshiyama et al. 1996). The present distribution has been greatly reduced (McEwan and Jackson 1996). The California Advisory Committee on Salmon and Steelhead (1988) reported a reduction of steelhead habitat from 6,000 miles historically to 300 miles today. Historically, steelhead probably ascended Clear Creek past the French Gulch area, but access to the upper basin was blocked by Whiskeytown Dam in 1964 (Yoshiyama et al. 1996). Steelhead also occurred in the upper drainages of the Feather, American, Yuba, and Stanislaus Rivers which are now inaccessible (McEwan and Jackson 1996; Yoshiyama et al. 1996).

Existing wild steelhead populations in the Central Valley are mostly confined to the upper Sacramento River and its tributaries, including Antelope, Deer, and Mill Creeks and the Yuba River. Populations may exist in Big Chico and Butte Creeks and a few wild steelhead are produced in the American and Feather Rivers (McEwan and Jackson 1996). Recent snorkel surveys (1999 to 2002) indicate that steelhead are present in Clear Creek (J. Newton, FWS, pers. comm. 2002, *in* Good et al. 2005). Because of the large resident *O. mykiss* population in Clear Creek, steelhead spawner abundance has not been estimated. Until recently, steelhead were thought to be extirpated from the San Joaquin River system. Recent monitoring has detected small self-sustaining populations of steelhead in the Stanislaus, Mokelumne, Calaveras, and other streams previously thought to be void of steelhead (McEwan 2001). On the Stanislaus River, steelhead smolts have been captured in rotary screw traps at Caswell State Park and Oakdale each year since 1995 (Demko et al. 2000). It is possible that naturally spawning populations exist in many other streams but are undetected due to lack of monitoring programs.

The Sacramento and San Joaquin Rivers offer the only migration route to the drainages of the Sierra Nevada and southern Cascade mountain ranges for anadromous fish. The CDFG considers all steelhead in the Central Valley as winter steelhead, although "three distinct runs," including summer steelhead, may have occurred there as recently as 1947 (CDFG 1995 in Good et al. 2005; McEwan and Jackson 1996). Steelhead in these basins travel extensive distances in fresh water (some exceed 300 km to their natal streams), making these the longest freshwater migrations of any population of winter steelhead. The upper Sacramento River essentially receives a single continuous run of steelhead in from July through May, with peaks in September and February. Spawning begins in late December and can extend into April (McEwan and Jackson 1996).

Status and Trends

NMFS originally listed California Central Valley steelhead as threatened in 1998; this status was reviewed and retained on January 5, 2006 (71 FR 834). Historic Central Valley steelhead run

size is difficult to estimate given the paucity of data, but may have approached one to two million adults annually (McEwan 2001). By the early 1960s, the steelhead run size had declined to about 40,000 adults (McEwan 2001). Over the past 30 years, the naturally spawned steelhead populations in the upper Sacramento River have declined substantially. Hallock et al. (1961) estimated an average of 20,540 adult steelhead occurred in the Sacramento River (upstream of the Feather River). Steelhead counts at Red Bluff Diversion Dam declined from an average of 11,187 for the period of 1967 to 1977, to an average of approximately 2,000 through the early 1990s, with an estimated total annual run size for the entire Sacramento-San Joaquin system at no more than 10,000 adults (based on Red Bluff Diversion Dam counts; McEwan and Jackson 1996; McEwan 2001). The five-year geometric mean, however, is just under 2,000 steelhead (Table 16), and the long-term trend suggests that the population is declining.

Table 16. California Central Valley steelhead and their long-term trend

Population	5-Year Mean (Min – Max)[a]	λ	Long-term trend[b]
Sacramento River	1,952 (1,425-12,320)	0.95 (0.90,1.02)	-0.09 (-0.13,-0.06)

[a]Refers to the period ending in 1993, when steelhead counts at Red Bluff Diversion dam ended. Data reported in Good et al. 2005.
[b] 90% confidence limits in parentheses.

The only consistent data available on steelhead numbers in the San Joaquin River basin come from CDFG mid-water trawling samples collected on the lower San Joaquin River at Mossdale. These data indicate a decline in steelhead numbers in the early 1990s, which have remained low through 2002 (Good et al. 2005). In 2004, a total of 12 steelhead smolts were collected at Mossdale (CDFG, unpublished data *in* Good et al. 2005).

Reynolds et al. (1993) reported that 95% of salmonid habitat in California's Central Valley has been lost, largely due to mining and water development activities. They also noted that declines in Central Valley steelhead populations are "due mostly to water development, inadequate instream flows, rapid flow fluctuations, high summer water temperatures in streams immediately below reservoirs, diversion dams which block access, and entrainment of juveniles into unscreened or poorly screened diversions." Thus, overall habitat problems in this ESU relate primarily to water development resulting in inadequate flows, flow fluctuations, blockages, and entrainment into diversions (McEwan and Jackson 1996). Other problems related to land use practices (agriculture and forestry) and urbanization have also contributed to population declines. It is unclear how harvest has affected California's Central Valley steelhead, although it is likely a continuing threat. A CDFG creel census in 2000 indicated that most fish are caught and released, but due to the size of the catch and release fishery (more than 14,000 steelhead were caught and released according to the survey) even a small amount of mortality in this fishery could cause declines in the populations.

Critical Habitat

NMFS designated critical habitat for California Central Valley steelhead on September 2, 2005 (70 FR 52488). Specific geographic areas designated include the following CALWATER hydrological units: Tehama, Whitmore, Redding, Eastern Tehama, Sacramento Delta, Valley-Putach-Cache, American River, Marysville, Yuba, Valley American, Colusa Basin, Butte Creek,

Ball Mountain, Shata Bally, North Valley Floor, Upper Calaveras, Stanislaus River, San Joaquin Valley, Delta-Mendota Canal, North Diablo Range, and the San Joaquin Delta. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this ESU identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The critical habitat designation (70 FR 52488) contains additional details on the sub-areas that are included as part of this designation, and the areas that were excluded from designation.

In total, California Central Valley steelhead occupy 67 watersheds (freshwater and estuarine). The total area of habitat designated as critical includes about 2,300 miles of stream habitat and about 250 square miles of estuarine habitat in the San Franciso-San Pablo-Suisan Bay estuarine complex. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. In estuarine areas the lateral extent is defined by the extreme high water because extreme high tide areas encompass those areas typically inundated by water and regularly occupied by juvenile salmon during the spring and summer, when they are migrating in the nearshore zone and relying on cover and refuge qualities provided by these habitats, and while they are foraging. Of the 67 watersheds reviewed in NMFS' assessment of critical habitat for California Central Valley steelhead, seven watersheds received a low rating of conservation value, three received a medium rating, and 27 received a high rating of conservation value for the species.

Lower Columbia River Steelhead

Distribution and Description of the Listed Species

Lower Columbia River steelhead include naturally produced steelhead returning to Columbia River tributaries on the Washington side between the Cowlitz and Wind rivers in Washington and on the Oregon side between the Willamette and Hood rivers, inclusive. In the Willamette River, the upstream boundary of this species is at Willamette Falls. This species includes both winter and summer steelhead. Two hatchery populations are included in this species, the Cowlitz Trout Hatchery winter-run population and the Clackamas River population but neither was listed as threatened. Table 17 identifies the populations that comprise Lower Columbia River steelhead and summarizes several measures available to characterize population viability.

Summer steelhead return sexually immature to the Columbia River from May to November, and spend several months in fresh water prior to spawning. Winter steelhead enter fresh water from November to April, are close to sexual maturation during freshwater entry, and spawn shortly after arrival in their natal streams. Where both races spawn in the same stream, summer steelhead tend to spawn at higher elevations than the winter forms.

Status and Trends

NMFS listed Lower Columbia River steelhead as threatened on March 19, 1998 (63 FR 13347), and reaffirmed their status as threatened on January 5, 2006 (71 FR 834). The 1998 status review noted that this ESU is characterized by populations at low abundance relative to historical levels, significant population declines since the mid-1980s, and widespread occurrence of hatchery fish in naturally spawning steelhead populations. During this review NMFS was unable to identify any natural populations that would be considered at low risk.

All populations declined between 1980 and 2000, with sharp declines beginning in 1995. Those with adequate data for modeling are estimated to have a high extinction risk (Good et al. 2005). Abundance trends are generally negative, showing that most populations are in decline, although some populations, particularly summer run, have shown higher return in the last 2 to 3 years. Historical counts in some of the larger tributaries (Cowlitz, Kalama, and Sandy Rivers) suggest the population probably exceeded 20,000 fish while in the 1990s fish abundance dropped to 1,000 to 2,000. Recent abundance estimates of natural-origin spawners range from completely extirpated for some populations above impassable barriers to over 700 for the Kalama and Sandy winter-run populations. A number of the populations have a substantial fraction of hatchery-origin spawners in spawning areas, and are hypothesized to be sustained largely by hatchery production. Exceptions are the Kalama, the Toutle, and East Fork Lewis winter-run populations. These populations have relatively low recent mean abundance estimates with the largest being the Kalama (geometric mean of 728 spawners).

Table 17. Lower Columbia River steelhead populations and select measures of population viability

Life History	Population	Historical Abundance[a]	Mean Number of Spawners	Percent Hatchery Contribution	Median Short-term Growth Rate (λ)[b]
Winter	Cispus River				
	Tilton River		2,787[c]	73	
	Upper Cowlitz River				
	Lower Cowlitz River	1,672			
	Coweeman River	2,243	466[d]	50	0.920, 0.787
	South Fork Toutle River	2,627	504[d]	2	0.933, 0.929
	North Fork Toutle River	3,770	196[d]	0	1.038, 1.038
	Kalama River	554	726[d]	0	0.984, 0.922
	North Fork Lewis River	713			
	East Fork Lewis River	3,131			
	Salmon Creek				
	Washougal River	2,497	323[d]	0	
	Clackamas River		560[e]	41	0.875, 0.830
	Sandy River		977e	42	0.866, 0.782
	Lower Columbia Gorge tributaries	793			
	Upper Columbia Gorge tributaries	243			
	Hood River		756[f]	52	
Summer	Wind River	2,288	472[g]	5	0.995, 0.903
	Hood River		931[f]	83	Unknown
	Washougal River	1,419	264[g]	8	1.029, 0.960
	East Fork Lewis River	422	434[g]	25	

North Fork Lewis River Kalama River	3,165	474[g]	32	0.900, 0.664

[a]All data reported by Good et al. 2005. Estimate of historical abundance derived through EDT model associated with large uncertainty. Model also incorporates presently available habitat that was not historically available and vice versa.
[b]λ calculation assumed either hatchery fish fail to reproduce or reproduce at the rate of wild individuals, respectively.
[c]Data from 2002.
[d]Data from 1998-2002.
[e]Data from 1997-2001.
[f]Data from 1996-2000.
[g]Data from 1999-2003.

Critical Habitat

NMFS designated critical habitat for Lower Columbia River steelhead on September 2, 2005 (70 FR 52630). Designated critical habitat includes the following subbasins: Middle Columbia/Hood subbasin, Lower Columbia/Sandy subbasin, Lewis subbasin, Lower Columbia/Clatskanie subbasin, Upper Cowlitz subbasin, Cowlitz subbasin, Clackamas subbasin, Lower Willamette subbasin, and the Lower Columbia River corridor. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this DPS identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The critical habitat designation (70 FR 52630) contains additional description of the watersheds that are included as part of this designation, and any areas specifically excluded from the designation.

In total, Lower Columbia River steelhead occupy 32 watersheds. The total area of habitat designated as critical includes about 2,340 miles of stream habitat. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. Of the 32 watersheds reviewed in NMFS' assessment of critical habitat for Lower Columbia River steelhead, two watersheds received a low rating of conservation value, 11 received a medium rating, and 26 received a high rating of conservation value for the species. Limiting factors identified for Lower Columbia River steelhead include: degraded floodplain and steam channel structure and function, reduced access to spawning/rearing habitat, altered stream flow in tributaries, excessive sediment and elevated water temperatures in tributaries, and hatchery impacts.

Middle Columbia River Steelhead

Distribution and Description of the Listed Species

The Middle Columbia River steelhead DPS includes all naturally spawned anadromous steelhead populations below natural and manmade impassible barriers in Oregon and Washington drainages upstream of the Hood and Wind River systems, up to and including the Yakima River (61 FR 41541). Steelhead from the Snake River Basin (described elsewhere) are excluded from this DPS. Seven artificial propagation program are part of this DPS: The Touchet River

endemic, Yakima River kelt reconditioning program (in Satus Creek, Toppenish Creek, Naches River, and the Upper Yakima River), and the Umatilla River and the Deschutes River steelhead hatchery programs. These artificially propagated populations are considered no more divergent relative to the local natural populations than would be expected between closely related natural populations within the DPS.

Middle Columbia River steelhead occupy the intermontane region of the Pacific Northwest, which includes some of the driest areas in the region generally receiving less than 15.7 inches of rainfall annually. Major drainages in this ESU are the Deschutes, John Day, Umatilla, Walla Walla, Yakima, and Klickitat river systems. The area is generally characterized by its dry climate and harsh temperature extremes. Almost all steelhead populations within this DPS are summer-run fish; the only exceptions are the only populations of inland winter steelhead, which occur in the Klickitat River and Fifteenmile Creek (Busby et al. 1996). According to Interior Columbia Basin Technical Recovery Team (ICBTRT 2003) this DPS is comprised of 16 putative populations in four major population groups (Cascades Eastern Slopes Tributaries, John Day River, Walla Walla and Umatilla Rivers, and Yakima River) and one unaffiliated independent population (Rock Creek). See Table 18 for a list of extant (putative) populations that compose this DPS. There are two extinct populations in the Cascades Eastern Slope major population group, the White Salmon River and Deschutes Crooked River above the Pelton/Round Butte Dam complex. Present population structure is delineated largely on the basis of geographical proximity, topography, distance, ecological similarities or differences. Additional genetic studies are needed to describe the DPS substructure, as well as the fine-scale genetic structure of the populations within a particular basin (e.g., John Day River).

Table 18. Middle Columbia River steelhead populations and select measures of population viability

Population[a]	Major Population Groups	Mean Number of Spawners (range)[b]	Percent Hatchery Contribution[c]	Long-term Growth Rate (λ)[d]
Klickitat River	Cascade Eastern Slope	155 redds (97-261)		
Fifteenmile Creek	Cascade Eastern Slope	2.87 rpm (1.3-6.0)	0	1.129
Deschutes River - eastside	Cascade Eastern Slope	13,455 (10,026-21,457)	72	1.022, 0.840, 0.942
Descutes River – westside	Cascade Eastern Slope			
John Day lower mainstem tributaries	John Day River	1.4 rpm (0-5.4)		1.013
North Fork John Day	John Day River	Upper NF - 2.57 rpm (1.6-5.0)[e]		1.011
		Lower NF - 3.52 rpm (1.5-8.8)		1.174
Middle Fork John Day	John Day River	3.70 rpm (1.7-6.2)		0.966
South Fork John Day	John Day River	2.52 rpm (0.9-8.2)		0.967
John Day upper mainstem	John Day River	2,122 (926-4,168)	4	0.975, 0.966
Rock Creek	Unaffiliated Area			
Umatilla River	Walla Walla & Umatilla	2,486 (1,480-5,157)	40	1.007, 0.969
Walla Walla	Walla Walla & Umatilla			
Touchet River	Walla Walla & Umatilla	345 (273-527)[f]	16	0.961, 0.939
Toppenish & Satus	Yakima River			

Creek				
Naches River	Yakima River			
Yakima River upper mainstem	Yakima River	1,801 (1,058-4,061)	3	1.009

[a]Population groups defined by the ICBTRT (2003).
[b]Values represent the 5-year geometric mean in spawners, redds, or redds per mile (RPM). Values calculated from data series using years 1997-2001 or 1998-2001. See Good et al. (2005) for details.
[c]Hatchery production in the recent past and at present consists of locally-derived broodstock, although straying of production fish into the Deschutes River has been a persistent problem. Data from Good et al. 2005.
[d]Multiple estimates for long-term growth (λ) presented for some populations representing two different assumptions on the contribution of hatchery fish to the natural production. Where two or more values are presented, the first value reflects the assumption that hatchery fish do not contribute to natural production, and the second value reflects the assumption that hatchery contribute to natural production at the same rate as natural-origin spawners. Deschutes River values are reflective of total population, not eastside only. The λ value is calculated from data (1980-1999) from Warm Springs area. Data series upon which values are calculated varies across basins. See Good et al. (2005) for details on the length and time of data series available by population.

Most Middle Columbia River steelhead smolt at 2 years of age and spend 1 to 2 years at sea prior to re-entering natal river systems. They may remain in such rivers for up to a year prior to spawning (Howell et al. 1985). Within this ESU, the Klickitat River is unusual, as it produces both summer and winter steelhead. The summer steelhead are dominated by year-class-two ocean steelhead, whereas most other rivers in this region produce about equal numbers of both age-one and age-two ocean steelhead. Factors contributing to the decline of Middle Columbia river steelhead include hydropower development and agriculture; these land uses impede or prevent migrations, alter water availability, and alter water chemistry and temperatures.

Status and Trends

Middle Columbia River steelhead were listed as threatened in 1999 (64 FR 14517), and their status was reaffirmed on January 5, 2006 (71 FR 834). The precise pre-1960 abundance of this species is unknown. Based upon the Washington Department of Fish and Wildlife's estimates of the historic run size for the Yakima River at 100,000 steelhead, Busby et al. (1996) surmised that total DPS abundance likely exceeded 300,000 returning adults. By 1993, the estimated 5-year average size (ending in 1993) of the Middle Columbia steelhead DPS was 142,000 fish (Busby et al. 1996). Survey data collected between 1997 and 2001 indicates that several populations within the DPS have increased since the last status review (Good et al. 2005). However, long-term annual population growth rate (λ) is negative for most populations (see Table 18).

In contrast, short term trends in major areas were positive for 7 of the 12 areas with available data (see Good et al. 2005). Spawner numbers in the Yakima River, the Deschutes River and sections of the John Day River system were substantially higher compared to numbers surveyed between 1992 and 1997 (Good et al. 2005). Similarly, spawner numbers substantially increased in the Umatilla River and Fifteenmile Creek relative to annual levels in the early 1990s. Nonetheless, most populations remain below interim target levels. For instance, the Yakima River returns are still substantially below interim target levels of 8,900 (the current 5-year average is 1,747 fish) and estimated historical return levels. In fact, the majority of spawning occurs in only one tributary, Satus Creek (Berg 2001 in Good et al. 2005). Based on recent 5-year geometric means, only the Deschutes River exceeded interim target levels (Good et al. 2005). While increases in short-term trends could suggest improvements within the DPS, given that the average population growth rate across all streams is negative (0.98 assuming hatchery spawners do not contribute to production, and 0.97 assuming that both hatchery and natural-

origin fish contribute equally) and evidence of large fluctuation in marine survival for the species, recent increases in population sizes must be viewed cautiously.

Critical Habitat

NMFS designated critical habitat for Middle Columbia River steelhead on September 2, 2005 (70 FR 52630). Designated critical habitat includes the following subbasins: Upper Yakima, Naches, Lower Yakima, Middle Columbia/Lake Wallula, Walla Walla, Umatilla, Middle Columbia/Hood, Klickitat, Upper John Day, North Fork John Day, Middle Fork John Day, Lower John Day, Lower Deschutes, Trout, and the Upper Columbia/Priest Rapids subbasins, and the Columbia River corridor. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this DPS identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The final rule (70 FR 52630) lists the watersheds that comprise the designated subbasins and any areas that are specifically excluded from the designation.

In total, there are 114 watersheds within the range of Middle Columbia River steelhead. The total area of habitat designated as critical includes about 5,800 miles of stream habitat. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. Of the 114 watersheds reviewed in NMFS' assessment of critical habitat for Middle Columbia River steelhead, nine watersheds received a low rating of conservation value, 24 received a medium rating, and 81 received a high rating of conservation value for the species. Although pristine habitat conditions are still present in some wilderness, roadless, and undeveloped areas, habitat complexity has been greatly reduced in many areas of designated critical habitat for Middle Columbia River steelhead. Limiting factors identified for Middle Columbia River steelhead include: hydropower system mortality, reduced stream flow, impaired passage, excessive sediment, degraded water quality, and altered channel morphology and floodplain.

Northern California Steelhead

Distribution and Description of the Listed Species

The Northern California DPS of steelhead includes all naturally spawned steelhead populations below natural and manmade impassible barriers in California coastal river basins from Redwood Creek south to, but not including the Russian river, and two artificial propagation programs (Yager Creek Hatchery, and North Fork Gualala River Hatchery). In the recent update on the status of this DPS, the southern boundary of the DPS was redefined to include the small coastal streams south of the Gualala River (between the Gualala River and the Russian River) that support steelhead. This DPS consists of winter and summer-run fish, as well as "half-pounders" – a sexually steelhead that returns from the sea after spending less than a year in the ocean. Generally, a half-pounder will overwinter in freshwater and return to the ocean in the spring.

Status and Trends

NMFS listed Northern California steelhead as threatened on June 7, 2000 (65 FR 36074), and reaffirmed their status as threatened on January 5, 2006 (71 FR 834). Long-term data sets are limited for Northern California steelhead. Before 1960, estimates of abundance specific to this DPS were available from dam counts in the upper Eel River (Cape Horn Dam; annual average number of adults was 4,400 in the 1940s), the South Fork Eel River (Benbow Dam; annual average number of adults was 18,000 in the 1940s), and the Mad River (Sweasey Dam; annual average number of adults was 3,800 in the 1940s). According to California Department of Fish & Game nearly 200,000 spawning steelhead may have comprised this DPS in the early 1960s (Good et al. 2005). At the time of the first status review on this population, adult escapement trends could be calculated for seven populations. Five of the seven populations exhibited declines, while two exhibited increases with a range of almost 6% annual decline to a 3.5% increase. At the time, little information was available on the actual contribution of hatchery fish to natural spawning, there was and continues to be insufficient information to calculate an overall abundance estimate for Northern California steelhead (Busby et al. 1996).

Recent time series data is also limited for this DPS, with recent abundance estimates available for only four populations, three summer-run and one winter-run. Similarly, Good et al. (2005) could only calculate the population growth rate for three populations (see Table 19). Population growth rates are negative for two of the three populations, the South Fork Eel River winter-run and the Middle Fork Eel River summer-run. Based on time series data for the Middle Fork Eel River, both the long-term and short-term trends are downward. Due to the lack of adult data on which to base their risk assessment, Good et al. (2005) also examined data on juvenile steelhead, and found both upward and downward trends. The lack of data for the populations within this DPS, particular winter-run fish is of continuing concern.

Table 19. Northern California steelhead salmon populations and select measures of population viability

River	Historical Abundance[a]	Mean Number (CI)[b]	Growth Rate (λ)[c]
Redwood Creek	10,000	3 (n/a)	
Mad River	6,000	162 (162-384)[d]	1.00 (0.93,1.05)[e]
Freshwater Creek winter run		32 (25-32)	
Eel River -Total	82,000		
South Fork Eel River	34,000		0.98 (0.92,1.02)
Middle Fork Eel River	23,000	418 (384-1,246)[e]	0.98 (0.93,1.04)[g]
Mattole River	12,000		
Ten Mile River	9,000		
Noyo River	8,000		
Big River	12,000		
Navarro River	16,000		
Garcia River	4,000		
Gualala River	16,000		
Other Humboldt County streams	3,000		
Other Mendocino County streams	20,000		

[a]Historical abundances (1963) are considered uncertain by the author, California Department of Fish & Game. All data are reported in Good et al. 2005.

[b]Value represents the geometric mean number of fish surveyed by snorkel counts or weir counts (e.g., Mad River and MF Eel counts are from snorkel surveys – for the MF Eel River these are snorkel counts of fish holding in pools of the main stem). See Good et al. 2005 for details.

[c]Growth rate calculated upon method where a λ=1.0 could describe a population that is in decline due to environmental stochasticity.

[d]Five year mean of Mad River summer-run steelhead only.
[e]Population growth rate calculated on Mad River winter-run steelhead only.

Critical Habitat

NMFS designated critical habitat for Northern California steelhead on September 2, 2005 (70 FR 52488). Specific geographic areas designated include the following CALWATER hydrological units: Redwood Creek, Trinidad, Mad River, Eureka Plain, Eel River, Cape Mendocino, and the Mendocino Coast. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this DPS identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The critical habitat designation (70 FR 52488) contains additional details on the sub-areas that are included as part of this designation, and the areas that were excluded from designation.

In total, Northern California steelhead occupy 50 watersheds (fresh water and estuarine). The total area of habitat designated as critical includes about 3,000 miles of stream habitat and about 25 square miles of estuarine habitat, mostly within Humboldt Bay. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. In estuarine areas the lateral extent is defined by the extreme high water because extreme high tide areas encompass those areas typically inundated by water and regularly occupied by juvenile salmon during the spring and summer, when they are migrating in the nearshore zone and relying on cover and refuge qualities provided by these habitats, and while they are foraging. Of the 50 watersheds reviewed in NMFS' assessment of critical habitat for Northern California steelhead, nine watersheds received a low rating of conservation value, 14 received a medium rating, and 27 received a high rating of conservation value for the species. Two estuarine areas used for rearing and migration (Humboldt Bay and the Eel River estuary) also received a rating of high conservation value.

Puget Sound Steelhead

Distribution and Description of the Listed Species

The Puget Sound DPS for steelhead includes all naturally spawned anadromous winter-run and summer-run steelhead populations in watersheds of the Strait of Juan de Fuca, Puget Sound and Hood Canal, Washington. Boundaries of this DPS extend to and include the Elwha River to the west, and the Nooksack River and Dakota Creek to the north. Hatchery production of steelhead is widespread throughout this DPS, but only two artificial propagation programs are part of this DPS: the Green River natural and Hamma Hamma winter-run steelhead hatchery populations. The remaining hatchery programs are not considered part of the Puget Sound steelhead DPS because they are more than moderately diverged from the local native populations (NMFS 2005c).

The oceanic distribution of Puget Sound steelhead is not well understood. Winter and summer runs from multiple DPS' comingle in the North Pacific Ocean and some may undergo extensive migrations as a result of the location of their natal streams and oceanic "centers of abundance" (Light et al. 1989). Tagging and genetic studies indicate that Puget Sound steelhead migrate to the central North Pacific ocean (see French et al. 1975, Hartt and Dell 1986, and Burgner et al. 1992 in NMFS 2005c). However, the fjord-like ecosystem of Puget Sound may affect steelhead migration patterns; for example, some populations of coho and Chinook salmon, at least historically, remained within Puget Sound and did not migrate to the Pacific Ocean itself. Even when Puget Sound steelhead migrate to the high seas, they may spend considerable time as juveniles or adults in the protected marine environment of Puget Sound. Oceanic residence times varies among populations within the DPS, with some populations spending only one season in the ocean and others spending three years in marine waters before returning to their natal stream for spawning. Generally, winter-run steelhead enter their natal freshwater systems later (November to April) in the year than summer-run steelhead (May to October), and thus have a shorter freshwater residence time just prior to spawning. The result is that winter-run steelhead have a lower pre-spawn mortality rate than summer-run steelhead (NMFS 2005c). Winter-run steelhead are also more prevalent than summer-run fish, comprising 37 of the 53 populations within this DPS.

Status and Trends

NMFS listed Puget Sound steelhead as a threatened species on May 11, 2007 (72 FR 26722). At the time of the listing, the biological review team concluded that: the viability of Puget Sound steelhead is at a high risk due to declining productivity and abundance; Puget Sound steelhead are at moderate risk due to reduced spatial complexity and connectivity among populations within the DPS, and reduction in life-history diversity within populations and from the threats posed by artificial propagation and harvest. The Puget Sound steelhead DPS includes 53 putative populations; most of which are composed of winter-run fish. Summer-run populations within Puget Sound are small, with most averaging less than 200 spawners, and most lack sufficient data to estimate population abundance. Table 20 lists several of the populations that comprise Puget Sound steelhead as well as some statistics summarizing their current status.

In general, steelhead are most abundant in the northern Puget Sound streams. The largest populations in this DPS are in the Skagit River and Snohomish River winter-run steelhead populations. The recent geometric mean escapement is 5,608 winter-run steelhead in the Skagit, and 3,230 winter-run steelhead in the Snohomish River. The Green River and Puyallup River populations, in central Puget Sound, are the next largest populations and average approximately 1,500 (Green) and 1,000 (Puyallup) winter-run steelhead spawners annually.

Table 20. Puget Sound steelhead salmon populations and a summary of available demographic data

Population	Life History	Historical Abundance (Percent Annual change[a]	Mean Number of Spawners[b]	Trends in escapement[c]	Median short-term growth rate (λ)[d]
Canyon	Summer Winter				
Skagit	Summer				

	Winter	7,700 (2.0)	5608.5	-0.002	0.997 (0.997-0.998)
Snohomish	Summer				
Snohomish	Winter	8,000 (3.1)	3230.1	-0.019	0.804 S
Dakota	Winter				
Nooksack	Winter	NA (-11.6)			
Samish	Winter		852.2	0.067**	0.988 (0.997-0.998)
Stillaguamish	Winter	NA (-6.3)	550.2	-0.065****	0.885 S (0.884-0.885)
Tolt	Summer		119.0	0.025	1.018 (1.017-1.018)
Green	Summer				
Green	Winter		1625.5	0.008	0.932 (0.932-0.933)
Cedar	Winter		36.8	-0.179**	0.808 S (0.804-0.811)
Lake Washington	Winter	NA (-17.5)	36.8	-0.180****	0.802 (0.800-0.803)
Nisqually	Winter	1,200 (-5.1)	392.4	-0.084****	0.918 (0.917-0.918)
Puyallup	Winter	2,000 (-5.2)	1001.0	-0.062****	0.882 (0.881-0.882)
Dewatto	Winter		24.7		1.020 (1.008-1.020)
Dosewallips	Winter		76.7		
Duckabush	Winter		17.7	0.017	
Hamma Hamma	Winter		51.9	0.291*	1.013
Quilcene	Winter		15.1	-0.006	0.988 S
Skokomish	Winter	NA (-3.5)	202.8	-0.075****	0.865 S
Tahuya	Winter	NA (-0.6)	117.0	0.009	0.983 (0.982-0.983)
Union	Winter		55.3	0.008	0.969 S
Elwha	Summer				
	Winter		210.0		0.966 (0.965-0.966)
Dungeness	Winter	NA (-5.5)	173.8	-0.076	0.924 (0.924-0.924)
Mc Donald	Winter			-0.031	0.732 S
Morse	Winter	200 (-12.3)		-0.006	0.945 (0.945-0.946)

[a]Values of historical abundance represent the total escapement for the subbasin. Data generally span the late 1970s to mid 1990s. All estimates are run reconstructions, except the Nooksack which comes from spawner surveys. Specific data years for each data set and other details are noted in Busby et al. 1996.

[b]Geometric mean estimates of escapement for Puget Sound steelhead are provided for the five year period from 2000-2004, and for hatchery plus natural spawners (NMFS 2005c).

[c]Estimates of temporal trends in escapement and total run size (transformed by natural log). Estimates are the slopes of the regressions of natural log (spawners or run size) on year. Estimates provided are for the entire available dataset and are based on natural fish (data years noted in NMFS 2005c). *, P<0.05; **, P<0.01; ***, P<0.001; ****, P<0.0001 (all other values are not significant (data from NMFS 2005c)).

[d]Estimates for each population were computed for the most recent 10 years of data (1995-2004). S – Denotes that the estimate is based on natural spawners alone. Values in parentheses represent the 95% Confidence Intervals of the estimate (data from NMFS 2005c).

Estimates of historical abundance for this DPS are largely based on catch data. The earliest catch records from commercial fisheries in the late 1880s indicate that the catch peaked at 163,796 steelhead in Puget Sound in 1895 (NMFS 2005c). Based on this catch data, NMFS (2005c) estimated that the peak run size for Puget Sound steelhead ranged between 300,000 and 550,000 fish. Given that most fish were harvested in terminal fisheries (nets set at the mouth of rivers) NMFS expects that this estimate is a fair estimate of the Puget Sound DPS as it is unlikely to include fish from neighboring rivers outside of the Puget Sound DPS. As early as 1898, Washington officials expressed concerns that the run had declined by half of its size in only three years (NMFS 2005c). Since 1925, Washington has managed steelhead as a game fish, and in 1932 the State prohibited the commercial catch, possession or sale of steelhead.

Run size for this DPS was calculated in the early 1980s at about 100,000 winter-run fish and 20,000 summer-run fish. It is not clear what portion were hatchery fish, but a combined estimate

with coastal steelhead suggested that roughly 70% of steelhead in ocean runs were of hatchery origin. Escapement of wild fish to spawning grounds would be much lower without the influx of hatchery fish (Busby et al 1996).

NMFS first status review for Puget Sound steelhead demonstrated that 80 % of the runs for which there was data had declining trends in abundance. Basinwide abundance estimates from Busby et al. (1996) are depicted in Table 20. Busby et al. (1996) noted that the largest decline, an 18% annual decline, occurred in the Lake Washington population. On the contrary, the largest increase in abundance occurred in the Skykomish River winter-run steelhead (the Skykomish River is a tributary to the Snohomish River) at a 7% annual increase. Estimates of spawner abundance in the Skagit and Snohomish rivers, the two largest steelhead producing basins in the DPS, were about 8,000 naturally spawning adult steelhead each (Table 20). These two basins exhibited modest overall upward trends at the time of the first status review. Recent data demonstrates significant declines in the natural escapement of steelhead throughout the DPS, especially in the southern Puget Sound populations. Significant positive trends have occurred in the Samish and the Hamma Hamma winter-run populations. The increasing trend in the Hamma Hamma River appears to be the result of a captive rearing program, rather than due to natural escapement. The predominant downward trends in escapement and run size of natural steelhead in the Puget Sound DPS, both over the long-term and short-term, is of concern particularly given that despite widespread reductions in direct harvest since the mid 1990s (NMFS 2005c).

Critical Habitat

NMFS has not designated critical habitat for Puget Sound steelhead.

Snake River Steelhead

Distribution and Description of the Listed Species

The Snake River Basin steelhead DPS includes all naturally spawned populations of steelhead in streams in the Snake River basins of southeast Washington, northeast Oregon and Idaho. Six artificial propagation programs are considered part of this DPS: The Tucannon River, Dworshak National Fish Hatchery, Lolo Creek, North Fork Clearwater, East Fork Salmon River, and the Little Sheep Creek/Imnaha river hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than what would be expected between closely related natural populations within the DPS.

Snake River Basin steelhead are distributed throughout the Snake River drainage basin, migrating a considerable distance from the ocean to use high-elevation tributaries (typically 1,000-2,000 m above sea live). Generally, classified as summer-run fish, Snake River steelhead enter the Columbia River from late June to October. After remaining in the river through the winter, Snake River steelhead spawn the following spring (March to May). Managers recognize two life history patterns within Snake River steelhead primarily based on ocean age and adult size upon return: A-run steelhead are typically smaller, have a shorter fresh water and ocean residence (generally 1 year in the ocean), and begin their up-river migration earlier in the year; whereas B-run steelhead are larger, spend more time in fresh water and the ocean (generally 2-years in ocean), and appear to start their upstream migration later in the year. Table 21 lists the

life-history type associated with each of the 24 demographically independent populations within this DPS.

Table 21. Snake River steelhead populations and a summary of available demographic data

Populations[a]	Life History	Historical Abundance (Percent Annual change[b]	Mean Number of Spawners (range)[c]	Percent Hatchery Contribution[d]	Long-term growth rate (λ)[e]
Tucannon River	A-run	400 (-18.3)	407 (257-628)	74	0.886, 0.733
Asotin Creek	A-run	200 (-19.7)	87 exp. redds (0-543)	Unknown	
Lower Clearwater	A-run				
South Fork Clearwater	B-run				
Lolo Creek	B-run				
Selway River	B-run				
Lochsa River	B-run				
North Fork Clearwater River					
Lower Grande Ronde	A-run	(-0.5)			
Joseph Creek	A-run		1,542 (1.077-2,385)	0	1.069
Wallowa River	A-run	(-3.0)			
Upper Grande Ronde	A-run		1.54 rpm (0.3-4.7)	23	0.967, 0.951
Little Salmon and lower Salmon tributaries	A-run				
South Fork Salmon River	B-run	(-8.0)			
Secesh River	B-run				
Chamberlain Creek	A-run				
Lower Middle Fork Salmon	B-run	(-25.8**)			
Upper Middle Fork Salmon	B-run				
Panther Creek	A-run				
North Fork Salmon	A-run				
Lemhi River	A-run				
Pahsimeroi River	A-run	1,400 (0.1)			
East Fork Salmon River	A-run	150*(-6.0)			
Upper Mainstem Salmon River	A-run				
Imnaha River	A-run	(81.2)	3.7 rpm (2.0-6.8)	20	1.042, 1.026
Hells Canyon tributaries	A-run				

[a] Demographically independent populations identified by ICBTRT 2003.

[b]Values of historical abundance represent total escapement as calculated in NMFS' first status review for the DPS. Values with a * are estimates of total run; no escapement estimate was available. Data generally span the late 1980s to mid 1990s. Estimates are calculated from different data types, and include data from spawner surveys, run reconstructions, or dam/weir counts. Specific data years for each data set and other details are noted in Busby et al. 1996. **=Middle Fork and tributaries.

[c]Geometric mean estimates of escapement represent total escapement (hatchery plus natural adult returns).

[d]Estimates of percentage of hatchery returns in Granite dam aggregate counts indicate that returns are predominantly composed of hatchery fish (about 85%). Values from Good et al. 2005.

[e]Multiple estimates for long-term growth (λ) presented for some populations represent two different assumptions on the contribution of hatchery fish to natural production. Where two or more values are presented, the first value reflects the assumption that hatchery fish do not contribute to

natural production, and the second value reflects the assumption that hatchery contribute to natural production at the same rate as natural-origin spawners. Data series upon which values are calculated, varies across basins. See Good et al. (2005) for details on the length and time of data series available by population.

Status and Trends

NMFS listed Snake River steelhead as threatened in 1997 (62 FR 43937), and reaffirmed their status as threatened on January 5, 2006 (71 FR 834). NMFS 1997 status review identified sharp declines in the returns of naturally produced steelhead, beginning in the mid-1980s. At the time nine of 13 trend indicators were in decline and the average abundance (geometric mean, 1992-1996) for the DPS was 75,000 adult steelhead (8,900 naturally produced). Of this, about 7,000 were A-run adults, and about 1,400 were B-run adults (Busby et al. 1996).

The paucity of information on adult spawning escapement for specific tributaries of the Snake River Basin DPS continues to make a quantitative assessment of viability difficult. Available data indicate that the overall long-term estimates of population trends have remained negative. Return estimates for the late 1990s to early 2000s are summarized in Table 21. Annual return estimates are limited to counts of the aggregate return over Lower Granite Dam, and spawner estimates for the Tucannon, Asotin, Grande Ronde, and Imnaha Rivers. The 2001 return over Lower Granite Dam was substantially higher relative to the low levels seen in the 1990s; the recent geometric 5-year mean abundance (Total escapement 106,175 with 14,768 natural returns) was approximately 28% of the interim recovery target level (52,000 natural spawners). The 10-year average for natural-origin steelhead passing Lower Granite Dam between 1996 and 2005 is 28,303 adults. Long-term trend estimates of the population growth rate (λ) across the available data set was 0.998 assuming that natural returns are produced only from natural-origin spawners, and 0.733 if both hatchery and wild spawners are contributing to production equally. Parr densities in natural production areas, which are another indicator of population status, have been substantially below estimated capacity for several decades. The Snake River supports approximately 63% of the total natural-origin production of steelhead in the Columbia River Basin. Genetic diversity is currently being depressed by the displacement of natural fish by hatchery fish (declining proportion of natural-origin spawners). Homogenization of hatchery populations occurs within basins and some populations exhibit high stray rates.

Critical Habitat

NMFS designated critical habitat for Snake River steelhead on September 2, 2005 (70 FR 52630). Designated critical habitat includes the following subbasins: Hells Canyon, Imnaha River, Lower Snake/Asotin, Upper Grand Ronde River, Wallowa River, Lower Grand Ronde, Lower Snake/Tucannon, Upper Salmon, Pahsimeroi, Middle Salmon-Panther, Lemhi, Upper Middle Fork Salmon, Lower Middle Fork Salmon, Middle Salmon, South Fork Salmon, Lower Salmon, Little Salmon, Upper and Lower Selway, Lochsa, Middle and South Fork Clearwater, and the Clearwater subbasins, and the Lower Snake/Columbia River corridor. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this DPS identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and

floodplain connectivity. The final rule (70 FR 52630) lists the watersheds that comprise the designated subbasins and any areas that are specifically excluded from the designation.

There are 289 watersheds within the range of Snake River steelhead. The total area of habitat designated as critical includes about 8,000 miles of stream habitat. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. Of the 289 fifth order streams reviewed in this DPS, 231 received a high conservation value rating, 44 received a medium rating, and 14 received a rating of low conservation value for the species. The lower Snake/Columbia rearing/migration corridor downstream of the spawning range has a high conservation value. Limiting factors identified for Snake River Basin steelhead include: hydrosystem mortality, reduced stream flow, altered channel morphology and floodplain, excessive sediment, degraded water quality, harvest impacts, and hatchery impacts.

South-Central California Coast Steelhead

Distribution and Description of the Listed Species

The South-Central California Coast steelhead DPS includes all naturally spawned populations of steelhead (and their progeny) in streams from the Pajaro River (inclusive) to, but not including the Santa Maria River, California. No artificially propagated steelhead populations that reside within the historical geographic range of this DPS are included in this designation. The two largest basins within this DPS are the inland basins of the Pajaro River and the Salinas River. Both of these watersheds drain intercoastal mountain ranges and have long alluvial lower stretches. Principle sub-basins in the Pajaro River that support steelhead include: Corralitos Creek, Pescadero Creek, Uvas Creek, and Pacheco Creek. Principle sub-basins in the Salinas River that support steelhead include the Arroyo Seco River, Gabilan Creek, Paso Robles Creek, Atascadero Creek and Santa Margarita Creek. Other important watersheds include the smaller coastal basins of the Carmel River, and St. Rosa and San Luis Obispos creeks.

Status and Trends

NMFS listed South-Central California Coast steelhead as threatened in 1997, and reaffirmed their status as threatened on January 5, 2006 (71 FR 834). Historical data on the South-Central California Coast steelhead DPS are sparse and no credible historic or recent estimates of total DPS size are available. Steelhead are present in a large portion of the historically occupied basins within this DPS (estimated 86-95 %) but observed and inferred abundance suggest many of this basins support a small fragment of their historic run size. Present population trends within individual watersheds continuing to support runs is generally unknown, but may vary widely between watersheds. No data are available to estimate the steelhead abundance or trends in the two largest watersheds in the DPS, the Pajaro and Salinas basins, although these basins are highly degraded and expected to support runs much reduced in size from historical levels.

Steelhead in the Carmel Basin have been monitored at San Clemente Dam since 1964, representing one of the longest data sets available for steelhead in this DPS. However, this data is also limited because a nine year gap exists in the series, a large portion of the run spawns

below the dam, and the older dam counts may be incomplete. Between NMFS' 1997 status review and 2005 status update, continuous data from San Clement dam suggests that the abundance of adult spawners in the Carmel River has increased. Carmel River time series data indicate that the population declined by about 22% per year between 1963 and 1993, and between 1991 and 1997 the population increased from one adult to 775 adults at San Clemente Dam. Good et al. (2005) deemed this increase too great to attribute simply to improved reproduction and survival of the local steelhead population. Other possibilities were considered, including that the substantial immigration or transplantation occurred, or that resident trout production increased as a result of improved environmental conditions within the basin. The five-year geometric mean calculated by Good et al. (2005) for the Carmel River population (1998-2002) was 611 steelhead (range 1-881).

Critical Habitat

NMFS designated critical habitat for South-Central California Coast steelhead on September 2, 2005 (70 FR 52488). Specific geographic areas designated include the following CALWATER hydrological units: Pajaro River, Carmel River, Santa Lucia, Salinas River, and Estero Bay. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this DPS identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The critical habitat designation (70 FR 52488) contains additional details on the sub-areas that are included as part of this designation, and the areas that were excluded from designation.

In total, South-Central California Coast steelhead occupy 30 watersheds (fresh water and estuarine). The total area of habitat designated as critical includes about 1,250 miles of stream habitat and about 3 square miles of estuarine habitat (e.g., Morro Bay). This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. In estuarine areas the lateral extent is defined by the extreme high water because extreme high tide areas encompass those areas typically inundated by water and regularly occupied by juvenile salmon during the spring and summer, when they are migrating in the nearshore zone and relying on cover and refuge qualities provided by these habitats, and while they are foraging. Of the 30 watersheds reviewed in NMFS' assessment of critical habitat for South-Central California Coast steelhead, six watersheds received a low rating of conservation value, 11 received a medium rating, and 13 received a high rating of conservation value for the species.

Southern California Steelhead

Distribution and Description of the Listed Species

The Southern California steelhead DPS includes all naturally spawned populations of steelhead in streams from the Santa Maria River, San Luis Obispo County, California (inclusive) to the United States-Mexico border. Artificially propagated steelhead that reside within the historical geographic range of this DPS are not included in the listing.

A comprehensive assessment of the distribution of steelhead within the Southern California DPS indicates that steelhead occur in most of the coastal basins (Boughton and Fish 2003 in Good et al. 2005). Major watersheds occupied by steelhead in this DPS include the Santa Maria, Santa Ynez, Ventura, Santa Clara rivers. Smaller watersheds that support steelhead include the Los Angeles, San Gabriel, San Luis Rey, and Sweetwater rivers, and San Juan and San Mateo creeks. Significant portions of several upper watersheds are contained with four national forests (Los Padres, Angeles, Cleveland, and San Bernardino National Forests), whereas coastal and inland valleys are dominated by urban development, with the Los Angeles basin being the most expansive and densest urban area in the DPS. Populations within the southernmost portion of the DPS (San Juan Creek, San Luis Rey River, and San Mateo Creek) are separated from the northernmost populations by about 80 miles.

Status and Trends

NMFS listed Southern California steelhead as endangered in 1997 (62 FR 43937), and reaffirmed their status as endangered on January 5, 2006 (71 FR 834). Historical and recent data is generally lacking for Southern California steelhead, making a general assessment of their status difficult. The historical run size estimate for the entire DPS was between 32,000-46,000 steelhead, but this estimate omits the Santa Maria system and basins south of Malibu Creek (Busby et al. 1996). Estimates for the Santa Ynez River Basin, probably the largest run historically, range from 13,000 to 30,000 spawners, although this number may underestimate the steelhead abundance in the basin prior to the construction of Juncal and Gibraltar dams (Busby et al. 1996; Good et al. 2005). No recent data are available for steelhead in the Santa Ynez basin, and most of the historical spawning habitat was blocked by Bradbury and Gibraltar dams. Steelhead and rainbow trout are known to occur in streams downstream of Bradbury Dam, but no estimates of abundance or trends are available. Similarly, Twitchell Dam in the Santa Maria River, and Casitas Dam on Coyote Creek and Matilija Dam on Matilija Creek block access to significant portions of historical spawning and rearing habitat, and alter the hydrology of the basins. A fish ladder and counting trap at the Vern Freeman Diversion Dam on the Santa Clara River is thought to be dysfunctional (Good et al. 2005). In general run sizes in river systems within the DPS are believed to range between less than five anadromous adults per year, to less than 100 anadromous adults per year. An estimated 26-52% of historically occupied basins are believed to still contain some steelhead, and about 30% are believed vacant, extirpated or nearly extirpated due to dewatering or barriers that block spawning habitat.

Critical Habitat

NMFS designated critical habitat for Southern California steelhead on September 2, 2005 (70 FR 52488). Specific geographic areas designated include the following CALWATER hydrological

units: Santa Maria River, Santa Ynez, South Coast, Ventura River, Santa Clara Calleguas, Santa Monica Bay, Callequas, and San Juan hydrological units. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this DPS identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The critical habitat designation (70 FR 52488) contains additional details on the sub-areas that are included as part of this designation, and the areas that were excluded from designation.

In total, Southern California steelhead occupy 32 watersheds (fresh water and estuarine). The total area of habitat designated as critical includes about 700 miles of stream habitat and about 22 square miles of estuarine habitat, mostly within Humboldt Bay. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. In estuarine areas the lateral extent is defined by the extreme high water because extreme high tide areas encompass those areas typically inundated by water and regularly occupied by juvenile salmon during the spring and summer, when they are migrating in the nearshore zone and relying on cover and refuge qualities provided by these habitats, and while they are foraging. Of the 32 watersheds reviewed in NMFS' assessment of critical habitat for Southern California steelhead, five watersheds received a low rating of conservation value, six received a medium rating, and 21 received a high rating of conservation value for the species.

Upper Columbia River Steelhead

Distribution and Description of the Listed Species

The Upper Columbia River steelhead DPS includes all naturally spawned populations of steelhead in streams in the Columbia River Basin upstream from the Yakima River, Washington, to the United States-Canada border. Six artificial propagation programs are part of this DPS: the Wenatchee River, Wells Hatchery (in the Methow and Okanogan rivers), Winthrop National Fish Hatchery, Omak Creek, and the Ringold steelhead hatchery programs. These artificially propagated populations are no more divergent relative to the local natural populations than would be expected between closely related populations within this DPS.

Rivers in this DPS primarily drain the east slope of the northern Cascade Mountains and include the Wenatchee, Entiat, Methow, and Okanogan River Basins. Some of these upper Columbia River subbasins, including the Okanogan River and the upper Columbia River proper, extend into British Columbia although steelhead do not occur in significant numbers in British Columbia and thus were not included in the DPS. Identified largely on the basis of spawning distributions, this DPS is composed of four putative populations defined by the Wenatchee, Entiat, Methow, and Okanogan rivers (Table 22). Historically (before the construction of Grand Coulee Dam blocked 50% of the river to Upper Columbia steelhead) major watershed that may

have supported steelhead within this DPS were the Sanpoil, Spokane, Colville, Kettle, Pend Oreille and Kootenai rivers (ICBTRT 2003).

All upper Columbia River steelhead are summer-run steelhead. Adults return in the late summer and early fall, with most migrating relatively quickly to their natal tributaries. A portion of the returning adult steelhead overwinters in mainstem reservoirs, passing over upper-mid-Columbia dams in April and May of the following year. Spawning occurs in the late spring of the year following river entry. Juvenile steelhead spend 1 to 7 years rearing in fresh water before migrating to sea. Smolt outmigrations are predominantly year class two and three (juveniles), although some of the oldest smolts are reported from this DPS (7 years). Most adult steelhead return to fresh water from sea after 1 or 2 years.

Status and Trends

NMFS originally listed Upper Columbia River steelhead as endangered in 1997 (62 FR 43937). On January 5, 2006, after reviewing the status of Upper Columbia River steelhead and noting an increase in abundance and more widespread spawning, NMFS reclassified the status of Upper Columbia River threatened (71 FR 834). In accordance with a United States District Court decision, NMFS reinstated the endangered status of Upper Columbia River steelhead in June 2007 (62 FR 43937). NMFS appealed the Court's decision, and on June 18, 2009, the District Court revised its ruling, effectively reinstating threatened status for Upper Columbia River steelhead (74 FR 42605). Thus, consistent with the court's rulings and the NMFS' listing determination of January 5, 2006, Upper Columbia River steelhead are listed as threatened under the ESA.

Since the 1940s, artificially propagated steelhead have seeded this DPS to supplement the numbers lost with the construction Grand Coulee Dam. Abundance estimates of returning naturally produced Upper Columbia River steelhead have been based on extrapolations from mainstem dam counts and associated sampling information (e.g., hatchery/wild fraction, age composition). Early estimates of steelhead in this DPS may be based on runs that were already depressed due to dams and steelhead fisheries. Nevertheless, these early dam counts are the best source of available data on the former size of the populations within this DPS. From 1933-1959 counts at Rock Island Dam averaged between 2,600 and 3,700 steelhead adults, which suggested the pre-fishery run size likely exceeded 5,000 adults destined for tributaries above Rock Island Dam (Chapman et al. 1994 in Busby et al. 1996). Using counts at Priest Rapids Dam (located below the production areas for this DPS) as an indicator of DPS size and trends suggests that the total number of spawners has increased since NMFS' 1996 status review. The 1992-1996 average annual total returns (hatchery plus natural) of steelhead spawners was 7,800, and the 1997-2001 average is 12,900 steelhead (hatchery plus natural). The natural component increased in these same periods from 1,040 to 2,200, respectively (Good et al. 2005).

Table 22. Upper Columbia River steelhead salmon populations and a summary of demographic data

Population	**Historical Abundance (Percent Annual change)[a]**	**Mean Number of Spawners (range)[b]**	Percent Hatchery Contribution[c]	Long-term growth rate (λ)[d]
Wenatchee River Entiat River	2,500 (2.6)	3,279** (1,899-8,036)	71 (65)	1.067, 0.733

Methow River Okanogan River	2,400* (-12.0)	3,714** (1,879-12,801)	91 (81)	1.086, 0.589

[a]Values of historical abundance represent total escapement as calculated in NMFS' first status review for the DPS. * = value represents a combined total escapement for the Methow and Okanogan rivers. Available data series: Wenatchee = 1962-1993, Methow and Okanogan = 1982-1993; calculations represent the geometric mean 1989-1993. Estimates are run reconstructions. Demographically independent populations identified by ICBTRT 2003.

[b]Geometric mean estimates of escapement represent total escapement (hatchery plus natural adult returns). ** Estimates of the mean number of spawners is a combined estimate for the Wenatchee and Entiat rivers, and the Methow and Okanogan rivers are also combined.

[c]Estimates of percentage of hatchery returns are from Good et al. 2005, and are based on extrapolations from mainstem dam counts and sampling. Parenthetical values are from Busby et al. 1996, and are provided for comparison.

[d]Multiple estimates for long-term growth (λ) are provided by Good et al. (2005) and represent two different assumptions on the contribution of hatchery fish to natural production. The first value reflects the assumption that hatchery fish do not contribute to natural production, and the second value reflects the assumption that hatchery fish contribute to natural production at the same rate as natural-origin spawners. Data series: 1976-2001.

While the total number of naturally produced fish in this DPS increased between status reviews, the proportion of naturally produced steelhead to hatchery-origin fish has declined. Total escapement increased in the combined estimate for the Wenatchee and Entiat rivers to a geometric mean of 3,279 spawners (900 natural spawners) over NMFS' previous estimate of 2,500 hatchery and natural steelhead spawners (1989 to 1993, natural component 800 steelhead). Estimates of the hatchery contribution to this population increased from 65% to 71% of total escapement (Table 22). A comparison of estimates for the Methow and Okanogan rivers during the same periods indicate that the total escapement increased from 2,400 to 3,714 while naturally produced steelhead declined from 450 to 358. Thus, the contribution of naturally produced steelhead declined from 19% to only 9% of total escapement between the 1993 and 2001 estimates (Good et al. 2005).

The assumptions of the role that hatchery fish play in the overall productivity and health of the DPS strongly influence estimates of population growth rates. Estimates based on the assumption that hatchery fish contribute to natural production at the same rate as natural-origin spawners consistently result in long-term population growth rates (expressed as λ) that are consistently below 1 (Table 22). Under the assumption that hatchery fish do not contribute to natural production, estimates of long term population growth rate suggest the population is growing. Determining the actual contribution of hatchery fish to natural production is important for understanding the true status of this DPS, particularly given that the proportion of naturally produced steelhead to hatchery-origin steelhead continues to decline. The extremely low replacement rate of naturally produced steelhead in this DPS is of concern, and the returns of natural steelhead remain well below recovery target levels.

The majority of the biological review team (54%) felt that this DPS warranted an "endangered" listing due to the growth rate and productivity, and uncertainty over the contribution of hatchery fish to natural production. NMFS, after convening a review of the artificial propagation programs of the six hatcheries in the DPS concluded that the programs collectively mitigate the immediacy of extinction risk in the DPS. Thus, NMFS listed the DPS as threatened rather than threatened (71 FR 834). NMFS concluded that the hatchery programs have increased total escapement and the distribution of spawning areas, and minimize the potential risks associated with artificial propagation. However, the abundance and productivity of naturally spawned steelhead remains a concern.

Critical Habitat

NMFS designated critical habitat for Upper Columbia River steelhead on September 2, 2005 (70 FR 52630). Designated critical habitat includes the following subbasins: Chief Joseph, Okanogan, Similkameen, Methow, Upper Columbia/Entiat, Wenatchee, Lower Crab, and the Upper Columbia/Priest Rapids subbasins, and the Columbia River corridor. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this DPS identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The final rule (70 FR 52630) lists the watersheds that comprise the designated subbasins and any areas that are specifically excluded from the designation.

There are 42 watersheds within the range of Upper Columbia River steelhead. The total area of habitat designated as critical includes about 1,250 miles of stream habitat. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. Of the 42 watersheds reviewed in NMFS' assessment of critical habitat for Upper Columbia River steelhead, three watersheds received a low rating of conservation value, eight received a medium rating, and 31 received a high rating of conservation value for the species. In addition, the Columbia River rearing/migration corridor downstream of the spawning range was rated as a high conservation value. Limiting factors identified for the Upper Columbia River steelhead include: mainstem Columbia River hydropower system mortality, reduced tributary stream flow, tributary riparian degradation and loss of in-river wood, altered tributary floodplain and channel morphology, and excessive fine sediment and degraded tributary water quality.

Upper Willamette River Steelhead

Distribution and Description of the Listed Species

The Upper Willamette River steelhead DPS includes all naturally spawned populations of winter-run steelhead in the Willamette River, Oregon, and its tributaries upstream from Willamette Falls to the Calapooia River (inclusive). No artificially propagated populations that reside within the historical geographic range of this DPS are included in this listing. Hatchery summer-run steelhead occur in the Willamette Basin but are an out-of-basin population that is not included in this DPS.

The native (late) winter-run steelhead, with spring Chinook salmon, are the only two populations of salmon believed to historically occur above Willametter Falls (RKm 77). The construction of a fish ladder at the falls in the late 1880s, allowed for the passage of summer steelhead from Skamania Creek and winter-run steelhead from Big Creek (i.e., Gnat Creek). The two groups of winter-run steelhead exhibit different return times. The later run exhibits the historical phenotype adapted to passing the seasonal barrier that existed at Willamette Falls prior to construction of the fish ladder. The early run of winter-run steelhead are considered non-native,

and were derived from Columbia River steelhead outside the Willamette River (Good et al. 2005). While the release of these hatchery winter-run fish was recently discontinued, some fish from earlier releases now reproduce naturally within the upper Willamette River Basin. Nonnative summer-run hatchery steelhead continue to be released within the upper basin (Good et al. 2005).

Native steelhead in the Upper Willamette are a late-migrating winter group that enters fresh water in January and February (Howell et al. 1985). They do not ascend to their spawning areas until late March or April (Dimick and Merryfield 1945) and spawning occurs from April to June 1. The smolt migration past Willamette Falls also begins in early April and proceeds into early June, peaking in early- to mid-May (Howell et al. 1985). Smolts generally migrate through the Columbia via Multnomah Channel rather than the mouth of the Willamette River. Most spend 2 years in the ocean before re-entering natal rivers to spawn (Busby et al. 1996). Steelhead in the Upper Willamette River DPS generally spawn once or twice, although some may spawn three times. Repeat spawners are predominantly female and generally account for less than 10% of the total run size (Busby et al. 1996).

Status and Trends

NMFS originally listed Upper Willamette River steelhead as threatened in 1999 (64 FR 14517), and reaffirmed their status as threatened on January 5, 2006 (71 FR 834). The Upper Willamette steelhead DPS consists of four demographically independent populations, each of which remains extant although depressed from historical levels (Table 23). Available data for this DPS comes from a combination of dam counts, redd count index surveys, and hatchery trap counts. Estimates of abundance from NMFS 1996 status review on this DPS, demonstrate a mix of trends with the largest populations, Mollala and North Santiam rivers, declining over the period of analysis. The 2005 review of the status of the Upper Willamette steelhead DPS indicated that each population showed a declining trend over the data series that extended to 2000 and 2001, while one population, the Calapooia River, increased over the short-term (1990-2000/1; Good et al. 2005).

More recently, data reported in McElhany et al. (2007) indicate that currently the two largest populations within the DPS are the Santiam River populations. Mean spawner abundance in both the North Santiam River and the South Santiam River is about 2,100 native winter-run steelhead. Long-term growth is negative for three of the populations within the DPS, with Calapooia River demonstrating a lambda of >1 indicating long-term growth in this population (McElhany et al. 2007). Spatial structure for the North and South Santiam populations has been substantially reduced by the loss of access to the upper North Santiam basin and the Quartzville Creek watershed in the South Santiam subbasin due dam construction lacking passage facilities (McElhany et al. 2007). Additionally, habitat in the Molalla subbasin has been reduced significantly by habitat degradation and in the Calapooia by habitat degradation as well as passage barriers. Finally, the diversity of some populations has been eroded by small population size, the loss of access to historical habitat, legacy effects of past winter-run hatchery releases, and the ongoing release of summer steelhead (McElhany et al. 2007).

Table 23. Upper Willamette river steelhead populations and a summary of available demographic data

Population[a]	Historical Abundance (Percent Annual change)[b]	Mean Number of Spawners (range)[c]	Long-term trend in redds per mile (95% CI)[d]	λ[e]
Mollala River	2,300 (-4.9)	914 (655-1275)	0.947 (0.918, 0.977)	0.988 (0.79, 1.235)
North Santiam River	2,000 (-4.0)	2,109 (1,485-2,994)	0.941 (0.906, 0.977)	0.983 (0.786, 1.231)
South Santiam River	550 (2.4)	2,149 (1,618-2,853)	0.936 (0.904, 0.907)	0.976 (0.855, 0.998)
Calapooia River	700	339 (206-560)	0.968 (0.933, 1.003)	1.023 (0.743, 1.409)

[a]Demographically independent populations identified by Myers et al. 2002 cited in Good et al. 2005.
[b]Values of historical abundance represent total escapement, with the exception of the Calapooia River which represents total run, as calculated in NMFS' first status review for the DPS. Data were collected using different methods (Angler Catch vs. Dam Counts) and represent data series ending in the early 1990s or earlier. Details on data types and the data series used for these calcuations are available in Busby et al. (1996).
[c]The geometric mean natural orgin spawner abudance calculated for the data series 1990-2005, and reported in McElhany et al. 2007.
[d]Long term trends are estimated using the entire data set, which is 1980 to 2000 for the Mollala River, and 1980-2001 for the remaining populations. Trends calculated by Good et al. 2005.
[e]Long-term growth rate (λ) reported by McElhany et al. 2007, and relects spawner escapement for the total available data series (1980-2005 – Molalla, Calappia & N Santiam Rivers; 1968-2005-S.Santiam River).

Critical Habitat

NMFS designated critical habitat for Upper Willamette River steelhead on September 2, 2005 (70 FR 52488). Designated critical habitat includes the following subbasins: Upper Willamette, North Santiam, South Santiam, Middle Willamette, Molalla/Pudding, Yamhill, Tualatin, and the Lower Willamette subbasins, and the lower Willamette/Columbia River corridor. These areas are important for the species' overall conservation by protecting quality growth, reproduction, and feeding. The critical habitat designation for this DPS identifies primary constituent elements that include sites necessary to support one or more steelhead life stages. Specific sites include freshwater spawning sites, freshwater rearing sites, freshwater migration corridors, nearshore marine habitat and estuarine areas. The physical or biological features that characterize these sites include water quality and quantity, natural cover, forage, adequate passage conditions, and floodplain connectivity. The final rule (70 FR 52630) lists the watersheds that comprise the designated subbasins and any areas that are specifically excluded from the designation.

There are 38 watersheds within the range of Upper Willamette River steelhead. The total area of habitat designated as critical includes about 1,250 miles of stream habitat. This designation includes the stream channels within the designated stream reaches, and includes a lateral extent as defined by the ordinary high water line. In areas where the ordinary high-water line is not defined the lateral extent is defined as the bankfull elevation. Of the 38 watersheds reviewed in NMFS' assessment of critical habitat for Upper Willamette River steelhead, 17 watersheds received a low rating of conservation value, six received a medium rating, and 15 received a high rating of conservation value for the species. In addition, the lower Willamette/Columbia River rearing/migration corridor downstream of the spawning range was rated as a high conservation value.

Marine Mammals

Cook Inlet Beluga Whale

Distribution and Description of the Listed Species

Beluga whales are widely distributed in Arctic and subarctic waters, and in Alaska five putative populations exist (Beaufort Sea, eastern Chukchi Sea, Bristol Bay, eastern Bering Sea, and Cook Inlet). Cook Inlet beluga whales are the only population that is listed under the ESA. Mitochondrial and nuclear DNA distinguish Alaskan beluga whales from those that occur in Hudson Strait, Baffin Bay and the St. Lawrence River, with the Cook Inlet population demonstrating the strong evidence of genetic isolation from the other Alaskan populations and other populations demonstrating weak to moderate evidence of genetic isolation (O'Corry-Crowe et al. 2007 in Hobbs et al. 2008; O'Corry-Crowe 2008; O'Corry-Crowe et al. 2008). Analysis of mtDN variation indicates strong philopatry to summering areas and low rates of dispersal between Cook Inlet beluga whales and other populations. The phylogenetic structure of the Cook Inlet beluga whale population suggests isolation of the population over evolutionary time scales.

Beluga whales are observed year-round in Cook Inlet although less is known about their winter movements than summer movements (see Hobbs et al. 2008 for a review). Data from satellite tagging studies suggest that movements of Cook Inlet beluga whales during summer months are short and largely focused around river estuaries and inlets (e.g., Chickaloon Bay, Turnagain Arm, Susitna River, and Knik Arm in the upper inlet and in many cases the animals exhibited very little movement for weeks during the summer (Hobbs et al. 2005). Dense groupings in these areas during June and July are the focus of NMFS aerial surveys, but numbers drop substantially in the upper inlet by November (Hobbs et al. 2005). Outside of Cook Inlet in the Gulf of Alaska beluga whale sightings are extremely rare (Laidre et al. 2000). Hobbs et al. (2005) found that tagged beluga whales moved to farther offshore during winter months, but remained within Cook Inlet. However travel distance appeared to increase during winter months, and exhibited more widely dispersed patterns both within and among individuals (Hobbs et al. 2005). Distribution during all months is likely influenced by prey distribution, where salmon and eulachon are concentrated in river mouths during summer months and other prey like sand lance are found in mid and bottom waters of the inlet during winter months, albeit in more dispersed patterns leading to the wider dispersal of the whales.

Based on past studies of the summer distribution of beluga whales in Cook Inlet, it appears that the population has experienced a contraction in its overall distribution (Speckman and Piatt 2000; Hobbs et al. 2008). Aerial surveys in the 1970s indicated that at least 10% of the population used areas south of Kenai River and Kalgin Island (mid- to lower Cook Inlet) during summer months, whereas more recent surveys (1993-2007) observed more than 90% of the beluga whales in upper Cook Inlet in shallow waters. According to Hobbs et al. (2008) 90% of the whales in the 1970s were observed within 70 nmi of the western tip of Anchorage (Point Woronzof), whereas more recently (1998-2007) 90% were detected within 20 nmi. Although the precise reason for the range contraction is not known, the shrinking summer distribution likely reflects the reduction in the population size over the same intervals and the beluga whale's preference for dense

aggregations of preferred prey species.

Analyses of beluga whale stomach contents indicate that beluga whales are opportunistic feeders, but specific species form the bulk of the prey when they are seasonally abundant (Hobbs et al. 2008). For instance, eulachon (*Thaleichthys pacificus*) also known as smelt or candlefish, are a small anadromous fish return that their natal rivers in spring for spawning. In the Susitna River, the eulachon spawning migration has a bimodal peak, with fish entering the estuary in May and again in June, and represents a significant biomass of prey, with estimates of several thousand fish entering the river in the first wave and several million entering the river in June (Calkins 1989). The common name candlefish is derived from the fact the fish is so high in fat content during spawning, with up to 15% of total body weight as fat, that when caught and dried and strung on a wick the fish could be burned like a candle. This high fat content confers a significant source of energy for beluga whales, including calving whales that occur in the upper inlet during the same period (Calkins 1989). The stomach contents of one beluga whale harvested in upper Cook Inlet in 1998 near the Susitna River contained only eulachon. Based on stomach sample analyses from 2002-2007 fish compose the majority of the prey species, with gadids (cod and walleye pollock) and salmonids composing the majority of the fish eaten (Hobbs et al. 2008). Anadromous salmonids begin concentrating at the river mouths and intertidal flats in upper Cook Inlet in late spring and early summer as emigrating smolts and immigrating adult spawners. Like eulachon, salmon are another source of lipid-rich prey for the beluga whale and represent the greatest percent frequency of occurrence of the prey species found in Cook Inlet beluga whale stomachs (Hobbs et al. 2008). As salmonid numbers dwindle in the fall and winter, beluga whales return to feed on nearshore or deeper water species including cod, sculpin, flounder, sole, shrimp, crab and others (Hobbs et al. 2008).

Cook Inlet experiences some of the most extreme tidal fluctuations in the world (see NMFS 2008 for a discussion), and beluga whales in the inlet have adapted to these tidal cycles and seemingly take advantage of them, although the precise causal reasons are not well known. Presumably, the feeding opportunities these tidal cycles proffer the beluga whale are a contributing factor. Aerial surveys and predictive models of habitat us indicate that beluga whale movement patterns are closely correlated to tidal patterns, flow accumulation and mudflats, with a preference for medium and high flow inlets of larger river basins (Ezer et al 2008; Goetz et al. 2007). More information, however, is needed to link these habitat attributes to causative reasons for this preference. Besides feeding, studies have suggested this preference for tidal mudflats may also be attributed to calving and breeding, molting, or shelter from predators like killer whales (Calkins 1989; Huntington 2000; Moore et al. 2000; Sheldon et al. 2003).

Beluga whale calving is not well documented but the presence of cow/calf pairs in large river estuaries in the upper inlet, and accounts of Alaskan Natives, suggests that calving and nursery areas are located near the mouths of the Beluga and Susitna Rivers, Chickaloon Bay and Turnagain Arm (NMFS 2008). According to surveys by LGL (Funk et al. 2005 as cited in NMFS 2008) cow/calf pairs also make extensive use of Knik Arm in the summer and fall. Neonates are often not seen until June in Cook Inlet (Burns and Seaman 1986a). NMFS (2008) and others have suggested that the shallow waters of Cook Inlet may be important for reproduction and calving, as the shallower water is warmer which may confer an important thermal advantage for calf survival as they have relatively limited fat deposits at birth. Breeding is presumed to occur

shortly after calving, in the late summer with a female's first parturition at age 5 or 6 after about 14-15 months of gestation (Calkins 1989). Lactation lasts about two years, with breeding occurring during lactation (Calkins 1989).

Calculation of beluga whale age is based on growth layers in teeth. Some debate exists as to whether a beluga whale tooth contains two growth layer groups (GLG) per year or one growth layer per year (See Hobbs et al. 2008 for discussion). Due to this ambiguity, Hobbs et al. (2008) summarized life history parameters according to tooth growth layers rather than years (Table 24 from Hobbs et al. 2008).

Table 24. Review of Female beluga life history parameters found in the published literature (from Hobbs et al. 2008; GLG=growth layer groups)

Parameter	Data	Sources
Age at sexual maturity	8-15 GLG	Brodie 1971; Sergeant 1973; Ognetov 1981; Seaman and Burns 1981; Braham 1984; Burns and Seaman 1986
	0% at 8-9 GLGs 33% at 10-11 GLGs 94% at 12-13 GLGs	Burns and Seaman 1986[a]
	9.1 +/- 2.8 GLGs	Robeck et al. 2005
Age at color change (gray to white)	12 GLGs	Brodie 1971
	22 GLGs	Sergeant 1973
Age at 1st conception	54% at 8-9 GLGs 41% at 10-11 GLGs 94% at 12-13 GLGs	Burns and Seaman 1986[b]
Age at senescence	42-43 GLGs	Brodie 1971
Pregnancy and birth rates	Small fetuses: 0.055 at 0-11 GLGs 0.414 at 12-21 GLGs 0.363 at 22-45 GLGs 0.267 at 46-57 GLGs 0.190 at 58-77 GLGs With full-term fetuses/neonates: 0.000 at 0-11 GLGs 0.326 at 12-21 GLGs 0.333 at 22-45 GLGs 0.278 at 46-51 GLGs 0.182 at 52-57 GLGs 0.125 at 58-77 GLGs	Burns and Seaman 1986
Lifespan	>60 GLGs (Oldest female estimated at 70+ GLGs)	Burns and Seaman 1986
	64-65 GLGs	Khuzin 1961 (cited in Ohsumi 1979)
	60-61 GLGs	Brodie 1971
	50-51 GLGs	Sergeant 1973
Adult annual survival	0.96-0.97	Béland et al. 1992
	0.955 (based on pilot whale data)	Brodie et al. 1981
	0.935	Lesage and Kingsley 1998
	0.91-0.92	Allen and Smith 1978
	0.906 (includes natural & human-caused mortality)	Burns and Seaman 1986

Parameter	Data	Sources
	0.84-0.905 (based on body length and lifespan	Ohsumi 1979
Immature annual survival	0.905 (for neonates in first half year)	Sergeant 1973
Reproductive rate	0.010-012	Perrin 1982[c]
	0.11[d]	Burns and Seaman 1986
	0.13[d]	Sergeant 1973
	0.09[d]	Brodie 1971
	0.09-0.12 [d]	Braham 1984
	0.09-0.14[e]	Braham 1984
	0.12 [e]	Sergeant 1973; Ray et al. 1984
	0.08-0.14[e]	Davis and Evans 1982
	0.06-0.10[e]	Davis and Finley 1979
	0.08-0.10[e]	Brodie et al. 1981
	0.08 (unknown)	Breton-Provencher 1981
Calving Interval	<3 years	Burns and Seaman 1986[f]
	2 yrs and 3 years	Sergeant 1973[g]

[a]Alaska sample (52 whales). Sampling occring in June when most Alaskan beluga whales are born. Hobbs et al. 2008 note that it is possible that non-pregnant 8-9 GLGs beluga whales would have conceived before their 10-11 GLG birth date.
[b]Alaska sample of 22 whales.
[c]Based on literature review and adopted by the International Whaling Commission
[d]Based on annual calf production rates
[e]Based on calf counts
[f]For some female beluga whales. This was a tentative conclusion based on high conception rates noted in some females between the ages of 12-13 GLGs and 44-45 GLGs.
[g]Two-year intervals were for 25% of mature female belugas in eastern Canada (7 of 29 sampled); presumed after noting pregnancies occurred during lactation. Three-year intervals were for 75% of mature females in eastern Canada. Sergeant (1973) concluded that the "overlap of pregnancy and previous lactation is infrequent so that calving occurs about once in three years."

Status

On October 22, 2008, NMFS listed the Cook Inlet beluga whale as endangered (73 FR 62919). Historic numbers of beluga whales in Cook Inlet are unknown. Dedicated surveys began in earnest in the 1990s when NMFS began conducting aerial surveys for beluga whales in Cook Inlet. Prior to then, survey efforts were inconsistent, part of larger sea bird and marine mammal surveys, made by vessel, or estimated following interviews with fishermen (Klinkhart 1966). In many cases the survey methodology or confidence intervals were not described. For instance, Klinkhart (1966) conducted aerial surveys in 1964 and 1965, where he describes having estimated the populations at 300-400 whales, but the methodology was not described nor did he report the variance around these estimates. Other estimates were incomplete due to the small area the survey focused upon (e.g. river mouth estimates; e.g., Hazard 1988). The most comprehensive survey effort prior to the 1990s occurred in 1979 and included transects from Anchorage to Homer, and covered the upper, middle and lower portions of Cook Inlet. From this effort, and using a correction factor of 2.7 to account for submerged whales Calkins (1989 cited in NMFS 2008) estimated the 1979 abundance at about 1,293 whales.

In 1993, NMFS began systematic aerial surveys of beluga whales in Cook Inlet and like the 1979 survey cover the upper, middle and lower portions of Cook Inlet. The survey protocol involves using paired observers who make independent counts at the same time a video of the whale grouping is recorded. Each group size estimate is corrected for subsurface and missed animals, or if video counts are not available then additional corrections are made (Allen and Angliss 2010).

Table 25. Estimated abundance of Cook Inlet beluga whales with coefficient of variation and 95% confidence intervals.

Year	Estimate[1]	CV	95% CI[2]	
			Lower	Upper
1979	1,293			
1994	653	0.43	291	1464
1995	491	0.44	215	1120
1996	594	0.28	347	1018
1997	440	0.14	335	578
1998	347	0.29	199	606
1999	367	0.14	279	482
2000	435	0.23	279	679
2001	386	0.087	326	458
2002	313	0.12	248	396
2003	357	0.107	290	440
2004	366	0.2	290	440
2005	278	0.18	196	394
2006	302	0.16	221	412
2007	375	0.14	285	492
2008	375	0.23	240	585
2009[2]	321	0.18	226	456

[1]All estimates, except 1979 estimate, reported in Hobbs & Shelden 2008. The 1979 estimate is from Calkins 1989 as cited in NMFS 2008.
[2]Data from R. Hobbs, pers. comm., to A. Garrett, Apr. 2010.

Between 1979 and 1994, according to above noted population estimates, Cook Inlet beluga whales declined by 50%, with another 50% decline observed between 1994 and 1998. Using a growth fitted model Hobbs et al. 2008 observed an average annual rate of decline of -2.91% (SE = 0.010) from 1994 to 2008, and a -15.1% (SE=0.047) between 1994 and 1998. A comparison with the 1999-2008 data suggests the rate of decline at -1.45% (SE=0.014) per year (Hobbs et al. 2008). Given that harvest was curtailed significantly between 1999 and 2008, NMFS had expected the population would begin to recover at a rate of 2-6% per year. However, abundance estimates demonstrate that this is not the case (Hobbs & Shelden 2008).

In conducting its status review, NMFS ran a number of population viability analyses (PVAs) to estimate the time to extinction for Cook Inlet beluga whales. The models were sensitive to a variety of parameters such as killer whale predation, allee effects, and unusual mortality events. The best approximation of the current population incorporated killer whale predation at only one beluga whale per year, and allowed for an unusual mortality event occurring on average every 20 years. According to this model, there is an 80% probability that the population is declining, a 26% probability that the population will be extinct in 100 years (by 2108) and a 70% probability that the population will be extinct within 300 years (by 2308).

Social Behavior

Beluga whales are highly social animals. The highly developed vocal repertoire of the beluga whale may play a substantial role in the formation of groups and communication among individuals. According to O'Corry-Crowe (2002), the beluga whale has long been called the "sea canary" by mariners because of the wide variety of sounds they make and can be heard reverberating through ship hulls. About 50 types of calls are recognized, typically ranging from

0.1 to 12 kHz, and include groans, whistles, buzzes, trills, roars and others, allow them to communicate over long distance and through icy arctic waters.

Belugas are typically observed in groups, which typically range from 2-25 individuals although they have been observed in groups of hundreds and even up to a thousand animals. There may be some seasonal segregation of sexes, as at times males form distinct groups and females are often tightly associated with one or more generations, at other times the groupings are a mixed social unit (O'Corry-Crowe 2002). Beluga whales also have a wide variety of facial expressions, as they can alter the shape of the mouth and melon. The lateral flexibility allows them to exploit shallow habitats and likely enhances visual signaling between animals (like vocalization, visual acuity is highly developed).

Threats

Natural Threats. Natural threats to Cook Inlet beluga whales include stranding, predation, parasitism and disease, environmental change, and genetic risks associated with small populations (e.g., inbreeding, loss of genetic variability). Beluga whales may strand accidentally as they occupy shallow water areas or escape predators, or as a result of diseases, illness or injury (NMFS 2008). Given the extreme tidal fluctuations in Cook Inlet, beluga whale strandings are not uncommon. According to NMFS (2008) killer whales have been observed in Cook Inlet concurrent to beluga whale strandings, and evidence of killer whale attacks is apparent in some beluga whale strandings (see Table 26).

According to NMFS (2008) over 700 beluga whales have stranded in Cook Inlet since 1988, many of which occurred in Turnagain Arm and often coincided with extreme tidal fluctuations (see Table 26 for a complete record). Where stranding occurs from extreme tidal fluctuations, and animals are out of the water for extended periods the risk of mortality increases from cardiovascular collapse. Ten hours may be the upper limit for out of the water for beluga whales before serious injury or death occurs (NMFS 2008). Strandings may represent a significant threat to the conservation and recovery of the Cook Inlet beluga whale population.

Table 26. Cook Inlet beluga whale stranding records from 1988 through September 2008 (from Hobbs and Shelden 2008, and NMFS 2008).

Year	Month	Location	No. w/evidence of Killer whale predation	Number of Whales	Known Associated Deaths	Total Mortalities* (live + dead stranded)
1988	October	Turnagain Arm		27	0	0
1989	-	-		-	-	4
1988	-	-		-	-	2
1991	August	Turnagain Arm	1	70-80	0	2
1992	October	Kenai River	2	2	2	5
1993	July	Turnagain Arm	1	10+	0	3
1994	June	Susitna River		186	0	7
1995	-	-		-	-	2
1996	June	Susitna River		63	0	12
	August	Turnagain Arm		60	4	
	September	Turnagain Arm		20-30	1	
	September	Knik Arm		1	0	

Year	Month	Location	No. w/evidence of Killer whale predation	Number of Whales	Known Associated Deaths	Total Mortalities* (live + dead stranded)
	October	Turnagain Arm		10-20	0	
1997	-	-		-	-	3
1998	May	Turnagain Arm		30	0	10
	September	Turnagain Arm		5	0	
1999	August	Turnagain Arm	5	58	5	12
	September	Turnagain Arm		12-13	0	
2000	August	Turnagain Arm	2	8	0	13
	September	Turnagain Arm		15-20	0	
	October	Turnagain Arm		1-2	0	
2001	-	-		-	-	10
2002	-	-		-	-	13
2003	April	Turnagain Arm	1	2	0	20
	August	Turnagain Arm		46+	5	
	September	Turnagain Arm		58	0	
	October	Turnagain Arm		9		
2004	-	-		-	-	13
2005	August	Knik Arm		6	1	6
2006	September	Knik Arm		12	0	8
2007	-	-		-	-	15
2008	August	Knik Arm	1	28-30	2	11

*Known subsistence harvested beluga whales are not included in these numbers.

Gaydos et al. (2004) identified 16 infectious agents in free-ranging and captive southern resident killer whales, but concluded that none of these pathogens were known to have high potential to cause epizootics. Many of these same infectious agents could pose a problem for Cook Inlet beluga whales. At this time little information is available to date to suggest bacterial or viral agents are actively contributing to the decline in the Cook Inlet population. About 80% of Cook Inlet beluga whales examined, however, have evidence of the parasite *Crassicauda giliakiana* in the kidneys, although it is presently unclear whether the parasite is affecting the status of the population (NMFS 2008). Necropsies have also revealed infestations of the common nematode anasakids, or whaleworm in the stomach of adult Cook Inlet beluga whales. While the parasite tends to favor the stomach and can cause gastritis or ulcerations, the infestations in beluga whales has not been considered severe enough to have caused clinical responses (NMFS 2008). Liver trematodes have also been identified in at least one beluga whale. At present, NMFS has no information to suggest that parasites are having a measureable impact on the survival and health of the Cook Inlet whale population (NMFS 2008).

Anthropogenic Threats. Human induced threats to Cook Inlet beluga whales include subsistence harvest, poaching and illegal harvest, incidental take during commercial fishing and reduction of prey through fishing harvests, pollution, oil and gas development, urban development, vessel traffic including from tourism and whale watching, noise, as well as research activities directed at beluga whales. Subsistence harvest of beluga whales by Alaskan natives has occurred since prehistoric times, but the effect of recent harvests has been significant. Although harvest levels have only recently been recorded, population declines in the mid 1990s are largely attributed to subsistence harvests during that period. In part, improved efficiencies of harvest techniques has allowed natives and others to increase catch of beluga whales. During the early 1900s there was

a short-lived commercial whaling company, The Beluga Whaling Company, which operated at the Beluga River in upper Cook Inlet. The Company during its 5 years of operation harvest 151 belugas from 1917-1921 (Mahoney and Shelden 2000). Another commercial hunt of beluga whales in 1930s is recollected by residents, but no record of the hunt exists in Alaska fishery and fur seal documents (Bower, 1931-41 as cited in Mahoney and Shelden 2000). In 1999 and 2000 there was a voluntary moratorium on subsistence harvest, and since substance harvest have been conducted under co-management agreements. Since 2000, no more than 2 beluga whales have been taken in subsistence harvests in any one year (NMFS 2008).

Commercial fisheries likely have varying levels of interactions with Cook Inlet beluga whales, according to the timing, gear types, targeted species, and location of activities (NMFS 2008). Reports of fatal interactions with commercial fisheries have been noted in the literature (Murray and Fay 1979 cited in Hobbs et al. 2008; Burns and Seaman 1986). Direct interactions with fishing vessels and nets are considered unusual, based on observer data, and unlikely to inhibit the recovery of Cook Inlet beluga whales. The reduction of prey species, however, is of more concern for the species. In 2000 NMFS recommended the closing of the eulachon fishery due to a lack of understanding of how this fishery interfered with beluga whale feeding, but in 2005 this fishery was reopened with a harvest limited at 100 tons of eulachon. Currently, it is unclear if fishery harvest of beluga whale prey species is having a significant impact on the population. Impacts from recreational fisheries, which are very popular in the region, likely include the reduction of fish prey species particularly salmonid species, and also the harassment from noise and risk of injury from vessel strikes from the operation of small watercraft in the estuarine/river mouths may (NMFS 2008).

Contaminants in beluga whales are of concern, both for whale health and the health of subsistence users. Tissue samples are regularly collected from subsistence harvested and stranded beluga whales and archived. Tissues and organs commonly collected include blubber, liver and kidneys, as well as muscle, heart, bone, skin and brain. Blubber is the most commonly collected; due to the lipid content it typically contains the most lipophilic substances (Becker 2000). The kidney and liver are used to analyze heavy metal compounds. Relatively high levels of PCBs, chlorinated pesticides and mercury are evident in beluga whales, although the more contaminated belugas are from the St. Lawrence River, Canada (Becker 2000). Concentrations of chlorinated hydrocarbons in Cook Inlet beluga whales range from 0.1-2.4 µg/g, w.w. DDT, 0.6-4.7 µg/g, w.w. PCB, 0.1-0.6 µg/g, w.w. chlordane, <0.1-4.3 µg/g, w.w. toxaphene. The higher levels of these compounds found in beluga whales in comparison to bowhead whales is probably reflective of the trophic levels of the species, as bowhead are baleen whales that feed on copepods while belugas are primarily fish eaters (Becker 2000). Studies indicate that PCBs and chlorinated pesticide concentrations are higher in male beluga whales than females, reflecting the transference of body loads to the offspring that occurs during gestation and lactation (Becker et al. 2000). Other contaminant detected in Cook Inlet beluga whales include heavy metals such as cadmium, mercury, selenium, copper, and zinc to name a few. Comparative studies suggest that Cook Inlet beluga whales generally carry less body burdens than beluga whales from other areas. An exception is copper, which is two to three times higher in Cook Inlet beluga whales than beluga whales from the eastern Beaufort Sea and the eastern Chukchi Sea, but is similar concentrations found in Hudson Bay beluga whales (Becker et al. 2000). To date, the health implications of high copper levels in Cook Inlet beluga whales is not clear.

Critical Habitat

NMFS proposed critical habitat for the Cook Inlet beluga whale on December 2, 2009 (74FR 63080). Two areas specific areas are proposed comprising 7,809 square kilometers of marine habitat. Area 1 encompasses 1,918 square kilometers (741 sq. mi.) of Cook Inlet northeast of a line from the mouth of Threemile Creek (61º 08.5'N., 151 º 04.4' W.) to Point Possession (61º 02.1'N., 150 º 24.3' W.). This area is bounded by Anchorage, the Matansuska-Susitna Borough, and the Kenai Peninsula Borough. This area contains shallow tidal flats, river mouths or estuarine areas and is important as foraging and calving habitats. Area 1 also has the highest concentrations of beluga whales in the spring through fall as well as the greatest potential for adverse impact from anthropogenic threats. Area 1 contains many rivers with large eulachon and salmon runs, including 2 rivers in Turnagain Arm (Twenty-mile River and Placer River) which are visited by beluga whales in the early spring. Use declines in the summer and increases again in August through the fall, coinciding with coho salmon returns. Also included in Area 1 is Knik Arm and the Susitna delta. Area 2 consists of 5,891 square kilometers (2,275 sq. mi.) of Cook Inlet, located south of Area 1, north of a line at 60º 25.0'N., and includes nearshore areas south of 60º 25.0'N. along the west side of the Inlet and Kachemak Bay on the east side of the lower inlet. Area 2 is used by Cook Inlet beluga whales in a dispersed fashion for fall and winter feeding and as transit waters. Area 2 includes near and offshore areas of the mid and upper Inlet, and nearshore areas of the lower Inlet. Area 2 includes Tuxedni, Chinitna, and Kamishak Bays on the west coast and a portion of Kachemak Bay of the east coast. Dive studies indicate that beluga whales in this area dive to deeper depths and are at the surface less frequently than they are when they inhabit Area 1. The primary constituent elements essential to the conservation of Cook Inlet beluga whales are: (1) intertidal and subtidal waters of Cook Inlet with depths <30 ft. (MLLW) and within 5 miles of high and medium flow accumulation anadromous fish streams; (2) primary prey species consisting of four species of Pacific salmon (Chinook, coho, sockeye, and chum salmon), Pacific eulachon, Pacific cod, walleye pollock, saffron cod, and yellowfin sole; (3) the absence of toxins or other agents of a type or amount harmful to beluga whales; (4) Unrestricted passage within or between the critical habitat areas; and (5) absence of in-water noise at levels result in the abandonment of habitat by Cook Inlet beluga whales. The comment period on this proposed rule closed on February 1, 2010.

Southern Resident Killer Whale

Distribution and Description of the Listed Species

Three kinds of killer whales occur along the Pacific Coast of the United States: Eastern North Pacific (ENP) southern resident killer whales, ENP Offshore killer whales, and ENP transient killer whales. Of these only the southern resident killer whales are listed as endangered or threatened under the ESA. Southern resident killer whales primarily occur in the inland waters of Washington State and southern Vancouver Island, although individuals from this population have been observed off the Queen Charlotte Islands (north of their traditional range) and off coastal California in Monterey Bay, near the Farallon Islands, and off Point Reyes (NMFS 2005; BOR 2008).

Southern resident killer whales spend a significant portion of the year in the inland waterways of

the Strait of Georgia, Strait of Juan de Fuca, and Puget Sound, particularly during the spring, summer, and fall, when all three pods regularly occur in the Georgia Strait, San Juan Islands, and Strait of Juan de Fuca (Heimlich-Boran 1988; Felleman et al. 1991; Olson 1998; Osborne 1999). The K and L pods typically arrive in May or June and remain in this core area until October or November, although both pods make frequent trips lasting a few days to the outer coasts of Washington and southern Vancouver Island (Ford et al. 2000). The J pod will occur intermittently in the Georgia Basin and Puget Sound during late fall, winter and early spring. During the warmer months, all of the pods concentrate their activities in Haro Strait, Boundary Passage, the southern Gulf Islands, the eastern end of the Strait of Juan de Fuca, and several localities in the southern Georgia Strait (Heimlich-Boran 1988; Felleman et al. 1991; Olson 1998; Ford et al. 2000).

Southern resident killer whales are fish eaters, and predominantly prey upon salmonids, particularly Chinook salmon, but are also known to consume more than 20 other species of fish and squid (Scheffer and Slipp 1948; Ford et al. 1998; Ford et al. 2000; Saulitis et al. 2000; Ford and Ellis 2005; Ford and Ellis 2006;). Throughout inland waters from May to September, southern resident killer whale diet is approximately 88% Chinook salmon, with a shift to chum salmon in fall. Chum salmon are also taken in significant amounts (11%), especially in autumn (Hanson et al. 2005; Ford and Ellis 2006; Hanson et al. 2007b). Chinook salmon are preferred despite much lower abundance in comparison to other salmonids (such as sockeye) presumably because of the species' large size, high fat and energy content, and year-round occurrence in the area. Killer whales also capture older (i.e., larger) than average Chinook salmon (Ford and Ellis 2006). Little is known about the winter and early spring diet of southern residents. Early results from genetic analysis of fecal and prey samples indicate that Southern Residents consume Fraser River-origin Chinook salmon, as well as salmon from Puget Sound, Washington and Oregon coasts, the Columbia River, and Central Valley of California (Hanson et al. 2007a). However, recent studies suggest that members of L pod have undergone dietary shifts from Chinook salmon during fall months over the past decade (Krahn et al. 2009).

The local movements of southern resident killer whales usually follow the distribution of salmon (Heimlich-Boran 1986a, 1988, Nichol and Shackleton 1996). Areas that are major corridors for migrating salmon, and therefore, for southern resident killer whales, include Haro Strait and Boundary Passage, the southern tip of Vancouver Island, Swanson Channel off North Pender Island, and the mouth of the Fraser River delta, which is visited by all three pods in September and October (Felleman et al. 1991, Ford et al. 2000, K.C. Balcomb, unpublished data).

Female southern resident killer whales give birth to their first surviving calf between the ages of 12 and 16 years (mean ~ 14.9 years) and produce an average of 5.4 surviving calves during a reproductive life span lasting about 25 years (Matkin et al. 2003; Olesiuk et al. 1990). Females reach a peak of reproduction around ages 20-22 and decline in calf production gradually until reproductive senescence (Ward et al. 2009a). Older mothers tend to have greater calving success than do their younger, less-experienced counterparts (Ward et al. 2009b). Calving success also appears to be aided by the assistance of grandmothers (Ward et al. 2009b). The mean interval between viable calves is four years (Bain 1990). Males become sexually mature at body lengths ranging from 17 to 21 feet, which corresponds to between the ages of 10 to 17.5 years (mean ~ 15 years), and are presumed to remain sexually active throughout their adult lives (Christensen

1984; Duffield and Miller 1988; Olesiuk et al. 1990; Perrin and Reilly 1984). Most mating is believed to occur from May to October (Matkin et al. 1997; Nishiwaki 1972; Olesiuk et al. 1990). However, conception apparently occurs year-round because births of calves are reported in all months. Newborns measure seven to nine feet long and weigh about 200 kg (Clark et al. 2000; Ford 2002; Nishiwaki and Handa 1958; Olesiuk et al. 1990). Mothers and offspring maintain highly-stable, life-long social bonds and this natal relationship is the basis for a matrilineal social structure (Baird 2000; Bigg et al. 1990; Ford et al. 2000). Some females may reach 90 years of age (Olesiuk et al. 1990).

Southern resident killer whales spend a significant portion of the year in the inland waterways of the Strait of Georgia, Strait of Juan de Fuca, and Puget Sound, particularly during the spring, summer, and fall, when all three pods are regularly present in the Georgia Basin (defined as the Georgia Strait, San Juan Islands, and Strait of Juan de Fuca) (Felleman et al. 1991; Heimlich-Boran 1988; Olson 1998; Osborne 1999). Typically, K and L pods arrive in May or June and primarily occur in this core area until October or November. During this stay, both pods also make frequent trips lasting a few days to the outer coasts of Washington and southern Vancouver Island (Ford et al. 2000); however, J pod's movements differ considerably and are present only intermittently in the Georgia Basin and Puget Sound. Late spring and early fall movements of Southern Residents in the Georgia Basin have remained fairly consistent since the early 1970s, with strong site fidelity shown to the region as a whole (NMFS 2005b). During late fall, winter, and early spring, the ranges and movements of the southern residents are less well known. Offshore movements and distribution are largely unknown for the southern resident population.

While the southern residents are in inland waters during the warmer months, all of the pods concentrate their activities in Haro Strait, Boundary Passage, the southern Gulf Islands, the eastern end of the Strait of Juan de Fuca, and several localities in the southern Georgia Strait (Felleman et al. 1991; Ford et al. 2000; Heimlich-Boran 1988; Olson 1998). Individual pods are similar in their preferred areas of use, although there are some seasonal and temporal differences in certain areas visited (Olson 1998). For example, J pod is the only group to venture regularly inside the San Juan Islands. The movements of southern resident killer whales relate to those of their preferred prey, salmon. Pods commonly seek out and forage in areas where salmon occur, especially those associated with migrating salmon (Heimlich-Boran 1986; Heimlich-Boran 1988; Nichol and Shackleton 1996).

Members of different pods do interact, but members generally remain within their matrilinear group (Parsons et al. 2009). However, additional interaction between pods has occurred over the past two decades, possibly in association with the decline of the Southern Resident population as a whole (Parsons et al. 2009).

Population Structure

Southern resident killer whale DPS consists of three pods, or stable familial groups: the J pod, K pod, and L pod. The J pod is seen most frequently along the western shore of San Juan Island and is the only pod observed regularly in Puget Sound throughout winter (Heimlich-Boran 1988; Osborne 1999). The K pod is most frequently observed during May and June when they occur along the western shore of San Juan Island while searching for salmon. The L pod is the largest of the three pods (Ford et al. 1994) and frequently breaks off into separate subgroups.

Status

Southern resident killer whales were listed as endangered under the ESA in 2005 (70 FR 69903). In the mid- to late-1800s, southern resident killer whales were estimated to have numbered around 200 individuals. By the mid-1960s, they had declined to about 100 individuals. As discussed in the preceding section, between 1967 and 1973, 43 to 47 killer whales were removed from the population to provide animals for displays in oceanaria and the population declined by about 30 percent as a result of those removals. By 1971, the population had declined to about 67 individuals. Since then, the population has fluctuated between highs of about 90 individuals and lows of about 75 individuals.

At population sizes between 75 and 90 individuals, we would expect southern resident killer whales to have higher probabilities of becoming extinct because of demographic stochasticity, demographic heterogeneity (Coulson et al. 2006; Fox et al. 2006) —including stochastic sex determination (Lande et al. 2003) — and the effects of phenomena interacting with environmental variability. Demographic stochasticity refers to the randomness in the birth or death of an individual in a population, which results in random variation on how many young that individuals produce during their lifetime and when they die. Demographic heterogeneity refers to variation in lifetime reproductive success of individuals in a population (generally, the number of reproductive adults an individual produces over their reproductive lifespan), such that the deaths of different individuals have different effects on the growth or decline of a population (Coulson et al. 2006). Stochastic sex determination refers to the randomness in the sex of offspring such that sexual ratios in population fluctuate over time (Melbourne and Hastings 2008). For example, the small number of adult male southern resident killer whales might represent a stable condition for this species or it might reflect the effects of stochastic sex determination. Regardless, a high mortality rates among adult males in a population with a smaller percentage of males would increase the imbalance of male-to-female gender ratios in this population and increase the importance of the few adult males that remain.

At these population sizes, population's experience higher extinction probabilities because stochastic sexual determination leaves them with harmful imbalances between the number of male or female animals in the population (which occurred to the heath hen and dusky seaside sparrow just before they became extinct), or because the loss of individuals with high reproductive success has a disproportionate effect on the rate at which the population declines (Coulson et al. 2006). In general, an individual's contribution to the growth (or decline) of the population it represents depends, in part, on the number of individuals in the population: the smaller the population, the more the performance of a single individual is likely to affect the population's growth or decline (Coulson et al. 2006). Given the small size of the southern resident killer whale population, the performance (= "fitness," measured as the longevity of individuals and their reproductive success over their lifespan) of individual whales would be expected to have appreciable consequences for the growth or decline of the southern resident killer whale population.

These phenomena would increase the extinction probability of southern resident killer whales and amplify the potential consequences of human-related activities on this species. Based on their population size and population ecology (that is, slow-growing mammals that give birth to

single calves with several years between births), we assume that southern resident killer whales would have elevated extinction probabilities because of exogenous threats caused by anthropogenic activities that result in the death or injury of individual whales (for example, ship strikes or entanglement) and natural phenomena (such as disease, predation, or changes in the distribution and abundance of their prey in response to changing climate) *as well as* endogenous threats resulting from the small size of their population. Based on the number of other species in similar circumstances that have become extinct (and the small number of species that have avoided extinction in similar circumstances), the longer southern resident killer whales remain in these circumstances, the greater their extinction probability becomes.

Social Behavior

Killer whales are highly social animals that occur primarily in groups or pods of up to 40-50 animals (Dahlheim and Heyning 1999; Baird 2000). Mean pod size varies among populations, but often ranges from 2 to 15 animals (Kasuya 1971; Condy et al. 1978; Mikhalev et al. 1981; Braham and Dahlheim 1982; Dahlheim et al. 1982; Baird and Dill 1996). Larger aggregations of up to several hundred individuals occasionally form, but are usually considered temporary groupings of smaller social units that probably congregate near seasonal concentrations of prey, for social interaction, or breeding (Dahlheim and Heyning 1999; Baird 2000; Ford et al. 2000).

In terms of gender and age composition, southern and northern resident killer whales social groups consisted of 19 percent adult males, 31 percent adult females, and 50 percent immature whales of either sex in 1987 (Olesiuk et al. 1990a). This composition is comparable with the composition of southern Alaska resident killer whales and killer whale populations in the Southern Ocean (Matkin et al. 2003; Miyazaki 1989).

Threats

Natural Threats. Southern resident killer whales like many wild animal populations (Nettles, 1992), experience highest mortality in the first year age class (Olesiuk et al. 1990; Krahn et al. 2002), although the reasons for these mortalities are still uncertain. The causes could include poor mothering, infectious or non-infectious diseases, and infanticide (Gaydos et al. 2004).

Gaydos et al. (2004) identified 16 infectious agents in free-ranging and captive southern resident killer whales, but concluded that none of these pathogens were known to have high potential to cause epizootics. They did, however, identify pathogens in sympatric odontocete species that could threaten the long-term viability of the small southern resident population.

Anthropogenic Threats. Several human activities appeared to contribute to the decline of southern resident killer whales. Southern resident killer whales were once shot deliberately in Washington and British Columbia (Scheffer and Slipp 1948; Pike and MacAskie 1969; Olesiuk et al. 1990; Baird 2001). Until 1970, about 25 percent of the killer whales that were captured for aquaria had bullet scars (Hoyt 1990). The effect of these attacks on individual whales or the population itself remains unknown. However, between 1967 and 1973, 43 to 47 killer whales were removed from the population for displays in oceanaria; because of those removals, the southern resident killer whale population declined by about 30%. By 1971, the population had declined to about 67 individuals. Since then, the population has fluctuated between highs of about 90 individuals and lows of about 75 individuals.

Over the same time interval, southern resident killer whales have been exposed to changes in the distribution and abundance of their prey base (primarily Pacific salmon) which has reduced their potential forage base, potential competition with salmon fisheries, which reduces their realized forage base, disturbance from vessels, and persistent toxic chemicals in their environment. The primary prey of killer whales, salmon, has been severely reduced due to habitat loss and overfishing of salmon along the West Coast (NRC 1996;Slaney et al. 1996; Gregory and Bisson 1997; Lichatowich 1999; Lackey 2003; Pess et al. 2003; Schoonmaker et al. 2003;). Several salmon species are currently protected under the ESA, and are generally well below their former numbers. A 50% reduction in killer whale calving has been correlated with years of low Chinook salmon abundance (Ward et al. 2009a).

Puget Sound also serves as a major port and drainage for thousands of square kilometers of land. Contaminants entering Puget Sound and its surrounding waters accumulate in water, benthic sediments and organisms (Krahn et al. 2009). Exposure to contaminants may harm southern resident killer whales. The presence of high levels of persistent organic pollutants, such as PCB, DDT, and flame –retardants have been documented in southern resident killer whales (Ross et al. 2000; Ylitalo et al. 2001; Herman et al. 2005; Ross 2006). Although the consequences of these pollutants on the fitness of individual killer whales and the population itself remain unknown, in other species these pollutants have been reported to suppress immune responses (Kakushke and Prange 2007), impair reproduction, and exacerbate the energetic consequences of physiological stress responses when they interact with other compounds in an animal's tissues (Martineau 2007). Because of their long life span, position at the top of the food chain, and their blubber stores, killer whales would be capable of accumulating high concentrations of contaminants.

Since the 1970s commercial shipping, whale watching, ferry operations, and recreational boat traffic have increased in Puget Sound and the coastal islands of southern British Columbia. This traffic exposes southern resident killer whales to several threats that have consequences for the species' likelihood of avoiding extinction and recovering if it manages to avoid extinction. First, these vessels increase the risks of southern resident killer whales being struck, injured, or killed by ships. In 2005, a southern resident killer whale was injured in a collision with a commercial whale watch vessel although the whale subsequently recovered from those injuries. However, in 2006, an adult male southern resident killer whale, L98, was killed in a collision with a tug boat; given the gender imbalances in the southern resident killer whale population, we assume that the death of this adult male would have reduced the demographic health of this population (see further discussion below).

Second, the number and proximity of vessels, particularly whale-watch vessels in the areas occupied by southern resident killer whales, represents a source of chronic disturbance for this population. Numerous studies of interactions between surface vessels and marine mammals have demonstrated that free-ranging marine mammals engage in avoidance behavior when surface vessels move toward them. It is not clear whether these responses are caused by the physical presence of a surface vessel, the underwater noise generated by the vessel, or an interaction between the two (Goodwin and Green 2004; Lusseau 2006). However, several authors suggest that the noise generated during motion is probably an important factor (Blane and Jackson 1994; Evans et al. 1992, 1994). These studies suggest that the behavioral responses of marine mammals to surface vessels are similar to their behavioral responses to predators.

Several investigators have studied the effects of whale watch vessels on marine mammals (Watkins 1986; Cockeron 1995; Au and Green 2000; Erbe 2002; Félix 2001; Magalhães et al. 2002; Williams et al. 2002; Richter et al. 2003; Scheidat et al. 2004; Amaral and Carlson 2005; Simmonds 2005;). The whale's behavioral responses to whale watching vessels depended on the distance of the vessel from the whale, vessel speed, vessel direction, vessel noise, and the number of vessels. The whales' responses changed with these different variables and, in some circumstances, the whales did not respond to the vessels. In other circumstances, whales changed their vocalizations, surface time, swimming speed, swimming angle or direction, respiration rates, dive times, feeding behavior, and social interactions.

In addition to the disturbance associated with the presence of vessels, the vessel traffic affects the acoustic ecology of southern resident killer whales, which would affect their social ecology. Foote et al. (2004) compared recordings of southern resident killer whales that were made in the presence or absence of boat noise in Puget Sound during three time periods between 1977 and 2003. They concluded that the duration of primary calls in the presence of boats increased by about 15% during the last of the three time periods (2001 to 2003). At the same time, Holt et al. (2007) reported that southern resident killer whales in Haro Strait off the San Juan Islands in Puget Sound, Washington, increased the amplitude of their social calls in the face of increased sounds levels of background noise. Although the costs of these vocal adjustments remains unknown, Foote *et al*. (2004) suggested that the amount of boat noise may have reached a threshold above which the killer whales needs to increase the duration of their vocalization to avoid masking by the boat noise.

Critical Habitat

NMFS designated critical habitat for the DPS of Southern Resident killer whales on November 29, 2006 (71 FR 69054). Three specific areas were designated; (1) the Summer Core Area in Haro Strait and waters around the San Juan Islands; (2) Puget Sound; and (3) the Strait of Juan de Fuca, which comprise approximately 6,630 square kilometers of marine habitat. Three primary constituent elements exist in these areas: water quality to support growth and development, prey species of sufficient quantity, quality, and availability to support individual growth, reproduction and development, as well as overall population growth, and passage conditions to allow for migration, resting, and foraging. Water quality has declined in recent years due to agricultural run-off, urban development resulting in additional treated water discharge, industrial development, and oil spills. The primary prey of southern residents, salmon, has also declined due to overfishing and reproductive impairment associated with loss of spawning habitat. The constant presence of whale-watching vessels and growing anthropogenic noise background has raised concerns about the health of areas of growth and reproduction as well.

Environmental Baseline

By regulation, the environmental baseline for biological opinions include the past and present impacts of all state, Federal or private actions and other human activities in the action area, the anticipated impacts of all proposed Federal projects in the action area that have already

undergone formal or early section 7 consultation, and the impact of State or private actions which are contemporaneous with the consultation in process (50 CFR 402.02). The environmental baseline for this biological opinion also includes a general description of the natural factors influencing the current status of the listed species, their habitats, and the environment within the action area. The baseline analysis "is not the proportional share of responsibility the federal agency bears for the decline in the species, but what jeopardy might result from the agency's proposed actions in the present and future human and natural contexts." Pacific Coast Federation, 426 F.3d at 1093.

Our summary of the environmental baseline complements the information provided in the status of the species section of this Opinion, provides information on the past and present ecological conditions of the action area that is necessary to understand the species' current risk of extinction, and provides the background necessary to understand information presented in the *Effects of the Action* and *Cumulative Effects* sections of this biological opinion. The "impact" of the activities we normally identify in the *Environmental Baseline* of our Opinons allows us to assess the prior experience and state (or condition) of the endangered and threatened individuals and areas of designated critical habitat that occur in an action area. This is important because, as noted in the *Approach to the Assessment* section of this Opinion, in some phenotypic states, listed individuals will commonly exhibit responses they would not exhibit in other phenotypic states. The same is true for populations of endangered and threatened species: the consequences of change in the performance of individual on a population depend on the prior state of the population. Designated critical habitat is not different: under some ecological conditions, the physical and biotic features of critical habitat will exhibit response that they would not exhibit in other conditions. When we "add" the effects of a new, continuing, or proposed action to the prior condition of endangered and threatened individuals and designated critical habitat, as our regulations require, our assessments are more likely to detect a proposed action's "true" consequences on endangered species, threatened species, and designated critical habitat.

Because this is a programmatic consultation on what is essentially a continuing action with a geographic scope that encompasses all waters of the United States and its territories, this environmental baseline serves a slightly different purpose. First, as both a programmatic and a national consultation this Opinion does not assess the consequences of the EPA's recommended aquatic life criteria for specific sites or the listed resources that occur those specific sites. Rather, the *Environmental Baseline* for this Opinion focuses on the status and trend of the aquatic ecosystems in the United States and the consequences of that status for listed resources. Since our action area and the environmental baseline encompass a very broad spatial scale with many distinct ecosystems, wherever possible we have focused on common indicators of the biological, chemical, and physical health of the nation's aquatic environments. The *Environmental Baseline* for this consultation provides the background information and context that is necessary for our assessment of the *Effects of the Action.*

We divided the environmental baseline for this consultation into five broad geographic regions of the United State: the Atlantic Northeast Region, the Atlantic Southeast Region, the Gulf Coast Region, the Southwest Region, and the Pacific Northwest Region. In some instances regions were further subdivided according to ecoregions, importance to NMFS' trust resources or other natural features. In each section we describe the biological and ecological characteristics of the

region such as the climate, geology, and predominant vegetation to provide landscape context and highlight some of the dominant processes that influence the biological and ecological diversity of the region where threatened and endangered species reside. We then described the predominant land and water uses within a region to illustrate how the physical and chemical health of regional waters and the impact of human activities have contributed to current status of listed resources.

Atlantic Northeast Region

This region encompasses Maine, New Hampshire, Massachusetts, Rhode Island, Vermont, Connecticut, New York, New Jersey, Delaware, Pennsylvania, Maryland, and Virginia. Major rivers in this region are the Penobscot, Connecticut, Hudson, Delaware, and Susquehanna rivers. Important estuarine areas include the Chesapeake Bay, Long Island Sound, Cape Cod Bay, and Massachusetts Bay.

The region is ecologically diverse, encompassing several broad ecoregions. According to Bailey's (1995) *Description of the Ecoregions of the United States,* this region encompasses the warm continental, the hot continental and the hot continental mountains divisions, and northern portions of the subtropical division – these ecoregions can be further subdivided into provinces based on vegetation. Climate is defined by hot humid summers and cold winters. Mean annual precipitation varies from about 35 to 45 inches per year. Vegetation in this region is characterized by tall broadleaf trees that provide a continuous dense canopy in summer, but shed their leaves completely in winter. Lower layers of small trees and shrubs are weakly developed. In spring, a luxuriant ground cover of herbs quickly develops, but is greatly reduced after trees reach full foliage and shade the ground. Needleleaf trees grow in colder, northern parts of the region and in mountain areas. Soils are generally rich in humus and strongly to moderately leached, although in the southern portions of this region, soils tend to be sandier and support second-growth forests of longleaf, loblolly, and slash pines (Bailey 1995).

Gulf of Maine

Natural History

This region encompasses drainages entering the Gulf of Maine, and is one of the most productive marine ecosystems in the world. Several significant rivers that drain into the gulf include the Merrimac, Kennebec, Androscoggin, Penobscot, and St. John Rivers (Table 24), and the significant estuaries that compose the larger Gulf of Maine include the Bay of Fundy, Massachusetts Bay, Merrymeeting Bay, and Cape Cod Bay. The Gulf of Maine is semi-enclosed, bounded to the south by Georges Banks and to the north by Brown's Bank. The area is strongly influenced by the Labrador Current, which makes the waters significantly colder and more nutrient rich than waters to the south, which are more strongly influenced by the Gulf Stream. The Gulf of Maine is characterized by salt marshes, kelp and seagrass beds, tidal mudflats, and underwater rocky outcrops, which form the foundation of a complex ecosystem and provide habitat for Atlantic herring, American lobster, Atlantic salmon, and several whale species. Merrymeeting Bay is the largest freshwater tidal estuary that enters the Gulf of Maine and has the largest freshwater outflow to the gulf (Kistner and Pettigrew 2001; Jackson et al. 2005). The

Kennebec and Androscoggin Rivers, along with four smaller tributaries, converge to form Merrymeeting Bay with the two larger rivers accounting for 98% of the inflow.

Table 27. Select rivers of the northeast United States that drain to the Gulf of Maine

Watershed	Approx. Length (mi)	Basin Size (mi^2)	Physiographic Provinces*	Mean Annual Precipitation (in)	Mean Discharge (cfs)	Number of Fish Species	Number of Endangered Species
Penobscot	275	8,592	NE	42	14,196	45	1 fish
Kennebec	230	5,383	NE	43	9,076	48	1 fish
Androscoggin	164	3,263	NE	44	6,180	33	1 fish
Merrimack	180	5,014	NE	36	8,299	50	1 fish

Data from Jackson et al 2005; Maine Rivers 2007a, b

*Physiographic Provinces: NE = New England, AD = Adirondack Mountains, VR = Valley Ridge, AP = Appalachian Plateau, PP = Piedmont Plateau, CP = Coastal Plain, BR = Blue Ridge.

Human Activities and Their Impacts

Land Use. Most of the watersheds within this region are heavily forested with relatively small areas of highly urbanized lands (Table 25). While there is not much urban development in the Penobscot watershed except in and around Bangor, Doggett and Sowles (1989) report that tanneries, metal finishing, pulp and paper mills, textile plants, chemical products, and municipal sewage contribute chromium, mercury, zinc, copper, lead, arsenic, hydrocarbons, dioxins, PAHs, pesticides, and other contaminants to the river. The only major town in the Kennebec River watershed is Augusta, Maine (Jackson et al. 2005). The heaviest population density occurs in the watershed of the Merrimack River, which flows through industrial centers Manchester and Concord, New Hampshire, and Lowell and Lawrence, Massachusetts.

Textile mills, as well as paper and pulp mills, have long influenced water quality in the Penobscot, Kennebec, and Androscoggin rivers. The Kennebec River exceeds recommended levels of dioxins, arsenic, cadmium, chromium, copper, lead, mercury, nickel, silver, zinc, and PAHs in the sediments and surface water (MDEP 1999, Harding Lawson Associates 1999, Harding Lawson Associates 2000). Since 1990, the levels of dioxins in other Maine rivers have been decreasing, but the levels in the Kennebec have remained constant (Kahl 2001). At one time, the Androscoggin River was considered one of the ten most polluted rivers in the country. The river has become much cleaner since the CWA was passed, but pesticides, mercury, lead, sedimentation, total suspended solids, PCBs, and dioxins are still considered too high (Chamberland et al. 2002).

The Merrimack River watershed is one of the most heavily urbanized watersheds in the region, and some of the biggest sources of pollution facing the river are from industrial and urban sources, such as combined sewage overflows, industrial discharge, and stormwater run-off (USACE 2003). The upper mainstem of the Merrimack River has problems with bacteria, *E. coli*, and acidity, while the lower mainstem has problems with bacteria, metals, nutrients, dioxins, turbidity and suspended solids, and un-ionized ammonia. In all, over 125 miles of mostly lower watershed areas do not support their designated uses (USACE 2003).

Toxins draining from river systems have produced significant toxin levels in regional estuarine

systems, particularly from New Hampshire south throughout the Cape Cod region. Casco Bay still harbors residual sediment contamination and organic carbon levels from industries of a century ago, including heavy metals, PCBs, pesticides, TBT, dioxins and furans, and PAHs (EPA 2006). Low dissolved oxygen and red tide from nutrient loading also remain issues in the area. Habitats here remain relatively coalesced, although fragmentation is on the rise, and eelgrass beds have undergone local reductions.

Toxic sediments have been identified in Merrymeeting Bay, although some pollutants like metals declined in the bay between 1980 and 1991, although copper levels have increased (Hayden 1998). Sediments associated with the Androscoggin River exhibit higher levels of PAHs and mercury, while sediments from the Kennebec River had higher levels of chromium, arsenic, and selenium (Hayden 1998). Merrymeeting Bay has more moderate levels of these toxins than the rivers themselves. Chilcote and Waterfield (1995) found that levels of arsenic are higher than levels identified by EPA as likely to have adverse effects. At one station, PAHs from the Androscoggin also exceeded EPA-identified levels of minimal effects. Commercially important fish also have elevated metal concentrations in their livers, which is thought to be from their time spent in Merrymeeting Bay (Kistner and Pettigrew 2001).

Human activities have impact the coasts of New Hampshire and Massachusetts. New Hampshire estuaries suffer from habitat fragmentation and degradation, bacterial and nutrient contamination, salt marsh degradation, and declines in the commercially valuable oyster and clam populations resulting from sewage and industrial pollution (EPA 2006). Several areas experience elevated nitrogen and phosphorus in water, high total organic carbon, and sediment contaminant levels in the benthos, as well as above average contaminants (PAHs, DDT, and PCBs) in fish and shellfish. A massive decline in eelgrass habitats occurred in 1989 and meadows have been relatively constant since.

Estuarine and bay systems of Massachusetts experience pressures from the major metropolitan region around Boston Harbor. The increased sewage and stormwater outflow results in a loss of roughly 1,000 acres of wetland habitat per year and cause closings in shellfish harvests due to bacterial contamination. Local wetland restoration projects have improved over 450 acres of wetland in the region. Over 26 invasive species have been identified in Massachusetts Bay, including the Asian shore crab and Pacific tunicate, and have contributed to a reduction in the industrial scallop fishery.

Table 28. Land uses and population density of several watersheds that drain to the Gulf of Maine

Watershed	Land Use Categories (%)				Density
	Agriculture	Forested	Urban	Other	(people/mi^2)
Penobscot	5	95			21
Kennebec	6	82	2	10	39
Androscoggin	5	86	2	7	65
Merrimack	6	75	13	6	404

Data from Jackson et al. 2005

Hydromodification Projects. There are five major hydroelectric dams along the mainstem of the Penobscot River as well as 111 other licensed dams located along the river and its tributaries.

Atlantic salmon historically migrated as far as 143 miles upstream of the mouth, but due to development along the river in the 1960s, Atlantic salmon were extirpated (Jackson et al. 2005). The population has since been re-established and runs of 2,000 to 4,000 occur with natural spawning as far upstream as 62 miles. Because 6,000 to 10,000 salmon are required for a sustainable population, the Penobscot run depends on fish from a local hatchery (Moore and Platt 1996).

The Kennebec River mainstem has eight large hydroelectric dams, which restrict fish passage both up and downstream. In 1999, the Edwards Dam was removed, opening 17 additional miles of habitat for fish and macroinvertebrates in the river. Removal of Edwards Dam restored full access to historical spawning habitat for species like Atlantic sturgeon, shortnose sturgeon, and rainbow smelt, but not for species like alewife, American shad, and Atlantic salmon that migrated much further up the river. Since the removal of Edwards Dam, dissolved oxygen levels and macroinvertebrate density have improved. Additionally, in 2007, the fish passage facilities on the lowest dam on the Kennebec River, as well as those on the Sebasticook River's second and third lowest dams, became operational.

The Androscoggin River has 14 hydroelectric dams on the mainstem of the river and 18 in the watershed. Fish ladders have been installed on the lower dams allowing anadromous fish passage to Lewiston Falls (Brown et al. 2006). The dams play a considerable role in the poor water quality of the river, causing reduced dissolved oxygen throughout the summer. During the 1960s, most of the river had oxygen levels of 0 ppm, resulting in massive fish kills. There is still a 14-mile stretch of river that requires aerators to provide dissolved oxygen to the river.

The Merrimack River watershed has over 500 dams, including three in Massachusetts and three in New Hampshire, that essentially make the mainstem into a series of ponds (Dunn 2002; Jackson et al. 2005). Flow alteration is considered a problem on the upper mainstem of the river and has resulted in the river not meeting EPA's flow requirements (USACE 2003).

Mining. Mining in watersheds of the Atlantic Northeast Region began before the Civil War. Since then, mining has been conducted for granite, peat, roofing slate, iron ore, sulfur, magnetite, manganese, copper, zinc, mica, and other materials. Currently, exploration for precious metals and basic metals is ongoing, but to a lesser extant than during the 1980s. Recent mining activities were conducted in this region by The Penobscot Nation, Champion Paper Company, Oquossoc Minerals, Boliden Resources, Inc., Black Hawk Mining, and BHP-Utah. There are several abandoned mines in the northeast watersheds that have become Superfund sites due to excessive pollutants being leached into groundwater, such as Elizabeth, Pike Hill, and Calhoun Mines, and others. Common pollutants leaked by mining operations in this area are lead, mercury, arsenic, and selenium (Ayuso et al. 2006; Piatak et al. 2006). Many of the abandoned mines are scheduled for cleanup; however, the impacts of their former use could persist for years after decommissioning.

Commercial and Recreational Fishing. The primary commercial fisheries along the Northeast coast by harvest weight exist for herring (39%), lobster (26%), blue mussel (6%), hatchery-origin sea-run Atlantic salmon (4%), groundfish (4%), quahog (4%), soft clam (3%), sea cucumber (3%), seaweed (3%), crabs (2%), and various other species (6%). Directed harvest of shortnose

sturgeon and wild Atlantic salmon is prohibited by the ESA; however, both are taken incidentally in other fisheries along the east coast and are probably targeted by poachers throughout their range (Dadswell 1979; Dovel et al. 1992; Collins et al. 1996).

Long Island and the Connecticut River

Natural History

South of the Gulf of Maine is the Long Island Sound watershed, which includes portions of Connecticut, New York, Massachusetts, New Hampshire, Rhode Island, and Vermont. Long Island Sound was designated a national estuary in 1987, due to its significance as an area where fresh water from the Connecticut, Thames, and Housatonic rivers (90% of the freshwater input) mixes with the Atlantic Ocean. The sound ranges in salinity from 23 ppt in the western end to 35 ppt on the eastern side. The surface area of Long Island Sound is 1,320 square miles, draining an area of over 16,000 square miles. Long Island Sound connects to the Atlantic Ocean on both the eastern and western side, called "The Race" and the East River, respectively. The sound substrate is primarily mud, sand, silt, and clay, with very small areas of exposed bedrock. The sound is home to more 120 species of fish and at least 50 species use Long Island Sound as spawning grounds.

The Connecticut River drains a watershed of 11,259 square miles and flows approximately 410 miles to Long Island Sound. The river flows from the highlands of New Hampshire and Quebec, and is bordered by the Green and White Mountains. The Connecticut River bed is composed of glacial deposits and granitic bedrock. The average annual precipitation is approximately 43 inches. At the mouth, the average discharge is 10.2 billion gallons per day, or 15,715 cubic feet per second, which accounts for approximately 70% of the freshwater inflow to Long Island Sound (Jackson et al. 2005). The final 56 miles of the river prior to Long Island Sound is a tidal estuary (Jackson et al. 2005). The river and estuary are also important for many fish species, with 64 fresh water and 44 estuarine species having been recorded in the river or estuary, but 20 of the fish are nonnative (Jackson et al. 2005).

Human Activities and Their Impacts

Land Use. More than eight million people live in the Long Island Sound watershed. With so many people in the watershed, both point and non-point source pollution is a major concern. Toxic substances often adsorb to the surface of sediments, which means sediments with high surface to volume ratios like sand, silt, and clay, can hold more pollutants than larger substrates. The sound has elevated levels of PCBs, PAHs, nitrogen, lead, mercury, cadmium, cesium, zinc, copper, and arsenic. Organic and metal contaminants in Long Island Sound are above national averages (Turgeon and O'Connor 1991). Much of the lead, copper, and zinc are likely deposited via the atmosphere (Cochran et al. 1998). Cadmium, chlordane, and lead appear to be decreasing while copper is increasing (Turgeon and O'Connor 1991). Studies on winter flounder showed PAHs and PCBs leading to alteration of DNA in the livers of those fish (Gronlund et al. 1991). One of the biggest problems facing the sound is dissolved oxygen depletion (Parker and O'Reilly 1991), resulting in dead zones. The governors of Connecticut and New York have signed agreements to reduce the total nitrogen input to Long Island Sound by 58.5% before 2015 in an effort to get dissolved oxygen levels above 5 ppm for surface water, above 3.5 ppm for deeper

water, and at or above 2 ppm for all water.

Within the Connecticut River watershed the dominant land use is forest (80%), with 11% used for agriculture and the remaining 9% in mixed uses (Jackson et al. 2005). Major towns in the Connecticut River watershed are Holyoke and Springfield, Massachusetts and Hartford, Connecticut. The human population in the watershed is approximately 179 people per square mile (Jackson et al. 2005). Throughout the 20th century, power plants, defense contractors, municipalities, and corporations such as General Electric, Union Carbide, and Pfizer contributed large quantities of pollutants to the river. Still to this day, approximately one billion gallons of raw sewage enters the river as a result of combined sewer overflow from Hartford, Connecticut alone (CRWC 2006). The river has become much cleaner since the CWA was passed, but chromium, copper, nickel, lead, mercury, and zinc, chlordane, DDT, DDE, PCBs, and PAHs are found in quantities above the EPA-recommended levels in sediments and fish tissue throughout the watershed (Jackson et al. 2005). Acid rain also affects rivers in the northeast, as it reduces the pH of rivers and causes metals to leach from bedrock at a faster rate (USFWS 2007).

Estuaries within Long Island Sound have historically been plagued by low dissolved oxygen, pathogens, habitat degradation and species decline, and sediment contamination (EPA 2006). These issues remain relevant today, with increasing human populations increasing contaminant loads and decreasing wetland habitat. Almost all measures of quality have been affected, including phosphorus load, low dissolved oxygen, and chlorophyll *a* concentrations, high sediment contaminants (DDT and metals) and total organic carbon, as well as excessive levels of PCBs in nearly all fishes sampled. Riverine and wetland restoration has been ongoing for several years and provided an additional 2,000 acres of wetland and over 50 miles of stream passage for migratory fishes. This may help curtail the decline of estuarine bird populations and oysters in recent years. Oyster harvest closures resulting from pathogen concentrations have been common for two decades and additional regulation of vessel discharges, illegal sewage connections to Long Island Sound, high volume of storm water effluent, and malfunctioning septic systems are identified as point sources for this.

Hydromodification Projects. The Connecticut River has 16 hydroelectric dams on the mainstem of the river and as many as an estimated 900 have been built in the watershed. Fish ladders have been installed at Vernon, Turner Falls, and Holyoke Dams allowing fish passage to areas above Holyoke Dam in Massachusetts since 1981 (USGS 2004). For some species, the ladders are not efficient, so fish passage continues to be compromised. For instance, overall passage efficiency at Turner Falls fish ladder is 17%, and has historically been inefficient at passing shad. Shortnose sturgeon are not able to migrate to spawning habitat above Holyoke Dam, which was recently re-licensed through 2039, so the only spawning shortnose sturgeon in the river are the fish that reside above the dam. The dams also affect the river's water quality, causing reduced dissolved oxygen and elevated water temperatures throughout the summer.

Mining. Dating back thousands of years, there is evidence of native people mining and extracting natural resources from the headwaters of the Connecticut River. Towns such as Plymouth, Vermont were famous for mining gold, iron, talc, soapstone, marble, asbestos, and granite (Ewald 2003). Other towns throughout New Hampshire and Vermont also mined gold, silver, soapstone, talc, granite, slate, and copper (Ewald 2003). There are many mines along the

Connecticut River, which currently degrade the river's water quality, including the country's first chartered copper mine. In many locations, far downstream of the mines, accumulated heavy metals are in concentrations high enough to threaten aquatic life. In other cases, the mines are abandoned or failing and need to be cleaned. Such is the case with Elizabeth Mine, an old copper mine perched above the Connecticut River that leaches heavy metals into the river. As a result, Elizabeth Mine has been declared a Superfund site. There is little to no mining in Long Island Sound although there has been and continues to be discussions about mining for sand and gravel.

Commercial and Recreational Fishing. Few commercial fisheries exist in the Connecticut River. Shad is the primary commercial fishery, although shellfish, bluefish, striped bass, and flounder can be caught in the tidal estuary near the mouth. There are many recreationally angled fish, such as shad, striped bass, bluefish, northern pike, largemouth and smallmouth bass, perch, catfish, and others.

Long Island Sound fisheries provide an estimated 5.5 million dollars to the Connecticut economy. The primary fisheries target oysters, lobsters, scallops, blue crabs, flounder, striped bass, and bluefish. Recently, due to dissolved oxygen deficiencies, the western portion of Long Island Sound has seen major declines in fish and shellfish populations. Despite these declines, the sound houses the largest oyster fishery in the US, providing 95% of the nation's oysters. At this same time, lobsters have been suffering from an unknown disease and their population has been declining. Simultaneously, menhaden have made a dramatic recovery over the past 10 years, which has resulted in much better fishing for larger predatory fish, such as striped bass.

Directed harvest of shortnose sturgeon is prohibited by the ESA. However, shortnose sturgeon are likely taken incidentally in fisheries in the Connecticut River and Long Island Sound. Moser and Ross (1993) found that captures of shortnose sturgeon in commercial shad nets disrupted spawning migrations in the Cape Fear River, North Carolina. Weber (1996) reported that these incidental captures caused abandonment of spawning migrations in the Ogeechee River, Georgia.

Hudson River

Natural History

The Hudson River flows approximately 315 miles to the ocean, with a watershed of 13,365 square miles. The river flows from the Adirondack Mountains, draining most of eastern New York State, to the Atlantic Ocean where the Hudson River Canyon continues onto the continental shelf, marking where the original mouth of the Hudson was covered by rising sea levels after the last ice age. The Hudson River bed is composed of metamorphosed plutonic rock in the Adirondack Mountains, then transitions to sedimentary rock, such as shale and limestone in the middle portion of the watershed. The lower portion of the watershed is a mixture of sedimentary, metamorphic, and igneous rocks. Average annual precipitation is approximately 36 inches per year. At the mouth, the average discharge is 13.5 billion gallons per day, or 20,906 cubic feet per second (Jackson et al. 2005). The Hudson River is a freshwater tidal estuary between Troy, New York at river mile 154 to Newburgh Bay at river mile 62, and then it is a tidal brackish estuary for the lower 62 miles to the Atlantic Ocean (Jackson et al. 2005). The river and estuary are

home to over 200 fish species, with approximately 70 native freshwater fish species and 95 estuarine species having been recorded (Jackson et al. 2005).

Human Activities and Their Impacts

Land Use. The Hudson River watershed usage is 25% agriculture, 65% forested, 8% urban, and 5% other (Jackson et al. 2005). Major towns in the Hudson River watershed are New York City, Albany, Poughkeepsie, and Hudson, New York as well as Jersey City, New Jersey. The average population density in the watershed is approximately 350 people per square mile, but the actual density within a reach of the watershed varies widely. For instance, according to Jackson et al. (2005) population density at the headwaters at Lake Tear of the Clouds is 0 people per square mile, while the population density in Manhattan is over 25,907 people per square mile.

Throughout the 20th century, power plants, municipalities, pulp and paper mills, and corporations such as IBM, General Motors, and General Electric in particular, whom the EPA estimates dumped between 209,000 and 1.3 million pounds of PCBs into the river, contributed large quantities of pollutants to the Hudson River. The PCB levels in the Hudson River are among the highest nationwide. The upper basin is mostly unaffected by humans, with clear, soft water with low nutrients. The middle Hudson River is more polluted, with 30 to 50% of the land in this region being used for agriculture and several cities such as Corinth, Glens Falls, Hudson Falls, and Fort Edward contributing industrial waste to the river. The tidal freshwater portion of the river is nutrient rich with exceptionally low gradient. High tide in this stretch causes the river to flow backwards due to the low gradient and this prevents stratification. The brackish tidal estuary portion of the Hudson River is nutrient rich with hard water. Two hundred miles of the Hudson River, from Hudson Falls to New York City, were designated as a Superfund site due to the amount of pollution. There are still elevated amounts of cadmium, copper, nickel, chromium, lead, mercury, zinc, DDT, PCBs, and PAHs in quantities above the EPA recommended levels in sediments and fish tissue throughout the watershed (Wall et al. 1998).

Estuarine conditions surrounding the Hudson River are extremely poor (EPA 2006). Most issues stem from the extremely dense human population center around New York City. Fish consumption warnings are commonplace due to high mercury levels, over 200 million gallons of untreated sewage enter the bay daily, and only 20% of the former wetland area remains. Nitrogen and phosphorus are generally very high, sediments are highly contaminated (PCB), and total organic carbon is generally elevated. Only about 20,000 acres of wetland remain in the region. Although these poor conditions persist, wading birds formerly absent are present today and osprey (indicator bird species) are showing resurgence.

Hydromodification Projects. The mainstem Hudson River has 14 dams and dams exist near the mouths of many tributaries, but the lower 154 miles of tidally influenced river is undammed. Several flood control dams on tributaries such as the Indian and Sacandaga rivers have drastically altered the flow of the mainstem Hudson River. The Hudson is an important river for anadromous fishes because it has a low number of physical obstructions, and contains one of the largest populations of endangered shortnose sturgeon in the United States. Prior to the Clean Water Act, the middle stretch of the Hudson River and much of the lower reaches had low dissolved oxygen as a result of reduced flow behind the dams, high nutrients, and the collection of waste with high biological oxygen demand (Jackson et al. 2005).

Mining. The Hudson River has been periodically important as a source of metals and mined resources. The Adirondack Mountains, in the headwaters, have been mined for silver, iron, titanium, coal, talc, vanadium, graphite, garnet, and zinc at various times over the past 300 years. McIntyre Mine is an example of a mine that has produced different minerals during different generations. Initially bought as an iron mine, McIntyre sat dormant for 75 years before titanium was discovered there and mined until 1982, when it was abandoned.

Commercial and Recreational Fishing. The Hudson River commercial fishery historically caught fish, blue crabs, and oysters. Now, the only fish that is caught commercially in the Hudson is American shad. Historically, Atlantic sturgeon, striped bass, American eel, and white perch were productive commercial fisheries. The striped bass fishery closed in 1976 due to PCBs in the river and fish tissue. Atlantic sturgeon were fished until the mid 1990s. Blue crabs are still fished in the estuary all the way to Troy, New York with recent catches over 88,185 pounds per year. There is no commercial fishery for oysters but they used to be taken commercially in the brackish tidal section of the Hudson River.

Delaware River

Natural History

The Delaware River flows approximately 329 miles to the ocean, with a watershed of 12,757 square miles. The river originates in the Catskill Mountains with over half of the river flowing through Pennsylvania and the rest of the watershed occupying parts of New Jersey, New York, and Delaware. The Delaware River's geology is sandstone with shale conglomerate in the upper watershed transitioning to sandstone, shale, and limestone in the middle watershed and igneous and metamorphic rock in the lower watershed. The average annual precipitation is approximately 43 inches. At the mouth, the average discharge is 9.6 billion gallons per day, or 14,903 cubic feet per second. Although it is only the 42nd largest river in the United States by discharge, Philadelphia is home to the largest freshwater port in the country (Jackson et al. 2005). The Delaware River estuary begins in Trenton, New Jersey and extends downstream for 144 miles (Jackson et al. 2005). The river and estuary are home to 105 species of fish, with approximately eight nonnative fish (Jackson et al. 2005).

Human Activities and Their Impacts

Land Use. The Delaware River watershed usage is 24% agriculture, 60% forested, 9% urban, and 7% surface water or other (Jackson et al. 2005). Major towns in the Delaware River watershed are Easton, Allentown, Reading, and Philadelphia, Pennsylvania; Trenton and Camden, New Jersey; and Wilmington, Delaware. The human population in the watershed is approximately 555 people per square mile with most distributed around the estuary (Fischer et al. 2004; Jackson et al. 2005). As the area's population grew, water quality significantly degraded around Philadelphia with serious water quality problems observed as early as 1799. By the 1960s, the average dissolved oxygen in the lower river was approximately 0.2 ppm. A survey in the 1970s of organochlorine frequency in rivers ranked the Delaware at Trenton and the Schuylkill, the largest tributary to the Delaware River, as the 8th and 1st worst, respectively in the nation (Jackson et al. 2005). Urban and agricultural activities continue to affect the basin water quality today. Organochlorines, pesticides, nutrients, organics, and trace elements were

widely detected in small tributaries and mainstream reaches (Fischer et al. 2004). In the Delaware River Basin, commonly detected organochlorines include DDTs, PCBs, chlordanes, and dieldrin. Fisher et al. (2004) found that 84% of the fish tissue sampled contained PCBs, while 21% of the sediment samples contained PCBs despite that the manufacture and use of these compounds ceased in the 1970s or earlier. These compounds are bioaccumulating in the food chain, and occasionally exceed wildlife protection guidelines (52% of the sites exceeded wildlife guidelines for PCBs, whereas 16% of the sites exceeded guidelines for dieldrin, and 12% exceeded wildlife guidelines for DDT [Fischer et al. 2004]).

Trace metal contamination is also a significant concern within the basin, and may be a particular concern for benthos including endangered shortnose sturgeon. Trace metals detected at high levels included arsenic, cadmium, chromium, copper, lead, mercury, nickel, and zinc (Fischer et al. 2004). Most trace metal contamination was associated with former or on-going industrial sites such as mines and metal smelters.

The Delaware Estuary is considered to be in poor condition primarily from historical and modern toxin contributions from population centers such as Philadelphia along the Delaware River (EPA 2006). Overfishing and habitat loss also play a role in and along the estuary system itself. Estuarine waters contain high nitrogen and phosphorus levels with low chlorophyll *a* concentrations. Water clarity is variable, but fish tissues contain unsatisfactory levels of PCBs, DDT, and PAHs. Invasive plant species, including the common reed and purple loosestrife, have displaced native marsh species. Disease and low recruitment in oyster populations have had significant effects in commercial and ecological parameters of Delaware Bay. Shad populations declined due to poor water quality and have not recovered, which may indicate environmental stress in several other finfish populations.

Hydromodification Projects. The Delaware River has 16 dams in its headwaters, but the middle and lower rivers are the longest undammed stretch of river east of the Mississippi. This stretch of free-flowing river is beneficial to anadromous and catadromous species, such as American shad, striped bass, and American eels.

Mining. The Delaware River watershed, particularly the eastern section, was home to the majority of the nation's anthracite coal. As a result, many mining towns were established in the watershed to exploit the abundant resources. By 1914, over 181,000 people were employed as miners in the region. Apart from the coal mining, other minerals such as sulfur, talc, mica, aluminum, titanium, and magnesium were mined. Mines were also established for sand and gravel.

Commercial and Recreational Fishing. In the Delaware River, commercial fisheries exist for American shad, weakfish, striped bass, Atlantic croaker, Atlantic silversides, bay anchovy, black drum, hogchoker, northern kingfish, and American eel. Commercial fishermen use gillnets and trawls as the primary means of capturing fish. Bycatch is a concern for the recovery of endangered shortnose sturgeon, where the highest mortality rates are recorded in gillnet fisheries. Recreational fishermen target weakfish, striped bass, croaker, drum, kingfish, and eel.

Chesapeake Bay Drainages

Natural History

Chesapeake Bay, the largest estuary in the United States, was formed by glacial activity more than 18,000 years ago. The bay stretches some 200 miles from Havre de Grace, Maryland to Norfolk, Virginia, with more than 11,000 miles of shoreline. At its widest point, Chesapeake Bay is about 35 miles wide (near the Potomac River). Despite its massive size, the bay is relatively shallow – average depth is only 21 feet – making it susceptible to significant fluctuations in temperature.

Chesapeake Bay lies totally within the Atlantic Coastal Plain but the watershed includes parts of the Piedmont Province and the Appalachian Province. Tributaries provide a mixture of waters with a broad geochemical range to the bay with its own mixture of minerals, nutrients, and sediments depending on the geology of the place where the waters originate. In turn, the nature of the bay itself depends on the characteristics and relative volumes of these contributing waters. More than 100 rivers and streams deliver fresh water to Chesapeake Bay, and major rivers include the Susquehanna and Potomac (see Table 26).

The Susquehanna River, rated as the 18th largest river in the United States based on average discharge at the mouth, flows approximately 448 miles to Chesapeake Bay (Kammerer 1990; Jackson et al. 2005). The river flows north to south from New York, through Pennsylvania, and reaches the Chesapeake Bay at Havre de Grace, Maryland. The Susquehanna River's bed is rocky throughout, being described as a mile wide and a foot deep with distinct pool/riffle formations even near the mouth. The Susquehanna drains into the most northern portion of Chesapeake Bay and is not tidally influenced. The Susquehanna River provides about 50% of the freshwater inflow into the Chesapeake Bay.

Table 29. Select rivers of the northeast United States that drain to Chesapeake Bay

Watershed	Approx. Length (mi)	Basin Size (mi^2)	Physiographic Provinces*	Mean Annual Precipitation (in)	Mean Discharge (cfs)	No. Fish Species	No. Endangered Species
Susquehanna	448	27,580	AP, VR, PP, BR	39	40,718	103	2 birds
Potomac	383	14,700	AP, VR, PP BR, CP	39	11,301	95	1 fish, 1 mussel
James	340	10,102	VR, BR, PP, CP	43	8,016	109	3 fish, 1 amphibian, 1 reptile, 6 mussels

Data from Jackson et al. 2005; Smock et al. 2005
*Physiographic Provinces: NE = New England, AD = Adirondack Mountains, VR = Valley Ridge, AP = Appalachian Plateau, PP = Piedmont Plateau, CP = Coastal Plain, BR = Blue Ridge.

The Potomac and James Rivers, on the other hand, are located south of the Susquehanna River basin and are tidally influenced. The Potomac River estuary begins two miles below the Washington, D.C.-Maryland border, just below the Little Falls of the Potomac River. The river's headwaters begin in the Allegheny Mountains of West Virginia and the Potomac and flows through Washington, D.C., to the western side of the Chesapeake Bay. The substrate of the

Potomac and its tributaries is mostly schist, phyllite, and metavolcanic rock. Ninety-five fish species live in the Potomac, but only 65 of those are native to the area (Jackson et al. 2005). The James River is one of the longest bodies of water in entirely one state, beginning in the Allegheny Mountains of western Virginia and flowing across the state to the Chesapeake Bay.

Human Activities and Their Impacts

Land Use. The Chesapeake Bay area, while dominated by forested lands, is more heavily influenced by agriculture than many other areas in the northeast and middle Atlantic (see Table 27 for land uses by watershed). Urbanized areas are scattered but dominate the landscape in certain areas (e.g., Washington D.C. metro area, in the Potomac River watershed, and Scranton and Harrisburg, Pennsylvania in the Susquehanna River watershed). Most of the bay's waters are degraded, and algal blooms are a chronic problem. Nutrient pollution and heavy sediment loads have lead to large anoxic areas within the bay, and fish kills in some areas. Agricultural practices, urban development, as well as natural sources of sediment influence water quality within the bay. Past logging practices in the basins draining to Chesapeake Bay also influenced sediment loads within several rivers. In the Susquehanna River watershed, sediment transport in the early 1900s was nine times higher than it was 200 years earlier, due to logging and agriculture.

Overall, in 2006, less than one-third of Bay water quality goals were met (Chesapeake Bay Program 2007). Direct discharges of sewage and industrial wastewater into the Susquehanna River watershed and contributes to degraded water quality in the basin. The number of outfalls totals over 400 in the watershed, generally with the number of outfalls being proportional to the size of the city (Binghamton, New York: 10, Harrisburg, Pennsylvania: 65, Scranton, Pennsylvania: 70). As a result, the Susquehanna River contributes 44% of the nitrogen and 21% of the phosphorous to the Chesapeake Bay. This has led to large algal blooms in the bay and a resulting "dead zone" between Annapolis, Maryland and Newport News, Virginia. In 2005, the Susquehanna River was named America's most endangered river by American Rivers, who produces an annual list. Even 35 years after the Clean Water Act, there are still elevated levels of copper, sulfur, selenium, arsenic, cobalt, chromium, lead, mercury, zinc, and pesticides (Beyer and Day 2004) as well as depressed pH associated with abandoned mines in the watershed (Hoffman 2008). Excessive nutrient and sediment loads also significantly impair the Susquehanna, stemming from urban and agricultural runoff and sewage treatment discharge (Hoffman 2008). Although water quality has significantly improved in the Potomac River over the past 50 years, the river remains threatened by elevated amounts of cadmium, chromium, copper, lead, dioxin, PCBs, and chlordane, which may have resulted in recent highly publicized reports of male fish producing eggs.

Similarly, the James River has elevated levels of zinc, copper, cadmium, nickel, chromium, lead, arsenic, dioxin, PCBs, and pesticides. The James River was also the site of one of the nation's most publicized pollution events when a manufacturing plant discharged, for nearly ten years, the chlorinated insecticide Kepone in the lower river (Smock et al. 2005). The insecticide is bioaccumulated, and resulted in a ban on commercial fishing in the lower river. Although the ban has been lifted, accumulations of Kepone in the riverine sediments are still cause for concern (Smock et al. 2005).

Table 30. Land uses and population density in several watersheds that drain to Chesapeake Bay

Watershed	Land Use Categories (%)				Density (people/mi^2)
	Agriculture	Forested	Urban	Other	
Susquehanna	27	63	9	---	145
Potomac	32	58	5	6	358
James	23	71	6	---	249

Data from Jackson et al. 2005; Smock et al. 2005

Hydromodification Projects. The Chesapeake Bay is home to several moderate to small sized dam projects. While only a few impoundments in the Potomac River and its tributaries are larger than 1.5 square miles, the Susquehanna River has over 100 dams along the mainstem and the first major dam is located just 10 miles upstream of the mouth. The Anacostia River, a major tributary to the Potomac River is scheduled to have some 60 dams removed or altered to improve water quality and fish passage. Dams in other basins have also been upgraded or are planned for upgrades. For instance, between 1928 and 1972, no shad passed Conowingo Dam on the Susquehanna River, 10 miles upstream of the mouth, but since passage facilities were installed fish abundance has increased from approximately 100 to more than 100,000 individuals. Similarly, many dams have been improved or removed in the James River. In 1999, a fish ladder added to Boscher Dam, which had prevented upstream fish runs since 1823 provided access to 137 additional miles of the James River and 168 miles of its tributaries.

Mining. In the Chesapeake Bay watershed, coal mining has likely had the most significant impact on water quality. Coal waste and acid mine drainage damaged much of the river and its tributaries. There was so much coal silt in the Susquehanna at one point that a fleet of over 200 vessels began harvesting the silt from the river's bed. From 1920 to 1950, over 3 million tons of coal was harvested from behind one dam. Later, between 1951 and 1973, over 10 million tons were harvested from behind another dam. Mining in this watershed was so extensive that while many mines have been reclaimed and others are currently being reclaimed, at the current level of funding, it will take decades to completely reclaim all of the old mines in the watershed. Abandoned coal mines leach sulfuric acid as a result of natural reactions with the chemicals found in coal mines. Much of the Appalachian Mountain chain that was mined for coal is now leaching sulfuric acid into tributaries of the Chesapeake Bay and requires some sort of treatment to improve the water quality of the region. Many of these abandoned coal mines must be treated with doses of limestone to balance the pH of the water draining from the mines. Coal is abundant through the watershed, amounting to nearly 30 billion tons of coal mined. Coal is no longer a primary industry in the watershed, but the impacts of the acid mine drainage are still prominent.

Commercial and Recreational Fishing. The Chesapeake Bay supports fisheries for American eel, croaker, blue crab, black sea bass, bluefish, oyster, red drum, spot, striped bass, summer flounder, weakfish, menhaden, and white perch (CFEPTAP 2004). Populations of striped bass got so low in the mid 1980s that a moratorium started in 1985, but they recovered so well that well-regulated harvests are now permitted. Since the mid 1990s, levels of blue crab and menhaden have dropped to the lowest levels in history. Species such as catfish and white perch are year round residents and managed by individual states around the bay. Species like Spanish mackerel, king mackerel, red drum, and summer flounder have ranges that extend beyond the bay

and are managed under multiple regional management plans. Some species such as American shad are allowed to be fished by some states (Virginia and Maryland) within the Chesapeake Bay, but not by other states (Delaware and Pennsylvania).

Atlantic Southeast Region

This region covers all drainages that ultimately drain to the Atlantic Ocean (South Carolina and parts of Georgia, North Carolina, Florida, and Virginia). The region encompasses three ecoregions—the hot continental division, subtropical division, and savanna division (southern most tip of Florida's panhandle). The hot continental division is characterized by its winter deciduous forests dominated by tall broadleaf trees, soils rich in humus and moderately leached (Inceptisols, Ultisols, and Alfisols), and rainfall totals that decrease with distance from the Atlantic Ocean (Bailey 1995).

Most of the Atlantic Southeast Region is contained within the subtropical ecoregion and is characterized by a humid subtropical climate with particularly high humidity during summer months, and warm mild winters. Soils are strongly leached and rich in oxides of iron and aluminum (Bailey 1995). The subtropical ecoregion is forested, largely by second growth forests of longleaf, loblolly and slash pines, with inland areas dominated by deciduous trees. Rainfall is moderate to heavy with annual averages of about 40 inches in the north, decreasing slightly in the central portion of the region, and increasing to 64 inches in southern Florida. The savanna ecoregion has a tropical wet-dry climate, controlled by moist warm topical air masses and supports flora and fauna that is adapted to fluctuating water levels (Bailey 1995).

In the sections that follow we describe several basins and estuaries to characterize the general ecology and natural history of the area, and past and current human activities and their impacts on the area. The region contains more than 22 river systems that generally flow in a southeasterly direction to the Atlantic Ocean. The diverse geology and climate ensures variability in biological productivity and hydrology. Major basins include the Albemarle-Pamlico watershed and its tributaries, the Cape Fear River, Winyah Bay and the Santee-Cooper Systems, the Savannah, Ogeechee, and the St. Johns Rivers. The more northern river, the Roanoke, which is part of the Albemarle-Pamlico watershed, is cooler and has a higher gradient and a streambed largely characterized by cobble, gravel and bedrock.

The southern rivers are characterized by larger portions of low gradient reaches, and streambeds that are composed of greater amounts of sand and fine sediments—are often high in suspended solids, and have neutral to slightly acidic waters with high concentrations of dissolved organic carbon. Rivers emanating entirely within the Coastal Plain are acidic, low alkalinity, blackwater systems with dissolved organic carbon concentrations often up to 50 mg/L (Smock et al. 2005). We describe several river basins in detail to provide additional context for evaluating the influence of the environmental baseline on listed species under NMFS' jurisdiction and the health of their environment.

Albemarle-Pamlico Sound Complex

Natural History

The Albemarle-Pamlico Sound Estuarine Complex, the largest lagoonal estuarine system in the United States, includes seven sounds including Currituck Sound, Albemarle Sound, Pamlico Sound and others (EPA 2006). The Estuarine Complex is separated from the Atlantic Ocean by the Outer Banks, a long barrier peninsula, and is characterized by shallow waters and wind-driven tides that result in variable patterns of water circulation and salinity. Estuarine habitats include salt marshes, hardwood swamp forests, and bald cypress swamps.

The Albemarle-Pamlico watershed encompasses four physiographic regions—the Valley and Ridge, Blue Ridge, Piedmont and Costal Plain Provinces. Basin geology strongly influences the water quality and quantity within the basin. The headwaters of the basin tributaries are generally steep and surface water flowing downstream has less opportunity to pick up dissolved minerals. As the surface water flows reaches the Piedmont and Coastal Plain, water velocity slows due to the low gradient and streams generally pick up two to three times the mineral content of surface waters in the mountains (Spruill et al. 1998). At the same time, much of the upper watershed is composed of fractured rock overlain by unconsolidated and partially consolidated sands. As a result more than half of the water flowing in streams discharging to the Albemarle-Pamlico Estuarine Complex comes from ground water.

Primary freshwater inputs to the estuary complex include the Pasquotank, Chowan and Roanoke rivers that flow into Albemarle Sound, and the Tar-Pamlico and Neuse rivers that flow into Pamlico Sound. The Roanoke River is approximately 410 miles long and drains a watershed of 9,580 square miles. The Roanoke River begins in the mountains of western Virginia and flows across the North Carolina border before entering Albemarle Sound. The upper Roanoke River's geology is primarily a high gradient boulder-rubble bedrock system. The middle Roanoke River is primarily course sand and gravel. The lower section of the river is almost entirely organic-rich mud. The average annual precipitation is approximately 43 inches. At the mouth, the average discharge is 5.3 billion gallons per day, or 8,193 cubic feet per second (Smock et al. 2005). The Roanoke River is home to 119 fish species, and only seven of those are not native to the area (Smock et al. 2005). The Roanoke is also home to nine endangered fish species, two amphibians, and seven mussels, including several important anadromous fish species.

The Neuse River is 248 miles long and has a watershed of 6,235 square miles (Smock et al. 2005). The Neuse River watershed is also located entirely within the state of North Carolina, flowing through the same habitat as the Cape Fear River, but ultimately entering Pamlico Sound. The river originates in weathered crystalline rocks of the Piedmont and crosses sandstone, shale, and limestone before entering Pamlico Sound (Turekian et al. 1967). The average annual precipitation is approximately 48 inches. At the mouth, the average discharge is 3.4 billion gallons per day, or 5,297 cubic feet per second (USGS 2005).

Human Activities and Their Impacts

Land Use. Land use in the Roanoke River is dominated by forest (68%) and the basin contains some of the largest intact, least disturbed bottomland forest floodplains along the eastern coast.

Three percent of the basin qualifies as urban land uses and 25% is used for agriculture (Smock et al. 2005). The only major town in the Roanoke watershed is Roanoke, Virginia and population in the watershed is approximately 80 people per square mile (Smock et al. 2005). In contrast, the Neuse River watershed is described as 35% agriculture, 34% forested, 20% wetlands, 5% urban, and 6% other, with a basin-wide density of approximately 186 people per square mile (Smock et al. 2005). While the population has increased in the Albemarle-Pamlico Complex more than 70% during the last 40 years, the rate of growth is relatively low for many coastal counties in the Southeast (EPA 2006). Much of the estuarine complex is protected by large tracts of state and federally protected lands, which may reduce development pressures.

Coal is mined from the mountainous headwaters of the Roanoke River in southwestern Virginia. Mining through the Piedmont and coastal areas of North Carolina was conducted for limestone, lead, zinc, titanium, apatite, phosphate, crushed stone, sand, and fossils. Many active mines in these watersheds are still in operation today. These mines contribute to increased erosion, reduced pH, and leached heavy metals.

Agricultural activities are major source of nutrients to the estuary and a contributor to the harmful algal blooms (HABs) in summer, although according to (McMahon and Woodside 1997 as cited in EPA 2006) nearly one-third of the total nitrogen inputs and one-fourth of the total phosphorus input to the estuary are from atmospheric sources. Primary agricultural activities within the watershed include corn, soybean, cotton, peanut, tobacco, grain, potato, and the production of chicken, hog, turkey, and cattle.

The Neuse River entered the national spotlight during the early 1990s due to massive and frequent fish kills within the basin. Over one billion American shad have died in the Neuse River since 1991. The problem is persistent but the cause of the kills differs among events; in 2004 more than 700,000 estuarine fish died and more than 5,000 freshwater fish died within the basin. Freshwater species most commonly identified during investigations included sunfishes, shad, and carp, while estuarine species most commonly reported included menhaden, perch, and croaker. Atlantic menhaden have historically been involved in a majority of estuarine kill events and have exhibited stress and disease in conjunction with fish kills. Fish kill events may often have different causative agents, and in many cases the precise cause is not clear, but high levels of nutrients, HABs, toxic spills, outbreaks of a marine organism, *Pfiesteria pescicida*, low dissolved oxygen concentrations and sudden wind changes that mix hypoxic waters, are some of contributing factors or causes to the basins persistent fish kills (NCDWQ 2004).

Both the Roanoke River and the Neuse River are fragmented by dams. The reservoirs are used for flood control and recreation, but the amount of agricultural and urban runoff that collects behind the dams has caused sanitation problems in the recent past. Three dams were removed recently in an effort to improve environmental conditions and fish passage. Widespread stream modification and bank erosion were rated high within the greater watershed relative to other sites nationally (Spruill et al. 1998).

Conditions within the Albemarle-Pamlico estuary system are relative good compared to other northeastern estuaries. Agricultural and urban runoff provide the majority of toxins to the region that can impair water and habitat quality, as well as degrade fishery resource quality and quantity,

including Atlantic sturgeon and numerous sport and commercially important fish species (EPA 2006). Chlorophyll *a* is the most significant detractor to water quality and total organic carbon has the greatest impact on sediment quality. Benthic quality and fish tissue contamination (PAHs and PCBs) also have suffered from human-introduced toxins. Losses of 25 to 50% of wetlands surrounding tributaries have lead to significant reduction in habitat as a result of human development.

Commercial and Recreational Fishing. The Albemarle and Pamlico Sounds and associated rivers support a dockside commercial fishery valued at over $54 million annually. The commercial harvest includes blue crabs, southern flounder, striped bass, striped mullet, white perch, croaker, and spot, among others. Roughly 100 species are fished commercially or recreationally in the region. The Neuse River supports many of the same species as the Roanoke River. Commercial and recreational fisheries exist for oyster, crab, clam, American shad, American eel, shrimp, and many other species. Shellfish can be collected by dredging, which has adverse effects to benthic organisms, including shortnose sturgeon that use estuarine areas for feeding. Commercial fisheries along the South Carolina coast use channel nets, fyke nets, gillnets, seines, and trawls. All of these methods can accidentally capture a shortnose sturgeon.

Major Southeast Coastal Plains Basins

Natural History

More than five major river basins flow through the Coastal Plains of the Southeast and directly enter the Atlantic Ocean, including the Cape Fear, Great Pee-Dee, Altamaha, and the St. Johns rivers (see Table 28 for a description of several basins within this region). Rainfall is abundant in the region and temperatures are generally warm throughout the year. Northern rivers originate in the Blue Ridge Mountains or the Piedmont Plateau, but all the rivers described in this section have sizeable reaches of slack water as they flow through the flat Coastal Plain. Two rivers, the Satilla in Georgia and the St. Johns in Florida, are located entirely within the Coastal Plain. The highest elevation of the St. Johns River is 26 feet above sea level, so the change in elevation is essentially one inch every mile, making it one of the most gradually flowing rivers in the country.

Smock et al. (2005) described the mountains and plateau as heavily dissected and highly metamorphosed rock of Paleozoic age, with occasional areas of igneous and sedimentary rock. Underlying rock is varied with bands of limestone, dolomite, shale, sandstone, cherts, and marble, with a number of springs and caves scattered throughout the area. At the fall line, where the Piedmont Plateau meets the sedimentary deposits of the Coastal Plain, steep changes in elevation result in rapids or falls before the rivers level off in their Coastal Plain reaches. Here, soils are acidic with a low cation exchange capacity and a sandy or loamy surface horizon, and a loamy or clay subsurface. The acidic characteristics, slow flowing water with poor flushing and high organic and mineral inputs gives these waters their characteristic blackwater (or brownwater, for those rivers that originate in the Piemont Plateau) appearance. The Satilla River is a blackwater river that has a naturally low pH (between four and six) and white sandbars. Due to the low pH, it also has naturally lower productivity than other rivers that originate within the mountains or the plateau.

Table 31. Rivers of the Southeast United States

Watershed	Approx Length (mi)	Basin Size (mi^2)	Physiographic Provinces*	Mean Annual Precip (in)	Mean Discharge (cfs)	No. Fish Species	Number of Endangered Species
Cape Fear	320	9,324	PP, CP	47	7,663	95	8 fish, 1 mammal, 15 mussels
Great Pee Dee	430	10,641	BR, PP, CP	44	13,102	>100	6 fish, 1 reptile
Santee-Cooper	440	15,251	BR, PP, CP	50	15,327	>100	5 fish, 2 reptiles
Savannah	300	10,585	BR, PP, CP	45	11,265	>100	7 fish, 4 amphibians, 2 reptiles, 8 mussels, 3 crayfish
Ogeechee	250	5,212	PP, CP	44	4,061	>80	6 fish, 2 amphibians, 2 reptiles, 1 mussel
Altamaha	140	14,517	PP, CP	51	13,879	93	1 mammal, 12 fish, 2 amphibians, 2 reptiles, 7 mussels, 1 crayfish
Satilla	200	3,530	CP	50	2,295	52	2 fish, 1 amphibian, 2 reptiles, 1 mussel
St. Johns	311	8,702	CP	52	7,840	>150	1 mammal, 4 fish, 2 reptiles, 2 birds

Data from NCDENR 1999; Smock et al. 2005

*Physiographic Provinces: BR = Blue Ridge, PP = Piedmont Plateau, CP = Coastal Plain

Human Activities and Their Impacts

Land Use. Across this region, land use is dominated by agriculture and industry, and to a lesser extent timber and paper production, although more than half of most basins remain forested. Basin population density is highly variable throughout the region with the greatest density in the St. Johns River watershed with about 200 people per square mile of catchment, most of whom are located near Jacksonville, Florida. In contrast, there are only 29 people per square mile in the Saltilla River watershed in Georgia (Smock et al. 2005). See Table 29 for a summary of land uses and population densities in several area basins across the region (data from Smock et al. 2005).

The largest population centers in the region include Miami and Jacksonville, Florida and Savannah, Georgia. Major towns include Greensboro, Chapel Hill, Fayetteville, and Wilmington, North Carolina in the Cape Fear River watershed; Winston-Salem, North Carolina and Georgetown, Florence, and Sumter, South Carolina in the Great Pee-Dee River Watershed; Charlotte, Hickory, and Gastonia, North Carolina and Greenville and Columbia, South Carolina in the Santee-Cooper River watershed; Savannah and Augusta, Georgia, in the Savannah River watershed; Louisville, Statesboro, and Savannah, Georgia, in the Ogeechee River watershed; Athens and Atlanta, Georgia, in the Altamaha River watershed; and Jacksonville, Florida in the St. Johns River watershed.

Several of the rivers in the region have elevated levels of metals including mercury, fecal coliform, ammonia, turbidity, and low dissolved oxygen. These impairments are caused by

municipal sewage overflows, mining, non-point source pollution, waterfowl, urban runoff, marinas, agriculture, and industries including textile manufacturing, power plant operations, paper mills, and chemical plants (Mehta 2008; Harned and Meyer 1983; Berndt et al. 1998; NCDENR 1998; Smock et al. 2005).

Several watersheds exhibit high nitrogen loads including the Cape Fear River, Winyah Bay, Charleston Harbor, St. Helena Sound, Savannah River, Ossabaw Sound, Altamaha River, and St. Mary's River and Cumberland Sound (Bricker et al. 2007). Nitrate concentrations (as nitrogen) tend to be higher in stream draining basins with agricultural and mixed land uses (Berndt et al. 1998). Based on studies in Georgia, however, nitrate loads did not vary with growing season of crops (periods of heaviest fertilizer application), but were influenced by high stream flow, which could be related to downstream transport by subsurface flows (Berndt et al. 1998).

Table 32. Land uses and population density in several Atlantic southeast basins

Watershed	Land Use Categories (%)				Density (people/mi^2)
	Agriculture	Forested	Urban	Other	
Cape Fear River	24	56	9	11	80
The Great Pee-Dee	28	58	8	6	127
Santee-Cooper River	26	64	6	4	168
Savannah River	22	65	4	9	91
Ogeechee River	18	54	1	17 (wetlands)	78
Altamaha River	--	64	3	7	73
Satilla River	26	72	1	1	29
St. Johns River	25	45	6	24 (wetlands & water)	202

Data from Smock et al. 2005

Sediment is the most serious pollutant in the Yadkin (Pee-Dee) River and has historically been blamed on agricultural runoff. In the mid 1990s, farmers in the region began using soil conservation techniques that have reduced sediment inputs by 77%. The reduction in sediment inputs from farms did not translate to a reduction in sediment in the river, and during this period there was a 25% reduction in agricultural land and a 38% increase in urban development.

Where data are available, estuaries throughout the region contain generally moderate to severe nitrogen loads from river systems (Bricker et al. 2007). This has lead to toxic blooms of algae in some areas. Eutrophication has been noted particularly in the St. Johns River region. Low dissolved oxygen levels have also been found in the area around the Savannah River.

Mining. Mining occurs throughout the region. South Carolina is ranked 25th in terms of mineral value and 13th among the eastern 26 states, and produces 1% of the total nonfuel mineral production value in the United States. There are currently 13 minerals being extracted from 485 active mines in South Carolina alone. Portland and masonry cement and crushed stone were South Carolina's leading nonfuel minerals in 2004 (NMA 2007). In contrast, Georgia accounts for 4%, Florida accounts for 5%, and North Carolina accounts for about 2% of the total non-fuel mineral production value in the United States. North Carolina's leading nonfuel minerals in 2004 were crushed stone, phosphate rock, and construction sand and gravel. Georgia produces 24% of the clay in the nation; other leading nonfuel minerals include crushed stone and Portland cement. Florida is the top phosphate rock mining state in the United States and produces about

six times more than any other state in the nation. Peat and zirconium concentrates are also produced in Florida.

The first gold mine operated in the United States is outside Charlotte, North Carolina in the Pee Dee watershed. Mines through Georgia are also major producers of barite and crude mica, iron oxide, and feldspar. There is a proposed titanium mine near the mouth of the Satilla River. Mines release toxic materials that negatively affect fish, as fish living around dredge tailings have elevated levels of mercury and selenium.

Hydromodification Projects. Several area rivers have been modified by dams and impoundments. In contrast to rivers along the Pacific Coast, considerable less information is available on other types of hydromodification projects in this area, such as levees and channelization projects. There are three locks and dams along the mainstem Cape Fear River and a large impoundment on the Haw River. The lower river and its tributaries are relatively undisturbed. The lower reach is naturally a blackwater river with naturally low dissolved oxygen, which is compounded by the reduced flow and stratification caused by upstream reservoirs and dams. The Yadkin (Pee Dee) River is heavily utilized for hydroelectric power. There are numerous dams on Santee-Cooper River System. The Santee River Dam forms Lake Marion and diverts the Santee River to the Cooper River, where another dam, St. Stephen Dam, regulates the outflow of the Santee River. Lake Moultrie is formed by both St. Stephen Dam and Pinopolis Dam, which regulates the flow of the Cooper River to the Atlantic Ocean. Below the fall line, the Savannah River is free-flowing with a meandering course, but above the fall line, there are three large dams that turn the Piedmont section of the river into a 100-mile long reservoir. Although the Altamaha River is undammed, hydropower dams are located on its tributaries, the Oconee and Ocmulgee rivers, above the fall lines. There are no dams along the entire mainstem Satilla River. There are no major dams on the mainstem St. Johns River either, but one of the largest tributaries has a dam on it. The St. Johns River's flow is altered by water diversions for drinking water and agriculture.

Commercial and Recreational Fishing. The region is home to many commercial fisheries targeting shrimp, blue crab, clams, American and hickory shad, oysters, whelks, scallops, channel catfish, flathead catfish, snapper, and grouper. Shortnose sturgeon can be caught in gillnets, but gillnets and purse seines account for less than 2% of the annual bycatch. Shrimpers are responsible for 50% of all bycatch in Georgia waters. There are approximately 1.15 million recreational anglers in the state as well.

Southwest Coast Region

The basins described in this section are encompassed by the State of California and parts of Oregon. Select watersheds described herein characterize the general ecology and natural history of the area, and the past, present and future human activities and their impacts on the area. Essentially, this region encompasses all Pacific Coast rivers south of Cape Blanco, California through southern California. The Cape Blanco area marks a major biogeographic boundary and has been identified by NMFS as a DPS/ESU boundary for Chinook and coho salmon, and steelhead on the basis of strong genetic, life history, ecological and habitat differences north and south of this landmark. Major rivers contained in this grouping of watersheds are the

Sacramento, San Joaquin, Salinas, Klamath, Russian, Santa Ana and Santa Margarita Rivers (see Table 30).

Table 33. Select rivers in the southwest coast region

Watershed	Approx Length (mi)	Basin Size (mi^2)	Physiographic Provinces*	Mean Annual Precipitation (in)	Mean Discharge (cfs)	No. Fish Species (native)	No. Endangered Species
Rogue River	211	5,154	CS, PB	38	10,065	23 (14)	11
Klamath River	287	15,679	PB, B/R, CS	33	17,693	48 (30)	41
Eel River	200	3,651	PB	52	7,416	25 (15)	12
Russian River	110	1,439	PB	41	2,331	41 (20)	43
Sacramento River	400	27,850	PB, CS, B/R	35	23,202	69 (29)	>50 T & E spp.
San Joaquin River	348	83,409	PB, CS	49	4,662	63	>50 T & E spp.
Salinas River	179	4,241	PB	14	448	36 (16)	42 T & E spp.
Santa Ana River	110	2,438	PB	13	60	45 (9)	54
Santa Margarita River	27	1,896	LC, PB	49.5	42	17 (6)	52

Data from Carter and Resh 2005
*Physiographic Provinces: PB = Pacific Border, CS = Cascades-Sierra Nevada Range, B/R = Basin & Range.

Natural History

The physiographic regions covered by the basins discussed herein include: (a) the Cascade-Sierra Nevada Mountains province, which extends beyond this region as we have defined it and continue north into British Columbia, (b) the Pacific Border province, and (c) the Lower California province (Carter and Resh 2005). The broader ecoregions division, as defined by Bailey (1995) is the Mediterranean Division. Three major vegetation types are encompassed by this region: the temperate coniferous forest, the Mediterranean shrub and savannah, and the temperate grasslands/savannah/shrub. The area, once dominated by native grasses, is naturally prone to fires caused by lightening during the dry season (Bailey 1995).

This region is the most geologically young and tectonically active region in North America. The Coast Range Mountains are folded and faulted formations, with a variety of soil types and nutrients that influence the hydrology and biology of the individual basins (Carter and Resh 2005). The region also covers the Klamath Mountains and the Sierra Nevada Range.

The climate is defined by hot dry summers and wet, mild winters, with precipitation generally decreasing in southern latitudes although precipitation is strongly influences by topography and generally increases with elevation. Annual precipitation varies from less than 10 inches to more than 50 inches in the region. In the Sierra Nevada about 50% of the precipitation occurs as snow (Carter and Resh 2005), as a result snowmelt strongly influences hydrological patterns in the area. Severe seasonal patterns of flooding and drought and high interannual variation in total precipitation makes the general hydrological pattern highly unpredictable within a basin, but consistant across years (Carter and Resh 2005). According to Carter and Resh (2005) this likely increases the variability in the annual composition of the fish assemblies in the region.

The San Joaquin River, draining the largest basin in the region, originates within the Sierra

Nevada Range near central California and flows in a northwesterly direction through the southern portion of the Central Valley. The alluvial fan of the Kings River separates the San Joaquin River from the Tulare River basin.

Human Activities and Their Impacts

Land Use. Land use is dominated by forest (and vacant land) in northern basins, and grass, shrubland, and urban uses dominate in southern basins (see Table 31). Overall, the most developed watersheds are the Santa Ana, Russian, and Santa Margarita rivers. The Santa Ana watershed encompasses portions of San Bernardino, Los Angeles, Riverside, and Orange counties. About 50% of coastal subbasin of the Santa Ana watershed is dominated by urban land uses and the population density is about 1,500 people per square mile. When steep and unbuildable lands are excluded from this area, then the population density in the watershed is 3,000 people per square mile. The most densely populated portion of the basin is near the City of Santa Ana, where density reaches 20,000 people per square mile (Burton 1998; Belitz et al. 2004). The basin is home to nearly 5 million people and the population is projected to increase two-fold in the next 50 years (Burton 1998; Belitz et al. 2004).

Table 34. Land uses and population density in several basins of the southwest coast region

Watershed	Land Use Categories (%)				Density (people/mi^2)
	Agriculture	Forest	Urban	Other	
Rogue River	6	83	<1	9 grass & shrub	32
Klamath River	6	66	<1	24 grass, shrub, wetland	5
Eel River	2	65	<1	31 grass & shrub	9
Russian River	14	50	3	31 (23 grassland)	162
Sacramento River	15	49	2	30 grass & shrub	61
San Joaquin River	30	27	2	36 grass & shrub	76
Salinas River	13	17	1	65 (49 grassland)	26
Santa Ana River	11	57	32	---	865
Santa Margarita River	12	11	3	71 grass & shrub	135

Data from Carter and Resh 2005

Not only is the Santa Ana watershed the most heavily developed watershed in the region, the Santa Ana is the most heavily populated study site out of more than 50 assessment sites studied across the nation by the United States Geological Survey (USGS) under the National Water-Quality Assessment (NAWQA) Program. Water quality and quantity in the basin reflects the influence of the high level of urbanization. For instance, the primary source of baseflow to the river is the treated wastewater effluent; secondary sources that influence peak flows include stormwater runoff from urban, agricultural, and undeveloped lands (Belitz et al. 2004). Concentrations of nitrates and pesticides are elevated within the basin, and were more frequently detected than in other national NAWQA sites (Leenheer et al. 2008; Kent et al. 2005; Belitz et al. 2004). Belitz et al. (2004) found that total nitrogen concentrations commonly exceeded 3 mg/L in the Santa Ana basin. In other NAWQA basins with elevated total nitrogen concentrations across the country, the primary influencing factor was the level of agriculture and the application of manure and pesticides within the basin. In the Santa Ana basin the elevated nitrogen is attributed largely to the wastewater treatment plants, where downstream reaches consistently exceeding 3 mg/L total nitrogen. Samples of total nitrogen taken upstream of the wastewater

treatment plants were commonly below 2 mg/L (Belitz et al. 2004). Other contaminants detected at high levels included volatile organic compounds (VOCs; including chloroform, which sometimes exceeded water quality standards), pesticides (diuron, diazinon, carbaryl, chlophyrifos, lindane, malathion, and chlorothalonil), and trace elements (lead, zinc, and arsenic). As a result of the changes, the biological community in the basin is heavily altered (Belitz et al. 2004).

In many basins, agriculture is the major water user and the major source of water pollution to surface waters. In 1990, nearly 95% of the water diverted from the San Joaquin River was diverted for agriculture, and 1.5% diverted for livestock (Carter and Resh 2005). During the same period, Fresno, Kern, Tulare, and Kings counties ranked top in the nation for nitrogen fertilizer use. Nitrogen fertilizer use increased 500% and phosphorus use increased 285% in the San Joaquin River basin over a 40-year period (Knatzer and Sheton 1998 *in* Carter and Resh 2005). A study conducted by USGS in the mid-1990s on water quality within San Joaquin River basin detected 49 pesticides in the mainstem and three subbasins; 22 pesticides were detected in 20% of the samples and concentrations of seven exceeded water quality standards (Dubrovsky et al. 1998). Water chemistry in the Salinas River is strongly influenced by intensive agriculture; water hardness, alkalinity, nutrients and conductivity are high in areas where agricultural uses predominate.

Estuary systems of the region are consistently exposed to anthropogenic pressures stemming from high human density sources. As an example, the largest west coast estuary, the San Francisco Estuary, provides drinking water to 23 million people, irrigates 4.5 million acres of farmland, and drains roughly 40% of California's land area. As a result of high use, many environmental measures of the estuary are poor. Water quality suffers from high phosphorus and nitrogen loads, primarily from agricultural, sewage, and storm water runoff. Water clarity is also compromised. Sediments contain high levels of the contaminants PCB, pesticides, mercury, copper, and silver from urban runoff and historical activities. As these persist in the marine environment, the estuary system will likely carry loads for years to come, even with strict regulation or banning. Gold mining has reduced estuary depths in much of the region, causing drastic changes to habitat. Large urban centers are foci for contaminants and levels near San Francisco, Oakland, and San Jose are highest and are also where water clarity tends to be at its worst. These water and sediment quality characteristics biomagnify into the food chain; fish tissues contain high levels of particularly PCB and mercury, the former being concentrated 10 times more than human health guidelines for consumption. Birds, some of whom are endangered (clapper rail and least tern), have also concentrated these toxins.

Invasive species have become an increasingly recognized issue. Giant reeds have displaced native marsh species in many areas. Marine invasive species include the green crab, shimofuri goby, Asiatic clams, and zooplankton; these species are cited in reducing the abundance of local species. The Asian clam has become the dominant infaunal species and has likely reduced primary production in the estuary system (Nichols et al. 1990; Ray 2005).

Red tide significantly affects the California coastline. Here, poisoning and mortality of California sea lions, fish, and birds have been recorded, the most recent of which was in 2007 (Chea 2007). California red tide events are correlated with El Niño oscillations. In addition to

the toxin produced by red tide diatoms, a pathogen associated with cholera has been identified in California red tide blooms (Mouriño-Pérez et al. 2003).

Hydromodification Projects. Several of the rivers within the area have been modified by dams, water diversions, and drainage systems for agriculture and drinking water, and some of the most drastic channelization projects in the nation. In all, there are about 1,400 dams within the State of California, more than 5,000 miles of levees, and more than 140 aqueducts (Mount 1995 *in* Carter and Resh 2005). While about 75% of the runoff occurs in basins in the northern half of California, 80% of the water demand is in the southern half. Two water diversion projects meet these demands—the Federal Central Valley Project and the California State Water Project. The Central Valley Project, one of the world's largest water storage and transport systems, has more than 20 reservoirs and delivers about 7 million acre-feet per year to southern California. The State Water Project has 20 major reservoirs and holds nearly 6 million acre-feet of water, delivering about 3 million acre feet. Together these diversions irrigate about 4 million acres of farmland and deliver drinking water to roughly 22 million residents and growing.

Both the Sacramento and San Joaquin rivers are heavily modified, each with hundreds of dams. In 2009, the Sacramento-San Joaquin river system was named America's most endangered river by American Rivers. In the prior year, the Rogue River was listed as the second most endangered river. The Rogue, Russian, and Santa Ana rivers each have more than 50 dams, and the Eel, Salinas and the Klamath rivers have between 14 and 24 dams each. The Santa Margarita, considered one the last free flowing rivers in coastal southern California has nine dams in its watershed. All major tributaries of the San Joaquin River are impounded at least once and most have multiple dams or diversions. The Stanislaus River, a tributary of the San Joaquin River, has over 40 dams. As a result, the hydrograph of the San Joaquin River is seriously altered from its natural state, and the temperature regime and sediment transport regime are altered. Such changes have had profound influences on the biological community within the basin. These modifications generally result in a reduction of suitable habitat for native species and frequent concomitant increases in suitable habitat for nonnative species. The Friant Dam on the San Joaquin River is attributed with the extirpation of spring-run Chinook salmon within the basin, a run once estimated as producing 300,000 to 500,000 fish (Carter and Resh 2005).

Mining. Famous for the gold rush of the mid 1800s, California has a long history of mining. In 2004, California ranked top in the nation for nonfuel mineral production with 8.23% of total production (NMA 2007). Today, gold, silver, and iron ore comprise only 1% of the production value. Primary minerals include construction sand and gravel, cement, boron and crushed stone. California is the only state to produce boron, rare-earth metals, and asbestos (NMA 2007).

California contains some 1,500 abandoned mines and roughly 1% are suspected of discharging metal-rich waters in the basins. The Iron Metal Mine in the Sacramento Basin releases more than 1,100 pounds of copper and more than 770 pounds of zinc to the Keswick Reservoir below Shasta Dam, as well as elevated levels of lead (Cain et al. 2000 *in* Carter and Resh 2005). Metal contamination seriously reduces the biological productivity within a basin and can result in fish kills at high levels or sublethal effects at low levels, including reduced feeding, overall activity levels, and growth. The Sacramento Basin and the San Francisco Bay watershed is one of the most heavily affected basins within the state from mining activities, largely because the basin

drains some of the most productive mineral deposits in the region. Methylmercury contamination within San Francisco Bay, the result of 19th century mining practices using mercury to amalgamate gold in the Sierra Nevada Mountains, remains a persistent problem today. Based on sediment cores, we know that pre-mining concentrations were about five times lower than concentrations detected within San Francisco Bay today (Conaway et al. 2003 in EPA 2006).

Commercial and Recreational Fishing. The region is home to many commercial fisheries. The largest in terms of total landings in 2006 were northern anchovy, Pacific sardine, Chinook salmon, sablefish, Dover sole, Pacific whiting, squid, red sea urchin, and Dungeness crab (CDFG 2007). Red abalone are also harvested. The first salmon cannery established along the west coast was located in the Sacramento River watershed in 1864, but it only operated for about two years because the sediment from hydraulic mining decimated the runs in the basin (NRC 1996).

Pacific Northwest Region

This region encompasses Washington, Oregon, Idaho, and includes parts of Nevada, Montana, Wyoming, and British Columbia. The region is ecologically diverse, encompassing northern marine lowland forests, mountain forests, alpine meadows, and northern desert habitat. In this section we focus on three primary areas that characterize the region, the Columbia River Basin and its tributaries, the Puget Sound Region, and the coastal drainages north of the Columbia River. The broader ecoregion divisions, as defined by Bailey (1995) and encompassed within this region, are the Marine Division, Marine Division – Mountain Provinces, Temperate Steppe Division, Temperate Steppe Division – Mountain Provinces, and portions of the Temperate Desert Davison. Puget Sound and the coastal drainages are contained within the Marine Division, while the Columbia River watershed encompasses portions of all five ecoregions.

Columbia River Basin

Natural History

The most notable basin within the region is the Columbia River. The largest river in the Pacific Northwest and the fourth largest river in terms of average discharge in the United States, it drains over 258,000 square miles, making it the sixth largest in terms of drainage area. The Columbia River Basin includes parts of Washington, Oregon, Nevada, Utah, Idaho, Wyoming, Montana, and British Columbia and encompasses 13 terrestrial and three freshwater ecoregions, including arid shrub-steppes, high desert plateaus, temperate mountain forests, and deep gorges (Kammerer 1990; Hinck et al. 2004; Stanford et al. 2005).

Major tributaries include the Snake, Willamette, Salmon, Flathead, and Yakima Rivers; smaller rivers include the Owyhee, Grande Ronde, Clearwater, Spokane, Methow, Cowlitz, and the John Day Rivers (see Table 32 for a description of select Columbia River tributaries). The Snake River is the largest tributary at more than 1,000 miles long; its headwaters originating in Yellowstone National Park, Wyoming. The second largest tributary is the Willamette River in Oregon (Kammerer 1990; Hinck et al. 2004) and the 19th largest river in the nation in terms of average annual discharge (Kammerer 1990). The basins drain portions of the Rocky Mountains,

Bitteroot Range, and the Cascade Range.

The average annual discharge at the mouth of the Columbia River is 265,000 cubic feet per second (Kammerer 1990). A saltwater wedge extends 23 miles upstream of the mouth with tidal influences extending up to 146 miles up river (Hinck et al. 2004). The climate within the basin is a mix of arid, dry summers, cold winters, and maritime air masses entering from the west. It is not uncommon for air temperatures in the Rocky Mountains to dip below zero in mid-winter, but summer air temperatures can reach more than 100° F in the middle basin.

Table 35. Select tributaries of the Columbia River

Watershed	Approx Length (mi)	Basin Size (mi^2)	Physiographic Provinces*	Mean Annual Precip. (in)	Mean Discharge (cfs)	No. Fish Species (native)	No. Endangered Species
Snake/Salmon	870	108,495	CU, NR, MR, B/R	14	55,267	39 (19)	5 fish (4 T, 1 E), 6 (1 T, 5 E) snails, 1 plant (T)
Yakima	214	6,139	CS, CU	7	3,602	50	2 (T)
Willamette	143	11,478	CS, PB	60	32,384	61 (~31)	5 fish (4 T, 1 E),

Data from Carter and Resh 2005
*Physiographic Provinces: CU = Columbia-Snake River Plateaus, NR = Northern Rocky Mountains, MR = Middle Rocky Mountains, B/R = Basin & Range, CS = Cascade-Sierra Mountains, PB = Pacific Border

The river and estuary were once home to more than 200 distinct runs of Pacific salmon and steelhead with unique adaptations to local environments within a tributary (Stanford et al. 2005). Salmonids within the basin include Chinook, chum, coho, sockeye salmon, steelhead and redband trout, bull trout, and cutthroat trout. Other fish species within the basin include sturgeon, eulachon, lamprey, and sculpin (Wydoski and Whitney 1979). According to a review by Stanford et al. (2005), the basin formerly contained 65 native fish species and at least 53 nonnative fishes. The most abundant non-native fish is the American shad, which was introduced to the basin in the late 1800s (Wydoski and Whitney 1979).

Human Activities and Their Impacts

Land Use. More than 50% of the United States portion of the Columbia River Basin is in Federal ownership (most of which occurs in high desert and mountain areas), 39% is in private land ownership (most of which occurs in river valleys and plateaus), and the remainder is divided among tribes, state, and local governments (Hinck et al. 2004). See Table 33 for a summary of land uses and population densities in several subbasins within the Columbia River watershed (data from Stanford et al. 2005).

Table 36. Land uses and population density in select tributaries of the Columbia River

Watershed	Land Use Categories (%)				Density ($people/mi^2$)
	Agriculture	Forest	Urban	Other	
Snake/Salmon rivers	30	10-15	1	54 scrub/rangeland/barren	39
Yakima River	16	36	1	47 shrub	80
Willamette River	19	68	5	--	171

Data from Stanford et al. 2005

The interior Columbia Basin has been altered substantially by humans causing dramatic changes and declines in native fish populations. In general the basin supports a variety of mixed uses. Predominant human uses include logging, agriculture, ranching, hydroelectric power generation, mining, fishing, a variety of recreational activities, and urban uses. The decline of salmon runs in the Columbia River is attributed to loss of habitat, blocked migratory corridors, altered river flows, pollution, overharvest, and competition from hatchery fish. Critical ecological connectivity (mainstem to tributaries and riparian floodplains) has been disconnected by dams and associated activities such as floodplain deforestation and urbanization. The most productive floodplains of the watershed are either flooded by hydropower dams or dewatered by irrigation diversions. Portions of the basin are also subject to impacts from cattle grazing and irrigation withdrawals. In the Yakima River, 72 stream and river segments are listed as impaired by the Washington Department of Ecology and 83% exceed temperature standards. In the Willamette River, riparian vegetation was greatly reduced by land conversion. By 1990, only 37% of the riparian area within 120 m was forested, 30% was agricultural fields and 16% was urban or suburban lands. In the Flathead River, aquatic invasive plants such as pondweed, hornwort, water milfoil, waterweed, cattail, and duckweed grow in the floodplain wetlands and shallow lakes. In the Yakima River, non-native grasses and other plant are commonly found along the lower reaches of the river (Stanford et al. 2005).

Agriculture and Ranching. Roughly 6% of the annual flow from the Columbia River is diverted for the irrigation of 7.3 million acres of croplands within the basin. The vast majority of these agricultural lands are located along the lower Columbia River, the Willamette, Yakima, Hood, and Snake rivers, and the Columbia Plateau (Hinck et al. 2004). The Yakima River Basin is one of the most agriculturally productive areas in the United States (Fuhrer et al. 2004). Croplands within the Yakima Basin account for about 16% of the total basin area of which 77% is irrigated.

Agriculture and ranching increased steadily within the Columbia River basin from the mid to late 1800. By the early 1900s, agricultural opportunities began increasing at a much more rapid pace with the creation of more irrigation canals and the passage of the Reclamation Act of 1902 (NRC 2004). Today, agriculture represents the largest water user within the basin (>90%). Agriculture, ranching, and related services employ more than nine times the national average (19% of the households within the basin; NRC 2004).

Ranching practices have led to increased soil erosion and sediment loads within adjacent tributaries, the worst of these effects may have occurred in the late 1800s and early 1900s from deliberate burning to increase grass production (NRC 2004). Several measures are in use to reduce the impacts of grazing, including restricting grazing in degraded areas, reduced grazing allotments, and lower stocking rates. Today, agricultural impacts to water quality within the basin are second to large-scale influences of hydromodification projects for both power generation and irrigation. Water quality impacts from agricultural activities include alteration of the natural temperature regime, and insecticide and herbicide contamination, and increased suspended sediments.

The USGS has a number of fixed water quality sampling sites throughout various tributaries of the Columbia River, many of which have been in place for decades. Water volumes, crop rotation patterns, crop-type, and basin location are some of the variables that influence the

distribution and frequency of pesticides within a tributary. Detection frequencies for a particular pesticide can vary widely. One study conducted by the USGS between May 1999 and January 2000 detected 25 pesticide compounds (Ebbert and Embrey 2001). Another study detected at least two pesticides or their breakdown products in 91% of the samples collected, with the median number of chemicals being eight, and a maximum of 26. The herbicide 2,4-D occurred most often in the mixtures, along with azinphos-methyl, the most heavily applied pesticide, and atrazine, one of the most aquatic mobile pesticides (Fuhrer et al. 2004). However, the most frequently detected pesticides in the Yakima River Basin are total DDT, as well as its breakdown products DDE and DDD, and dieldrin (Johnson and Newman 1983; Joy 2002; Joy and Madrone 2002; Furher et al. 2004). In addition to current-use chemicals, these legacy chemicals continue to pose a serious problem to water quality and fish communities despite their ban in the 1970s and 1980s (Hinck et al. 2004).

Fish and macroinvertebrate communities exhibit an almost linear decline in condition as the level of agriculture intensity increases within a basin (Cuffney et al. 1997; Fuhrer et al. 2004). A study conducted in the late 1990s examined 11 species of fish, including anadromous and resident fish collected throughout the Columbia River Basin for a suite of 132 contaminants, including 51 semi-volatile chemicals, 26 pesticides, 18 metals, seven PCBs, 20 dioxins, and 10 furans. The study revealed PCBs, metals, chlorinated dioxins and furans (products of wood pulp bleaching operations) and other contaminants within fish tissues; white sturgeon tissues contained the greatest concentrations of chlorinated dioxins and furans (Hinck et al. 2004).

Urban and Industrial Development. The largest urban area in the basin is the greater Portland metropolitan area, located at the mouth of the Columbia River. Portland's population exceeds 500,000, and the next largest cities, Spokane, Salem, Eugene, and Boise, have over 100,000 people (Hinck et al. 2004). Overall, the basin's population density is one-third the national average, and while the basin covers about 8% of United States land, only about 1.2% of the United States population lives within the basin (Hinck et al. 2004).

Discharges from sewage treatment plants, paper manufacturing, and chemical and metal production represent the top three permitted sources of contaminants within the lower basin according to discharge volumes and concentrations (Rosetta and Borys 1996 in Hinck et al. 2004). According to Rosetta and Borys (1996 in Hinck et al. 2004), based on their review of 1993 data, 52% of the point source waste water discharge volume is from sewage treatment plants, 39% from paper and allied products, 5% from chemical and allied products, and 3% from primary metals (Rosetta and Borys 1996 in Hinck et al. 2004). The paper and allied products industry is the primary source of the suspended sediment load (71%), while 26% comes from sewage treatment plants, and 1% is from the chemical and allied products industry. Non-point source discharges (urban stormwater runoff) account for significant pollutant loading to the lower basin, including most organics and over half of the metals. Although rural non-point sources contributions were not calculated, Rosetta and Borys (1996 in Hinck et al. 2004) surmised that in some areas and for some contaminants, rural areas may contribute a large portion of the load. This is particularly true for pesticide contamination in the upper river basin where agriculture is the predominant land use.

The Columbia River Estuary is under threat from several anthropogenic sources. Habitat loss has

fragmented habitat and human density increase has created additional loads of pollutants and contaminants (EPA 2006). Water quality has been reduced by phosphorus loads and decreased water clarity, primarily along the lower and middle sections of the Columbia River Estuary. Although sediment quality is generally very good, benthic indices have not been established within the estuary, and fish tissue contaminant loads (PCBs, DDT, DDD, DDE, and mercury) are high, presenting a persistent and long lasting effect on estuary biology. Health advisories have been recently issued for people eating fish in the area that contain high levels of dioxins, PCBs, and pesticides. Habitat loss has been significant; 77% of swamps, 57% of marshes, and over 20% of tree cover has been lost to development and industry. Twenty-four threatened and endangered species occur in the estuary, some of whom are recovering and others (i.e., Chinook salmon) are not. Issues surrounding damming and environmental toxins have played key roles in original decline and subsequent recovery of several species and will be vital for future management. Invasive species in the estuary are pervasive; at least 81 have currently been identified, composing one-fifth of all species in some areas, and new species are being identified presently.

Hydromodification Projects. More than 400 dams exist in the basin, ranging from mega dams that store large amounts of water to small diversion dams for irrigation. Every major tributary of the Columbia River except the Salmon River is totally or partially regulated by dams and diversions. More than 150 dams are major hydroelectric projects of which 18 dams are located on mainstem Columbia River and its major tributary, the Snake River. The Federal Columbia River Power System encompasses the operations of 14 major dams and reservoirs on the Columbia and Snake rivers, operated as a coordinated system. The Army Corps of Engineers operates nine of 10 major Federal projects on the Columbia and Snake rivers, and Dworshak, Libby and Albeni Falls dams. The Bureau of Reclamation operates Grand Coulee and Hungry Horse dams. These Federal projects are a major source of power in the region, and provide flood control, navigation, recreation, fish and wildlife, municipal and industrial water supply, and irrigation benefits.

The Bureau of Reclamation has operated irrigation projects within the basin since 1904. The irrigation system delivers water to about 2.9 million acres of agricultural lands; 1.1 million acres of land are irrigated using water delivered by two structures, the Columbia River Project (Grand Coulee Dam) and the Yakima Project. Grand Coulee Dam delivers water for the irrigation of over 670,000 acres of croplands and the Yakima Project delivers water to nearly 500,000 acres (BOR 2007).

The Bonneville Power Administration, an agency under the U.S. Department of Energy, wholesales electric power produced at 31 Federal dams (67% of its production) and non-hydropower facilities in the Columbia-Snake Basin, selling about half the electric power consumed in the Pacific Northwest. The Federal dams were developed over a 37-year period starting in 1938 with Bonneville Dam and Grand Coulee in 1941, and ending with construction of Libby Dam in 1973 and Lower Granite Dam in 1975.

Development of the Pacific Northwest regional hydroelectric power system, dating to the early 20th century, has had profound effects on the ecosystems of the Columbia River Basin (ISG 1996). These effects have been especially adverse to the survival of anadromous salmonids. The

construction of the Federal power system modified migratory habitat of adult and juvenile salmonids, and in many cases presented a complete barrier to habitat access. Both upstream and downstream migrating fish are impeded by the dams, and a substantial number of juvenile salmonids are killed and injured during downstream migrations. Physical injury and direct mortality occurs as juveniles pass through turbines, bypasses, and spillways. Indirect effects of passage through all routes may include disorientation, stress, delays in passage, exposure to high concentrations of dissolved gases, warm water, and increased predation. Dams have also flooded historical spawning and rearing habitat with the creation of massive water storage reservoirs. More than 55% of the Columbia River Basin that was accessible to salmon and steelhead before 1939 has been blocked by large dams (NWPPC 1986). Construction of Grand Coulee Dam blocked 1,000 miles of habitat from migrating salmon and steelhead (Wydoski and Whitney 1979). The mainstem habitats of the lower Columbia and Willamette rivers have been reduced primarily to a single channel. As a result, floodplain area is reduced, off-channel habitat features have been eliminated or disconnected from the main channel, and the amount of large woody debris in the mainstem has been reduced. Remaining areas are affected by flow fluctuations associated with reservoir management for power generation, flood control and irrigation. Overbank flow events, important to habitat diversity, have become rare as a result of controlling peak flows and associated revetments. Consequently, estuary dynamics have changed substantially.

Artificial Propagation. There are several artificial propagation programs for salmon production within the Columbia River Basin, many of which were instituted under Federal law to ameliorate the effects of lost natural salmon production within the basin from the dams. The hatcheries are operated by Federal, state, and tribal managers. For more than 100 years, hatcheries in the Pacific Northwest have been used to produce fish for harvest and replace natural production lost to dam construction, and have only minimally been used to protect and rebuild naturally produced salmonid population (e.g., Redfish Lake sockeye salmon). In 1987, 95% of the coho salmon, 70% of the spring Chinook salmon, 80% of the summer Chinook salmon, 50% of the fall Chinook salmon, and 70% of the steelhead returning to the Columbia River Basin originated in hatcheries (CBFWA 1990). More recent estimates suggest that almost half of the total number of smolts produced in the basin come from hatcheries (Mann et al. 2005).

The impact of artificial propagation on the total production of Pacific salmon and steelhead has been extensive (Hard et al. 1992). Hatchery practices, among other factors, are a contributing factor to the 90% reduction in natural coho salmon runs in the lower Columbia River over the past 30 years (Flagg et al. 1995). Past hatchery and stocking practices have resulted in the transplantation of salmon and steelhead from nonnative basins, and the impacts of these practices are largely unknown. Adverse effects of these practices likely included loss of genetic variability within and among populations (Busack 1990 *in* Hard et al. 1992; Riggs 1990; Reisenbichler 1997), disease transfer, increased competition for food, habitat, or mates, increased predation, altered migration, and displacement of natural fish (Steward and Bjornn 1990; Fresh 1997). Species with extended freshwater residence are likely to face higher risk of domestication, predation, or altered migration than are species that spend only a brief time in fresh water (Hard et al. 1992). Nonetheless, artificial propagation also may contribute to the conservation of listed salmon and steelhead although it is unclear whether or how much artificial propagation during the recovery process will compromise the distinctiveness of natural population (Hard et al. 1992).

Currently, NMFS is working on a hatchery reform project in the Columbia River Basin, which will include a collaborative review of how harvest and hatcheries (particularly Federally funded hatcheries) are affecting the recovery of listed salmon and steelhead in the basin. This effort was mandated by Congress in 2005, and is in its early stages. Eventually, the project team would create a management approach that allows tribal, state and Federal managers to effectively manage Columbia River Basin hatcheries to meet conservation and harvest goals consistent with their respective legal responsibilities.

Mining. Most of the mining in the basin is focused on minerals such as phosphate, limestone, dolomite, perlite, or metals such as gold, silver, copper, iron, and zinc. Mining in the region is conducted in a variety of methods and places within the basin. Alluvial or glacial deposits are often mined for gold or aggregate, and ores are often excavated from the hard bedrocks of the Idaho batholiths. Eleven percent of the nation's output of gold has come from mining operations in Washington, Montana, and Idaho, and more than half of the nation's silver output has come from a few select silver deposits, with 30% coming from two deposits in the Columbia River Basin (the Clark Fork River and Coeur d'Alene deposits; Hinck et al. 2004, Butterman and Hilliard 2005). According to Wydoski and Whitney (1979), one of the largest mines in the region, located near Lake Chelan, once produced up to 2,000 tons of copper-zinc ore with gold and silver on a daily basis. Most of the phosphate mining within the basin occurs in the headwaters of the Snake River; the overall output from these deposits accounts for 12% of United States phosphate production (Hinck et al. 2004).

Many of the streams and river reaches in the basin are impaired from mining and several abandoned and former mining sites are designated as Superfund cleanup areas (Stanford et al. 2005; EPA 2007). According to the United States Bureau of Mines, there are about 14,000 inactive or abandoned mines within the Columbia River Basin of which nearly 200 pose a potential hazard to the environment (Quigley et al. 1997 *in* Hinck et al. 2004). Contaminants detected in the water include lead and other trace metals. Mining of copper, cadmium, lead, manganese, and zinc in the upper Clark Fork River have contributed wastes to this basin since 1880 (Woodward et al. 1994). Benthic macroinvertebrates and fish within the basin have bioaccumulated metals, which are suspected of reducing their survival and growth (Farag et al. 1994; Woodward et al. 1994). In the Clark River, several fish kills have occurred since 1984 and are attributed to contamination from trace metals such as cadmium, copper, lead, and zinc (Hinck et al. 2004).

Commercial, Recreational, and Subsistence Fishing. Archeological records indicate that indigenous people caught salmon in the Columbia River more than 7,000 years ago. One of the most well known tribal fishing sites within the basin was located near Celilo Falls, an area in the lower river that has been occupied by Dalles Dam since 1957. Salmon fishing increased with better fishing methods and preservation techniques, such as drying and smoking, such that harvest substantially increased in the mid-1800s with canning techniques. Harvest techniques also changed over time, from early use of hand-held spears and dip nets, to riverboats that used seines and gill-nets, eventually, transitioning to large ocean-going vessels with trolling gear and nets and the harvest of Columbia River salmon and steelhead from California to Alaska (Mann et al. 2005).

During the mid-1800s, an estimated 10 to 16 million adult salmon of all species entered the Columbia River each year. Large harvests of returning adult salmon during the late 1800s ranging from 20 million to 40 million pounds of salmon and steelhead annually significantly reduced population productivity (Mann et al. 2005). The largest known harvest of Chinook salmon occurred in 1883 when Columbia River canneries processed 43 million pounds of salmon (Lichatowich 1999). Commercial landings declined steadily from the 1920s to a low in 1993, when just over one million pounds were harvested (Mann et al. 2005).

Harvested and spawning adults reached 2.8 million in the early 2000s, of which almost half are hatchery produced (Mann et al. 2005). Most of the fish caught in the river are steelhead and spring/summer Chinook salmon, while ocean harvest consists largely of coho and fall Chinook salmon. Most ocean catches are made north of Cape Falcon, Oregon. Over the past five years, the number of spring and fall salmon commercially harvested in tribal fisheries has averaged between 25,000 and 110,000 fish (Mann 2004 in Mann et al. 2005). Recreational catch in both ocean and in-river fisheries varies from 140,000 to 150,000 individuals (Mann et al. 2005).

Puget Sound Region

Natural History

The Puget Sound watershed is defined by the crest lines of the Olympia Mountain Range (and the Olympic Peninsula) to the west and the Cascade Range to the east. The Olympic Mountains reach heights of about 8,000 feet, and are extremely rugged and steeply peaked with abrupt descents into the Puget Lowland. The Cascade Mountains range in heights of 4,000 to 8,000 feet with the highest peak, Mount Rainer, towering at 14,410 feet above sea level. As the second largest estuary in the United States, Puget Sound has about 1,330 miles of shoreline and extends from the mouth of the Strait of Juan de Fuca east, including the San Juan Islands and south to Olympia, and is fed by more than 10,000 rivers and streams.

Puget Sound is generally divided into four major geographic marine basins: Hood Canal, South Sound, Whidbey Basin, and the Main Basin. The Main Basin has been further subdivided into two sub-basins: Admiralty Inlet and Central Basin. Each of the above basins forms a depression on the sea floor in which a shallower ledge or sill separates the relatively deep water from the adjacent basin. The waters of Puget Sound function as a partially mixed, two-layer system, with relatively fresh water flowing seaward at the surface and salty oceanic water entering at depth.

The main ledge of Puget Sound is located at the north end of Admiralty Inlet where the water shoals to a depth of about 200 feet at its shallowest point (King County 2001). The deepest point in Puget Sound is in the Central Basin at over 920 feet in depth. Approximately 43% of the Puget Sound's tideland is located in the Whidbey Island Basin. This reflects the large influence of the Skagit River, which is the largest river in the Puget Sound system and whose sediments are responsible for the extensive mudflats and tidelands of Skagit Bay.

Habitat types that occur within the nearshore environment include eelgrass meadows, kelp forest, mud flats, tidal marshes, subestuaries (tidally influenced portions of river and stream mouths), sand spits, beaches and backshore, banks and bluffs, and marine riparian vegetation. These

habitats provide critical functions such as primary food production and support habitat for invertebrates, fish, birds, and other wildlife.

The Puget Sound ecoregion is a glaciated area consisting of glacial till, glacial outwash and lacustrine deposits with high quality limestone in the San Juan Islands (Wydoski and Whitney 1979). Relief in the valley is moderate, with elevation ranging from sea level to about 1,300 feet. Geology in the region consists of mostly Tertiary sedimentary bedrock formations.

The land and vegetation surrounding Puget Sound waters is classified as Puget Lowland Forest and occupies the depression or valley between the Olympic Peninsula on the west and the Cascade Range to the east (Franklin and Dyrness 1973). The alpine zone is expressly devoid of trees. Vegetation changes abruptly along the mountain slopes and across minimal horizontal distances as a result of steep topography, soil, and microclimate (sun exposure, temperature, and precipitation). Dominant vegetation types include the Puget lowland region – the lowland forest and the mid-montane forest of Pacific silver fir and Alaska yellow cedar; the subalpine forest of mountain hemlock with subalpine fir and Alaska yellow cedar; and the alpine tundra or meadow above the treeline (Kruckeberg 1991).

The Puget Sound region has a Mediterranean-like climate, with warm, dry summers, and mild wet winters (Franklin and Dyrness 1973). Annual precipitation varies from 28 to 35 inches, and falls predominantly as rain in lowland areas. Annual snowpack in the mountain ranges is often high; although the elevation of the Olympic Mountains is not as high as that of the Cascade Mountain Range, abundant accumulation occurs, such that it will sometimes persist throughout much of the summer. Average annual rainfall in the north Cascades at Mount Baker Lodge is about 110 inches, and at Paradise Station at Mount Rainer is about 105 inches, while average annual snowfall is 550 inches and 582 inches respectively, sometimes reaching more than 1,000 inches on Mount Rainer (Wydoski and Whitney 1979; Kruckeberg 1991).

Major rivers draining to Puget Sound from the Cascade Mountains include the Skagit, Snohomish, Nooksack, Puyallup, and Green rivers, as well as the Lake Washington/Cedar River watershed. Major rivers from the Olympic Mountains include the Hamma Hamma, the Duckabush, the Quilcene, and the Skokomish rivers. Numerous other smaller rivers drain to the Sound, many of which provide important salmonid habitats despite their small size.

The Puget Sound basin is home to more than 200 fish and 140 mammalian species. Salmonids within the region include coho, Chinook, sockeye, chum, and pink salmon, kokanee, steelhead, rainbow, cutthroat, and bull trout, as well as Dolly Varden (Wydoski and Whitney 1979; Kruckeberg 1991). Important commercial fishes include the five Pacific salmon and several rockfish species. A number of introduced species occur within the region, including brown and brook trout (*Salvelinus fontinali*s), Atlantic salmon, bass, tunicates (sea squirts), and a saltmarsh grass (*Spartina* spp.). Estimates suggest that more than 90 species have been intentionally or accidentally introduced in the region (Ruckelshaus and McClure 2007). At present over 40 species in the region are listed as threatened and endangered under the ESA.

Human Activities and Their Impacts

Land Use. Land use in the Puget Sound lowland is composed of agricultural areas (including

forests for timber production), urban areas (industrial and residential use), and rural areas (low density residential with some agricultural activity). In the 1930s, all of western Washington contained about 15.5 million acres of "harvestable" forestland and by 2004 the total acreage was nearly half that originally surveyed (PSAT 2007). Forest cover in Puget Sound alone was about 5.4 million acres in the early 1990s and about a decade later the region had lost another 200,000 acres of forest cover with some watersheds losing more than half the total forested acreage. The most intensive loss of forest cover occurred in the Urban Growth Boundary, which encompasses specific parts of the Puget Lowland; in this area forest cover declined by 11% between 1991 and 1999 (Ruckelshaus and McClure 2007). Projected land cover changes (reviewed in Ruckelshaus and McClure 2007) indicate that trends are likely to continue over the next several decades with population changes; coniferous forests are projected to decline at an alarming rate as urban uses increase.

The Puget Sound Lowland contains the most densely populated area of Washington. The regional population in 2003 was an estimated 3.8 million people, with 86% residing in King, Pierce and Snohomish counties (Snohomish, Cedar-Sammamish Basin, Green-Duwamish, and Puyallup River watersheds), and the area is expected to attract four to six million new human residents in the next 20 years (Ruckelshaus and McClure 2007). According to the State of the Sound report (PSAT 2007) in 2001, impervious surfaces covered 3.3% of the region, with 7.3% of lowland areas (below 1,000 feet elevation) covered by impervious surfaces. In one decade, 1991 – 2001 impervious surfaces increased 10.4% region wide. The Snohomish River watershed, one of the fastest growing watersheds in the region, increased about 16% in the same period.

Much of the region's estuarine wetland losses have been heavily modified, primarily from agricultural land conversion and urban development (NRC 1996). Although most estuarine wetland losses result from conversions to agricultural land by ditching, draining, or diking, these wetlands are also experiencing increasing effects from industrial and urban causes. The most extreme case of river delta conversion is observed in the Duwamish Waterway in Seattle. As early as the mid-1800s, settlers in the region began discussing the need for a ship canal that linked Lake Washington directly with Puget Sound. After several private and smaller attempts, by the early 1900s locks were built achieving this engineering feat. The result was that the Black River, which formerly drained Lake Washington to the Green and White rivers (at their confluence, these rivers formed the Duwamish River), dried up. The lower White River, which historically migrated sporadically between the Puyallup and the Green/Duwamish basins, was permanently diverted into the Puyallup River basin in 1914 with the construction of a concrete diversion at river mile 8.5, resulting in a permanent increase of Puyallup River flow by about 50% and a doubling of the drainage area (Kerwin 1999). The Cedar River, on the other hand was permanently diverted to Lake Washington. The oxbow in the lower Duwamish River was lost with the lower river dredging in the early 1900s, reducing the lower nine miles of the river to 5 miles in length. Over time, the Duwamish Waterway has been heavily armored and diked, result in the loss of all tidal swamps, 98% of the tidal forests, marshes, shallows and flats and 80% of the riparian shoreline (Blomberg et al. 1988). By 1980, an estimated 27,180 acres of intertidal or shore wetlands had been lost at eleven deltas in Puget Sound (Bortleson et al. 1980). Tidal wetlands in Puget Sound amount to roughly 18% of their historical extent (Collins and Sheikh 2005). Coastal marshes close to seaports and population centers have been especially vulnerable

to conversion with losses of 50-90%.

More than 100 years of industrial pollution and urban development have affected water quality and sediments in Puget Sound. Many different kinds of activities and substances release contamination into Puget Sound and the contributing waters. Positive changes in water quality in the region are also evident. One of the most notable improvements was the elimination of sewage effluent to Lake Washington in the mid 1960s, which significantly reduced problems within the lake from phosphorus pollution and triggered a concomitant reduction in cyanobacteria (Ruckelshaus and McClure 2007). Even so, as the population and industry has risen in the region a number of new and legacy pollutants are of concern. According to the State of the Sound Report (PSAT 2007) in 2004, more than 1,400 fresh and marine waters in the region were listed as "impaired." Almost two-thirds of these water bodies were listed as impaired due to contaminants, such as toxics, pathogens, and low dissolved oxygen or high temperatures, and less than one-third had established cleanup plans. More than 5,000 acres of submerged lands (primarily in urban areas; 1% of the study area) are contaminated with high levels of toxic substances, including polybrominated diphenyl ethers (PBDEs; flame retardants), and roughly one-third (180,000 acres) of submerged lands within Puget Sound are considered moderately contaminated. Primary pollutants of concern in Puget Sound include heavy metals, organic compounds, PAHs, PCBs, dioxins, furans, DDT, phthalates, and PBDEs. Areas of highest concern in Puget Sound are Southern Hood Canal, Budd Inlet, Penn Cove, Commencement Bay, Elliott Bay, Possession Sound, Saratoga Passage, and Sinclair Inlet (PSAT 2007). Hypoxic or low dissolved oxygen concentration were found at a number of monitoring stations, including Saratoga Passage, Discovery Bay, Bellingham Bay, Elliott Bay, Budd Inlet, and Commencement Bay. Many of the contaminants in the Sound, including several that were banned years ago, continue to bioaccumulate in the food web to top level predators (NMFS 2008a).

Hydromodification Projects. More than 20 dams occur within the region's rivers and overlap with the distribution of salmonids, and a number of basins contain water withdrawal projects or small impoundments that can impede migrating salmon. The impact of these and land use changes (forest cover loss and impervious surface increases) has been a significant modification in the seasonal flow patterns of area rivers and streams, and the volume and quality of water delivered to Puget Sound waters. Several rivers have been hydromodified by other means including levees and revetments, bank hardening for erosion control, and agriculture uses. Since the first dike on the Skagit River delta was built in 1863 for agricultural development (Ruckelshaus and McClure 2007), other basins like the Snohomish River are diked and have active drainage systems to drain water after high flows that top the dikes. Dams were also built on the Cedar, Nisqually, White, Elwha, Skokomish, Skagit, and several other rivers in the early 1900s to supply urban areas with water, prevent downstream flooding, allow for floodplain activities (like agriculture or development), and to power local timber mills (Ruckelshaus and McClure 2007).

In the next couple of years, a highly publicized and long discussed dam removal project is expected to begin in the Elwha River. The removal of two dams in the Elwha River, a short but formerly very productive salmon river, is expected to open up more than 70 miles of high quality salmon habitat (Wunderlich et al. 1994). Estimates suggest that nearly 400,000 salmon could

begin using the basin within 30 years after the dams are removed (PSAT 2007).

About 800 miles of Puget Sound's shorelines are hardened or dredged (PSAT 2004). The area most intensely modified is the urban corridor – the eastern shores of Puget Sound from Mukilteo to Tacoma). Here, nearly 80% has been altered, mostly from shoreline armoring associated with the Burlington Northern Railroad tracks (Ruckelshaus and McClure 2007). Levee development within the rivers and their deltas has isolated significant portions of former floodplain habitat that was historically used by salmon and trout during rising flood waters.

Mining. Mining has a long history in the Washington, and in 2004 the state was ranked 13th nationally in total nonfuel mineral production value and 17th in coal production (Palmisano et al. 1993; NMA 2007). Metal mining for all metals (zinc, copper, lead, silver, and gold) peaked between 1940 and 1970 (Palmisano et al. 1993). Today, construction sand and gravel, Portland cement, and crushed stone are the predominant materials mined. Where sand and gravel is mined from riverbeds (gravel bars and floodplains) it may result in changes in channel elevations and patterns, instream sediment loads, and instream habitat. In some cases, instream or floodplain mining has resulted in large scale river avulsions. The effect of mining in a stream or reach depends upon the rate of harvest and the natural rate of replenishment, as well as flood and precipitation conditions during or after the mining operations.

Commercial and Recreational Fishing. Most of the commercial landings in the region are groundfish, Dungeness crab, shrimp, and salmon. Many of the same species are sought by tribal fisheries and by charter and recreational anglers. Nets and trolling are used in commercial and tribal fisheries, whereas recreational anglers typically use hook and line, and may fish from boat, river bank, or docks. Entanglement of marine mammals in fishing gear is not uncommon and can lead to mortality or serious injury.

Oregon-Washington-Northern California Coastal Drainages

Natural History

This region encompasses drainages originating in the Klamath Mountains, the Oregon Coast Mountains and the Olympic Mountains, all of which form the Coast Range ecoregion where elevations range from sea level to about 4,000 feet. More than 15 watersheds drain the region's steep slopes including the Umpqua, Alsea, Yaquima, Nehalem, Chehalis, Quillayute, Queets, and Hoh rivers. Numerous other small to moderately sized streams dot the coastline. Many of the basins in this region are relatively small; the Umpqua River drains a basin of 4,685 square miles and is a slightly over 110 miles long, and the Nehalem River drains a basin of 855 square miles and is almost 120 miles long. However, systems here represent some of the most biologically diverse basins in the Pacific Northwest (Johnson 1999; Carter and Resh 2005).

The region is part of a coastal, temperate rainforest system, and is characterized by a moderate maritime climate marked by long wet seasons with short dry seasons and mild to cool year-round temperatures. Average annual precipitation ranges from about 60 inches to more than 180 inches, much of which falls as rain, and supports a rich temperate forest. Vegetation is characterized by giant coniferous forests of Sitka spruce, western hemlock, Douglas fir, western

red cedar, red alder, and black cottonwood

The Oregon Coast supports a unique coastal sand dune system. The sand dunes were largely created by the sand deposited from the coastal rivers, in particular the Umpqua and Columbia rivers. North, steep headlands and cliffs are separated by stretches of flat coastal plain and large estuaries. Significant estuaries in the region (outside of the Columbia River Estuary) include Coos Bay, Tillamook Bay, and the Nehalem River Estuary in Oregon, as well as Grays Harbor and Willapa Bay in Washington.

Human Activities and Their Impacts

Land Use. The rugged topography of the western Olympic Peninsula and the Oregon Coastal Range has limited the development of dense population centers. For instance, the Nehalem River and the Umpqua River basins consist of less than 1% urban land uses. Most basins in this region have long been exploited for timber production, and are still dominated by forestlands. In Washington State, roughly 90% of the coastal region is forested (Palmisano et al. 1993). Approximately 92% of the Nehalem River basin is forested, with only 4% considered agricultural (Johnson 1999). Similarly, in the Umpqua River basin, about 86% is forested land, 5% agriculture, and 0.5% are considered urban lands. Roughly half the basin is under Federal management (Carter and Resh 2005).

Hydromodification Projects. Compared to other areas in the greater Northwest Region, the coastal region has fewer dams and several rivers remain free flowing (e.g., Clearwater River). The Umpqua River is fragmented by 64 dams, the fewest number of dams on any large river basin in Oregon (Carter and Resh 2005). According to Palmisano et al. (1993) dams in the coastal streams of Washington permanently block only about 30 miles of salmon habitat. In the past, temporary splash dams were constructed throughout the region to transport logs out of mountainous reaches. The general practice involved building a temporary dam in the creek adjacent to the area being logged, the pond was filled with logs and when the dam broke the floodwater would carry the logs to downstream reaches where they could be rafted and moved to market or downstream mills. Thousands of splash dams were constructed across the Northwest in the late 1800s and early 1900s. While the dams typically only temporarily blocked salmon habitat, in some cases they remained long enough to wipe out entire runs, since effects of the channel scouring and loss of channel complexity resulted in the long term loss of salmon habitat (NRC 1996).

Mining. Oregon is ranked 35th nationally in total nonfuel mineral production value in 2004, while Washington was ranked 13th nationally in total non-fuel mineral production value and 17th in coal production (Palmisano et al. 1993; NMA 2007). Metal mining for all metals (e.g., zinc, copper, lead, silver, and gold) peaked in Washington between 1940 and 1970 (Palmisano et al. 1993). Today, construction sand and gravel, Portland cement, and crushed stone are the predominant materials mined in both Washington and Oregon. Where sand and gravel is mined from riverbeds (gravel bars and floodplains) it may result in changes in channel elevations and patterns, instream sediment loads, and seriously alter instream habitat. In some cases, instream or floodplain mining has resulted in large scale river avulsions. The effect of mining in a stream or reach depends upon the rate of harvest and the natural rate of replenishment, as well as flood and precipitation conditions during or after the mining operations.

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Impact of the Environmental Baseline on Listed Resources

In 2007, the population of the United States increased to more than 300 million people for the first time in its history. That population growth and increase in population density was accompanied by dramatic changes in the landscapes of the United States. By 2000, half of the population in the United States lived in the suburbs (Hobbs and Stoops 2002). About 75% of all Americans now live in areas that are urban or suburban in character; that is, about 75% of the people in the lower 48 States live in less than 2% of the land area of the lower 48 states. Most modern metropolitan areas encompass a mosaic of different land covers and uses (Hart 1991). The mosaic or land uses associated with urban and suburban centers has been cited as the primary cause of declining environmental conditions in the United States (Flather et al. 1998) and other areas of the world (Houghton 1994).

The direct and indirect effects of these changes in land-use and land-cover have had lasting effects on the quantity, quality, and distribution of every major terrestrial, aquatic, and coastal ecosystem in the United States, its territories, and possessions. Many native ecosystems exist as small isolated fragments, surrounded by expanses of urban and suburban landscapes or by natural areas dominated by non-native species. As a result, many of the native plant and animal species that inhabited those native ecosystems over the past have become extinct, endangered, or threatened over the past 200 years. Even marine ecosystems, once deemed by many as the most resilient of ecosystems, a vast source of fish for harvest and a limitless sink for waste material, are threatened by human activities on a global scale. The most pervasive threats to marine ecosystems include ocean-based destructive demersal fishing practices, increasing sea temperatures, coastal development, increased sediment loading, point-source organic pollution, and hypoxia (Halpern et al. 2007).

The rapid growth of commercial fishing of what was once considered an endless food supply has resulted in drastic over-exploitation of fisheries resources and modification of the marine environment (Hall 1999). Increases in national and global populations have lead to a dramatic increase in demand for seafood, resulting in expansion of fishing fleets by orders of magnitude, development of new technology to capture resources more efficiently, and greater ability to exploit areas once considered out of reach. In particular, fishing practices have lead to pressures not only on target species, but changes to whole habitats and the protected species that are either caught directly, or whose habitat is degraded because of them. It has been estimated that global commercial fishing industries catch and discard 27 million metric tons of fish, sea turtles, marine mammals, and other organisms annually (Hall 1999). Gill nets set for several miles can entrap, drown, or disable any organisms larger than their mesh size, from salmon to large whales. Although gill nets may be set thousands of miles from domestic waters, individuals of protected species caught in these nets can be the same that nest, breed, or feed in United States waters.

Dredging and trawling gears clear bottom habitat of any sizeable material, eliminating habitat of small fishes and invertebrates on which other species feed (Hall 1999). This process also displaces large amounts of sediment into the water, dramatically altering water clarity and chemistry. There are likely additional factors that influence listed species directly or indirectly, which are thus far unknown.

The process of global warming is a developing concern to protected species management. Widespread habitat alteration or loss can also stem from even moderate, but prolonged, increases in temperature. Although many effects of climate change are unknown, the instability and environmental change that has been measured to occur thus far support the likelihood that global warming will have negative impacts on protected species and the habitats that they occupy.

Coastal development has more localized effects on marine environments, but is so extensive that most, if not all, nearshore environments are affected by it in some way. Development may be so detrimental as to extirpate populations or species in very short periods of time. Such is the case with several populations of salmon along the United States Pacific coast, where dam construction blocked fish movement to and from spawning and feeding habitats (Lichatowich 1999). As a result, entire populations are now considered extinct. In general, coastal development without environmental consideration has resulted in direct mortality to protected species, modification of habitat to displace individuals or populations from a region, or reduced reproductive success. In such cases, survivorship declines can be significant, resulting in protection of species not formerly listed, or moderate in species already listed that can ill-afford further impediments to recovery. As with fishing, coastal development in foreign countries can affect marine species protected in the United States by affecting habitat to which these species migrate for breeding or feeding. Environmental impacts, particularly to strategically important or listed species, of coastal development have received more global interest in recent years and changes, such as EIS statements, outreach and education, and environmentally friendly design have mitigated some impacts. However, many countries continue developing coastal regions without significant concern for protected or sensitive species or their habitats and these distant activities can have negative consequences for listed species in this country.

Additional activities on land have significant effects in ocean environments. This is particularly true for sedimentation as well as agricultural, industrial, and municipal pollution. Soils are normally covered by tracts of forest, grassland, marsh, or other vegetation preventing significant erosion. However, development activities tend to disturb these areas, or bring in large amounts of soil during construction, allowing for wind, rain, and other mechanisms to move the soil to local water bodies. Salmon nests become covered with sediment, or highly localized spaces for nests become covered, resulting in high hatching mortality or elimination of entire stretches of spawning habitat (NMFS and USFWS 2005).

Agricultural development and use has its own unique contribution to marine pollution. Fertilizers applied to tracts of land, from front lawns to large fields, can run-off in rainwater if not applied properly these fertilizers contain concentrated nutrients that dissolve in water and enter streams, rivers, lakes, estuaries, and the marine environment (Kennish 1992; Soares 1999). Along with nutrients contained in sediments, these elevated nutrient concentrations provide fodder for potentially exponential bacterial, algal, and plant growth. This rapid growth process

can create algal “blooms” (red tide), which can make toxic metabolic byproducts in such concentrations that fish, seabirds, and marine mammals can become ill or die as a result. Such events happen continually along Gulf of Mexico states and instances are known for the west and east coasts. After nutrients have been used up, large numbers of small organisms die and the natural breakdown of their bodies results in areas of oxygen depleted water, sometimes hundreds of square miles in size, called “dead zones” in which organisms requiring oxygen in water to breathe cannot survive. Such an area occurs off the coast of Louisiana. This process of eutrophication can eliminate large areas of nearshore and oceanic habitat, resulting in direct mortality to or adverse modification of habitat utilized by listed species. Shortnose sturgeon are generally believed to be absent from numerous rivers feeding into and sections of the Chesapeake Bay itself because of eutrophication issues stemming from fertilizer use on lawns and fields. Unlike most other forms of pollution, eutrophication can eliminate or displace large sections of habitat and all animals within it. This issue has received more interest in recent years. Regulations are being installed to regulate fertilizer runoff and public outreach has been growing.

Although sedimentation and agricultural pollution comes from general areas, point-source pollution comes from specific effluents and can have additional effects. These drainages frequently come from municipal wastewater treatment plants, commercial and industrial discharges, as well as recreational and commercial vessels (Kennish 1992). Point-sources tend to contain specific chemical components that result from anthropogenic activity, as opposed to excessive sediments entering a waterbody. These components can be toxic and require regulation. However, the effects of components on species and their environment is generally unknown and it is only after several years of research that enough evidence is collected to initiate regulation. Such has been the case with pesticides, such as DDT and DDE, which caused severe fragility in bird eggs and led to the listing of several avian species, including bald eagles. Such is now the case with pharmaceuticals in wastewater. Hormones are currently released in wastewater from treatment plants. It is unknown what effects these chemicals have on endocrine disruption to species in habitats near wastewater discharges. It has been suggested that humans reconsuming these waters may have intra-sex children (Soares 1999), which indicates that these chemicals may affect other exposed organisms. What is known is that point-source discharges can introduce chemicals into fresh water, estuarine, and marine habitats whose effects can cause significant decline in a variety species, but the effects may not be known for years later.

Salmonids originally underwent dual pressures that led to their decline: dam construction and commercial fishing (Lichatowich 1999). Although fishing had occurred extensively through time, more widespread and technologically advanced methods were developed in the past two centuries to harvest salmon beyond the rate at which they could reproduce (Lichatowich 1999). More importantly and at the same time, dam construction occurred that cut the connection between two necessary salmon habitats: streams and ocean (Lichatowich 1999). This lead almost immediately to large-scale salmon declines or extinctions in several local areas. Now, dams have generally been modified or removed to re-establish communication between habitats for salmon in most areas. Commercial fishing is also closely monitored to prevent excessive pressures on populations. However, new threats in the forms of habitat loss, pollution, and genetic dilution of populations specialized for certain habitats impede recovery efforts (Reisenbichler 1997). As predators, salmon tend to bioaccumulate toxins as whales do, but generally accumulate more because they eat other fish instead of krill, which are lower on the food chain. Pollution is

identified as a contributing factor for 38% of ESA listed species overall (Hoffman et al. 2003). Contaminants can cause reproductive disruption, immune dysfunction, and other physiological effects accumulate in vertebrates and can cause reduced reproductive fitness and subsequent population decline (Rand and Petrocelli 1985).

EFFECTS of the Action

The *Description of the Proposed Action* describes EPA's proposal to continue to recommend the 1985 304(a) aquatic life criteria for cyanide and approve state and tribal water quality standards, or federal water quality standards promulgated by EPA for the protection of aquatic life that are identical to or are more stringent than the section 304(a) cyanide aquatic life criteria. The *Status of the Species and Critical Habitat* section of this Opinion identified the endangered and threatened species, and designated critical habitat that may be affected by the proposed action, as well as those species and critical habitat that are currently proposed for listing under the ESA. The *Status* also summarized the status and trends of those species, and other ecological information relevant to our effect's analyses, while the *Environmental Baseline* summarized the consequences of a variety of human activities, including land and water uses that impact the listed species and critical habitat considered herein.

In this section, we identify specific stressors and subsidies associated with the proposed action, the likelihood endangered species, threatened species and designated critical habitat are exposed to those stressors and subsidies, the responses of listed species and critical habitat to their exposure, and the consequences of those responses to the different listed resources. Based on the results of these analyses, we assess the risks EPA's proposal to recommend and approve of water quality standards for cyanide poses to listed resources. For endangered and threatened species, our assessment focuses on the risk of increasing the extinction probability of these species, for designated critical habitat our assessment focuses on the risk of reducing the conservation value of the habitat designated for the endangered and threatened species.

As discussed in the *Approach to the Assessment* section of this Opinion, as part of this national consultation, our consultation examines the decision-making process that EPA uses to recommend and approve water quality criteria and the outcome of that decision making process. In particular, this consultation focuses on how EPA determines what constitutes a "safe and healthful level in waterbodies for a pollutant, which a regulatory authority can use to guide the control, reduction and eventual elimination of that pollutant (BE, page 15)" in the environment for the protection of fish and wildlife species consistent with the goals of the CWA, and threatened and endangered species in particular.

When EPA recommends 304(a) aquatic life criteria, that recommendation means that water quality standards identical to or more stringent than EPA's criteria will protect the designated uses of water that receive pollutants at levels consistent with the aquatic life criteria. As a default, EPA uses "fishable and swimmable" as the designated uses when it establishes their aquatic life criteria. That is, EPA has determined that the adoption of their criteria to be protective of aquatic life designated uses consistent with the objective and goals articulated in

CWA sections 101(a) and 101(a)(2) (EPA 2008b). Therefore, when EPA recommended 304(a) aquatic life criteria for cyanide, EPA also determined that cyanide at the recommended numeric value would protect designated uses consistent with the objective of the CWA to "restore and maintain the chemical, physical and biological integrity of the Nation's waters (CWA §101(a))," and the goal to provide "for the protection and propagation of fish, shellfish, and wildlife and provides for recreation in and on the water…. (CWA §101(a)(2))."

If EPA recommends 304(a) aquatic life criteria, then fish and wildlife that might be exposed to pollutants at those criteria levels generally should not experience physical, physiological, behavioral, or ecolgocal consequences that would interfere with reproduction or reduce the long-term persistence of their populations resulting from that exposure. That is, EPA expects that the criteria would generally provide a "reasonable level of protection" of all but a small fraction of the "appropriate" taxa (0.05; Stephan et al 1985). Restated, there is 5% probability that an aquatic species would not be protected by EPA's national criteria. Provided EPA considers threatened and endangered species part of the taxa that would be protected by their national criteria, then we would expect that EPA's national criteria would generally protect endangered and threatened species and designated critical habitat. Specifically, we would expect that when EPA recommends the CMC of 22.36 μg/L and the CCC of 5.221 μg/L in fresh water, or at the CMC of 1.015 μg/L or the CCC of 1.015 μg/L in salt water as its 304(a) aquatic life criteria for cyanide, then endangered or threatened species or designated critical habitat exposed to cyanide at these concentrations should not experience physical, physiological, behavioral, or ecological consequences that would reduce the long-term persistence of their populations resulting from that exposure. Certainly this would be the case if (a) EPA considered aquatic listed species as an indicator of or part of the aquatic assemblage that defines the biological integrity of the Nation's waters, or part of the fish, shellfish and wildlife the CWA intends to protect; or (b) listed species are expressly or indirectly listed as a designated use by a state or tribe.

We begin our assessment of the *Effects of the Action* by evaluating the decision making process EPA uses to develop 304(a) aquatic life criteria and establish numeric values for the CMC and CCC for a particular pollutant, and EPA's 1985 published values for the cyanide CMC and CCC. These values represent EPA's recommended 304(a) aquatic life criteria for cyanide, upon which EPA intends to subsequently approve for use in state or tribal water quality standards. Our evaluation focuses on whether it is reasonable to expect that endangered species, threatened species, and designated critical habitat are exposed to cyanide at concentrations similar to national criteria values; and whether it is reasonable to expect that endangered species, threatened species, and designated critical habitat are not likely to respond to any exposures to cyanide at the CMC of 22.36 μg/L or the CCC of 5.221 μg/L in fresh water, or at the CMC of 1.015 μg/L or the CCC of 1.015 μg/L in salt water.

If listed resources are likely to respond to exposures to cyanide at the CMC of 22.36 μg/L or the CCC of 5.221 μg/L in fresh water, or at the CMC of 1.015 μg/L or the CCC of 1.015 μg/L in salt water, then we would evaluate the likelihood that endangered species, threatened species, and designated critical habitat would be exposed to: a) the one-hour average exposure concentrations of cyanide that would not exceed the CMC more than once every three years; and b) four-day average exposure concentrations of cyanide that would not exceed the CCC more frequently than

once every three years on average. If we conclude that, endangered species, threatened species, and designated critical habitat would be exposed to cyanide at concentrations that deviate from the one-hour and four-day average, we would examine the variability in concentrations to which endangered species, threatened species, and designated critical habitat would be exposed. As part of this evaluation, we would examine whether endangered species, threatened species, and designated critical habitat "should not be affected unacceptably (EPA 1985)" if the four-day average concentration of cyanide does not exceed 5.2 μg/CN in fresh water or 1.015 μg/CN in salt water more than once every three years on average and if the one-hour average concentration does not exceed 22.36 μg/CN in fresh water or 1.015 μg/CN in salt water more than once every three years on average. Finally, we would evaluate the context for probable exposure events including whether environmental conditions in which listed species reside or the physiological state of the individual organism would influence the severity of probable responses.

EPA's Decision-Making Process

Derivation of Criteria

In order to evaluate whether the cyanide aquatic life criteria and any water quality standards that would be based on those criteria are not likely to jeopardize listed species or adversely modify critical habitat, we first examine how EPA derived the aquatic life criteria. The EPA document, *Guidelines for Deriving Numerical National Water Quality Criteria for* the *Protection* of *Aquatic Organisms and Their Uses* (the "Guidelines") outlines the process that EPA uses to derive water quality recommendations that intend to protect aquatic assemblages (Stephan et al. 1985; EPA 2008b). According to the guidelines, once a decision is made that a criterion is needed EPA collects and reviews all available information on the toxicity of the chemical is collected and reviewed for acceptability, and sorted.

The decision-making process for deriving aquatic life criteria involves a mix of quantified estimates of the effects a particular contaminant would have on a sample of test subjects, and professional judgement. That is, criterion development involves quantifying the sensitivity of a suite of species to toxic compounds in controlled studies; professional judgment comes into the process in several areas including the setting aside of data, determining whether a species is commercially or recreationally important and whether data on that species deserves additional attention in the final derivation of the criterion, determining whether particular data is useful or should be set aside (e.g., determining if water quality characteristics of a test are acceptable, or whether the degree of agreement between species is reasonable).

As a general matter, the *Guidelines* require the use of acute and chronic toxicity tests on a broad range of aquatic species to provide an indication of the sensitivities of untested species. These data are used by EPA to develop chronic and acute criteria for both salt and fresh water, the CCC and the CMC respectively. EPA's development of two values for fresh and salt water, the CMC and CCC, is premised on the assumption that doing so more accurately reflects toxicological and practical realities while not being as restrictive as a one-number criterion would have to be in order provide the same degree of protection (Stephan et al. 1985).

To derive an acute criterion for fresh water, the *Guidelines* suggest that toxicity data be available for at least one species of freshwater animal in at least eight different families. The families include:

1) Salmonidae (e.g., salmon or trout),
2) a second family in the class Osteichthyes, preferably a commercially or recreationally important warmwater species (e.g., bass, bluegill),
3) a third family in the phylum Chordata (e.g, salamander, frog),
4) a planktonic crustacean (e.g, daphnia),
5) a benthic crustacean (e.g, crayfish, amphipod),
6) an insect (e.g., dragonfly, mayfly),
7) a family in a phylum other than Arthropoda or Chordata (e.g., mussel, snail, worm), and
8) a family in any order of insect or any phylum not already represented.

For deriving a saltwater acute criterion the *Guidelines* suggest that acute tests with at least one species of saltwater animal in at least eight different families should be used. The represented families should include:

1) two families in the phylum Chordata,
2) a family other than Arthropoda or Chordata,
3) either the Mysidae or Penaeidae family,
4) three other families not in the phylum Chordata,
5) and any other family.

Additionally, at least one acceptable test is required for saltwater and freshwater plants, and at least one acceptable bioconcentration factor determined with an appropriate saltwater species. Data that is rejected from further consideration may include: data from studies that did not contain control treatment; too many organisms in the control treatment died or showed signs of stress or disease; data from tests using distilled or deionized water as the dilution water without adding appropriate salts; data from species that do not have reproducing wild populations in North America; data on organisms that were previously exposed to substantial concentrations of the test material or other contaminants (Stephan et al. 1985).

Studies used for determining the CMC are acute tests, which are performed with 48 or 96 hours of exposure, and measure the concentration at which the toxin causes death in 50% of the test population (LC_{50}). The LC_{50} values for each species are pooled and averaged to determine the species mean acute value (SMAV). If EPA has data on several species within a genus, then the data are pooled again to calculate a genus mean acute value (GMAV). If data are available from only one species, then that species mean value becomes the GMAV. Once calculated, the GMAV is ranked from high to low (least to most sensitive species) and the lowest four values are used in regression to estimate the concentration that would cause death for the fifth percentile of the most sensitive species. This fifth percentile value represents the final acute value or FAV. In the event a commercially or recreationally important species has a SMAV below the FAV, the SMAV can be substituted for the FAV to protect that important species. Once EPA has determined the FAV (or the lowest SMAV for an important species) then that value is divided by two, in an effort to avoid the death of exposed organisms. The resulting value is the criterion maximum concentration or CMC. EPA calculates the CMC under the assumption, that the CMC

averaging period would be substantially less than the lengths of the acute tests upon which it is based (Stephan et al. 1985). As such, EPA recommends that the CMC be applied as a limit on the 1-hour average concentration in the environment to provide an addition level of protection.

Chronic toxicity values are calculated either in the same general manner as the acute values, or by dividing the FAV by the final acute-to-chronic (ACR). The ACR is a way of relating the acute toxicities to chronic toxicities and is more commonly employed because it allows EPA to make use of a smaller data set. Chronic toxicity test data must be available from at least three different families, so long as at least one is a fish, an invertebrate, and one is an acutely sensitive species, in order to derive a final chronic value. In contrast to acute studies, chronic tests may last weeks or more, at sublethal exposure concentrations and focus on the endpoints of growth and reproduction. Chronic studies focus on two levels of effect for a concentration: the NOEC and the LOEC that cause a statistically significant change in the endpoint of interest (growth or reproduction). Similar to the FAV, the CCC is derived by pooling values and calculating the geometric mean of the two effect levels.

EPA's decision-making process was developed under the assumptions that:

1) Effects that occur on a species in laboratory tests generally occur on the same species in comparable field situations (Stephan et al. 1985);
2) Effect levels defined by chronic toxicity tests are conducted on the "most sensitive life stages" and therefore should protect all other (less sensitive) life stages (Stephan et al. 1985)
3) When the minimum data requirements are satisfied, but few data are available, then restrictive criteria values are derived (BE 2006).
4) The averaging recommendation is based in part on the assumption that most bodies of water could tolerate exceedences once every three years on the average provided the body of water is not subject to anthropogenic stress other than the exceedences of concern (Stephan et al. 1985).

Important caveats to the general approach in EPA's decision-making process include:

1) The development of water quality standards may need to take into account additional factors such as hydrological considerations, environmental and analytical chemistry, extrapolation from lab to field situations, and relationships between species for which data are available and species in the water of concern (Stephan et al. 1985),
2) It may be necessary to derive site-specific criteria by modifying national criteria to reflect local conditions of water quality, temperature, or ecologically important species.
3) Some untested locally important species might be very sensitive to the contaminant of concern (Stephan et al. 1985),
4) Some aquatic organisms in the wild may be stressed by disease, parasites, predators, other pollutants, contaminated or insufficient food, and fluctuating and extreme conditions of flow, water quality and temperatures (Stephan et al. 1985),
5) The decision-making approach is meant to derive criterion that prevent unacceptable long-term and short-term effects, which is not the same as threshold of adverse effects. Some adverse effects (e.g., small reductions in growth, survival or reproduction) will probably occur at or below criterion values (Stephan et al. 1985),
6) The frequency, magnitude and duration of the exceedences should be based on the ability

of the aquatic ecosystem to recover, which will differ greatly according to the pollutant and the state or health of the ecosystem (Stephan et al. 1985)

Understanding the assumptions and the caveats inherent to EPA's decision-making process is important to understanding the uncertainty around the values EPA recommends to states and tribes for use as their water quality standards. For instance, according to EPA laboratory tests conducted at constant exposures simulates "worst case" field conditions. In limited circumstances this assertion is probably true, but in many cases it is not. In the wild, species will typically not be exposed to continuous concentrations of a particular chemical. Rather, concentrations typically vary temporally and spatially and would result in doses that are both higher and lower than the tested dose. This in itself does not make the laboratory exposure approach a reasonable simulation of a worst-case field (or natural) condition. Responses of organisms tested in controlled laboratory systems do not necessarily provide reasonable predictors of organisms' responses to similar chemicals in the wild, although admittedly in many cases this is the only type of data available to us from which to conduct an evaluation. In many cases, the conditions simulated in a laboratory test have little to do with the environment in which most species live in the wild, and as such are unlikely to resemble "worst case field conditions."

In laboratory tests, species are generally isolated from multiple stressors so that researchers are able to isolate the species responses to the chemical (or stressor) under study. In the wild, species are typically exposed to a wide range of stressors, from natural to human induced. For instance, lab studies do not replicate typical environmental conditions where intraspecific competition for food or shelter occurs. Instead, all the test organisms are about the same size, provided with abundant food, and minimal habitat complexity. Interspecific competition generally does not occur in lab tests either, as most lab environments isolate the species under study from typical predators. Physical conditions are maintained at optimal or constant levels (e.g., velocities, water temperature, and dissolved oxygen are not representative of fluctuating conditions in a natural aquatic environment) and generally, there are no other chemical stressors present. Typically, lab specimens are generally not exposed to other stressors such other chemicals, or environmental factors that can influence toxicity (e.g., some chemical or environmental changes in temperature or other parameters can increase or decrease toxicity, some times in a greater than additive fashion). Wild taxa are exposed to a myriad of factors that can influence their responses to a particular chemical at a particular concentration, and at best the laboratory tests are an indication of how species may respond to that chemical in nature. The actual physical and chemical conditions within a waterbody can, for some chemicals, alter the toxicity of the chemical evaluated in the laboratory under controlled conditions. Knowing this, the authors of EPA's decision-making process noted that it may be necessary to account for local conditions when setting water quality standards and permit limitations (see caveat 1 above).

Another important assertion is that EPA's decision-making process uses the most sensitive life stages for defining chronic toxicity. Unfortunately, chronic values, as is the case of acute values, are calculated on available data and generally, chronic studies are few in comparison to studies that examine mortality as the endpoint of concern. The species used for lab tests are also often not representative of the composition and sensitivities of species in a natural community or ecosystem. EPA's aquatic life criteria guidelines require species from eight different families be

tested to determine acute toxicity values for both marine and fresh water. To derive chronic numeric criteria, however, only three chronic tests are necessary, despite the fact that chemical concentrations in the natural environment are likely to occur more often at chronic low levels. Use of such a small data set to make inferences to a much larger community in the wild is cause for concern. Further, it is unclear whether the assumption that the most sensitive life stage is tested, is regularly met. Certain life stages or the transition between life stages, which are inherently stressful as a result of the physiological changes the animal is undergoing (e.g., osmoregulation), are rarely tested.

Even if the tests are conducted on the most sensitive life stage to a particular toxicant, it does not necessarily follow that the critical concentration determined by these sensitive stages are correlated with the vulnerability of the species to the toxicant. For instance, Kammenga et al. 1996 demonstrated that the fitness implication of a toxicant was measureable on the least sensitive stage of the tested species, whereas the most sensitive trait did not have any effect on the fitness of organism. Equally important, however, is that the smaller the data set used to extrapolate responses, the lower the confidence can be in the outcome of the final value. As is the case for many compounds, for cyanide the most robust data set that EPA had available for making its decision was the acute data set. Only five studies were used for deriving the CCC for fresh water, and two from saltwater taxa ---none of which represent empirical evidence of how any of the species addressed in this Opinion would respond to low-level or prolonged exposures of cyanide.

According to EPA when the minimum data requirements are satisfied, however, then *restrictive* criteria values are derived. Unfortunately, extrapolating the stress responses of individuals in a limited number of lab tests to organisms exposed to similar chemical concentrations while in highly complex natural environment provides for weak gross scale predictions at best, particularly when few or none of the species of interest were evaluated by direct empirical evidence. The greatest utility in simple laboratory tests is that they facilitate faster (and cheaper) data on generalized responses of a range of taxa to a defined chemical exposure (Cairns and Niederlehner 2003). Models, mesocosm or field studies, transparent reasoning, and validation studies should temper the results of such lab studies in decision-making, particularly when extrapolating potential environmental outcomes to a complex environment and in situations, like the management of threatened and endangered species and their designated critical habitat, where a low tolerance for error is warranted. Stephan (2002) summed it up best when he said: "Unless species are selected from a field population using an appropriate procedure (e.g., using random or stratified random sampling), use of the resulting benchmark(s) to protect field populations requires a leap of faith that the distribution of the sensitivities of tested species is representative of the distribution of the sensitivities of field species."

Consideration of Listed Resources in EPA's Decision-Making Process

EPA's decision-making process (a.k.a. the *Guidelines*) does not explicitly require EPA to consider toxicity data on endangered or threatened species, although one species in particular, *Oncorhynchus mykiss* (specifically the freshwater phenotype, rainbow trout) is a commonly tested fish species. How EPA incorporates threatened and endangered species into their approval

of state and tribal water quality standards varies across regions. Certain regional offices of EPA have completed Section 7 consultations on their approval of state water quality standards for a subset of the numeric standards. This national consultation represents the first of a series of Section 7 consultations with EPA on their recommended criteria and EPA's subsequent approval of state and tribal water quality standards that are based on the recommended 304(a) aquatic life criteria. This enhanced coordination at the national level was envisioned under an MOA between the Services and EPA (66 FR 11202).

There is a critical difference between decision-making for the purpose of criteria setting and conducting a risk assessment on a particular species or group of species (Suter and Cormier 2008). The benchmark calculation used in EPA's decision to recommend a particular criterion "rests on an assumption that selecting a percentile [e.g. 95%] is an appropriate way of specifying a level of protection (Stephan 2002)." Whereas, the Section 7 consultation solves (or attempts to solve) the risk of exposing listed species to a particular federal action or set of actions, in this case the risk of exposing listed species to chemicals at particular concentrations. Unlike criteria development, Section 7 consultations begin by assessing the effect of the chemical to the individual of a listed species. This endpoint differs greatly from the population level response evaluated during criteria development.

To bridge the gap between the aquatic-life criteria decision-making process and information needed to conduct Section 7 consultation, EPA with the assistance of the Services, developed the *Draft Framework for Conducting Biological Evaluations of Aquatic Life Criteria: Methods Manual*. The Methods Manual describes a process for evaluating whether the CMC protects acute mortality of listed species, and whether the CCC protects listed species under longer exposures. Additionally the Method introduces a process for evaluating the effects expected from a diet of aquatic organism contaminated with the chemical of interest to levels that would result from concentrations consistent with the criterion. The Method also addresses toxicity of the criterion chemical to the food items of listed species to determine if listed species are likely to be adversely affected by a loss of food. The basic goal of the Methods Manual was to produce robust decisions for determining when the aquatic life criteria for a specific chemical is likely to adversely affect (or not) a particular listed species, and whether formal consultation is required.

The Methods Manual Approach to Estimating Acute Responses. To evaluate whether a listed or proposed species would respond to a particular chemical when exposed at the criterion value, the Methods Manual uses a risk paradigm or risk ratio for conducting toxicity screening that is based on the numeric value represented by the CMC[9] as the "assessment exposure concentration" (represented by C_A), divided by the "assessment effects concentration" (EC_A).

$$R = C_A/EC_A$$

The EC_A is an estimate of the highest chemical concentration that EPA portends would cause an acceptable small adverse effect and for acute effects that estimate is derived when the mean acute

[9] The BE and the *Methods Manual* refer to this as the "maximum exposure concentrations allowed by the criteria", but this is deceptive as the approved standard allows that discharges may exceed the established value for the CMC under certain circumstances. See our discussion under *Concentrations of Cyanide in U.S. Waters*. Therefore, we note here that the C_A = CMC (or the CCC).

value divided by 2.27[10]. For acute toxicity, the small level of effect is EC_0 to EC_{10}. Under this simple paradigm when $C_A < EC_A$ then the chemical concentration established by the aquatic life criteria "is not likely to adversely affect" listed species. Conversely, when $C_A \geq EC_A$ then the chemical concentration established by the aquatic life criteria is considered "likely to adversely affect" listed species (see *Methods Manual*, page 9). This risk paradigm, defined by the risk ratio, forms the foundation of the each aquatic life criteria consultation.

For listed species for which acute data exist, the relationship is straightforward. Using the mean acute value calculated for rainbow trout or steelhead exposed to cyanide we illustrate the calculation. For example,

If the steelhead mean acute value = 44.73 µg CN/L,

Then $EC_A = 44.73/2.27 = 19.70$

And, $R = 22.36/19.70 = 1.14$[11]

Under this framework, a species with an $R < 1$ is not likely to suffer lethal consequences when exposed at the CMC, and a species with an $R \geq 1$ is more likely to suffer lethal consequences when exposed to the pollutant of concern at the CMC. Using this framework, the farther the species' R-value is away from 1, the more confidence there is in the determination that the species is (or is not) protected when exposed to cyanide at the CMC.

For listed and proposed species without species-specific data the EC_A is calculated using data from surrogate species. Since we do not have species-specific data for most listed species; most of the assessments will likely either estimate LC_{50}s for species using the Interspecies Correlation Estimations (ICE) model or Species Sensitivity Distributions (SSD). EPA developed the ICE model using taxonomic level information for endangered species. ICE models are based on regression analyses of LC_{50}s measured for a listed species to LC_{50}s measured for the same chemicals for commonly used surrogate species, preferably based on a minimum of five test chemicals. If surrogate species have been tested for the chemical of interest, but the listed species of interest have not, the relationships are used to estimate the LC_{50} for the chemical and species of interest. When an ICE model is not available for a listed species, then an ICE model for the genus or family is used. In this instance, each higher order ICE model must contain at least two species that represent the genus or family for it to be useful. Due to the uncertainty in the correlations, EPA stated in the *Methods Manual* that they intended to estimate the LC_{50} using the lower 95% confidence bound of the ICE. On the other hand, the SSD is calculated from several surrogate species within the same taxonomic unit as the species of interest, to define possible LC_{50}s for the species of interest. According the *Methods Manual*, to increase the confidence in protecting listed species the 5th percentile in this distribution will be used, such that

[10] Note that we previously refer to this divisor as 2. The actual factor is 2.27, the inverse of 0.44, and "is based on 219 acute toxicity tests which showed that the mean concentration lethal to 0-10 percent of the test population was 0.44 times the LC50 (43 FR 21506, 18 May 1978)." In practice, such as the 1985 CN criteria document, EPA uses 2, but in the *Methods Manual* and this consultation EPA chose to not round to the nearest whole number but to use the fractional component as it was originally published in the *Federal Register*.

[11] If the CMC and the EC_A had been calculated with the same divisor, either 2 or 2.27, then in this example the rainbow trout R value would equal 1 because the CMC for cyanide was set using the rainbow trout SMAV.

the actual toxicity for the listed species should be higher than the chemical concentration estimated. When an ICE model was available for the listed species, or within the genus of the species of interest, the ICE model was given preference over the SSD. The *Methods Manual* lists a six-step approach for deriving EC_A estimates using surrogate data given the data that are available for closely related surrogates.

Given the lack of empirical information on the effects of many toxics on listed and proposed species, the Services and EPA will have to estimate to the best of their ability the potential effect using information from other species. Clearly, the validity and robustness of this risk ratio approach as a conceptual framework depends upon the value calculated for the EC_A. That is, the strength of the value (or range of values) represented by the EC_A depends ultimately on the identification, assimilation, and interpretation of evidence (i.e., the use best available scientific and commercial data) used in its calculation, which we expect will for most consultation predominantly come from surrogate species.

The Methods Manual Approach to Estimating Chronic Responses. To evaluate whether a listed or proposed species would respond to a particular chronic exposure to a particular chemical, the Methods Manual uses the same risk paradigm as described previously. For chronic toxicity, we used the numeric value represented by the CCC as the as the "assessment exposure concentration" (represented by C_A), divided by the EC_A.

As with the acute EC_A, the chronic EC_A represents an estimate of the highest chemical concentration in water or food that would cause an acceptable small adverse effect. For chronic toxicity, the acceptably small level of effect is the NOEC. Studies on the chronic effects of cyanide on listed species are few, and the literature search conducted by EPA was for a wide variety of species that have been tested. For chronic toxicity, the EC_A is based on the acute toxicity to the listed species, and the acute to chronic ratio (ACR) of surrogate species. The ACR is calculated as follows:

$$ACR = SS\ LC_{50}/SS\ NOEC$$

Where: SS LC_{50} is the LC_{50} for the surrogate species

SS NOEC is the No Observable Effects Concentration for the surrogate species

EC_As are estimated using the following equation:

$$EC_A = LS\ LC_{50}/\ ACR$$

Where: LS LC_{50} is the LC_{50} for the listed species

So for example, if the fathead minnow SS LC_{50} = 138 µg CN/L,
And, the NOEC = 13 µg CN/L
Then, ACR = 10.6

The listed species LC_{50} is then divided by the ACR to derive an EC_A, which is compared to the CCC. Using this framework, when the $C_A < EC_A$ then the chemical concentration established by

the aquatic life criteria "is not likely to adversely affect" listed species. Conversely, when the $C_A \geq EC_A$ then the chemical concentration established by the aquatic life criteria is considered "likely to adversely affect" listed species (see the *Methods Manual*, page 9).

Once the analysis produces a $C_A \geq EC_A$ for a particular listed species and contaminant combination, the *Methods Manual* provides little insight on the next step in EPA's evaluation. The *Methods Manual* merely states that when a particular chemical combination is classified as "likely to adversely affect" a particular listed species, these will require additional consideration and analysis to determine "under what circumstances risks are unacceptable (*Methods Manual*, page 9)." Unfortunately, the *Methods Manual* does not clarify for the reader what type or extent of "additional consideration and analysis" is necessary in such circumstances, nor does it provide a definition of when risks would be considered unacceptable (or acceptable). In contrast, the implementing regulations for Section 7 consultation state that "Each Federal agency shall review its actions at the earliest possible time to determine whether any action *may affect* listed species or critical habitat. If such a determination is made, formal consultation is required… (emphasis added; 50 CFR 402.14)."

Neither the implementing regulations for Section 7 consultation nor the ESA use the terminology "unacceptable" as a qualifier to describe effects to listed species. Therefore, it is unclear what EPA intended by this statement in the *Methods Manual* in terms of their Section 7(a)(2) consultations. An obvious unacceptable effect under Section 7 would be when an agency's action is likely to jeopardize the continued existence of listed species, or destroy or adversely modify critical habitat. Arguably, a reduction in the fitness of an individual of a listed species may also be considered unacceptable. The term's use is not defined under the ESA.

EPA's *Guidelines* may provide some insight into what EPA considers an unacceptable effect (Stephan et al. 1985). According to the *Guidelines*, the "protection of aquatic organisms and their uses should be defined as the prevention of *unacceptable* long-term and short-term effects on (1) commercially, recreationally, and other important species and (2)(a) fish and benthic invertebrate assemblages in rivers and streams, and (b) fish, benethic invertebrate, and zooplankton assemblages in lakes, reservoirs, estuaries, and oceans (emphasis added; Stephan et al. 1985)." According to Stephan (1986) his use of the term "unacceptable" in EPA's *Guidelines* was intentional because it allows for flexibility in determining the level of protection that a waterbody might receive and recognizes that such decisions are based on value judgements. When the validity of a criterion derived for a particular body of water is "based on an operational definition of 'protection of aquatic organisms and their uses' that take into account the practicalities of field monitoring and the concerns of the public" as suggested by EPA's *Guidelines*, then what drives the decision as to what constitutes an unacceptable risk is the level of protection (or conversely, adverse effect) that a particular criterion would have on a particular state or tribe's *designated uses* for their waters. The designated uses assigned to a particular waterbody by a state or tribe are explicit value-statements of what a particular state or tribe wants to protect their water resources for.

It follows that an unacceptable risk under EPA's decision-making process is one that fails to protect the designated uses for a waterbody. This is also consistent with EPA's review and approval of state standards. If EPA's line of inquiry as established through the *Methods Manual*

leads them to a "may affect" or more specifically, a "may affect, likely to adversely affect" and assuming that the only risk that would be considered unacceptable is if the critierion under review fails to protect designated uses, the question that remains is whether EPA generally considers endangered and threatened aquatic species (and aquatic dependent as defined by the *Methods Manual*) a designated use. If listed aquatic species, however, are not specifically identified as a designated use by a particular state or tribe, we would ask whether EPA, states, and tribes would generally protect listed aquatic species, as part of the broader definition to protect species that are defined as "important", part of the aquatic "assemblage", or "fish and wildlife." That is, would listed species fall into any of the categories identified by the *Guidelines*:

1. commercially, recreationally, and other important species, and
2. (a) fish and benthic invertebrate assemblages in rivers and streams, and
   (b) fish, benethic invertebrate, and zooplankton assemblages in lakes, reservoirs, estuaries, and oceans?

The third category, "fish and wildlife" comes not specifically from the guidelines but from the language adopted by many states to describe their designated uses. The answer to the question "does EPA consider threatened and endangered fish or benthic invertebrates part of any of these categories?" is critical to understanding EPA's decision-making process both pursuant to the *Guidelines* and the *Methods Manual*, and this consultation.

Designated Uses

The *Approach to the Assessment* section of this Opinion identified that EPA's approval of a state water quality standard involves more than merely establishing a numeric value for a particular chemical pollutant, but also requires a positive finding from EPA that a state has adopted uses that are consistent with the requirements of the CWA and that their proposed criteria protect those designated uses. Thus, state designated uses are an action interrelated to EPA's approval of any state standards that rely on EPA's recommended CMC and CCC values. When a state modifies EPA's criteria or proposes their own water quality criteria, then EPA must evaluate and find that the criteria protect a state's designated uses. When EPA recommends a criterion or promulgates a federal water quality standard, EPA states that it would generally find its criterion support the designated uses and the goals of the CWA: to restore and maintain the chemical, physical, and biological integrity of the Nation's water (objective of the CWA); and provide for the protection and propagation of fish, shellfish, and wildlife (the interim goal). Thus a state or tribe that has identified acceptable designated uses under the CWA can expect that if they adopt EPA's recommended water quality criteria that EPA would approve the standard. When EPA approves state or tribal water quality standards, that approval implies that those standards protect the designated uses of the state's waters when state waters are exposed to chemical pollutants at levels consistent with the criteria.

Whether a state's water quality standards actually protect the designated uses is unclear, and likely varies by circumstance (e.g., pollutant, state, and use). A designated use is a goal statement for a water body that reflects the social and political value of the water. Like numeric criteria, each state has discretion to set their own designated uses. As a minimal standard, the

CWA requires states adopt use designations consistent with the provisions of sections 101(a)(2) and 303(c)(2) of the CWA. Thus, a state must adopt uses that provide for the protection and propagation of fish, shellfish, and wildlife, and other uses such recreation, agriculture and industry. If a state designates a use that does not address the "fishable and swimmable" goal, the state must complete a use attainability analysis (UAA) that justifies why such uses are not feasible, and that the state is establishing the highest attainable use, instead. A state has the discretion to make their uses as restrictive or loose as they desire, as long as they meet the "fishable and swimmable" goals of the CWA. While the designated use is a qualitative value statement for a waterbody, a criterion represents a scientific determination as to whether a particular water body can, given an ambient concentration of a pollutant, can still support the designated use (Gaba 1983). However, the designated use, while written in qualitative form, should be as specific as possible so as to be measurable or have meaningful and measureable surrogate indicators of goal (designated use) attainment (NRC 2001). According to the Government Accountability Office (2002), many states recognized that the linkage between their designated uses and their ability to measure attainment (or failure to reach attainment) was missing and acknowledged that they needed evaluation criteria to determine whether designated uses are being protected that are measured by reasonably obtainable monitoring data.

Accordingly, part of the problem that GAO (2002) and National Research Council (NRC 2001) noted was that many states' designated uses may be overly broad. Many states designated uses were established in the 1970s when they had only 180 days to do so. Consequently, many states adopted the very general goal of the CWA to provide for the protection and propagation of fish and wildlife (GAO 2002). According to the NRC (2001) the problem with such broadly defined designated uses is that broader the use designation and the weaker the linkage between the use and any measurable indicator of attainment, the greater uncertainty and higher likelihood of error in subsequent determinations of use attainment. We found that many of the coastal states and states that contain listed species under NMFS jurisdiction have updated their designated uses in the past ten years (Designated Use Table -see Appendix B). Currently, designated uses include such uses as fishing/harvest, propagation of fish, protection, natural state, viable populations, diversity, species richness, and species assemblages. In our review we found only a few specified that the use was for a native fish community, and a few that did not appear to have a designated use that included wildlife. We were also curious whether listed aquatic species are directly or indirectly protected as part of the designated uses coastal states had adopted.

We found only one state, California, and one territory, Puerto Rico that explicitly addressed threatened or endangered species as part of their designated use. California's designated uses include a broad statement that the waters must support the survival and maintenance of aquatic species that are protected, and Puerto Rico's designated uses note that endangered and threatened species are included as part of the broader category of desirable species (Table 34). Other states have revised their designated uses to incorporate the specific needs of certain threatened or endangered species (e.g., Oregon and Washington adopted designated uses for the protection of Pacific salmon). Washington's designated uses explicitly denote the following categories of aquatic life uses: char spawning and rearing; core summer salmonid habitat; salmonid spawning; rearing and migration; salmonid rearing and migration only and several others (WAC 173-201A-200). Washington's designated uses should provide additional protection for Washington's native char, bull trout and Dolly Varden, and several species of Pacific salmon that are listed as

threatened or endangered, as well as others that are not listed. This is likely an improvement over the more generalized goals of "for the protection and propagation of fish, shellfish, and wildlife" or "fishable".

Table 37. State designated uses that explicitly address threatened and endangered species.

State	Designated Use Name	Designated Use Description	EPA Effective Date
CA Regions 1, 2, 3, 4, 5, 6, 7, 8, 9	Rare, Threatened, Or Endangered Species	Uses of water that support aquatic habitats necessary, at least in part, for the survival and successful maintenance of plant or animal species established under state or federal law as rare, threatened or endangered.	8/18/1994
PR	Class SB, SC, SD	Coastal waters and estuarine waters intended for use in primary and secondary contact recreation, and for propagation and preservation of desirable species, including threatened or endangered species.	6/26/2003

Careful consideration of the relationship between the value statement of use and the manner of evaluating attainment of the use is essential. When the relationship between the endpoint and the indictor is weak particular life stages of regionally important species and regional biota may be under-protected. Portions of the native aquatic community may be left unprotected by omission and unique life histories may be overlooked. For instance, Washington's designated uses may generally protect spawning salmon, but are under protective of early or summer migrating adult salmon for water temperature where warm water temperatures may interfere with gamete development during the migration and holding of the early migrating spawners (T. Hooper, pers. comm., October 28, 2008). Additionally, broadly defined designated uses are difficult to translate into meaningful and measurable criteria for determining whether uses have been attained. The closer a designated use is linked to its indicator, the chance of falsely concluding that the designated uses are being attained, when they are not, decreases.

To address this problem the NRC (2001) recommended greater stratification of designated uses at the state level to provide a logical link between designated uses and attainment of that use (NRC 2001). Considering that the designated use is the description of the desired endpoint for a waterbody and the criterion is the measurable indicator for determining attainment, using a stratified designated use framework could allow state's to measure ranges of attainment, create stronger linkages between endpoint and indicator, decrease decision risk, etc. The further the criterion for determining attainment is apart from the desired condition (the designated use) the greater chance for introducing (or magnifying) error into the decision-making process.

Figure 4 illustrates some examples of water quality criteria as the measurable indicator for attainment of designated uses in relationship to the desired endpoint, attainment of uses (after NRC 2001). The unnumbered square represents the designated use for the water (depicted by a value statement such as "fishable" or "swimmable"). Square 1, the furthest from the designated use, represents measures of pollutants at their source (end of pipe measurements). Square 2 represents the chemical criterion as the measure of the ambient water quality condition, but may

also include non-chemical measures (criteria) for physical attributes of ambient water quality such as dissolved oxygen and temperature. Square 3 represents criteria that are associated with physical or biological sources of pollution, and might include such measures as flow timing, pattern, non-indigenous taxa, channel sinuosity, etc. Square 4 represents biological measures of ambient water quality condition, such as those represented by indexes of biological community.

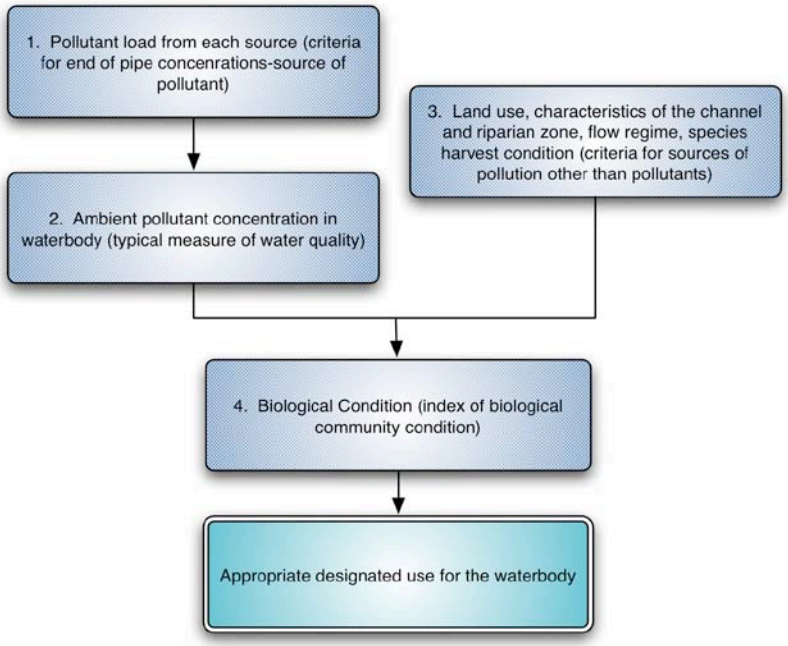

Figure 4. Types of water quality criteria and their position relative to designated uses (After NRC 2001).

A criterion, as described by NRC (2001) could be positioned at any point along the causal chain. However, if the desired endpoint is to restore and maintain the chemical, physical, and biological integrity of the Nation's waters, the biological condition is closest indicator to the desired endpoint. Not only is the proximate position of the biological indicator closer to the designated uses that describe the desired biological community, the biological community reflects the interplay between the physical, biological and chemical conditions of its environment. Under the stratified designated uses framework as suggested by the NRC (2001), states would adopt biological indicators as an intermediate and measurable indicator of designated use attainment. An index of biological health that considers a balanced community of native species versus the abundance and viability of alien species, loss of sensitive species and long-lived species; hydrological regime shifts (alterations in peak flows versus low flows, timing, intensity and duration), and so on, would provide a more holistic view of water body health and it's ability to meet public goals.

If the outcome or desired state for a designated use is preserving the biological integrity of the native community, then more meaningful measures as to whether that designated use is being supported by the aquatic life criteria are necessary. One advantage of a more explicit biological framing of designated uses is that threatened and endangered species can be expressly incorporated into the designated uses. When the designated uses are explicit, and provided the

criteria properly support such designated uses, the broader biological community should be protected. In turn, it would be reasonable to expect that enhanced aquatic conditions may prevent more aquatic species from becoming listed under the ESA, and promote the survival and recovery of currently listed threatened and endangered species suffering from poor water quality. In contrast, when the biological community is not a measured indicator of what EPA intends to protect through its chemical indicators, then EPA and the states are engaged in a water quality process, including designation of uses, to "merely to justify the specific numbers contained in pollutant criteria (Gaba 1983)." Absent robust indicators, Gaba (1983) notes that EPA, in reviewing the adequacy of state water quality standards is also engaged in an "ad hoc" assessment of whether the states are satisfying the minimum requirements of the CWA, and what kinds of fish or other wildlife are to be protected under a particular designation (Gaba 1983; Stephan 1985).

NMFS is particularly concerned about those instances where EPA finds that a criterion can adversely affect certain populations of listed species, while simultaneously protecting designated uses. Although individual listed species and the population they represent are part of the native aquatic assemblage within a waterbody and depend upon quality waters for protection and propagation, according to EPA it cannot disapprove a state's designated use solely on the basis that the designated use does not provide for the protection against "take" of listed species (EPA 2008b). Yet, where a listed fish species is failing to mate, rear, feed, migrate, or maintain viable populations for reasons attributable, in part, to water quality, it follows that the standard is not providing for the protection and propagation of at least some fish.

States and tribes that wish to avoid water quality related impacts to listed species could write their designated uses to include the protection of listed species, as a general category and, if necessary, include species specific designated uses. When states include the protection of the viability of listed species as a designated use, as a general matter, those states should be able to demonstrate that they would not be likely to increase a listed species risk of extinction due to chemical water quality impacts so long as they are meeting their designated uses. To demonstrate this level of protection would require a strong linkage between the designated use and the criteria states use for evaluating attainment. States that rely on chemical criteria without biological criteria to measure the attainment of designated uses, and fail to designate biologically meaningful indicators of use, may miss important changes in environmental health attributable to water quality impacts, including changes in the viability of listed species populations (see for instance, Karr et al. 2003). Currently, however, the approach used by most states in evaluating the effectiveness of the criteria (and other water pollution control efforts) at meeting the designated uses is unlikely to present a very complete or comprehensive picture of the biological health of their waters from chemical or physical stressors, and therefore cannot provide a very complete picture as to the successfulness of the water quality control program (GAO 2002; Karr et al. 2003). According to Gaba (1983) EPA has allowed states to trivialize designated uses as a scientifically credible endpoint by allowing designated uses to justify the specific numbers contained in pollutant criteria, which EPA has predetermined support any designated uses that would comply with the very general goal of the CWA.

Arguably it is even more important that EPA recognize that confidence in the ability of aquatic life criteria to protect the aquatic assemblage is increased when chemical and biological criteria

are used in concert to evaluate environmental impacts. The traditional laboratory based studies used as the basis for recommending aquatic life criteria require validation using more definitive and biologically rigorous metrics of biological integrity of natural systems. According to Adler et al. (1993, citing CRS, 1972 Legislative History, 76-77), the definition of "biological integrity" includes a condition in which the natural structure and function of ecosystems is maintained, and natural levels of biological integrity are those "levels believed to have existed before irreversible perturbations caused by man's activities." While the Senate report instructed that integrity under the CWA ought to be determined by reference to historical records on species composition (Adler et al. 1993). Biological integrity as defined by Karr and Dudley (1985) is "the capability of supporting and maintain a balanced, integrated, adaptive community of organisms having a species composition, diversity, and functional organization comparable to that of natural habitats of the region". If it were EPA's intent to design aquatic life criteria that protect the designated use for "fishable" waters, they would test the validity of *whether criteria are protecting the aquatic assemblage* in a waterbody, using rigorous biological indicators of aquatic ecosystem health.

The *native* aquatic assemblage is, arguably, the relevant endpoint envisioned by the Congress in establishing the CWA – when they stated the objective of the CWA is to restore and maintain the chemical, physical, and biological integrity of the Nation's waters -- and is certainly is that envisioned by Congress in adopting the ESA. Regardless, EPA could engage states in better defining the objectives of their uses classifications, and identifying measureable indicators of attainment. More importantly, EPA should review their operational definition of protecting the aquatic assemblage in a waterbody and how the definition should be expanded beyond the limited indicators of species richness and species evenness to better reflect current science for a biological healthy aquatic community, and incorporate their affirmative duties under both the CWA and ESA (EPA 2008b). Species richness and species evenness are not necessarily indicators of the health of the native aquatic fauna. They can, however, be combined with other important variables for assessing the biological condition of a water body such as: species diversity, trophic composition, fish abundance, fish health metrics (e.g., body condition), presence (or absence) of non-native species, presence (or absence) of tolerant (or sensitive) species. EPA might also adopt a stratified designated use approach, rigorous and measurable indicators of the native aquatic assemblage in those states where EPA retains primacy for setting water quality standards, and engage in meaningful field studies for assessing the status of surface water integrity that integrates chemical criteria with indicators of biological and physical condition. While EPA has stated the guidelines for establishing aquatic life criteria are meant to protect aquatic assemblages, the laboratory studies used in setting the aquatic life criteria for cyanide do not represent species' compositions from a natural community or ecosystem and consequently may fail to identify toxicant/population/community interrelationships. If field monitoring is not feasible, then mesocosm studies could provide EPA an opportunity to take a replicatable, laboratory-controlled approach to evaluate higher order effects in aquatic systems. Such studies may be useful in examining the indirect effects of reduced water quality and community response.

Stressors and Subsidies Associated with the Proposed Action

The primary stressor associated with the proposed action is aqueous cyanide. The following sections provide background on the characteristics of cyanide as a pollutant; including its uses and sources, observed concentrations, and other information that helps establish the exposure profile---the magnitude and spatial and temporal patterns of cyanide occurrence in the environment to which listed resources are exposed---for this analysis.

Exposure Analysis

Cyanide Sources and Production

We examined the typical sources of cyanide and the geographic distribution of those sources of cyanide to determine whether we would expect cyanide would co-occur with listed resources. This effort was based on the presumption that the fewer sources of cyanide there are across the United States, and the more limited their spatial distribution, the less likely that listed resources would be exposed to cyanide during their lifetime. If through this examination we would find that cyanide does not co-occur with listed resources, then we would conclude there is no exposure. The evidence leads us to conclude, however, that this is not the case. That is, based on the large number of sources of cyanide, their wide spatial distribution, and the increasing production of cyanide in the United States, we expect listed resources are more likely than not to be exposed to one or more sources of cyanide during their lifetime.

A common misconception is that cyanide is predominantly associated with gold mining or other mineral processing operations, which would tend to make this predominantly fresh water and perhaps rural pollutant. While cyanide is widely used in ore-extraction and cyanide related mine accidents have been widely publicized particularly when they have led to massive fish kills and human impacts, cyanide enters waterways from a wide variety of sources. Cyanide is ubiquitous in the environment, at least at low levels, as it is produced by a number of plants and microorganisms. However, cyanide is also produced synthetically to support industrial uses and is a byproduct of certain industrial processes (Leduc 1984; Eisler 1991; Dzombak et al. 2006).

Humans contribute the vast majority of cyanide to the environment. Cyanides are used widely in steel and heavy metal industries (e.g. electroplating), the manufacture of synthetic fabrics and plastics, as a pesticide and as an intermediate ingredient in herbicides, in road salts, and some fire retardants. Cyanide is also a byproduct of other activities such as municipal waste and sludge incineration and coking and gasification of coal (see Table 35). Of these sources metal industries and organic chemical industries are major contributors of cyanide into the freshwater aquatic environment, whereas, atmospheric cyanide, a by-product of forests fires, may be the primary source of oceanic cyanide except where cyanide enters coastal waters from fresh water sources (Leduc 1984; EPA 2005; Dzombak et al. 2006). Wastewater treatment plants across the United States can also be unexpected, but significant sources of cyanide to both fresh water and saltwater environments through several chemical processes, including dissociation of thiocyanide by chlorination or UV disinfection, chlorination in the presence of residual ammonia, nitrosation, and photolysis of ferrocyanate (Kavanaugh et al. 2003).

According to the 2002 United States Economic Census, there are 180 facilities engaged in gold-ore mining in 27 states across the nation, including Alaska, California, Idaho, Massachusetts, and Florida (U.S. Census Bureau 2002). The top four states, in terms of number of facilities, were Nevada, Colorado, California, and Alaska. In contrast, the manufacturing of photographic film, paper, plate, and associated chemicals occurs in more than 400 facilities and 24 states across the nation, and more than 3,000 establishments engage in electroplating and related activities in 41 states across the nation (U.S. Census Bureau 2002). The influx of cyanide to aquatic environments is likely as widely distributed across the landscape as the industries that use cyanide as part of their routine operations.

Cyanide is also synthetically produced in several states across the nation including Texas, Wyoming, West Virginia, Nevada, and Ohio (CMR 2008). In fact, the synthetic production of cyanide in the United States is a growing industry. The United States production of hydrogen cyanide (HCN) more than doubled in the past two decades from 330,000 tons in 1983 to 750,000 tons in 2001. Production growth between 1997 and 2000 increased about 1.7% per year (Dzombak et al. 2006; CMR 2008). The Chemical Market Reporter indicated that production demand in 2004 was estimated at nearly 2 million pounds. With demand exceeding current production of HCN, and price growth positive for the producers, HCN production and availability is expected to continue to increase in the United States. Incidentally, the United States does not export domestically produced HCN (CMR 2008).

The largest portion of the HCN produced in the United States is used in the textiles industry, for nylon production (47% is used for adiponitrile). Whereas, 27% is used in the production of acetone cyanohydrin for methyl methacrylate, the monomer for the transparent plastic polymethyl methacrylate also known as acrylic, 8% is for the production of sodium cyanide (NaCN), 6% is for methionine, 2% are chelating agents, 2% for cyanuric chloride, and 8% goes to miscellaneous uses including nitrilotriacetic acid and salts (CMR 2008). The demand for nylon remains high, with new growth and new applications still strong. According to CMR (2008) one such new application is in the automobile industry where metal components are being replaced by nylon parts. At the same time acrylic demands remain high, while the declining price of gold has reduced the demand for NaCN production, which had formerly been the primary driver for HCN production. With overall demand for HCN production growing in the United States, clearly cyanide is not a chemical that is being phased out of production or practical use but remains in prominent use. In fact, acrylonitrile (vinyl cyanide), a monomer in the synthesis of adiponitrile, is among the top 50 chemicals produced in the United States (Dzombak et al. 2006). While HCN facilities that support acrylonitrile production are in several states across the United States, several of the largest producers are in Texas (CMR 2008).

Table 38. Industrial Sources and Uses of Cyanide Compounds.

Source/Use	Form	Reference
Energy Production - Coal Gasification	Cyanide salts (potassium cyanide, sodium cyanide)	Way 1981; EPA 2008c
Steel manufacturing & heat-treating facilities, metal cleaning, electroplating		WHO 2004; Leduc 1984; EPA 2005
Ore-extraction (gold-mining, coke extraction)		WHO 2004; Leduc 1984; EPA 2005

Dyeing, printing of photographs		WHO 2004; EPA 2005
Production of resin monomers (acrylates)		WHO 2004
Pigments, paints	Ferrocyanides	Dzombak et al. 2006
Fire retardants		Little and Calfee 2002
Anti-caking agent for road salts		Dzombak et al. 2006
Detergents, dyeing of textiles		Dzombak et al. 2006
Pharmaceuticals (antibiotics, steroids, chemotherapy)		Dzombak et al. 2006
Fumigant/pesticide	Hydrogen cyanide, metallo-cyanide compounds	WHO 2004, Dzombak et al. 2006
Herbicides (dichlobenil, bromoxynil, bantrol)		EPA 2005, Dzombak et al. 2006
Road salts		EPA 2005
Production of other cyanides (e.g., sodium cyanide for gold mining)		EPA 2005, Dzombak et al. 2006
Pyrolysis of paper, wool, polyurethane		WHO 2004
Chelating agents for water and wastewater treatment		EPA 2005, Dzombak et al. 2006
Production of clear plastics		Dzombak et al. 2006
Methionine for animal food supplement		Dzombak et al. 2006
Wastewater Treatment Facilities (secondary treatment and/or disinfection w/ chlorine or UV)		Kavanaugh et al. 2003
Automobiles (with older or malfunctioning catalytic converters)		Voorhoeve et al. 1975; Karlsson 2004

With increasing uses and increasing production of cyanide we would expect that the amount of cyanide entering the environment would also be increasing (Way 1981). However, we have little data to ascertain if this is the case. According to the Toxics Release Inventory (TRI) data, total reported hydrogen cyanide releases have been increasing over the past 20 years (Figure 5). In 2008, some 4.5 billion pounds of HCN were released; over 432,000 pounds represent air emissions, while 58,000 pounds were discharged to surface waters (EPA 2008d). In comparison the long-term trend in releases to surface waters is declining, although this may not be a reflection of trends in actual ambient instream concentrations for several reasons. First, the data reflect one type of cyanide compound for which release data exists and does not include an assessment of the fate and transport of the released HCN including the ability of cyanide compounds to undergo transformation as under some environmental conditions that can increase or decrease its toxicological impact, and the TRI data does not include non-point sources of cyanide to the environment. Nonetheless, the TRI data, with its many caveats represents one of the only sources of data upon which trends in potential ambient cyanide can be discerned.

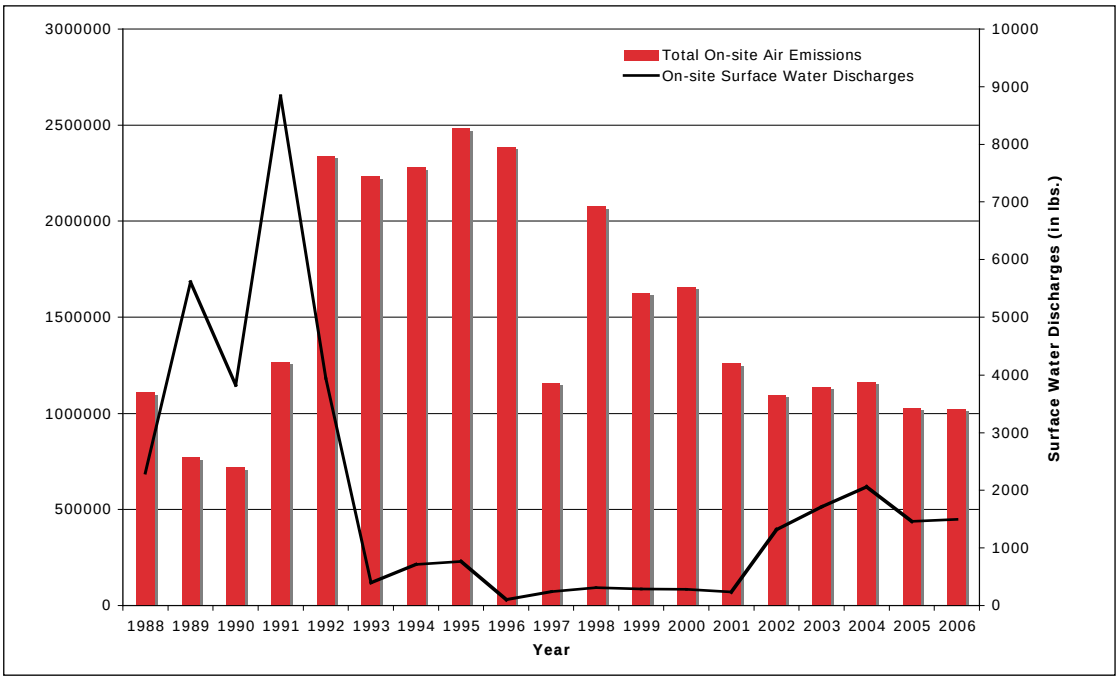

Figure 5. Toxics Release Inventory Data for HCN Releases in the United States to Air and Surface Waters, 1998 to 2006 (Source EPA 2008c).

The TRI data can also be used as indicator for understanding the geographic distribution of cyanide, in this case HCN, across the nation. The TRI data set, together with information on the distribution of manufacturers and user groups provide some insight into the distribution of cyanide sources and those areas where species might be at a higher risk of being exposed to ambient cyanide. While it is not clear that the volumes of cyanide discharged in these states typically resulted in aqueous concentrations that were problematic for listed species, the foregoing discussion illustrates that cyanide sources are widely distributed, and cyanide production and use is far from waning. On the contrary, cyanide production has increased in the past and is expected to increase in the future. As a result, we would expect listed aquatic resources are likely be exposed to one or more sources of cyanide during their lifetime. Due to the nature of the industrial sources, most exposure would occur in fresh water and marine coastal waters influenced by human activities. The predominant sources of cyanide to marine waters would be from direct discharges to marine waters (typically coastal outfalls), downstream transport from freshwater sources, and incidental releases from vessels (Dzombak 2006), which generally suggests that the further from shore a species or critical habitat occurs, the less likely it would be exposed to a wide variety of cyanide sources. However, with a large portion of cyanide entering the environment in gaseous form, we would expect some cyanide likely enters marine and fresh waters through atmospheric deposition.

Concentrations of Cyanide in U.S. Waters

As noted earlier, cyanide enters waterways through a variety of pathways and sources; however, the direct discharges (from point and nonpoint sources) pose the greatest concern for aquatic habitats because these sources are likely the dominant sources of cyanide loading to United States waters. To further characterize the exposure of listed resources in the aquatic

environment, we asked whether and to what degree we would expect listed resources would be exposed to cyanide concentrations at or near EPA's recommended CCC or CMC for cyanide. We examined data in EPA's data base STORET (STORage and RETrieval data warehouse) for information on potential concentrations of cyanide in the environment, as well as individual studies of cyanide loading from various sources. Based on our evaluation, we expect listed resources will be exposed to a wide range of concentrations of cyanide, and a wide number of cyanide compounds with varying toxicity. We expect that most waters likely have some low-level background concentrations of cyanide at most times. When exacerbated by anthropogenic sources, in-water concentrations may exceed EPA's approved numeric criteria for cyanide and the averaging recommendations adopted in state standards.

Studies have detected low levels of cyanide as a natural condition in some waterways, likely resulting from plant and microbial input. There also appears to be a seasonal component to the cyanide loading in waterways, which presumably varies with cyanogenic plant production, atmospheric deposition and rainfall patterns. A study of the occurrence of cyanides (free and combined) in small streams in the North-West Germany, using a technique that allowed a detection limit of 0.1 µg/L, found annual values of total cyanide in rural watersheds was 3 µg/L, while mean annual values of total cyanide in industrial watersheds were 20 µg/L with values reaching over 200 µg/L (Krutz 1979 in Leduc 1981, Krutz 1981). Cyanide concentrations varied seasonally, with the lowest concentrations occurring in spring and late summer and highest concentrations occurred in winter. Krutz (1979 in Leduc 1981) calculated maximum winter loads at 6 g CN^-/day and summer loads at 0.2 g CN^-/day. Principal factors attributed to winter peak loading included increased potassium loads that induced cyanogenic microorganism activity and winter precipitation and runoff events that increased delivery of atmospheric cyanide and cyanide formed by plants and terrestrial microorganisms to the water. Seasonal peaks were more frequently observed in the small catchments, although seasonal peaks were also observed in medium to large sized catchments (Krutz 1979 and PPWB 1978 in Leduc 1981). On the other hand, Tarras-Walberg et al. (2001) found concentrations were highest when the river under study was in a low flow period. In many cases, the low flow period for a catchment would correspond with low-flows and peak vegetative growth within a basin. Consequently, small catchments tend to be more closely associated with streamside vegetation and allochthonous input of cyanogenic (and other) plants, which would explain the summer and low-flow peaks observed by Krutz (1981) and Tarras-Walberg et al. (2001).

Cyanide also enters waterways through the indirect pathway from airborne sources, such as burning waste biomass for energy conversion, crop burning, prescribed forest fires and wildfires, and through the atmospheric release of cyanide from industrial sources and the eventual transformation to aqueous cyanide. Barber et al. (2003) found that free cyanide concentrations in stormwater runoff collected after a wildfire in North Carolina averaged 49 µg/L, an order of magnitude higher than in samples from an adjacent unburned area (Barber et al. 2003). Atmospheric deposition of HCN may be one of the most significant sources of HCN to ocean waters, excluding coastal areas. However, according to Dzombak et al. (2006) the concentration of HCN in ocean waters is likely to be low (less than 1 µg/L than the criterion value for salt water).

Studies evaluating the direct discharge of cyanide to waterways indicate that the concentrations

entering water are as variable as the sources themselves. Studies have shown that stormwater melting off roadside snow has a much greater capacity to accumulate and retain heavy metals and other pollutants than summer stormwater runoff. In a study of urban highway sites, concentrations of cyanide and metals were orders of magnitude higher than at the control sites and exceeded storm water (rain) runoff concentrations by one to two orders of magnitude. Cyanide concentrations, although demonstrating some variability, remained relatively constant at all sites (averaging 154 µg/L) or increased according increasing application rates of deicing salts that contained cyanide compounds as anti-caking agents (Glenn and Sansalone 2002). A study on the effect of cyanide on the anaerobic treatment of synthetic wastewater noted that cyanide is produced on an industrial scale of 2–3 million tons per year and, therefore is in many different industrial wastewaters. The concentrations encountered in industrial waste generally are in the range 0.01–10,000 mg/L, most of it in complexed forms of cyanide, which are less toxic than free cyanide but can transform to free cyanide or HCN. Cyanide contamination also occurs in the processing of agricultural crops containing high concentrations of this compound, such as cassava[12]. Systematic surveys of large wastewater effluents in Southern California suggest that free cyanide is routinely found in wastewaters, at low levels. In different years reported from 1992 – 2002, mean cyanide concentrations in effluents ranged from <2 to 30 µg/L (Steinberger and Stein 2003). Data from the US National Urban Runoff Program in 1982, revealed that 16% of urban runoff samples collected from four cities (Denver, Colorado; Long Island, New York; Austin Texas; and Bellevue, Washington) contained cyanide concentrations ranging from 2 to 33 µg/L (Cole et al. 1984 in ASTDR 2006). While demonstrating variability in the concentrations of cyanide found in some discharges, these studies also indicate that cyanide concentrations can be quite high at times.

The Difficulties of Measuring Cyanide in Water

Dzombak et al. (2006) refer to measuring cyanides as "a regulatory dilemma" because most analytical methods used in the field do not target specific cyanide compounds, rather the methods report various cyanide groups. EPA's recommended aquatic life criteria are specified in terms of free cyanide, yet the conventional sampling methods provide for measurement of a group of cyanide compounds. Methods include total cyanide, weak-acid-dissociable cyanide (WAD), cyanide amenable to chlorination (CATC), available cyanide by ligand exchange, and free cyanide. Total cyanide, the most frequently conducted sampling method, measures free cyanide and metal-complexed forms of inorganic cyanide, while WAD measures weak metal-cyanide complexes plus free cyanide. Much of the older data available in such databases like STORET were measured and reported in terms of total cyanide, which although it could be used as a surrogate of the amount free cyanide in a sample, doing so would lead to an overestimate in the amount of free cyanide in the samples because total cyanide includes free cyanide, WAD cyanide plus the relatively non-toxic iron-cyanide complexes. When EPA published their recommended aquatic life criteria for cyanide in 1985, they recognized the incongruity between publishing numeric criteria for free cyanide, and the fact that no EPA approved sampling method was available at the time that would measure free cyanide (EPA 1985). Therefore, in 1985 EPA recommended that states apply the criteria to total cyanide, acknowledging that doing so may

[12] Although we are not aware whether the U.S. has any cassava processing plants, there are over 1,000 cyanogenic plants including many sorghum grains that may contribute to cyanide contamination when processed. Cassava is merely one of the best documented sources of cyanide contamination attributable to cyanogenic plant processing.

make the water quality standard over-protective. An approved method for measuring free cyanide is now available, but unfortunately a translator has not been developed to convert data on total cyanide to free cyanide (Kavanaugh et al. 2003).

At the same time, there is a concern over measurement precision with data found in sources such as STORET. Measurement precision varies among sampling methods and certain chemicals and procedures can interfere with measurements as well. Measurements are frequently conducted via colorimetric, titrimetric, or electrochemical finish techniques (Dzombak et al. 2006). Measurements of total cyanide are limited to detection in reagent water matrix of about 1 to 5 µg/L and do not measure: cyanates, thiocyanates, most organic-cyanide compounds, and most cobalt and platinum cyanide complexes (Dzombak et al. 2006). Problems with sample storage, regulatory criteria, and the methods for testing and their sensitivity are a concern (Eisler 1991; Dzombak et al. 2006). Eisler (1991) notes that due to the volatilization of cyanide, periodic monitoring is not informative (for example, monitoring once per quarter [for instance, see the permit requirements in EPA 2008e) except perhaps, where continuing or chronic conditions persist. Consequently, Eisler (1991) and others recommend that continuous monitoring systems are necessary, with particular emphasis on industrial dischargers, to understand the fate and transport, critical exposures, and relative contributions of human and natural sources of cyanide in the aquatic environment. The availability of data from case studies using continuous monitoring systems would significantly increase our understanding of cyanide in the aquatic environment, and provide us important exposure profiles for evaluating approved water quality standards. Unfortunately, we were not aware of any such data sets that we could examine as part of this analysis.

STORET – EPA's Main Repository for Water Quality Data

Since we do not have data on long-term studies using continuous monitoring systems to evaluate cyanide discharges, we conducted a query of EPA's STORET database to further characterize cyanide entering the action area for this consultation. STORET, EPA's main repository for water quality data, contains information on water quality collected from a variety of organizations across the United States, from small volunteer watershed groups to state and federal agencies (http://www.epa.gov/STORET/index.html). Our review of STORET data indicates that many dischargers reported no-detectable amount of cyanide in their samples, which in some case may have been a limitation of the sampling method and does not necessarily suggest that the water contained no cyanide or alternatively it may suggest that the discharges were free of cyanide – either way, we do not know. We searched the STORET database and found records spanning 1964 to 2008 (August), most of which were recorded as total cyanide. Some states of particular interest, like Washington, where NMFS has listed salmonids and where the TRI database suggests there have been large discharges of HCN to surface waters, were not represented in STORET. While data were available for several other states, data was often sparse for many of the coastal states where NMFS' listed resources occur. When we queried according to the data fields for "rivers, lakes, reservoirs, and canals" the database returned only one sample for Alaska and Oregon, 13 to 51 samples for states such as California, New Jersey and North Carolina. The largest number of samples in this category was from Florida. We compared the sample data to approved water quality standards for cyanide and found that 4 of 13 values reported for California (31%) exceeded the CCC and the CMC. Upon closer inspection it appears that most

of the California data came from reservoirs and streams in the Mojave Desert that were presumably impacted by gold mining. The minimum value above the water quality standards was 300 µg/L and the highest reported value was 5,000 µg/L. For New Jersey, 17% of the reported values were above the CCC and the CMC. The highest reported concentration of cyanide above the approved water quality standard was 130,000 µg/L CN_T –there were two samples at this concentration in the data set, taken two weeks apart. Two months later, during the same year a concentration was sampled of 84,000 µg/L CN_T. All three of these samples were taken from the Ramapo River (near Mawhaw, New Jersey). Similarly, in Mississipi data show that state water quality standards were exceeded in 13% of the reported samples. When we queried STORET for data from marine waters we found only 5 reported values. All the samples were taken in Puerto Rico in about a 9-month span beginning late 2005. The mean concentration for this sampling station was 4.6 µg/L CN_T, four times higher than the approved water quality standard for marine waters, while the minimum reported concentration was 0 µg/L CN_T and the maximum concentration was 20 µg/L CN_T.

There are several very strong arguments that can be made questioning the utility of the STORET data for establishing an exposure profile for aquatic species. Not the least of which are the arguments that (a) the vast majority of information on cyanide in STORET is represented by total cyanide (CN_T) and a translator is yet to be developed that would allow us to determine the proportion of free cyanide (the most biologically toxic form) represented by the data on total cyanide, (b) the scarcity of data generally provides us little understanding of the spatial or temporal patterns of cyanide concentrations in United States waters, particularly since some states do not report their monitoring to STORET (or perhaps those states are not monitoring for cyanide), and (c) there are insufficient replicate data in STORET to provide any meaningful illustration of the trends in cyanide discharges within a particular locality. Despite the limitations of the data in STORET, it (with TRI data) represents some of the best available information we have on cyanide discharges across the United States. The STORET data however, does illustrate that listed resources may be exposed to a wide range of cyanide concentrations in receiving waters and that those concentrations may vary widely relative to EPA's approved (and recommended) national numeric criteria.[13]

Given typical monitoring schemes in many permits the probability that a particular facility would detect an exceedence event is quite low. A typical permit may require sampling once a week, once a month, or less frequently, and will often conduct their sampling using grab samples[14] (see for instance, permit requirements in EPA's 2008 Multisector General Permit). To determine whether or to what degree grab samples might detect events in which water quality criteria had been exceeded, we considered several scenarios. In the first scenario, we considered a facility that has 52 discharge events a year that result in elevated cyanide concentrations and assumed each discharge event lasted eight hours. In this scenario, there would be a 95% probability that the event would not be detected by a grab sample. Conversely, there would be a 5% probability that the event would be detected by a grab sample. If we increased the number of discharge events to 110 events per year with each event exceeding a particular criterion value and each

[13] An interesting question that merits exploration is whether EPA and/or the respective states considered many of the recorded peak discharges events that are included within the STORET database as violations of water quality standards.

[14] The grab sample technique is a rapid collection single point sampling method that does not integrate vertical or cross sectional variability, but captures point concentrations near the water's surface.

event lasts 8 hours, there would be a 90% probability that the event would not be detected by grab samples, and a 10% probability that the event would be detected by a grab sample [15].

A discharge containing a high concentration of cyanide would have to occur for more than 180 days a year (24 hours/day) to have a high probability of detection, which suggests that random grab samples generally are not likely to detect an exceedence event. Therefore, the sample data we found in our query of STORET may not have been produced by truly random samples, but instead, were produced by samples taken after known discharges containing high concentrations of cyanide. The fact that some samples data points reported high concentrations of cyanide could also be attributed to serendipity during the timing of sampling, or it could be that the discharged concentrations are high for frequently long intervals of time (e.g., more likely than low concentrations).

Allowable averaging schemes contained in many NPDES permits would further mask the true distribution of sample concentrations to which listed resources are exposed. That is, recall that the approved standards include a provision that allows for the average of the 1-hour concentration for the CMC, and the average of the 4-day concentration for the CCC. A facility that takes ten samples a year may have one sample exceeding 200 µg/L CN and nine samples with "non-detectable" concentrations and that facility would still fall within the recommended limit recommend by EPA. Figure 6 illustrates three hypothetical scenarios that demonstrate how individual discharges may exceed the approved numeric standards for the cyanide CMC, but still fall within allowable standard when averaged accordingly. The three alternatives presented illustrate three scenarios, all with the same central tendency despite widely different sample distributions. The result is that all three scenarios would be presumed equal in perceived risk under the recommended averaging scheme, despite the actual and widely disparate concentrations to which fish and wildlife would be exposed. As a result of the averaging and infrequent sampling schemes, the power of the data to detect problems is exceedingly low, and the fact that so many samples reported in STORET are unusually high is cause for concern and suggests that in some areas cyanide concentrations may exceed the numeric values defined by the cyanide CCC and CMC fairly often. Consequently, based on the best available data it appears that at least some listed resources would be exposed to cyanide at concentrations well above the approved CMC of 22.36 µg/L CN and the CCC of 5.221 µg/L CN, and at a frequency and duration which may result in demonstrable harm to aquatic life.

Factors That Influence Cyanide Toxicity

The risk to aquatic environments from cyanide releases depends on several factors including: the cyanide compound and concentrations released, pH, presence of iron and other metallic trace elements, solar radiation, air and water temperature, and dissolved oxygen levels to name a few (Doudoroff 1976; Smith et al. 1978; Dzombak et al. 2006). There are several compounds in the cyanide group, with varying degrees of complexity. Cyanide is formed by carbon and nitrogen, attached by three molecular bonds (C ≡ N). Complex cyanide compounds are formed when one or more CN compound forms with other atoms, such as hydrogen (H - C ≡ N). The resulting

[15] The probabilities in this paragraph were derived using the equation: Probability = $1 - (1-p)^n$ where p is the probability of a water quality exceedance event in a sample, n is the number of samples, so $(1-p)^n$ is the probability of not detecting a water quality exceedance event, *and* then $1 - (1-p)^n$ is the probability of detecting a water quality exceedance event in a sample of size n. Adapted from McArdle 1990.

compound is HCN. Hydrogen cyanide (HCN(g)) is a gas that is miscible in water, and its water form, hydrocyanic acid (HCN(aq)), is weakly acidic and the most toxic cyanide compound. Other compounds include sodium cyanide (NaCN), potassium cyanide (KCN), adiponitrile ($C_6H_8N_2$), and copper cyanide (CuCN).

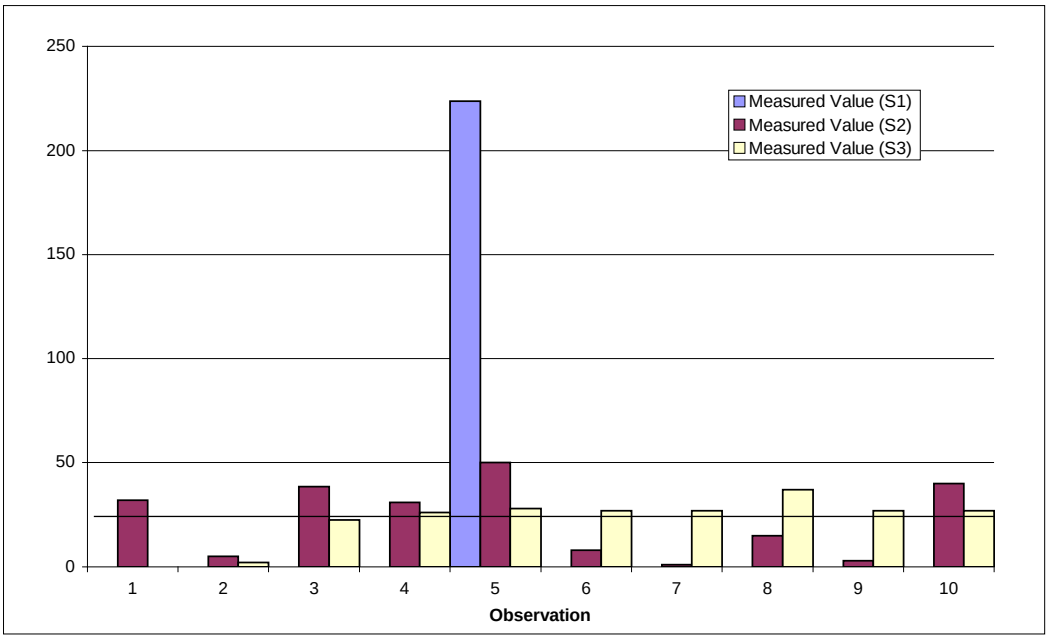

Figure 6. Three Hypothetical Discharge Scenarios that Comport with the Acute Water Quality Standard for Cyanide (Avg. CMC = 22.36 µg CN/L).

Free cyanide readily biodegrades, but the degradation is influenced by several factors including availability of oxygen, pH, carbon and nitrogen and the initial concentration of the cyanide compound released. Cyanide, through degradation, is converted to simple molecules like ammonia and carbon dioxide or it may it may be assimilated into the primary metabolism of bacteria, fungi, or plants (Dzombak 2006). Many forms of cyanide exist in the aquatic environment, including NaCN, KCN, metal-cyanide complexes, and organocyanides (e.g., acetonitrile), and metal-cyanide solids (e.g., ferric ferrocyanide). These forms have different chemical and toxicological properties. For instance, simple solids, like KCN and NaCN, are more soluble in solution and readily release free cyanide and HCN, which is the subject of this consultation. Solid forms of cyanide may exist in the soil of sites for years, and once exposed to water may result in dissolved cyanide reaching ground water and eventually surface waters (see Dzombak et al.'s [2006] discussion about the industrial legacy of cyanide box wastes at thousands of former manufactured gas plants in the United States). The chemical transformation of the cyanide compounds to HCN or CN^-, determines their toxicological significance to protected species, and their transport and fate in the environment.

The iron-cyanide complexes are the dominant form of cyanide found in soils and those most frequently encountered in dissolved form at concentrations in surface waters, making them the compound of most concern in managing water quality (Dzombak et al. 2006). The mobility of cyanide in soil and through groundwater depends upon precipitation, pH, the types of trace

minerals present, and organic matter among other things. In water, cyanide transport, fate and toxicity vary according to volume dispersed, pH, temperature, mixing and turbulence, dissolved oxygen concentrations, form and abundance of alternative nitrogen sources, biological use, and incidental light as some cyanide complexes display photochemical reactivity (Leduc 1981; Kavanaugh et al. 2003; Dzombak et al. 2006). Regardless of what form of cyanide is introduced into a system, cyanide transformation mechanisms are variable according to environmental factors, and Kavanaugh et al. (2003) caution that managers need to acknowledge that multiple species of cyanide typically coexist, introconvert, and degrade in a system, and through its transformation the toxicological effect of cyanide may increase or decrease. Consequently, knowledge of cyanide compounds and their ability to undergo transformation is important to managing it in aquatic environments (Kavanaugh et al. 2003).

The Exposure Profile – Summarized

As noted earlier in this section (*Exposure – Cyanide Sources and Production*), we began our exposure assessment by examining general sources of cyanide across the United States, their spatial distribution and their production trends. We also examined available data to characterize CN concentrations in waters of the United States (the action area), and we compared the recommended (approved) numeric standards for cyanide to those values represented in data sets collected by EPA to determine if the numeric standards are representative of actual cyanide concentrations observed in United States waters. We also evaluated the ability of the data generally collected by EPA and authorized states to provide information that would help us make these comparisons.

Based on our analysis, we were unable to conclude that cyanide discharged in accordance with EPA's approved water quality standard has not co-occurred with listed and protected resources under NMFS' jurisdiction in the past, or that listed and protected resources would not be exposed to cyanide at some time in the future, such as over the course of the next 10 years[16]. As a result, we were unable to conclude that any particular listed or proposed resources should be excluded from our exposure analysis. The wide number of cyanide sources and uses and their broad geographic distribution suggests that some individuals of listed species, their designated critical habitat, and some individuals of species proposed for listing or their critical habitat proposed for listing, are all reasonably likely to be exposed to cyanide at some stage of their lives. Certainly, as the numbers of cyanide sources vary, the risk of exposure would also vary spatially and temporally across the action area. It appears that the potential for exposure may increase in urbanized areas, but rural areas are not free from potential sources of cyanide and some listed species would likely be exposed in these areas (e.g., gold mining and road maintenance activities are likely some of the sources in rural areas). In general anadromous fishes like salmonids and sturgeon, that traverse fresh and salt waters, would potentially be exposed to a greater number of cyanide sources throughout their life cycle, whereas listed marine species are more likely to be exposed to elevated concentrations of cyanide along the coasts than in deep or open ocean waters areas due to the combined effect of point and non-point sources from human activities. Both marine and fresh water species would likely be exposed to cyanide through deposition of

[16] We requested EPA identify a reasonable analysis period for their action, but did not receive assistance on this issue. Because the existing cyanide criteria were published in 1985 and have not been updated since, and we do not have any indication from EPA that they have plans to revisit their cyanide criteria, we used an analytical period of ten years. That is, our effects analysis considers the effects of continuing the approved cyanide criteria for an additional ten years into the future.

airborne releases. We did not find sufficient information to suggest that there were particular areas where listed species are not likely to be exposed to cyanide.

In a typical site specific assessment we would characterize the intensity of the listed resources and proposed resources exposure over time and space; however due to the inherent nature of this assessment, and the variability across sites and over time, such an estimate does not exist. Nor could we find data that we could use to assemble a case study of aquatic exposures in a particular space over a particular time. However, based on data collected by EPA (which is limited so the possibility for false positive errors (Type 1) and false negative errors (Type II) is high) it is clear that concentrations of cyanide have exceeded the approved standards in some locations and at some times. The data illustrate that the exceedances are sometimes orders of magnitude higher than the approved standards. Further, typical monitoring methods and the use of measures of central tendency on the collected data will often mask biologically important exposure scenarios. That is, we presented three alternative hypothetical sample data sets to illustrate that despite the distributions varied widely (i.e., even when individual events exceed the approved standards by 10 times) the perceived risk of the hypothetical sample sets would be presumed equal and in compliance with the approved water quality standard when using the central tendency as the measure of risk. In general, the monitoring and reporting practices routinely adopted in water quality standards severely reduce the utility of the data collected by EPA and states for characterizing typical exposure scenarios. Unfortunately, at the scale of this consultation and given the wide variability of the data available, it is not clear what might be a reasonable daily or longer-term potential dose for this analysis. Clearly, many factors influence the actual exposure of listed species in the wild and insufficient data are collected to evaluate the concentration, frequency and duration of allowable excursions, as well as the ambient concentrations to which authorized discharges are added. Simply, the criteria, as approved by EPA in state and tribal water quality standards, are the "protection level" to which the water quality based approach to pollution is applied. Absent better data to inform below and above criterion exposure events and other factors that influence exposure, we cannot confidently characterize the rarity or commonness of exposure scenarios that differ from the proposed criteria. Therefore, to anchor our response analysis for this consultation, we proceed with the core assumption that one or more life stages (all aquatic life stages) of all listed resources and resources proposed for listing would be exposed to cyanide at concentrations equivalent to EPA's approved (and recommended) numeric water quality criteria. Since the CMC and the CCC represent the basis for administering water quality programs under the water quality-based approach to pollution control, including monitoring to determine whether waters are attaining designated uses, benchmarks for evaluating BMP performance in NPDES permits, evaluating whether waters should be listed as impaired, and as effluent limits for TMDL permits, we believe this is a reasonable core assumption for this analysis.

Response Analysis

As noted in our Approach to the Assessment, response analyses determine how listed resources are likely to respond after being exposed to an action's effects on the environment or directly on listed species themselves. For the purposes of consultations on recommended or approved water quality standards, our assessments try to detect the probability of lethal responses, physiological

responses, and behavioral responses that might result in reducing the fitness of listed individuals. Ideally, our response analyses consider and weigh evidence of adverse consequences, beneficial consequences, or the absence of such consequences.

It is important to begin these analyses by stating that, to the best of our knowledge, few data are available from the actual exposures of endangered or threatened species to cyanide in the laboratory or natural settings. We are aware of a few studies on rainbow trout, the resident form of *O. mykiss*; however, these studies are typically conducted on artificially propagated individuals that come from populations with a long history of artificial propagation such that their genetic make-up may be altered from their wild counterparts, and as a result there is some risk that their responses could differ from their wild counterparts. That said we have no information that would suggest this is the case and are assuming that there would be no difference in responses between artificially propagated individuals and wild individuals. Therefore, rainbow trout are the best surrogate available for predicting the response of wild steelhead, and many other species as well, because we lack species-specific data for several anadromous salmonids. We also have very little data for marine species as a group and no data on listed marine mammals. In fact, a recent reexamination of EPA's 1985 nationally recommended criteria for cyanide conducted by the Water Environment Research Foundation (Gensemer et al. 2007) concluded that "due to the lack of cyanide toxicity data for these species or reasonable surrogates", there was insufficient information available to evaluate the protectiveness of the saltwater cyanide criteria to threatened and endangered marine species. Instead, more research is needed on these species (Gensemer et al. 2007). Without empirical information on the actual responses of endangered and threatened species to cyanide, we reviewed the best scientific and commercial information available on the responses of fish and wildlife to cyanide. We also relied on estimates of sensitivity produced by EPA's Interspecies Correlation Estimations (ICE) model. We used this information to make inferences about the probable responses of endangered and threatened species when exposed to cyanide at the approved CCC and CMC.

Generalized Review of Responses

Individual aquatic organisms are exposed to cyanide by inhalation, ingestion, and absorption through epidural layers and mucus membranes. Cyanide is rapidly absorbed and distributed through the body. Once exposed, the primary manner of transport is via the bloodstream. In the bloodstream cyanide inhibits cellular respiration. Cyanide inhibits cytochrome c oxidase, an important hemeoprotein found in the mitochondria, by attaching to the iron in the protein it blocks the electron transfer to oxygen causing cellular respiration to cease. As a result many enzymes and biological systems are inhibited by cyanide, including succinic dehydrogenase, carbonic anhydrase and others (see Ballantyne 1987). Inhibition of cytochrome c oxidase activity, and the mitochondrial electron transport system will cause the cell to no longer aerobically produce ATP for energy, and the tissue then switches to anaerobic metabolism and the depletion of energy rich compounds (Eisler 1991; Dzombak et al. 2006). The result is rapid depression of central nervous system and the autonomic control of respiration. The heart is also a likely target of toxicity. Several species have shown consistently high concentrations within the myocardium, similar to brain concentrations, irrespective of the route exposure (Ballantyne 1987). Symptoms of acute poisoning in fish may include distress, increased ventilation – gill movement, surfacing, frantically swimming in circles at the surface, violently swimming against

the bottom, convulsions, tremors, and finally death (Leduc 1981).

As a powerful and rapid asphyxiant, cyanide will cause death in a manner of minutes by hypoxic apoxia at lethal concentrations. Releases of cyanide at extreme lethal doses are likely rare based on known fish kills and STORET data, but they do occasionally occur. However, when they do occur, massive fish kills result. Some such events occurred in:

- Wissahickon Creek, Pennsylvania, where more than 1,000 fish were killed in 2006 due to the dumping of about 25 gallons of potassium thiocyanate, which is suspected of having interacted with chlorine in the nearby wastewater treatment plant (EPA 2006).
- Alamosa River, Colorado, where the Summitville Mine was responsible for contaminating 17 miles of the river and killing more than 15,000 trout in 1990 due to the escape of cyanide-laden pit waters. By the 1992, the site was abandoned by the mining company and was a notable superfund site, at high risk of additional leaks (Gavin 2004).
- Fall River, Oregon, where more than 22,000 trout died in 2002 when 1,000 to 2,000 gallons of fire retardant, which was released during fire fighting activities reached the waterway. The fire retardant was mixed with sodium ferrocyanide, which was used as corrosion inhibitor to protect the tanks the retardant was stored in (ODFW 2002).

Other events like these have occurred in the United States, and there have also been several events in other countries such as Ghana, Kyrgyzstan, and Canada, to name a few. Such events, while severe when they occur, tend to occur infrequently. Typically, we would find cyanide at much lower concentrations in the environment.

Cyanide although a potent asphyxiant, is also rapidly detoxified. The major determinant of the severity and rapidity of a response depends upon the rate of absorption versus the rate of detoxification, which are influenced by the rate and severity of exposure. Detoxification occurs primarily through the enzymatic transformation to thiocyanate, which is excreted by the kidney (Ballantyne 1987).

At sublethal doses, individuals may act stunned, which is why cyanide is widely used for the collection of tropical fish for aquariums. Sublethal doses can also inhibit reproduction, metabolic rate, egg production, spermatogenesis, oocyte development, lead to tissue necrosis, aggressiveness, impair food capture, and interrupt ion regulation and swimming ability (see Doudoroff 1976, Kimball et al. 1978, Leduc 1984, Eisler 1991). On the other hand, low-level exposure may also stimulate growth (Negliski 1973 in Dzombak et al. 2006; McCracken and Leduc 1980). Whether there are concomitant adverse effects to other physiological development process associated with growth stimulated by cyanide exposure is unclear. Rapid detoxification occurs at lower doses, as cyanide is metabolized to thiocyanate by two enzymes that are widely distributed in the body, and then excreted in urine over a period of days. Although thiocyanate (SCN^-) is the principle form of cyanide that is eliminated, it can also accumulate in tissues and is known to have antithyroidal properties. SCN^- inhibits iodine uptake by thyroid tissues and disrupts thyroid hormone homeostasis, which can result in the development of goiter. Cyanide does not bioaccumulate through the food web; however, the damage associated with prolonged exposure at low levels, recovery, and re-exposure may be cumulative. There is no evidence to suggest cyanide is mutagenic or carcinogenic (Ballantyne 1987).

Calculating a Response

Studies on the responses of listed resources and resources proposed for listing to cyanide and cyanide compounds are few. Directed studies on listed and proposed resources would generally rank highest for consideration, provided the studies were carefully designed, had large sample sizes (with small variances), and measured cyanide using a reliable test method. Such studies would generally provide the most reliable indicator of a listed species response, when exposed to cyanide in the wild. However, because data are not available for large number of fish and wildlife species EPA's *Guidelines* establish some minimum standards for deriving water quality criteria.

Generally, EPA would use the GMAV, which are calculated as the geometric means of the available SMAV to set the acute criterion, although this was not the case for their recommended aquatic life criterion for cyanide in fresh water. EPA calculated the acute freshwater value or CMC for cyanide (22.36 µg/L) to protect the recreationally and commercially important rainbow trout, the most sensitive of the species tested. Data were available for 15 different genera, and the most sensitive species of those tested was rainbow trout. At the time, rainbow trout was classified as *Salmo gairdneri*, and the other species in the same genera for which EPA had test data was the Atlantic salmon, which incidentally had a SMAV double that rainbow trout. Therefore, EPA chose to use the rainbow trout SMAV to set the acute criterion for cyanide. The acute criterion for saltwater was calculated using the GMAV from eight different genera, with *Cancer irratus* representing the lowest ranked GMAV. EPA then divided the FAV by 2[17] to derive the CMC. There was however, insufficient chronic toxicity data available to meet the minimum standards established by the *Guidelines* therefore EPA applied the ACR to the FAV to estimate the final chronic value. Unless there are other data to suggest the FCV is not sufficiently protective, the CCC is set to the FCV. For cyanide, once the ACR for four species was calculated, EPA took the geometric mean of the four freshwater species to derive the final ACR. Next the FAV was divided by the final ACR, to derive the final CCC. For saltwater, the CCC was set equal to the CMC because it was assumed that the acute sensitivity of the rock crab was a better indicator of the chronic sensitivity of the species than would be obtained otherwise. Table 36 contains a summary of the cyanide water quality standards and the top-ranked values used to calculate the CMC and the CCC.

Table 39. Summary of cyanide test results and subsequent water quality criteria[1].

GMAV	Fresh water		Saltwater	
Rank	Genus	GMAV (µg CN/L)	Genus	GMAV (µg CN/L)
4	Lepomis	99.28	Mysidopsis	118.4
3	Perca	92.64	Menidia	59
2	Salvelinus	85.80	Acartia	30
1	Salmo	63.45	Cancer	4.893
FAV (calculated from GMAVs)		62.68		2.030
FAV (SMAV for rainbow trout)		44.73		
CMC		22.36		1.015
ACR		8.568		2

[17] By dividing the FAV by 2, EPA believes that they have derived a CMC concentration "that will not severely adversely affect too many of the organisms (EPA 1985)".

CCC	5.221	1.015

[1]Table adapted from Gensemer et al. 2006; data from EPA 1985.

Acute Toxicity

Knowledge of the acute lethal effects of cyanide on fish has been gained through observations following accidental spills, intentional field application for lake/stream management and controlled laboratory studies. Cyanide is highly toxic with a relatively short half-life. At high levels of exposure, acute toxicity occurs rapidly (Leduc 1984). During intentional field applications exposed fish were observed exhibiting symptoms that include increased ventilation, surfacing, gulping for air, frantic swimming in circles, convulsions and tremors prior to death (Leduc 1984). Laboratory tests under controlled situations have revealed that not all life stages of fish are equally sensitive to acute cyanide exposure, that cyanide toxicity can be modulated by abiotic factors, and that there is a wide range in sensitivity among aquatic organism. For instance, Smith et al. (1978) demonstrated that bluegill, yellow perch, and brook trout juveniles were more sensitive than newly-hatched fry, where, as swim-up fry were the most sensitive fathead minnow life stages.

EPA and the Services conducted an extensive search for data for the consultation, which included a review of studies that had been used in the derivation of the cyanide criteria in 1985 and any new studies that had been conducted since 1984. EPA compiled toxicity data for 83 species of aquatic animals and plants (61 freshwater species and 22 saltwater species) as part of their BE for the cyanide consultation (EPA 2007). Based on this compilation, there appears to be a large range in sensitivity between the most sensitive (rock crab LC_{50} 4.89 µg CN/L) and the least sensitive species tested (river snail LC_{50} 760,000 µg CN/L). Freshwater species represented 9 phyla, 15 classes, 29 orders, 36 families, and 52 genera. Fishes were among the most sensitive freshwater taxa although there was substantial variability in sensitivity. Among the 24 freshwater fish species included in the list there was a 33 fold difference in sensitivity between the most sensitive (rainbow trout, *Oncorhynchus mykiss*, LC_{50} 59 µg CN/L) and the least sensitive (bata, *Labeo bata*, LC_{50} 1970 µg CN/L). The 8 most sensitive fish species belong to 3 different families, Salmonidae (3 species, 3 genera), Percidae (2 species, 1 genera), and Centrarchidae (3 species, 3 genera). Because of the relatively low number of species that have been tested within these families it is difficult to get a sense of the amount of intra-family variability in species sensitivity on the low end of the species sensitivity distribution. By contrast, the family cyprinidae was well represented with 10 different species representing 8 genera. Among those 10 species there is an 18-fold difference in sensitivity between the most sensitive (roach LC_{50} 108 µg CN/L) and the least sensitive (bata, *Labeo bata*, LC_{50} 1970 µg CN/L) species. Because of pronounced intra-family variation it is unlikely that the 8 species within the 3 most sensitive families represent the most sensitive species within those families.

Within the compiled data set, empirical data on the acute effects of cyanide was available for only two biological species under NMFS' jurisdiction—steelhead (representing 11 listed species (DPSs) of *O. mykiss*) and Atlantic salmon (*Salmo salar*)[18]. Consequently, EPA estimated EC_As

[18] Atlantic salmon are jointly managed with FWS. This species is addressed in the FWS' biological opinion on this action.

were calculated using ICE or SSD for six biological fish species under NMFS' jurisdiction, representing 19 listed species (ESUs and DPSs).

Steelhead/Rainbow Trout. Previous work by EPA and others suggest that rainbow trout are the most sensitive freshwater test species to cyanide. That is, the concentration of cyanide that induces mortality is lower than it is for many other species, with warmwater species generally exhibited greater tolerance. We found one additional study on the acute response of rainbow trout to cyanide that has been conducted since EPA calculated the 1984 aquatic life criteria. The study by McGeachy and Leduc was published in 1988 and analyzed the influence of season and exercise on the acute responses of rainbow trout to cyanide. The other studies on the lethal responses of rainbow trout to cyanide were available at the time EPA published their cyanide criteria in 1985. In 1985, EPA chose to use only 4 values for calculating the SMAV for rainbow trout (Table 37). EPA's reasoning for choosing those studies at the time, was because in a comparison of acute toxicity values for fishes they confirmed what Doudoroff (1976) had concluded earlier, that static toxicity tests generally produced higher response values than flow-through tests of equal, fairly prolonged duration (EPA 1985). As a result, they based the SMAV on the results of flow-through tests in which the concentrations were measured (EPA 1985). This comports with direction provided by the *Guidelines* (Stephan et al. 1985) which states:

- For some highly volatile, hydrolyzable, or degradable materials it is probably appropriate to use only results of flow-through tests in which the concentrations of test material in the test solutions were measured often enough using acceptable analytical methods
- For each species for which at least one acute value is available the SMAV should be calculated as the geometric mean of the results of all flow-through tests in which the concentrations of test material were measured.

Thus, the estimated mean acute value influences the estimated assessment effects concentration and the preliminary screen for making Section 7 effects determinations (also the estimated level of protection) under the *Method Manual*. For instance, Table 38 compares acute data from: all referenced studies used by EPA in their 1985 published recommendation for cyanide and used by Gensemer et al. (2007) in their recent review of the cyanide criteria, an approximation of EPA's calculated LC_{50} that they used in the BE to make their effects determination[19], and two alternative data sets to calculate the SMAV and EC_As for steelhead. Using only flow-through test data EPA (1985) and Gensemer et al. (2007) derived SMAVs of 44.73 µg CN/L and 46.53 µg CN/L, respectively. The difference in SMAVs is attributed to Gensemer et al.'s (2007) addition of values from the flow-through tests conducted by McGeachy and Leduc (1988), which were not available at the time the criteria document was published. Because the precise values EPA (2007) used in their BE calculation were not clear to NMFS when there were multiple test values available within a particular study, we used data values that allowed us to approximate their final LC_{50} value. For instance, we are aware EPA used data from Markings et al. (1984) but are not clear what particular values influenced their final LC_{50} calculation.

Marking et al. (1984), Bills et al. (1977), and Skibba were not used in the calculation by EPA (1985) or Gensemer et al. (2007) because the data were derived from static tests, which as noted

[19] EPA calculated a mean LC50 for steelhead (rainbow trout) of 59.22 (µg CN/L). We tried, but could not precisely replicate this value.

earlier tend to produce responses at higher concentrations. Neither EPA (1985) nor Gensemer et al. (2007) stated why the data from Dixon and Sprague (1981) were not used in the calculation. Although these studies were not used in the mean LC_{50} calculation, EPA (1985) and Gensemer et al. (2007) considered the studies as "other data".

Alternatives 1 and 2 in Table 38, NMFS followed suit with the *Guidelines* and relegated static-test data for later consideration but did not include these data in the LC_{50} calculation. The primary difference between Alternatives 1 and 2, however, was in the test data we included from McGeachy and Leduc (1988). McGeachy and Leduc (1988) compared the toxicity of cyanide under different swimming conditions-- "exercised" versus "non-exercised" conditions. The non-exercised trout were placed in white polyethylene tanks and surrounded with Styrofoam and black plastic to minimize disturbance. It appears that Gensemer et al. (2007) chose to use the data from "non-exercised" fish in their calculation. For comparison, we used only the data from "exercised" trout in Alternative 1 because these fish were kept in more realistic test conditions (i.e., more natural), whereas all the data from McGeachy and Leduc (1988) are used in Alternative 2.

Table 40. Comparison of Toxicity Values To Support Species Mean Acute Value Calculations for Rainbow Trout

Mean LC_{50} Value	**LC_{50} Value used to calculate SMAV (µg CN/L)**					**Reference**
	EPA 1985	**Gensemer et al. 2007**	**EPA 2007***	**Alt. 1**	**Alt. 2**	
90			90			Bills et al. 1977
57	57	57	57	57	57	Smith et al. 1978; Broderius and Smith 1979
27	27	27	27	27	27	Kovacs 1979
40	40	40	40	40	40	
65	65	65	65	65	65	
98			98			Dixon and Sprague 1981
98			98			
97						
96						
97						
67						
83						
95						
46			46			Marking et al. 1984
52			52			
54			54			
62			62			
75			75			
55			55	55	55	McGeachy and Leduc 1988
53		53	53		53	
50				50	50	
42		42	42		42	
56				56	56	
53		53	53		53	
56				56	56	
66					66	
97			97			Skibba 1981

64.28	44.73	46.53	59.15	49.28	50.49	SMAV
28.32	19.70	20.50	26.06	21.71	22.24	EC_A using EPA's 2.27 LTAF
49.07	34.15	35.52	45.15	37.62	38.54	EC_A using Gensemer et al. LTAF of 1.31
0.79	1.13	1.09	0.86	1.03	1.01	R

*Values used in the calculation were not provided, and are assumed approximately equivalent to those provided herein.

As noted earlier, this risk paradigm was designed to estimate the relative risk of a chemical, such that the farther away from 1 an R-value, the greater the assurance the assessor would have in their Section 7 effect determination. However, the strength of the EC_A (and the effects determination) depends on the availability of pertinent evidence, and ultimately on the identification, appraisal, assimilation, and interpretation of that evidence. A strict interpretation of the risk paradigm indicates that four of the six scenarios illustrated in Table 37 would warrant a preliminary "likely to adversely affect" determination until additional data is provided that demonstrates otherwise (e.g., "other data" not used in the SMAV calculation, and a closer review of the data used in the LC_{50} calculation). While the risk ratio is merely an indication of potential risk, it is clear that the values chosen to calculate the species' LC_{50} value can influence the preliminary screen risk prediction. Based on our comparison, it also appears that the values EPA used to calculate the SMAV for rainbow trout was conservative, given the larger data set.

Nevertheless, we are concerned that this analytical approach can generate misleading results by ignoring meaningful differences among studies. That is, when the data are normalized first by calculating the geometric mean of the LC_{50}s without regard to the underlying distribution of the data, resolution is lost. In addition to examining the pooled data set to see that it is comprehensive, we must also closely examine the distribution of the underlying data, and differences in test methods (doses, schedules, modes of treatment, etc.) to ensure important differences in data are not drowned in a single estimate generated from a pooled data set (Lau et al. 1998). Uncertainty is incorporated in our analysis when we "focus on the tails of the distribution rather than on the measure of central tendency (the mean or best estimate)…. (Taylor and Wade 2002)." A careful examination of the pooled data set is warranted to ensure we have appropriately incorporated uncertainty and to ensure that the method provides a high degree of conservatism (e.g., errs on the side of the protecting the species when data are not sufficient to reasonably conclude the action is "not likely to adversely affect" the listed species or its critical habitat). When we examine the distribution of the data for rainbow trout we see that the lowest test value presented by Kovacs (1979) approaches the CMC (LC_{50} at 6 ºC = 28 µg CN/L). When we apply EPA's extrapolation factor of 2.27 to the lowest LC_{50} value available for rainbow trout we can estimate of the lowest concentration likely to be lethal to 0 to 10 percent of the population. The resulting LC_{10} for very cold-water conditions (6 ºC) is 12 µg CN/L. That is, when exposed to as little as 12 µg CN/L in cold waters, as much as 10% of the exposed threatened and endangered steelhead may die.

EPA derived the lethality threshold adjustment factor, 2.27, from a combined data set on fresh water and marine fish and invertebrates, a number of chemicals tested, as well as whole effluent data. In comparison, Dwyer et al. (2005) looked at five chemicals and seventeen species, including a few listed species, and also found the average factor to calculate a no- or low-effect concentration varied among pollutants and species (0.50 - 0.66), with the geometric mean for all species as 0.56 (f^{-1} = 1.8). More recently, DeForest et al. (in Gensemer et al. 2007) compiled

concentration-response curves for rainbow trout, using data from McGeachy and Leduc (1988), and Kovacs and Leduc (1982), estimated the lethality threshold adjustment factor as 0.76 (f^{-1} = 1.316). Applying the extrapolation factor from Dwyer et al. (2005) results in a low effect concentration of about 16 µg CN/L, and DeForest et al. (in Gensemer et al. 2007) would result in a low effect concentration of 21 µg CN/L. DeForest et al. (in Gensemer et al. 2007) estimated the mean LC_{01}:LC_{50} ratio based on the steepness of the concentration-response curves to produce an estimated effect level lower than the LC_{01} (DeForest et al. in preparation, cited in Gensemer et al. 2007). Using DeForest et al.'s calculated adjustment factor, we would expect that 1% of the sample population would be expected to die as a result of their exposure at that calculated cyanide concentration.

In a separate analysis of the lethality threshold adjustment factor, FWS found EPA's 1978 data set upon which the 2.27 was derived from widely varied data and thus recalculated the adjustment factor as a standardized LC_{50}/LC_{10} using 62 acute exposure-response regression equations for cyanide (Appendix C). FWS' recalculated adjustment factor calculated for rainbow trout was 1.14. Had EPA used this, or any of these revised adjustment factors, more species would have been screened out as not likely to be adversely affected by their exposure to cyanide at the CMC. This further suggests that at least for cyanide, EPA's lethality threshold adjustment factor of 2.27, despite having introduced an additional source of uncertainty into estimates of the EC_A, likely produced preliminary estimates in accordance with the approach in the *Methods Manual* that erred on the side of inclusion rather than screening out species. Again, if we look at the distribution of the acute data for rainbow trout, using Kovacs' (1979) LC_{50} of 28 µg CN/L, which was derived in very cold water temperatures, and apply EPA's adjustment factor of 2.27 then an estimated 1 to 10% of individual steelhead may die when exposed when exposed to as little as 12 µg CN/L in very cold waters (6 ºC or less). Alternatively, if we apply the FWS' recalculated adjustment factor for cyanide to the same data, then the LC_{10} concentration would be above the CMC (at 24.56 µg CN/L).

Other Pacific Salmon Species. Based on species-specific estimates for coho and Chinook salmon and estimates for the genus *Oncorhynchus*, ICE predicts that coho, Chinook, sockeye, and chum salmon are relatively more sensitive than steelhead to cyanide (see Table 38). That is, based on the lower bound of the ICE predicted LC_{50}, coho, Chinook, sockeye, and chum salmon are all "likely to be adversely affected" when exposed to cyanide. Of these four fish within the genus *Oncorhynchus*, EPA's ICE results suggest that coho salmon are the most sensitive Pacific salmon with a predicted acute EC_A of 15.51 µg CN/L, with an estimated LC_{50} of 53.16 µg CN/L. In comparison, also using ICE, DeForest (pers. comm.) estimated the LC_{50} for coho salmon as 41.9 µg CN/L and the LC_{50} for Chinook salmon as 50.9 µg CN/L (Table 5). When we recalculated the EC_A using the divisor 2.27 and the expected LC_{50} value, for both species, the EC_A fell above the CMC using EPA's LC_{50} value, but below the CMC using the estimated LC_{50} calculated by DeForest (pers. comm.). Whereas, DeForest concluded, based use of the LC01 divisor, 1.316, that coho salmon and Chinook salmon were protected by the CMC (both LC01 values are greater than 30 µg CN/L). Based on the work by Gensemer et al. (2007), and DeForest (in Gensemer et al. 2007), the ICE model is likely to conservatively overestimate the sensitivity of most species (i.e., produce lower LC_{50} values than would likely be measured). DeForest (pers. comm.) concluded, based on his analysis of the empirical cyanide SMAVs, that there is an eight percent probability that a fish species would be more sensitive to cyanide than rainbow trout; whereas, if

the ICE-estimated LC_{50} values are considered in the SSD, then there is about a 20% probability that a fish species would be more sensitive than rainbow trout. Based on our recalculations and information from DeForest (pers. comm.), and EPA's use of the lower 95 confidence level to calculate the EC_A for these species, it appears that EPA's preliminary effects determination that these species should not be screened out would be conservative (i.e., that is it erred on the side of protecting listed species given the uncertainty in the estimates).

The Influence of Other Data

The preliminary screen in the *Methods Manual* was designed to be a first step for reviewing robust response data, and conclusions based on this screen should be carefully reviewed by rechecking each step. That is, studies that have been dismissed because they do not meet basic requirements for the calculation of EC_A require review as "other data". EPA's *Guidelines* explicitly state that

> *Pertinent information that could not be used in earlier sections might be available concerning adverse effects on aquatic organisms and their uses. The most important of these are data on cumulative and delayed toxicity, flavor impairment, reduction in survival, growth, or reproduction, or any other adverse effect that has been shown to be biologically important. Especially important are data for species for which no other data are available. Data from behavioral, biochemical, physiological, microcosm, and field studies might also be available. Data might be available from tests conducted in unusual dilution water, from chronic tests in which the concentrations were not measured, from tests with previously exposed organism, and from tests on formulated mixtures or emulsifiable concentrates. Such data might affect a criterion if the data were obtained with an important species, the test concentrations were measured, and the endpoint was biologically important (Stephan et al. 1985).*

According to the *Guidelines*, EPA ought to consider "other data" in its decision to recommend a criterion. Unfortunately, it's not apparent that this "other data" influenced EPA's final value for the cyanide CMC (or CCC) in their 1985 cyanide recommendation. Nor is there evidence to suggest that particular states incorporated the "other data" in their final state water quality criteria, such that particular exceptions or special management actions were written into the final adopted water quality standard, when applicable. We were particularly interested in the effects of temperature and dissolved oxygen on EPA's decision to recommend the cyanide criteria because these are two factors known to affect cyanide toxicity, and because studies that have directly explored these relationships with listed resources (steelhead and Atlantic salmon). We explore this "other data" in the following sections.

Table 41. Species Specific Toxicity Estimates (EPA 2007**).

Species*	Saltwater v. Freshwater Exposure	Acute EC_A (µg CN/L)	Chronic EC_A (µg CN/L)	Species Specific Toxicity Data	Estimation Method Used	Taxon Represented by EC_A
Coho salmon	SW (adult & smolt) FW (all life stages)	15.51	3.33	N	ICE	*Oncorhynchus kisutch*
Chinook salmon	SW (adult & smolt) FW (all life stages)	16.26	3.49	N	ICE	*Oncorhynchus tschawytscha*
Chum salmon	SW (adult & smolt) FW (all life stages)	21.41	4.60	N	ICE	*Oncorhynchus* (genus)
Sockeye salmon	SW (adult & smolt) FW (all life stages)	21.41	4.60	N	ICE	*Oncorhynchus* (genus)
Steelhead	SW (adult & smolt) FW (all life stages)	26.08	9.80	Y		
Shortnose sturgeon	SW (adult & juveniles) FW (all life stages)	29.28	6.39	N	SSD	*Actinopterygii* (class)
Green sturgeon	SW (adult & juveniles) FW (all life stages)	29.28	6.39	N	SSD	*Actinopterygii* (class)

*EPA also included Totoaba. This is a foreign-listed species and would not be adversely affected by the proposed action because it is not exposed to United States waters.
**Data are from EPA 2007. Life stages exposed were adjusted to account for all possible life stages that could be exposed in fresh water and salt water.

Table 42. Comparisons of LC50 values for coho and Chinook salmon (µg CN/L)

Species	Estimated Mean LC_{50}	Lower 95% CL	Estimated EC_A using expected LC_{50}	Estimated LC01 (f^{-1}= 1.316)
Coho salmon	53.16*	35.21*	23.42*	40.40
	41.9**	25.2**	18.46	31.84**
Chinook salmon	64.35*	36.91*	28.35*	48.90
	50.9**	24.7**	22.42	38.68**

**Data from DeForest, pers. comm.
*Data from EPA 2007

The Influence of Temperature on Tolerance Limits

As a general matter the tolerance of fish to many pollutants tends to decrease with increases in water temperatures. Studies have demonstrated that the effects of temperature on the toxicity of cyanide can vary with concentration and temperature such that cyanide toxicity increases at high temperatures and at very low temperatures. Studies that have evaluated the effects of cyanide at high temperatures have found that the toxic action of cyanide increases with increasing temperatures, but many of these studies were conducted with extremely high doses of cyanide (see Doudoroff 1976). Early studies indicated that the 72-hour median lethal concentration or tolerance limit increased almost threefold with increased temperatures, when rainbow trout were exposed to test temperatures ranging from 4 to 20 ˚C (Great Britain, Ministry of Technology 1969 in Doudoroff 1976). Unfortunately, it is not clear what cyanide concentrations were used in the Great Britain study (Doudoroff 1976). Later, Kovacs (1979) confirmed that there are significant differences in 96-hour LC_{50} values between 6, 12 and 18 ˚C, such that it took 2.4 times less cyanide to kill 50% of the trout in 96 hours at 6 ˚C than it did at 18 ˚C. One of the primary differences between work by Kovacs (1979) and earlier researchers is the rate and concentration of the doses administered. Kovacs (1979) administered cyanide at slowly lethal concentrations, whereas earlier studies tended to focus on rapidly lethal concentrations, suggesting that the potency of cyanide is both temperature and concentration dependent. Doudoroff (1976) suggested that the lethal response at low temperatures is likely a result of a decrease in the rate of detoxification at lower temperatures, which is affected by the decline in the metabolic rate at lower temperatures. Death at lower temperatures may also be caused by the disruption of cytochrome oxidase activity (Kovacs 1979).

Since steelhead rearing and spawning typically occurs in temperatures ranging from about 4˚C to about 15˚C (Barnhart 1991). Consequently, increased cyanide toxicity at lower temperatures could have serious consequences for steelhead fitness. We chose four river basins that we felt were representative steelhead rivers—one from each of the western states, Oregon, Washington, Idaho, and California, where there are listed steelhead populations—and examined the mean monthly water temperatures for comparison to the low temperatures measured by Kovacs (1979). Figure 7 compares the mean monthly water temperatures to the generalized life history stages of steelhead in the Clearwater River, Idaho. Steelhead in this system compose two-runs, an “A” and “B” run, which are distinguished according to their size and ocean life history. Spawning occurs from mid-April to late June, with “A-run” fish returning after one year in the ocean and “B-run”

fish returning after two years in the ocean. Due to the long freshwater rearing period of juvenile steelhead and the long holding period of adults, at least two to three age classes of steelhead can be found in the basin during winter. As illustrated in Figure 7, winter water temperatures are at or below 6˚C for several months each year (about 5 months). Similarly, water temperatures are at or below 6˚C in the Puyallup River in Washington for about three months when adult and juvenile life stages would be in the basin (Figure 8). In the North Umpqua River in Oregon water temperatures are at or below 6˚C for about four months of the year, when additional life stages are present including migrating and spawning adults, eggs, fry, and juvenile fish (Figure 9). In the Klamath River in Oregon/California average water temperatures are below 6˚C for a brief period of time (about a month), but these temperatures occur when adults are migrating and spawning, and juvenile steelhead are rearing (Figure 10). Due to the iteroparous life history of steelhead and the propensity for multiple juvenile age-classes to rear together, these basins would generally have at least two age-classes but may have four or more age-classes in the basin during winter.

We looked but did not find information to suggest that states or EPA would generally modify the cyanide water quality standards to minimize the impacts to salmonids in cold water. We looked for this information particularly in state water quality standards for Idaho, California, Washington and Oregon. Generally, we found that when states modified EPA's nationally recommended criteria they did so to increase the cyanide concentration, not decrease acceptable limits. However, we did not search specific permit conditions to evaluate whether permits were adjusted to account for increased toxicity of cyanide during low temperatures.

All of the Pacific salmonids under NMFS' jurisdiction, green and shortnose sturgeon, and Atlantic salmon are exposed to very cold water temperatures during their life cycle. We would not expect that the general response of increased toxicity at low temperatures is species specific response, but is a generalized physiological response of fish that occupy cold streams. The low acute response of steelhead is likely a reasonable predictor of other Pacific salmonids, but we do not know the lowest response value of sturgeon or Atlantic salmon nor do we have a suitable surrogate to estimate this response. Clearly, more studies are warranted in this area.

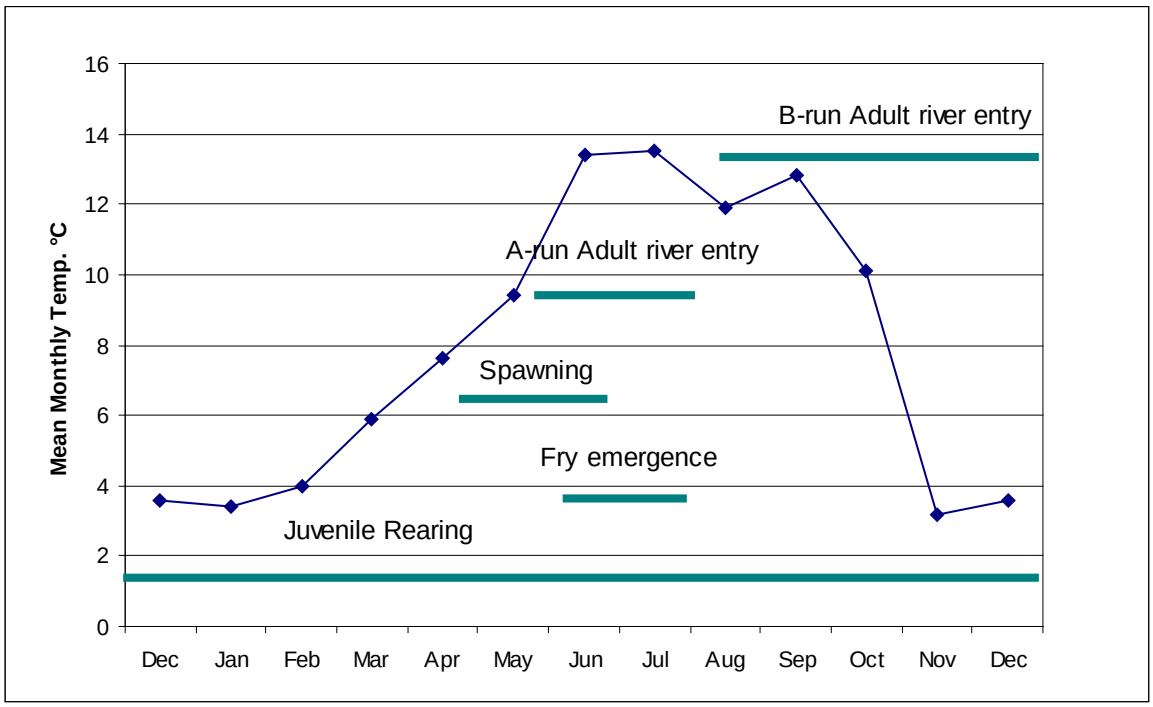

Figure 7. Steelhead life history and mean monthly water temperatures in the Clearwater River, Idaho (Sources: Idaho Department of Fish and Game[20] and USGS Surface-Water Monthly Statistics for the Nation, USGS 13342500 Clearwater River at Spalding ID[21]).

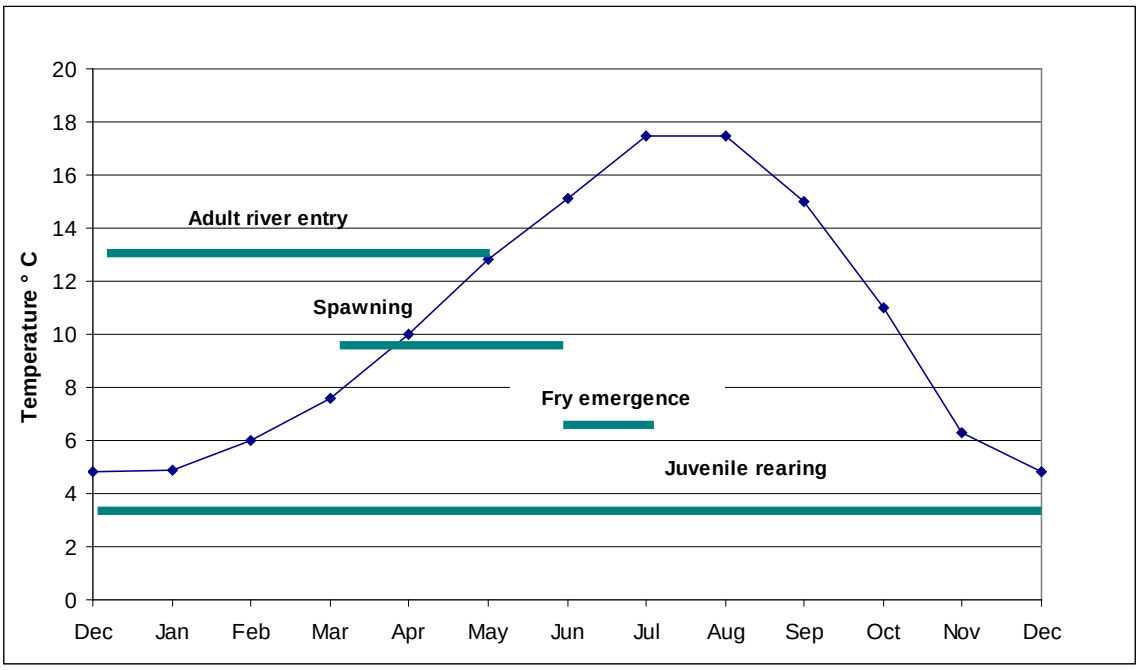

Figure 8. Winter steelhead life-history and mean monthly water temperatures in the Puyallup River Basin, Washington (Ball 2004; and B. Smith, Puyallup Tribe Fisheries, pers. comm., Oct. 14, 2008).

[20] URL:http:fishandgame.adaho.gov/fish/fish_id/steelhead.cfm
[21] URL:http://waterdata.usgs.gov/nwis/monthly?

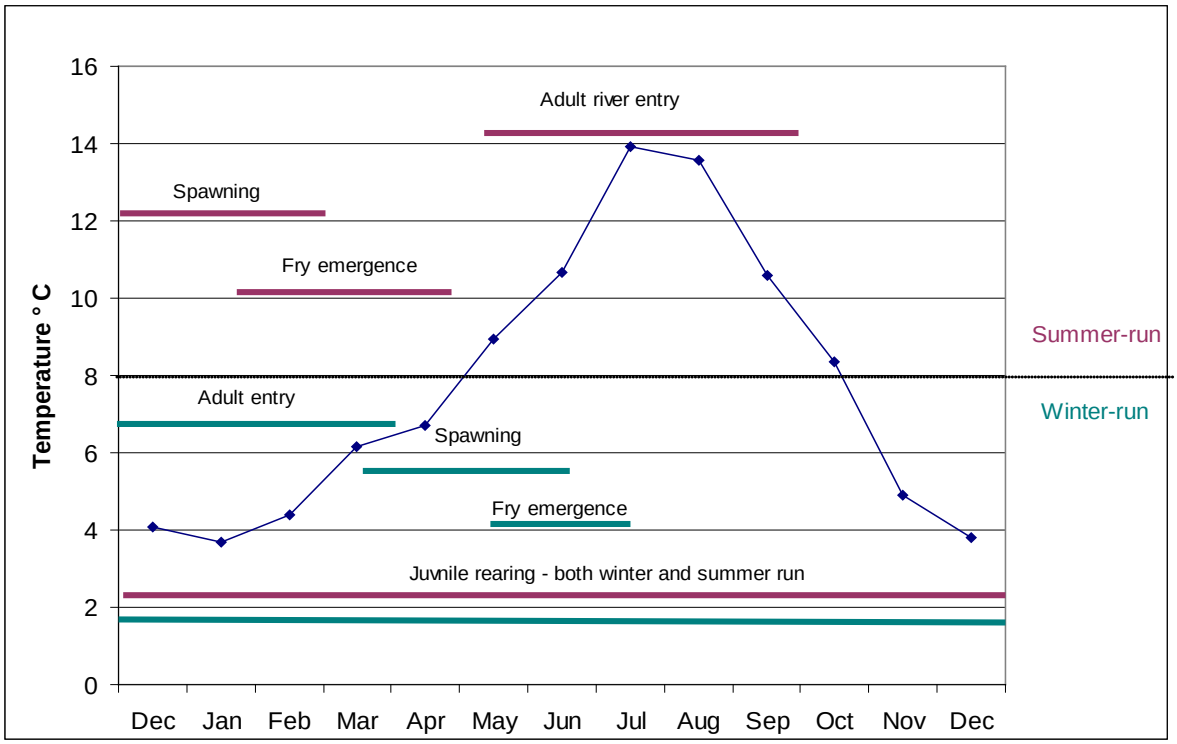

Figure 9. North Umpqua River steelhead life history and average monthly water temperatures (Source: USGS National Water Information System, URL: http://nwis.waterdata.usgs.gov)

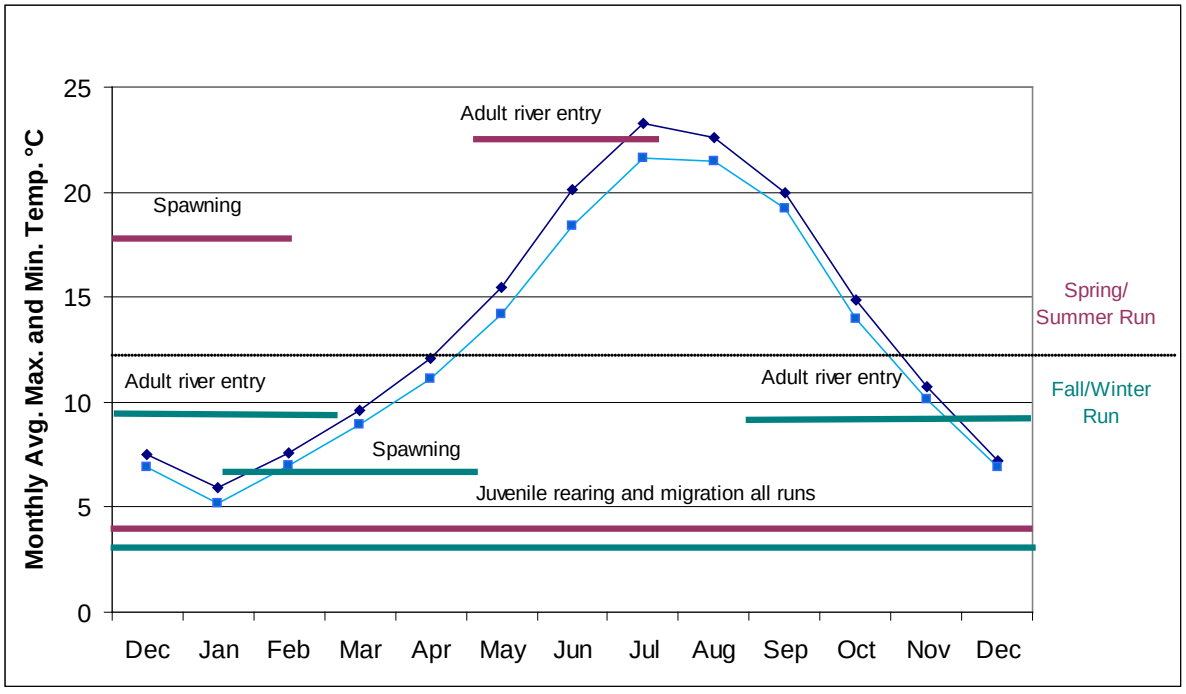

Figure 10. Klamath River steelhead life history and average min. & max. monthly water temperatures (Sources: USFWS 1998 and USGS 2007).

The Influence of Dissolved Oxygen on Tolerance Limits

Generally, in environments where DO is less than optimal fish will compensate for the reduction in DO by increasing gill movement and ventilation volume, in an attempt to maintain adequate oxygen volumes. Cyanide is a powerful asphyxiant, and the addition of cyanide in waters with low DO further stresses fish, reducing the lethal concentration at which survival is typically expected. That cyanide toxicity is influenced by DO is well known (Downing 1954; Smith et al. 1978; Doudoroff 1976; Towill et al. 1978; Alabaster et al. 1983; EPA 1985; Dzombak et al. 2006). Smith et al. (1978) found that a about a 40% reduction in DO levels lead to a 20 to 30% reduction in lethal thresholds for brook trout and rainbow trout. Similarly, Downing (1954) found that rainbow trout survival time increased as DO increased, and the rate of increase did not fall off as DO approach saturation. Alabaster et al. (1983) also demonstrated that the 24-hr LC_{50} value varies with DO concentrations, but not with salinity, and when DO was as low as 3.5 mg/L the LC_{50} value for Atlantic salmon was 24 µg CN/L HCN (well below the acute EC_A reported in Table 4 of the final cyanide BE of 39.65 µg CN/L).

Since the conditions under which this study was conducted are very important to a comprehensive effects analysis, we asked that EPA consider it further in their BE. Unfortunately, EPA responded that (a) it was not considered in the criteria derivation (or was relegated to "other data"), (b) that considerations of toxicity values obtained under a combination of low DO and chemical toxicity is generally not included in their BE because such exposure conditions are not wide-spread across the nation, and (c) confound the toxicity of cyanide alone to which the criteria apply. The first part of the statement, that the data in Alabaster et al. (1983) was not considered in the derivation of the cyanide criteria is interesting, but not necessarily pertinent for the purposes of a Section 7 consultation. First, the criteria were derived without consideration of listed species, but more importantly the question of risk depends upon the environmental decision-making context. That is, Section 7 is first concerned with the risk an action poses individuals of a listed species –this is the level at which a federal agency makes their effect determination. Not until individual effects can be dismissed as insignificant or discountable, would we conclude that an action is "not likely to adversely effect" listed species. The CWA decision-making process begins by focusing not on the individual, but whether community level effects are likely. The effect threshold is considerably different. By the time community level effects are measurable (and noticed) the hazard's risk may pose substantial impacts to small populations. If EPA meant to imply that because a study was not used to derive a particular criterion that it did not warrant consideration in their Section 7 effects analysis, then EPA is missing the point of Section 7 under the ESA. The relevant inquiry is not whether such a study was used by EPA in their 1985 criteria decision, but whether there is information to suggest that environmental conditions to which listed species are exposed may influence the toxicity of the chemical under review—in this case cyanide. Cyanide can be more toxic to freshwater fish at low dissolved oxygen concentrations.

According to our assessment of water quality conditions across the nation, low DO conditions are a problem in many basins at various times of the year (see the Environmental Baseline section of this opinion, also see EPA 2006). The susceptibility of fish to cyanide at low DO may be correlated with the rate of breathing. That is, as a general matter the rate of gill movement increases with decreasing DO, causing the fish to pump additional water through the gills to

obtain more oxygen. When cyanide is present in the water column, this may increase the rate at which tissue that is more susceptible to absorption is exposed to cyanide. Although EPA did not consider the relationship between DO and cyanide to be one that should drive the nationally recommended criteria, there is sufficient information indicating the toxicity threshold for salmonids is reduced in low DO conditions such that additional studies are warranted to make definitive conclusions regarding the effect on fish and whether the criteria would fully protect salmonids at the local or site-specific scale.

Effects of Mixtures

Relatively few studies have been performed to measure the effects of free cyanide in combination with other contaminants. Concurrent exposure to cyanide and ammonia produced greater than additive effects to acute lethality in rainbow trout, salmon, and chub (Smith et al, 1979; Alabaster et al., 1983; and Douderoff 1976), and to chronic sublethal effects to growth in rainbow trout (Smith et al 1979). In rainbow trout and salmon, effects to acute lethality were 1.2 and 1.63 times greater than would be expected by additivity. Concurrent exposure to cyanide and zinc also resulted in synergistic effects to acute lethality in fathead minnows, where toxicity was 1.4 times that predicted by additivity (Smith et al 1979). Though we are unable to quantify the effect of these synergistic mechanisms for this analysis, they should be considered when assessing effects of cyanide to aquatic organisms in waterways with elevated concentrations of ammonia and zinc.

Chronic Toxicity

Chronic cyanide toxicity tests have been conducted with relatively few fish species, however, the available data indicate that cyanide not only reduces survival but also affects reproduction, weight gain, growth and development, swimming performance, condition, and development. Few studies have examined the sublethal responses at cyanide concentrations below the freshwater CCC (i.e., <5 µg CN/L) and many have evaluated the effect of concentrations double that of the CCC, making it difficult to evaluate the effect of exposing individuals at the CCC.

Dixon and Leduc (1981) also found evidence of liver necrosis in rainbow trout from low-level exposures of cyanide; however the lowest concentration that they examined was 10 µg HCN/L (~9.8 µg CN/L). In calculating the chronic EC_A value for rainbow trout, it appears that EPA used the reported NOEC from only one study, Dixon and Leduc (1981). In Table 1 of the final cyanide BE, EPA reports a chronic EC_A value for rainbow trout of 9.8µg/L. However, since Dixon and Leduc (1981) did not evaluate rainbow trout response to cyanide concentrations below 9.8 µg/L, it is equivocal to equate this value to a NOEC for the species since adverse effects could not be distinguished at concentrations below this value.

Given the available data reproduction appears to be one of the most sensitive (and most studied) endpoints. Full and partial life cycle tests with fathead minnow and brook trout have shown that fish exposed to sublethal concentrations of cyanide spawned fewer eggs than non-exposed fish (Koenst et al. 1977; Lind et al. 1977). Fecundity was reduced by 57.8% and 46.9% (compared to controls) in female fathead minnows exposed to cyanide at 19.6 µg HCN/L (the LOEC) and 12.9 µg HCN/L (the NOEC), respectively. Similarly, the mean number of eggs spawned by brook trout was reduced by 53.3% at 11.2 µg HCN/L and by 17.7% at 5.7 µg HCN/L. Koenst et al.

(1977) exposed brook trout to nominal HCN concentrations between 5.7 and 77 µg HCN/L, and found that at the mean number of eggs spawned per female decreased with increasing HCN concentrations above 5.7 µg HCN/L. Using a mean temperature of 13.5 ˚C, to convert to CN^- results in a NOEC value is 5.6 µg CN/L, just above the CCC. In the same study, Koenst et al. (1977) found that exposure to 5.5 HCN/L (5.4 µg CN/L) reduced the length of brook trout at hatching and the percentage of eggs that hatched.

Kimball et al. (1978) studied the chronic toxicity of HCN to bluegill and found that bluegill ceased spawning at 5 µg HCN/L (~4.8 µg CN/L). Of eight tests with different concentrations, ranging from 5 to 80.0 µg HCN/L, no spawning was recorded in seven of the tests. Interestingly, at the highest concentration 80.0 µg HCN/L, one female survived and managed to spawn, although her egg production was markedly reduced in comparison to controls. Although the single spawning is difficult to explain, the fact that spawning was completely inhibited in 42 of 43 cyanide-exposed females suggests that bluegill may be particularly sensitive to cyanide at low levels. Results of the tests conducted by Kimball et al. (1978) suggest there was a 3% probability that a female would spawn at $\geq$4.8 µg CN/L, and since levels less than 5.2 µg HCN/L were not tested the only we can only safely conclude that this is a LOEC and that the NOEC lies at a threshold concentration below 5.2 µg HCN/L. Considering the overwhelming evidence of an adverse effect, it is surprising that additional studies on the effects of cyanide on bluegill reproduction have not been conducted over the past 30 years. Cheng and Ruby (1981) studied the effects of pulsed exposures of cyanide on flagfish reproduction. Unlike the studies describe above, where fish were exposed over an extended period of time to a constant concentration, flagfish were exposed to sublethal concentrations of cyanide for 5 day pulses. Flagfish exposed to cyanide (65 µg/L) for 5 days following fertilization, i.e. as eggs, and then reared to maturity in clean water, spawned 25.6% fewer eggs than flagfish that had not been exposed. In another experiment by the same authors, flagfish that received a second 5-day pulse of cyanide as juveniles had an even greater reduction (39.3%) in number of eggs spawned. These studies demonstrate that cyanide can affect an apical reproductive endpoint in fish.

The mechanism by which cyanide induces these reproductive effects is not fully understood, however, key physiological, biochemical, histological (morphological), and endocrine functions known to be involved in sexual maturation are affected by cyanide. For instance, Lesniak and Ruby (1982) reported abnormal oocyte development in sexually maturing female rainbow trout exposed to cyanide (10 and 20 µg HCN/L) for 20 days. Ovaries from cyanide-exposed fish contained fewer mature oocytes, exhibited altered patterns of secondary yolk deposition (in developing oocytes), had nearly twice the frequency of atresia (oocyte resorption), and had an overall reduction in the number of viable eggs. Ruby et al. (1986) reported that vitellogenic female rainbow trout exposed for 12 days to 10 µg HCN/L had lower levels of plasma vitellogenin and a lower gonadosomatic index (GSI) compared to controls. In two similar studies, oocyte diameter (an indicator of gonadal growth and development) was reduced in sexually maturing female rainbow trout exposed for 12 days to 10 µg HCN/L (Ruby et al. 1993a, Szabo et al. 1991). Reduced oocyte diameter was accompanied by reductions in plasma vitellogenin, 17β-estradiol (E2), and GSI (Ruby et al. 1993a), as well as increased whole brain dopamine levels (Szabo et al. 1991).

Dopamine has an inhibitory effect on gonadotropin-releasing hormone (GnRH) neurons in some

fish species and it is GnRH which stimulates the release of gonadotropins (GtH I and GtH II) from the pituitary (Patino 1997; Saligaut et al. 1999). GtH I and GtH II are believed to function similar to follicle-stimulating hormone and luteinizing hormone, respectively, in tetrapods (Patino 1997). In female fish, GtH I acts on target cells in the gonad, stimulating E2 synthesis. E2 induces vitellogenin synthesis in the liver. Vitellogenin is the egg yolk precursor in fish which is produced by the liver, transported via blood, taken up by ovaries, and incorporated into developing oocytes. GtH II also acts on the gonad by inducing the synthesis of maturation-inducing steroid (MIS). MIS induces oocyte maturational competence and ovulation (Park et al. 2007; Patino 1997). The control exerted by dopamine over gonadal maturation has been recognized by fish culturists, who have been successful in treating captive-reared fish with anti-dopaminergic drugs (which block dopamine receptors), such as pimozide and domperidone, to induce ovulation (Jensen 1993; Park et al. 2007; Patino 1997; Szabo et al. 2002). Thus, oocyte development, maturation and ovulation are under the control of gonadotropins and E2 which in turn, are modulated in part by GnRH and dopamine. This interaction between the neuroendocrine system and reproductive organs is referred to as the hypothalamus-pituitary-gonadal (HPG) axis (IPCS 2002).

Cyanide has also been shown to affect male reproductive processes. Exposure of male rainbow trout to cyanide (10 and 30 µg HCN/L) for 18 days disrupted spermatogenesis as evidenced by a reduction in the number of dividing spermatogonia and a blockage of mitotic progress (Ruby et al. 1979). Exposure of rainbow trout for 12 days to 10 µg HCN/L resulted in higher numbers of spermatogonial cysts in testes of male trout as well as higher levels of whole brain dopamine (Szabo et al. 1991). Similar results were reported by Ruby et al. (1993) where the number of spermatocytes decreased and the number of spermatocyte precursors (spermatogonial cysts) increased in two-year-old sexually maturing rainbow trout after 12 day exposure to 10 µg HCN/L. There are indications that the transformation of spermatogonial cysts to spermatocytes is hormonally regulated through GtH along the HPG axis and that, within the pituitary, GtH is released from type I granular basophils (Ruby et al. 1993). Histological examination of pituitary glands from cyanide-exposed fish showed a reduction in the number of type I granular basophils. The authors suggested that elevated levels of brain dopamine may be responsible for the selective loss of type I granular basophils and subsequent alteration of spermatocyte formation.

Ruby et al. (1979, 1993) and Szabo et al. (1991) hypothesized that cyanide acts through the HPG axis to affect reproduction in fish. Their studies (described above) demonstrated (1) that cyanide caused an increase in brain dopamine levels, consistent with neuronal effects observed on mammals, (2) that levels of reproductive hormones (E2) and egg-yolk precursors (vitellogenin) were altered following exposure to cyanide, (3) the selective loss of putative GtH releasing pituitary cells (type I granular basophils) and (4) retarded gonad development in cyanide-exposed male and female rainbow trout. Taken together, these results appear to be consistent with HPG axis involvement. In addition, the authors found that these effects occurred following relatively short, sublethal exposures to cyanide (12 – 18 days). Whether these effects would result in the same type of reduced fecundity and spawning, as was observed in cyanide-exposed female fathead minnow (Lind et al. 1977), bluegill (Kimball et al. 1978), and brook trout (Koenst et al. 1977), was not addressed in the rainbow trout studies because they were terminated before the fish reached full sexual maturity, however, it does seem likely. Results from Cheng and Ruby (1981) indicate that continuous exposure to cyanide through the spawning period may not be

necessary to affect fecundity. Short-term, pulsed exposures of cyanide to flagfish were sufficient to induce later effects on the number of eggs spawned, and exposed fish did not appear to recover once the exposure had ceased. Even exposure of eggs, one of the most tolerant life stages in terms of acute toxicity (Smith et al. 1979), resulted in latent effects on fecundity once embryos hatched and survived to maturity. Interestingly, it is during early developmental stages that the HPG endocrine axis is set up and feedback sensitivity of the hypothalamus and pituitary gonadotropes to gonadal steroids is established (IPCS 2002). Although Cheng and Ruby (1981) did not measure specific indicators of endocrine axis function, they did find that the pituitary gland of cyanide-exposed flagfish embryos was significantly smaller than the pituitaries from control fish. It would appear that cyanide, like many EDCs (endocrine disrupting compounds, IPCS 2002), may affect the "set up" of the HPG axis and that these early developmental effects may have long term consequences on reproduction.

Chronic exposure of eggs and larvae to cyanide can result in reduced embryo/larvae survival and altered development. Leduc (1978) exposed newly fertilized Atlantic salmon eggs to cyanide (10 - 100 µg HCN/L) and observed teratogenesis, as well as, delayed hatching and reduced hatching success at higher concentrations. There was a dose-dependent increase in the frequency of abnormal fry, ranging from 5.8% to 18.5%. Abnormalities included malformed and/or absence of eyes, defects in the mouth and vertebral column and yolk-sac dropsy (*Hydrocoele embryonalis*, also known as blue sac disease). Similar eye abnormalities were reported by Cheng and Ruby (1981) in flagfish larvae exposed, as eggs, to cyanide (65, 75, 87, 150 µg HCN/L). Hatching success was also reduced and time to hatch was delayed in all cyanide treatments. In a 28-day embryo/juvenile toxicity test, sheepshead minnow survival was significantly reduced in all treatments >29 µg HCN/L (Schimmel 1981). The author noted there was considerable embryonic mortality and that there was no larval mortality during the last two weeks of exposure, indicating a greater sensitivity during early development. Kimball et al. (1978) exposed bluegill eggs and larvae to cyanide (4.8 – 82.1 µg/HCN/L) and reported that most deaths occurred within the first 30 days after hatching. Survival was reduced in all cyanide treatments and the effects were statistically significant at cyanide concentrations >9.1 µg HCN/L.

As previously mentioned, cyanide effects oxidative metabolism, energy production, and thyroid function; all are important for normal growth and performance. Therefore, it is not surprising that sublethal exposure of fish to cyanide has been shown to impact growth, condition and swimming performance. There is also evidence that the effect of cyanide on these physiological endpoints can be modulated by other factors such as diet/ration and temperature. When cichlids (*Cichlasoma bimaculatum*) were fed unlimited rations and exposed to cyanide for 24 days, those fish exposed to lower concentrations of cyanide (< 0.06 µg HCN/L) were larger than controls, where as, at higher concentrations weight gain was depressed (Leduc 1984). The increased weight gain in the low-dose treatments was attributed to higher food consumption, which was allowed to occur because ration was not restricted. Low-dose stimulation is a common effect across a broad range of chemical and non-chemical stressors (Calabrese 2008). Dixon and Leduc (1981) held juvenile rainbow trout on restricted rations and exposed them to cyanide (10, 20, 30 µg HCN/L) for 18 days and observed significantly reduced weight gain in all treatments compared to controls. The effect was characterized by an initial decrease in specific growth during the first 9 days followed by a significant increase from day 9 through 18. The growth surge during the latter half of the exposure period was not sufficient to offset early reductions.

Cyanide-affected fish were in poorer condition, as indicated by lower fat content, and had higher respiration rates for several days post exposure. In addition, fish in all cyanide treatments exhibited degenerative necrosis of hepatocytes, i.e. liver tissue damage, which increased in severity with the level cyanide exposure. Kovacs (1979) held juvenile rainbow trout on restricted rations and exposed them to cyanide for 20 days. The results were similar to those of Dixon and Leduc (1981). Cyanide reduced the mean specific growth rate and affected-fish gained less fat during the exposure period. Kovacs (1979) also examined the effects of temperature on rainbow trout growth and sensitivity to cyanide, and found that the growth rate of rainbow trout was inversely related to holding temperature (6, 12 and 18˚C), as would be expected, and that trout held at colder temperatures were more sensitive to cyanide. The NOECs for mean specific growth rate were 5, 20, and 30 µg HCN/L for trout held at 6, 12, and 18˚C, respectively. Based on the exposure response curves the author estimated thresholds for effects on growth to be <5 µg HCN/L at 6˚C (<4.9 µg CN/L, just below the freshwater CCC), 10 µg HCN/L at 12˚C, and 30 µg HCN/L at 30˚C. In the same study, swimming performance was found to be affected by cyanide and the effect was also temperature-sensitive. Fish from the growth study were placed in swimming chambers and tested for swimming stamina. Among non-exposed trout, swimming stamina, measured as distance travelled (meters), decreased with decreasing temperature, i.e. fish held a 6˚C travelled a shorter distance than fish held at 18˚C. Cyanide-exposed fish had reduced swimming stamina compared to non-exposed fish and the effect was more severe at colder temperatures. Based on the exposure-response regression equations reported by Kovacs (1979) the predicted reduction in swimming stamina (compared to controls) for fish exposed to cyanide at the chronic water quality criterion (5.2 µg CN/L) would be 52% at 6˚C, 20% at 12˚C, and 3% at 18˚C. Several other authors have studied swimming performance as well. Leduc (1966) studied the effect of sublethal concentrations of cyanide on cichlids and coho salmon; the lowest concentration examined was 7 µg CN/L. At 7 and 8 µg CN/L cichlids exhibited reduced swimming speeds, similar to fish exposed to higher concentrations (30 µg CN/L; Leduc 1966). Neil (1957 in Kovacs 1979, Koenst et al. 1977) also showed that cyanide concentrations as low as 10 µg CN/L reduced the swimming stamina of brook trout by 98%. Similarly Broderius (1970) and Speyer (1975) observed reduced the swimming ability of coho salmon and rainbow trout at concentrations of 10 and 20 µg HCN/L. Thus, chronic exposure of fish to cyanide at sublethal concentrations, can affect growth, condition and swimming performance, and factors such as temperature and diet/ration can modulate cyanide toxicity. Neither fat synthesis nor swimming performance, however, are endpoints that EPA would typically use to establish water quality criteria, yet the two endpoints can significantly influence an individual's fitness. Fat is an indicator of growth, and is important during migration and reproduction as an energy reserve. Poor swimming performance can reduce ability to escape predators, maintain stream position, migratory performance. That adverse effects occur below the CCC appears unequivocal; a question that merits further investigation is just how far below the CCC is the threshold response for most species?

Chronic Effects Estimation

Ideally, we would use concentration (dose)-response data to build predictive models of the potential sublethal effects of cyanide. Unfortunately, such data do not exist for cyanide or listed species. As recently reviewed by Gensemer et al. (2007), the current inventory of concentration-response data from chronic toxicity testing with cyanide consists of four datasets; one each for

reproductive endpoints among fathead minnow (*Pimephales promelas*; Lind et al. 1977) and brook trout (*Salvelinus fontinalis*; Koenst et al. 1977); and for juvenile survivorship among bluegill (*Lepomis macrochirus*; Kimball et al. 1978) and sheepshead minnow (*Cyprinodon variegates*; Schimmel et al. 1981). Upon closer inspection, Gensemer et al. (2007) found the dataset for sheepshead minnow to be insufficient for meaningful predictive modeling and we agree with that conclusion. Thus, we are left with three datasets as the best available scientific basis for estimating toxic effects at the chronic criterion value of 5.2 µg CN/L. In addition to our three useable concentration-response datasets we also possess estimates of LC_{50} values for our listed species as per the procedures established in the *Methods Manual*.

To estimate the chronic effects of the proposed action on listed species, we transformed our three concentration-response data sets into the most precise predictive concentration-response models that the data can support and then used these models to predict the response of chronic toxicity test species to the CCC for cyanide. We assume that the predicted response of a listed fish species to the CCC is the same as the response observed for a chronic toxicity test species at an adjusted chronic CN exposure level based on the ratio of their respective LC_{50} values (example below). This approach is based on two simplifying assumptions:

1. That relative differences in sensitivity to chronic CN exposures between our listed evaluation species and our chronic toxicity test species (i.e., fathead minnow, brook trout, and bluegill) are approximated by the ratio of their respective LC_{50} values, and

2. The slopes of the concentration-response curves are also approximately comparable between our listed evaluation species and our chronic toxicity test species.

These assumptions create a clearly defined basis for a default hypothesis that allows us to proceed within the constraints of minimal data until such time as more data become available. As more data become available appropriate modification (or validation) of our default approach is necessary.

To provide an illustrative example of the outcome from our simplifying assumptions, suppose that one chronic toxicity test species is predicted to exhibit a 20% adverse effect from 5.2 µg CN/L. If a listed species happens to have an estimated LC_{50} value equal to that of the chronic toxicity test species, then a 20% adverse effect would also be predicted for the listed species. If the ratio of LC_{50} values was 1.5 (rather than 1.0) in the direction of greater sensitivity for the listed species than the chronic toxicity test species, then the predicted response at the concentration of interest of 5.2 µg/L for the listed species would be the same as the response observed for the chronic toxicity test species at a CN concentration 1.5 times 5.2 µg/L, i.e., at 7.8 µg/L. We refer to such surrogate currency equivalents for our listed species as $SSEC_x$ values (or sometimes shortened to SS_x). In this example, the predicted adverse effect for chronic toxicity test species at the $SSEC_x$ of 7.8 µg/L would be our surrogate currency predicted effect for the listed species at 5.2 µg CN/L (from that one of three prediction models) for the purposes of this Opinion. A more detailed derivation and explanation of the $SSEC_x$ concept is provided in Appendix D.

Because groups of taxonomically related listed species were assigned identical LC_{50} values from

the same ICE model, there are only 17 $SSEC_x$ values that need to be evaluated for any given (chronic toxicity test species) prediction model, but they are different for each prediction model (3 x 17 = 51 total $SSEC_x$ values of interest). For the prediction model based on fathead minnow chronic toxicity data the $SSEC_x$ values range from 6.7 to 45.8 µg CN/L (Table 40). As indicated by the entire range of $SSEC_x$ values being greater than 5.2 µg CN/L, all listed evaluation species have LC_{50} values that are more sensitive to cyanide than the fathead minnow LC_{50} value. For the prediction model based on brook trout chronic toxicity data the $SSEC_x$ values range from 4.2 to 28.4 µg CN/L (Table 40). For the prediction model based on bluegill chronic toxicity data the $SSEC_x$ values range from 6.1 to 41.7 µg CN/L (Table 40). Those $SSEC_x$ ranges define for each prediction model the range of cyanide concentrations over which model fit will be of most relevance to this Opinion. Detailed $SSEC_x$ results and the origins of the LC_{50} values used to calculate the $SSEC_x$ values are presented in Table 40 and Appendix D.

Table 43. Surrogate currency equivalents ($SSEC_x$[1]) for each LC_{50} surrogate taxon/chronic toxicity test species combination

Surrogate taxa used to estimate listed species (LS) LC_{50}	**$LSEC_x$ (µg CN/L)**	**LS LC_{50} (µg CN/L)**	**Effects on Fecundity**		**Effects on Early Life Stage Survival**
			Fathead Minnow SS LC_{50}=138.4 (µg CN/L)	**Brook Trout SS LC_{50}=85.7 (µg CN/L)**	**Bluegill SS LC_{50}=126.1 (µg CN/L)**
			$SSEC_X$ (µg CN/L)	**$SSEC_X$ (µg CN/L)**	**$SSEC_X$ (µg CN/L)**
Actinopterygii (class)	5.2	66.5[2]	10.8	6.7	9.9
Cypriniformes (order)	5.2	84.55[2]	8.5	5.3	7.8
Family Catostomidae					
Xyrauchen texanus (species)	5.2	83.8[3]	8.6	5.3	7.8
Family Cyprinidae					
Cyprinella monacha (species)	5.2	36.4[3]	19.8	12.2	18.0
Gila elegans (species)	5.2	50.9[3]	14.1	8.8	12.9
Notropis mekistocholas (species)	5.2	48.5[3]	14.8	9.2	13.5
Ptychocheilus lucius (species)	5.2	43.5[3]	16.6	10.3	15.1
Perciformes (order)	5.2	90.8[2]	7.9	4.9	7.2
Percidae (family)	5.2	42.3[3]	17.0	10.5	15.5
Etheostoma (genus)	5.2	40.0[3]	18.0	11.1	16.4
Etheostoma fonticola (species)	5.2	21.5[3]	33.4	20.7	30.5
Order Salmoniformes, Family Salmonidae					
Oncorhynchus (genus)	5.2	47.0[3]	15.3	9.5	13.9

Oncorhynchus apache (species)	5.2	16.5[3]	43.6	27.0	39.7
Oncorhynchus tschawytscha (species)	5.2	32.0[3]	22.5	13.9	20.5
Oncorhynchus kisutch (species)	5.2	32.4[3]	22.2	13.8	20.3
Oncorhynchus clarki henshawi (species)	5.2	22.8[3]	31.5	19.5	28.7
Salvelinus (genus)	5.2	15.7[3]	45.8	28.4	41.7
Salmo salar (species)	5.2	90[4]	8	5	7.3

[1] $SSEC_x$ values were calculated using equation 5 in Appendix D. Surrogate taxa were used to estimate LC_{50} values for listed species.
[2] LC_{50} based on 5th percentile estimate from species sensitivity distribution (SSD), Table 2 – Cyanide BE.
[3] LC_{50} estimate based on lower bound of the 95% CI from ICE model
[4] LC_{50} based on measured value from the Cyanide BE

Prediction models. We applied statistical regression techniques to model, or "fit", the relationship between cyanide concentrations and toxic effects based on data for our chronic toxicity test species. For nuances of statistical regression specific to toxicological applications we relied substantively on two recent technical guidance documents: (1) Environment Canada (2005: "Guidance Document on Statistical Methods for Environmental Toxicity Tests"), and (2) OECD (2006: "Current Approaches in the Statistical Analysis of Ecotoxicity Data: A Guidance to Application"). We also reviewed other relevant guidance such as that provided in the documentation for EPA's Toxicity Relationship Analysis Program (TRAP) (EPA 2002) and in discipline-specific statistical textbooks such as Gad and Weil (1988) and Sparks (2000).

As noted by Environment Canada (2005) an important principle of regression techniques is to keep the model simple, if that can reasonably be done. We have further incentive to follow that principle because we have a strong interest in evaluating the uncertainty (confidence) associated with point estimates and therefore an interest in avoiding what Environment Canada (2005) noted as the "...obstacle of calculating confidence intervals around nonlinear regression estimates..." Throughout this exercise we have been mindful that because our models are not based on biological or chemical mechanisms of action, but are purely statistical constructs, they have no biological meaning. A statistical concentration-response model only serves to smooth the observed concentration-response, to estimate effect concentrations by interpolating between treatment concentrations, and to provide a tool for assessing confidence intervals. Therefore the choice of model is to some extent arbitrary (OECD 2006). That being noted, we constructed models that conformed to the data we are working with and with statistical standard practices (such as data transformations). The degree of model fit achieved is an artifact of those specific decisions not the result of *post hoc* "model shopping" (EPA 2002).

Generic concentration-response relationship. Figure 11 illustrates a generic concentration-response relationship which typically takes on a sigmoidal form due to threshold effects on the low concentration end of the x-axis and to asymptotic effects at the high concentration end of the x-axis.

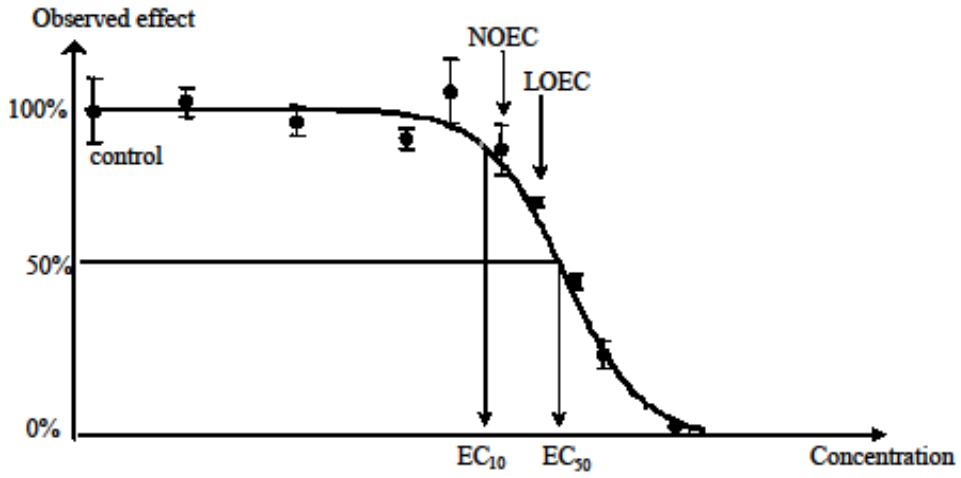

Figure 11. Generalized concentration-response relationship adapted from OECD (2006:Figure 3.2). Note that the illustrated curve is a plot fitted to a real dataset, thus the identification of NOEC and LOEC concentrations. For the purposes of this figure think of the y-axis as a positive attribute that becomes diminished by toxicity, such as percent survivorship.

Note that the superimposed straight line in Figure 11 represents the region of concentrations that induce an intermediate toxic response that are well approximated by a linear fit. This "linear region" is strongest within one probit (also known as "normal equivalent deviate") either side of the median response concentration (EC_{50}), or roughly for concentrations that induce 16 to 84% response (Environment Canada 2005). The narrow ranges of $SSEC_x$ values that we need to evaluate can be expected to overwhelmingly fall within those boundaries as a result of the methods EPA used to set the chronic criterion at 5.2 µg CN/L (see the next section titled, *Derivation of the Criterion Continuous Concentration*). Our approach is conceptually similar to the TRAP program's Piecewise Linear regression option (EPA 2002). Even with regard to the nonlinear regression options in TRAP, EPA (2002) provides a recommendation for segmented analysis when there is a focal region (or subset) of test concentrations of particular concern:

> *Within the limitations of this program, one useful approach can be to exclude (censor) high effects data from the analysis if (a) only low levels of effect are of interest and (b) there are sufficient low-to-moderate effects data to support a good analysis.*

Prediction model based on fathead minnow dataset. Lind et al. (1977) examined fathead minnow fecundity (number of eggs per spawn) and egg hatchability in relation to a series of cyanide treatments (concentrations). The experimental structure and fecundity results are summarized in Table 41. There were five control replicates, and two replicates each for ten exposure concentrations. The response data are reasonably monotonic, especially within the intermediate response range covered by the lowest six treatments. Those treatments range (on a free cyanide basis) from 6 to 45.6 ug/L CN; a span that closely corresponds to the $SSEC_x$ range we want to evaluate (Table 4).

Table 44. Egg production of adult fathead minnows exposed for 256 days (from larvae through adult) to various concentrations of cyanide (from Lind et al. 1977; Table II).

Treatment HCN (µg/L)	Mean HCN (µg/L)	Free cyanide as CN (µg/L)	Mean eggs per female	Mean eggs per female per treatment	Reduction in the number of eggs per female - percent of control
Control			2530	3476	
Control			4483		
Control			3990		
Control			2718		
Control			3660		
5.7	5.8	6.0	1886	2512	27.7
5.9			3138		
13.0	12.9	13.3[N]	1701	1845	46.9
12.7			1989		
19.6	19.6	20.2[L]	1694	1468	57.8
19.6			1241		
27.1	27.3	28.2	1093	1367	60.7
27.5			1640		
36.0	35.8	36.9	678	1010	71.0
35.6			1341		
43.7	44.2	45.6	2054	1124	67.7
44.7			194		
62.5	63.5	65.6	74	72	97.9
64.5			70		
73.1	72.8	75.1	573	319	90.8
72.4			64		
81.5	80.7	83.3	266	243	93.0
79.8			219		
96.1	100.8	103.9	0	0	100.0
105.4			0		

[N]NOEC
[L]LOEC

To "build" our prediction model we transformed both the concentration data and the fecundity data for *a priori* reasons. We log-transform the concentration data for two reasons: (1) statistically, toxicological tolerance distributions have long been confirmed as log-normal (OECD 2006), and (2) biologically, organisms experience toxicants on a log scale. Toxicological custom is to use log base-10 for the log transformations of test concentrations (Environment Canada 2005). Count data, such as "number of eggs per spawn" typically conform to a Poisson distribution rather than a normal distribution. To normalize such data for regression analysis a square-root transformation is recommended (EPA 2002). Thus, we use the square-root transformed response data for statistical analysis and then back-transform for reporting results. This transform does not change the model, but affects what the best parameter estimates and confidence limits are (EPA 2002). Thus, our model of choice is a log-square root linear regression over our focal segment (subset) of test concentrations.

In agreement with Gensemer et al.'s (2007) treatment of the same dataset, we collapse the fecundity and egg hatchability endpoints into a single endpoint, "eggs hatched per spawn" which

is the product of (eggs per spawn) x (egg hatchability) at each treatment concentration. We went a step further than Gensemer et al. (2007) and additionally apply a data smoothing procedure to meet the assumption of monotonicity of response inherent in a linear regression. We did that by calculating three-point moving averages for both the fecundity and hatchability endpoints. This is a standard statistical technique for separating the "signal" from the "noise" in epidemiological and earth sciences (e.g., Borradaile 2003; Rothman et al. 2008). Although we didn't use the control data in our focal segment linear regression, we estimate where the smoothed data would cross the y-axis by double-weighting the control value, which then along with its nearest neighboring data point provided the basis of a three-point moving average for the "endpoint" of the concentration series. This double-weighting is justified conceptually because a treatment to the left of the controls on the concentration axis would be expected to respond the same as the controls (Environment Canada 2005). This enables us to avoid comparing point estimates of eggs hatched per spawn from models fitted to smoothed data with "unsmoothed" control reference points. Note that our "smoothed" estimate of a control reference point is obtained using the actual data nearest to the y-axis and is not extrapolated from our estimated regression equation. Also note that we do not control-adjust the results prior to model fitting, a practice that leads to serious upward bias in EC_x point estimates (Environment Canada 2005; OECD 2006). A summary of response data smoothing and transformation is presented in Table 42.

Table 45. Fathead minnow input data for effects modeling

Treatment (free µg CN/L)	Pooled mean eggs/female	Pooled Proportion Hatch[a]	Unsmoothed Pooled mean hatch/female[b]	3-pt moving average of proportion hatch	Smoothed Pooled mean hatch/female[b]	SQRT transform
Control Mean	3476	0.842	2927	0.763[c]	2652	51.5
6.00	2512	0.606	1522	0.754	1894	43.52
13.30	1845	0.813	1500	0.682	1258	35.47
20.20	1468	0.626	919	0.612	898	29.97
28.20	1367	0.396	541	0.527	720	26.83
36.90	1010	0.559	565	0.354	358	18.92
45.60	1124	0.108	121	0.271	305	17.46
65.60	72	0.147	11	0.149	11	3.31
75.10	319	0.192	61	0.181	58	7.62
83.30	243	0.204	50	0.132	32	5.66
103.90	0	0	0	0.068[c]	0	0

[a]Means weighted by replicate sample sizes; excludes hatchability result for Control B as per authors' (Lind et al. 1977:264-265) recommendation
[b]Rounded to the nearest whole number
[c]Based on double-weighted observed value; assuming any doses to the left of 0% response will be constant and any points to the right of 100% response will be constant
[d]Final effects model based upon the shaded subset of data

The resulting log-square root focal segment linear regression model shows a very close fit to the data with an adjusted r-square of 0.964. The regression equation is:

Square-root (hatched eggs per spawn) = -30.19(LOG CN) + 68.36

The regression plot (Figure 12) and summary regression statistics (Table 43) are presented below. The regression was conducted using the multiple linear regression module of the *Statistica* software package (StatSoft 2006). Because we are dealing with small samples, i.e., six points in this case, we report the adjusted r-squared value which adjusts for the limited degrees of freedom in the model (StatSoft 2006).

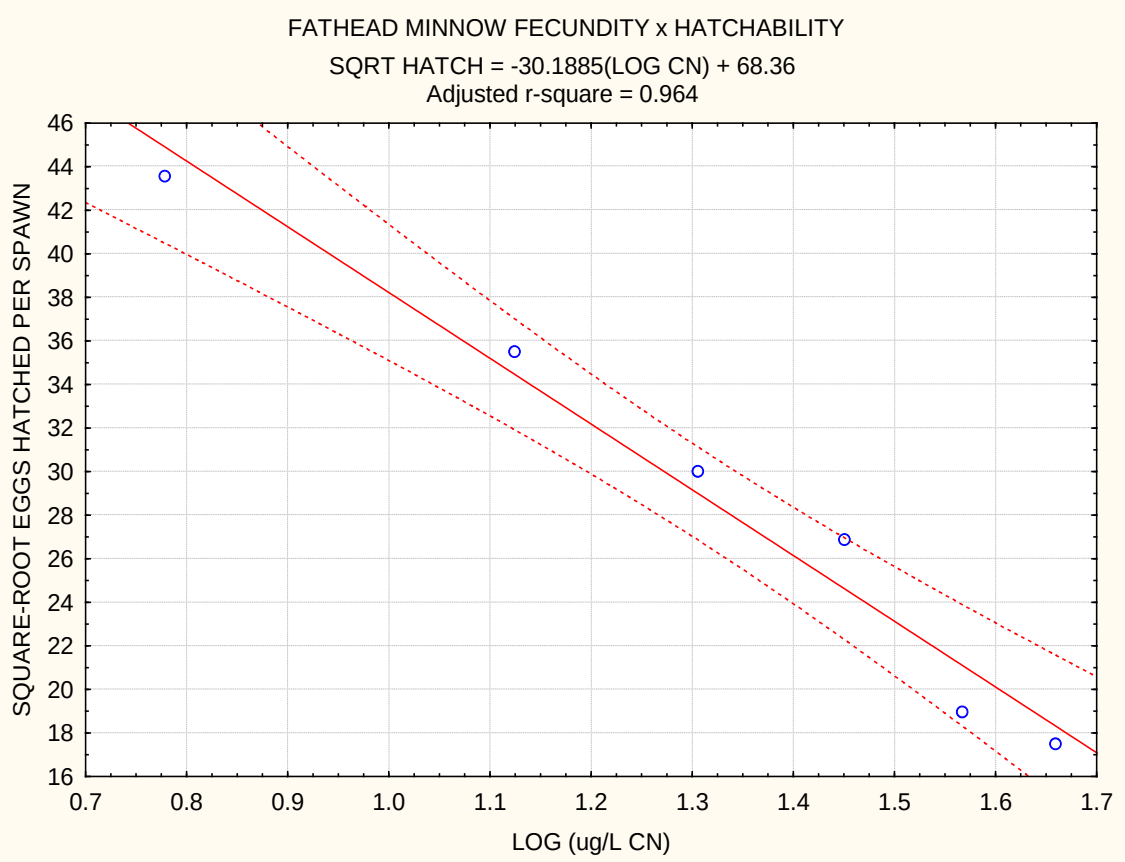

Figure 12. Log- Square Root Focal Segment Regression Plot for Fathead Minnow Fecundity x Hatchability (= Eggs Hatched Per Spawn)

Table 46. Summary regression statistics

Effects Surrogate	N	F value	p-level	Intercept	Std Err	p-level	Slope	Std Err	p-level
Fathead Minnow	6	134.6	<0.00032	68.36	3.505	0.000041	-30.19	2.602	0.00032
Brook Trout	5	12.34	<.039	24.85	2.595	0.0024	-6.594	1.877	0.039
Bluegill	5	11.75	<0.042	0.3514	0.9277	0.73	-2.533	0.7919	0.042

Prediction model based on brook trout dataset. Koenst et al. (1977) examined brook trout

fecundity (number of eggs per spawn) and egg viability in relation to a series of cyanide treatments (concentrations). The experimental structure, as well as the fecundity results are summarized below (Table 44). There were two control replicates, and seven cyanide treatments. The lowest five treatments produced intermediate effects responses and covered a range of concentrations from 5.6 to 53.2 µg/L CN; a span that closely corresponds to the $SSEC_x$ range we want to evaluate (Table 40). There was substantive variability in the results for the two control replicates. This lead Koenst et al. (1977) to exclude control replicate B, but noting that additional testing might indicate that the control results should be averaged. As noted in the footnote to Table 44, subsequent studies with brook trout (Holcombe et al. 2000) have confirmed that control replicate B should be averaged with control replicate A and therefore we use the control mean as our reference point for evaluating model predictions.

Table 47. Egg production of adult brook trout exposed to HCN for 144 days prior to the start of spawning (from Koenst et al. 1977)

HCN (µg/L)	Free cyanide as CN (µg/L)	Mean eggs spawned per female	Reduction in the number of eggs per female - percent of control*
Control A		502	
Control B		744	
Control Mean		623	
5.7	5.6	513	17.7
11.2	11.1	291	53.3
32.3	31.9	246	60.5
43.6	43.1	442	29.1
53.9	53.2	262	57.9
64.9	64.1	124	80.1
75.3	74.4	0	100.0

* Reductions in the number of eggs spawned relative to controls were calculated using the Control mean (623 eggs per female). Koenst et al. 1977 performed the same calculation using only Control A (502 eggs per female) and reported that the MATC (Maximum Acceptable Toxicant Concentration) lies between 5.7 and 11.2 µg HCN/L. However, the authors went on to say that "When compared to the mean of the two controls, 5.7 µg/L HCN would appear to show a substantial reduction in eggs spawned per female, but due to the high variability in spawning in the two controls, further study would be required to reach this conclusion." Since that time other studies with brook trout have been conducted (Holcombe et al. 2000). The mean number of eggs spawned per female observed by Koenst et al. 1977 is within the range reported for these other studies, which supports the use of data from both controls in estimating the effect of cyanide on brook trout fecundity.

Again, in agreement with Gensemer et al.'s (2007) treatment of the same dataset, we collapse the fecundity and egg viability endpoints into a single endpoint, "viable eggs per spawn" which is the product of (eggs per spawn) x (egg viability) at each treatment concentration. In the five-point segment of the data that we focus on, there was a substantive deviation from monotonicity at the 43.1 µg/L CN concentration. Therefore, once again we employed data smoothing with a 3-point moving average to restore a monotonic progression of responses. Because the endpoint here is virtually the same as the endpoint for the fathead minnow dataset, other aspects of our treatment of the data for "building" a prediction model are the same as already presented above. A summary of response data smoothing and transformation is presented in Table 45 below.

Table 48. Brook trout input data for effects modeling

Treatment (free CN µg/L)	Mean eggs/female	3-pt moving average of mean eggs/spawn	Proportion Viable	3-pt moving average of proportion viable	Smoothed mean viable/female[a]	SQRT transform
Control Mean	623	586[b]	0.935	0.923[b]	541	23.26
5.60	513	476	0.899	0.872	415	20.37
11.10	291	350	0.781	0.803	281	16.76
31.90	246	326	0.729	0.792	258	16.06
43.10	442	317	0.866	0.745	236	15.36
53.20	262	276	0.641	0.502	139	11.79
64.10	124	129	0	0.214	28	5.29
74.40	0	41[b]	0	0[b]	0	0

[a]Rounded to the nearest whole number
[b]Based on double-weighted observed value; assuming any doses to the left of 0% response will be constant and any points to the right of 100% response will be constant
[c]Final effects model based upon the shaded subset of data

The resulting log-square root focal segment linear regression model does not show as strong a fit to the data as the fathead minnow model does, but still shows a reasonably good fit with an adjusted r-square of 0.739. The regression equation is:

Square-root (viable eggs per spawn) = -6.594(LOG CN) + 24.85

The regression plot is presented in Figure 13 and summary regression statistics are presented in Table 43. The regression was conducted using the multiple linear regression module of the *Statistica* software package (StatSoft 2006). Because we are dealing with small samples, i.e., five points in this case, we report the adjusted r-squared value which adjusts for the limited degrees of freedom in the model (StatSoft 2006).

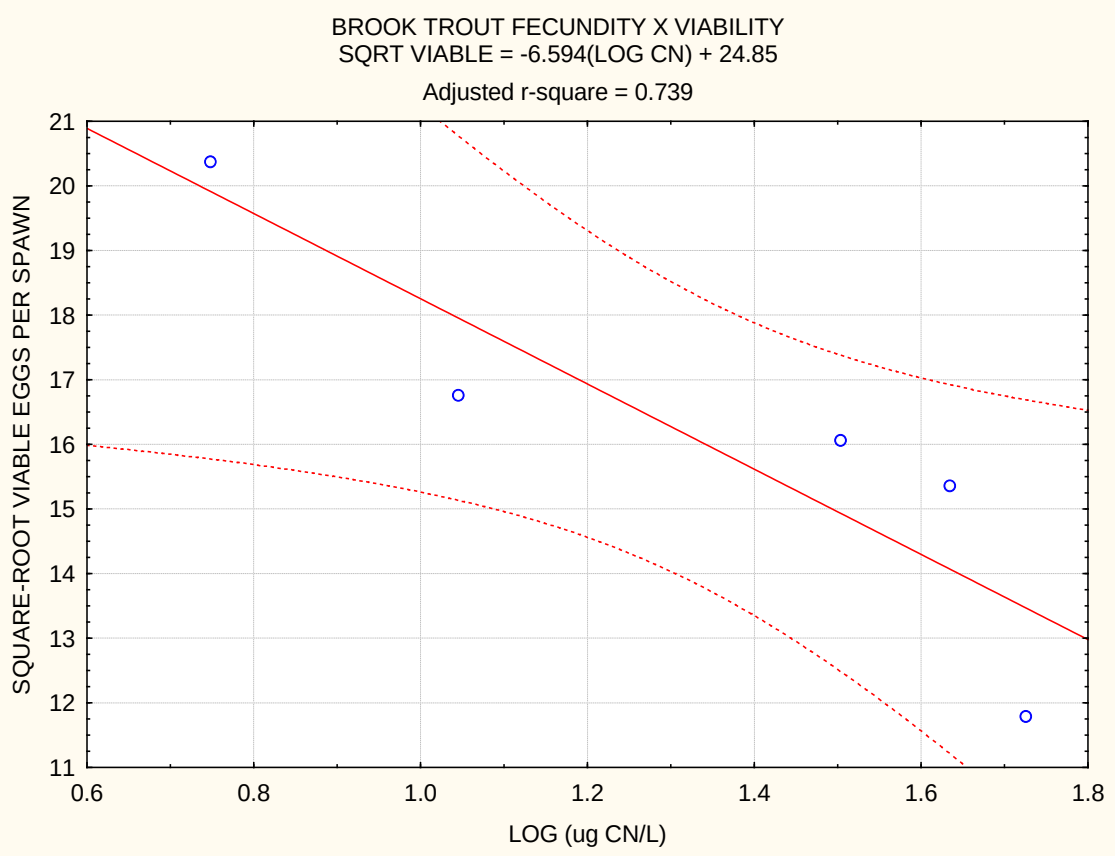

Figure 13. Log-Square Root Focal Segment Regression Plot for Brook Trout Fecundity x Viability(= Viable Eggs per Spawn)

Prediction model based on bluegill dataset. Kimball et al. (1978) examined bluegill juvenile survivorship in relation to a series of cyanide treatments (concentrations). The experimental structure, as well as the survivorship results are summarized in Table 46. There were four control replicates, and two replicates each for eight cyanide treatments. The lowest five treatments produced intermediate effects responses and covered a range of concentrations from 4.9 to 40.6 µg/L CN; a span that closely corresponds to the $SSEC_x$ range we want to evaluate (Table 40).

Table 49. Survival of bluegill from fertilized egg to the 57-day juvenile state in various HCN concentrations (from Kimball et al. 1978)

HCN (µg/L)	Mean HCN (µg/L)	Free cyanide as CN (µg/L)	Percent survival	Number of surviving juveniles *	Mean percent survival	Reduction in survival compared to controls
Control			37.5	75	23.3	
Control			20.0	40		
Control			10.0	20		
Control			25.5	51		
4.8	4.8	4.9	18.5	37	18.5	20.6%
5.2			lost			

8.9	9.1	9.4[N]	25.0	50	16.3	30.0%
9.2			7.5	15		
19.2	19.4	19.9[L]	3.0	6	2.8	88.0%
19.6			2.5	5		
28.5	29.1	29.9	2.5	5	2.5	89.3%
29.7			2.5	5		
38.7	39.5	40.6	3.0	6	3.8	83.7%
40.2			4.5	9		
49.3	49.3	50.7	13.5	27	13.5	42.1%
51.9			lost			
61.8	62.9	64.6	0.0	0	0.0	100.0%
64			0.0	0		
80.4	82.1	84.4	0.0	0	0.0	100.0%
83.8			0.0	0		

*Number of surviving juveniles was calculated by multiplying the reported percent survival times the starting number of fertilized eggs per treatment (200).
[N]NOEC
[L]LOEC

The bluegill dataset differs qualitatively from the fathead minnow and brook trout datasets because the response variable, juvenile survivorship is a quantal (binary) rather than continuous variable. Quantal variables conform to a binomial distribution. Such data are typically analyzed via either probit transformation, as employed by Gensemer et al. (2007), or logit transformation of the proportions of responding and non-responding test subjects. Probits are normal equivalent deviates and logits are logistic equivalent deviates. These two transforms usually yield similar estimates of EC_{50} values, but differ appreciably in their EC estimates in the tails of the distributions.

Environment Canada (2005) recommends logistic methods over probits for "... mathematical simplicity and other good reasons." Logit = ln (p/1-p), where p is the proportion of effected test subjects (e.g., if juvenile survival were 30% for a particular treatment concentration, p would equal 0.3 and the logit transform would equal -0.8473). The logit transform linearizes the sigmoidal logistic response curve (Environment Canada 2005; StatSoft 2006). Furthermore, in fitting the logit model, the control observations can be excluded, as they do not provide any information, unless a background parameter in included (OECD 2006).

Both Environment Canada (2005) and OECD (2006) note that it is common practice to correct the data for background response prior to analysis (for example via Abbott's correction), but that such pre-treatment of the data is unsound statistical practice that can result in substantive overestimation of EC_x values. The bias increases as the control effect being adjusted for increases. We fit a focal segment of the bluegill dataset to a log-logit regression using results that are not control-adjusted prior to analysis. Thus, our prediction model yields unbiased estimates of proportion effect that can be control-adjusted for reporting purposed after the fact. The dataset is reasonably monotonic until the highly anomalous result for the treatment at a concentration of 50.7 µg/L CN. Gensemer et al. (2007) censored that point as an outlier. Because our $SSEC_x$ range extended up to only 41.7 µg/L CN (Table 40) the 50.7 µg/L CN treatment did not fall within our focal segment of concern. The last three treatments in our focal

segment produced results of greater than 84% effect which would place them in the nonlinear upper tail of the sigmoidal curve (Figure 11), but unlike a log-square root regression the logit transform will linearize points in the tails relative to intermediate effects points. Thus, for log-logit regression points that fall in tails do not have to be avoided in order to apply linear regression. The minor deviation from monotonicity in the last two points of our focal segment did not warrant data smoothing. A summary of the logit transformed response data is presented in Table 47.

Table 50. Bluegill input data for effects modeling

Treatment (free CN µg/L)	Mean surviving juveniles	Proportion Survival	Logit Proportion Survival
Control Mean	46.5	0.2325	-1.1942
4.9	37	0.1850	-1.4828
9.4	32.5	0.1630	-1.6361
19.9	5.5	0.0280	-3.5472
29.9	5	0.0250	-3.6636
40.6	7.5	0.0380	-3.2314
50.7	27	0.1350	-1.8575
64.6	0	0.0000	
84.4	0	0.0000	

[a]Final effects model based upon the shaded subset of data

The resulting log-logit focal segment linear regression model does not show as strong a fit to the data as the fathead minnow model does, but with an adjusted r-square of 0.729 shows a reasonably good fit comparable to that achieved for the brook trout dataset. The regression equation is:

$$\text{Logit (proportion juvenile survival)} = -2.533\ (\text{LOG CN}) + 0.3514$$

The regression plot is presented in Figure 14 and summary regression statistics are presented in Table 43. The regression was conducted using the multiple linear regression module of the *Statistica* software package (StatSoft 2006). Because we are dealing with small samples, i.e., five points in this case, we report the adjusted r-squared value which adjusts for the limited degrees of freedom in the model (StatSoft 2006).

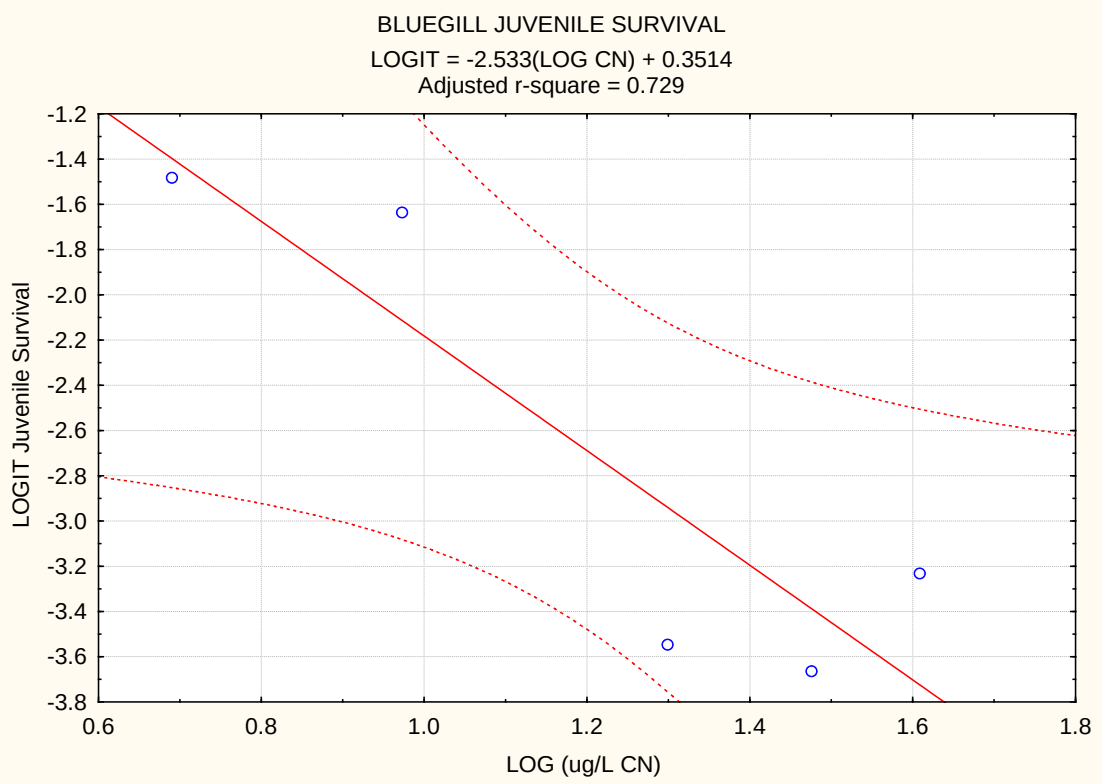

Figure 14. Log-logit focal segment regression plot for bluegill juvenile survival

Prediction Results

Effects predictions are generated by substituting LOG ($SSEC_x$) for LOG (CN) into the prediction regression equations. This was accomplished via the "predict dependent variable" algorithm in the multiple linear regression module of Statistica (StatSoft 2006). That algorithm also uses the estimated standard error of the regression coefficient to generate 95% confidence limits for the predicted point estimates (maximum likelihood estimates). For the fathead minnow and brook trout prediction regressions, the prediction and confidence limit output are in the form of square-roots of numbers of eggs. To convert those predictions to a percent effect, the predicted results were first squared and then scaled for percent change compared to the applicable smoothed control value according to the formula:

$$\% \text{ Effect} = [1 - (\text{predicted egg count} / \text{smoothed control value})] \times 100$$

Any predicted egg counts exceeding the smoothed control value were automatically converted to 0% effect. For the bluegill prediction regression, the prediction and confidence limit output are in the form of logit transforms for proportions of juvenile survivorship. The logit transforms are back-transformed to proportions by the formula:

$$\text{Proportion survival} = e^{(\text{logit})} / 1 + e^{(\text{logit})}$$

The predicted survival proportions are scaled for percent change compared to the reported control value according to the formula:

% Effect = [1- (predicted proportion survival / mean control proportion survival)] x 100

Again, any predicted survivorship exceeding the observed mean control survivorship results in a percent effect prediction that is automatically converted to 0% effect. The raw input and output data for effects predictions are presented in Appendix E.

A summary of predicted effects and their estimated 95% confidence limits from each of the three prediction models for each of the 14 surrogate taxa from which listed-species' LC_{50} values were derived are presented in Table 48. The effects estimates are presented in Table 49for the listed species (i.e., matches up the effects estimates for surrogate taxa in Table 48 with the listed species linked to each surrogate taxon).

The EC_{10} and EC_{20} concentrations for each of our three regression models were also estimated. The fathead minnow regression yielded an estimated EC_{10} of 4.4 µg/L CN (95% CI = 2.6-6.2 µg/L CN) and an estimated EC_{20} of 5.5 µg/L CN (95% CI = 3.5-7.4). By comparison, Gensemer et al. (2007) estimated an EC_{20} of 6.0 µg/L CN from a log-probit analysis of the fathead minnow data, but did not report confidence limits for that estimate. The brook trout regression yielded an estimated EC_{10} of 2.6 µg/L CN (95% CI = 0.0-8.4 µg/L CN) and an estimated EC_{20} of 4.1 µg/L CN (95% CI = 0.0-11.1). Gensemer et al. (2007) estimated an EC_{20} of 7.7 µg/L by linear interpolation of the brook trout data, and again did not report confidence limits for that estimate. The bluegill regression yielded an estimated EC_{10} of 4.6 µg/L CN (95% CI = 0.0-10.5 µg/L CN) and an estimated EC_{20} of 5.3 µg/L CN (95% CI = 0.0-11.5). Gensemer et al. (2007) estimated an EC_{20} of 5.6 µg/L CN from a log-probit analysis of the bluegill data, and also estimated an EC_{20} of 8.9 µg/L CN for the bluegill data from EPA's TRAP program. All of Gensemer et al.'s (2007) estimates fall within our 95% confidence limits, and in general show excellent agreement with our results even though Gensemer et al's methods differed from ours. This suggests that our results are not highly dependent on the particular statistical approach that we chose for our analysis.

Table 51. Estimated magnitude of effect of cyanide (at the CCC, 5.2 µg CN/L) on surrogate taxa for listed fish species (95% CL)*

Surrogate taxa used to estimate magnitude of effect on listed species	Surrogate species		
	Fathead Minnow	**Brook Trout**	**Bluegill**
	Reduction in the mean number of hatched eggs per spawn compared to controls	**Reduction in the mean number of viable eggs per spawn compared to controls**	**Reduction in the number of surviving larvae/juveniles compared to controls**
Actinopterygii (class)	48% (39%, 56%)	30% (1%, 55%)	56% (3%, 82%)
Order Cypriniformes	39% (28%, 49%	26% (0%, 54%)	44% (0%, 80%)

Family Catostomidae			
Xyrauchen texanus (species)	39% (28%, 49%)	26% (0%, 54%)	44% (0%, 80%)
Cyprinidae (family)	29% (15%, 42%)	21% (0%, 53%)	30% (0%, 78%)
Cyprinella monacha (species)	68% (63%, 72%)	42% (23%, 58%)	76% (50%, 89%)
Gila elegans (species)	57% (51%, 63%)	36% (12%, 56%)	66% (30%, 84%)
Notropis mekistocholas (species)	59% (53%, 65%)	37% (14%, 56%)	68% (34%, 85%)
Ptychocheilus lucius (species)	63% (57%, 68%)	39% (18%, 57%)	71% (41%, 86%)
Order Perciformes	36% (24%, 47%)	24% 0%, 53%)	40% 0%, 79%)
Percidae (family	63% (58%, 68%)	39% (18%, 57%)	72% (43%, 87%)
Etheostoma (genus	65% (60%, 70%)	40% (20%, 58%)	74% (46%, 88%)
Etheostoma fonticola (species)	81% (76%, 85%)	52% (37%, 64%)	86% (64%, 95%)
Order Salmoniformes, Family Salmonidae			
Oncorhynchus (genus)	60% (54%, 65%)	37% (15%, 57%)	69% (36%, 85)
Oncorhynchus apache (species)	87% (82%, 91%)	56% (42%, 68%)	90% (67%, 97%)
Oncorhynchus tschawytscha (species)	71% (67%, 76%)	45% (27%, 59%)	79% (55%, 91%)
Oncorhynchus kisutch (species)	71% (66%, 75%)	44% (27%, 59%)	79% (55%, 90%)
Oncorhynchus mykiss (species)	52% (45%, 59%)	33% (6%, 55%)	61% (16%, 83%)
Oncorhynchus clarki henshawi (species)	80% (75%, 84%)	51% (36%, 63%)	85% (63%, 94%)
Salvelinus (genus)	87% (83%, 92%)	57% (43%, 69%)	90% (68%, 97%)

*The magnitude of effect was estimated using the regression model for each surrogate response species and SS EC_X value for each surrogate taxa (Table 40). For each surrogate taxa there were two estimates of effects on reproductive performance and one estimate of effects on early life stage survival.

Table 52. Estimated magnitude of effect of cyanide (at the CCC, 5.2 µg CN/L) on listed fish species (95% CL). There are two estimates for effects on fecundity and one estimate for effects on early life stage survival for seven listed species due to exposure at based on surrogate species data.

Listed Species	Surrogate Taxa	Estimated reduction in fecundity and larvae/juvenile survival due to cyanide exposure at the CCC based on surrogate species data sets		
		Fathead minnow[1] (Percent reduction in the mean number of hatched eggs per spawn compared to controls)	Brook trout[2] (Percent reduction in the mean number of viable eggs per spawn compared to controls)	Bluegill[3] (Percent reduction in the number of surviving larvae/juveniles compared to controls)
Coho salmon *(Oncorhynchus kisutch)*	*Oncorhynchus kisutch*	71 (66, 75)	44 (27, 59)	79 (55, 90)
Chinook salmon (*Oncorhynchus tschawytscha)*	*Oncorhynchus tschawytscha*	71 (67, 76)	45 (27, 59)	79 (55, 91)
Chum salmon *(Oncorhynchus keta)*	*Oncorhynchus (genus)*	60 (54, 65)	37 (15, 57)	69 (36, 85)
Sockeye salmon *(Oncorhynchus nerka)*	*Oncorhynchus (genus)*	60 (54, 65)	37 (15, 57)	69 (36, 85)
Steelhead (*Oncorhynchus mykiss)*	*Oncorhynchus mykiss*	52 (45, 59)	33 (6, 55)	61 (16, 83)
Shortnose sturgeon *(Acipenser brevirostrum)*	Actinopterygii (class)	48 (39, 56)	30 (1, 55)	56 (3, 82)
Green sturgeon *(Acipenser medirostris)*	Actinopterygii (class)	48 (39, 56)	30 (1, 55)	56 (3, 82)

[1]Based on data contained in Lind et al. 1977
[2]Based on data contained in Koenst et al. 1977
[3]Based on data contained in Kimball et al. 1978
[4]Based on data contained in Schimmel 1981

Other Effects Estimates

The estimates of effects presented in Table 49 are based largely on ICE LCL (lower confidence limit) LC_{50} values for listed fish evaluation species. Those are the LC_{50} values that we accept as sufficiently accounting for the uncertainties inherent in relying on surrogate data and numerous other untested assumptions to estimate the sensitivity of listed species to cyanide. The Service, NMFS, and EPA agreed that using ICE LCL values was preferable to the practice of applying arbitrary uncertainty factors.

However, EPA has, at various times, questioned whether the use of ICE LCL values might not be overly conservative. Therefore, we also estimated effect levels using ICE MLE (maximum likelihood estimates) LC_{50} values for listed fish evaluation species (via revised $SSEC_x$ estimates). Those results are presented in Appendix F. Based on the fathead minnow prediction model, which was the strongest model, the median levels of effect predicted for the 15 ICE surrogate taxa were 51% and 65%, respectively, for ICE MLE and ICE LCL. The number of surrogate taxa with a predicted effect of 35% or greater was 11 and 14, respectively, for ICE MLE and ICE LCL. Those differences indicate only modest conservatism conferred by ICE LCL-based effects estimates as compared to ICE MLE-based estimates. Such modest differences would not have a decision-making impact. For both sets of results, unacceptably high levels of effect would overwhelmingly be the predominant prediction.

Empirical Test of Method Performance

Because only three concentration-response datasets are available, there is almost no basis for testing our method performance (i.e., there are no known directly measured "true" values for effects to our listed fish evaluation species at a concentration of 5.2 µg/L CN). However, because the fathead minnow and brook trout datasets focused on essentially the same response variable (number of hatchable/viable eggs produced per spawn) we can perform two tests of method performance. For each species, we can directly estimate a predicted effect level at 5.2 µg/L CN using the species-specific regressions. Those would be our estimates of the "true" effect level. Next, we can use our surrogate method and estimate an $SSEC_x$ for each species on the other species' response curve and evaluate the predicted effect level for that $SSEC_x$ value and compare the surrogate estimate to the estimated "true" value. The results are as follows:

> The directly estimated fathead minnow effect level at 5.2 µg/L CN is 18% with a 95% CI of 0%-34%. The fathead minnow $SSEC_x$ value on the brook trout response curve would be 3.2 µg/L CN, which yields an effects estimate of 15%. That is nearly identical to estimated "true" value and easily within the 95% CI for the "true value".
>
> The directly estimated brook trout effect level at 5.2 µg/L CN is 25% with a 95% CI of 0%-54%. The brook trout $SSEC_x$ value on the fathead minnow response curve would be 8.4 µg/L CN, which yields an effects estimate of 38%. Again, that is within the 95% CI for the "true" value, although our estimate of the "true" value is not very precise and therefore the 95% CI is fairly wide.

In summary, in both test cases, the estimated effect level derived from our surrogate methodology is not significantly different from the estimated "true" value in a statistical sense, but the second

comparison has low statistical power. Further validation testing of this sort should be done as concentration-response datasets become available for more species using a comparable response variable, but it is reassuring that in these test cases our method yielded results that were nearly identical to the "true" value in one case and reasonably close to the "true" value in the other case.

Derivation of the Criterion Continuous Concentration (CCC)

Our analysis predicts that the listed fish species considered in this Opinion would be severely affected by exposure to cyanide at the CCC. National criteria are typically derived using chronic toxicity data from laboratory tests. As noted earlier, most aquatic life criteria that have been derived thus far, including the cyanide criterion, chronic values have been obtained by calculating the geometric mean of the lower and upper chronic limits. In practice, the upper and lower chronic limits are often statistically determined by hypothesis testing. The lower limit is typically the NOEC, which is defined as the highest test concentration where the effects are not statistically significantly different from controls. The upper limit is typically the LOEC, which is defined as the lowest test concentration where the effects are statistically significantly different from controls. The guidelines recommend that the magnitude of effect associated with the upper and lower chronic limits should be considered when determining values that appropriately estimate acceptable and unacceptable levels of adverse effect:

> *Because various authors have used a variety of terms and definitions to interpret and report results from chronic tests, reported results should be reviewed carefully. The amount of effect that is considered unacceptable is often based on a statistical hypothesis test, but might also be defined in terms of a specified percent reduction from the controls. A small percent reduction (e.g., 3%) might be considered acceptable even if it is statistically significantly different from the control, whereas, a large percent reduction (e.g., 30%) might be considered unacceptable even if it is not statistically significant."*

Based on this guidance, the threshold for unacceptable adverse effects would be estimated by the chronic value. The magnitude of effect at the threshold would then be equivalent to the magnitude of effect at the chronic value. For chronic criteria derived using hypothesis tests, this would be the magnitude of effect occurring at a concentration equal to the geometric mean of the NOEC and LOEC, that is, somewhere between an acceptable and unacceptable level of adverse effect. The guidelines do not specify a level of adverse effect on which the threshold for unacceptability should be based. The only mention of a numeric value or range is provided in the guidance for selecting chronic limits (mentioned above) and suggests that this threshold may lie between 3% and 30%.

Thus, for a given species or test the magnitude of effect at the chronic value will depend on the magnitude of effect at the lower and upper chronic limits. We followed this approach for estimating the magnitude of effect occurring at the cyanide CCC. The freshwater cyanide CCC was derived based on chronic toxicity data for 4 species (Table 50): 3 fish (fathead minnow, brook trout, and bluegill) and 1 invertebrate (*Gammarus pseudolimnaeus*). Chronic values for each species were obtained by calculating the geometric mean of the lower and upper chronic limits. The magnitude of effect at the lower and upper chronic limits was calculated by comparing responses at the lower and upper limits to controls. For fathead minnow and brook trout these effects were expressed as reduction in the mean number of eggs spawned per female

compared to controls; for the bluegill the effect was reduction in larvae/juvenile survival compared to controls; and for *G. pseudolimnaeus* the effect was a reduction in the mean number of eggs or young per gravid female relative to controls.

We then estimated the magnitude of effect at the chronic value by linear interpolation between lower and upper chronic limits (Table 50). Based on these calculations the magnitude of effect at the chronic values for the fathead minnow, brook trout, bluegill and *G. pseudolimnaeus* would be 52%, 32%, 54%, and 47%, respectively. According to the guidelines, if there were a sufficient number of chronic values (i.e., chronic values for species in 8 phylogenetic families) the chronic criterion could be computed directly from the distribution of chronic values (see earlier discussion under *Derivation of Criteria*). If there were fewer chronic values, as was the case for cyanide, the chronic criterion would be computed using ACRs. ACRs for the 4 freshwater species were reported in the cyanide criterion document and are shown in Table 50. The ACRs were calculated by dividing the species mean acute value (i.e., mean LC_{50} for the species) by the chronic value. For example, the ACR for fathead minnow (7.633) was computed by dividing 125.1 µg CN/L (the mean LC_{50} for the species) by 16.39 µg/ CN/L (the chronic value). Thus, the ACR is the ratio between the concentration of cyanide causing 50% lethality (following acute exposure) and the concentration following chronic exposure that causes a level of adverse effect that is at the threshold of unacceptability, i.e., 52% for fathead minnow. The guidelines require that, for criteria derivation, the geometric mean of individual species ACRs is used to obtain the Final ACR. For cyanide, the freshwater Final ACR was 8.562 (Table 50). We estimated the magnitude of chronic effects associated with the Final ACR to be about 45% (Table 50).

The Final ACR and the FAV were then used to derive the CCC. The guidelines describe how the FAV is computed. In short, the FAV is set equal to the 5^{th} percentile estimate from the distribution of genus mean acute values. In other words, the FAV represents the genus with acute sensitivity (LC_{50}) in the *sensitive tail* of the distribution where, theoretically, approximately 5% of the genera would be more sensitive and about 95% of the genera would be less sensitive. Based on this analysis, the FAV for cyanide was determined to be 62.68 µg CN /L. Since the guidelines include provisions for adjusting the FAV to protect commercially and recreationally important species, EPA lowered the FAV from 62.68 µg/L to 44.73 µg/L because the SMAV for rainbow trout (44.73 µg/L) was below the calculated FAV. The cyanide criterion (5.2 µg/L) was then derived by division of the FAV (44.73 µg/L) by the Final ACR (8.562). Thus the chronic criterion, 5.2 µg CN/L, was based on the concentration intended to protect rainbow trout from unacceptable adverse effects. Based on our estimate of the magnitude of effect associated with the Final ACR, we estimate the magnitude of adverse effects occurring to rainbow trout at the chronic criterion to be approximately 45%. This value is higher than we would have expected considering it is intended to represent the threshold for unacceptable adverse effects. However, the magnitude is in line with effects we predicted for the other listed fish species, most of which were estimated to be as (or more) sensitive to cyanide as rainbow trout.

Table 53. Chronic toxicity data used by EPA to derive the freshwater chronic criterion for cyanide. **Effect levels were calculated using data from the original papers.**

Species	Chronic Limits[1]				Chronic Value[2]		LC_{50}[3] (µg CN/L)	ACR[3]
	Lower		Upper					
	(µg CN/L)	Effect	(µg CN/L)	Effect	(µg CN/L)	Effect		
Fathead Minnow	13.3	47%	20.2	58%	16.39	52%	125.1	7.633
Brook Trout	5.6	18%	11.0	53%	7.849	32%	83.14	10.59
Bluegill	9.3	30%	19.8	88%	13.57	54%	99.28	7.3
Gammarus	16	0%	21	100%	18.33	47%	167	9.111
Geometric mean						45%		8.562

[1] Lower and upper chronic limits were taken from the cyanide criteria document. For fathead minnow and bluegill these values were determined statistically (i.e., NOEC and LOEC identified via hypothesis tests). Effect levels were take from Tables 5, 8 and 10 in the Effects section of the BO and from Oseid and Smith 1979.

[2] Chronic values were taken from the cyanide criteria document. Effect levels associated with the chronic values were estimated by linear interpolation between the effects at the lower and upper chronic limits.

[3] Acute-Chronic Ratios were taken from the cyanide criteria document.

This same conclusion, that NOEC/LOEC-based estimates of "chronic values" can correspond to $\geq$ 40% adverse effect, has also been reached by others. Decades ago Suter et al. (1987) reported that MATC's for fish fecundity, on average, corresponded to a 42% level of adverse effect (MATC = Maximum Acceptable Toxicant Concentration; a term for the geometric mean of the NOEC and LOEC from a given toxicity test and often assigned by EPA as the estimated "chronic value" from a test). Other response endpoints were found to correspond to average adverse effect levels of 12-35%. More recently, SETAC (Society for Environmental Toxicology and Chemistry) convened a panel of experts (Reiley et al. 2003) who concluded that "*...[toxicity] tests with high variability may result in an(sic) NOEC corresponding to a response greater than 40% different from the control.*" Moore and Caux (1997) statistically examined nearly 200 toxicity data sets and found that most NOEC's (76.9%) exceeded a 10% adverse effect level and most LOEC's (62.4%) exceeded a 30% effect level. Various other researchers have noted a variety of adverse effect levels for NOEC's, such that Crane and Newman (2000) were led in summary to conclude that "… *[NOEC] effect levels from individual tests ranged from nearly 0% to nearly 100%.*" For seven cyanide toxicity tests with sufficient data for comparison, Gensemer et al. (2007: Figure 3-7) found in all cases that the geometric mean of the NOEC and LOEC corresponded to an adverse effect level of $\geq$ 20% (how much greater was not reported).

Because of the highly variable and often substantive levels of effect associated with NOEC's, LOEC's, MATC's, and with the "chronic values" based on them, and for numerous other reasons, a strong professional consensus recommendation to avoid using NOEC/LOEC-based estimates for regulatory thresholds (when possible) has been expressed repeatedly. For example, there was an ISO (International Organization for Standardization) resolution (ISO TC147/SC5/WG10 Antalya 3) as well as an OECD (Organisation for Economic Co-operation and Development) workshop recommendation (OECD 1998) that the NOEC should be phased out from international standards (OECD 2006:14). Environment Canada (2005) notes, that there is a growing literature which points out many deficiencies of the NOEC approach (Andersen et

al. 2000; Bailer and Oris 1999; Chapman 1996; Chapman et al. 1996; Crane and Godolphin 2000; Crane and Newman 2000; Miller et al. 1993; Moore and Caux 1997; Noppert et al. 1994; Pack 1993; Pack 1998; Suter et al. 1987; Suter 1996). Moving away from the NOEC/LOEC approach was also among the recommendations of the SETAC panel for improving the scientific basis of water-quality criteria (Reiley et al. 2003).

Accordingly, EPA has begun employing a regression approach for estimating "chronic values" whenever sufficient data are available to do so. For example, in the 1999 update for ammonia water quality criteria EPA used regression analyses to estimate 20% effect concentrations (EC_{20}s) from individual toxicity tests and used those EC_{20}s as estimates of chronic values (EPA 1999). Likewise, estimated EC_{20}s have been the basis for estimating chronic values in recently proposed updates for copper and selenium water quality criteria (EPA 2003a, 2004). EPA's choice of the EC_{20} as a basis for estimating chronic values was justified from statistical considerations rather than from biological or demographic considerations:

> *To make [chronic values] reflect a uniform level of effect, regression analysis was used here both to demonstrate that a significant concentration-effect relationship was present and to estimate [chronic values] with a consistent level of effect. Use of regression analysis is provided for on page 39 of the 1985 Guidelines (Stephan et al. 1985). The most precise estimates of effect concentrations can generally be made for 50 percent reduction (EC50); however, such a major reduction is not necessarily consistent with criteria providing adequate protection. In contrast, a concentration that caused a low level of reduction, such as an EC5 or EC10, is rarely statistically significantly different from the control treatment. As a compromise, the EC20 is used here as representing a low level of effect that is generally significantly different from the control treatment across the useful chronic datasets that are available for ammonia.*

Pack (1993) asserted that most ecotoxicologists consider effects in the range of 5-20% to be biologically acceptable depending on the species involved and the type of effect. However, EPA appears to have chosen the top end of that range based more on the expected statistical power of toxicity tests than on a serious examination of the typical demographic sensitivity of biotic populations to a 20% adverse effect on survival, growth, or reproduction. Furthermore, 95% statistical confidence limits for most EC_{20} estimates are likely to extend well into adverse effect levels that would be of unquestionably serious demographic concern for most organisms. As evident from the above discussion, most chronic criteria derived by EPA, including for cyanide, are highly likely to be associated with $\geq$ 20% adverse effect level for species at the vulnerable end of species sensitivity distributions (such as the subset of ESA-listed species we are evaluating). Therefore, it should be no surprise that our estimated effect levels for such species at the current cyanide CCC of 5.2 µg/L are almost always higher than 20% and in some cases substantially higher.

Population Responses to Reductions in Fecundity and Juvenile Survival

Laboratory experiments have demonstrated that even closely related fish species can demonstrate great differences in sensitivity when exposed to the same chemical, as measured by differences in acute or chronic toxicity values. This variability in sensitivity has been related to differences in species' physiology and life history strategies. Similarly, population modeling and experimental

studies have shown that variation in population-level responses to environmental toxicity can also be expected among species as a consequence of factors such as life history strategies, life stage affected, and density dependence. Studies have also demonstrated that chronic toxicity can lead to population decline and extirpation.

Under the ESA, in determining whether a proposed Federal action is likely to jeopardize the continued existence of a listed species under the ESA, we assess whether the proposed activity reasonably would be expected to appreciably reduce the likelihood of survival and recovery of a listed species by reducing its reproduction, numbers, or distribution. Two common metrics used in population modeling to assess effects of perturbations on populations are population growth rate and time to or probability of extinction.

Population growth rate is the change in a population size over a unit time period. Long-term reductions in population growth rate as low as 5% has been shown to significantly increase a population's likelihood of extinction (Snell and Serra 2000). Population growth rate can be positive when the population is increasing, negative when decreasing, or zero when the net difference between births, deaths, and migration is zero and the population is stable. For listed species, populations may exist in any of these states depending on its recovery status. Our analysis determines the relative predicted effects of the action to the population growth rate, regardless of its starting value.

Using known parameters of a species' life history, sensitivity analyses can be conducted to determine which parameters, when modified, will have the greatest impact on the species' population growth rate. Elasticity analysis is one type of sensitivity analysis that is commonly used in conservation biology to demonstrate the relative contributions to population growth rate made by life cycle transitions, based on vital rate statistics for survival, growth and fertility. While these types of analyses cannot predict absolute effects to population size, because they quantify the relative importance of an element to changes in population growth rate, they can help focus management decisions on those demographic parameters that exhibit the largest elasticity, and thus, the largest impact on population growth (de Kroon et al. 2000). However, elasticity analysis requires the development of a population model, for which adequate data are often scarce. Because this type of demographic data is often lacking for threatened and endangered species in particular, the need to develop generalized approaches for classifying population responses to perturbation for rare species has been recognized (Dennis et al. 1991; Heppell et al. 2000).

Several authors have examined the effect of life history strategies on the elasticities of various demographic measures. In evaluating demographic parameters of 50 mammal populations with different life history strategies, Heppell et al. (2000) found that phylogeny alone is often not a reliable indicator of which vital rates (survival, growth and fertility) will have the greatest impact on elasticity. Instead, the authors found that species that mature early and have high reproductive output had high fertility elasticities and low adult survival elasticities. Conversely, for those which mature late and have long lifespans, fecundity and early offspring survival are less important than survival of juveniles to maturity to changes in population growth rate. Calow et al. (1997) also found that the relative importance of juvenile fish survival can vary according to reproductive strategy. These authors concluded that reductions in juvenile survival would have

the greatest impact on semelparous fish species, in which adults die after reproduction, a lesser impact on a moderately iteroparous population, in which adult postreproductive survival is intermediate, and the least impact on strongly iteroparous species, in which adult survival after reproduction is high. These assumptions held true for elasticity analysis of the green sturgeon, a fish species with life history patterns such as late-maturity and long-life that are common to other sturgeon (Heppell 2007).

Juvenile survival had relatively lower elasticity values than adult and subadult survival, with compensation for the loss of adults requiring much larger increases in young-of-the-year survival than would be commensurate with the loss. However, other authors have found increased importance of juvenile survival for sturgeon, despite their lifespan (Gross et al. 2002; Paragamian and Hansen 2008). Gross et al. (2002) hypothesized that this difference was due to the vastly larger fecundity of sturgeon as compared to other long-lived species.

Vélez-Espino et al. (2006) argue the need for a broadscale summary of species' population dynamics to help guide the conservation biology of freshwater fishes, for which information on life history is often limited. Using information, on adult survival, juvenile survival, and fecundity, the authors performed elasticity analyses on 88 species of freshwater fish and found that they could be classified into 4 functional groups with regard to the sensitivity of their population growth rates:

1. species most sensitive to perturbations in adult survival
2. species most sensitive to perturbations to adult and juvenile survival
3. species most sensitive to perturbations to juvenile survival
4. species most sensitive to perturbations to juvenile survival and fecundity

These groups are characterized by decreased age at maturity, longevity, and reproductive lifespan as one moves from group 1 to group 4. Age at maturity, reproductive lifespan, fecundity, juvenile survivorship, and longevity were all correlated with adult survival and fecundity. However, the best predictors of elasticity patterns were longevity, which explained 93% of the variability in the elasticity of adult survival, and age at maturity, which explained 92% of the variability in the elasticity of fecundity. The authors also found that elasticities are highly conserved among genera within the same taxonomic family

Spromberg and Birge (2005) also found that life history strategies influence effects to populations. The five life history strategies they modeled encompassed differences in stage-specific survival, fecundity and hatch success, number of spawning events, and life-span. The authors found that regardless of strategy, changes in the number of young-of-the-year stage individuals had the greatest impact on population growth rate. However, the relative contribution of this parameter was greatest for life history strategies with multiple spawnings, high fecundity, and short lifespans as opposed to those with longer lifespan, which had increased elasticity of adult survival.

Spromberg and Meador (2005) linked toxicant effects on immune suppression, reproductive development, and growth reduction to demographic traits in Chinook salmon and modeled their influence on population growth rate. Overall, effects to first- and second-year survival had the greatest elasticities, with constant reductions to first year survival as low as 10% achieving

population declines ranging from 35-78% compared to controls. Other studies have demonstrated the importance of first year survival in this species (Kareiva et al. 2000). Spromberg and Meador (2005) also found that models which incorporated effects to both survival and reproduction were additive, indicating the importance of evaluating the overall impact of all potential impacts to population growth.

Many listed species populations are limited by the amount of adequate habitat or resources and experience some degree of density dependence. Density-dependence at any life stage must be considered in elasticity analysis in order to yield appropriate results (Grant and Benton 2000; Hayashi et al. 2008). In a review of toxicant impacts on density-limited populations, Forbes et al. (2001) noted that the full range of interactions have been found between toxicant stress and density dependence, including less than additive, additive, and more than additive effects. Also, the type of effect may vary with increasing toxicant concentration from one that ameliorates density dependent effects at low toxicant concentrations to one that exacerbates density dependent effects at higher toxicant concentrations. Case studies which incorporate density-dependence into population modeling demonstrate this variability, with overall impacts to populations shown to be both lesser (Van Kirk and Hill 2007) and greater (Hayashi et al. 2008) than the level of effect that would be predicted from individual response depending on the situation. In time, density-dependant populations may rebound, stabilize at a lower absolute population number, or continue to decline until the population is extirpated (Forbes et al. 2001). Modeling exercises have demonstrated cases in which populations stabilize at new, lower equilibrium abundances in response to a constant impact (van Kirk and Hill 2007; Spromberg and Meador 2005).

A species' likelihood of persistence can also be estimated a number of ways. There are no standard methods or protocols to estimate the risk of extinction. Instead, the method used is usually dependent on the availability of data available on the species in question and species' biology. Extinction risk analyses methodologies may be qualitative, semi-quantitative, or quantitative. One quantitative method that is used widely for modeling a species' time to extinction or probability of extinction is Population viability analysis (PVA). PVAs use simulation modeling to identify threats to species and to assess the vulnerability of populations to extinction risks. These models incorporate demographic parameters such as fecundity, survivorship, age structure, and population size, but can also incorporate effects to the environment such as habitat degradation and catastrophic events. As for the evaluation of population growth rate, sensitivity analysis is used to determine which factors have the greatest impact on population persistence, and many experts feel that parsing out these influential factors for management purposes is the best utilization of these models, as opposed to absolute predictions of population decline. However, PVA models require a depth of demographic data that is often lacking for listed species.

For Pacific salmon, NMFS has not found a PVA that completely represents the various risks facing salmon populations (McElhany et al. 2000). Consequently NMFS created the viable salmonid population concept to provide useful benchmarks for evaluating actions that directly affect natural populations and for which incremental increases in extinction risk may be difficult or impossible to accurately quantify. Where PVAs have been conducted for specific populations of salmon, these have informed NMFS in status assessments. While the VSP concept isn't meant

to replace quantitative models where they can be properly used because the VPS employs a combination of quantitative and qualitative methods for determining the extinction risk of listed species it is more flexible and easier to use where data are limited (McElhany et al. 2000). Under the VSP approach, risk is first addressed at the population level and then the ESU. Individual populations are assessed according to four parameters: abundance, growth rate/productivity, spatial structure, and diversity. NMFS focuses on these parameters because they are reasonable indicators of extinction risk (viability). Although, there is no formal link between VSP and jeopardy under Section 7, the same population level parameters used in VSP, whenever available, are a significant part of our analysis in determining whether an agency's action is likely to jeopardize the continued existence of a listed species.

Summary of Population Responses to Reductions in Fecundity and Juvenile Survival

Modeling and experimental studies have shown that chronic toxicity to pollutants can lead to population decline and extirpation. Variation in population-level responses to environmental toxicity can be expected among species as a consequence of factors like species life history strategies, life stage affected, density dependence, and magnitude of toxicant stress. Although the degree varied among different life history strategies, fecundity and juvenile survival remained a highly influential demographic parameter throughout modeled scenarios, with adult survival taking on greater importance in long-lived species. These results must be coupled with other influences on the population status, such as the degree of density dependence and additional environmental perturbations such as catastrophes. Although population modeling often requires more demographic information than is available for threatened and endangered species, careful selection of surrogates and use of their data may allow for extrapolation from models for species with similar life histories.

Summary of the Direct Effects

According to our analysis, Chinook, chum, coho, and sockeye salmon, and green and shortnose sturgeon exposed to cyanide are likely to experience reduced survival, reproduction, and may also experience effects on growth, swimming performance, condition, and development, as described above. Our analysis demonstrates that acute and chronic toxicity may be exacerbated by other stressors such as dissolved oxygen concentration, temperature, and the presence of other pollutants in the water column. That is, the threshold of adverse effects is diminished in the very cold waters and low dissolved oxygen conditions.

Relatively few studies were available for estimating the magnitude of effects that could occur following exposure to cyanide at criterion concentrations. Because no data for cyanide toxicity to sturgeon exist, LC50 values for sturgeon were derived from the 5% SSD concentration for the class Actinoptergyii, which encompasses all known cyanide toxicity data for fish. From this data, we developed quantitative estimates of the effects on fecundity, hatching success, and survival of young first-year fish (Table 49). Given the limited data set, our estimates are the same for green sturgeon and shortnose sturgeon, as well as sockeye salmon and chum salmon (the latter are based on data from the genus *Oncorhynchus*). Based on our analysis, we estimate that green and shortnose sturgeon exposed to cyanide at the CCC may experience a reduction in juvenile survival that is as high as, but not likely to be greater than, 56%. Our estimates reveal

that the green and shortnose sturgeon may experience a reduction in the number of hatched eggs and that reduction could be as high as, but is not likely to be greater than, 30%.

Similarly, we expect that coho and Chinook salmon would experience a reduction of juvenile survival and that reduction could be as much as, but is not likely to be greater than 79%. We estimate that, when exposed to cyanide at the CCC, coho and Chinook salmon may experience a reduction in the number of hatched eggs and that reduction could be as much as, but is not likely to be greater than, 45%. Similarly we expect that chum and sockeye salmon would experience a reduction in juvenile survival and that reduction could be as much as, but is not likely to be greater than 69%. We estimate that, when exposed to cyanide at the CCC, chum and sockeye salmon may experience a reduction in the number of hatched eggs and that reduction could be as much as, but is not likely to be greater than, 37%. Our estimates reveal that steelhead would experience a reduction in juvenile survival and that reduction could be as much as, but is not likely to be greater than 61%. We estimate that, when exposed to cyanide at the CCC, steelhead may experience a reduction in the number of hatched eggs and that reduction could be as high as, but is not likely to be greater than, 33%.

Young of the year fish, and juvenile fish that do survive exposure to cyanide could experience reduced growth rates which would increase their vulnerability to a host of potential stressors, including temperature, flow, and inter- and intraspecific competition for food and cover. We expect that such exposure could also delay reproductive maturity and productivity. These reductions in reproductive performance and survival represent reductions in the fitness of the individuals exposed to cyanide at the chronic criterion concentration. Changes in the fitness of the individuals in a population will affect the population as a whole, and could be measured in terms of changes in population growth rates and changes in risk of extinction.

Sturgeon have naturally high adult survival, and the loss of juvenile life stages is particularly problematic. Several authors have suggested that the rate of survival may be so high that management at the levels of these age classes is unlikely to improve their survival or increase population growth rate (Gross et al 2002; Heppell 2007). As such, recovery efforts are often based upon increasing survival in juvenile age classes. Gross et al (2002) modeled population growth rates for three species of sturgeon that varied in life history traits such as size, lifespan, age to maturity, and migration. All three sturgeons showed similar elasticity profiles, and thus the authors concluded that general interpretation could be applied to sturgeon across species. In contrast to other elasticity profiles for long-lived species, elasticity in sturgeon was highest in individual young-of-the-year and juvenile age classes, dropped at the onset of maturity, and continued to decline for each successive adult age class. Fecundity had relatively low elasticity, as the effects of changes in fecundity are shared among all adult age classes of these long-lived species, and the value of changes to egg numbers is lessened by the high mortality of the young-of-the-year age class. The authors concluded that population growth rate would show little response to improvements in fecundity, but greater responses in survival at either the young-of-the-year or juvenile age classes. However, since survival of the juvenile and adult age classes is naturally high, improvements at these stages will have smaller effects to improving population growth rate than increases to survival of young-of-the-year, when natural mortality is greater. The authors note that among biologists and managers involved in sturgeon conservation, habitat improvement was regarded as the most important conservation undertaking for sturgeon. Results

from this study indicate that restoration efforts should target the survival of age classes with high elasticity, specifically young-of-year and juvenile. Paragamian and Hansen (2008) drew similar conclusions in modeling effects on population growth of the Kootenai River white sturgeon. The authors found that subadult and adult survival (>90%) was much higher than that of juveniles (40% in the first year), and recovery was dependent on increasing first-year survival. The authors suggested that to have the largest effect on recovery, managers should increase the current targeted recruitment rate.

Unlike sturgeon, most Pacific salmon (with the exception of steelhead and cutthroat trout) are semelparous, such that they spawn only once. Consequently, reductions in the number of viable eggs and juvenile survival through their first year would likely have greater population-level effects on Chinook, coho, sockeye, and chum salmon. Low fresh water is survival is considered typical of most salmon populations, although estimates for many populations are nonexistent, mortality rates are recorded from 40-90% (Sandercock 1991; Bradford 1997). According to Brandford, the coefficient of variation (CV) for interannual survival in fresh water is about 30% averaged over all species. The factors that influence the freshwater survival rate for the likely differs somewhat between widely-dispersed spawning species (e.g., steelhead, coho and Chinook salmon) compared to those that spawn in dense aggregations (e.g., sockeye and chum salmon), as well as the length of time spent in freshwater rearing (e.g., coho salmon versus early migrant Chinook salmon or chum salmon). For Pacific salmon, mortality appears to be roughly equally divided between fresh water and marine waters, suggesting that each habitat contributes to recruitment variation (Bradford 1997). Consequently, significant reductions in freshwater production would be expected to significantly affect the number of adults returning to fresh water to spawn.

As discussed earlier, there are several factors that can influence the relative toxicity of chemical contaminants under natural exposure conditions. When organisms are stressed due to environmental factors outside their normal optima they may become more sensitive to a given toxicant. This can occur when homeostasis is disrupted in organisms that are infected with a pathogen, outside their normal range for various water quality parameters (salinity, pH, or temperature), diseased, or debilitated due to other toxic insults. Very cold temperatures and low DO conditions increase the toxicity of cyanide. Despite the limited number of studies on these influencing factors, until more work can be done we have little evidence to suggest species specific responses to cyanide under low DO conditions or low water temperatures. Considering that cyanide is a respiratory toxin that inhibits oxidative metabolism, it is not surprising that the effects are exacerbated under conditions where oxygen availability is limited. Any factor that affects gill ventilation will also likely affect the amount and speed at which the toxin is distributed in the body. A fish is under stressed conditions like oxygen depletion, would typically increase their ventilation rate to compensate for the low DO and would, in this situation also increase their rate of uptake of aqueous cyanide.

In summary, exposure to aqueous cyanide at the approved CCC and CMC is likely to lead to the fitness consequences for Chinook, coho, sockeye, and chum salmon, and green and shortnose sturgeon. In particular, exposure to cyanide concentrations at the chronic criterion could substantially reduce reproduction by reducing the number of eggs spawned by females, reducing the hatchability of spawned eggs, and reducing the survival of young fish through the first year.

Sturgeon and salmon may also experience effects on growth, swimming performance, condition, and development. While sturgeon have developed a life history that allows them to cope with low survivorship to maturity and occasional decreases in recruitment, these adaptations are unlikely to compensate for a constant reduction in both fecundity and early life stage survival. The reductions we estimate in survival of young fish through the first year in particular would substantially decrease survival and recovery of this species. Because of the high magnitude of effects, we would expect density-dependent compensatory mechanisms, if they exist, to be overwhelmed.

Based on our analysis, we expect that the proposed action would significantly reduce the absolute numbers of green sturgeon, shortnose sturgeon, Chinook salmon, chum salmon, sockeye salmon, coho salmon, and steelhead. Based upon the magnitude of effects we anticipate could occur, the distributions of green and shortnose sturgeons are likely to be reduced in waters where they are exposed to cyanide at the levels defined by the chronic criterion, and may be reduced when cyanide exposure overlaps with low water temperatures or low DO concentrations.

Critical Habitat

We evaluated the effect of EPA's approval of the cyanide water quality standards on the effect of critical habitat by first reviewing the essential features or primary constituent elements of critical habitat for listed and proposed designations. Based on our analysis, the primary features that may be affected by EPA's approved water quality criteria are those designated as "water quality" areas for growth, development and reproduction (salmon and green sturgeon). We evaluated the "water quality" feature according to whether the acute or chronic criteria were likely to reduce the amount of clean water available for supporting essential patterns of growth, development or reproduction.

Approval of the CCC in state water quality standards would allow states to manage cyanide in waters to these levels. Even if waters never systematically reached these levels, the use of the aquatic life criteria in NPDES permits, TMDL limits, indicates the importance that these numeric values play in the overall success and operation of the water quality program. Our analysis demonstrates that where cyanide concentrations reach the approved standard, the proposed action would likely adversely affect the quality of water to the degree that it would impair individual reproduction and survival of green sturgeon, Chinook salmon, coho salmon, chum salmon, sockeye salmon and steelhead, and would cause these species to experience adverse effects to growth, swimming performance, condition, and development. For green sturgeon, we estimate the reduction in the number of hatched eggs could be as high 48% and the reduction in the survival of young fish through the first year as high as 56%. For coho and Chinook salmon, we estimate the reduction in the number of hatched eggs could be as high 45% and the reduction in the survival of young fish through the first year as high as 79%. For chum and sockeye salmon, we estimate the reduction in the number of hatched eggs could be as high 37% and the reduction in the survival of young fish through the first year as high as 69%. For steelhead, we estimate the reduction in the number of hatched eggs could be as high 33% and the reduction in the survival of young fish through the first year as high as 61%. These effects are estimated to be of a magnitude great enough to reduce numbers of both sturgeon and salmon. Approval of the CCC would adversely affect the quality of water to the degree that normal population growth would be

severely reduced, and sturgeon and salmon may be extirpated from critical habitat containing cyanide at approved values. Not only would impacts to water quality resulting from management of cyanide to the CCC diminish the ability of critical habitat to provide for conservation of the these species, our analysis also suggests that the conservation value of critical habitat for these species would likely be diminished at concentrations below EPA's recommended CCC for fresh water.

The Impacts of Reduced Salmon Populations – Summary of Indirect Effects

Salmon are a significant contributor to the overall ecological food web throughout their range, whether they are from listed populations or unlisted populations. Two significant indirect effects of the proposed action, attributable not to the direct toxicity of cyanide, but the action's impact on Chinook, coho, sockeye and chum salmon and steelhead population abundance would include the further loss of primary prey species for southern resident killer whales and Cook Inlet beluga whales, and the loss of salmon nutrient transport to freshwater systems, which indirectly affects their own productivity. Bilby et al. (1996) demonstrate that juvenile and older age classes of salmon grow more rapidly with the appearance of spawners because these younger fish will feed on eggs and spawner carcasses. Salmon carcasses strewn along river reaches and streambanks are a significant source of food to a wide number of animals and affect the overall productivity of nutrient-poor systems (Bilby et al. 1996; Cederholm et al. 2000). The loss of these "marine derived nutrients" likely significantly reduces the survival of their own species, particularly in nutrient poor streams. Bilby et al. (1996) demonstrated that the mean fork length of juveniles and up to 45% of the carbon in cutthroat trout and 40% of the carbon in young of the year coho comes from the decaying carcasses of the previous generation of salmon. The increased body size is directly correlated to increases in over winter survival and marine survival. Based on historical cannery records and current records of escapement, Gresh et al. (2000) estimate this nutrient source has declined to about 13 to 17 percent of the historic biomass of return salmon to Pacific Northwest streams (Washington, Oregon, Idaho, and California). They suggest that this loss is one important indicator of ecosystem failure, contributing to the observed reductions in abundance we have seen in many salmon populations, and could further hamper recovery efforts. Thus, while we may have estimated the direct loss of individuals attributable to the proposed action, further reductions in many populations would be expected as adult spawner numbers decline from reduced recruitment attributable to the proposed action.

Similarly, although not obligate feeders, southern resident killer whales feed primarily on salmon and salmon are seasonally an important prey for Cook Inlet beluga whales. The reductions in salmon populations anticipated as a result of this action can be expected to have significant affects on southern resident killer whales and their critical habitat, and Cook Inlet beluga whales and their proposed critical habitat. Based on killer whale stomach contents from stranded whales and field observations of predation, Ford et al. (1998) determined that 95% of the diet of resident killer whales consists of fish, with a significant portion being Chinook salmon (about 2/3 of the samples that were identified to species). The authors suggested that Chinook salmon may be preferentially hunted by killer whales because of their large body size, high fat content, and seasonal distribution patterns. Although, Cook Inlet beluga whales feed on a variety of other fish species Pacific salmon are an important prey species for these animals as they build their lipid

body stores essential to their winter survival. The significant reduction in the southern resident killer whale's primary prey species, Pacific salmon in general and in particular Chinook salmon, from the proposed action is likely to have significant effects on the fitness of southern resident killer whales and their population viability. As noted earlier, a 50% reduction in killer whale calving has been correlated with years of low Chinook salmon abundance (Ward et al. 2009a). Cook Inlet beluga whales would similarly experience a significant reduction in their most abundant summer and fall prey species (most of which, are non-listed Chinook, coho, sockeye, and chum species, although some listed species may be consumed during their marine migrations to Alaska). The proposed action, based on our analysis would significantly reduce freshwater production of all listed salmon species, as well as non-listed salmon species where cyanide concentrations are allowed to reach EPA's recommended aquatic life criteria concentrations. As noted earlier, we expect the proposed action would cause as high as a 79% reduction in the survival of juvenile (young fish through their first year) Chinook salmon, and as high as a 45% reduction in the number of viable eggs. These losses would severely reduce the number of adult Chinook salmon in the Puget Sound ESU, and would reduce the forage base for southern resident killer whales. Southern resident killer whales are not restricted to Puget Sound, but do spend a large portion of time in Puget Sound, the Strait of Juan de Fuca, and Haro Strait. Prey losses would also be realized throughout their range, including Oregon and California. Consequently, we expect that the proposed action would significantly reduce the absolute numbers of southern resident killer whales by reducing the absolute numbers of their primary prey. Based upon the magnitude of effects estimated to salmon, we expect the numbers, distribution and reproduction of southern killer whales would likely to be reduced due to significantly a reduced forage base.

Similarly, we expect the proposed action would cause as high as a 79% reduction in the survival of juvenile (young fish through their first year) coho salmon, as high as a 69% reduction in the survival of juvenile sockeye and chum salmon, and as high as a 44% reduction in the number of viable coho salmon eggs, and as high as a 37% reduction in the number of viable sockeye and chum salmon eggs. These losses would severely reduce the forage base of Cook Inlet beluga whales, and as a result we expect that the proposed action would significantly reduce the absolute numbers of Cook Inlet beluga whales by reducing important prey species. Based upon the magnitude of effects estimated to salmon, we expect the numbers, distribution and reproduction of Cook Inlet beluga whales would likely be reduced due to a significantly a reduced forage base.

Critical Habitat of Southern Resident Killer Whales

We evaluated the effect of EPA's approval of the cyanide water quality standards on the effect of critical habitat by first reviewing the essential features or primary constituent elements of critical habitat for listed designations. Based on our analysis, the primary features that may be affected by EPA's approved water quality criteria are those designated as "prey species of sufficient quantity, quality, and availability to support individual growth, reproduction and development, as well as overall population growth." Based on our analysis, we estimate that coho and Chinook salmon will experience reductions in the number of hatched eggs as high 45% and the reduction in the survival of young fish through the first year as high as 79%. For chum and sockeye salmon, we estimate the reduction in the number of hatched eggs could be as high 37% and the reduction in the survival of young fish through the first year as high as 69%. For steelhead, we estimate the reduction in the number of hatched eggs could be as high 33% and the reduction in

the survival of young fish through the first year as high as 61%. These effects are estimated to be of a magnitude great enough to reduce numbers of these listed salmon species. Approval of the CCC would adversely affect the quality of water to the degree that normal salmon population growth would be severely reduced, and salmon may be extirpated from areas containing cyanide at approved values. These losses would severely diminish the ability of critical habitat to provide for conservation of the southern resident killer whales.

Proposed Critical Habitat of Cook Inlet Beluga Whales

We evaluated the effect of EPA's approval of the cyanide water quality standards on the effect of critical habitat by first reviewing the essential features or primary constituent elements of critical habitat for the proposed designation for Cook Inlet beluga whales. Based on our analysis, the primary features that may be affected by EPA's approved water quality criteria are those primary prey species consisting of Chinook, coho, sockeye, and chum salmon. Based on our analysis, we estimate that coho and Chinook salmon will experience reductions in the number of hatched eggs as high 45% and the reduction in the survival of young fish through the first year as high as 79%. For chum and sockeye salmon, we estimate the reduction in the number of hatched eggs could be as high 37% and the reduction in the survival of young fish through the first year as high as 69%. These effects are estimated to be of a magnitude great enough to reduce numbers of these salmon species. Approval of the CCC would adversely affect the quality of water to the degree that normal salmon population growth would be severely reduced, and salmon may be extirpated from areas containing cyanide at approved values. These losses would severely diminish the ability of critical habitat to provide for conservation of Cook Inlet beluga whales.

Cumulative Effects

Cumulative effects include the effects of future state, tribal, local, or private actions that are reasonably certain to occur in the action area considered in this biological opinion. In this section we focus on the status and trends of land-uses across the United States and the consequences of those land uses for listed and proposed resources. Since our action area encompasses a very broad spatial scale, we focused on key properties of ecosystem condition and the actions that influence those properties. According to the Consultation Handbook (USFWS and NMFS 1998), the "reasonably certain to occur" clause may include such indicators of actions such as approval of an action by a state, tribal or local agencies or government; indications that granting authorities for the action are imminent; project sponsor's assurance that actions will proceed, etc. Although speculative non-federal actions are not factored into the analysis, at the same time "reasonably certain to occur" does not require a guarantee that an action will occur, therefore a degree of uncertainty is acceptable when characterizing cumulative effects.

Due to the scale at which a national consultation occurs, the degree of uncertainty increases, particularly with respect to anticipating the cumulative effects of future non-federal actions across the action area. We necessarily relied on types of human activity (e.g., regional trends and projections in population increases, and associated industrial and commercial development) as proxies for the suite of hydrological, chemical, and biological changes that would reasonably be expected in the surrounding landscape. Metrics of land use (e.g., percent impervious or urbanization; road density) are strongly correlated to a variety of ecological indicators of stress

(e.g., changes in aquatic community; increases in chemical constituents, physical stream-channel condition; NRC 2008). Based on our knowledge of past changes within a watershed and the effects landscape changes have had on aquatic ecosystems, we can anticipate the general types and patterns of future land uses will have on the physical, chemical and biological conditions of downstream waterways. The specific factors that are important within a specific locality will vary from place to place, and over time.

The information we present herein is based on data produced by recognized organizations using demographic data, and economic and labor statistics and include their reasoned rough-trend estimates of population and economic change stemming from these data. Changes in the near-term (5-year; 2013) are more likely to occur than longer-term projections (10-year; 2018). Because the anticipated effects are based upon projections that are subject to error and alteration by complex economic and social interactions, our analysis does not address small or localized changes in aquatic habitats. Further, since the effects of future federal actions that are unrelated to the proposed action are not to be considered herein because they require separate consultation pursuant to Section 7, wherever possible, we eliminated known or typical future federal actions from our analysis (e.g., construction of new oil platforms). Many of the actions we discuss herein, such as construction and industrial development, are planned, approved and permitted through wholly local and state approvals and with private funds. However, in many instances we found it impossible to differentiate between non-federal and federal actions, and therefore we erred on including a general type of action in our analysis recognizing that a portion may qualify as federal actions and would not normally be included in our cumulative effects analysis. For example, transportation projects may be undertaken by local and state entities, and others may qualify as federal actions for reasons of federal funding, permitting, etc. In this instance, we were unable to discern federal transportation related actions from future non-federal transportation-related actions therefore we focused on general patterns we might in various regions and the generalized impacts of transportation projects on water quality.

Sources queried for the information herein include the United States Census Bureau, Department of Labor, and Lexis-Nexis information system. With the latter (which was our source for state legislation), we reviewed bills passed in 2007 to 2008 and pending bills under consideration were included as further evidence that actions “are reasonably certain to occur”. Bills that died in process or were vetoed are not included in our review.

Northeast Projection

We began our review for each region by examining current and pending state legislation for regional and local policy and political trends that may impact future development and management directions within the area. For instance, we looked for regulatory and political impetus for changes in zoning, fisheries, environmental standards, and development of commerce and industry. For the Northeast, we selected Maine as a representative state for this effort because of the extent of coastline and waterways, as well as the presence of habitat for several listed species from different taxa. We found that legislation in the state shows tendencies towards controlling invasive species, chemical (wastewater, pesticide, oil, nutrients, bacteria, and other toxic contamination) and sedimentation impacts humans have on rivers and nearshore waters, emissions associated with global warming, and the ability of fish to migrate past river

infrastructure. As a general matter, we expect that other coastal states within this region likely have programs or interests engaged in many similar activities, many of which are designed to minimize some of the adverse effects associated with increasing development and extraction industries.

In general, the northeast region is one of the most densely populated regions in the United States. Based upon 2000 United States census data, the northeast United States was predicted to contain 54.8 million people in 2005, and population growth is predicted to decrease over the foreseeable future from 0.41%/year between 2000 and 2010 to 0.24%/year from 2010 to 2020 (USCB 2005a). Much of the regional population is contained in concentrated metropolitan centers. If these cities were to continue to grow at the rate which they did from 2000 to 2007 (USCB 2008), the largest growth will occur in Dover, DE (2.89%/yr), Washington, D.C. metro (1.51%/year), and York-Hanover, PA (1.47%/year). The only population center greater than one million people growing at greater than one percent per year is Washington, D.C. Overall, the northeast United States is predicted to have 55.8 million people in 2010, 56.6 million in 2015, and 57.1 million in 2015. Growth of metropolitan centers will increase discharge of wastewater from water treatment systems into rivers and streams, which will increase the loads of contaminants carried by these waterways to the marine environment, and would have concomitant effects on such parameters as biological oxygen demand, chemical oxygen demand, DO, and water temperature. It is likely that development will continue along the coast and waterways, which will add sediment to river systems and potentially alter spawning habitat. Oil and other roadway pollutants may increase as a result of additional vehicular traffic. Additional recreational use of lakes, waterways, and coastal areas will increase fish takes and add additional discharges from vessels.

Industrial changes can indirectly add pressures to ESA listed species' survival and the health of their habitats. From 2006 to 2016, output of the mining industry is expected to increase by 1.0%/year (Figueroa and Woods 2007), which is a 25% decline in growth from what it was between 1996 and 2006. However, technological advancements will likely increase output in this sector. It should be noted that 60% of this industry is comprised of oil and natural gas, very little of which exists in the northeast United States. Coal output is likely to increase with demand for power through the electrical grid. Most significantly for the northeast, metal mining is anticipated to increase 4.3%/year with demand by various technologies and rising metal process. Currently, granite, peat, roofing slate, iron ore, sulfur, magnetite, manganese, copper, zinc, mica, and precious metals are mined in the region, with numerous others on an infrequent or historical basis (see baseline for additional information). Increasing output by existing and new mines can place additional pressures on species recovery in the foreseeable future by increasing waste runoff into streams and rivers.

Nationwide, construction is forecasted to be one of the most extensively growing industries in the United States. From 2006 to 2016, the construction industry is expected to grow by 1.4%/year and employ an additional 600,000 people during that time (Figueroa and Woods 2007). However, this represents a 30% slow-down from the 1996 to 2006 time period. Construction will be most likely to occur in school, industrial, and medical areas, as well as infrastructure (bridge and road) repair and replacement. An increase in construction will entail additional development in urban and non-urbanized areas that can introduce large amounts of sediment into

waterways via run-off, altering riverine habitat relied upon by salmonids. Sediments can also reduce water clarity and food availability resulting from loss of primary productivity. Sediment run-off can also introduce nutrients into marine environments that can cause algal blooms, which have been documented in nearshore habitats of the northeast United States, and introduce neurotoxins to large areas and cause wide-scale mortality (Vitousek et al. 1997).

Output of the transportation industry is expected to increase by 2.9%/year from 2006 to 2016 (Figueroa and Woods 2007), placing additional pollution pressures on listed species and their habitats. Although this rate is slower than the trend from 1996 to 2006, additional movement of freight by truck, plane, and train introduces pollutants, especially oils, to waterways that can increase petroleum concentrations in streams and estuaries. Greater potential for moderate- to large-scale pollutant release by spills and accidents also exists. Carbon dioxide released from petroleum combustion is a significant component of global warming (Vitousek et al. 1997; Nordhaus 2007; EIA 2007) and increases in the transportation will likely mean greater contributions of carbon dioxide and exacerbation of the global warming phenomenon. Based upon these factors, additional recovery pressures are likely to occur from the future growth of the transportation industry.

With increasing population, the leisure and hospitality industry is forecasted to grow by 2.1%/year from 2006 to 2016 (Figueroa and Woods 2007). As with other industries, this is a decline from the 1996 to 2006 rate by about 25%. In addition, most growth will likely occur in food services or drinking places, which is not expected to have impacts to listed species. However, this industry includes personnel and activities that utilize natural and protected areas. Additional use will likely include more debris and pollution discharge into areas frequently used by protected species. It can be contended that additional use of parks can increase outreach and public awareness of protected species and their habitats, which can benefit recovery of these species and areas. It is not known whether growth in the leisure and hospitality industry will have a net positive or negative impact on ESA listed species, but likely will include both helpful and hurtful aspects.

In contrast to other industries, agriculture is forecasted to increase in rate of growth from 2006 to 2016 versus the growth experienced from 1996 to 2006 (Figueroa and Woods 2007). Growth will increase from 1.3%/year to 2.2%/year, a change of roughly 75%. The increase results from increased efficiency from technological improvements and the rise of ethanol from crops. In this sector, agriculture accounts for over 80% of production, which masks regionally important factors. Agriculture in the northeast overshadows a projected output decline in forestry (-0.9%/year) and fisheries/hunting/trapping (-2.9%/year). Agriculture is not as extensive as in other regions of the United States and growth. However, additional growth will increase pollution and sediment runoff into streams, placing additional stress on salmon habitat and making bloom conditions more likely in marine areas where rivers discharge. Based upon the declines in fisheries and forestry, it is unlikely that extensive additional pressures will be placed on ESA listed species recovery by these two industries.

Southeast and Mid-Atlantic Projection

State legislation frequently shows regional and local policy and political trends that can significantly impact future directions within the area. Florida was selected as an example of

legislative trends in the mid-Atlantic and Gulf of Mexico because of the extent of coastline, presence of diverse and numerous listed species, socio-economic similarities to other states, large population, and progressive tendencies. Here, legislative regulation is moving towards management of beaches, control of watersheds and vessel discharges, protecting marine resources, restoration of freshwater habitats, identifying issues and contributing factors to climate change, limitation of oil and gas development, and lowering harmful chemical inputs into systems.

Mid-Atlantic states (including Florida) are predicted to increase in population from 55.7 million people in 2005 to 59.8 million in 2010 and 64.0 million in 2015. This is the fastest rate of anticipated regional growth in the nation except for western states (USCB 2005b). The rate of regional growth is anticipated to remain above 10% through 2030 and will be greatest in Florida and North Carolina and lowest in West Virginia. Although this region includes a larger area than the northeast, urban growth is much more extensive in the mid-Atlantic; 12 metropolitan areas experienced population growth of 3%/year or greater from 2000 to 2007, including the Atlanta area, once considered the most rapidly developing area in human history. However, half of these urban centers were in Florida. Cities of over one million people that grew at a rate of 1%/year or greater from 2000 to 2007 included Raleigh, NC (4.49%/year), Atlanta, GA (3.47%/year), Charlotte, NC (3.44%/year), Orlando, FL (3.37%/year), Jacksonville, FL (2.27%/year), Tampa-St. Petersburg, FL (1.96%/year), Richmond, VA (1.51%/year), and Miami, FL (1.16%/year). This rapid and concentrated population increase places much larger demand upon natural systems. Wastewater systems must handle larger loads of sewage. As soil is covered by asphalt and concrete, run-off must be channeled into local stormwater drains increasing contaminant load in streams. Regional areas of development are frequently in low-elevation locations, limiting water retention and movement. Both of these are sources of concern for sediment and contaminants entering local waterways and flowing into rivers, estuaries, and nearshore marine habitats.

Economic development will contribute additional pressures to ESA-listed species of the mid-Atlantic region. West Virginia is mined extensively for coal and demand for this resource to meet the needs of coal-fired power plants will drive increasing production (Figueroa and Woods 2007). Production of North Carolina's cement constituents, Georgian clay, and Florida's phosphate rock are likely to increase with demand in other sectors, such as construction. These and other mining sources can produce excessive sedimentation in streams as well as affect pH and metal concentrations. Expansion or increased production from regional mines is expected to have increased negative impacts to freshwater systems, estuaries, and bay systems in the foreseeable future.

Changes in the leisure and hospitality, transportation, and construction sectors are likely to have similar effects in the mid-Atlantic as were identified for the northeast. However, regional differences will likely lead to different local effects. Low-lying estuaries can collect oil and contaminant run-off from rapidly developing roads, leading to habitat degradation.

The mid-Atlantic region has significantly greater agriculture than in the northeast; a difference that will likely affect the health of streams, estuaries, and marine habitats. Extensive agriculture in the region requires the use of pesticides, fertilizers, and other chemicals in large scale that migrate into freshwater systems. The expansion of agriculture, regardless of crop, will likely

entail additional chemicals entering freshwater systems. This can have negative impacts on the survival and recovery of sturgeon populations in fresh water and bay systems, both by accumulation in fish tissues, and general degradation of habitat (i.e., Chesapeake Bay).

West Coast Projection

For the west coast, we selected California as a state representative in legislation. This is because of the large population, complex geography, diverse socio-economic and demographic structure, extent of waterway and coastline, and presence of several listed species of varied taxa. Trends in legislation address the impact and causal regulation of climate change, control of marine debris and harmful substances in waterways and marine areas, regulation of fisheries and invasive species, limitation of oil and gas development, clarification of state listed species takes, and aid for salmon recovery.

States along the Pacific coast, or which contribute water to major river systems here, are projected to have the most rapid growth of any area in the United States within the next few decades. This is particularly true for coastal states and those of the desert southwest. California, Oregon, Washington State, Arizona, Idaho, Utah, Nevada, and Alaska are forecasted to have double digit increases in population growth rates for each decade from 2000 to 2030 (USCB 2005b). New Mexico, Montana, and Wyoming will have far slower growth, with Wyoming forecasted to eventually experience population contraction. Overall, this region had a projected population of 65.6 million people in 2005 and will likely grow to 70.0 million in 2010 and 74.4 million in 2015, making it by far the most populous region (but also containing the greatest land area). As with other regions, growth stems from development of metropolitan areas. However, western growth will come generally from enlargement of smaller cities than from major metropolitan areas. Of the 42 metropolitan areas that experienced 10% growth or greater between 2000 and 2007, only seven have populations greater than one million people. These major cities include Las Vegas, NV (4.79%/year), Phoenix, AZ (4.07%/year), Riverside-San Bernadino-Ontario, CA (3.63%/year), Sacramento-Arden-Arcade-Roseville, CA (2.34%/year), Salt Lake City, UT (1.93%/year), Denver, CO (1.87%/year), and Portland-Vancouver-Beaverton, OR (1.83%/year). It should be noted that most of these metroplexes border coastal or riverine systems. Diffuse, but extensive, growth in the region will increase contaminants from wastewater treatment plants and sediments from sprawling urban and suburban development that enter riverine, estuarine, and marine habitats. This is of particular concern in western states, where numerous rivers and their tributaries are designated critical habitat for listed salmon. Increased contaminant loads have the potential to influence fry and smolt development in freshwater systems. Sediments may alter spawning grounds so as to make them unusable by salmon. Unlike other areas of the United States, the west coast region has extensive fluctuations in elevation and pooling oil and pollutants from developing roadways will likely not be as significant an issue in this region as elsewhere. Western states are widely known for scenic and natural beauty. Increasing resident and tourist use will place additional strain on maintaining the natural state of park and nature areas, also utilized by protected species.

Mining has historically been a major component of western state economies. With national output for metals increasing at 4.3% annually (little oil, but some gas is drawn from western states), output of western mines should increase markedly (Figueroa and Woods 2007). This will

increase already significant levels of mining contaminants entering river basins. This future increase is all the more problematic because many western streams feed into or provide spawning habitat for threatened and endangered salmonid populations. These fishes rely upon healthy streams for breeding and their offspring spend the first parts of their lives feeding in rivers, lakes, and streams that heavier contaminant burdens will be affecting. Sturgeon also live in these waterways and will similarly experience negative impacts from growth in the mining sector.

Western states boast large tracts of irrigated agriculture. The rise in agricultural output (Figueroa and Woods 2007) will likely result in two negative impacts upon protected species. With increased production, pesticide, fertilizer, and herbicide use will be used in greater amounts and enter freshwater systems in greater concentrations. Like mining, this has the potential to harm salmonids and sturgeon or their habitats. Further, increased output could place greater demands upon limited water resources. This will reduce flow rates and alter habitat throughout freshwater systems, and likely lead to increased water temperatures and decreases in DO. As water is drawn off, contaminants will become more concentrated in these systems, exacerbating contamination issues in habitats and protected species.

Summary of Cumulative Effects

At the large spatial scale of this consultation, we could not identify specific future state, tribal, local or private actions that were reasonably certain to occur in the action area. Instead we looked at demographic and economic trends to discern general patterns of land use change anticipated by states and federal organizations and their potential effects on listed species. Assuming recent increases in unemployment and poor performance of the dollar are fair indicators of rates potential land use change, regional growth is expected to continue on a slower pace than observed in the past decade. In January 2010, however, unemployment dropped a modest amount from 10 percent to 9.7 percent, which may signal a shift to a more promising economy. However, much uncertainty surrounds whether we will see near term measurable increases in the construction and industrial arenas. We suspect that spatial patterns of growth and development, and redevelopment would likely continue as it has in the past for the near future, but expect that the pace of new development and redevelopment will continue to remain at a slower pace than the past decade.

In general, we expect that the threatened and endangered aquatic species and designated critical habitats considered in this biological opinion are likely to be adversely affected by non-federal activities that affect the quantity, and quality of water, waterways, and habitats important to listed aquatic species and their critical habitat. Non-federal activities that change vegetative cover, soil structure, and water use ways that increase erosion and sedimentation, increase introduction of pollutants into waterways, and result in introductions and spread of non-native invasive species will likely continue to directly and indirectly affect listed species and critical habitats. These species and their critical habitats could also be affected by illegal harvest. At the same time, states or private entities may also engage in activities to restore, enhance, and improve water quality and quantity and restore more natural hydrographic patterns that benefit listed species and their habitats. All of the species and critical habitats considered in this document are likely to be exposed to these types of activities in the future to varying extents.

Integration and Synthesis

The U.S. Environmental Protection Agency proposes to approve state or tribal water quality standards, or federal water quality standards promulgated by EPA, that are identical to or more stringent than EPA's recommended 304(a) aquatic life criteria for cyanide. This approval would authorize states and tribes and EPA to establish source controls (e.g., permits, 401 certifications, waste load allocations, etc.), define and allocate control responsibilities (allocate loads under TMDLs), measure and enforce compliance with the CWA, and measure progress in meeting the goals of the CWA (whether a water body should be listed as impaired; see *Understanding the Water Quality Program* earlier in this Opinion for a summary of the activities that are influenced by or rely upon the water quality standards approved by EPA and implemented by states, tribes and EPA.

In the *Approach to the Assessment* section of this Opinion, NMFS explained that we would assess the effects of EPA's programmatic approval of state, tribal, and federal water quality standards that rely upon their nationally recommended 304(a) aquatic life criteria for cyanide at the CCC and the CMC, by asking:

> Is EPA's approval of state, tribal and federal water quality standards consistent with (or more stringent than) the 304(a) criteria for cyanide, likely to prevent the exposure of endangered species, threatened species, and designated critical habitat to aqueous cyanide concentrations that are toxic, given the approach EPA uses to approve a water quality standards?

If, after considering the best scientific and commercial data available, we conclude that listed resources are not likely to be exposed to activities the water quality standards would authorize, both individually and cumulatively, we stated we would conclude that EPA's proposal to continue recommending the 304(a) aquatic life criteria for cyanide is not likely to jeopardize the continued existence of endangered species, threatened species, or result in the destruction or adverse modification of designated critical habitat under NMFS' jurisdiction. When an agency's national action is likely to prevent exposure of listed resources to their activities, then we would expect an agency's program would generally ensure that actions taken under the program are not likely to individually, or cumulatively, jeopardize the continued existence of threatened and endangered species, and are not likely to result in the destruction or adverse modification of critical habitat that has been designated for those species.

If our assessment determined that listed resources are likely to be exposed to these activities, we stated we would examine whether and to what degree listed species are likely to respond to their exposure, given the approach EPA uses to approve a water quality standards. As part of this analysis, we stated we would examine whether and to what degree EPA has identified chemical, physical and biological scenarios that influence cyanide toxicity and presence in the environment inhabited by listed species and their critical habitat, the nature of any in situ effects, and the consequences of those effects for listed resources under NMFS' jurisdiction, to determine if EPA can insure that the approval of state, tribal and federal water quality standards that they are proposing is not likely to jeopardize the continued existence of endangered species or threatened species, or result in the destruction or adverse modification of critical habitat that has been

designated for these species. We stated that we measure risks to listed individuals using changes in the individual's "fitness" or the individual's growth, survival, annual reproductive success, and lifetime reproductive success. When we do not expect listed plans or animals exposed to an action's effects to experience reductions in fitness, we would not expect that action to have adverse consequences on the viability of the populations those individuals represent or the species those population comprise (Mills and Beatty 1979; Stearns 1992; Anderson 2000). As a result, if we conclude that listed plants or animals are not likely to experience reductions in their fitness we would conclude our assessment.

Based on the analysis contained in their BE and on the results of the preliminary screen as introduced by the *Methods Manual*, EPA was able to screen out (or make not likely to adversely affect) determinations on all but 32 species. The 32 species included: several darters, perch, salmonids, and one amphipod. Next, EPA applied a secondary screen that relied primarily on evaluating whether the waters where the 32 listed species occurred were listed as impaired pursuant to the CWA as well as data that would indicate the species had been (1) listed for reasons attributed to cyanide, (2) or whether there were known dischargers of cyanide within the range of the listed species. Using these metrics EPA concluded that of the 32 potentially sensitive species, none would be adversely affected by their action of approving state or tribal water quality standards or federal water quality standards that are equal to or more stringent than the nationally recommended section 304(a) aquatic life water quality criteria for cyanide.

Based on data available in STORET and TRI, as well as information about cyanide in general, the patterns of cyanide exposure are variable and probably not reflective of only permitted discharges. A number of non-permitted (non-point) sources likely also contribute to ambient cyanide concentrations in waters of the United States. Since state, tribal and federal water quality standards form the foundation for, not only permitting, but also evaluating the measuring the progress of the goals of the CWA, it is important to consider non-point sources of a contaminant in evaluating exposure scenarios. Our analysis also demonstrates that permitted discharges likely exceed criterion values from time to time, and can be as much as ten times higher than criterion values without being in violation of CWA. Because we lacked long term data sets for our analysis, we could not evaluate an upper exposure limit nor do we know what a typical exposure scenario would necessarily look like. Our analysis demonstrates that all listed species considered herein would likely be exposed to cyanide during the course of their typical life histories. However, because we could not determine the typical concentrations of exposure, our analysis is premised on the assumption that a suitable concentration for evaluating exposure and response are the proposed criteria values. We believe this is a reasonable threshold for evaluating the effects of cyanide at the national level, since it forms the foundation for a host of water quality management actions in waters of the United States and is the basis for EPA's proposed approval of state, tribal and federal water quality standards.

Our analysis demonstrates that EPA may identify chemical and biological scenarios that influence cyanide toxicity and presence in the environment, but that such information often has little influence (or at least no obvious influence) on the concentration of cyanide that EPA recommends to states and tribes as a "safe dose" for water quality standards. Since the information relegated to "other data" is not considered at the national level in publishing a 304(a) recommendation, then we looked for information to suggest that states would use the information

to modify their water quality standards to incorporate site or situation specific modifications as appropriate. That is, we found no evidence that states adopted cyanide water quality standards that were modified by expected water temperatures, unless it was to increase the accepted concentration of the cyanide standard. For instance, since the cyanide standard is driven by rainbow trout data, states with warm water basins often increased the threshold of their water quality standard. In contrast, states where cold water species (e.g., steelhead and salmon) reside did not have modified standards for winter (very cold) water situations that account for the increased toxicity of cyanide at cold temperatures.

In general for cyanide, EPA's decision to recommend and approve water quality standards for cyanide was based on a paucity of data in general, and in particular for listed species. The paucity of data was particularly apparent for saltwater species. However, data was also extremely poor for characterizing a few good case studies on cyanide or what might be considered typical cyanide exposures. Based on our limited review of a few general permits, which incidentally, happen to be one of the most routinely issued permit types issued by EPA and states, generally too few samples are required to result in meaningful monitoring data by which to manage cyanide discharges, or to evaluate the frequency and severity of cyanide entering most basins.

EPA's strict interpretation of what they deemed adequate data for the purposes of decision-making under the CWA is also particularly disconcerting. While both EPA and NMFS are required to use the best available data in their decision-making, when there is data on the listed taxa despite whether there are numerous studies that confirm the findings, NMFS would generally consider that data and the strength of the data in its decision. For instance, EPA often narrowly constrains their decision on a criterion to "avoid confounding factors". However, what might be considered a "confounding factor" in a laboratory setting is often a realistic mixture of conditions in the wild and is relevant for the purposes of evaluating whether a particular action or set of actions is not likely to jeopardize the continued existence of listed resources. For instance, the interplay between DO and cyanide or cyanide and temperature received little attention in EPA's 304(a) aquatic life criteria, despite that there is a wide problem of low DO in many watersheds inhabited by anadromous fish species both on the west coast and the east coast, and salmonids generally inhabit very cold waters during winter months. At least with cyanide, EPA's decision-making process is based on limited very controlled test situations that may be poor predictors of real exposure scenarios and at a minimum, would be strengthened by some field experiments or at least mesocosm studies that are more representative of typical aquatic communities.

Based on our analysis, it also appears that guidance to states and tribes may be prudent for recognizing the potential impacts of cyanide, and the ability of the various forms of cyanide to interact and change within a system. Although we did not search for specific examples of guidance, sources of cyanide within a watershed are numerous and are not limited to expected dischargers and certainly are not limited to the mining industry, which is often the misconception. Based on a review of wastewater treatment facilities, Kavanaugh et al. (2003) caution that managers need to acknowledge that multiple forms of cyanide typically coexist, introconvert, and degrade in a waterbody. It is for this reason, that Kavanaugh et al. (2003) recommended that water quality standards ought to reflect the ability of cyanide compounds to

undergo transformation, increasing or decreasing in impact; in so doing, EPA could establish water quality standards for certain classes of cyanide that would be measured using appropriate analytical methods. Kavanaugh et al. (2003) also recommend that the water quality criteria and discharge standards for cyanide be revised to ensure that monitoring methods can distinguish between cyanide forms, and that methods with the greatest potential for use should receive EPA and state approval.

Nevertheless, based upon our analysis we concur with EPA's effect determination that a number species are not likely to be adversely affected when exposed to cyanide at criterion values. Our determination, however, is based on uncertain evidence because for the most part suitable data upon which to make this determination is weak at best. As noted earlier, Gensemer et al. (2007) declined to evaluate the effects of several marine species, acknowledging that the data is too poor to evaluate the protectiveness of the saltwater cyanide criteria on marine species. We concur with Gensemer et al. (2007) that "this represents an area requiring further research" since only three fish genera and five invertebrate genera were used to establish the saltwater criteria. That said, based on the available data as discussed in the preceding analysis, we would not expect the following threatened or endangered species to respond physically, physiologically, or behaviorally to exposure at the CMC or the CCC, whether exposed in saltwater or fresh water or both: Blue whales, bowhead whales, fin whales, humpback whales, North Atlantic right whales, North Pacific right whales, sei whales, sperm whales, beluga whales, southern resident killer whales, Guadalupe fur seals, Hawaiian monk seals, Western Steller sea lions, Eastern Steller sea lions, Florida green sea turtles, Mexico green sea turtles, hawksbill sea turtles, Kemp's ridley sea turtles, loggerhead sea turtles, leatherback sea turtles, Mexico's breeding colonies of olive ridley sea turtles, other olive ridley sea turtles, smalltooth sawfish, elkhorn coral, staghorn coral, white abalone, black abalone and Johnson's seagrass.

Species under NMFS' jurisdiction that demonstrate sensitivity to cyanide at criterion values are: chum salmon, coho salmon, sockeye salmon, Chinook salmon, steelhead, shortnose sturgeon, and green sturgeon, representing 30 DPS/ESUs of these species. Of these species, empirical and modeled evidence suggests that some salmon may die when exposed to cyanide at the CMC of 22 µg/L. According to modeled estimates chum, coho, Chinook, and sockeye salmon, are all more sensitive to cyanide than steelhead, suggesting that some individuals may die when exposed to cyanide at the CMC. However, lethal effects on steelhead salmon are predicated on an exposure to cyanide at low temperatures. That is, the risk of death increases at lower temperatures, while exposure to cyanide in waters at about the average test temperature of 12-13 ºC would probably not lead to the death of steelhead.

While, the relationship between temperature and cyanide may merit further examination to increase confidence in the relationship, existing information suggests that coldwater species may be more sensitive to cyanide at temperatures that are typical of winter months. We have no evidence that the interplay between cyanide and temperature is species specific. If temperature influences the sensitivity of other salmonids, then that would increase the risk of death for not only steelhead, but also coho, sockeye, chum, and Chinook salmon. Our best estimate of effect for steelhead is that roughly 1% of steelhead exposed to cyanide in winter months may die from their exposure, since coho, Chinook, sockeye and chum salmon are all more sensitive to cyanide than steelhead, the percent lethal effect would also increase. We do not know which ages or

stages of salmon are most likely to be affected at low temperatures.

Based on our review of chronic studies, we estimate that female sturgeon and Pacific salmon may experience a 40-60% reduction in the number of eggs spawned, and these species would experience a 40 to 70 % reduction in early life stage survival. This should only be considered a rough estimate of the magnitude of the true effect expected at the CCC of 5.2 µg CN/L. Other sublethal responses to low levels of cyanide include reduced swimming performance and reduced weight gain.

In the *Status of the Species* section of this Opinion, we established that Chinook, coho, sockeye, and chum salmon, steelhead, and green and shortnose sturgeon species have declined throughout their range. Some ESUs have demonstrated modest increases in recent years, like Lower Columbia River Chinook salmon and Hood Canal chum salmon, and others like Sacramento winter-run Chinook salmon, Puget Sound steelhead, and Lower Columbia River coho salmon continue to decline. For some ESUs like California coastal Chinook salmon and Central California coast coho salmon, current trends are unknown.

In the *Environmental Baseline* section of this Opinion, we established that salmon and sturgeon are exposed to a myriad of habitat alterations attributable to urban and agricultural development, as well as fishing pressure. Land-use patterns have a profound impact on the contribution of chemicals to the waterways where salmon, steelhead, and sturgeon migrate, rear, spawn, feed and grow. In many basins, these fish are exposed to persistent "legacy" chemicals, as well as there is a relatively constant influx of common-use chemicals like copper and PAHs. At the same time, migratory barriers continue to impact population movement and expansion, loss of riparian forest has lead to increased water temperatures in some areas and the loss of allochthonous input, reduced stream bank complexity, loss of spawning gravels, and altered flow regimes, to name a few. Salmon and sturgeon are also commonly impacted by low DO in many areas throughout their ranges. In the *Cumulative Effects* section of this Opinion, we established that salmon and sturgeon are likely to be exposed to the combined effects of similar habitat modifications for the next ten years, and given expected human population increases and economic development in many regions these impacts will likely increase. The combined effect of these habitat alterations means that chemical loading in many watersheds and coastal areas will likely continue to increase, despite pollution control efforts. Non-point sources for pollutant loading will likely continue to be a significant portion of the problem.

Killing 30-45% of the viable eggs spawned per salmon and sturgeon and killing 56-79% of their larvae is certain to reduce the likelihood of survival and the reproductive success of coho salmon, Chinook salmon, chum salmon, sockeye salmon, green sturgeon, and shortnose sturgeon populations. Reducing the swimming performance of these species would likely reduce their fitness and possibly their survival, through reductions in prey capture, weight gain, displacement, predator escapement, and possibly lead to death. Although there is uncertainty in this analysis, which incidentally is not limited to these calculations, based on the evidence available, we do not believe EPA's decision-making process mitigates or minimizes these potential losses. Worse yet, EPA and the states are not in a position to detect these losses if or when they occur.

If the intent of the 304(a) aquatic life criteria is to define a level in the waterbody of a pollutant

that will be fully protective of the designated uses of a water body and that a state or tribe identify as part of their water quality standards (see BE page 11, and also 40 CFR 131.2), then it would follow that EPA would have to review whether their recommended criteria can protect the specific uses that states and tribes have identified in their designated uses. Instead, our analysis suggests that Gaba (1983) was correct when he noted that EPA and the states are engaged in a water quality process "merely to justify the specific numbers contained in pollutant criteria." That uses are designated without meaningful linkages between the chemical criteria indicators and the biological condition of the waters they are meant to protect, means neither EPA or states or tribes can know how well the chemical criteria are protecting the aquatic assemblages or biological community diversity they are meant to protect. That is, available evidence suggests that EPA (nor states or tribes) is not likely to monitor (a) the direct, indirect, and cumulative impacts of the activities their approvals would authorize on biological community diversity, (b) the nature of those effects on the aquatic assemblages in which they occur, or (c) the consequences of those effects on listed resources. Given the lack of measured endpoints for biological condition, EPA will not know if the aquatic assemblages or species identified as designated are actually protected by the water quality standards, much less whether those water quality standards protect endangered species, threatened species or designated critical habitat under NMFS' jurisdiction.

Based on our review, it is not even clear that EPA would consider listed species as part of the biological community to which Congress directed them to consider in establishing 304(a) aquatic life criteria. EPA's decision-making process (the *Guidelines*) places special emphasis on commercially, recreationally, and other important species, and aquatic assemblages. If, as EPA stated, their only metrics for evaluating the protection of the aquatic assemblage are species richness and species evenness (see EPA 2008a), then EPA could argue (albeit a poor argument) that they are protecting aquatic assemblages if their recommended aquatic life criteria and approved state water quality standards protect non-native aquatic assemblages. Yet, listed species, arguably, are "important" as Congress saw fit to provide for their protection under the ESA and ensure federal agencies have a prominent role in providing for their protection. Moreover, many of NMFS' listed species are also commercially and recreationally valued, and many of the species discussed herein are part of the same aquatic assemblage. Given, EPA's lack of clarity on what constitutes an "important" species, and the indicators they stated they use to evaluate an aquatic assemblage (species richness and species evenness) EPA has placed themselves in a position to exclude the needs of native species in general, and listed species in particular, as part of the biological communities they intend to protect.

All of the endangered species, threatened species, and designated critical habitat under NMFS' jurisdiction depend upon the health of the aquatic ecosystems they occupy for their survival and recovery. EPA's 304(a) aquatic life criteria are designed to reflect the latest scientific knowledge including on the kind and extent of all identified effects on …. fish, shellfish, wildlife, and plants…which may be expected from the presence of pollutants in any body of water…; the concentration and dispersal of pollutants or their byproducts, through biological, physical and chemicals processes; and on the effects of pollutants on biological community diversity, productivity, and stability….. (CWA section 304(a)(1)). As such, 304(a) aquatic life criteria have a prominent role in the success of the overall water quality program designed "to restore and maintain the chemical, physical and biological integrity of the Nation's waters."

Nevertheless, degraded water quality has been one of the contributing factors for the decline of almost all of the anadromous fish species NMFS has listed since the mid-1980s. While cyanide has not been identified as a specific concern in any listing, poor water quality has generally been identified as cause contributing to their need for listing. Generally, it has not been the case that NMFS has isolated poor water quality to only one chemical, physical, or biological stressor for the species that have been listed. To use this lack of evidence, as evidence that an effect is lacking is simply not a persuasive argument that cyanide is not problem for listed species.

Based on our analysis we believe it is reasonable to expect that the number of cyanide sources is likely to increase commensurate with land use changes and expansion of industrial and extraction activities. Our analysis illustrates that the exposure of listed salmon and sturgeon species to cyanide at the proposed chronic criterion concentration is likely to substantially reduce their reproduction by reducing the number of eggs spawned by females, reducing the hatchability of spawned eggs, and by reducing the survivorship of young fish in their first year. These fish may also experience effects on growth, swimming performance, condition, and development. Based upon the magnitude of adverse effects caused by the exposure of these listed species to cyanide at the proposed criteria concentrations, these fish species are likely to become extirpated from waters where they are exposed to approved cyanide discharges that are compliant with approved water quality standards. Continued approval of the EPA's aquatic life criteria for cyanide at the range wide scale of these listed species is likely to reduce their reproduction, numbers, and distribution. Unfortunately, it appears that not only does EPA fail to consider biologically, chemically, and physically relevant exposure scenarios that influence cyanide toxicity, EPA is not and has not put themselves in a position of knowing whether their 304(a) aquatic life recommendations and subsequent approvals of state and tribal water quality standards are in fact, protecting the biological community diversity, productivity and stability they intend to protect. Therefore, we do not believe the EPA can insure that the approval of water quality standards for cyanide are not likely to jeopardize the continued existence of endangered species or threatened species or result in the destruction or adverse modification of critical habitat that has been designated for these species.

Because the proposed action, based on our analysis, is likely to reduce the viability of one or more populations throughout the range of listed Pacific salmon, steelhead, and sturgeon species, we expect that the action is likely to reduce the viability (that is, increase the extinction probability or appreciably reduce their likelihood of both surviving and recovering in the wild) of the listed species as a whole. The specific listed species at risk are: California coastal Chinook salmon, Central Valley spring-run Chinook salmon, Lower Columbia River Chinook salmon, Upper Columbia River spring-run Chinook salmon, Puget Sound Chinook salmon, Sacramento River winter-run Chinook salmon, Snake River fall-run Chinook salmon, Snake River spring/summer-run Chinook salmon, Upper Willamette River Chinook salmon, Columbia River chum salmon, Hood Canal summer-run chum salmon, Central California Coast coho salmon, Lower Columbia River coho salmon, Southern Oregon and Northern California Coast coho salmon, Oregon Coast coho salmon, southern green sturgeon, shortnose sturgeon, Lake Ozette sockeye salmon, Snake River sockeye salmon, Central California Coast steelhead, California Central Valley steelhead, Lower Columbia River steelhead, Middle Columbia River steelhead, Northern California steelhead, Puget Sound steelhead, Snake River steelhead, South-Central California Coast steelhead, Southern California coast steelhead, Upper Columbia river steelhead,

and Upper Willamette River steelhead.

Finally, a reduction in Puget Sound Chinook salmon would in turn significantly reduce the forage base of southern-resident killer whales. Therefore, while we agree that southern resident killer whales are not likely to respond physically, physiological, or behaviorally to their direct exposure to cyanide at the CCC or the CMC, we expect that the action, through indirect effects to their primary prey, Pacific salmon, is likely to appreciably reduce the likelihood of southern-resident killer whales surviving and recovering in the wild. Similarly, a reduction in Chinook, coho, sockeye, and chum salmon would in turn significantly reduce the forage base of Cook Inlet beluga whales. We also agree with EPA that Cook Inlet beluga whales are not likely to respond physically, physiological, or behaviorally to their direct exposure to cyanide at the CCC or the CMC, we expect that the action, through indirect effects to their primary prey, Pacific salmon, is likely to appreciably reduce the likelihood of Cook Inlet beluga whales surviving and recovering in the wild.

The proposed action is likely to reduce the habitat qualities for these species that are essential to their conservation. Specifically, reduced availability of clean quality water for the purpose of reproduction, rearing and growth, and a reduction in prey species of sufficient quantity and quality would affect the conservation value of designated critical habitat for these species. The functional value of critical habitat exposed to cyanide at criterion values would be severally reduced and could not serve the intended conservation role for the species. Based on our analysis, the functional value of critical habitat would be reduced throughout the areas designated as critical habitat for: southern resident killer whale, California coastal Chinook salmon, Central Valley spring-run Chinook salmon, Lower Columbia River Chinook salmon, Upper Columbia River spring-run Chinook salmon, Puget Sound Chinook salmon, Sacramento River winter-run Chinook salmon, Snake River fall-run Chinook salmon, Snake River spring/summer-run Chinook salmon, Upper Willamette River Chinook salmon, Columbia River chum salmon, Hood Canal summer-run chum salmon, Central California Coast coho salmon, Lower Columbia River coho salmon, Southern Oregon and Northern California Coast coho salmon, Oregon Coast coho salmon, southern green sturgeon, Lake Ozette sockeye salmon, Snake River sockeye salmon, Central California Coast steelhead, California Central Valley steelhead, Lower Columbia River steelhead, Middle Columbia River steelhead, Northern California steelhead, Snake River steelhead, South-Central California Coast steelhead, Southern California coast steelhead, Upper Columbia river steelhead, and Upper Willamette River steelhead. Similarly, the proposed action would significantly reduce the functional value of proposed critical habitat for Cook Inlet beluga whales when their salmon prey species are exposed to cyanide at criterion values. The result of the exposure of salmon species outside of the geographic area designated as critical habitat would severally reduce the numbers of salmon available to beluga within proposed critical habitat and therefore, the critical habitat could not serve the intended conservation role for the species.

Conclusion

Listed Species and Critical Habitat

After reviewing the current status of the listed species, the environmental baseline for the action area, the effects of the EPA's continuing approval of state water quality standards that rely on their nationally recommended criteria for cyanide and the cumulative effects, it is NMFS' biological opinion that EPA's approval of state water quality standards for cyanide is likely to jeopardize the continued existence of the following species:

> California coastal Chinook salmon, Central Valley spring-run Chinook salmon, Lower Columbia River Chinook salmon, Upper Columbia River spring-run Chinook salmon, Puget Sound Chinook salmon, Sacramento River winter-run Chinook salmon, Snake River fall-run Chinook salmon, Snake River spring/summer-run Chinook salmon, Upper Willamette River Chinook salmon, Columbia River chum salmon, Hood Canal summer-run chum salmon, Central California Coast coho salmon, Lower Columbia River coho salmon, Southern Oregon and Northern California Coast coho salmon, Oregon Coast coho salmon, southern green sturgeon, shortnose sturgeon, Lake Ozette sockeye salmon, Snake River sockeye salmon, Central California Coast steelhead, California Central Valley steelhead, Lower Columbia River steelhead, Middle Columbia River steelhead, Northern California steelhead, Puget Sound steelhead, Snake River steelhead, South-Central California Coast steelhead, Southern California coast steelhead, Upper Columbia river steelhead, Upper Willamette River steelhead, southern resident killer whales, and beluga whales.

After reviewing the current status of the listed species, the environmental baseline for the action area, the effects of the EPA's continuing approval of state water quality standards that rely on their nationally recommended criteria for cyanide and the cumulative effects, it is NMFS' biological opinion that EPA's approval of state water quality standards for cyanide is likely to destroy or adversely modify designated critical habitat for the following species:

> Southern resident killer whale, California coastal Chinook salmon, Central Valley spring-run Chinook salmon, Lower Columbia River Chinook salmon, Upper Columbia River spring-run Chinook salmon, Puget Sound Chinook salmon, Sacramento River winter-run Chinook salmon, Snake River fall-run Chinook salmon, Snake River spring/summer-run Chinook salmon, Upper Willamette River Chinook salmon, Columbia River chum salmon, Hood Canal summer-run chum salmon, Central California Coast coho salmon, Southern Oregon and Northern California Coast coho salmon, Oregon Coast coho salmon, southern green sturgeon, Lake Ozette sockeye salmon, Snake River sockeye salmon, Central California Coast steelhead, California Central Valley steelhead, Lower Columbia River steelhead, Middle Columbia River steelhead, Northern California steelhead, Snake River steelhead, South-Central California Coast steelhead, Southern California coast steelhead, Upper Columbia river steelhead, and Upper Willamette River steelhead.

For species that have no designated critical habitat, then none can be affected.

Species and Critical Habitat Proposed for Listing

After reviewing the current status of bocaccio, canary rockfish, spotted seal, and yelloweye rockfish, the environmental baseline for the action area, the effects of the EPA's continuing approval of state water quality standards that rely on their nationally recommended criteria for cyanide and the cumulative effects, it is NMFS' conference opinion that EPA's approval of state water quality standards for cyanide is not likely to jeopardize the continued existence of bocaccio, canary rockfish, spotted seal, and yelloweye rockfish. NMFS' conclusion for these proposed species is based on the limited data available on marine species. Based on the foregoing analysis, NMFS expects that the approval of cyanide water quality standards is likely to destroy or adversely modify the proposed critical habitat for beluga whales because salmon are an important prey species for beluga whales and are identified as a PCE. NMFS' conclusion for the area designated as proposed critical habitat for Cook Inlet beluga whales is based on the proposed action's effects on salmonids.

Reasonable and Prudent Alternatives

This Opinion has concluded that EPA's approval of state or tribal water quality standards, or federal water quality standards promulgated by EPA for aquatic life criteria that are identical the section 304(a) aquatic life criteria for cyanide, is likely to jeopardize the continued existence of 31 species under NMFS' jurisdiction, and result in the destruction or adverse modification of critical habitat that has been designated for these species. The clause "jeopardize the continued existence of" means "to engage in an action that reasonably would be expected, directly or indirectly, to reduce appreciably the likelihood of both the survival and recovery of listed species in the wild by reducing the reproduction, numbers or distribution of that species (50 CFR §402.02).

Regulations implementing Section 7 of the Act (50 CFR 402.02) define reasonable and prudent alternatives as alternative actions, identified during formal consultation, that: (1) can be implemented in a manner consistent with the intended purpose of the action; (2) can be implemented consistent with the scope of the action agency's legal authority and jurisdiction; (3) are economically and technologically feasible; and (4) would, NMFS believes, avoid the likelihood of jeopardizing the continued existence of listed species or resulting in the destruction or adverse modification of critical habitat.

NMFS reached this conclusion because the evidence available suggests that EPA does not (a) use biological, chemical, or physically relevant information of the natural conditions to which aquatic species would be exposed to derive their numeric recommendations for 304(a) aquatic life criteria or to approve state and tribal water quality standards that rely on their recommended criteria, (b) that EPA is not in a position to know whether the water quality standards they approve actually protect native biological communities, or (c) the listed species that are part of the native biological community. Given the decision structure employed by EPA, EPA will not know whether designated uses are protected, much less whether the direct, indirect, or cumulative impacts of their approval of state and tribal water quality standards that rely on their

304(a) aquatic life criteria recommendations protect endangered species, threatened species, or designated critical under NMFS' jurisdiction.

To satisfy its obligation pursuant to section 7(a)(2) of the ESA of 1973, as amended, EPA must put itself in a position to (a) use biological, chemical, or physically relevant information of the natural conditions to which aquatic species would be exposed to derive their numeric recommendations for 304(a) aquatic life criteria or to approve state and tribal water quality standards that rely on their recommended criteria, (b) monitor whether the water quality standards they approve actually protect native biological communities, and (c) the listed species that are part of the native biological community. What follows is a single reasonable and prudent alternative, consisting of several sub-elements that must be implemented in its entirety to insure that the activities EPA's approval of state and tribal water quality standards would authorize are not likely to jeopardize endangered or threatened species under the jurisdiction of the NMFS or destroy or adversely modify critical habitat that has been designated for these species.

The U.S. Environmental Protection Agency must, by December 1, 2012:

A). Revise the *Guidelines* and any relevant regulatory guidance to:

1. Address how they will incorporate relevant information on biological, chemical, or physical processes that alter a particular chemical's toxicity in nature, in their recommendations such that states and tribes that adopt 304(a) aquatic life criteria as recommended will be required to account for relevant exposure scenarios that affect chemical toxicity, in their state water quality standards.

2. Explicitly address (a) endangered species, threatened species, and designated critical habitat as part of the "important" species the aquatic life criteria are designed to protect, and (b) the native biological community, of which listed species are a part, as the relevant community endpoint to which they intend to protect.

B). Develop and implement the research necessary to replace modeled estimates of species sensitivities to cyanide with direct evidence, using listed species or more closely related surrogates, as the basis for defining cyanide criteria to insure an appropriate level of protection is afforded to listed species and critical habitats addressed by this RPA.

Because this biological opinion has concluded that the U.S. Environmental Protection Agency's proposed approval of state water quality standards that rely on their 304(a) aquatic life criteria is likely to jeopardize the continued existence of endangered and threatened species under the jurisdiction of NMFS, and is likely to result in the destruction or adverse modification of critical habitat, the Environmental Protection Agency is required to notify NMFS of its final decision on the implementation of the reasonable and prudent alternatives.

Incidental Take Statement

Section 9 of the ESA and Federal regulation pursuant to section 4(d) of the ESA prohibits the take of endangered and threatened species, respectively, without special exemption. Take is defined as to harass, harm, pursue, hunt, shoot, wound, kill, trap, capture or collect, or to attempt to engage in any such conduct. Harm is further defined by NMFS to include significant habitat modification or degradation that results in death or injury to listed species by significantly impairing essential behavioral patterns, including breeding, feeding, or sheltering. Incidental take is defined as take that is incidental to, and not the purpose of, the carrying out of an otherwise lawful activity. Under the terms of section 7(b)(4) and section 7(o)(2), taking that is incidental to and not intended as part of the agency action is not considered to be prohibited taking under the Act provided that such taking is in compliance with the terms and conditions of this Incidental Take Statement.

Amount or Extent of Take

As described earlier in this Opinion, this NMFS' review of EPA's national approval of state and tribal water quality standards that are consistent with or more stringent than the nationally recommended 304(a) criteria for cyanide. The goal of this national level Opinion is to evaluate the general impacts to NMFS' listed resources from the national approval of the 304(a) cyanide criteria when adopted by states and tribes for implementation as part of their water quality standards. It is not possible to identify take that would occur from specific permitted actions or the specific exposure scenarios typical in a particular state. Instead, this Opinion anticipates the general effects that would occur from the approval of cyanide water quality standards across the landscape. Therefore, this Opinion does not exempt incidental take of listed fish from the prohibitions of section 9 of the ESA for the EPA's approval of cyanide water quality standards.

NMFS anticipates that with implementation of the RPA, incidental take of the listed species considered in this biological opinion is not likely to occur from exposure to cyanide at revised criteria concentrations. However, other elements of water quality standards could allow for exceedance of criteria concentrations and may result in incidental take. The other elements of water quality standards will be the focus of subsequent tiered consultations on individual state and tribal water quality standards. In each of these instances, EPA must conduct a separate, tiered consultation, and if necessary NMFS would issue a separate biological opinion before any endangered or threatened species might be "taken"; the amount or extent of "take" would be identified in those subsequent consultation on site-specific, state or tribal specific, or permit specific activities. Therefore, no incidental take exemptions are provided in this programmatic biological opinion.

Conservation Recommendations

Section 7(a)(1) of the Act directs Federal agencies to utilize their authorities to further the purposes of the Act by carrying out conservation programs for the benefit of endangered and threatened species. Conservation recommendations are discretionary agency activities to minimize or avoid adverse effects of a proposed action on listed species or critical habitat, to help implement recovery plans, or to develop information.

The following conservation recommendations would provide information for future consultation involving EPA's approval of state water quality standards:

1. The EPA should work with states to develop more meaningful linkages between designated uses and the water quality standards they intend to protect, to create monitoring programs that are capable of actually evaluating whether designated uses are being protected by approved water quality standards.

In order to keep NMFS' Endangered Species Division informed of actions minimizing or avoiding adverse effects or benefiting listed species or their habitats, the United States Environmental Protection Agency should notify the Endangered Species Division of any conservation recommendations they implement in their final action.

Reinitiation Notice

This concludes formal consultation on the United States Environmental Protection Agency's approval of water quality standards that are identical to or are more stringent than the section 304(a) cyanide aquatic life criteria. As provided in 50 CFR 402.16, reinitiation of formal consultation is required where discretionary Federal agency involvement or control over the action has been retained (or is authorized by law) and if: (1) the amount or extent of incidental take is exceeded; (2) new information reveals effects of the agency action that may affect listed species or critical habitat in a manner or to an extent not considered in this opinion; (3) the agency action is subsequently modified in a manner that causes an effect to the listed species or critical habitat not considered in this opinion; or (4) a new species is listed or critical habitat designated that may be affected by the action. In instances where the amount or extent of authorized take is exceeded, the United States Environmental Protection Agency must immediately request reinitiation of section 7 consultation.

UNITED STATES DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
NATIONAL MARINE FISHERIES SERVICE
Northwest Region
7600 Sand Point Way N.E., Bldg. 1
Seattle, WA 98115

Refr to NMFS No.:
2008/00148

August 14, 2012

Dennis McLerran
Regional Administrator
U.S. Environmental Protection Agency, Region 10
1200 Sixth Avenue, Suite 900
Seattle, Washington 98101-3140

Re: Jeopardy and Adverse Modification of Critical Habitat Biological Opinion for the Environmental Protection Agency's Proposed Approval of Certain Oregon Administrative Rules Related to Revised Water Quality Criteria for Toxic Pollutants

Dear Mr. McLerran: Dennis

Enclosed is a biological opinion (opinion) prepared by the National Marine Fisheries Service (NMFS) pursuant to section 7(a)(2) of the Endangered Species Act (ESA) on the Environmental Protection Agency's proposed approval of certain Oregon administrative rules related to revised water quality criteria for toxic pollutants.

In this opinion, NMFS concludes that the proposed action is likely to jeopardize the continued existence of LCR Chinook salmon (*Oncorhynchus tshawytscha*), UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon (*O. keta*), LCR coho salmon (*O. kisutch*), SONCC coho salmon, OC coho salmon, SR sockeye salmon (*O. nerka*), LCR steelhead (*O. mykiss*), UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon (*Acipenser medirostris*), eulachon (*Thaleichthys pacificus*), and Southern Resident killer whales (*Orcinus orca*).

NMFS also concludes that the proposed action will result in the destruction or adverse modification of designated critical habitats for LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, SONCC coho salmon, and OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, and eulachon.

NMFS concludes that the proposed action is not likely to adversely affect the following species: Steller sea lion (*Eumetopias jubatus*), blue whale (*Balaenoptera musculus*), fin whale, (*Balaenoptera physalus*), Sei whale (*Balaenoptera borealis*), sperm whale (*Physeter macrocephalus*), humpback whale (*Megaptera novaeangliae*), North Pacific right whale (*Eubalaena glacialis*), loggerhead turtle (*Caretta caretta*), green sea turtle (*Chelonia mydas*), leatherback turtle, (*Dermochelys coriacea*), and Olive Ridley turtle (*Lepidochelys olivacea*); or designated critical habitats for Steller sea lion, North Pacific right whale, green sea turtle, or leatherback turtle.

Section 7(b)(3)(A) of the ESA requires that, if jeopardy or destruction or adverse modification of critical habitat is found, NMFS must provide a Reasonable and Prudent Alternative (RPA), which is an alternative action that the Federal agency could take which would not violate section 7(a)(2). NMFS has developed an RPA, which, if implemented, will change the action such that NMFS would conclude no jeopardy or destruction or adverse modification of critical habitat.

This opinion assesses effects to listed species that occur in the State of Oregon pursuant to the ESA. It does not address EPA's obligation under the Magnuson-Stevens Fishery Conservation and Management Act to consult on effects to essential fish habitat (EFH) for Federally-managed species. Please contact the Oregon State Habitat Office regarding the EFH consultation process.

If you have questions regarding this consultation, please contact Robert Anderson, Fishery Biologist, NMFS Northwest Region, at 503.231.2226.

Sincerely,

William W. Stelle, Jr.
Regional Administrator

cc: Paul Henson, USFWS

Jeopardy and Destruction or Adverse Modification of Critical Habitat Endangered Species Act Biological Opinion for

Environmental Protection Agency's Proposed Approval of Certain Oregon Administrative Rules Related to Revised Water Quality Criteria for Toxic Pollutants

NMFS Consultation Number: 2008/00148

Federal Action Agency: U.S. Environmental Protection Agency

Affected Species and Determinations:

ESA-Listed Species	Status	Is Action Likely to Adversely Affect Species or Critical Habitat?	Is Action Likely to Jeopardize Species?	Is Action Likely to Destroy or Adversely Modify Critical Habitat?
Lower Columbia River Chinook salmon (*Oncorhynchus tshawytscha*)	Threatened	Yes	Yes	Yes
Upper Willamette River Chinook salmon (*Oncorhynchus tshawytscha*)	Threatened	Yes	Yes	Yes
Upper Columbia River spring-run Chinook salmon (*Oncorhynchus tshawytscha*)	Endangered	Yes	Yes	Yes
Snake River spring/summer run Chinook salmon (*Oncorhynchus tshawytscha*)	Threatened	Yes	Yes	Yes
Snake River fall-run Chinook salmon (*Oncorhynchus tshawytscha*)	Threatened	Yes	Yes	Yes
Columbia River chum salmon (*Oncorhynchus keta*)	Threatened	Yes	Yes	Yes
Lower Columbia River coho salmon (*Oncorhynchus kisutch*)	Threatened	Yes	Yes	Yes
Southern Oregon/Northern California Coasts coho salmon (*Oncorhynchus kisutch*)	Threatened	Yes	Yes	Yes
Oregon Coast coho salmon (*Oncorhynchus kisutch*)	Threatened	Yes	Yes	Yes
Snake River sockeye salmon (*Oncorhynchus nerka*)	Endangered	Yes	Yes	Yes
Lower Columbia River steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes
Upper Willamette River steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes
Middle Columbia River steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes

ESA-Listed Species	Status	Is Action Likely to Adversely Affect Species or Critical Habitat?	Is Action Likely to Jeopardize Species?	Is Action Likely to Destroy or Adversely Modify Critical Habitat?
Upper Columbia River steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes
Snake River Basin steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes
Green sturgeon Southern DPS (*Acipenser medirostris*)	Threatened	Yes	Yes	Yes
Eulachon (*Thaleichthys pacificus*)	Threatened	Yes	Yes	Yes
Southern Resident killer whale (*Orcinus orca*)	Endangered	No	Yes	No
Steller sea lion (*Eumetopias jubatus*)	Threatened	No	No	No
Blue whale (*Balaenoptera musculus*)	Endangered	No	No	N/A
Fin whale (*Balaenoptera physalus*)	Endangered	No	No	N/A
Sei whale (*Balaenoptera borealis*)	Endangered	No	No	N/A
Sperm whale (*Physeter macrocephalus*)	Endangered	No	No	N/A
Humpback whale (*Megaptera novaeangliae*)	Endangered	No	No	N/A
North Pacific Right whale (*Eubalaena glacialis*)	Endangered	No	No	No
Loggerhead turtle (*Caretta caretta*)	Threatened	No	No	N/A
Green sea turtle (*Chelonia mydas*)	Threatened	No	No	No
Leatherback turtle (*Dermochelys coriacea*)	Endangered	No	No	No
Olive Ridley turtle (*Lepidochelys olivacea*)	Threatened	No	No	N/A

Consultation Conducted By: National Marine Fisheries Service, Northwest Region

Issued by:

William W. Stelle, Jr.
Regional Administrator

Date: August 14, 2012

TABLE OF CONTENTS

1. INTRODUCTION

This Introduction section provides information relevant to the other sections of this document and is incorporated by reference.

1.1 Background

The biological opinion (opinion) and incidental take statement portions of this document were prepared by the National Marine Fisheries Service (NMFS) in accordance with section 7(b) of the Endangered Species Act (ESA) of 1973, as amended (16 U.S.C. 1531, *et seq.*), and implementing regulations at 50 CFR 402.

The opinion is in compliance with section 515 of the Treasury and General Government Appropriations Act of 2001 (Public Law 106-5444) ("Data Quality Act") and underwent pre-dissemination review.

1.2 Consultation History

On June 9, 2004, and September 15, 2004, NMFS, the U.S. Fish and Wildlife Service (FWS), and the U.S. Environmental Protection Agency (EPA) met to develop a work plan for the consultation on EPA's proposed approval of the 2004 Oregon revisions to state water quality standards for toxic pollutants.

Between September 2005 and February 2007, NMFS, EPA, and FWS participated in a series of technical and policy workgroup meetings, conference calls, and e-mail exchanges, and discussed and reviewed EPA's draft methodology for conducting biological evaluations (BE) of EPA's aquatic life criteria methods manual (Methods Manual, EPA 2005). Key events covered over this period are summarized below.

On August 9, 2005, EPA provided NMFS with a copy of the methods manual.

On October 3, 2005, EPA provided NMFS with a preliminary analysis for saltwater zinc and saltwater cadmium to review.

On November 9, 2005, November 10, 2005, and November 17, 2005, NMFS provided EPA several issue papers detailing technical issues with the methods manual and the preliminary analyses for saltwater zinc and saltwater cadmium.

On April 7, 2006, Northwest Environmental Advocates (NWEA) sent EPA a 60-day notice of intent to sue for violations of the Clean Water Act (CWA).

On August 21, 2006, EPA provided NMFS with a draft BE on the effects of its proposed approval of 39 freshwater and 16 saltwater criteria for toxics to review.

On November 2, 2006, NMFS provided EPA with detailed comments on the draft BE for toxics. In our letter, we identified several fundamental problems with the

application of the methods manual and the draft BE. Subject areas that needed substantial revision or a new approach are summarized below by category.

- Median lethal concentration (LC_{50}) toxicity data interpretation and application
- No observable effect concentration (NOEC) toxicity data interpretation and application
- Exclusion of published toxicity data in the BE analysis
- Acute adjustment factor
- Sublethal effects analysis
- Chemical mixture analysis
- Scale of effect determinations—effects of the action as a whole versus effects based on individual criterion

On December 20, 2006, NMFS, FWS and EPA met to discuss issues with the draft BE and the methods manual.

On February 2, 2007, NMFS, FWS, and EPA developed a draft issues paper as a means to resolve outstanding issues with the BE.

On February 6, 2007, NMFS, FWS, and EPA met to discuss a path forward for resolving outstanding issues with the BE.

On January 16, 2008, EPA submitted a BE with a letter requesting formal consultation on its proposed approval of the Oregon revisions to state water quality standards for toxic pollutants.

On April 4, 2008, NMFS submitted a data request via letter to EPA.

On May 23, 2008, EPA and NWEA settled their lawsuit via consent decree.

October 3, 2008, EPA provided the last of the data requests to NMFS.

On May 26, 2009, NWEA sent NMFS a 60-day notice of intent to sue for failing to timely complete ESA section 7 consultation.

On August 23, 2010, NMFS and NWEA settled their lawsuit via a stipulated order of dismissal.

Between January 2012 through May 2012, NMFS and EPA participated in a series of meetings to discuss the findings in the draft opinion and develop the reasonable and prudent alternative, including meeting with EPA region 10 staff on April 19, 2012, to discuss the reasonable and prudent alternatives and reasonable and prudent measures.

On February 24, 2012, NMFS provided EPA with a preliminary draft opinion.

On March 8, 2012, NMFS meet with representatives of the Columbia River Inter-Tribal Fish Commission for a technical-level meeting on the consultation.

On March 20, 2012, NMFS meet with representatives of the Yakama Nation for a technical-level meeting on the consultation.

On March 28, 2012, NMFS sent EPA a letter regarding the court-ordered deadline and key dates for interagency coordination to finalizing the opinion.

On April 11, 2012, NMFS received a letter from EPA recognizing the court-ordered deadline and key dates for interagency coordination to finalizing the opinion.

On May 7, 2012, NMFS received a letter from EPA with comments on the February 24, 2012, draft opinion.

On May 7, 2012, NMFS provided EPA with a final draft opinion.

Between May 17, 2012, and August 1, 2012, NMFS and EPA exchanged information on the development of the reasonable and prudent alternative (RPA).

On August 9, 2012, EPA sent NMFS a letter withdrawing their request for consultation on Oregon's acute and chronic aluminum criteria as "EPA has determined that the BE submitted to NMFS in January 2008 incorrectly described the proposed federal action under consultation for aluminum (*i.e.*, CW A § 303(c)(3) approval of Oregon's submission of aluminum criteria). Specifically, Oregon's submitted description of the pollutant refers to aluminum in waters with a pH of 6.5- 9.0, but a footnote in the criterion itself indicates that the criterion is meant to apply to waters with pH less than 6.6 and hardness less than 12 mg/L (as $CaCO_3$)." Due to the court-ordered deadline of August 14, 2012, NMFS did not have time to modify its opinion to exclude acute and chronic aluminum from the document. The NMFS acknowledges EPA's revision to the proposed action, however, and notes it does not anticipate EPA will carry out the RPA for aluminum in light of this change. The NMFS will await a further request from EPA relating to EPA's potential future actions regarding Oregon's aluminum criteria.

1.3 Proposed Action

The proposed action is EPA's, Region 10, proposed approval of portions of Oregon Administrative Rules (340-041-0033) related to revised water quality criteria for toxic pollutants for aquatic life (Table 1.1) under section 303(c) of the Clean Water Act (CWA), and 40 CFR 131. The CWA requires all states to adopt water quality standards (WQS) to restore and maintain the physical, chemical, and biological integrity the Nation's waters. Section 303(c) of the act requires states to adopt chemical-specific, numeric criteria for priority toxic pollutants. The criteria must protect state-designated beneficial uses of water bodies. Development of WQS is primarily the responsibility of the states, but adoption of the WQS is subject to approval by EPA. The EPA is proposing to approve or disapprove Oregon's proposed numeric water quality criteria for 20 toxic pollutants that include 39 freshwater criteria and 26 saltwater criteria.

Oregon's proposed aquatic life criteria are listed in Table 1.1. The Oregon criteria are identical to the national criteria developed by EPA and recommended by EPA to states for adoption. Table 1.2 provides a comparison of the Oregon's existing numeric criteria with the proposed numeric criteria for aquatic life subject to this consultation. Table 1.3 lists all the toxic criteria with numeric criteria (regulated by Oregon) and those without numeric criteria (unregulated). In the BE, EPA evaluated the proposed criteria as continuous water quality conditions, *i.e.*, EPA assumed that listed species would be exposed to waters meeting the proposed water quality criteria listed in Table 1.1. The EPA assumed that the numeric criteria would be met outside the State's applicable mixing zone boundaries, *i.e.*, that the criteria represent ambient water quality conditions.

Proposed aquatic life criteria that are the same or more stringent than previously approved by EPA may be used prior to EPA approval in national pollution elimination system [NPDES and stormwater (MS4)] permits issued by the Oregon Department of Environmental Quality (ODEQ) unless they are (1) formula-based metals, (2) ammonia, (3) were previously total recoverable criteria, or (4) would discharge into a 303(d)-listed impaired water, and are otherwise not in effect until approved by EPA. Compounds subject to pre-approval use are lindane, dieldrin, endosulfan-alpha, endosulfan-beta, and heptachlor epoxide, all legacy compounds, *i.e.*, compounds that are either no longer in use or their use is highly restricted within the U.S.

The acute criterion is the Criterion Maximum Concentration (CMC) and is EPA's acute criterion recommendation. The CMC is set to one-half of the fifth percentile of the average acute toxicity values for the various genera tested. The EPA's technical support document (EPA 1991) recommends that the one-hour average exposure concentrations should not exceed the CMC more than once every three years on the average.

The chronic criterion is the Criterion Continuous Concentration (CCC), criterion for indefinite exposures, and is EPA's chronic criterion recommendation. The CCC is derived from a set of chronic toxicity values, which are the geometric mean of the highest no observed effect concentrations (NOEC) and lowest observed effect concentrations (LOEC) for survival, growth, or reproduction in tests which range from seven days to several months or more. The EPA's technical support document (EPA 1991) recommends that the four-day average exposure concentrations should not exceed the CCC more frequently than once every three years on the average.

For ammonia, the numeric criteria are based on the following equations (numeric criteria for ammonia are calculated based on site-specific pH and temperature):

1) Acute ammonia criterion, salmonid fishes present:

$$CMC = \frac{0.275}{1 + 10^{7.204 - pH}} + \frac{39.0}{1 + 10^{pH - 7.204}}$$

2) Acute ammonia criterion, salmonid fishes absent:

$$CMC = \frac{0.411}{1 + 10^{7.204 - pH}} + \frac{58.4}{1 + 10^{pH - 7.204}}$$

3) Chronic ammonia criterion, early life stages present:

$$CCC = \frac{0.577}{1+10^{7.688 - pH}} \frac{2.487}{+} \quad 1+10^{pH - 7.688} \quad * MIN (2.85, 1.45^{*} 10)^{0.028(25-T)}$$

4) Chronic ammonia criterion, early life stages not present:

$$CCC = \frac{0.577}{1+10^{7.688 - pH}} + \frac{2.487}{1+10^{pH - 7.688}} \quad *1.45^{*} 10^{0.028 (25- (MAX T, 7))}$$

The freshwater criterion for cadmium, chromium (III), copper, lead, nickel, silver, and zinc are expressed as a function of hardness (CaCO3 mg/L) in the water column (refer to Appendix A in the BE, pages 16-26, for equations and conversion factors).

Table 1.1 Proposed Oregon aquatic life criteria for toxics. All values are expressed as micrograms per liter (μg/L) except where noted. Shaded cells denote no criteria proposed for EPA approval.

Compounds	Freshwater Acute Criteria (μg/L)	Freshwater Chronic Criteria (μg/L)	Saltwater Acute Criteria (μg/L)	Saltwater Chronic Criteria (μg/L)
Aluminum	750	87		
Ammonia*	5.6 mg/L	1.7 mg/L		
Arsenic	340	150	69	36
gamma-BHC (Lindane)	0.95			
Cadmium	2.0	.25	40	8.8
Chromium (III)	570	74		
Chromium (VI)	16	11	1100	50
Copper	13	9.0	4.8	3.1
Dieldrin	0.24	0.056		
alpha- Endosulfan	0.22	0.056	0.034	0.0087
beta- Endosulfan	0.22	0.056	0.034	0.0087
Endrin	0.086	0.036		
Heptachlor epoxide	0.52	0.0038	0.053	0.0036
Lead	65	2.5	210	8.1
Nickel	470	52	74	8.2
Pentachlorophenol	19	15		7.9
Selenium	190	5.0	290	71
Silver	3.2	0.10	1.9	
Tributyltin	.46	.063	.37	.01
Zinc	120	120	90	81

* See equations 1, 2, 3, and 4.

Table 1.2 Existing and proposed numeric criteria for aquatic life in Oregon.

Compound	Existing Acute Criteria	Proposed Acute Criteria	Existing Chronic Criteria	Proposed Chronic Criteria	Existing Acute Criteria	Proposed Acute Criteria	Existing Chronic Criteria	Proposed Chronic Criteria
	FW	FW	FW	FW	SW	SW	SW	SW
Ar	360	340	190	150	69	69	36	36
Cd	3.9	2	1.1	0.25	43	40	9.3	8.8
CrIII	1700	570	210	74				
CrVI	16	16	11	11	1100	1100	50	50
Cu	18	13	12	9	2.9	4.8	2.9	3.1
Pb	82	65	3.2	2.5	140	210	5.6	8.1
Ni	1400	470	160	52	75	74	8.3	8.2
Se	260	190	35	5	410	290	54	71
Ag	4.1	3.2	0.12	0.1	2.3	1.9		
Zn	120	120	110	120	95	90	86	81
PCP	20	19	13	15				7.9
Dieldrin	2.5	0.24	0.0019	0.056				
Endrin	0.18	0.086	0.0023	0.036				
Ammonia	6	5.6	0.76	1.7				
Lindane	2	0.95						
TBT		0.46		0.063		0.37		0.01
Al		750		87				
Hept E		0.52		0.0038		0.053		0.0036
Endo-a		0.22		0.056		0.034		0.0087
Endo-b		0.22		0.056		0.034		0.0087
same	7							
more strict	30							
less strict	9							
previously unregulated	19							
	No criteria proposed							
Boldtype=legacy compounds								

Table 1.3 Regulated and unregulated toxic compounds in the State of Oregon (ODEQ 2003). Compounds considered in this opinion for approval by EPA are shaded.

Aquatic Life Criteria				
	Freshwater	**Freshwater**	**Marine**	**Marine**
	Acute Criteria	**Chronic Criteria**	**Acute Criteria**	**Chronic Criteria**
Compound (µg/L)				
Antimony				
Arsenic *	360	190	69	36
Cadmium ***	3.9	1.1	43	9.3
Chromium III ***	1700	210		
Chromium VI *	16	11	1100	50
Copper ***	18	12	2.9	2.9
Lead ***	82	3.2	241	5.6
Mercury	2.4	0.012	2.1	0.025
Nickel ***	1400	160	75	8.3
Selenium *	260	35	410	54
Silver **	4.1	0.12	2.3	
Thallium				
Zinc ***	120	110	95	86
Cyanide	22	5.2	1	1
Asbestos				
Dioxin (2,3,7,8-TCDD)				
Acrolein				
Acrylonitrile				
Benzene				
Bromoform				
Carbon Tetrachloride				
Chlorobenzene				
Chlorodibromomethane				
Chloroform				
Dichlorobromomethane				
Dichloroethane 1,2-				
Dichloroethylene 1,1-				
Dichloropropane 1,2-				
Dichloropropene 1,3-				
Ethylbenzene				
Methyl Bromide				
Methylene Chloride				
Tetrachloroethane 1,1,2,2-				
Tetrachloroethylene				
Toluene				
Dichloroethylene 1,2-Trans-				
Trichloroethane 1,1,2-				
Trichloroethylene				
Vinyl Chloride				
Chlorophenol 2-				
Dichlorophenol 2,4-				

Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
Dimethylphenol 2,4-				
Methyl-4,6-Dinitrophenol 2-				
Dinitrophenol 2,4-				
Pentachlorophenol	20	13	13	7.9
Phenol				
Trichlorophenol 2,4,6-				
Acenaphthene				
Anthracene				
Benzidine				
BenzoaAnthracene				
BenzoaPyrene				
BenzobFluoranthene				
BenzokFluoranthene				
ChloroethylEther, Bis2-				
ChloroisopropylEther, Bis2-				
EthylhexylPhthalate, Bis2-				
Butylbenzyl Phthalate				
Chloronaphthalene 2-				
Chrysene				
Dibenzoa,hAnthracene				
Dichlorobenzene 1,2-				
Dichlorobenzene 1,3-				
Dichlorobenzene 1,4-				
Dichlorobenzidine 3,3'-				
DiethylPhthalate				
Dimethyl Phthalate				
Di-n-Butyl Phthalate				
Dinitrotoluene 2,4-				
Diphenylhydrazine 1,2-				
Fluoranthene				
Fluorene				
Hexachlorobenzene				
Hexachlorobutadiene				
Hexachlorocyclopentadiene				
Hexachloroethane				
Ideno1,2,3-cdPyrene				
Isophorone				
Nitrobenzene				
Nitrosodimethylamine, N-				
Nitrosodi-n-Propylamine, N-				
Nitrosodiphenylamine, N-				
Pyrene				
Trichlorobenzene 1,2,4-				
Aldrin	3.0		1.3	

Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
BHC, alpha-				
BHC, beta-				
BHC, gamma- (Lindane)	2	0.08	0.16	
Chlordane	2.4	0.0043	0.09	0.004
DDT 4,4'-	1.1	0.001	0.13	0.001
DDE 4,4'-				
DDD 4,4'-				
Dieldrin	2.5	0.0019	0.71	0.0019
Alpha-Endosulfan				
Beta-Endosulfan				
Endosulfan Sulfate				
Endrin	0.18	0.0023	0.037	0.0023
Endrin Aldehyde				
Heptachlor	0.52	0.0038	0.053	0.0036
Heptachlor Epoxide				
Polychlorinated biphenyls PCBs:	2	0.014	10	0.03
Toxaphene	0.73	0.0002	0.21	0.0002
Aluminum				
Ammonia (mg/L)	6	0.76		
Barium				
Chloride	860000	230000		
Chlorine	19	11	13	7.5
Chlorophenoxy Herbicide 2,4,5,-TP				
Chlorophenoxy Herbicide 2,4-D				
Chloropyrifos	0.083	0.041	0.011	0.0056
Demeton		0.1		0.1
Ether, Bis Chloromethyl				
Guthion		0.01		0.01
Hexachlorocyclo-hexane-Technical				
Iron		1000		
Malathion		0.1		0.1
Manganese				
Methoxychlor		0.03		0.03
Mirex		0.001		0.001
Nitrates				
Nitrosamines				
Dinitrophenols				
Nitrosodibutylamine,N				
Nitrosodiethylamine,N				
Nitrosopyrrolidine,N				
Parathion	0.065	0.013		
Pentachlorobenzene				
Phosphorus Elemental				0.1
Sulfide-Hydrogen Sulfide		2.0		2.0

Aquatic Life Criteria				
	Freshwater	**Freshwater**	**Marine**	**Marine**
	Acute Criteria	**Chronic Criteria**	**Acute Criteria**	**Chronic Criteria**
Compound (µg/L)				
Tetrachlorobenzene,1,2,4,5				
Tributyltin TBT				
Trichlorophenol 2,4,5				
* all criteria expressed as dissolved metal ** all criteria expressed as dissolved metal. FW acute criteria are hardness dependent (concentration shown is hardness = 100 mg/L $CaCO_3$) *** all criteria expressed as dissolved metal. FW criteria are hardness dependent (concentration shown is hardness = 100 mg/L $CaCO_3$)				

1.4 Action Area

'Action area' means all areas to be affected directly or indirectly by the Federal action and not merely the immediate area involved in the action (50 CFR 402.02). The species occurring within the action area that are the subject of this consultation are listed in Table 1.4.1 and Table 1.4.2.

References for listing status and dates, ESA section 4(d) take prohibitions, and critical habitat designations are provided in Table 1.4.1 and Table 1.4.2.

Table 1.4.1. Federal Register notices for final rules that list threatened and endangered species, designate critical habitats, or apply protective regulations to listed species considered in this consultation (anadromous fishes).

Species	Listing Status	Critical Habitat	Protective Regulations
Chinook salmon (*Oncorhynchus tshawytscha*)			
Lower Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Upper Willamette River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Upper Columbia River spring-run	E 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	ESA section 9 applies
Snake River spring/summer run	T 8/15/11; 76 FR 50448	10/25/99; 64 FR 57399	6/28/05; 70 FR 37160
Snake River fall-run	T 8/15/11; 76 FR 50448	12/28/93; 58 FR 68543	6/28/05; 70 FR 37160
Chum salmon *(O. keta*)			
Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Coho salmon (*O. kisutch*)			
Lower Columbia River	T 8/15/11; 76 FR 50448	Not applicable	6/28/05; 70 FR 37160
Southern Oregon/northern California coasts	T 8/15/11; 76 FR 50448	5/5/99; 64 FR 24049	6/28/05; 70 FR 37160
Oregon coast	T 2/11/08; 73 FR 7816	2/11/08; 73 FR 7816	2/11/08; 73 FR 7816
Sockeye salmon (*O. nerka*)			
Snake River	E 8/15/11; 76 FR 50448	12/28/93; 58 FR 68543	ESA section 9 applies
Steelhead (*O. mykiss*)			
Lower Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Upper Willamette River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Middle Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Upper Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	2/1/06; 71 FR 5178
Snake River basin	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Green sturgeon (*Acipenser medirostris)*			
Southern DPS	T 4/7/06; 71 FR 17757	10/9/2009: 74 FR 52300	6/2/10; 75 FR 30714
Eulachon (*Thaleichthys pacificus)*			
Eulachon	3/18/10; 75 FR 13012	10/20/11; 76 FR 65324	Not applicable

Table 1.4.2. Federal Register notices for final rules that list threatened and endangered species, designate critical habitats, or apply protective regulations to listed species considered in this consultation (marine mammals and turtles).

Species	Listing Status	Critical Habitat	Protective Regulations
Southern Resident killer whale (*Orcinus orca*)	E 11/18/05; 70 FR 69903	11/29/06; 71 FR 69034	ESA section 9 applies
Steller sea lion (*Eumetopias jubatus*)	T 11/26/90; 55 FR 49204	8/27/93; 58 FR 45269	11/26/90; 55 FR 49204
Blue whale (*Balaenoptera musculus*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
Fin whale (*Balaenoptera physalus*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
Sei whale (*Balaenoptera borealis*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
Sperm whale (*Physeter macrocephalus*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
Humpback whale (*Megaptera novaeangliae*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
North Pacific right whale (*Eubalaena glacialis*)	E 12/2/70; 35 FR 19319	7/6/06; 71 FR 38277	ESA section 9 applies
Loggerhead turtle (*Caretta caretta*)	T 7/28/78; 43 FR 32800	Not applicable	7/28/78; 43 FR 32800
Green sea turtle (*Chelonia mydas*)	T 7/28/78; 43 FR 32800	9/2/98; 63 FR 46693	7/28/78; 43 FR 32800
Leatherback turtle (*Dermochelys coriacea*)	E 12/2/70; 35 FR 18319	1/26/2012; 77 FR 4170	ESA section 9 applies
Olive Ridley turtle (*Lepidochelys olivacea*)	T 7/28/78; 43 FR 32800	Not applicable	7/28/78; 43 FR 32800

The fish considered in the opinion occur in the action area and use freshwater and marine habitats for multiple life history events, including incubation; emergence (residence in gravel); juvenile rearing, smoltification and migration; and adult migration, holding and spawning.

Marine mammals and sea turtles considered in this opinion occur in the marine portion of the below stated action area and use freshwater (Steller sea lions only) and marine habitats for multiple life history events, including foraging, rearing, and migration. Chinook salmon that originate from Oregon will disperse both north (to the coastal waters of Washington and the west coast of Vancouver Island), and south off the coast of California (Weitkamp 2010). Therefore, the action area for Southern Resident killer whales encompasses the whales' entire coastal range from California to Vancouver, British Columbia where the marine ranges of Southern Residents and affected Chinook salmon overlap.

The action area for this consultation includes the freshwater, estuarine, and ocean areas subject to the jurisdiction of the State of Oregon, where the criteria apply, as well as areas beyond the state's jurisdiction where the regulated pollutants area likely to be transported. The action area includes the Pacific Ocean, limited to the entire coastal range from California to Vancouver, British Columbia, where the marine ranges of some of the species subject to this consultation (Southern Resident killer whales and Chinook salmon) overlap, and to which the particular compounds under consultation (Table 1.1) are transported beyond these limits by such biotic and abiotic factors as river runoff, tidal energy, topography, stratigraphy, biota trapping/assimilation), that may influence chemical transport processes beyond original areas of dispersion.

Based on the chemical processes (sources, transport, fate, transformation) of compounds listed in Table 1.1, which are described later in this opinion, the action area, in addition to the Pacific Ocean area delineated above, includes all inland basins that provide access to the species listed in Table 1.1 (Figure 1.4.1 and Figure 1.4.2), including the Columbia River, bank-to-bank, from the mouth to the Washington-Oregon border [river mile (RM) 292]; and the Snake River, from RM 169 to RM 247.5 (Figure 1.4.1 and Figure 1.4.2). The Klamath River originates in southwest Oregon. However, the Iron Gate dam prevents up-river migration of (southern Oregon/Northern California coasts) SONCC coho salmon across the Oregon-California border. Iron Gate dam is located on the Klamath River at river mile 190.2 in California. Based on the fact that no southern Oregon/Northern California coasts SONCC coho salmon from the Klamath Strata occur in Oregon, NMFS determined that individuals of populations in the Klamath, Trinity, or central strata are not at risk of direct exposure to the toxics listed in Table 1.1 in association with this action.

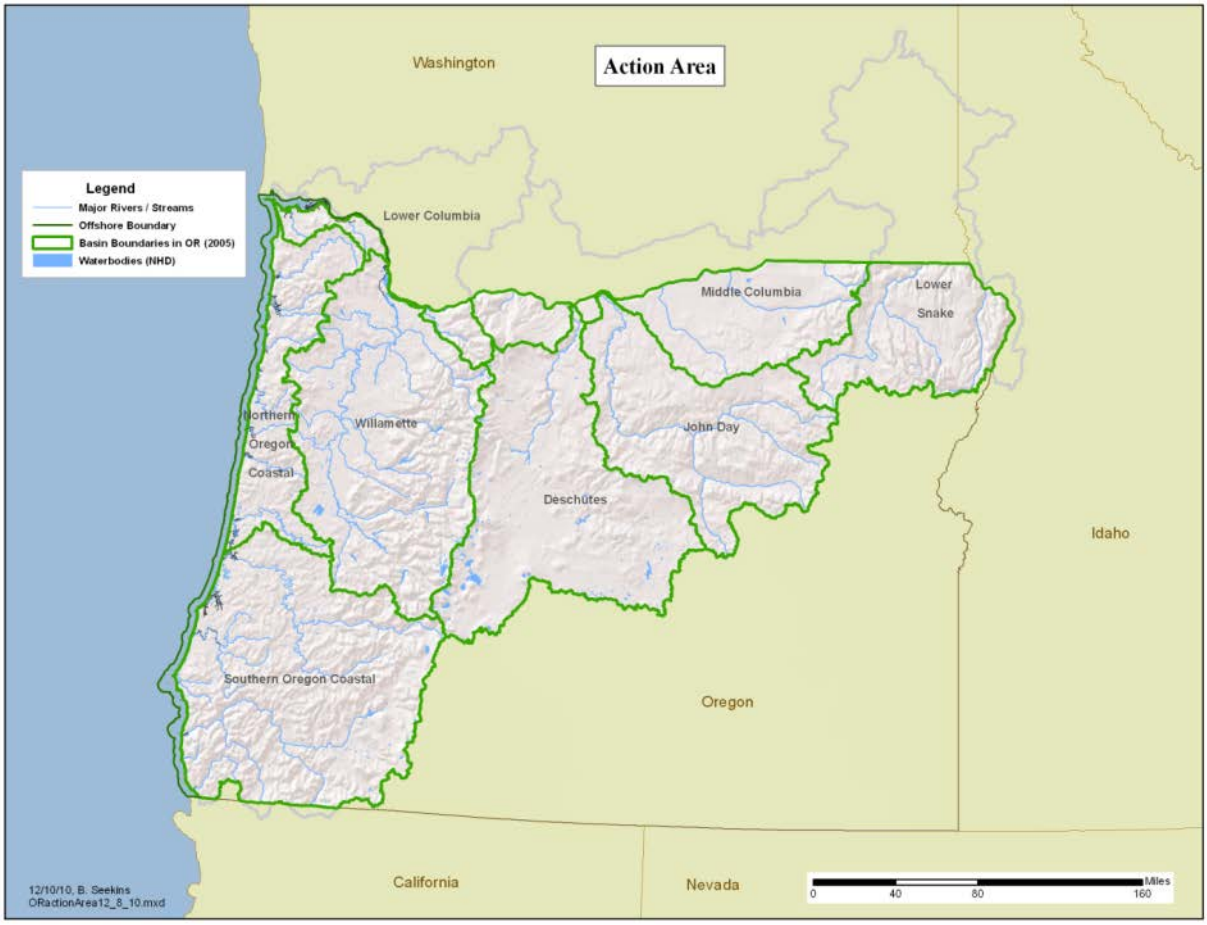

Figure 1.4.1. Overview of the of the action area (highlighted subbasins and the Pacific Ocean, not inclusive of the action area for Southern Resident killer whales).

Figure 1.4.2. Action area (light shading) for southern resident killer whales. Reprinted from Wiles (2004).

2. ENDANGERED SPECIES ACT BIOLOGICAL OPINION AND INCIDENTAL TAKE STATEMENT

The ESA establishes a national program for conserving threatened and endangered species of fish, wildlife, plants, and the habitat on which they depend. Section 7(a)(2) of the ESA requires Federal agencies to consult with the U.S. Fish and Wildlife Service, NMFS, or both, to ensure that their actions are not likely to jeopardize the continued existence of endangered or threatened species or adversely modify or destroy their designated critical habitat. Section 7(b)(3) requires that at the conclusion of consultation, the Service provide an opinion stating how the agencies' actions will affect listed species or their critical habitat. If incidental take is expected, section 7(b)(4) requires the provision of an incidental take statement (ITS) specifying the impact of any incidental taking, and including reasonable and prudent measures to minimize such impacts.

2.1 Introduction to the Biological Opinion

Section 7(a)(2) of the ESA requires Federal agencies, in consultation with NMFS, to insure that their actions are not likely to jeopardize the continued existence of endangered or threatened species, or adversely modify or destroy their designated critical habitat. The jeopardy analysis considers both survival and recovery of the species. The adverse modification analysis considers the impacts to the conservation value of the designated critical habitat.

"To jeopardize the continued existence of a listed species" means to engage in an action that would be expected, directly or indirectly, to reduce appreciably the likelihood of both the survival and recovery of a listed species in the wild by reducing the reproduction, numbers, or distribution of that species (50 CFR 402.02).

This opinion does not rely on the regulatory definition of "destruction or adverse modification" of critical habitat at 50 CFR 402.02. Instead, we have relied upon the statutory provisions of the ESA to complete the following analysis with respect to critical habitat.[1]

2.2 Approach to the Assessment

We will use the following approach to determine whether the proposed action described in Section 1.4 is likely to jeopardize listed species or destroy or adversely modify critical habitat:

- *Identify the rangewide status of the species and critical habitat likely to be adversely affected by the proposed action*. This section describes the current status of each listed species and its critical habitat relative to the conditions needed for recovery. For listed salmon and steelhead, NMFS has developed specific guidance for analyzing the status of the listed species' component populations in a "viable salmonid populations" paper (VSP; McElhany *et al.* 2000). The VSP approach considers the abundance, productivity, spatial structure, and diversity of each population as part of the overall review of a species' status. For listed salmon and steelhead, the VSP criteria therefore encompass the

[1] Memorandum from William T. Hogarth to Regional Administrators, Office of Protected Resources, NMFS (Application of the "Destruction or Adverse Modification" Standard Under Section 7(a)(2) of the Endangered pecies Act) (November 7, 2005).

species' "reproduction, numbers, or distribution" (50 CFR 402.02). In describing the range-wide status of listed species, we rely on viability assessments and criteria in technical recovery team documents and recovery plans, where available, that describe how VSP criteria are applied to specific populations, major population groups, and species. We determine the rangewide status of critical habitat by examining the condition of its physical or biological features (also called "primary constituent elements" or PCEs in some designations) – which were identified when the critical habitat was designated. Species and critical habitat status are discussed in Section 2.4 of this opinion.

- *Describe the environmental baseline for the proposed action*. The environmental baseline includes the past and present impacts of Federal, state, or private actions and other human activities *in the action area*. It includes the anticipated impacts of proposed Federal projects that have already undergone formal or early section 7 consultation and the impacts of state or private actions that are contemporaneous with the consultation in process. The environmental baseline is discussed in section 2.5 of this opinion.

- *Analyze the effects of the proposed actions*. In this step, NMFS considers how the proposed action would affect the species' reproduction, numbers, and distribution or, in the case of salmon and steelhead, their VSP characteristics.

- *Analyze the effects of the proposed actions*. In this step, NMFS considers how the proposed action would affect the conservation value of critical habitat for the affected species.

- *Describe any cumulative effects.* Cumulative effects, as defined in NMFS' implementing regulations (50 CFR 402.02), are the effects of future state or private activities, not involving Federal activities, that are reasonably certain to occur within the action area. Future Federal actions that are unrelated to the proposed action are not considered because they require separate section 7 consultation. Cumulative effects are considered in Section 2.6.8 of this opinion.

- *Integrate and synthesize the above factors to assess the risk that the proposed action poses to species and critical habitat.* In this step, NMFS adds the effects of the action (section 2.6) to the environmental baseline (section 2.5) and the cumulative effects (section 2.6.8) to assess whether the action could reasonably be expected to: (1) appreciably reduce the likelihood of both survival and recovery of the species in the wild by reducing its numbers, reproduction, or distribution; or (2) reduce the value of designated or proposed critical habitat for the conservation of the species. These assessments are made in full consideration of the status of the species and critical habitat (section 2.4). Integration and synthesis occurs in section 2.7 of this opinion.

- *Reach jeopardy and adverse modification conclusions*. Conclusions regarding jeopardy and the destruction or adverse modification of critical habitat are presented in section 2.9 of this opinion. These conclusions flow from the logic and rationale presented in the Integration and Synthesis section (2.7) of this opinion.

- *If necessary, define a reasonable and prudent alternative to the proposed action.* If, in completing the last step in the analysis, NMFS determines that the action under consultation is likely to jeopardize the continued existence of listed species or destroy or adversely modify designated critical habitat, NMFS must identify a reasonable and prudent alternative (RPA) to the action. The RPA must not be likely to jeopardize the continued existence of ESA-listed species nor destroy or adversely modify their designated critical habitat, and it must meet other regulatory requirements.

2.3. Species and Critical Habitat not considered further in this Opinion

In this opinion NMFS concludes that the proposed action is not likely to adversely affect (NLAA) Steller sea lions, humpback whales, blue whales, fin whales, Sei whales, sperm whales, North Pacific Right whales, loggerhead sea turtles, green sea turtles, leatherback sea turtles, and Olive Ridley sea turtles. Refer to section 2.14 for NLAA determinations.

2.4 Rangewide Status of the Species and Critical Habitat

The summaries that follow describe the status of the listed species, and their designated critical habitats, that occur within the action area of this proposed action and are considered in this opinion. More detailed information on the status and trends of these listed resources, and their biology and ecology, can be found in the listing regulations and critical habitat designations published in the Federal Register (Table 1.4.1 and Table 1.4.2, above).

2.4.1 Climate Change

Climate change is likely to play an increasingly important role in determining the abundance of listed species, and the conservation value of designated critical habitats, in the Pacific Northwest. These changes will not be spatially homogeneous across the Pacific Northwest. Areas with elevations high enough to maintain temperatures well below freezing for most of the winter and early spring would be less affected. Low-lying areas that historically have received scant precipitation contribute little to total streamflow and are likely to be more affected.

During the last century, average regional air temperatures increased by 1.5°F, and increased up to 4°F in some areas (USGCRP 2009). Warming is likely to continue during the next century as average temperatures increase another 3 to 10°F (USGCRP 2009). Overall, about one-third of the current cold-water fish habitat in the Pacific Northwest is likely to exceed key water temperature thresholds by the end of this century (USGCRP 2009).

Precipitation trends during the next century are less certain than for temperature but more precipitation is likely to occur during October through March and less during summer months, and more of the winter precipitation is likely to fall as rain rather than snow (ISAB 2007, USGCRP 2009). Where snow occurs, a warmer climate will cause earlier runoff so stream flows in late spring, summer, and fall will be lower and water temperatures will be warmer (ISAB 2007, USGCRP 2009).

Higher winter stream flows increase the risk that winter floods in sensitive watersheds will damage spawning redds and wash away incubating eggs (USGCRP 2009). Earlier peak stream flows will also flush some young salmon and steelhead from rivers to estuaries before they are physically mature, increasing stress and the risk of predation (USGCRP 2009). Lower stream flows and warmer water temperatures during summer will degrade summer rearing conditions, in part by increasing the prevalence and virulence of fish diseases and parasites (USGCRP 2009). Other adverse effects are likely to include altered migration patterns, accelerated embryo development, premature emergence of fry, variation in quality and quantity of tributary rearing habitat, and increased competition and predation risk from warm-water, non-native species (ISAB 2007).

The earth's oceans are also warming, with considerable interannual and inter-decadal variability superimposed on the longer-term trend (Bindoff *et al.* 2007). Historically, warm periods in the coastal Pacific Ocean have coincided with relatively low abundances of salmon and steelhead, while cooler ocean periods have coincided with relatively high abundances (Scheuerell and Williams 2005, Zabel *et al.* 2006, USGCRP 2009). Ocean conditions adverse to salmon and steelhead may be more likely under a warming climate (Zabel *et al.* 2006).

2.4.2 Status of the Species

The status of species and critical habitat sections below are organized under four recovery domains (Table 2.4.2.1) to better integrate recovery planning information that NMFS is developing on the conservation status of the species and critical habitats considered in this consultation. Recovery domains are the geographically-based areas that NMFS is using to prepare multi-species recovery plans. Southern green sturgeon are under the jurisdiction of NMFS' Southwest Region. The first meeting of the recovery team for this species was announced to be held in December, 2009. A recovery team has not yet been convened for eulachon, a species under the jurisdiction of NMFS' Northwest Region. Green sturgeon and eulachon may occur in multiple recovery domains.

Table 2.4.2.1. Recovery planning domains identified by NMFS and their ESA-listed salmon and steelhead species.

Recovery Domain	Species
Willamette-Lower Columbia (WLC)	LCR Chinook salmon UWR Chinook salmon CR chum salmon LCR coho salmon LCR steelhead UWR steelhead
Interior Columbia (IC)	UCR spring-run Chinook salmon SR spring/summer Chinook salmon SR fall-run Chinook salmon SR sockeye salmon UCR steelhead MCR steelhead SRB steelhead
Oregon Coast (OC)	OC coho salmon
Southern Oregon/Northern California Coasts (SONCC)	SONCC coho salmon

For each recovery domain, a technical review team (TRT) appointed by NMFS has developed, or is developing, criteria necessary to identify independent populations within each species, recommended viability criteria for those species, and descriptions of factors that limit species survival. Viability criteria are prescriptions of the biological conditions for populations, biogeographic strata, and ESUs that, if met, would indicate that the ESU will have a negligible risk of extinction over a 100-year time frame.[2]

The definition of a population used by each TRT to analyze salmon and steelhead is set forth in the "viable salmonid population" document prepared by NMFS for use in conservation assessments of Pacific salmon and steelhead (McElhany *et al.* 2000). That document defines population viability in terms of four variables: abundance, population growth rate (productivity), population spatial structure, and genetic diversity.

Abundance is of obvious importance since, in general, small populations are at greater risk of extinction than large populations, primarily because many processes that affect population dynamics may operate differently in small populations than in large populations (Shaffer 1987, McElhany *et al.* 2000).

[2] For Pacific salmon, NMFS uses its 1991 ESU policy, that states that a population or group of populations will be considered a distinct population segment if it is an evolutionarily significant unit (ESU). An ESU represents a distinct population segment of Pacific salmon under the Endangered Species Act that 1) is substantially reproductively isolated from conspecific populations and 2) represents an important component of the evolutionary legacy of the species. The species *O. mykiss* is under the joint jurisdiction of NMFS and the Fish and Wildlife Service, so in making its listing January, 2006 determinations NMFS elected to use the 1996 joint FWS-NMFS DPS policy for this species.

Population growth rate, the productivity over the entire life cycle, and factors that affect population growth rate provide information about how well a population is performing in the various habitats it occupies during the life cycle. Examining population growth rate allows one to assess if populations are able to replace themselves. Populations that consistently fail to replace themselves are at greater risk of extinction than populations that are consistently at or above replacement levels.

Spatial structure refers to the distribution of individuals within a population at a certain life stage throughout the available habitats, recognizing the abiotic and biotic processes that give rise to that structure. McElhany *et al.* (2000) gave two main reasons why spatial structure is important to consider when evaluating population viability: 1) overall extinction risk at longer time scales may be affected in ways not apparent from short-term observations of abundance and productivity, because there can be a time lag between changes in spatial structure and the resulting population-level effects, and 2) spatial population structure affects the ability of a population to respond to changing environmental conditions and therefore can influence evolutionary processes. Maintaining spatial structure within a population, and its associated benefits to viability, requires appropriate habitat conditions and suitable corridors linking the habitat and the marine environment to be consistently available.

Diversity relates to the variability of phenotypic characteristics such as life histories, individual size, fecundity, run timing, and other attributes exhibited by individuals and populations, as well as the genetic diversity that may underlie this variation. There are many reasons diversity is important in a spatially and temporally varying environment. Three key reasons are: (1) Diversity allows a species to use a wide array of environments; (2) diversity protects a species against short-term spatial and temporal changes in the environment; and (3) genetic diversity provides the raw material for surviving long-term environmental change (McElhany *et al.* 2000).

Although the TRTs operated from the common set of biological principals described in McElhany *et al.* (2000), they worked semi-independently from each other and developed criteria suitable to the species and conditions found in their specific recovery domains. All of the criteria have qualitative as well as quantitative aspects. The diversity of salmonid species and populations makes it impossible to set narrow quantitative guidelines that will fit all populations in all situations. For this and other reasons, viability criteria vary among species, mainly in the number and type of metrics and the scales at which the metrics apply (*i.e.*, population, major population group (MPG, or strata, or ESU) (Busch *et al.* 2008).

Overall viability risk scores (high to low) are based on combined ratings for the abundance and productivity (A/P) and spatial structure and diversity[3] (SS/D) metrics. WLC scores (Table 2.4.2.2) are based on population persistence established by McElhany *et al.* (2006). IC-TRT viability criteria were based on (McElhany *et al.* 2000 and 2006), as well as the results of previous applications in other TRTs and a review of specific information available relative to listed IC ESU populations (IC-TRT 2007). The A/P score considers the TRT's estimate of a populations' minimum threshold population, natural spawning abundance and the productivity of

[3] The WLC-TRT provided ratings for diversity and spatial structure risks. The IC-TRT provided spatial structure and diversity ratings combined as an integrated SS/D risk.

the population. Productivity over the entire life cycle and factors that affect population growth rate provide information on how well a population is "performing" in the habitats it occupies during the life cycle. Estimates of population growth rate that indicate a population is consistently failing to replace itself are an indicator of increased extinction risk. The four metrics (abundance, productivity, spatial structure, and diversity) are not independent of one another and their relationship to sustainability depends on a variety of interdependent ecological processes (Wainwright *et al.* 2008).

Table 2.4.2.2. Population persistence categories from McElhany *et al.* (2006). A low or negligible risk of extinction is considered "viable" (Ford *et al.* 2011). Population persistence categories correspond to: 4 = very low (VL), 3 = low (L), 2 = moderate (M), 1 = high (H), and 0 = very high (VH) in Oregon populations, which corresponds to "extirpated or nearly so" (E) in Washington populations (Ford *et al.* 2011).

Population Persistence Category	Probability of population persistence in 100 years	Probability of population extinction in 100 years	Description
0	0-40%	60-100%	Either extinct or "high" risk of extinction
1	40-75%	25-60%	Relatively "high" risk of extinction in 100 years
2	75-95%	5-25%	"Moderate" risk of extinction in 100 years
3	95-99%	1-5%	"Low" (negligible) risk of extinction in 100 years
4	>99%	<1%	"Very low" risk of extinction in 100 years

Integrated SS/D risk combines risk for likely, future environmental conditions, and diversity (McElhany *et al.* 2000, McElhany *et al.* 2007, Ford *et al.* 2011). Diversity factors include:

- Life history traits: Distribution of major life history strategies within a population, variability of traits, mean value of traits, and loss of traits.
- Effective population size: One of the indirect measures of diversity is effective population size. A population at chronic low abundance or experiencing even a single episode of low abundance can be at higher extinction risk because of loss of genetic variability, inbreeding and the expression of inbreeding depression, or the effects of mutation accumulation.
- Impact of hatchery fish: Interbreeding of wild populations and hatchery origin fish can be a significant risk factor to the diversity of wild populations if the proportion of hatchery fish in the spawning population is high and their genetic similarity to the wild population is low.
- Anthropogenic mortality: The susceptibility to mortality from harvest or habitat alterations will differ depending on size, age, run timing, disease resistance or other traits.
- Habitat diversity: Habitat characteristics have clear selective effects on populations, and changes in habitat characteristics are likely to eventually lead to genetic changes through

selection for locally adapted traits. In assessing risk associated with altered habitat diversity, historical diversity is used as a reference point.

The boundaries of each population were defined using a combination of genetic information, geography, life-history traits, morphological traits, and population dynamics that indicate the extent of reproductive isolation among spawning groups. The overall viability of a species is a function of the VSP attributes of its constituent populations. Until a viability analysis of a species is completed, the VSP guidelines recommend that all populations should be managed to retain the potential to achieve viable status to ensure a rapid start along the road to recovery, and that no significant parts of the species are lost before a full recovery plan is implemented (McElhany *et al.* 2000).

The size and distribution of the species and their component populations considered in this opinion generally have declined over the last few decades due to natural phenomena and human activity, including climate change (as described in section 2.4.1), the operation of hydropower systems, over-harvest, effects of hatcheries, and habitat degradation. Enlarged populations of terns, seals, California sea lions, and other aquatic predators in the Pacific Northwest may be limiting the productivity of some Pacific salmon and steelhead populations (Ford *et al.* 2011).

Southern distinct population segment (DPS) green sturgeon (southern green sturgeon) occur in all coastal recovery domains, although they only spawn in the Sacramento River system. Therefore, only subadults and adults may be present in recovery domains north of San Francisco Bay. Southern DPS eulachon (eulachon) also occur in all coastal recovery domains. However, the status of these species will only be presented once, with information presented for the Willamette and Lower Columbia (WLC) recovery domain. Each species consist of a single population.

Viability status is described below for each of the populations considered in this opinion.

Southern Green Sturgeon. Two DPSs have been defined for green sturgeon (*Acipenser medirostris*), a northern DPS (spawning populations in the Klamath and Rogue rivers) and a southern DPS (spawners in the Sacramento River). There are no empirical data on population size and trends for green sturgeon in the Southern DPS. The estimated abundance (based on the percent of viable spawners) was 1,500 (NMFS 2010). Southern green sturgeon includes all naturally-spawned populations of green sturgeon that occur south of the Eel River in Humboldt County, California. When not spawning, this anadromous species is broadly distributed in nearshore marine areas from Mexico to the Bering Sea. Although it is commonly observed in bays, estuaries, and sometimes the deep riverine mainstem in lower elevation reaches of non-natal rivers along the west coast of North America, the distribution and timing of estuarine use are poorly understood.

Southern green sturgeon occur in the Willamette and Lower Columbia (WLC), Oregon Coast (OC), and Southern Oregon/Northern California Coasts (SONCC) recovery domains. The principal factor for the decline of southern green sturgeon is the reduction of its spawning area to a single known population limited to a small portion of the Sacramento River. It is currently at risk of extinction primarily because of human-induced ‘‘takes’’ involving elimination of

freshwater spawning habitat, degradation of freshwater and estuarine habitat quality, water diversions, fishing, and other causes (USDC 2010). Adequate water flow and temperature are issues of concern. Water diversions pose an unknown but potentially serious threat within the Sacramento and Feather Rivers and the Sacramento River Delta. Poaching also poses an unknown but potentially serious threat because of high demand for sturgeon caviar. The effects of contaminants and nonnative species are also unknown but potentially serious threats. Retention of green sturgeon in both recreational and commercial fisheries is now prohibited within the western states, but the effect of capture/release in these fisheries is unknown. There is evidence of fish being retained illegally, although the magnitude of this activity likely is small (NOAA Fisheries 2011).

The viability of this species is still under assessment.

Eulachon. The southern distinct population segment of eulachon occur in four salmon recovery domains: Puget Sound, the Willamette and Lower Columbia, Oregon Coast, and Southern Oregon/Northern California Coasts. The 5-year geometric mean abundance (2006-2010) for eulachon (based on converting fish landings per pound to numbers of fish at 10.8 fish per pound) was 879,669 (NMFS 2010a). The ESA-listed population of eulachon includes all naturally-spawned populations that occur in rivers south of the Nass River in British Columbia to the Mad River in California. Core populations for this species include the Fraser River, Columbia River and (historically) the Klamath River. Eulachon leave saltwater to spawn in their natal streams late winter through early summer, and typically spawn at night in the lower reaches of larger rivers fed by snowmelt. After hatching, larvae are carried downstream and widely dispersed by estuarine and ocean currents. Eulachon movements in the ocean are poorly known although the amount of eulachon bycatch in the pink shrimp fishery seems to indicate that the distribution of these organisms overlap in the ocean.

In the early 1990s, there was an abrupt decline in the abundance of eulachon returning to the Columbia River with no evidence of returning to their former population levels since then (Drake *et al.* 2008). Persistent low returns and landings of eulachon in the Columbia River from 1993 to 2000 prompted the states of Oregon and Washington to adopt a Joint State Eulachon Management Plan in 2001 that provides for restricted harvest management when parental run strength, juvenile production, and ocean productivity forecast a poor return (WDFW and ODFW 2001). Despite a brief period of improved returns in 2001–2003, the returns and associated commercial landings have again declined to the very low levels observed in the mid-1990s (JCRMS 2009), and since 2005, the fishery has operated at the most conservative level allowed in the management plan (JCRMS 2009). Large commercial and recreational fisheries have occurred in the Sandy River in the past. The most recent commercial harvest in the Sandy River was in 2003. No commercial harvest has been recorded for the Grays River from 1990 to the present, but larval sampling has confirmed successful spawning in recent years (USDC 2011a).

The primary factors responsible for the decline of the southern DPS of eulachon are changes in ocean conditions due to climate change (Gustafson *et al.* 2010, Gustafson *et al.* 2011), particularly in the southern portion of its range where ocean warming trends may be the most pronounced and may alter prey, spawning, and rearing success. Additional factors include climate-induced change to freshwater habitats, dams and water diversions (particularly in the

Columbia and Klamath Rivers where hydropower generation and flood control are major activities), and bycatch of eulachon in commercial fisheries (NOAA Fisheries 2011). Other limiting factors include (Gustafson *et al.* 2010, Gustafson *et al.* 2011):

- Adverse effects related to dams and water diversions
- Artificial fish passage barriers
- Increased water temperatures, insufficient streamflow
- Altered sediment balances
- Water pollution
- Over-harvest
- Predation

Willamette-Lower Columbia Recovery Domain. Species in the Willamette-Lower Columbia (WLC) Recovery Domain include LCR Chinook salmon, UWR Chinook salmon, CR chum salmon, LCR coho salmon, LCR steelhead, UWR steelhead, southern green sturgeon, and eulachon. The WLC-TRT has identified 107 demographically independent populations of Pacific salmon and steelhead (Table 2.4.2.3). These populations were further aggregated into strata, groupings above the population level that are connected by some degree of migration, based on ecological subregions. All 107 populations use parts of the mainstem of the Columbia River and the Columbia River estuary for migration, rearing, and smoltification.

Table 2.4.2.3. Populations in the WLC recovery domain.

Species	Populations
LCR Chinook salmon	32
UWR Chinook salmon	7
CR chum salmon	17
LCR coho salmon	24
LCR steelhead	26
UWR steelhead	4

LCR Chinook Salmon. This species includes all naturally-spawned populations of Chinook salmon in the Columbia River and its tributaries from its mouth at the Pacific Ocean upstream to a transitional point between Washington and Oregon east of the Hood River and the White Salmon River; the Willamette River to Willamette Falls, Oregon, exclusive of spring-run Chinook salmon in the Clackamas River; and progeny of seventeen artificial propagation programs. LCR Chinook populations exhibit three different life history types base on return timing and other features: fall-run (a.k.a. “tules”), late-fall-run (a.k.a. “brights”), and spring-run. The WLC-TRT identified 32 historical populations of LCR Chinook salmon; seven in the Coast Range, six in the Columbia Gorge, and 19 in the Cascade Range (Table 2.4.2.4). The 5-year geometric mean abundance for LCR Chinook salmon (2005-2009) was 31,305 total spawners (NOAA 2011, CBFWA 2011).

Table 2.4.2.4. LCR Chinook salmon strata, ecological subregions, run timing, populations, and scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH) in Oregon populations. VH corresponds to "extirpated or nearly so" (E) in Washington populations.

Stratum		Spawning Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Viability Risk
Ecological Subregion	**Run Timing**					
Coast Range	Fall	Grays River (WA)	E	E	L	E
		Elochoman River (WA)	E	H	L	E
		Mill, Germany, and Abernathy creeks (WA)	E	H	L	E
		Young Bay (OR)	H to VH	H	L	VH
		Big Creek (OR)	H to VH	H	L to M	VH
		Clatskanie River (OR)	H	M to H	L	VH
		Scappoose River (OR)	H to VH	M to H	L to M	VH
Columbia Gorge	Spring	White Salmon River (WA)	E	E	E	E
		Hood River (OR)	VH	VH	L	VH
	Fall	Upper Gorge (OR)	E	H	H	VH
		Upper Gorge (WA)	H to VH	H	L to M	E
		White Salmon River (WA)	E	H	H	E
		Lower Gorge (OR)	H to VH	H	L to M	VH
		Lower Gorge (WA)	E	H	H	E
		Hood River (OR)	H to VH	H to VH	L	VH
Cascade Range	Spring	Upper Cowlitz River (WA)	E	M	H	E
		Cispus River (WA)	E	M	H	E
		Tilton River (WA)	E	E	E	E
		Toutle River (WA)	E	H	L	E
		Kalama River (WA)	E	H	L	E
		Sandy River (OR)	M to H	L to M	M	M
		Lewis (WA)	E	M	H	E
	Fall	Lower Cowlitz River (WA)	E	M	M	E
		Upper Cowlitz River (WA)	E	M	E	E
		Lewis River (WA)	E	L	M	E
		Salmon Creek (OR)	E	M	M	E
		Sandy River (OR)	H to VH	H	L	VH
		Toutle River (WA)	E	M	M	E
		Coweeman River (WA)	E	L	M	E
		Kalama River (WA)	E	M	L	E
		Clackamas River (OR)	H to VH	H	L	H
		Washougal River (WA)	E	M	M	E
	Late Fall	Lewis River (WA)	VL	L	L	VL
		Sandy River (OR)	L	L to M	L	L

A/P ratings for most LCR Chinook salmon populations are currently "high" risk to "extirpated or nearly so." Spatial structure was generally rated "low" to "moderate" risk for most populations. Other than the Sandy River, Oregon LCR Chinook salmon populations were rated "high" or "very high" risk for diversity. In 2005, diversity risk for Clackamas River and Lower Gorge

tributary fall Chinook salmon was rated “moderate”; now the risk is rated “high.” Most Washington LCR Chinook salmon populations are currently at “moderate” or “high” risk for diversity (Table 2.4.2.4).

Of the 32 historical populations in the ESU, 28 are extirpated or at “very high” risk. Based on the recovery plan analyses, all of the tule populations are “very high” risk except one that is considered at “high” risk. The modeling conducted in association with tule harvest management suggests that three of the populations (Coweeman, Lewis and Washougal) are at a somewhat lower risk. However, even these more optimistic evaluations suggest that the remaining 18 populations are at substantial risk because of very low natural origin spawner abundance (<100/population), high hatchery fraction, habitat degradation and harvest impacts (Ford *et al.* 2011).

Limiting factors and threats to LCR Chinook salmon include (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and near-shore marine habitat resulting from cumulative impacts of land use and flow management by the Columbia River hydropower system Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas, stream substrate, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development.
- Reduced access to spawning and rearing habitat mainly as a result of tributary hydropower projects
- Hatchery-related effects
- Harvest-related effects on fall Chinook salmon
- An altered flow regime and Columbia River plume has altered the temperature regime and estuarine food web, and has reduced ocean productivity
- Reduced access to off-channel rearing habitat in the lower Columbia River
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Juvenile fish strandings that result from ship wakes
- Contaminants affecting fish health and reproduction

CR Chum Salmon. This species includes all naturally-spawned populations of chum salmon in the Columbia River and its tributaries in Washington and Oregon, and progeny of three artificial propagation programs. The WLC-TRT identified 17 historical populations of CR chum salmon and aggregated these into four strata (Myers *et al.* 2006; Table 2.4.2.5). Unlike other species in the WLC recovery domain, CR chum salmon spawning aggregations were identified in the mainstem Columbia River. These aggregations generally were included in the population associated with the nearest river basin. Three strata and eight historical populations of CR chum salmon occur within the action area (Table 2.4.2.5); of these, none are “viable” (McElhany *et al.* 2007). The 5-year geometric mean abundance for CR chum salmon (2005-2009) was 4,068 total spawners (NOAA 2011, CBFWA 2011).

Table 2.4.2.5. CR chum salmon strata, ecological subregions, run timing, populations, and scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk (Ford *et al.* 2011). Risk ratings are very low (VL), low (L), moderate (M), high (H), and "extirpated or nearly so" (E).

Stratum		Spawning Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Viability Risk
Ecological Subregion	**Run Timing**					
Coast Range	Fall	Young's Bay (OR)	*	*	*	*
		Grays River (WA)	VL	L	M	M
		Big Creek (OR)	*	*	*	*
		Elochoman River (WA)	E	E	L	E
		Clatskanie River (OR)	*	*	*	*
		Mill, Abernathy and Germany creeks (WA)	E	E	L	E
		Scappoose Creek (OR)	*	*	*	*
Columbia Gorge	Fall	Lower Gorge (OR)	*	*	*	*
		Lower Gorge (WA)	VL	VL	L	L
		Upper Gorge (OR)	*	*	*	*
		Upper Gorge (WA)	E	E	H	E
Cascade Range	Summer	Cowlitz River (WA)	E	E	H	E
	Fall	Cowlitz River (WA)	E	E	L	E
		Kalama River (WA)	E	E	L	E
		Salmon Creek (WA)	E	E	H	E
		Lewis River (WA)	E	E	L	E
		Clackamas River (OR)	*	*	*	*
		Washougal River (WA)	E	E	L	E
		Sandy River (OR)	*	*	*	*

* No viability risk was completed for Oregon chum salmon populations. Oregon rivers have occasional reports of a few chum salmon. Populations are functionally extinct, or the risk of extinction is very high.

The vast majority (14 out of 17) chum salmon populations remain "extirpated or nearly so". The Grays River and Lower Gorge populations showed a sharp increase in 2002, but have since declined back to relatively low abundance levels in the range of variation observed over the last several decades. Chinook and coho salmon populations in the Lower Columbia and Willamette similarly increased in the early 2000s, then declined to typical recent levels, suggesting the increase in chum salmon may be related to ocean conditions. The Grays and Lower Gorge populations were rated "very low" risk for A/P, but all other populations were rated "extirpated or nearly so." Spatial structure was rated "low" for seven populations, one was has moderate risk and three have a "high" risk. Diversity risk was "high" for all populations except Grays ("moderate") and Lower Gorge ("very low"). Recent data on the Washougal/mainstem Columbia population are not available, but they likely follow a pattern similar to the Grays and Lower Gorge populations (Ford *et al.* 2011).

Limiting factors and threats to CR chum salmon include (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and nearshore marine habitat resulting from cumulative impacts of land use and flow management by the Columbia River hydropower system
- Degraded freshwater habitat, in particular of floodplain connectivity and function, channel structure and complexity, stream substrate, and riparian areas and large wood recruitment as a result of cumulative impacts of agriculture, forestry, and development
- Degraded stream flow as a result of hydropower and water supply operations
- Loss of access and loss of some habitat types as a result of passage barriers such as roads and railroads
- Reduced water quality
- Current or potential predation from hatchery-origin salmonids, including coho salmon
- An altered flow regime and Columbia River plume has altered the temperature regime and estuarine food web, and has reduced ocean productivity
- Reduced access to off-channel rearing habitat in the lower Columbia River
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Juvenile fish strandings that result from ship wakes
- Contaminants affecting fish health and reproduction

LCR Coho Salmon. This species includes all naturally-spawned populations of coho salmon in the Columbia River and its tributaries in Washington and Oregon, from the mouth of the Columbia up to and including the Big White Salmon and Hood rivers; in the Willamette River to Willamette Falls, Oregon; and progeny of 25 artificial propagation programs. The WLC-TRT identified 24 historical populations of LCR coho salmon and divided these into two strata based on major run timing: early and late (Myers *et al.* 2006). Three strata and nine historical populations of LCR coho salmon occur within the action area (Table 2.4.2.6). Of these nine populations, Clackamas River is the only population characterized as "viable" (McElhany *et al.* 2007). The 5-year geometric mean abundance for LCR coho salmon (2004-2008) was 6,375 total spawners (NOAA 2011, CBFWA 2011).

Table 2.4.2.6. LCR coho salmon strata, ecological subregions, run timing, populations, and scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH) in Oregon populations. VH corresponds to "extirpated or nearly so" (E) in Washington populations.

Stratum		Spawning Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Viability Risk
Ecological Subregion	**Run Type**					
Coast Range	N*	Young's Bay (OR)	VH	VH	L	VH
		Big Creek (OR)	VH	H	L to M	VH
		Clatskanie River (OR)	H to VH	M	L	H
		Scappoose River (OR)	M to H	M	L to M	M
		Grays River (WA)	E	E	L	E
		Elochoman Creek (WA)	E	E	L	E
		Mill, Germany, and Abernathy Creeks (WA)	E	H	L	E
Columbia Gorge	N	Lower Gorge Tributaries (OR)	VH	H	L to M	VH
		Lower Gorge Tributaries (WA)	E	E	M	E
	S**	Upper Gorge Tributaries (WA)	E	E	M	E
		Hood River (OR)	VH	H	L	H
Cascade Range	N	Lower Cowlitz River (WA)	E	M	M	E
		Coweeman River (WA)	E	M	L	E
		Salmon Creek (WA)	E	E	M	E
	N and S	Upper Cowlitz River (WA)	E	H	M	E
		Cispus River (WA)	E	H	M	E
		Tilton River (WA)	E	H	M	E
		South Fork Toutle River (WA)	E	M	L	E
		North Fork Toutle River (WA)	E	H	M	E
		Kalama River (WA)	E	M	L	E
		North Fork Lewis River (WA)	E	H	H	E
		East Fork Lewis River (WA)	E	M	L	E
		Washougal River (WA)	E	H	L	E
		Clackamas River (OR)	M	L to M	L	M
		Sandy River (OR)	H	L to M	M to H	H

*"Type N" are late-run fish that tend to undertake oceanic migrations to the north of the Columbia River, extending as far as northern British Columbia and southeast Alaska.
**"Type S" are early coho salmon that spawn in the upper reaches of larger rivers in the lower Columbia River and in most rivers inland of the Cascade Crest that tend to migrate to the south of the Columbia River.

Three status evaluations of LCR coho salmon status, all based on WLC-TRT criteria, have been conducted since the last NMFS status review in 2005 (McElhany *et al.* 2007, Beamesderfer *et al.* 2010, LCFRB 2010). Of the 27 historical populations in the ESU, 24 are at "very high" risk. The remaining three populations (Sandy, Clackamas and Scappoose) are at "moderate" or "high" risk (Ford *et al.* 2011).

In Oregon, the Scappoose Creek and Clackamas River populations have "moderate" risk ratings for A/P, while the rest are rated "high" or "very high" risk. All of the Washington populations have "extirpated or nearly so" A/P ratings. Spatial diversity is rated "moderate" or "low" risk for all the populations, except the North Fork Lewis River, which has a "high" risk rating for spatial structure. All LCR coho salmon populations, except the Clackamas and Sandy river populations (low risk), are at "moderate" or "high" risk for diversity. All of the Washington side populations are at "very high" risk, although uncertainty is high because of a lack of adult spawner surveys. As was noted in the 2005 status review, smolt traps indicate some natural production in Washington populations, though given the high fraction of hatchery origin spawners suspected to occur in these populations it is not clear that any are self-sustaining (Ford *et al.* 2011).

Limiting factors and threats to LCR coho salmon include (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and near-shore marine habitat resulting from cumulative impacts of land use and flow management by the Columbia River hydropower system
- Fish passage barriers that limit access to spawning and rearing habitats
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Hatchery-related effects
- Harvest-related effects
- An altered flow regime and Columbia River plume has altered the temperature regime and estuarine food web, and has reduced ocean productivity
- Reduced access to off-channel rearing habitat in the lower Columbia River
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Juvenile fish strandings that result from ship wakes
- Contaminants affecting fish health and reproduction

LCR Steelhead. This species includes all naturally-spawned steelhead populations below natural and manmade impassable barriers in streams and tributaries to the Columbia River between and including the Cowlitz and Wind rivers, Washington; in the Willamette and Hood rivers, Oregon; and progeny of ten artificial propagation programs; but excluding all steelhead from the upper Willamette River basin above Willamette Falls, Oregon, and from the Little and Big White Salmon rivers, Washington. Summer steelhead return to freshwater long before spawning. Winter steelhead, in contrast, return from the ocean much closer to maturity and spawn within a few weeks. Summer steelhead spawning areas in the Lower Columbia River are found above waterfalls and other features that create seasonal barriers to migration. Where no temporal barriers exist, the winter-run life history dominates. Six strata and 23 historical populations of LCR steelhead occur within the action area (Table 2.4.2.7). The 5-year geometric mean abundance for LCR steelhead (2006-2010) was 5,863 total spawners (NOAA 2011, CBFWA 2011).

Table 2.4.2.7. LCR steelhead strata, ecological subregions, run timing, populations, and scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH) in Oregon populations. VH corresponds to "extirpated or nearly so" (E) in Washington populations.

Stratum		Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Viability Risk
Ecological Subregion	**Run Timing**					
Columbia Gorge	Summer	Wind River (WA)	VL	L	VL	L
		Hood River (OR)	H	M	L	VH
	Winter	Lower Gorge (OR)	H	L	L	M to H
		Lower Gorge (WA)	H	M	VL	H
		Upper Gorge (OR)	M	M to H	L	VH
		Upper Gorge (WA)	H	M	M	E
		Hood River (OR)	M	M	L	M
West Cascade Range	Summer	Kalama River (WA)	L	M	VL	M
		North Fork Lewis River (WA)	E	E	E	E
		East Fork Lewis River (WA)	E	M	VL	E
		Washougal River (WA)	M	M	VL	M
	Winter	Cispus River (WA)	E	M	M	E
		Tilton river (WA)	E	H	M	E
		Upper Cowlitz River (WA)	E	M	M	E
		Lower Cowlitz River (WA)	H	M	M	H
		North Fork Toutle River (WA)	E	L	L	E
		South Fork Toutle River (WA)	M	L	VL	M
		Coweeman River (WA)	H	VL	VL	H
		Kalama River (WA)	H	L	VL	H
		North Fork Lewis River (WA)	E	M	M	E
		East Fork Lewis River (WA)	M	M	VL	M
		Salmon Creek (WA)	E	M	VL	E
		Washougal River (WA)	H	M	VL	H
		Sandy River (OR)	H	M	M to H	VH
		Clackamas River (OR)	L	L to M	L	L to M

All of the populations increased in abundance during the early 2000s, generally peaking in 2004. Most populations have since declined back to levels within one standard deviation of the long term mean. Exceptions are the Washougal summer-run and North Fork Toutle winter-run, which are still higher than the long term average, and the Sandy, which is lower (Ford *et al.* 2011).

Limiting factors and threats to LCR steelhead include (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and nearshore marine habitat resulting from cumulative impacts of land use and flow management by the Columbia River hydropower system
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and recruitment of large wood, stream substrate, stream flow,

and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development

- Reduced access to spawning and rearing habitat mainly as a result of tributary hydropower projects and lowland development
- Avian and marine mammal predation in the lower mainstem Columbia River and estuary.
- Hatchery-related effects
- An altered flow regime and Columbia River plume has altered the temperature regime and estuarine food web, and has reduced ocean productivity
- Reduced access to off-channel rearing habitat in the lower Columbia River
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Juvenile fish strandings that result from ship wakes
- Contaminants affecting fish health and reproduction

UWR Chinook Salmon. This species includes all naturally spawned populations of spring-run Chinook salmon in the Clackamas River; in the Willamette River and its tributaries above Willamette Falls, Oregon; and progeny of seven artificial propagation programs. All seven historical populations of UWR Chinook salmon identified by the WLC-TRT occur within the action area and are contained within a single ecological subregion, the western Cascade Range (Table 2.4.2.8); only the Clackamas population is characterized as "viable" (McElhany *et al.* 2007). The 5-year geometric mean abundance for UWR spring Chinook salmon (2004-2008) was 4,177 total spawners (NOAA 2011, CBFWA 2011).

Table 2.4.2.8. Scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk for UWR Chinook salmon (ODFW and NMFS 2011). All populations are in the Western Cascade Range ecological subregion. Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH).

Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Extinction Risk
Clackamas River	M	M	L	M
Molalla River	VH	H	H	VH
North Santiam River	VH	H	H	VH
South Santiam River	VH	M	M	VH
Calapooia River	VH	H	VH	VH
McKenzie River	VL	M	M	L
Middle Fork Willamette River	VH	H	H	VH

Consideration of data collected since the last status review in 2005 has confirmed the high fraction of hatchery origin fish in all of the populations of this species (even the Clackamas and McKenzie rivers have hatchery fractions above WLC-TRT viability thresholds). All of the UWR Chinook salmon populations have "moderate" or "high" risk ratings for diversity. The Clackamas and McKenzie river populations currently have the best risk ratings for A/P, spatial structure, and diversity. Clackamas River Chinook salmon have a "low" risk rating for spatial structure.

The new data have also highlighted the substantial risks associated with pre-spawning mortality. Although recovery plans are targeting key limiting factors for future actions, there have been no significant on-the-ground-actions since the last status review to resolve the lack of access to historical habitat above dams nor have there been substantial actions removing hatchery fish from the spawning grounds (Ford *et al.* 2011).

Limiting factors and threats to UWR Chinook salmon include (ODFW and NMFS 2011, NOAA Fisheries 2011):

- Significantly reduced access to spawning and rearing habitat because of tributary dams
- Degraded freshwater habitat, especially floodplain connectivity and function, channel structure and complexity, and riparian areas and large wood recruitment as a result of cumulative impacts of agriculture, forestry, and development
- Degraded water quality and altered temperature as a result of both tributary dams and the cumulative impacts of agriculture, forestry, and urban development
- Hatchery-related effects
- Anthropogenic introductions of non-native species and out-of-ESU races of salmon or steelhead have increased predation on, and competition with, native UWR Chinook salmon
- Ocean harvest rates of approximately 20%

UWR Steelhead. This species includes all naturally-spawned steelhead populations below natural and manmade impassable barriers in the Willamette River, Oregon, and its tributaries upstream from Willamette Falls to the Calapooia River. The WLC-TRT identified five historical populations of UWR steelhead, all with winter-run timing (Myers *et al.* 2006). UWR steelhead are currently found in many tributaries that drain the west side of the upper Willamette River basin. Analysis of historical observations, hatchery records, and genetic analysis strongly suggested that many of these spawning aggregations are the result of recent introductions and do not represent a historical population. Nevertheless, the WLC-TRT recognized that these tributaries may provide juvenile rearing habitat or may be temporarily (for one or more generations) colonized during periods of high abundance. One stratum[4] and five historical populations of UWR steelhead occur within the action area (Table 2.4.2.9), although the west-side tributaries population was included only because it is important to the species as a whole, and not because it is independent. Summer steelhead have become established in the McKenzie River where historically no steelhead existed, although these fish were not considered in the identification of historical populations. Hatchery summer-run steelhead that are produced and released in the subbasins are from an out-of-basin stock and are not part of the DPS (ODFW and NMFS 2011). The 5-year geometric mean abundance for UWR steelhead (2004-2008) was 6,392 total spawners (NOAA 2011, CBFWA 2011).

[4] The WLC-TRT defined the hierarchy by grouping the independent populations into larger aggregates that share similar genetic, geographic (hydrographic and ecoregion), and/or habitat characteristics. They called these "major groupings" stratum (plural: strata).

Table 2.4.2.9. Scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk for UWR steelhead (ODFW and NMFS 2011). All populations are in the Western Cascade Range ecological subregion. Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH).

Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Extinction Risk
Molalla River	VL	M	M	L
North Santiam River	VL	M	H	L
South Santiam River	VL	M	M	L
Calapooia River	M	M	VH	M

Since the last status review in 2005, UWR steelhead initially increased in abundance but subsequently declines and current abundance is at the levels observed in the mid-1990s when the DPS was first listed. The DPS appears to be at lower risk than the UWR Chinook salmon ESU, but continues to demonstrate the overall low abundance pattern that was of concern during the last status review. The elimination of winter-run hatchery release in the basin reduces hatchery threats, but non-native summer steelhead hatchery releases are still a concern for species diversity (Ford *et al.* 2011).

Limiting factors and threats to UWR steelhead include (ODFW and NMFS 2011, NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large wood recruitment, and stream flow have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Degraded water quality and altered temperature as a result of both tributary dams and the cumulative impacts of agriculture, forestry, and urban development
- Reduced access to spawning and rearing habitats mainly as a result of artificial barriers in spawning tributaries
- Hatchery-related effects: impacts from the non-native summer steelhead hatchery program
- Anthropogenic introductions of non-native species and out-of-ESU races of salmon or steelhead have increased predation and competition on native UWR steelhead.

Interior Columbia Recovery Domain. Species in the Interior Columbia (IC) recovery domain include UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, SR sockeye salmon, UCR steelhead, MCR steelhead, and SRB steelhead. The IC-TRT identified 82 populations of those species based on genetic, geographic (hydrographic), and habitat characteristics (Table 2.4.2.10). In some cases, the IC-TRT further aggregated populations into “major groupings” based on dispersal distance and rate, and drainage structure, primarily the location and distribution of large tributaries (IC-TRT 2003). All 82 populations identified use the lower mainstem of the Snake River, the mainstem of the Columbia River, and the Columbia River estuary, or part thereof, for migration, rearing, and smoltification.

Table 2.4.2.10. Populations of ESA-listed salmon and steelhead in the IC recovery domain.

Species	Populations
UCR spring-run Chinook salmon	3
SR spring/summer-run Chinook salmon	31
SR fall-run Chinook salmon	1
SR sockeye salmon	1
UCR steelhead	4
MCR steelhead	17
SRB steelhead	25

The IC-TRT also recommended viability criteria that follow the VSP framework (McElhany *et al.* 2006) and described biological or physical performance conditions that, when met, indicate a population or species has a 5% or less risk of extinction over a 100-year period (IC-TRT 2007; see also NRC 1995).

UCR Spring-run Chinook Salmon. This species includes all naturally-spawned populations of Chinook salmon in all river reaches accessible to Chinook salmon in Columbia River tributaries upstream of the Rock Island Dam and downstream of Chief Joseph Dam in Washington (excluding the Okanogan River), the Columbia River upstream to Chief Joseph Dam in Washington, and progeny of six artificial propagation programs. The IC-TRT identified four independent populations of UCR spring-run Chinook salmon in the upriver tributaries of Wenatchee, Entiat, Methow, and Okanogan (extirpated), but no major groups due to the relatively small geographic area affected (IC-TRT 2003, Ford *et al.* 2011)(Table 2.4.2.11). The 5-year geometric mean abundance for UCR spring-run Chinook salmon (2005-2009) was 3,134 total spawners (NOAA 2011, CBFWA 2011). The current estimate (2003-2008 5-year average) of natural origin spawning abundance ranges from 29% to 46% across populations.

Table 2.4.2.11. Scores for the key elements (A/P, diversity, and SS/D) used to determine current overall viability risk for spring-run UCR Chinook salmon (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH).

Population	A/P	Diversity	Integrated SS/D	Overall Viability Risk
Wenatchee River	H	H	H	H
Entiat River	H	H	H	H
Methow River	H	H	H	H
Okanogan River	n/a	n/a	n/a	n/a

TUCR spring-run Chinook salmon is not currently meeting the viability criteria (adapted from the IC-TRT) in the Upper Columbia recovery plan. A/P remains at “high” risk for each of the three extant populations in this MPG/ESU (Table 2.4.2.11). The 10-year geometric mean abundance of adult natural origin spawners has increased for each population relative to the levels for the 1981-2003 series, but the estimates remain below the corresponding IC-TRT thresholds. Estimated productivity (spawner to spawner return rate at low to moderate

escapements) was on average lower over the years 1987-2009 than for the previous period. The combinations of current abundance and productivity for each population result in a "high" risk rating. The composite SS/D risks for all three of the extant populations in this MPG are at "high" risk. The spatial processes component of the SS/D risk is "low" for the Wenatchee River and Methow River populations and "moderate" for the Entiat River (loss of production in lower section increases effective distance to other populations). All three of the extant populations in this MPG are at "high" risk for diversity, driven primarily by chronically high proportions of hatchery-origin spawners in natural spawning areas and lack of genetic diversity among the natural-origin spawners (Ford *et al.* 2011).

Increases in natural origin abundance relative to the extremely low spawning levels observed in the mid-1990s are encouraging; however, average productivity levels remain extremely low. Overall, the viability of UCR Chinook salmon has likely improved somewhat since the last status review, but the ESU is still clearly at "moderate-to-high" risk of extinction (Ford *et al.* 2011).

Limiting factors and threats to the UCR spring-run Chinook salmon ESU include (UCSRB 2007, NOAA Fisheries 2011):

- Mainstem Columbia River hydropower–related adverse effects: upstream and downstream fish passage, ecosystem structure and function, flows, and water quality
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Degraded estuarine and nearshore marine habitat
- Hatchery related effects: including past introductions and persistence of non-native (exotic) fish species continues to affect habitat conditions for listed species
- Harvest in Columbia River fisheries

SR Spring/summer-run Chinook Salmon. This species includes all naturally-spawned populations of spring/summer-run Chinook salmon in the mainstem Snake River and the Tucannon River, Grande Ronde River, Imnaha River, and Salmon River subbasins; and progeny of fifteen artificial propagation programs. The IC-TRT identified 27 extant and 4 extirpated populations of SR spring/summer-run Chinook salmon, and aggregated these into major population groups (IC-TRT 2003, Ford *et al.* 2011). Each of these populations faces a "high" risk of extinction (Ford *et al.* 2011) (Table 2.4.2.12). The 5-year geometric mean abundance for SR Spring/Summer Chinook salmon (2005-2009) was 6,365 total spawners (Ford *et al.* 2011). The current estimate (2005-2009 5-year average) of natural origin spawning abundance ranges from 25% to 100% across populations.

Table 2.4.2.12. SR spring/summer-run Chinook salmon ecological subregions, populations, and scores for the key elements (A/P, diversity, and SS/D) used to determine current overall viability risk for SR spring/summer-run Chinook salmon (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH) and extirpated (E).

Ecological Subregions	Spawning Populations (Watershed)	A/P	Diversity	Integrated SS/D	Overall Viability Risk
Lower Snake River	Tucannon River	H	M	M	H
	Asotin River				E
Grande Ronde and Imnaha rivers	Wenaha River	H	M	M	H
	Lostine/Wallowa River	H	M	M	H
	Minam River	H	M	M	H
	Catherine Creek	H	M	M	H
	Upper Grande Ronde R.	H	M	H	H
	Imnaha River	H	M	M	H
	Big Sheep Creek				E
	Lookingglass Creek				E
South Fork Salmon River	Little Salmon River	*	*	*	H
	South Fork mainstem	H	M	M	H
	Secesh River	H	L	L	H
	EF/Johnson Creek	H	L	L	H
Middle Fork Salmon River	Chamberlin Creek	H	L	L	H
	Big Creek	H	M	M	H
	Lower MF Salmon	H	M	M	H
	Camas Creek	H	M	M	H
	Loon Creek	H	M	M	H
	Upper MF Salmon	H	M	M	H
	Pistol Creek				E
	Sulphur Creek	H	M	M	H
	Bear Valley Creek	H	L	L	H
	Marsh Creek	H	L	L	H
Upper Mainstem Salmon	N. Fork Salmon River	H	L	L	H
	Lemhi River	H	H	H	H
	Pahsimeroi River	H	H	H	H
	Upper Salmon-lower mainstem	H	L	L	H
	East Fork Salmon River	H	H	H	H
	Yankee Fork	H	H	H	H
	Valley Creek	H	M	M	H
	Upper Salmon main	H	M	M	H
	Panther Creek				E
* Insufficient data.					

Population level status ratings remain at high risk across all MPGs within the ESU, although recent natural spawning abundance estimates have increased, all populations remain below

minimum natural origin abundance thresholds (Table 2.4.2.12). Spawning escapements in the most recent years in each series are generally well below the peak returns but above the extreme low levels in the mid-1990s. Relatively low natural production rates and spawning levels below minimum abundance thresholds remain a major concern across the ESU.

The ability of SR spring/summer-run Chinook salmon populations to be self-sustaining through normal periods of relatively low ocean survival remains uncertain. Factors cited by Good *et al.* (2005) remain as concerns or key uncertainties for several populations (Ford *et al.* 2011). Limiting factors and threats to the SR spring/summer-run Chinook salmon ESU include (NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, elevated water temperature, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Mainstem Columbia River and Snake River hydropower impacts
- Harvest-related effects
- Predation

SR Fall-run Chinook Salmon. This species includes all naturally-spawned populations of fall-run Chinook salmon in the mainstem Snake River below Hells Canyon Dam, and in the Tucannon River, Grande Ronde River, Imnaha River, Salmon River, and Clearwater River, and progeny of four artificial propagation programs. The IC-TRT identified three populations of this species, although only the lower mainstem population exists at present, and it spawns in the lower main stem of the Clearwater, Imnaha, Grande Ronde, Salmon and Tucannon rivers. The extant population of Snake River fall-run Chinook salmon is the only remaining population from an historical ESU that also included large mainstem populations upstream of the current location of the Hells Canyon Dam complex (IC-TRT 2003, Ford *et al.* 2011). The 5-year geometric mean abundance for SR fall-run Chinook salmon (2004-2008) was 11,321 total spawners. The current estimate (1999-2008 10-year geometric mean) of natural origin spawning abundance of SR fall-run Chinook is just over 2,200 (Ford *et al.* 2011).

The recent increases in natural origin abundance are encouraging. However, hatchery origin spawner proportions have increased dramatically in recent years – on average, 78% of the estimated adult spawners have been hatchery origin over the most recent brood cycle. The apparent leveling off of natural returns in spite of the increases in total brood year spawners may indicate that density dependent habitat effects are influencing production or that high hatchery proportions may be influencing natural production rates. The A/P risk rating for the population is "moderate." The population is at moderate risk for diversity and spatial structure. (Ford *et al.* 2011). Given the combination of current A/P and SS/D ratings summarized above, the overall viability rating for Lower SR fall Chinook salmon would be rated as "maintained."[5]

[5] "Maintained" population status is for populations that do not meet the criteria for a viable population but do support ecological functions and preserve options for ESU/DPS recovery.

Limiting factors and threats to SR fall-run Chinook salmon include (NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, and channel structure and complexity have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Harvest-related effects
- Lost access to historic habitat above Hells Canyon and other Snake River dams
- Mainstem Columbia River and Snake River hydropower impacts
- Hatchery-related effects
- Degraded estuarine and nearshore habitat

SR Sockeye Salmon. This species includes all anadromous and residual sockeye salmon from the Snake River basin, Idaho, and artificially-propagated sockeye salmon from the Redfish Lake captive propagation program. The IC-TRT identified historical sockeye salmon production in at least five Stanley Basin and Sawtooth Valley lakes and in lake systems associated with Snake River tributaries currently cut off to anadromous access (*e.g.,* Wallowa and Payette Lakes), although current returns of SR sockeye salmon are extremely low and limited to Redfish Lake (IC-TRT 2007). The 5-year geometric mean abundance for SR sockeye salmon (2005-2009) was 166 total spawners (NOAA 2011, CBFWA 2011).

This species is still at extremely high risk across all four basic risk measures (abundance, productivity, spatial structure and diversity. Although the captive brood program has been successful in providing substantial numbers of hatchery produced *O. nerka* for use in supplementation efforts, substantial increases in survival rates across life history stages must occur in order to re-establish sustainable natural production (Hebdon *et al.* 2004, Keefer *et al.* 2008).

The key factor limiting recovery of SR sockeye salmon ESU is survival outside of the Stanley Basin. Portions of the migration corridor in the Salmon River are impeded by water quality and temperature (Idaho Department of Environmental Quality 2011). Increased temperatures may reduce the survival of adult sockeye returning to the Stanley Basin. The natural hydrological regime in the upper mainstem Salmon River Basin has been altered by water withdrawals. In most years, sockeye adult returns to Lower Granite suffer catastrophic losses (*e.g.*, > 50% mortality in one year; Reed *et al.* 2003) before reaching the Stanley Basin, although the factors causing these losses have not been identified. In the Columbia and lower Snake River migration corridor, predation rates on juvenile sockeye salmon are unknown, but terns and cormorants consume 12% of all salmon smolts reaching the estuary, and piscivorous fish consume an estimated 8% of migrating juvenile salmon (NOAA Fisheries 2011).

MCR Steelhead. This species includes all naturally-spawned steelhead populations below natural and artificial impassable barriers in streams from above the Wind River, Washington, and the Hood River, Oregon (exclusive), upstream to, and including, the Yakima River, Washington, excluding steelhead from the Snake River basin; and progeny of seven artificial propagation programs. The IC-TRT identified 17 extant populations in this DPS (IC-TRT 2003). The populations fall into four major population groups: the Yakima River Basin (four extant populations), the Umatilla/Walla-Walla drainages (three extant and one extirpated populations);

the John Day River drainage (five extant populations) and the Eastern Cascades group (five extant and two extirpated populations) (Table 2.4.2.13) (NMFS 2009, Ford *et al.* 2011). The 5-year geometric mean abundance for MCR steelhead (2006-2010) was 15,723 total spawners (NOAA 2011, CBFWA 2011). The current estimate (2005-2009 5-year average) of natural origin spawning abundance ranges from 70% to 97% across populations.

Table 2.4.2.13. Ecological subregions, populations, and scores for the key elements (A/P, diversity, and SS/D) used to determine current overall viability risk for MCR steelhead (NMFS 2009, Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH). Maintained (MT) population status indicates that the population does not meet the criteria for a viable population but does support ecological functions and preserve options for recovery of the DPS.

Ecological Subregions	**Population (Watershed)**	**A/P**	**Diversity**	**Integrated SS/D**	**Overall Viability Risk**
Cascade Eastern Slope Tributaries	Fifteenmile Creek	L	L	L	Viable
	Klickitat River	M	M	M	MT?
	Eastside Deschutes River	L	M	M	Viable
	Westside Deschutes River	H	M	M	H*
	Rock Creek	H	M	M	H?
	White Salmon	Extinct	n/a	n/a	Extinct*
	Crooked River	Extinct	n/a	n/a	Extinct*
John Day River	Upper Mainstem	M	M	M	MT
	North Fork	VL	L	L	Highly Viable
	Middle Fork	M	M	M	MT
	South Fork	M	M	M	MT
	Lower Mainstem	M	M	M	MT
Walla Walla and Umatilla rivers	Umatilla River	M	M	M	MT
	Touchet River	M	M	M	H
	Walla Walla River	M	M	M	MT
Yakima River	Satus Creek	M	M	M	Viable (MT)
	Toppenish Creek	M	M	M	Viable (MT)
	Naches River	H	M	M	H
	Upper Yakima	H	H	H	H

* Re-introduction efforts underway (NMFS 2009).

There have been improvements in the viability ratings for some of the component populations, but the MCR steelhead DPS is not currently meeting the viability criteria (adopted from the IC-TRT) in the MCR steelhead recovery plan (NMFS 2009). In addition, several of the factors cited by Good *et al.* (2005) remain as concerns or key uncertainties. Natural origin spawning estimates of populations have been highly variable with respect to meeting minimum abundance thresholds. Straying frequencies into at least the Lower John Day River population are high. Returns to the Yakima River basin and to the Umatilla and Walla Walla Rivers have been higher

over the most recent brood cycle, while natural origin returns to the John Day River have decreased. Out-of-basin hatchery stray proportions, although reduced, remain very high in the Deschutes River basin (Ford *et al.* 2011).

The limiting factors and threats to MCR steelhead include (NMFS 2009, NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas, fish passage, stream substrate, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, tributary hydro system activities, and development
- Mainstem Columbia River hydropower–related impacts
- Degraded estuarine and nearshore marine habitat
- Hatchery-related effects
- Harvest-related effects
- Effects of predation, competition, and disease

UCR Steelhead. This species includes all naturally-spawned steelhead populations below natural and manmade impassable barriers in streams in the Columbia River Basin upstream from the Yakima River, Washington, to the U.S.-Canada border, and progeny of six artificial propagation programs. Four independent populations of UCR steelhead were identified by the IC-TRT in the same upriver tributaries as for UC spring-run Chinook salmon (*i.e.*, Wenatchee, Entiat, Methow, and Okanogan; Table 2.4.2.14) and, similarly, no major population groupings were identified due to the relatively small geographic area involved (IC-TRT 2003, Ford *et al.* 2011). All extant populations are considered to be at high risk of extinction (Table 22; Ford *et al.* 2011). The 5-year geometric mean abundance for UCR steelhead (2005-2009) was 7,884 total spawners (Ford *et al.* 2011). The current estimate (2003-2008 5-year average) of natural origin spawning abundance ranges from 9% to 47% across populations.

Table 2.4.2.14. Summary of the key elements (A/P, diversity, and SS/D) and scores used to determine current overall viability risk for UCR steelhead populations (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH).

Population (Watershed)	A/P	Diversity	Integrated SS/D	Overall Viability Risk
Wenatchee River	H	H	H	H
Entiat River	H	H	H	H
Methow River	H	H	H	H
Okanogan River	H	H	H	H

UCR steelhead populations have increased in natural origin abundance in recent years, but productivity levels remain low. The proportions of hatchery origin returns in natural spawning areas remain extremely high across the DPS, especially in the Methow and Okanogan River populations. The modest improvements in natural returns in recent years are probably primarily the result of several years of relatively good natural survival in the ocean and tributary habitats.

With the exception of the Okanogan population, the Upper Columbia populations rated as "low" risk for spatial structure. The "high" risk ratings for SS/D are largely driven by chronic high levels of hatchery spawners within natural spawning areas and lack of genetic diversity among the populations (Ford *et al.* 2011).

The limiting factors and threats to the UCR steelhead DPS include (UCSRB 2007, NOAA Fisheries 2011):

- Mainstem Columbia River hydropower–related adverse effects.
- Impaired tributary fish passage.
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development.
- Effects of predation, competition, and disease mortality: Fish management, including past introductions and persistence of non-native (exotic) fish species continues to affect habitat conditions for listed species.
- Hatchery-related effects.
- Harvest-related effects.

SRB Steelhead. This species includes all naturally-spawned steelhead populations below natural and manmade impassable barriers in streams in the Snake River Basin of southeast Washington, northeast Oregon, and Idaho, and progeny of six artificial propagation programs. The IC-TRT identified 25 historical populations in five major groups (Table 2.4.2.15) (IC-TRT 2006, Ford *et al.* 2011). The IC-TRT has not assessed the viability of this species. The 5-year geometric mean abundance for SRB steelhead (2005-2009) was 3,546 total spawners (NOAA 2011, CBFWA 2011).

The level of natural production in the two populations with full data series and the Asotin Creek index reaches is encouraging, but the status of most populations in this DPS remains highly uncertain. Population-level natural origin abundance and productivity inferred from aggregate data and juvenile indices indicate that many populations are likely below the minimum combinations defined by the IC-TRT viability criteria. The relative proportion of hatchery fish in natural spawning areas near major hatchery release sites is highly uncertain. There is little evidence for substantial change in ESU viability relative to the previous BRT and IC-TRT reviews (Ford *et al.* 2011).

Limiting factors and threats to the SRB steelhead DPS include (IC-TRT 2006, NOAA Fisheries 2011):

- Mainstem Columbia River hydropower–related adverse effects
- Impaired tributary fish passage
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development

- Impaired water quality and increased water temperature
- Related harvest effects, particularly for B-run steelhead
- Predation
- Genetic diversity effects from out-of-population hatchery releases

Table 2.4.2.15. Ecological subregions, populations, and scores for the key elements (A/P, diversity, and SS/D) used to determine current overall viability risk for SRB steelhead (Ford *et al.* 2011, NMFS 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH). Maintained (MT) population status indicates that the population does not meet the criteria for a viable population but does support ecological functions and preserve options for recovery of the DPS.

Ecological subregions	Spawning Populations (Watershed)	A/P	Diversity	Integrated SS/D	Overall Viability Risk*
Lower Snake River	Tucannon River	**	M	M	H
	Asotin Creek	**	M	M	MT
Grande Ronde River	Lower Grande Ronde	**	M	M	Not rated
	Joseph Creek	VL	L	L	Highly viable
	Upper Grande Ronde	M	M	M	MT
	Wallowa River	**	L	L	H
Clearwater River	Lower Clearwater	M	L	L	MT
	South Fork Clearwater	H	M	M	H
	Lolo Creek	H	M	M	H
	Selway River	H	L	L	H
	Lochsa River	H	L	L	H
Salmon River	Little Salmon River	**	M	M	MT
	South Fork Salmon	**	L	L	H
	Secesh River	**	L	L	H
	Chamberlain Creek	**	L	L	H
	Lower MF Salmon	**	L	L	H
	Upper MF Salmon	**	L	L	H
	Panther Creek	**	M	H	H
	North Fork Salmon	**	M	M	MT
	Lemhi River	**	M	M	MT
	Pahsimeroi River	**	M	M	MT
	East Fork Salmon	**	M	M	MT
	Upper Main Salmon	**	M	M	MT
Imnaha	Imnaha River	M		M	MT

* There is uncertainty in these ratings due to a lack of population-specific data.

** Insufficient data.

Oregon Coast Recovery Domain. The OC recovery domain includes OC coho salmon, southern green sturgeon, and eulachon, covering Oregon coastal streams south of the Columbia River and north of Cape Blanco. Streams and rivers in this area drain west into the Pacific Ocean, and vary in length from less than a mile to more than 210 miles in length.

OC Coho Salmon. This species includes all naturally-spawned populations of coho salmon in Oregon coastal streams south of the Columbia River and north of Cape Blanco, including the Cow Creek population, which is stock #37 of Oregon Department of Fish and Wildlife's (ODFW) coho hatchery program. OC Coho salmon were first listed in February 2008. As part of a legal settlement agreement in 2008, NMFS completed a new status review for the ESU. In 2011, NMFS issued a final rule re-promulgating the threatened listing for Oregon Coast coho salmon (USDC 2011b).

The OC-TRT identified 56 populations — 21 independent and 35 dependent. The dependent populations were dependent on strays from other populations to maintain them over long time periods. The TRT also identified 5 biogeographic strata (Table 2.4.2.16) (Lawson *et al.* 2007). The 5-year geometric mean abundance for OC coho salmon (2006-2010) was 162,769 total spawners (ODFW 2011).

Table 2.4.2.16. OC coho salmon populations. Dependent populations (D) are populations that historically would not have had a high likelihood of persisting in isolation for 100 years. These populations relied upon periodic immigration from other populations to maintain their abundance. Independent populations are populations that historically would have had a high likelihood of persisting in isolation from neighboring populations for 100 years and are rated as functionally independent (FI) and potentially independent (PI) (McElhany *et al.* 2000, Lawson *et al.* 2007).

Stratum	Population	Type	Stratum	Population	Type
North Coast	Necanicum	PI	Mid-Coast (cont.)	Alsea	FI
	Ecola	D		Big (Alsea)	D
	Arch Cape	D		Vingie	D
	Short Sands	D		Yachats	D
	Nehalem	FI		Cummins	D
	Spring	D		Bob	D
	Watseco	D		Tenmile	D
	Tillamook	FI		Rock	D
	Netarts	D		Big (Siuslaw)	D
	Rover	D		China	D
	Sand	D		Cape	D
	Nestucca	FI		Berry	D
	Neskowin	D		Sutton	D
Mid-Coast	Salmon	PI	Lakes	Siuslaw	FI
	Devils	D		Siltcoos	PI
	Siletz	FI		Tahkenitch	PI
	Schoolhouse	D		Tenmile	PI
	Fogarty	D	Umpqua	Lower Umpqua	FI
	Depoe	D		Middle Umpqua	FI
	Rocky	D		North Umpqua	FI
	Spencer	D		South Umpqua	FI
	Wade	D	Mid-South Coast	Threemile	D
	Coal	D		Coos	FI
	Moolack	D		Coquille	FI
	Big (Yaquina)	D		Johnson	D
	Yaquina	FI		Twomile	D
	Theil	D		Floras	PI
	Beaver	PI		Sixes	PI

Wainwright *et al.* (2008) determined that the weakest strata of OC coho salmon were in the North Coast and Mid-Coast of Oregon, which had only "low" certainty of being persistent. The strongest strata were the Lakes and Mid-South Coast, which had "high" certainty of being persistent. To increase certainty that the ESU as a whole is persistent, they recommended that restoration work should focus on those populations with low persistence, particularly those in the North Coast, Mid-Coast, and Umpqua strata.

A 2010 BRT (Stout *et al.* 2011) noted significant improvements in hatchery and harvest practices have been made. However, harvest and hatchery reductions have changed the population

dynamics of the ESU. It has not been demonstrated that productivity during periods of poor marine survival is now adequate to sustain the ESU. Recent increases in adult escapement do not provide strong evidence that the century-long downward trend has changed. The ability of the OC coho salmon ESU to survive another prolonged period of poor marine survival remains in question.

Current concerns for spatial structure focus on the Umpqua River. Of the four populations in the Umpqua stratum, the North Umpqua and South Umpqua, were of particular concern. The North Umpqua is controlled by Winchester Dam and has historically been dominated by hatchery fish. Hatchery influence has recently been reduced, but the natural productivity of this population remains to be demonstrated. The South Umpqua is a large, warm system with degraded habitat. Spawner distribution appears to be seriously restricted in this population, and it is probably the most vulnerable of any population in this ESU to increased temperatures.

Current status of diversity shows improvement through the waning effects of hatchery fish on populations of OC coho salmon. In addition, recent efforts in several coastal estuaries to restore lost wetlands should be beneficial. However, diversity is lower than it was historically because of the loss of both freshwater and tidal habitat loss coupled with the restriction of diversity from very low returns over the past 20 years.

The BRT concluded that there is a moderate certainty of ESU persistence over the next 100 years and a low-to-moderate certainty that the ESU is sustainable for the foreseeable future, assuming no future trends in factors affecting the ESU. The NMFS issued a final determination to retain the ESA listing status, effective June 20, 2011. Thus, the February 2008 critical habitat designation and 4(d) regulations remain in effect (USDC 2011b).

Limiting factors and threats to the OC coho salmon ESU include (Stout *et al.* 2011, NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, instream mining, dams, road crossings, dikes, levees, etc.
- Fish passage barriers that limit access to spawning and rearing habitats
- Adverse climate, altered past ocean/marine productivity, and current ocean ecosystem conditions have favored competitors and predators and reduced salmon survival rates in freshwater rivers and lakes, estuaries, and marine environments

Southern Oregon and Northern California Coasts Recovery Domain. The SONCC recovery domain includes coho salmon, southern green sturgeon, and eulachon. The SONCC recovery domain extends from Cape Blanco, Oregon, to Punta Gorda, California. This area includes many small-to-moderate-sized coastal basins, where high quality habitat occurs in the lower reaches of each basin, and three large basins (Rogue, Klamath and Eel) where high quality habitat is in the lower reaches, little habitat is provided by the middle reaches, and the largest amount of habitat is in the upper reaches.

SONCC Coho Salmon. This species includes all naturally-spawned populations of coho salmon in coastal streams between Cape Blanco, Oregon, and Punta Gorda, California, and progeny of three artificial propagation programs. The SONCC-TRT identified 42 extant populations within this ESU, as well as 3 artificial propagation programs (Williams *et al.* 2011). In some cases, the SONCC-TRT also identified groups of populations referred to as "diversity strata" largely based on the geographical arrangement of the populations and basin-scale environmental and ecological characteristics. Of those populations, 13 strata and 17 populations occur in Oregon (Table 2.4.2.17).

The estimated abundance for SONCC coho salmon was 6,705 total spawners (ODFW 2010, Williams *et al.* 2011).

In most cases, populations appear to be well below the proposed viability thresholds, and the steps needed to move them toward viability will be similar, regardless of the specific recovery targets, which can be refined as more information becomes available. The SONCC-TRT developed a framework to assess the viability of this species and recommended: (1) Securing all extant populations, (2) collecting distribution and abundance data, (3) minimizing straying from hatcheries to natural spawning areas, and (4) beginning critical research on climate change and its potential impacts (Williams *et al.* 2008). Although long-term data on abundance of SONCC coho salmon are scarce, available evidence from shorter-term research and monitoring efforts indicate that conditions have worsened for populations since the last formal status review was published (Good *et al.* 2005, Williams *et al.* 2011). Many independent populations are well below low-risk abundance targets, and several are likely below the high-risk depensation thresholds specified by the TRT (Williams *et al.* 2011).

Table 2.4.2.17. SONCC coho salmon populations in Oregon. Dependent populations (D) are populations that historically would not have had a high likelihood of persisting in isolation for 100 years. These populations relied upon periodic immigration from other populations to maintain their abundance. Independent populations are populations that historically would have had a high likelihood of persisting in isolation from neighboring populations for 100 years and are rated as functionally independent (FI) and potentially independent (PI). Two ephemeral populations (E) are defined as populations both small enough and isolated enough that they are only intermittently present (McElhany *et al.* 2000, Williams *et al.* 2011).

Population		Population Type
River Basin	**Subbasin**	
Elk River		FI
Mill Creek		D
Hubbard Creek		E
Brush Creek		D
Mussel Creek		D
Euchre Creek		E
Rogue River*	Lower Rogue River	PI
	Illinois River*	FI
	Mid Rogue/Applegate*	FI
	Upper Rogue River	FI
Hunter Creek		D
Pistol River		D
Chetco River		FI
Winchuck River		PI
Smith River*		FI
Klamath River*	Middle Klamath River	PI
	Upper Klamath River	FI

* Populations that also occur partly in California.

Limiting factors and threats to SONCC coho salmon include (NMFS 2012, NOAA Fisheries 2011):

- Lack of floodplain and channel structure
- Impaired water quality
- Altered hydrologic function due to altered amount and timing of river flows
- Degraded riparian forest conditions and large wood recruitment
- Altered sediment supply
- Degraded stream substrate
- Impaired estuarine function
- Impaired fish passage
- Hatchery-related adverse effects
- Effects of predation, competition, and disease mortality

Threats from natural or man-made factors have worsened in the past 5 years, primarily due to four factors: small population dynamics, climate change, multi-year drought, and poor ocean survival conditions (NOAA Fisheries 2011).

2.4.3 Status of the Critical Habitats

We based our ratings of the status of critical habitat primarily on a watershed-scale analysis of conservation value that focused on the presence of listed ESA-listed species and physical features (*i.e.*, the primary constituent elements or PCEs) that are essential to their conservation. The physical or biological features of freshwater spawning and incubation sites include water flow, water quality, water temperatures, suitable substrate for spawning and incubation, and migratory access for adults and juveniles. These features are essential to conservation because without them the species cannot successfully spawn and produce offspring. The physical or biological features of freshwater migration corridors associated with spawning and incubation sites include water flow, water quality and water temperatures to support larval and adult mobility; abundant prey items to support larval feeding after the yolk sac is depleted; and free passage (*i.e.*, no obstructions) for adults and juveniles. These features are essential to conservation because they allow adult fish to swim upstream to reach spawning areas, and they allow juvenile fish to proceed downstream and reach the ocean.

The analysis for the 2005 designations of critical habitat for 12 species of listed salmon and steelhead species in the Columbia River basin was completed by interagency critical habitat analytical review teams (CHARTs). These teams focused on large geographical areas corresponding approximately to recovery domains (NOAA Fisheries 2005). A CHART also did an initial assessment of PCEs for coho salmon on the Oregon Coast (NOAA Fisheries 2005). The CHARTs ranked the conservation value of each watershed based on the quantity of stream habitat with PCEs, the present condition of those PCEs, the likelihood of achieving PCE potential (either naturally or through active restoration), support for rare or important genetic or life history characteristics, support for abundant populations, and support for spawning and rearing populations. In some cases, we have refined our understanding of these conservation values of these watersheds based on the work of TRTs and other recovery planning efforts that have better explained the habitat attributes, ecological interactions, and population characteristics important to each species.

Salmon and Steelhead Critical Habitat. Tables 2.4.3.1 and 2.4.3.2 identify the PCEs (*i.e.*, site types, site attributes) and corresponding life history events for the critical habitats of listed salmon and steelhead.

Table 2.4.3.1. PCEs of critical habitats designated for listed salmon and steelhead species (except SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, SR sockeye salmon, and SONCC coho salmon), and corresponding species life history events.

Primary Constituent Elements		**Species Life History Event**
Site Type	**Site Attribute**	
Freshwater spawning	Substrate Water quality Water quantity	Adult spawning Embryo incubation Alevin growth and development
Freshwater rearing	Floodplain connectivity Forage Natural cover Water quality Water quantity	Fry emergence from gravel Fry/parr/smolt growth and development
Freshwater migration	Free of artificial obstruction Natural cover Water quality Water quantity	Adult sexual maturation Adult upstream migration and holding Kelt (steelhead) seaward migration Fry/parr/smolt growth, development, and seaward migration
Estuarine areas	Forage Free of artificial obstruction Natural cover Salinity Water quality Water quantity	Adult sexual maturation and "reverse smoltification" Adult upstream migration and holding Kelt (steelhead) seaward migration Fry/parr/smolt growth, development, and seaward migration
Nearshore marine areas	Forage Free of artificial obstruction Natural cover Water quantity Water quality	Adult growth and sexual maturation Adult spawning migration Nearshore juvenile rearing
Offshore marine areas	Forage Water quality	Adult growth and sexual maturation Adult spawning migration Subadult rearing

Table 2.4.3.2. PCEs of critical habitats designated for SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, SR sockeye salmon, and SONCC coho salmon, and corresponding species life history events.

Primary Constituent Elements		Species Life History Event
Site	**Site Attribute**	
Spawning and juvenile rearing areas	Access (sockeye) Cover/shelter Food (juvenile rearing) Riparian vegetation Space (Chinook, coho) Spawning gravel Water quality Water temp (sockeye) Water quantity	Adult spawning Embryo incubation Alevin growth and development Fry emergence from gravel Fry/parr/smolt growth and development
Adult and juvenile migration corridors	Cover/shelter Food (juvenile) Riparian vegetation Safe passage Space Substrate Water quality Water quantity Water temperature Water velocity	Adult sexual maturation Adult upstream migration and holding Kelt (steelhead) seaward migration Fry/parr/smolt growth, development, and seaward migration
Areas for growth and development to adulthood	Ocean areas – not identified	Nearshore juvenile rearing Subadult rearing Adult growth and sexual maturation Adult spawning migration

We give descriptions of the status of critical habitat for each species of salmon and steelhead below.

LCR Chinook salmon. Designated critical habitat for LCR Chinook salmon includes all Columbia River estuarine areas and river reaches from the mouth to the confluence with the Hood River, as well as specific stream reaches in the following subbasins: Middle Columbia/Hood, Lower Columbia/Sandy, Lewis, Lower Columbia/Clatskanie, Upper Cowlitz, Cowlitz, Lower Columbia, Grays/Elochoman, Clackamas, and Lower Willamette (NMFS 2005b). There are 48 watersheds within the range of this ESU. Four watersheds received a low rating, 13 received a medium rating, and 31 received a high rating of conservation value for the species (*i.e.*, for recovery) (NOAA Fisheries 2005). The lower Columbia River has a high conservation value. It connects every population with the ocean, and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,655 miles of habitat eligible for designation, NMFS designated 1,311 miles as critical habitat.

The major factors affecting the condition of the PCEs for this species are (LCFRB 2010, NOAA Fisheries Service 2011):

- Degraded estuarine and near-shore marine habitat resulting from the cumulative impacts of land use and flow management by the Columbia River hydropower system
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- In freshwater habitats, degradation of floodplain connectivity and function, channel structure and complexity, riparian areas, stream substrate, stream flow, and water quality, all as a result of the cumulative impacts of agriculture, forestry, and development
- Elevated concentrations of contaminants in sediments and water
- Reduced access to spawning and rearing habitats in tributaries, mainly as a result of hydropower projects
- Reduced access to off-channel rearing habitat in the Lower Columbia River

UWR Chinook salmon. Designated critical habitat for UWR Chinook salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence with the Willamette River, as well as specific stream reaches in the following subbasins: Middle Fork Willamette, Coast Fork Willamette, Upper Willamette, McKenzie, North Santiam, South Santiam, Middle Willamette, Molalla/Pudding, Clackamas, and Lower Willamette (NMFS 2005b). There are 60 watersheds within the range of this species. Nineteen watersheds received a low rating, 18 received a medium rating, and 23 received a high rating of conservation value for the species (NOAA Fisheries 2005). The lower Willamette/Columbia River rearing/migration has a high conservation value. It connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,796 miles of habitat eligible for designation, NMFS designated 1,472 miles as designated critical habitat.

The major factors affecting the condition of the PCES for this species are (ODFW and NMFS 2011, NOAA Fisheries 2011):

- Significantly reduced access to spawning and rearing habitat because of tributary dams
- Degraded freshwater habitat, especially floodplain connectivity and function, channel structure and complexity, and riparian areas and large wood recruitment as a result of the cumulative impacts of agriculture, forestry, and development
- Degraded water quality and altered water temperatures as a result of both tributary dams and the cumulative impacts of agriculture, forestry, and urban development

UCR spring-run Chinook salmon. Designated critical habitat for UCR spring Chinook includes all Columbia River estuarine areas and river reaches from the mouth upstream to Chief Joseph Dam, as well as specific stream reaches in the following subbasins: Chief Joseph, Methow, Upper Columbia/Entiat, and Wenatchee (NMFS 2005b). There are 31 watersheds within the range of this species. Five watersheds received a medium rating and 26 received a high rating of conservation value to the species. The Columbia River downstream of the specie's spawning range has a high conservation value and is the only habitat area designated in 15 of the high-value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,002 miles of habitat eligible for designation, NMFS designated 974 miles as critical habitat.

The major factors affecting the condition of the PCES for this species are (UCSRB 2007, NOAA Fisheries 2011):

- Altered upstream and downstream fish passage, ecosystem structure and function, flows, and water quality, all due to the Columbia River hydropower system
- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development
- Degraded estuarine and nearshore marine habitats

SR SS Chinook salmon. Designated critical habitat for SR spring/summer-run Chinook salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence of the Columbia and Snake rivers, and all Snake River reaches from the confluence of the Columbia River upstream to Hells Canyon Dam (NMFS 1999a). Critical habitat also includes river reaches presently or historically accessible (except those above impassable natural falls, including Napias Creek Falls, and Dworshak and Hells Canyon dams) in the following subbasins: Hells Canyon, Imnaha, Lemhi, Little Salmon, Lower Grande Ronde, Lower Middle Fork Salmon, Lower Salmon, Lower Snake-Asotin, Lower Snake-Tucannon, Middle Salmon-Chamberlain, Middle Salmon-Panther, Pahsimeroi, South Fork Salmon, Upper Middle Fork Salmon, Upper Grande Ronde, Upper Salmon, and Wallowa.

Designated areas of critical habitat consist of the water, waterway bottom, and the adjacent riparian zone (defined as an area 300 feet from the normal high water line on each side of the river channel) (NMFS 1999a). Designation did not involve rating the conservation value of specific watersheds as was done in subsequent designations (NMFS 2005b). The lower Columbia River is among the areas of high conservation value to this species because it connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats.

The major factors affecting the condition of the PCES for this species are (NOAA Fisheries 2011):

- Degradation of floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, water temperatures, stream flows, and water quality, all as a result of the cumulative impacts of agriculture, forestry, and development
- Impacts from the mainstem Columbia River hydropower system

SR fall-run Chinook salmon. Designated critical habitat for SR fall-run Chinook salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence of the Columbia and Snake rivers; all Snake River reaches from the confluence of the Columbia River upstream to Hells Canyon Dam; the Palouse River from its confluence with the Snake River upstream to Palouse Falls; the Clearwater River from its confluence with the Snake River upstream to its confluence with Lolo Creek; and the North Fork Clearwater River from its confluence with the Clearwater River upstream to Dworshak Dam. Critical habitat also includes river reaches

presently or historically accessible (except those above impassable natural falls and Dworshak and Hells Canyon dams) in the following subbasins: Clearwater, Hells Canyon, Imnaha, Lower Grande Ronde, Lower North Fork Clearwater, Lower Salmon, Lower Snake, Lower Snake-Asotin, Lower Snake-Tucannon, and Palouse. The lower Columbia River is among the areas of high conservation value to this species because it connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Designated areas consist of the water, waterway bottom, and the adjacent riparian zone (defined as an area 300 feet from the normal high water line on each side of the river channel).

The major factors affecting the condition of the PCES for this species are (NOAA Fisheries 2011):

- Degraded floodplain connectivity and function, and channel structure and complexity, as a result of the cumulative impacts of agriculture, forestry, and development
- Lost access to historical habitat above Hells Canyon and other Snake River dams
- Impacts of the mainstem Columbia River hydropower system
- Degraded estuarine and nearshore habitat

CR chum salmon. Designated critical habitat for CR chum salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence with the White Salmon River, as well as specific stream reaches in the following subbasins: Middle Columbia/Hood, Lower Columbia/Sandy, Lewis, Lower Columbia/Clatskanie, Cowlitz, Lower Columbia, and Grays/ Elochoman (NMFS 2005b). There are 20 watersheds within the range of this ESU. Three watersheds received a medium rating and 17 received a high rating for their conservation value to the ESU (*i.e.*, for recovery). The lower Columbia River has a high conservation value and is the only habitat area designated in one of the high value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 725 miles of habitat eligible for designation, NMFS designated 708 miles as critical habitat.

The major factors affecting the condition of the PCES for this species are (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and nearshore marine habitats resulting from the cumulative impacts of land use and flow management by the Columbia River hydropower system
- Degraded floodplain connectivity and function, channel structure and complexity, stream substrate, and riparian areas and large wood recruitment as a result of the cumulative impacts of agriculture, forestry, and development
- Altered stream flows as a result of hydropower and water supply operations
- Reduced access to off-channel rearing habitat
- Reduced water quality
- Alterations of the Columbia River's flow regime and the Columbia River plume that have altered the water temperature regime and estuarine food web, and have reduced ocean productivity
- Contaminants that have affected fish health and reproduction

SONCC coho salmon. Critical habitat for SONCC coho salmon includes all accessible waterways, substrate, and adjacent riparian zones between the Mattole River in California, and the Elk River in Oregon, inclusive (USDC 1999). Excluded are: (1) areas above specific dams identified in USDC (1999), (2) areas above longstanding natural impassible barriers (*i.e.*, natural waterfalls), and (3) tribal lands.

The major factors affecting the condition of the PCES for this species are (NOAA Fisheries 2011, NMFS 2012):

- Lack of floodplain function and channel structure
- Impaired water quality
- Altered hydrologic function (timing of volume of water flow)
- Impaired estuary functioning
- Degraded riparian forest conditions
- Altered sediment supply
- Barriers to migration

Oregon Coast coho salmon. Critical habitat for OC coho salmon includes areas specified in USDC (2008) south of the Columbia River and north of Cape Blanco including the Nehalem River, Nestucca River, Siletz River, Yaquina River, Alsea River, Siuslaw River, Umpqua River, Coos River, and Coquille River.

The major factors affecting the condition of the PCES for this species are (Stout *et al.* 2011, NOAA Fisheries 2011):

- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, instream mining, dams, road crossings, dikes, and levees
- Fish passage barriers that limit access to spawning and rearing habitats

SR sockeye salmon. Designated critical habitat for SR sockeye salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence of the Columbia and Snake rivers; all Snake River reaches from the confluence of the Columbia River upstream to the confluence of the Salmon River; all Salmon River reaches from the confluence of the Snake River upstream to Alturas Lake Creek; Stanley, Redfish, Yellow Belly, Pettit, and Alturas lakes (including their inlet and outlet creeks); Alturas Lake Creek; and that portion of Valley Creek between Stanley Lake Creek and the Salmon River (USDC 1993).

Designated areas consist of the water, waterway bottom, and the adjacent riparian zone (defined as an area 300 feet from the normal high water line on each side of the river channel) (USDC 1993). Designation did not involve rating the conservation value of specific watersheds as was done in subsequent designations. The lower Columbia River is among the areas of high conservation value to this species because it connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a

unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats.

The major factors affecting the condition of the PCES for this species are (NOAA Fisheries 2011):

- High water temperatures in portions of the migration corridor in the Salmon
- Alteration of the natural hydrological regime in the upper mainstem Salmon River Basin by water withdrawals
- Impacts of the mainstem Columbia River hydropower system

LCR steelhead. Designated critical habitat for LCR steelhead includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence with the Hood River, as well as specific stream reaches in the following subbasins: Middle Columbia/Hood, Lower Columbia/Sandy, Lewis, Lower Columbia/Clatskanie, Upper Cowlitz, Cowlitz, Clackamas, and Lower Willamette (NMFS 2005b). There are 32 watersheds within the range of this DPS. Two watersheds received a low rating, 11 received a medium rating, and 29 received a high rating of conservation value to the DPS. The lower Columbia River has a high conservation value. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 2,673 miles of habitat areas eligible for designation, NMFS designated 2,324 miles as critical habitat.

The major factors affecting the condition of the PCES for this species are (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and nearshore marine habitat resulting from the cumulative impacts of land use and flow management by the Columbia River hydropower system
- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and recruitment of large wood, stream substrate, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development
- Reduced access to spawning and rearing habitat as a result of tributary hydropower projects and lowland development
- Alterations of the Columbia River's flow regime and the Columbia River plume that have altered the water temperature regime and estuarine food web, and have reduced ocean productivity
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Contaminants that are affecting fish health and reproduction

UWR steelhead. Designated critical habitat for UWR steelhead includes all Columbia River estuarine areas and river reaches proceeding upstream to the confluence with the Willamette River, as well as specific stream reaches in the following subbasins: Upper Willamette, North Santiam, South Santiam, Middle Willamette, Molalla/Pudding, Yamhill, Tualatin, and Lower Willamette (NMFS 2005b). There are 38 watersheds within the range of this DPS. The lower Willamette/Columbia River has a high conservation value and is the only habitat area designated in one of the high value

watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,830 miles of habitat eligible for designation, 1,276 miles of stream are designated critical habitat.

The major factors affecting the condition of the PCES for this species are (ODFW and NMFS 2011, NOAA Fisheries 2011):

- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large wood recruitment, stream substrate, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development
- Reduced access to spawning and rearing habitat as a result of tributary hydropower projects and lowland development
- Reduced access to spawning and rearing habitats, mainly as a result of artificial barriers in tributaries

MCR steelhead. Designated critical habitat for MCR steelhead includes all Columbia River estuarine areas and river reaches in the following subbasins: Upper Yakima, Naches, Lower Yakima, Middle Columbia/Lake Wallula, Walla Walla, Umatilla, Middle Columbia/Hood, Klickitat, Upper John Day, North Fork John Day, Middle Fork John Day, Lower John Day, Lower Deschutes, Trout, and Upper Columbia/Priest Rapids (NMFS 2005b). There are 114 watersheds within the range of this DPS. Nine watersheds received a low rating, 24 received a medium rating, and 81 received a high rating of conservation value to the DPS (see Chapter 4 for more detail). The lower Columbia River downstream of the specie's spawning range has a high conservation value and is the only habitat area designated in three of the high value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. Of the 6,529 miles of habitat areas eligible for designation, 5,815 miles of stream are designated critical habitat.

The major factors affecting the condition of the PCES for this species are (NMFS 2009, NOAA Fisheries 2011):

- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas, fish passage, stream substrate, stream flow, and water quality as a result of cumulative impacts of agriculture, forestry, tributary hydropower projects, and development
- Impacts from the mainstem Columbia River hydropower system
- Degraded estuarine and nearshore marine habitats

UCR steelhead. Designated critical habitat for UCR steelhead includes all Columbia River estuarine areas and river reaches from the mouth upstream to Chief Joseph Dam, as well as specific stream reaches in the following subbasins: Chief Joseph, Okanogan, Similkameen, Methow, Upper Columbia/Entiat, Wenatchee, Lower Crab, and Upper Columbia/Priest Rapids (NMFS 2005b). There are 42 watersheds within the range of this DPS. Three watersheds received a low rating, 8 received a medium rating, and 31 received a high rating of conservation

value to the DPS. The Columbia River downstream of the specie's spawning range has a high conservation value and is the only habitat area designated in 11 of the high value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,332 miles of habitat areas eligible for designation, NMFS designated 1,262 miles as critical habitat.

The major factors affecting the condition of the PCES for this species are (UCSRB 2007, NOAA Fisheries 2011):

- Impacts from the mainstem Columbia River hydropower system
- Impaired tributary fish passage
- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development

SRB steelhead. Designated critical habitat for SRB steelhead includes all Columbia River estuarine areas and river reaches proceeding upstream to the confluence of the Columbia and Snake rivers as well as specific stream reaches in the following subbasins: Hells Canyon, Imnaha River, Lower Snake/Asotin, Upper Grande Ronde River, Wallowa River, Lower Grande Ronde, Lower Snake/Tucannon, Lower Snake River, Upper Salmon, Pahsimeroi, Middle Salmon-Panther, Lemhi, Upper Middle Fork Salmon, Lower Middle Fork Salmon, Middle Salmon-Chamberlain, South Fork Salmon, Lower Salmon, Little Salmon, Upper Selway, Lower Selway, Lochsa, Middle Fork Clearwater, South Fork Clearwater, and Clearwater (NMFS 2005b). There are 289 watersheds within the range of this DPS. Fourteen watersheds received a low rating, 44 received a medium rating, and 231 received a high rating of conservation value to the DPS. The lower Snake/Columbia River downstream of the specie's spawning range has a high conservation value and is the only habitat area designated in 15 of the high value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 8,225 miles of habitat areas eligible for designation, NMFS designated 8,049 miles as critical habitat.

The major factors affecting the condition of the PCES for this species are (IC-TRT 2006, NOAA Fisheries 2011):

- Impacts from the mainstem Columbia River hydropower system
- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development
- Increased water temperature

Green sturgeon. Critical habitat for green sturgeon includes: freshwater rivers, the bypasses, the Sacramento-San Joaquin Delta, coastal bays and estuaries, and coastal marine areas

(within 110 m depth) extending from the California/Mexico border north to Monterey Bay, California, and from the Alaska/Canada border northwest to the Bering Strait; and certain coastal bays and estuaries in California, Oregon, and Washington (USDC 2009b).

For freshwater rivers north of and including the Eel River, NMFS did not consider the areas upstream of the head of the tide to be part of the geographical area occupied by southern DPS green sturgeon. However, the critical habitat designation recognizes not only the importance of natal habitats, but of habitats throughout their range. Critical habitat has been designated in coastal U.S. marine waters within 60 fathoms depth from Monterey Bay, California (including Monterey Bay), north to Cape Flattery, Washington, including the Strait of Juan de Fuca, Washington, to its United States boundary; the Sacramento River, lower Feather River, and lower Yuba River in California; the Sacramento-San Joaquin Delta and Suisun, San Pablo, and San Francisco bays in California; the lower Columbia River estuary; and certain coastal bays and estuaries in California (Humboldt Bay), Oregon (Coos Bay, Winchester Bay, Yaquina Bay, and Nehalem Bay), and Washington (Willapa Bay and Grays Harbor) and freshwater (USDC 2009b). Table 2.4.3.1 lists the PCEs of critical habitat for southern DPS green sturgeon and corresponding life history events.

Table 2.4.3.3. PCEs of critical habitat designated for southern DPS green sturgeon and corresponding species life history events.

Primary Constituent Elements		**Species Life History Event**
Site Type	**Site Attribute**	
Freshwater riverine system	Food resources Migratory corridor Sediment quality Substrate type or size Water depth Water flow Water quality	Adult spawning Embryo incubation, growth and development Larval emergence, growth and development Juvenile metamorphosis, growth and development
Estuarine areas	Food resources Migratory corridor Sediment quality Water flow Water depth Water quality	Juvenile growth, development, seaward migration Subadult growth, development, seasonal holding, and movement between estuarine and marine areas Adult growth, development, seasonal holding, movements between estuarine and marine areas, upstream spawning movement, and seaward post-spawning movement
Coastal marine areas	Food resources Migratory corridor Water quality	Subadult growth and development, movement between estuarine and marine areas, and migration between marine areas Adult sexual maturation, growth and development, movements between estuarine and marine areas, migration between marine areas, and spawning migration

The major factors affecting the condition of the PCEs for this species within freshwater rivers, bypasses, and the Sacramento-San Joaquin Delta (the Delta) are (USDC 2009b):

- Dams and diversions that obstruct migration, alter water flows and temperature, and modify substrate composition within the rivers
- Low water levels may obstruct passage through the bypasses, resulting in stranded fish

- Pollution from agricultural runoff and water returns, as well as from other point- and non-point sources, degrades water quality within the rivers, bypasses and the Delta.
- Dredging and pile driving can adversely affect water quality and prey resources, and alter the composition and distribution of bottom substrates within the Delta

Within bays and estuaries, the major factors affecting the condition of the PCEs for this species are (USDC 2009b):

- The application of pesticides that adversely affects prey resources and water quality
- Disturbance of bottom substrates by dredging or certain other activities that adversely affects prey resources, or degrades water quality through re-suspension of contaminated sediments.
- Commercial shipping and other sources of point- and non-point source pollution that discharge contaminants
- Disposal of dredged materials that bury prey resources
- Bottom trawl fisheries that disturb the bottom and may result in beneficial or adverse effects on prey resources for green sturgeon

Within coastal marine areas, the major factors affecting the condition of the PCEs for this species are (USDC 2009b):

- Disturbance of bottom substrates by dredging or certain other activities that adversely affects prey resources, or degrades water quality through re-suspension of contaminated sediments.
- Commercial shipping and other sources of point- and non-point source pollution that discharge contaminants
- Disposal of dredged materials that bury prey resources
- Bottom trawl fisheries that disturb the bottom and may result in beneficial or adverse effects on prey resources for green sturgeon

Eulachon. Critical habitat for eulachon includes portions of 16 rivers and streams in California, Oregon, and Washington (USDC 2011c). All of these areas are designated as migration and spawning habitat for this species. In Oregon, NMFS designated 24.2 miles of the lower Umpqua River, 12.4 miles of the lower Sandy River, and 0.2 miles of Tenmile Creek as critical habitat. The NMFS also designated the mainstem Columbia River from the mouth to the base of Bonneville Dam, a distance of 143.2 miles, as critical habitat. Table 2.4.3.2 lists the designated Physical and Biological Features (PBFs) for eulachon and associated species life history events.

Table 2.4.3.4. PBFs of critical habitats designated for eulachon and corresponding species life history events.

Essential Features		Species Life History Event
Site Type	**Site Attribute**	
Freshwater spawning and incubation	Flow, Water quality Water temperature Substrate	Adult spawning Incubation
Freshwater migration	Flow, Water quality Water temperature, Food	Adult and larval mobility Larval feeding

The major factors affecting the condition of the PCEs for this species include (Gustafson *et al.* 2010, Gustafson *et al.* 2011, NOAA Fisheries 2011):

- Changes in ocean conditions due to climate change
- Adverse effects related to dams and water diversions
- Artificial fish passage barriers
- Water pollution
- Increased water temperatures
- Insufficient stream flow
- Altered sediment balances

2.4.4 Marine Mammals

2.4.4.1 Southern Resident Killer Whales

Current Rangewide Status of the Species. The Southern Resident killer whale DPS, composed of J, K and L pods, was listed as endangered under the ESA on November 18, 2005 (70 FR 69903). Southern Residents are designated as "depleted" and "strategic" under the Marine Mammal Protection Act (MMPA)(68 FR 31980, May 29, 2003).

This section summarizes the status of the Southern Resident killer whales throughout their range. The final recovery plan for Southern Residents was issued in January 2008 (NMFS 2008a). This section summarizes information taken largely from the recovery plan and recent 5-year status review (NMFS 2011), as well as new data that became available more recently. For more detailed information about this population, please refer to NMFS (2008a).

Abundance, Productivity and Trends. Southern Resident killer whales are a long-lived species, with late onset of sexual maturity (review in NMFS 2008a). Females produce a low number of surviving calves over the course of their reproductive life span (Bain 1990, Olesiuk *et al.* 1990). Southern Resident females appear to have reduced fecundity relative to Northern Residents; the average interbirth interval for reproductive Southern Resident females is 6.1 years,

which is longer than that of Northern Resident killer whales (Olesiuk *et al.* 2005). Mothers and offspring maintain highly stable social bonds throughout their lives, which is the basis for the matrilineal social structure in the Southern Resident population (Baird 2000, Bigg *et al.* 1990, Ford *et al.* 2000). Groups of related matrilines form pods. Three pods – J, K, and L – make up the Southern Resident community. Clans are composed of pods with similar vocal dialects and all three pods of the Southern Residents are part of J clan.

The historical abundance of Southern Resident killer whales is estimated from 140 to an unknown upper bound. The minimum historical estimate (~140) included whales killed or removed for public display in the 1960s and 1970s added to the remaining population at the time the captures ended. Several lines of evidence (*i.e.,* known kills and removals [Olesiuk *et al.* 1990], salmon declines [Krahn *et al.* 2002] and genetics [Krahn *et al.* 2002, Ford *et al.* 2011a]) all indicate that the population used to be much larger than it is now, but there is currently no reliable estimate of the upper bound of the historical population size. When faced with developing a population viability analysis for this population, NMFS' biological review team found it reasonable to assume an upper bound of as high as 400 whales to estimate carrying capacity (Krahn *et al.* 2004).

At present, the Southern Resident population has declined to essentially the same size that was estimated during the early 1960s, when it was considered as likely depleted (Olesiuk *et al.* 1990) (Figure 2.4.4.1). Since censuses began in 1974, J and K pods have steadily increased their sizes. However, the population suffered an almost 20 percent decline from 1996-2001 (from 97 whales in 1996 to 81 whales in 2001), largely driven by lower survival rates in L pod. Since then the overall population has increased slightly from 2002 to present (from 83 whales in 2002 to 88 whales in August, 2011). Over the last 28 years (1983-2010), population growth has been variable, with an average annual population growth rate of 0.3 percent and standard deviation of ± 3.2 percent. Seasonal mortality rates among Southern and Northern Resident whales may be highest during the winter and early spring, based on the numbers of animals missing from pods returning to inland waters each spring. Olesiuk *et al.* (2005) identified high neonate mortality that occurred outside of the summer season. At least 12 newborn calves (nine in the southern community and three in the northern community) were seen outside the summer field season and disappeared by the next field season. Additionally, stranding rates are higher in winter and spring for all killer whale forms in Washington and Oregon (Norman *et al.* 2004). Southern Resident strandings in coastal waters offshore include three separate events (1995 and 1996 off of Northern Vancouver Island and the Queen Charlotte Islands, and 2002 offshore of Long Beach, Washington State), but the causes of death are unknown (NMFS 2008a).

There are 26 whales in J pod, 20 whales in K pod and 42 whales in L pod. There are currently 2 adult males and one nearly matured male in J pod, three adult males in K pod, and 10 adult males in L pod. The population is 35.6 percent juveniles, 34.5 percent reproductive females, 10.3 percent post-reproductive females and 18.4 percent adult males. This age distribution is similar to that of Northern Residents that are a stable and increasing population (Olesiuk *et al.* 2005). However, there are several demographic factors of the Southern Resident population that are cause for concern, namely the small number of breeding males (particularly in J and K pods), reduced fecundity, sub-adult survivorship in L pod, and the total number of individuals in the population (review in NMFS 2008a). The current population abundance of 87 whales is small, at

most half of its likely previous abundance (140 to an unknown upper bound that could be as high at 400 whales, as discussed above). The estimated effective size of the population (based on the number of breeders under ideal genetic conditions) is very small at approximately 26 whales or roughly 1/3 of the current population size (Ford *et al.* 2011a). The small effective population size and the absence of gene flow from other populations may elevate the risk from inbreeding and other issues associated with genetic deterioration, as evident from documented breeding within pods (Ford *et al.* 2011a). As well, the small effective population size may contribute to the lower growth rate of the Southern Resident population in contrast to the Northern Resident population (Ford *et al.* 2011a, Ward *et al.* 2009).

Because of this population's small abundance, it is also susceptible to demographic stochasticity – randomness in the pattern of births and deaths among individuals in a population. Several other sources of stochasticity can affect small populations and contribute to variance in a population's growth and extinction risk. Other sources include environmental stochasticity, or fluctuations in the environment that drive fluctuations in birth and death rates, and demographic heterogeneity, or variation in birth or death rates of individuals because of differences in their individual fitness (including sexual determinations). In combination, these and other sources of random variation combine to amplify the probability of extinction, known as the extinction vortex (Gilpin and Soule 1986, Fagen and Holmes 2006, Melbourne and Hastings 2008). The larger the population size, the greater the buffer against stochastic events and genetic risks. A delisting criterion for the Southern Resident killer whale DPS is an average growth rate of 2.3% for 28 years (NMFS 2008a). In light of the current average growth rate of 0.3%, this recovery criterion reinforces the need to allow the population to grow quickly.

Population growth is also important because of the influence of demographic and individual heterogeneity on a population's long-term viability. Population-wide distribution of lifetime reproductive success can be highly variable, such that some individuals produce more offspring than others to subsequent generations, and male variance in reproductive success can be greater than that of females (*i.e.*, Clutton-Brock 1988, Hochachka 2006). For long-lived vertebrates such as killer whales, some females in the population might contribute less than the number of offspring required to maintain a constant population size (n = 2), while others might produce more offspring. The smaller the population, the more weight an individual's reproductive success has on the population's growth or decline (*i.e.*, Coulson *et al.* 2006). This further illustrates the risk of demographic stochasticity for a small population like Southern Resident killer whales – the smaller a population, the greater the chance that random variation will result in too few successful individuals to maintain the population.

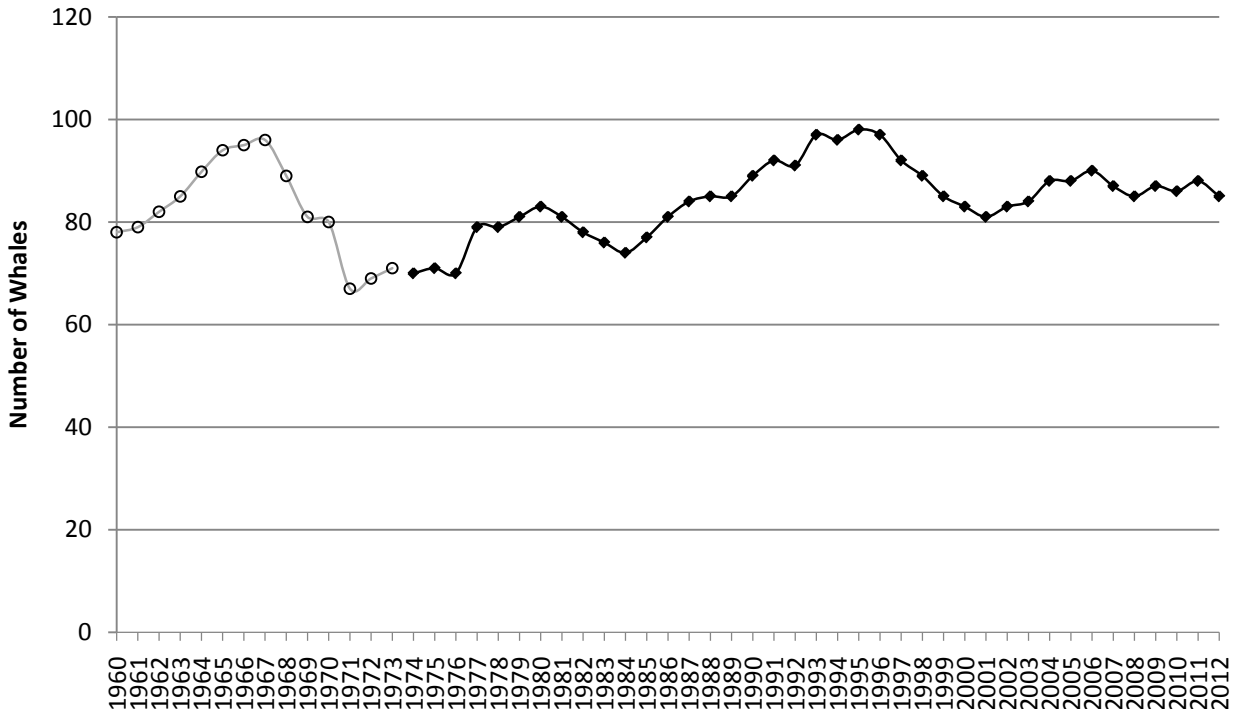

Figure 2.4.4.1.1. Population size and trend of Southern Resident killer whales, 1960-2012. Data from 1960-1973 (open circles, gray line) are number projections from the matrix model of Olesiuk *et al.* (1990). Data from 1974-2012 (diamonds, black line) were obtained through photo-identification surveys of the three pods (J, K, and L) in this community and were provided by the Center for Whale Research (unpubl. data) and NMFS (2008). Data for these years represent the number of whales present at the end of each calendar year, except for 2012, when data only extend to July.

Range and Distribution. Southern Residents occur throughout the coastal waters of Washington, Oregon, and Vancouver Island and are known to travel as far south as central California and as southeast Alaska (one sighting occurred in Chatham Strait, Alaska; Figure 2.4.4.1.2.). The Figure 2.4.4.1.2. does not reflect the recent sighting in Alaska. There is limited information on the distribution and habitat use of Southern Residents along the outer Pacific Coast.

Figure 2.4.4.1.2. Geographic Range (light shading) of the Southern Resident Killer Whale DPS. Reprinted from Wiles (2004).

Southern Residents are highly mobile and can travel up to 86 miles in a single day (Erickson 1978, Baird 2000). To date, there is no evidence that Southern Residents travel further than 50 km offshore (Ford *et al.* 2005). Although the entire Southern Resident DPS has potential to occur in coastal waters at any time during the year, occurrence is more likely from November to May (Table 2.4.4.1.1).

Southern Residents spend a substantial amount of time from late spring to early autumn in inland waterways of Washington State and British Columbia (Strait of Georgia, Strait of Juan de Fuca, and Puget Sound. Bigg 1982, Ford *et al.* 2000, Krahn *et al.* 2002, Table 2.4.4.1.1). Typically, J,

K and L pods are increasingly present in May or June and spend considerable time in the core area of Georgia Basin and Puget Sound until at least September. During this time, pods (particularly K and L) make frequent trips from inland waters to the outer coasts of Washington and southern Vancouver Island, which typically last a few days (Ford *et al.* 2000).

Table 2.4.4.1.1. Average number of days spent by Southern Resident killer whales in inland and coastal waters by month[1], 2003-2007 (Hanson and Emmons 2010).

Months	Lpod		Jpod		Kpod	
	Days Inland	Days Coastal	Days Inland	Days Coastal	Days Inland	Days Coastal
Jan	5	26	3	29	8	23
Feb	0	28	4	24	0	28
March	2	29	7	24	2	29
April	0	30	13	17	0	30
May	2	29	26	5	0	31
June	14	16	26	5	12	18
July	18	13	24	7	17	14
Aug	17	15	17	15	17	14
Sep	20	10	19	11	17	13
Oct	12	19	14	17	8	24
Nov	5	25	13	17	7	23
Dec	1	30	8	23	10	21

[1]Hanson and Emmons report sightings in inland waters. For purposes of this consultation analysis, and because the population is highly visible when in inland waters, NMFS assumes that when not sighted in inland waters the whales are in their coastal range.

Late summer and early fall movements of Southern Residents in the Georgia Basin are consistent, with strong site fidelity shown to the region as a whole and high occurrence in the San Juan Island area (Hanson and Emmons 2010, Hauser *et al.* 2007). There is inter-annual variability in arrival time and days present in inland waters from spring through fall, with late arrivals and fewer days present during spring in recent years potentially related to weak returns of spring and early summer Chinook salmon to the Fraser River (Hanson and Emmons 2010). Similarly, recent high occurrence in late summer may relate to greater than average Chinook salmon returns to South Thompson tributary of the Fraser River (Hanson and Emmons 2010). During fall and early winter, Southern Resident pods, and J pod in particular, expand their routine movements into Puget Sound, likely to take advantage of chum and Chinook salmon runs (Hanson *et al.* 2010a, Osborne 1999). During late fall, winter, and early spring, the ranges and movements of the Southern Residents are less known. Sightings through the Strait of Juan de Fuca in late fall suggest that activity shifts to the outer coasts of Vancouver Island and Washington (Krahn *et al.* 2002).

The Southern Residents were formerly thought to range southward along the coast to about Grays Harbor (Bigg *et al.* 1990) or the mouth of the Columbia River (Ford *et al.* 2000). However, recent sightings of members of K and L pods in Oregon (in 1999 and 2000) and California (in 2000, 2003, 2005, 2006, 2007, 2008, and 2009) have considerably extended the southern limit of their known range (NMFS 2008a). There have been verified visual sightings or

strandings of J, K or L pods along the outer coast from 1975 to present with most made from January through April (summarized in NMFS 2008a, and NWFSC unpubl. data). These include 16 records off Vancouver Island and the Queen Charlottes, 15 off Washington, four off Oregon, and 10 off central California. Most records have occurred since 1996, but this may be because of increased viewing effort along the coast for this time of year.

Sightings in Monterey Bay, California coincided with occurrence of salmon, with feeding witnessed in 2000 (Black *et al.* 2001). Southern Residents were also sighted in Monterey Bay during 2008, when salmon runs from California were expected to be near record lows (PFMC 2010). L pod was also seen feeding on unidentified salmon off Westport, Washington, in March 2004 during the spring Chinook salmon run in the Columbia River (M. B. Hanson, personal observation as cited in Krahn *et al.* 2004). In March, 2005 L pod was sighted working a circuit across the Columbia River plume from the North Jetty across to the South Jetty during the spring Chinook salmon run in the Columbia River (Zamon *et al.* 2007). Also in March of 2006, K and L pods were encountered off the Columbia River (Hanson *et al.* 2008). L pod was again seen feeding off Westport, Washington in March 2009, and genetic analysis of prey remains collected from two predation events identified one fish as spring Chinook salmon and the other as a summer/fall Chinook salmon from Columbia River stocks (Hanson *et al.* 2010b).

The Northwest Fisheries Science Center (NWFSC) also deploys and collects data from remote autonomous acoustic recorders in coastal waters of Washington State, and in 2009 alone documented 52 Southern Resident killer whale detections from this acoustic system (Emmons *et al.* 2009). The Department of Fisheries and Oceans (DFO), Canada also maintains acoustic recorders in British Columbia. When the NWFSC and DFO analyze these data, more information will be available about the seasonal distribution, movements and habitat use of Southern Resident killer whales, specifically in coastal waters off Washington and British Columbia.

Limiting Factors and Threats. Several factors identified in the final recovery plan for Southern Residents may be limiting recovery. These are quantity and quality of prey, toxic chemicals that accumulate in top predators, disturbance from sound and vessels. Oil spills are also a risk factor. It is likely that multiple threats are acting in concert to impact the whales. Although it is not clear which threat or threats are most significant to the survival and recovery of Southern Residents, all of the threats identified are potential limiting factors in their population dynamics (NMFS 2008a). Here we focus on the quantity and quality of prey, and the toxic chemicals in the whales because these are affected by the proposed action. The discussion in the Environmental Baseline and Cumulative Effects sections contain a thorough evaluation of all threats in the action area.

Prey. Healthy killer whale populations depend on adequate prey levels. First, we discuss the prey requirements of Southern Residents followed by an assessment of threats to the quantity and quality of their prey.

Prey Requirements. Southern Resident killer whales consume a variety of fish species (22 species) and one species of squid (Scheffer and Slipp 1948; Ford *et al.* 1998, 2000; Ford and Ellis 2006; Saulitis *et al.* 2000; Hanson *et al.* 2010c), but salmon are identified as their primary prey (*i.e.*, a high percent of prey consumed during spring, summer and fall, from long-term studies of resident killer whale diet; Ford and Ellis 2006, Hanson *et al.* 2010c). Feeding records

for Southern and Northern Residents show a predominant consumption of Chinook salmon during late spring to fall (Ford and Ellis 2006). Chum salmon are also taken in significant amounts, especially in fall. Other salmon eaten include coho, pink, steelhead (*O. mykiss*), and sockeye (*O. nerka*). The non salmonids included Pacific herring, sablefish, Pacific halibut, quillback and yelloweye rockfish (*Sebastes maliger*), lingcod (*Ophiodon elongates*), and Dover sole (*Microstomus pacificus*) (Ford *et al.* 1998, Hanson *et al.* 2010c). Chinook salmon were the primary prey despite the much lower abundance of Chinook salmon in the study area in comparison to other salmonids (primarily sockeye), for mechanisms that remain unknown but factors of potential importance include the species' large size, high fat and energy content, and year-round occurrence in the area. Killer whales also captured older (*i.e.*, larger) than average Chinook salmon (Ford and Ellis 2006). Recent research suggests that killer whales are capable of detecting, localizing and recognizing Chinook salmon through their ability to distinguish Chinook salmon echo structure as different from other salmon (Au *et al.* 2010).

Southern Residents are the subject of ongoing research, including direct observation, scale and tissue sampling of prey remains, and fecal sampling. A recent publication by Hanson *et al.* (2010c) provides the best available scientific information on diet composition of Southern Residents in inland waters during summer months. The results provide information on (1) the percentage of Chinook in the whales' diet, and (2) the predominant river of origin of those Chinook. Other research and analysis provides additional information on the age of prey consumed (Hanson, unpubl. data, as summarized in Ward *et al.* 2010), indicating that the whales are consuming mostly larger (*i.e.*, older) Chinook.

Scale and tissue sampling in inland waters from May to September indicate that the Southern Residents' diet consists of a high percentage of Chinook, with an overall average of 88% Chinook across the timeframe and monthly proportions as high as >90% Chinook (*i.e.*, July: 98% and August: 92%, see S/T sample type in Table 2 Hanson *et al.* 2010c). Fecal samples are also available in Hanson *et al.* (2010c) but were not used to estimate proportion of the Southern Residents' diet, because the data from these samples represents presence or absence of prey species, but not proportion of diet. DNA quantification methods can be used to estimate the proportion of diet from fecal samples (*i.e.*, Deagle *et al.* 2005). This technique is still in the developmental stages. However, preliminary DNA quantification results from Hanson *et al.* (2010c) samples indicate that Chinook make up the bulk of the prey DNA in the fecal samples (Ford *et al.* 2011b).

Genetic analysis of the Hanson *et al.* (2010c) samples indicate that when Southern Resident killer whales are in inland waters from May to September, they consume Chinook stocks that originate from regions including the Fraser River (including Upper Fraser, Mid Fraser, Lower Fraser, N. Thompson, S. Thompson and Lower Thompson), Puget Sound (N. and S. Puget Sound), the Central British Columbia Coast and West and East Vancouver Island. Hanson *et al.* (2010c) find that the whales are likely consuming Chinook salmon stocks at least roughly proportional to their local abundance, as inferred by Chinook run-timing pattern and the stocks represented in killer whale prey for a specific area of inland waters, the San Juan Islands. Ongoing studies also confirm a shift to chum salmon in fall (Ford *et al.* 2010a, Hanson *et al.* 2010a).

Although less is known about the diet of Southern Residents off the Pacific coast, the available information indicates that salmon, and Chinook salmon in particular, are also important when the whales occur in coastal waters. To date, there are direct observations of two different predation events (where the prey was identified to species and stock from genetic analysis of prey remains) when the whales were in coastal waters. Both were identified as Columbia River Chinook stocks (Hanson *et al.* 2010b). Chemical analyses also support the importance of salmon in the year round diet of Southern Resident killer whales (Krahn *et al.* 2002, 2007, 2009). Krahn *et al.* (2002), examined the ratios of DDT (and its metabolites) to various PCB compounds in the whales, and concluded that the whales feed primarily on salmon throughout the year rather than other fish species. The predominance of Chinook in their diet in inland waters, even when other species are more abundant, combined with information to date about prey in coastal waters (above), makes it reasonable to expect that Chinook salmon is equally predominant in the whales' diet when available in coastal waters. It is also reasonable to expect that the diet of Southern Residents is predominantly larger Chinook when available in coastal waters. The diet of Southern Residents in coastal waters is a subject of ongoing research.

Quantity of Prey. Human influences have had profound impacts on the abundance of many prey species in the northeastern Pacific during the past 150 years, including salmon. The health and abundance of wild salmon stocks have been negatively affected by altered or degraded freshwater and estuarine habitat, including numerous land use activities, from hydropower systems to urbanization, forestry, agriculture and development. Harmful artificial propagation practices and overfishing have also negatively affected wild salmon stocks. Section 2.4 provides a comprehensive overview of limiting factors for Puget Sound Chinook, as does the Puget Sound Salmon Recovery Plan (Shared Strategy 2007 and NMFS 2007). Predation also contributes to natural mortality of salmon. Salmonids are prey for pelagic fish, birds, and marine mammals including killer whales.

While wild salmon stocks have declined in many areas, hatchery production has supplemented additional prey. Currently, hatchery production contributes a significant component of the salmon prey base returning to watersheds within the range of Southern Resident killer whales (*i.e.*, review PFMC 2011 for Puget Sound, Barnett-Johnson *et al.* 2007 for Central Valley California, and NMFS 2008b for Columbia River Basin). Although hatchery production has contributed some offset of the historical declines in the abundance of wild salmon within the range of Southern Residents, hatcheries also pose risks to wild salmon populations (*i.e.*, Ford 2002, Nickelson *et al.* 1986, Levin and Williams 2002, Naish *et al.* 2007). In recent decades, managers have been moving toward hatchery reform, and are in the process of reducing risks identified in hatchery programs, through region-wide recovery planning efforts and hatchery program reviews. Healthy wild salmon populations are important to the long-term maintenance of prey populations available to Southern Resident killer whales, because it is uncertain whether a hatchery dominated mix of stocks is sustainable indefinitely.

Salmon abundance is also substantially affected by climate variability in freshwater and marine environments, particularly by conditions during early life-history stages of salmon (NMFS 2008b). Sources of variability include inter-annual climatic variations (*e.g.*, El Niño and LaNiña), longer term cycles in ocean conditions (*e.g.*, Pacific Decadal Oscillation, Mantua *et al.* 1997), and ongoing global climate change. For example, climate variability can affect ocean

productivity in the marine environment and water storage (*e.g.* snow pack) and in-stream flow in the freshwater environment. Early life-stage growth and survival of salmon can be negatively affected when climate variability results in conditions that hinder ocean productivity (*e.g.*, Scheuerell and Williams 2005) and/or water storage (*e.g.*, ISAB 2007) in marine and freshwater systems, respectively. Severe flooding in freshwater systems can also constrain salmon populations (NMFS 2008c). The availability of adult salmon may be reduced in years following unfavorable conditions to the early life-stage growth and survival of salmon.

When prey is scarce, whales likely spend more time foraging than when it is plentiful. Increased energy expenditure and prey limitation can cause nutritional stress. Nutritional stress is the condition of being unable to acquire adequate energy and nutrients from prey resources and as a chronic condition can lead to reduced body size and condition of individuals and lower reproductive and survival rates of a population (*e.g.*, Trites and Donnelly 2003). The Center for Whale Research has observed the very poor body condition in 13 members of the Southern Resident population, and all but two of those whales subsequently died (Durban *et al.* 2009). Both females and males across a range of ages were found in poor body condition (Durban *et al.* 2009). Food scarcity could also cause whales to draw on fat stores, mobilizing contaminants stored in their fat that are at relatively high levels (Krahn *et al.* 2007, 2009; Mongillo 2009) and affecting reproduction and immune function (as discussed above).

Here we examine potential symptoms of chronic nutritional stress by considering the available data on poor body condition of individual Southern Residents and discussing demographic modeling conducted to date that identifies Chinook abundance as strongly correlated with changes in demographic rates of the Southern Resident killer whale population.

Body Condition of Whales. The Center for Whale Research is the primary source of data for body condition of Southern Resident killer whales and retains photographs of all individual Southern Resident killer whales identified during annual census. They document body condition with boat-based visual observation and photographs. This technique is not able to detect fine scale differences in condition, because from the dorsal vantage a detectable change is only visible when a whale's condition has become very poor (Durban *et al.* 2009). Very poor condition is detectable by a depression behind the blowhole that presents as a "peanut-head" appearance. The Center for Whale Research has observed the "peanut-head" condition in 13 members of the Southern Resident population, and all but two of those whales subsequently died (Table 2.4.3.2). Durban *et al.* (2009) are currently refining methods to detect changes in body condition at a finer scale with aerial photogrammetry. Ayres *et al.* (2012) also examined potential symptoms of nutritional stress in the whales by measuring fecal hormones.

None of the whales that died were subsequently recovered, and therefore definitive cause of death could not be identified. Both females and males across a range of ages were found in poor body condition (Table 2.4.4.1.2). Regardless of the cause(s) of death, it is possible that poor nutrition could contribute to mortality through a variety of mechanisms. To demonstrate how this is possible, we reference studies that have demonstrated the effects of energetic stress (caused by incremental increases in energy expenditures or incremental reductions in available energy) on adult females and juveniles, which have been studied extensively (*e.g.*, adult females: Gamel *et al.* 2005, Daan *et al.* 1996, juveniles: Noren *et al.* 2009, Trites and Donnelly 2003). Small,

incremental increases in energy demands should have the same effect on an animal's energy budget as small, incremental reductions in available energy, such as one would expect from reductions in prey. Ford and Ellis (2006) report that resident killer whales engage in prey sharing about 76% of the time. Prey sharing presumably would distribute more evenly the effects of prey limitation across individuals of the population than would otherwise be the case (*i.e.*, if the most successful foragers did not share with other individuals). Therefore, although cause of death for these specific individuals is unknown, poor nutrition could contribute to additional mortality in this population.

Demographic Modeling. Ford *et al.* (2005 and 2010b) evaluated 25 years of demographic data from Southern and Northern Resident killer whales and found that changes in survival largely drive their population trends, and the populations' survival rates are strongly correlated with coast-wide availability of Chinook salmon (from Pacific Salmon Commission [PSC] abundance indices that estimate abundance between Southeast Alaska and Oregon). Ward *et al.* (2009) found that Northern and Southern Resident killer whale fecundity is highly correlated with Chinook abundance indices, and reported the probability of calving increased by 50 percent between low and high Chinook abundance years. PSC Chinook abundance indices from the West Coast of Vancouver Island (WCVI) were the most important predictor of the relationship. Recently, Ward (2010) considered new information to update the 2009 fecundity model with new birth data and a singular focus on the Southern Resident killer whale population. Ward (2010) also conducted the updated analysis for survival, where the survival of L pod was evaluated separately from the survival of J and K pods because of the apparent lower survival in L pod (Ward *et al.* 2011, Krahn *et al.* 2004). Best-ranked models all included one of the PSC Chinook indices (the Northern British Columbia indices performed best, and WCVI, Southeast Alaska and inland WCVI indices performed equally well at second best). The results are consistent with findings from Ford *et al.* 2010b.

Quality of Prey. The quality of Chinook salmon, Southern Resident killer whales' primary prey, is likely influenced by a variety of factors, including contaminant load, size of the fish, their fat content, and origin (natural vs. hatchery). Overall, Chinook have the highest lipid content (Stansby 1976, Winship and Trites 2003), largest size, and highest caloric value per kg of any salmonid species (Ford and Ellis 2006, Osborne 1999). Details about contaminant load, size, and origin are provided below.

Table 2.4.4.1.2. Dates of observed "peanut-head" condition of individual Southern Resident killer whales and their fates (Durban *et al.* 2009).

Year	Whale ID	Whale Sex/Age	Description	Fate
1994	L42	M / 21	A slight depression behind the blowhole was first noticed in mid-June; a prominent depression by mid-July; the dorsal fin was drooping by mid August; the depression had become large by early September exposing the shape of the back of the cranium and vertebrae; last seen in late September.	Died
	K17	M / 28	A slight depression behind the blowhole was first noticed in mid July; prominent depression by mid August; last seen in mid September with the fin severely drooping.	Died
1995	J3	M / 43	A slight depression behind the blowhole noticeable by the end of March; moderate depression by mid May with the fin beginning to droop; last seen late May.	Died
	L63	M / 11	A prominent depression behind the blowhole noticeable by late July; last seen late July.	Died
	L68	M / 10	A moderate depression behind the blowhole was noticeable in mid May; depression prominent by mid June; last seen in late June.	Died
1996	J12	F / 24	A slight depression behind the blowhole first noticed in mid February; depression moderate by April with the base of the cranium apparent; prominent depression by early June, with ribs beginning to show on flanks; depression very prominent by early September, revealing the shape of the base of the cranium and vertebrae, and ribs visible on flanks showing; last seen late September.	Died
	L9	F / 65	A slight depression behind the blowhole noticeable in early July; depression prominent by mid August, exposing the shape of the base of the cranium; last seen mid August.	Died
1997	J5	F / 59	A slight depression noticeable in early April; last seen early April.	Died
2002	L102	Unk / Calf	Moderate depression behind the blowhole noticeable in early December- only time the calf was seen; last seen early December.	Died
2005	K25	M / 14	A moderate depression was noticeable behind the blowhole in late July, with a laceration on the whale's back following a collision with a whale-watch boat in early July; depression slight by early September; whale survived.	Survived
2006	K28	F / 12	A prominent depression behind the blowhole was noticeable in mid September; whale not seen afterward.	Died
2008	L106	M / 3	A prominent depression behind the blowhole was noticeable in mid June; depression just slight by mid July; depression barely noticeable by early August; whale survived the year, and seen in early 2009.	Survived
	L67	F / 23	A slight depression behind the blowhole was first noticeable in late June; depression still slight in early August; depression prominent by mid September; last seen mid September.	Died

Contaminant Load. Levels of persistent organic pollutants (POPs) in killer whales are primarily determined by contaminant levels in their prey and the geographic region, although the age, gender, and birth order of the whale will also influence accumulation. Various studies have documented a range of concentrations of POPs in many populations of adult Pacific salmon (see Table 2.4.4.1.3). POP accumulation in Pacific salmon is primarily determined by geographic proximity to contaminated environments (Mongillo *et al.* in prep.). Because Chinook salmon are distributed in more coastal waters, they are more readily exposed to contaminants that are present in coastal waters than other species. In contrast, sockeye, pink, and chum salmon have lower POP concentrations because by the end of their first year, they have migrated through the coastal waters and are found in the open waters of the North Pacific, Gulf of Alaska, and Bering Sea (Quinn 2005). Measured average concentrations of PCBs and polybrominated diphenyl ethers (PBDEs) were highest for Chinook intermediate for coho, less for sockeye, and lowest for pink and chum salmon (see Table 2.4.4. 1.3). Similarly, average DDT values were higher in Chinook and coho salmon compared to sockeye and lowest for pink and chum salmon (see Table 2.4.4. 1.3). Intermediate levels of PCB and PBDEs were measured in California and Oregon populations and the lowest average levels were measured in populations off Alaska (Mongillo *et al.* in prep.). The biological traits in Pacific salmon (*e.g.* trophic status, lipid content, age, exposure duration, metabolism, and detoxification) may also affect the degree to which POPs accumulate (Mongillo *et al.* in prep.).

Size. Size of individual salmon is an aspect of prey quality that could affect the foraging efficiency of Southern Resident killer whales. As discussed above, available data suggests that Southern Residents consume larger prey. The degree to which this is a function of the availability of all sizes of fish in the coastal range of the whales, their ability to detect all sizes or a true preference of only large fish is unknown. It is possible although not conclusive that there has been a historical decrease in salmon age, size, or size at a given age (*i.e.*, Bigler *et al.* 1996, but also see PFMC data (PFMC 2011). Fish size is influenced by factors such as environmental conditions, selectivity in fishing effort through gear type, fishing season or regulations, and hatchery practices. The available information on size is also confounded by factors including inter-population difference, when the size was recorded, and differing data sources and sampling methods (review in Quinn 2005).

Origin. Southern Resident killer whales likely consume both natural and hatchery salmon (Hanson *et al.* 2010c). The best available information does not indicate that natural and hatchery salmon generally differ in size, run-timing, or ocean distribution (*e.g.*, Nickum *et al.* 2004, NMFS 2008c, Weitkamp and Neely 2002, regarding differences that could affect Southern Residents); however, there is evidence of size and run-timing differences between hatchery and natural salmon from specific river systems or runs (*i.e.*, size and run timing differences as described for Willamette River Chinook in NMFS 2008d). Potential run-specific differences in the quality of natural and hatchery salmon are evaluated where data are available.

Table 2.4.4. 1.3. Lipid and persistent organic pollutant concentrations (ng/g wet weight) of adult and subadult Pacific salmon sampled in terminal areas. Terminal areas include coastal marine water and river mouths through which fish migrate en route to their natal stream. From Mongillo *et al.* (in prep).

Species	Region	sub-region	Population	n	Tissue Analyzed	Lipid (%)	PCBs	DDTs	PBDEs	Citation
Chinook salmon	Alaska	unknown	unknown	2	muscle w/o skin	NR	5.6	NR	0.95	4
	Alaska	Aleutian Islands	unknown	3	muscle w/skin	7.6	5.0	22	0.71	13, 14*
	Alaska	SE Alaska/ Gulf of Alaska/ Bering Sea	unknown	35	muscle w/o skin	9.7	11	7.1	0.53	20
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	8.0	NR	0.50	5*, 6*
	Alaska	South Central	River	10	muscle w/o skin	NR	9.1	9.8	NR	12
		Alaskan Chinook salmon Average				8.7	7.7	13.0	0.67	
	British Columbia	BC North Coast	Skeena	30	whole body	NR	7.3	7.3	0.08	10
	British Columbia	Fraser River	Thompson	6	muscle w/o skin	10	9.1	1.5	NR	1
	British Columbia	Fraser River		13	whole body	NR	9.4	6.6	0.80	10
	British Columbia	Fraser River	Thompson	7	muscle w/o skin	12	8.6	7.7	1.54	16**
	British Columbia	Fraser River	Shuswap	2	muscle w/o skin	3.0	9.8	5.5	NR	16**
	British Columbia	Fraser River	Harrison	6	muscle w/o skin	5.4	47	4.3	17.7	1
		Fraser River Chinook salmon Average (excluding Harrison)				8.3	10	5.7	1.67	
		British Columbia Chinook salmon Average				7.6	15	5.5	4.87	
	Washington	Puget Sound	Nooksack River	28	muscle w/o skin	3.5	37	NR	NR	11
	Washington	Puget Sound	Skagit River	29	muscle w/o skin	4.8	40	NR	NR	11
	Washington	Puget Sound	Duwamish River	65	muscle w/o skin	7.3	56	NR	NR	11
	Washington	Puget Sound	Nisqually River	20	muscle w/o skin	3.8	41	NR	NR	11
	Washington	Puget Sound	Deschutes River	34	muscle w/o skin	1.7	59	NR	NR	11
	Washington	Puget Sound	PS mixed	28	muscle w/o skin	4.8	76	NR	NR	11
	Washington	Puget Sound	Duwamish River	3	whole body	6.4	35	18.3	6.43	1
	Washington	Puget Sound	Deschutes River	4	whole body	4.3	56	NR	NR	1
	Washington	Puget Sound	Deschutes River	10	muscle w/o skin	1.0	49	NR	NR	8
	Washington	Puget Sound	Issaquah Creek	10	muscle w/o skin	0.6	49	NR	NR	8
	Washington	Puget Sound	PS mixed	36	whole body	NR	43	29.1	18.9	10

Species	Region	sub-region	Population	n	Tissue Analyzed	Lipid (%)	PCBs	DDTs	PBDEs	Citation
	Washington	Puget Sound	PS mixed	34	whole body	NR	91	16.4	42.2	10
	Washington	WA Coast	Makah	10	muscle w/o skin	1.5	19	NR	NR	8
	Washington	WA Coast	Quinault	10	muscle w/o skin	1.8	16	NR	NR	8
		Puget Sound Chinook salmon Average				3.8	53	21.3	22.5	
		Washington Coast Chinook salmon Average				1.7	17	NR	NR	
		Washington Chinook salmon Average				3.5	48	21.3	22.5	
	Oregon	unknown	unknown	3	muscle w/skin	NR	10	NR	2.10	5*, 6*
	Oregon	Columbia River	unknown Fall	17	whole body	NR	18	19.9	3.69	10
	Oregon	Columbia River	unknown Spring	20	whole body	NR	33	34.8	9.77	10
	Oregon	Columbia River	mixed fall Chinook	15	muscle w/skin	7.0	37	21.0	NR	17
	Oregon	Columbia River	mixed spring Chinook	24	muscle w/skin	9.0	38	22.0	NR	17
	Oregon	Columbia River	fall Chinook	4	whole body	9.4	15	NR	2.30	15
	Oregon	Columbia River	Clackamas River	3	muscle w/skin	8.8	13	NR	1.80	15
	Oregon	Columbia River	Clackamas River	3	muscle w/o skin	6.1	10	NR	1.50	15
		Oregon Chinook salmon average				8.1	22	24.4	3.53	
	California	Sacramento /San Joaquin	unknown	29	whole body	NR	14	33.6	2.56	10
	Chinook salmon Average					5.6	29	15.7	6.22	
Sockeye salmon	Alaska	unknown	Alaska	2	muscle w/o skin	NR	3.6	NR	0.21	4
	Alaska	Aleutian Islands	unknown	13	muscle w/o skin	5.8	130	6.9	NR	3
	Alaska	Kodiak	unknown	3	muscle w/skin	NR	5.0	NR	0.10	5*, 6*
	Alaska	Gulf of Alaska/ Berring Sea	unknown	24	muscle w/o skin	8.2	13	12.0	0.22	20
	Alaska	Gulf of Alaska/ Berring Sea	Copper River	97	muscle w/o skin	5.5	37	12.2	NR	18**
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	13.3	NR	0.10	5*, 6*
		Alaskan sockeye salmon average				6.5	14.4#	10.4	0.16	
	British Columbia	unknown	unknown	3	muscle w/skin	NR	8.0	NR	0.10	5*, 6*
	British Columbia	Fraser River	Early Stuart	3	soma	16	13	NR	NR	7**
	British Columbia	Fraser River	Early Stuart	5	muscle w/o skin	4.0	3.9	NR	NR	7**
	British Columbia	Fraser River	Early Stuart	6	muscle w/o skin	5.0	6.9	NR	NR	7**
	British Columbia	Fraser River	Adams	5	muscle w/o skin	8.8	7.7	6.6	NR	16**

Species	Region	sub-region	Population	n	Tissue Analyzed	Lipid (%)	PCBs	DDTs	PBDEs	Citation
	British Columbia	Fraser River	Weaver Creek	3	muscle w/o skin	1.4	6.8	NR	NR	7**
	British Columbia	Fraser River	Weaver Creek	2	muscle w/o skin	1.1	3.6	NR	NR	7**
	British Columbia	Fraser River	Weaver Creek	2	muscle w/o skin	1.5	5.3	NR	NR	7**
	British Columbia	Fraser River	Weaver Creek	1	muscle w/o skin	1.1	4.0	NR	NR	7**
	British Columbia	Fraser River	Weaver	8	muscle w/o skin	3.9	6.8	5.4	NR	16**
	British Columbia	West Coast VI	Great Central Lk.	6	muscle	6.1	1.7	NR	NR	7**
	British Columbia	West Coast VI	Great Central Lk.	3	muscle	6.6	1.6	NR	NR	2**
	British Columbia	West Coast VI	Great Central Lk.	2	muscle	1.0	1.5	NR	NR	2**
	British Columbia	West Coast VI	Great Central Lk.	3	muscle	1.0	2.4	NR	NR	2**
	British Columbian sockeye salmon Average					4.4	5.2	6.00	0.10	
	Sockeye salmon Average					4.8	7.6#	8.6	0.15	
Steelhead	Oregon	Columbia River		21	muscle w/skin	6.0	34	21.0	NR	17
Coho Salmon	Alaska	unknown	unknown	2	muscle w/o skin	NR	1.6	NR	0.32	4
	Alaska	Kodiak	unknown	3	muscle w/skin	NR	4.0	NR	0.10	5*, 6*
	Alaska	seak/goa	unknown	14	muscle w/o skin	2.9	2.0	1.5	0.19	20
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	4.0	NR	0.10	5*, 6*
	Alaskan coho salmon Average					2.9	2.9	1.5	0.18	
	British Columbia	unknown	unknown	3	muscle w/skin	NR	6.0	NR	0.30	5*, 6*
	Washington	Puget Sound	unknown	32	muscle w/o skin	3.1	35	NR	NR	9
	Washington	Puget Sound	PS mixed	125	muscle w/o skin	3.1	27	NR	NR	9
	Washington	Puget Sound	PS mixed	266	muscle w/o skin	3.3	NR	11.7	NR	19
	Washington coho salmon Average					3.2	31	11.7	NR	
	Oregon	Columbia River	Umatilla River	3	muscle w/skin	2.5	35	41.0	NR	17
	Coho salmon Average					3.0	14	18.1	0.20	
Pink salmon	Alaska	Kodiak	unknown	3	muscle w/skin	NR	3.0	NR	0.10	5*, 6*
	Alaska	northern Alaska	unknown	7	canned	6.3	2.6	1.8	NR	21
	Alaska	SE Alaska/GOA	unknown	12	muscle w/o skin	3.5	1.3	0.6	0.22	20
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	2.0	NR	0.10	5*, 6*
	Alaskan pink salmon Average					4.9	2.2	1.2	0.14	
	British Columbia	unknown	unknown	3	muscle w/skin	NR	3.0	NR	0.30	5*, 6*

Species	Region	sub-region	Population	n	Tissue Analyzed	Lipid (%)	PCBs	DDTs	PBDEs	Citation
	Pink salmon Average					4.9	2.4	1.2	0.18	
Chum salmon	Alaska	Kodiak	unknown	3	muscle w/skin	NR	2.0	NR	0.10	5*, 6*
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	3.0	NR	0.10	5*, 6*
	Alaska	Bering Sea	unknown	18	muscle w/o skin	4.8	3.2	1.9	0.16	20
	Alaskan chum salmon Average					4.8	2.7	1.9	0.12	
	British Columbia	unknown	unknown	3	muscle w/skin	NR	2.0	NR	0.20	5*, 6*
	Chum salmon Average					4.8	2.6	1.9	0.14	
(1) Cullon *et al.* 2009, (2) Debruyn *et al.* 2004, (3) Hardell *et al.* 2010, (4) Hayward *et al.* 2007, (5) Hites *et al.* 2004a, (6) Hites *et al.* 2004b,										
(7) Kelly *et al.* 2007, (8) Missildine *et al.* 2005, (9) O'Neill *et al.* 1998, (10) O'Neill *et al.* 2006, (11) O'Neill and West 2009,										
(12) Rice and Moles 2006, (13) Shaw *et al.* 2008, (14) Shaw *et al.* 2006, (15) Stone 2006, (16) Veldhoen *et al.* 2010,										
(17) US EPA 2002, (18) Ewald *et al.* 1998, (19) West *et al.* 2001, (20) ADEC 2011, (21) O'Hara *et al.* 2005										
* estimated values from figure										
** estimated value from reported lipid weight										
#excluded value as an outlier										

Toxic Chemicals. Contaminants enter fresh and marine waters and sediments from numerous sources such as atmospheric transport and deposition, ocean current transport, and terrestrial runoff (Iwata *et al.* 1993, Grant and Ross 2002, Hartwell 2004), but are typically concentrated near populated areas of high human activity and industrialization. Oceans act as a repository for domestic and industrial wastes and significant contaminant concentrations have been measured in the sediment, water, and biota. Persistent contaminants can biomagnify or accumulate up the food chain in such a degree where levels in upper trophic-level mammals can have significantly higher concentrations than that found in the water column or in lower trophic-level species. Southern Resident killer whales are exposed to relatively high levels of persistent pollutants because they are long-lived, upper trophic-level predators that are in close proximity to industrial and agricultural areas. Consequentially, Southern Residents are a highly contaminated whale population.

Persistent pollutants are highly lipophilic (*i.e.*, fat soluble) and are primarily stored in the fatty tissues in marine mammals (O'Shea 1999, Reijnders and Aguilar 2002). Therefore, when killer whales consume contaminated prey they store the contaminants primarily in their blubber. However, some persistent contaminants (*e.g.*, the butyltins) are primarily stored in the liver and kidneys of marine mammals (Iwata *et al.* 1997). Persistent pollutants can resist metabolic degradation and can remain stored in the tissues or organs of an individual whale for extended periods of time. When prey is scarce and when other stressors reduce foraging efficiency (*e.g.*, as possible from vessel disturbance, disease, *etc.*), killer whales metabolize their blubber lipid stores and the contaminants can become mobilized to other organs or they can remain in the blubber and become more concentrated (Krahn *et al.* 2002). Nursing mothers can also transmit large quantities of contaminants to their offspring, particularly during lactation. The mobilized contaminants can reduce the whales' resistance to disease, can affect reproduction, disrupt the endocrine system, disrupt enzyme function and vitamin A physiology, induce developmental neurotoxicity, and cause skeletal deformities (see NMFS 2008a for a review).

There are several persistent pollutants of concern that have been highlighted in the Southern Resident killer whale Recovery Plan (Table 2.4.4. 1.4). Some of these pollutants do not need to be in high concentration in a species to be toxic and have long been recognized as problematic for the Southern Resident killer whales. The organochlorines (*e.g.*, PCBs and DDTs) are thought to pose the greatest risk to killer whales (Ross *et al.* 2000, Center for Biological Diversity 2001, Krahn *et al.* 2002). Organochlorines are a diverse group of lipophilic compounds. Designed for their stability, most are highly persistent in the environment and can resist metabolic degradation. These persistent pollutants can accumulate in the food webs and are at relatively high concentrations in upper trophic-level species such as killer whales. PCBs were designed for chemical stability and were historically used in paints and sealants, industrial lubricants and coolants, and flame-retardants. DDTs were primarily used to control insects in commercial and agricultural areas, forests, homes and gardens. PCBs and DDTs were banned in the 1970s and 1980s due to their toxicity in humans and wildlife. Although levels of PCBs and DDTs have dramatically decreased in environmental samples since the mid 1970s (Mearns *et al.* 1988, Lieberg-Clark *et al.* 1995, Calambokidis *et al.* 2001, Rigét *et al.* 2010), these compounds continue to be measured in marine biota around the world, including killer whales and their prey.

Many studies have found organochlorines in marine mammal tissues (*e.g.*, Appendices 10-1 through 10-4, O'Shea 1999). Several marine mammal populations have high levels of organochlorines associated with adverse health effects. For example, the St. Lawrence beluga population contains high levels of organochlorines, as well as lead, mercury, and selenium (Martineau *et al.* 1987, Muir *et al.* 1990, Wagemann *et al.* 1990). This beluga whale population has a high prevalence for tumors, and lesions in the digestive tract and mammary glands, which are thought to be associated with the high levels of contaminants, particularly PCBs (Martineau *et al.* 1994, De Guise *et al.* 1995).

The majority of Southern Residents have high levels of PCBs (Ross *et al.* 2000, Krahn *et al.* 2007a, 2009) that exceed a health-effects threshold (17,000 ng/g lipid) derived by Kannan *et al.* (2000) and Ross *et al.* (1996) for PCBs in marine mammal blubber. The PCB health-effects threshold is associated with reduced immune function and reproductive failure in harbor seals (Reijnders 1986, de Swart *et al.* 1994, Ross *et al.* 1996, Kannan *et al.* 2000). Hickie *et al.* (2007) projected that it will take at least 50 years for the Southern Residents to drop below the threshold. Moreover, juvenile Southern Resident killer whales had blubber concentrations that were 2 to 3.6 times higher than the established health-effects threshold (Krahn *et al.* 2009). Similarly, Southern Residents also have high levels of measured DDTs in their blubber (Krahn *et al.* 2007a, 2009).

Recent decades have brought rising concern over a list of the so-called "emerging" contaminants and other pollutants, such as the PBDEs. PBDEs have been used as additive flame-retardants in many products including electronics, textiles, and plastics. Additive flame-retardants can readily disassociate from the products they are added to and discharge into the environment. Due to the increase in fire regulations in many countries, the use of PBDEs has increased in the last few decades. PBDEs have been identified as a growing concern and have a ubiquitous distribution with increasing levels found in various matrices including surface water, sewage sludge, sediment, air, and biota (Hale *et al.* 2003, Hites 2004). PBDEs are structurally comparable to PCBs and share some similar toxicological properties (Hooper and McDonald 2000). In January 2006, the Washington State Department of Ecology (DOE) and the Washington State Department of Health (DOH) issued a Final PBDE Chemical Action Plan (DOE and DOH 2006) that recommended the Legislature prohibit the three main types of PBDEs used in consumer products (*e.g.*, penta-, octa-, and deca-BDEs). The penta and octa forms are currently being phased out in Washington State because manufacturers agreed to voluntarily stop producing these two forms of PBDEs by the end of 2004, and following a bill (ESHB1024) that was passed in 2007. This bill banned the use of the penta and octa forms by 2008, banned the use of the deca form in mattresses by 2008, and banned the use of the deca form in televisions, computers, and furniture by 2011.

Although specific regional data is limited for PBDE levels, the environmental levels of a few PBDE congeners appear to have surpassed PCBs in some areas in North America (Hale *et al.* 2003, Ross *et al.* 2009). Recent studies have documented relatively high concentrations of PBDEs in Southern Resident killer whales (Krahn *et al.* 2007a, 2009, Mongillo 2009). Although PBDE levels in the whales are lower than PCBs or DDTs (Krahn *et al.* 2007a, 2009), concern is growing because PBDE exposure and accumulation will likely continue in the future increasing the risk to the health of the killer whales. Several other marine species have recently experienced

an almost exponential increase in PBDE concentrations (*e.g.*, Ikonomou *et al.* 2002, Lebeuf *et al.* 2004).

Recent studies suggest that certain pharmaceuticals and personal care products (PPCPs) may also accumulate in killer whales. Synthetic musks and antibacterial chemicals (*e.g.* Triclosan) have been detected in dolphins and porpoises in coastal waters off Japan and the southeastern United States and in harbor seals off the California Coast (Fair *et al.* 2009, Kannan *et al.* 2005, Nakata 2005, Nakata *et al.* 2007). A wider range of PPCPs, including anti-depressants, cholesterol lowering drugs, antihistamines, and drugs affecting blood pressure and cholesterol levels have been detected in tissues of fish from urban areas and sites near wastewater treatment plants (Brooks *et al.* 2005, Ramirez *et al.* 2009), suggesting possible contamination of prey. As yet we have no data on concentrations of PPCPs in either killer whales or their prey species, but they could be a concern because of their widespread occurrence, potential for biomagnification, and biological activity.

Table 2.4.4. 1.4. Persistent pollutants that may pose a risk to resident killer whales. From Table 1 in Killer Whale Recovery Team (2007). Updated from NMFS (2008a).

Pollutant	Use/Source	Persistent	Bio-accumulate	Risk
DDT (Dichlorodi-phenyl trichloroethane	pesticide used in some countries, banned in North America, persists in terrestrial runoff 30 years post ban, enters atmosphere from areas where still in use	yes	yes	Reproductive impairment, immunosuppression, adrenal and thyroid effects
PCBs Polychlorinated Biphenyls	electrical transformer and capacitor fluid, limited use in North America but enters environment from runoff, spills and incineration	yes	yes	reproductive impairment, skeletal abnormalities, immunotoxicity and endocrine disruption
Dioxins and Furans	by-product of chlorine bleaching, wood product processing and incomplete combustion. Mills less of a source now. Current sources include burning of salt-laden wood, municipal incinerators, and residential wood and wood waste combustion, in runoff from sewage sludge, wood treatment	yes	yes	thymus and liver damage, birth defects, reproductive impairment, endocrine disruption, immunotoxicity and cancer
PAHs Persistent Polycyclic aromatic hydrocarbons	by-product of fuel combustion, aluminum smelting, wood treatment, oil spills, metallurgical and coking plants, pulp and paper mills	yes	no	Carcinogenic
flame retardants, esp. PBBs and PBDEs Polybrominated diphenyl ethers	flame retardants; in electrical components and backings of televisions and computers, in textiles and vehicle seats, ubiquitous in environment. 2/3 product PBDEs banned in Europe. Same two products withdrawn from North American marketplace in 2005, but one (deca) product still used globally.	yes	yes	endocrine disruption, impairs liver and thyroid
PFOs Perfluro-octane sulfonate	stain, water and oil repellent (included in Scotchgard until recently), fire fighting foam, fire retardants, insecticides and refrigerants, ubiquitous in environment	yes	yes but in blood, liver, kidney and muscle	promotes tumor growth
TBT, DBT Tributyltin Dibutyltin	antifoulant pesticide used on vessels	yes	yes	unknown but recently associated with hearing loss

Pollutant	Use/Source	Persistent	Bio-accumulate	Risk
PCPs (Polychlorinated paraffins)	flame retardants, plasticizers, paints, sealants and additives in lubricating oils	yes	yes	endocrine disruption
PCNs Polychlorinated napthalenes	ship insulation, electrical wires and capacitors, engine oil additive, municipal waste incineration and chlor-alkali plants, contaminant in PCBs	yes	yes	endocrine disruption
APEs Alkyl-phenol ethoxylates	detergents, shampoos, paints, pesticides, plastics, pulp and paper mills, textile industry found in sewage effluent and sediments	moderate	moderate	endocrine disruption
PCTs Polychlorinated terphenyls	fire retardants, plasticizers, lubricants, inks and sealants, enters environment in runoff	yes	yes	endocrine disruption and reproductive impairment
References: Primarily Grant and Ross 2002, but also Lindstrom *et al.* 1999, Hooper and MacDonald 2000, Kannan *et al.* 2001, Hall *et al.* 2003; Van de Vijver *et al.* 2003, Rayne *et al.* 2004, Song *et al.* 2005.				

Below we highlight the available information about marine mammal toxicity, storage, concentration levels, and detoxification mechanisms for toxic chemicals considered in the proposed action, as introduced in Table 1.1. We first discuss the organic compounds: dieldrin, endrin, endosulfan, heptachlor epoxide, Lindane, pentachlorophenol (PCP), and tributyltin (TBT). Second, we discuss the metals and elemental pollutants: cadmium, lead, aluminum, ammonia, arsenic, copper, chromium (III and VI), nickel, selenium, silver, and zinc. Of all the chemicals described below that are a part of this action, the organic compounds are of highest concern, followed by the metals and elemental pollutants.

Dieldrin and Endrin. Dieldrin and endrin are organochlorine insecticides that are more acutely toxic than DDT. They are highly neurotoxic and can cause reproductive defects in laboratory mammals (O'Shea 1999). Reproductive effects can include reduced fertility, reduced litter size, and increased pup mortality in mice, rats, and dogs (AMAP 1998). Furthermore, dieldrin has shown to be estrogenic, cause immunosuppression in laboratory animals, and increase benign and malignant tumors in mice (AMAP 1998).

By the end of the 1960s, dieldrin had been reported in tissues of marine mammals (O'Shea and Tanabe 2003). Dieldrin is commonly found in marine mammals throughout the world, whereas endrin, which is more toxic, is reported less often (see Appendices 10-1 to 10-4, O'Shea 1999). In the late 1980s, dieldrin was measured in the tissues of killer whales of the west coast of North America (Jarman *et al.* 1996). Concentration values revealed a geometric mean of 340 μg/kg wet weight (ww); this average level was appreciably less than the total DDT (32,000 μg/kg ww) and total PCB (22,000 μg/kg ww) in the six killer whales that were sampled (Jarman *et al.* 1996). Similarly, in a separate study, dieldrin levels in stranded or dead North Atlantic killer whales were measurably less than PCBs and DDTs (McHugh *et al.* 2007). Ylitalo *et al.* (2009) measured persistent organic pollutant concentrations including dieldrin in the false killer whale from the Hawaiian Islands. Dieldrin measured in these whales were relatively low. Subadults had significantly higher mean dieldrin levels compared to those measured in other age classes. Concentrations of dieldrin measured in blubber of Southern Residents sampled from 2004-2007 ranged from 9.2 ng/g wet weight (ww) to 440 ng/g ww, whereas the lipid-normalized levels ranged from 32 ng/g lipid to 1,100 ng/g lipid (G. Ylitalo NWFSC, pers. comm.).

Endosulfan. Endosulfan is a semi-volatile and relatively persistent organochlorine. It has shown to be estrogenic and cause reproductive effects in laboratory animals (AMAP 1998). It has high acute oral and inhalation toxicity as well as moderate dermal toxicity in humans (http://www.epa.gov/oppsrrd1/REDs/factsheets/endosulfan_fs.htm). Small and Solomon (2005) concluded that risk from endosulfan in marine mammals was negligible because the range of exposure concentrations were lower than the no observed adverse effect level (NOAEL) doses in laboratory species (*e.g.*, rat and grey partridge, see Figure 2.4.4. 1.3).

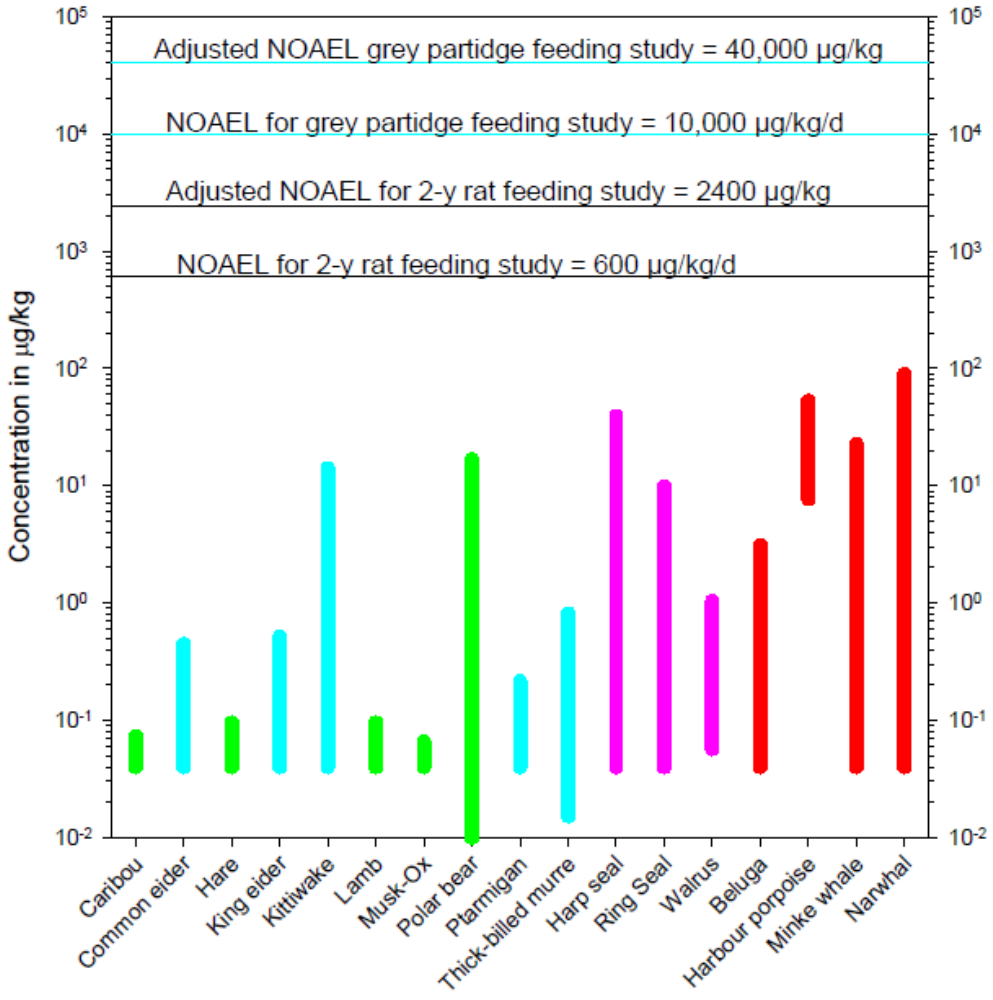

Figure 2.4.4. 1.3. Range of exposure concentrations measured in various polar marine and terrestrial wildlife species as compared to NOAEL doses in test species (reprinted from Small and Solomon 2005).

Endosulfan is present in several cetaceans such as the narwhal, beluga, and minke whales (Vorkamp *et al.* 2004, Small and Solomon 2005). The beluga whale appears to have varying levels depending on geographic location but no significant difference in concentration between sexes (Stern *et al.* 2005). Several studies focusing on the Arctic have shown the continued deposition of endosulfan from use at lower latitudes. Endosulfan is one of the few persistent organic pollutants that increased in concentration from the 1970s to the 1990s in the Canadian Arctic (Braune *et al.* 2005). However, there appears to be uncertainty in some of the datasets because of differences in analytical techniques (Weber *et al.* 2010). Endosulfan I (alpha endosulfan) levels in the blubber of false killer whales from the Hawaiian islands were below the limits of quantification (Ylitalo *et al.* 2009). Alpha endosulfan levels determined in blubber of the Southern Residents sampled between 2004 – 2007 were below the limits of quantification (< 2.2 - < 14 ng/g ww) for all samples analyzed and thus do not appear to currently pose a health risk (G. Ylitalo NWFSC, pers. comm.).

Heptachlor Epoxide. Heptachlor epoxide is a more toxic metabolite of heptachlor (which is prepared from chlordane and has a higher acute toxicity). Laboratory animals fed high levels in a short time period experienced tremors and convulsions (EPA 2008). Long term exposure can lead to liver and kidney tissue damage, enlarged liver, increased red blood cells, and liver cancer (EPA 2008).

Similar to dieldrin, heptachlor epoxide is found in marine mammals throughout the world but in relatively low concentrations (O'Shea 1999). Heptachlor epoxide can be offloaded from mother to offspring and is the primary metabolite of heptachlor found in marine mammals tissues (see Appendices 10-1 through 10-4, O'Shea 1999). In the late 1980s, heptachlor epoxide was measured in the tissues of killer whales of the west coast of North America (Jarman *et al.* 1996). Concentration values revealed a geometric mean of 120 μg/kg ww, respectively, which were appreciably less than DDTs and PCBs (Jarman *et al.* 1996). Blubber levels of heptachlor epoxide measured in Southern Residents sampled from 2004 – 2007 ranged from < 5.3 ng/g ww to 660 ng/g ww whereas the lipid-normalized values ranged from below the limits of quantification to 5,400 ng/g lipid (G. Ylitalo NWFSC pers. commun.).

Lindane. Hexachlorocyclohexane (HCH), also referred to as benzene hexachloride (BHC), is an organochlorine insecticide and consists of a number of isomers: γ-HCH (Lindane), α-HCH, and β-HCH. Lindane is the most biologically active isomer and is a neurotoxin; it affects the nervous system, liver and kidneys, and may act as an endocrine disruptor (http://www.epa.gov/oppsrrd1/REDs/factsheets/lindane_fs_addendum.htm). HCH isomers have caused tumors in laboratory mammals (O'Shea 1999). Lindane has shown to reduce immune responses in laboratory animals and may have both estrogenic and antiestrogenic effects (AMAP 1998).

Between 1986 and 1989, the average concentration of total HCHs (or the sum of Lindane, α-HCH, and β-HCH) measured in killer whales from the west coast of North America was 708 μg/kg ww, of that, the average lindane concentration was only 31 μg/kg ww (Jarman *et al.* 1996). More recently, total HCH was measured in Southern Resident killer whales (Krahn *et al.* 2007a, 2009). Similar to the previous study, total HCHs were measurably lower than PCBs or DDTs. The juvenile whales had significantly higher HCH levels than adult males and total HCH levels were strongly correlated with total PBDEs and did not correlate with age (Krahn *et al.* 2007a, 2009). Lindane concentrations in killer whales are relatively low, likely because it is less bioaccumulative than some of the other organochlorines, and it is potentially regulated by the whales' metabolic system (McHugh *et al.* 2007). Concentrations of total HCHs in the Southern Residents ranged from 62 ng/g to 1,700 ng/g lipid based on biopsy blubber samples collected from 2004 to 2007 (Table 2.4.4. 1.5).

Table 2.4.4. 1.5. Persistent organic pollutants (ng/g lipid) and percent lipid in blubber of biopsy samples from Southern Resident killer whales (data from Krahn *et al.* 2007a, 2009).

Whale ID	Age	Sex	Lipid %	ΣPCBs	ΣDDTs	ΣPBDEs	ΣHCHs
J39	3	M	40.9	34,000	24,000	15,000	1,300
J38	4	M	20.9	41,000	24,000	14,000	1,000
J22	22	F	28.4	4,600	1,500	880	62
J19	27	F	29.4	45,000	26,000	7,500	310
K36	4	F	18.3	62,000	95,000	15,000	1,700
K34	6	M	22.3	39,000	61,000	10,000	1,200
K21	21	M	26.6	38,000	73,000	2,900	410
K13	35	F	22	8,900	11,000	1,200	300
K7	est 97	F	28.5	120,000	44,000	6,700	1,100
L78	15	M	15.2	22,000	38,000	2,600	630
L85	15	M	24.8	50,000	120,000	2,500	530
L87	15	M	25.6	24,000	44,000	2,600	410
L71	18	M	9.6	36,000	72,000	2,600	920
L74	18	M	18	45,000	86,000	3,100	720
L73	21	M	23.8	32,000	55,000	3,400	450
L67	22	F	29.2	5,600	4,300	680	150
L57	29	M	19.4	56,000	110,000	3,300	640
L26	est 51	F	22.1	17,000	27,000	4,400	580
L21	est 57	F	18.7	55,000	99,000	4,200	750

Total HCH levels in Southern Resident killer whales are generally higher than resident killer whales from Central Aleutian Islands, and less than transient killer whales from the Eastern Aleutian Islands (EAI) and from California (Krahn *et al.* 2007b). In fact, the transients from the EAI had significantly higher total HCHs than all other whale groups sampled (Krahn *et al.* 2007b). Herman *et al.* (2005) also found higher total HCH levels in transient killer whales from the eastern North Pacific (mean of 11,500 ng/g lipid) compared to residents (mean of 470 ng/g lipid) followed by the offshore ecotype (mean of 120 ng/g lipid). Relatively low levels of HCH are not uncommon in other killer whale populations. In a separate study, organochlorines were measured in live stranded or dead North Atlantic killer whales (McHugh *et al.* 2007). Similar to previous studies, lindane in individual blubber tissues were relatively low compared to PCBs and DDTs. Blubber levels of Lindane measured in Southern Residents sampled from 2004 – 2007 ranged from < 1.9 ng/g ww to 17 ng/g ww, whereas the lipid-normalized valued ranged from below the limits of quantification to 42 ng/g lipid (G. Ylitalo NWFSC pers. commun.).

Pentachlorophenol (PCP). Pentachlorophenol (PCP) is an organochlorine pesticide and disinfectant, however its greatest use is as a fungicide (wood preservative). PCP is still currently used, but to a lesser degree than in the 1990s. The use of chlorophenol-based chemicals for wood treatment was a major source of dioxins and furans to the Georgia Basin (Garrett and Ross 2010). Although adverse health effects are unknown in marine mammals, chlorophenols (such as

PCP) can adversely affect the survival, reproduction, growth, and metabolism of fish and shellfish (Garrett and Ross 2010).

Data are limited on PCP concentrations in marine mammals, with no information available for Southern Residents. These compounds are less persistent than other organic compounds because they readily degrade in the environment, and there is no evidence of biomagnification in upper trophic-level species (Garrett and Ross 2010). However, PCP was measured in bowhead whale plasma and was relatively abundant compared to similar phenolic compounds (Hoekstra *et al.* 2003). Because long-range transport of PCPs is limited due to rapid photolysis, they do not readily bioaccumulate. It is assumed that PCPs found in these whales result from biotransformation of hexachlorobenzene or potentially a biotransformation of pentachloroanisole (Hoekstra *et al.*2003).

Tributyltin (TBT). Tributyltin has been used as an antifoulant on ships, buoys, nets and piers to restrict or retard growth of fouling organisms. It has been identified as a persistent organic pollutant that may pose a toxic threat to the Southern Resident killer whales (NMFS 2008a). However, bioaccumulation appears to be less than other persistent pollutants (*e.g.*, PCBs, DDTs, and PBDEs).

TBT acts as an endocrine disruptor and has shown to competitively inhibit aromatase cytochrome P450 activity (Heidrich *et al.* 2001). Aromatase plays a significant role in sustaining the ratio between male and female hormones during sexual differentiation during embryonic development. TBT inhibits the conversion of androgens to estrogens. TBT can also act synergistically with a PCB congener (PCB-126) known to induce P4501A, and produce opposite effects than when the chemicals are isolated at higher doses. For example, female mice exposed to high doses of TBT combined with PCB-126 inhibited P450 activity, whereas low doses of TBT combined with the PCB congener enhanced the activity (DeLong and Rice 1997). Although TBT can significantly inhibit P450 activities, the concentration levels in the liver at which this inhibition occurs is almost 25 times higher than that found in free-ranging marine mammals (Kim *et al.* 1998). However, some marine mammal populations are at or above TBT levels that cause immunotoxicity in laboratory species (Figure 2.4.4. 1.4).

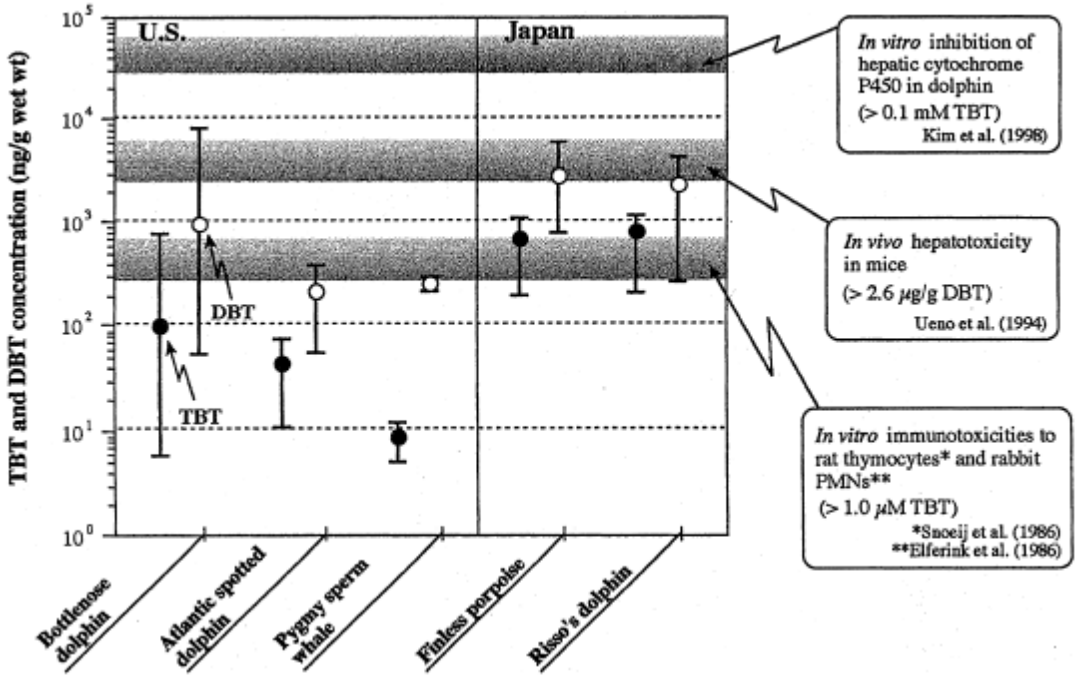

Figure 2.4.4. 1.4. Range of tributyltin (TBT) and a metabolite, dibutyltin (DBT), concentrations in the liver of cetaceans from the U.S. and Japanese coastal waters, and toxic effects threshold levels of TBT and the DBT metabolite. Reprinted from Tanabe (1999).

The distribution of TBT in the tissues and organs of marine mammals is similar to that of other species and are primarily in the liver and kidneys and lower in the muscles and blubber (Iwata *et al.* 1997, Tanabe 1999). Currently, butyltin concentrations in Southern Residents are unknown. Therefore, the extent of contamination relative to effect thresholds is unknown. Cetaceans distributed near more developed nations have elevated TBT levels compared to cetaceans adjacent to developed nations (Tanabe *et al.* 1998). Therefore, it is likely that the Southern Residents have relatively high TBT concentrations compared to cetaceans in less industrialized regions. Butyltin concentrations in cetaceans off of Japan and USA are similar. For example, the mean TBT liver concentration in killer whales off Japan (n=3) was 180 ng/g ww (Tanabe *et al.* 1998), and the mean TBT liver concentration in bottlenose dolphins off southeast Atlantic and Gulf coasts was 100 ng/g ww (Kannan *et al.* 1997). These levels are higher than concentrations in cetaceans near the Philippines, India, and China (Kannan *et al.* 1997, Tanabe *et al.* 1998). Transplacental transfer of TBT from mother to fetus is relatively low compared to other persistent pollutants. For example, TBT concentrations in the liver of a pregnant female killer whale (150 ng/g ww) was much higher compared to concentrations in the liver of the fetus (26 ng/g ww) (Tanabe *et al.* 1998). TBTs do not appear to differ between males and females, however increasing levels have been observed in immature stages of Risso's dolphins (Tanabe 1999).

Metals and Elemental Pollutants. Unlike the persistent pollutants described above, metals are naturally found in the environment and some are essential to an animals' nutrition. Heavy metals in marine mammals are primarily determined by the levels in prey and the geographic region, as well as age and gender of the individual. For example, marine mammals that feed on squid can be exposed to higher levels of cadmium, copper, and zinc because squid have the ability to retain these elements (Reijnders and Aguilar 2002). Human activities can increase the concentrations and metals can become toxic at certain exposure levels. Currently, there is little information on metals in killer whales or in their prey. Most metals, like persistent pollutants, settle to the ocean floor where they can accumulate in sediment. Therefore, areas with high human activity can become hotspots of multiple toxic chemicals.

The distribution or storage of heavy metals in marine mammals is dependent on the metal. In general, heavy metals are found in the liver, kidneys, muscles, and bones (O'Shea 1999, Reijnders and Aguilar 2002, Das *et al.* 2003). Some metals may transfer from mother to offspring during gestation and lactation, although not to the same degree as the persistent organic pollutants. For example, Honda *et al.* (1987) found the hepatic concentrations of iron, lead, nickel, and cobalt decreased in adult female southern minke whales with progress of gestation. Pregnant pilot whales had less mercury in the serum than non-pregnant females, indicating a potential transplacental transfer to the fetus (Nielsen *et al.* 2000). However, it may also be possible that a change in the diet of the pregnant pilot whales can explain the change in mercury levels (Nielsen *et al.* 2000).

Non-essential metals that can be toxic to marine mammals, even at low doses, include mercury, cadmium, and lead. Mercury, cadmium, and lead in the tissues of marine mammals have been the focus of several studies because of their known toxicity to humans and other wildlife, such as damage to the central nervous system, skeletal deformities, kidney lesions and kidney or liver damage, as well as carcinogenic, mutagenic, and teratogenic effects (O'Shea 1999, Das *et al.* 2003). However, little information is known about toxic effects of heavy metals in marine mammals. Essential metals that occur naturally in the environment can also be toxic and their concentrations can be elevated in areas of high human activities. These essential metals include copper, zinc, iron, and selenium. Below is a brief description of toxicity, storage, concentration levels, and detoxification mechanisms for the metals and elements discussed in this opinion.

Cadmium. Adverse health effects from high exposure to cadmium (or cadmium compounds) in mammals include reduced growth, impaired immunity, cancer, and renal dysfunction, whereas acute exposure can cause dystrophic changes in several organs including the liver, heart, and kidneys (Grant and Ross 2002 as cited in Government of Canada *et al.* 1993). Dietz *et al.* (1998) suggests that marine mammals in the Arctic regions may have habituated to naturally high levels of cadmium. For example, cadmium concentrations in ringed seals from Greenland are higher than the health-effects threshold for kidney damage (200 µg/g wet weight, WHO 1992). This health effects threshold has been more recently considered an overestimation, and that renal dysfunction from cadmium exposure has been observed at concentrations of only 50 µg/g wet weight (Elinder and Järup 1996). The ringed seals that had cadmium concentrations above both of the thresholds still displayed normal renal structure (Dietz *et al.* 1998). Despite the high levels of cadmium found in marine mammals (*e.g.*, Nielsen *et al.* 2000, O'Shea 1999 and Government of Canada *et al.* 1993), no toxic effect has been

observed indicating a potential detoxification mechanism (described further below). Liver levels of cadmium in an adult female transient killer whale that stranded at Dungeness Spit in 2002 were < 0.15mg/kg ww (G. Ylitalo NWFSC, pers. comm.).

Lead. Chronic exposure to lead in mammals can cause disorders of the nervous system, renal system, and gastrointestinal tract, impaired or weakened mental function, anemia, and variable immunotoxic effects (O'Shea 1999, Grant and Ross 2002, De Guise *et al.* 2003). Exposure to high concentrations of lead in mammals has lead to hypertension, reproductive disorders, and metabolic and neurological issues (Grant and Ross 2002). Long-term storage of lead primarily occurs in the bone; however, lead can be released with calcium into the bloodstream (Grant and Ross 2002).

Only a limited number of studies have measured lead concentrations in the bone of marine mammals. The few studies that have measured lead in the bone reported negligible concentrations (O'Shea 1999, Das *et al.* 2003, O'Hara *et al.* 2003). One of the highest concentrations of lead measured in the bone of marine mammals was approximately 61.6 ppm (wet weight) in a bottlenose dolphin from an area known for emissions from a lead smelter (O'Shea 1999 as cited in Kemper *et al.* 1994). In most studies, levels in tissues of marine mammals have not been reported at levels that were a cause for concern and were within normal ranges and included concentrations less than 1ppm (O'Shea 1999). Liver levels of lead in an adult female transient killer whale that stranded at Dungeness Spit in 2002 were < 0.15mg/kg ww (G. Ylitalo NWFSC, pers. comm.).

Detoxification Mechanisms. Some marine mammals (particularly from the northern arctic regions) appear to tolerate high levels of mercury, lead, and cadmium and are able to detoxify them through several processes. Cadmium and mercury can combine with selenium or metallothionein (MT, a protein molecule) to mitigate the toxic effects of exposure (Rooney 2007, Klaassen *et al.* 2009). These new complexes (mercury and selenium or cadmium and MT) in the liver or kidneys mitigate toxic effects and change the metals into non-toxic forms (Klaassen *et al.* 2009). This detoxification mechanism appears to be species-specific. For example, unlike in sperm whales that did not show an obvious relationship between mercury and selenium, pilot whales demonstrated a strong correlation between mercury and selenium with an almost fourfold higher molar ratio than that found in the sperm whales (Nielsen *et al.* 2000).

Other Metals and Elements. Aluminum, ammonia, arsenic, copper, chromium (III and VI), nickel, selenium, silver, and zinc are not primary toxic chemicals of concern for marine mammals compared to mercury, cadmium, or lead, because they are either essential to the nutrition of the animal and are found at relatively low concentrations (*e.g.*, aluminum, nickel, selenium, and zinc), the available data does not support a health risk from exposure (O'Shea 1999, O'Hara *et al.* 2003), or because the element does not build up in the food chain (*e.g.*, ammonia). Arsenic has been measured in marine mammals, but not at levels considered to be toxic (O'Shea 1999). Concentrations of arsenic tend to be higher in lower trophic level species and there is no evidence that arsenic biomagnifies (Garrett and Ross 2010). Selenium, zinc, and copper are all essential elements for the nutrition of animals. Effects in mammals exposed to high copper concentrations include genetic and developmental abnormalities, and renal failure (Grant and Ross 2002). Although low concentrations of copper have been measured in marine

mammals, chronic exposure to copper may be of concern to killer whales because anthropogenic activities can result in increased levels near urban and industrial areas (Grant and Ross 2002). Copper in the liver of marine mammals declines with age, however differences in copper concentrations in populations have been reported after accounting for age (Stein *et al.* 2003). For example, copper concentrations declined in the livers of bottlenose dolphins in Florida and Texas, however the dolphins from Florida had lower concentrations (Stein *et al.* 2003). In general, mammals are more sensitive to chromium (VI) than to chromium (III) and biomagnification factors are relatively low and increased concentrations up the food chain have not been observed (Garrett and Ross 2010). Recent evidence indicates chromium (VI) is cytotoxic and genotoxic to North Atlantic right whale lung and testes cells, indicating chromium (VI) may be a significant risk factor to these whales (Wise *et al.* 2008). They suggest inhalation is likely an important exposure route. Chromium (VI) was also cytotoxic and clastogenic to Steller sea lion lung cells (Wise *et al.* 2009). Lastly, research on selenium in marine mammals has been primarily focused on its ability to form a non-toxic complex with mercury.

Extinction Risk. In conjunction with the 2004 status review, NMFS conducted a population viability analysis (PVA) for Southern Resident killer whales (Krahn *et al.* 2004). Demographic information from the 1970s to fairly recently (1974-2003, 1990-2003, and 1994-2003) were considered to estimate extinction and quasi-extinction risk. The NMFS defined "quasi-extinction" as the stage at which 10 or fewer males or females remained a threshold from which the population was not expected to recover.

The model evaluated a range in Southern Resident survival rates, based on variability in mean survival rates documented from past time intervals (highest, intermediate, and lowest survival). The model used a single fecundity rate for all simulations. The study considered seven values of carrying capacity for the population ranging from 100 to 400 whales, three levels of catastrophic event (*e.g.*, oil spills and disease outbreaks) frequency ranging from none to twice per century, and three levels of catastrophic event magnitude in which 0, 10, or 20 percent of the animals died per event.

The analysis indicated that the Southern Resident killer whales have a range of extinction risk from 0.1 to 18.7 percent in 100 years and 1.9 to 94.2 percent in 300 years, and a range of quasi-extinction risk from 1 to 66.5 percent in 100 years and 3.6 to 98.3 percent in 300 years (Table 2.4.4. 1.6). The population is generally at greater risk of extinction as survival rate decreases and over a longer time horizon (300 years) than over a shorter time horizon (100 years) (as would be expected with long-lived mammals). There is a greater extinction risk associated with increased probability and magnitude of catastrophic events. The NWFSC continue to evaluate mortality rates and reproduction, and will complete work on a PVA similar to the analysis summarized above. Until these updated analyses are completed, the Krahn *et al.* (2004) analysis represents the best available science on extinction risk of Southern Resident killer whales.

Table 2.4.4. 1.6. Range of extinction and quasi-extinction risk for Southern Resident killer whales in 100 and 300 years, assuming a range in survival rates (depicted by time period), a constant rate of fecundity, between 100 and 400 whales, and a range catastrophic probabilities and magnitudes (Krahn *et al.* 2004).

Time Period	Extinction Risk (%)		Quasi-Extinction Risk (%)	
	100 yrs	300 yrs	100 yrs	300 yrs
Highest survival	0.1 – 2.8	1.9 – 42.4	1.0 – 14.6	3.6 – 67.7
Intermediate survival	0.2 – 5.2	14.4 – 65.6	6.1 – 29.8	21.4 – 85.3
Lowest survival	5.6 – 18.7	68.2 – 94.2	39.4 – 66.5	76.1 – 98.3

2.5 Environmental Baseline

The 'environmental baseline' includes the past and present impacts of all Federal, state, or private actions and other human activities in the action area, the anticipated impacts of all proposed Federal projects in the action area that have already undergone formal or early section 7 consultation, and the impact of state or private actions which are contemporaneous with the consultation in process (50 CFR 402.02).

In this section, NMFS first provides information on water body segments in Oregon that currently fail to meet applicable water quality standards. Second, NMFS provides information on stormwater (MS4) and point-source (NPDES) permits in Oregon, in terms of spatial distribution and chemical-specific constituents, and species distribution, exposure potential via point-source discharges. And third, NMFS summarizes past and current human activities and describes how these activities influence current habitat conditions within the action area.

2.5.1 303(d)-Listed Waterbody Segments in Oregon

Under section 303(d) of the CWA, states and tribes are required to provide EPA a biennial list of water body segments that do not meet water quality standards. On its 2004/2006 303(d) list, the Oregon Department of Environmental Quality (ODEQ) identified more than 15,000 stream miles listed for at least one pollutant. Pollutants identified on the 303(d) list fall into several major groups which include sediment, nutrients, metals, bacteria, oxygen demand, and toxic organics. For this consultation NMFS focused on metals, toxic organics, and conventional pollutants, (*i.e.*, temperature, pH, and dissolved oxygen) as these pollutants can affect the toxicity of metal and organic pollutants. Figure 2.5.1.1.1 identifies toxics associated with those listed in Table 1.1 that were detected in one or more watersheds in Oregon by the USGS. Figures 2.5.1.1.2 through 2.5.1.1.19 identify 303(d)-listed waters in Oregon for toxins, temperature, dissolved oxygen, and pH.

A query by NMFS of the National Aquatic Water Quality Assessment (NAWQA) database (http://water.usgs.gov/nawqa/about.html) determined that all but three compounds listed in Table 1.1 were detected in one or more watersheds in Oregon (Figure 2.5.1.1.1).

Compound	NAWQA Detection—Oregon
Aluminum	Yes
Ammonia	Yes
Arsenic	Yes
Lindane	Yes
Cadmium	Yes
Chromium III	Yes
Chromium VI	Yes
Copper	Yes
Dieldrin	Yes
Endosulfan-alpha	Yes
Endosulfan-beta	Yes
Endrin	No
Heptachlor Epoxide	No
Lead	Yes
Nickel	Yes
PCP	No
Selenium	Yes
Silver	Yes
TBT	Yes
Zinc	Yes

Figure 2.5.1.1.1 NAWQA database search results for compounds listed in Table 1.1.

2.5.1.1 303(d)-Listed Waters in Oregon

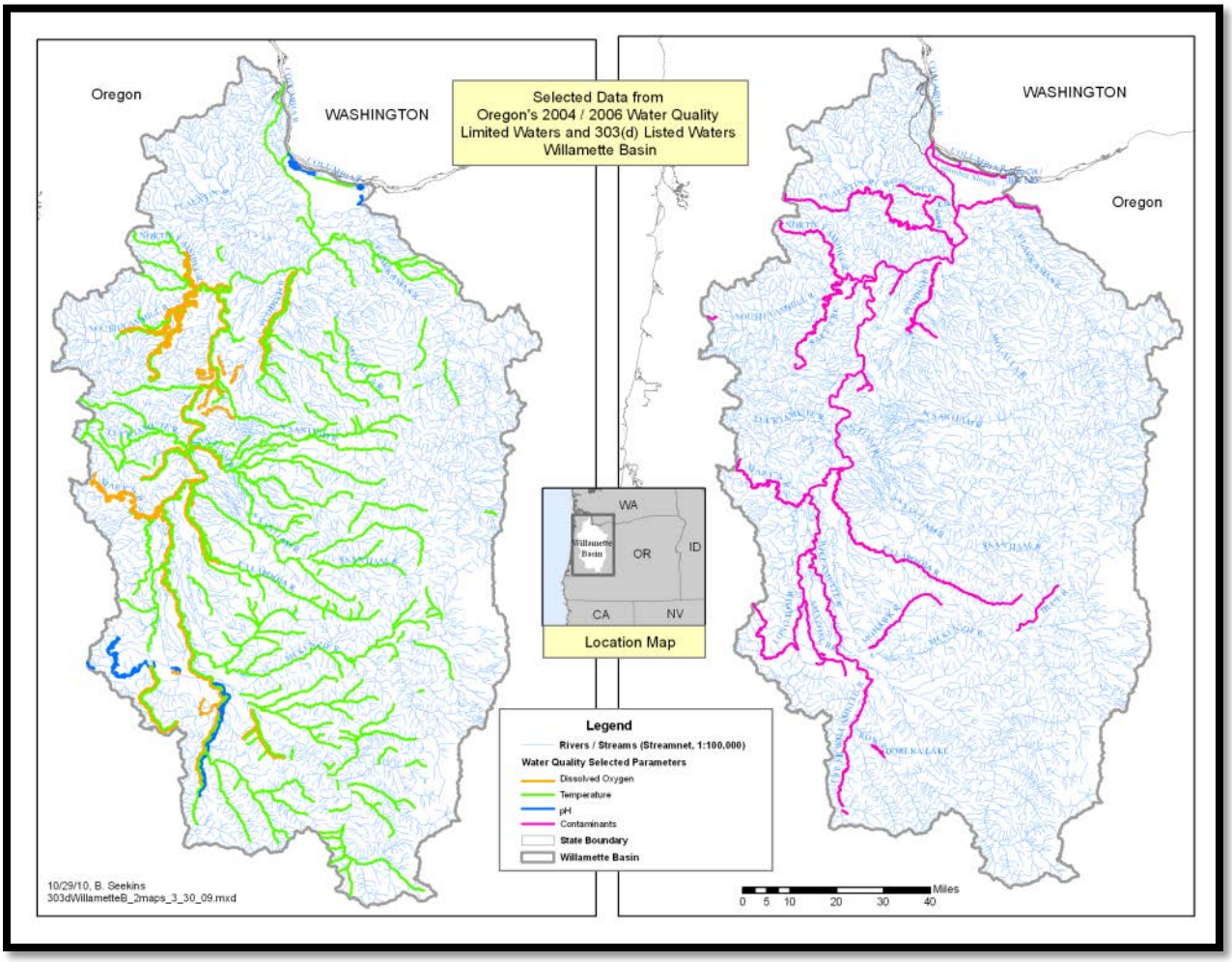

Figure 2.5.1.1.2 303(d) listed waters in the Willamette River Basin, Oregon for dissolved oxygen, pH, temperature, and non-specified toxins.

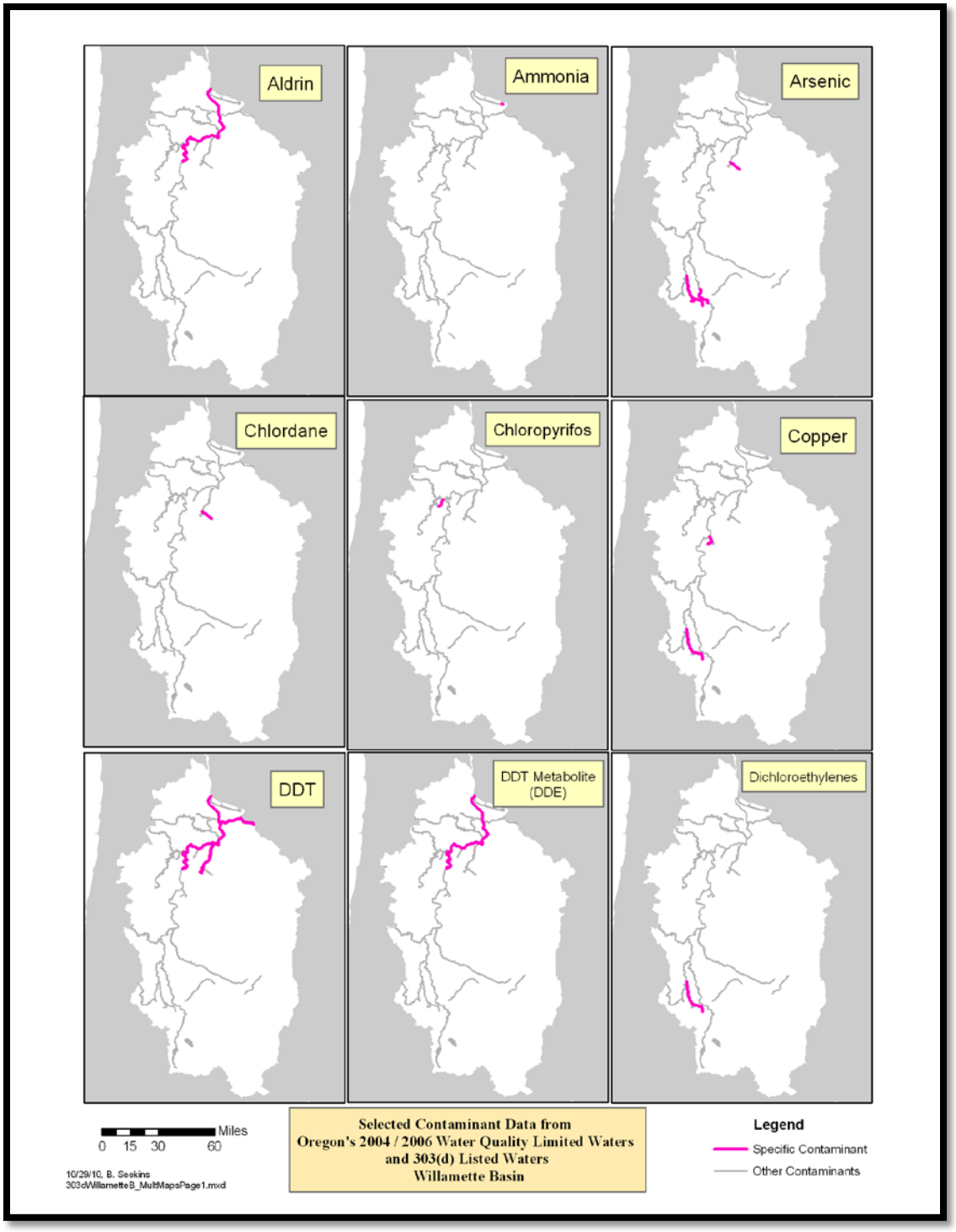

Figure 2.5.1.1.3 303(d) listed waters in the Willamette River Basin, Oregon for specified toxins.

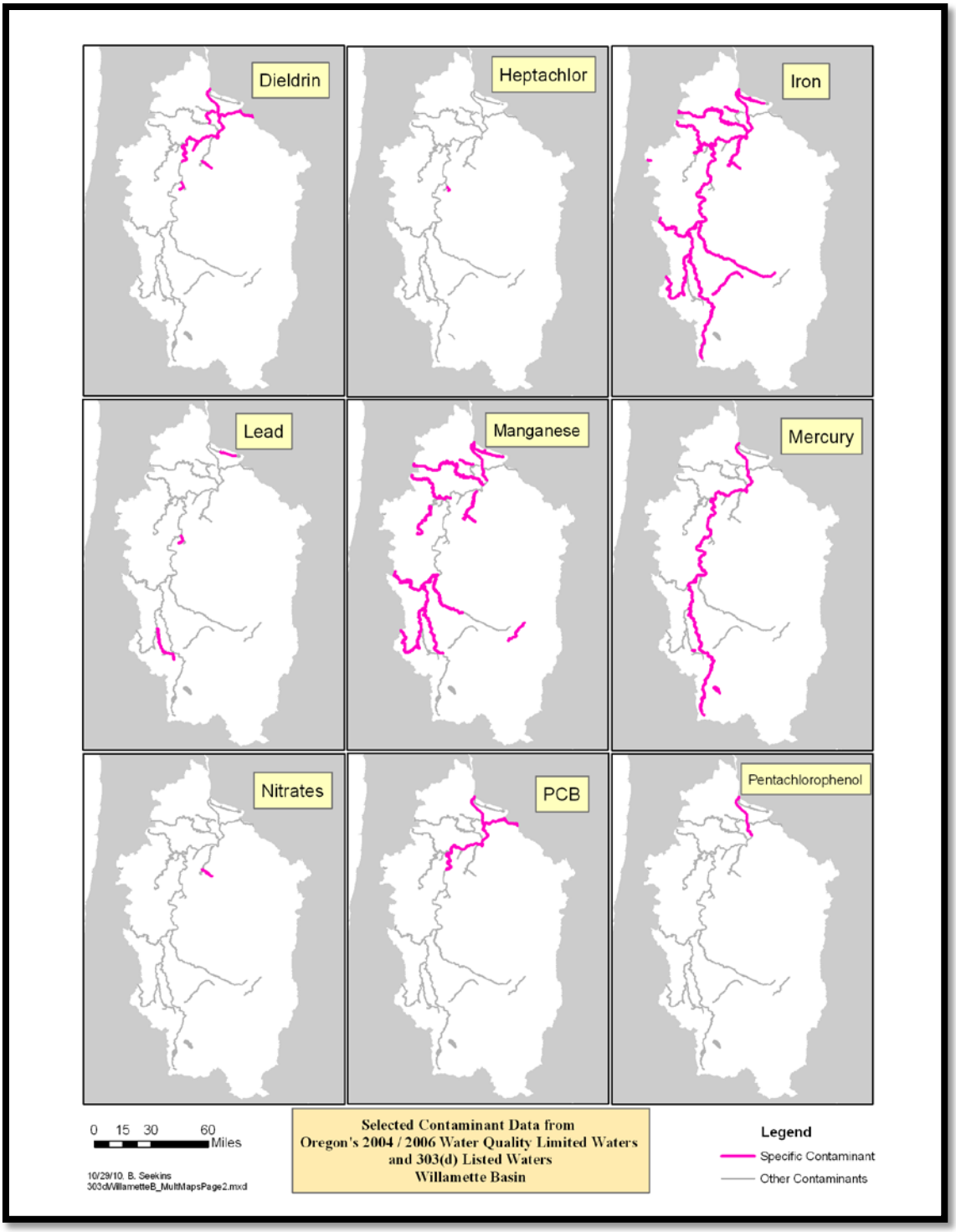

Figure 2.5.1.1.4 303(d) listed waters in the Willamette River Basin, Oregon for specified toxins.

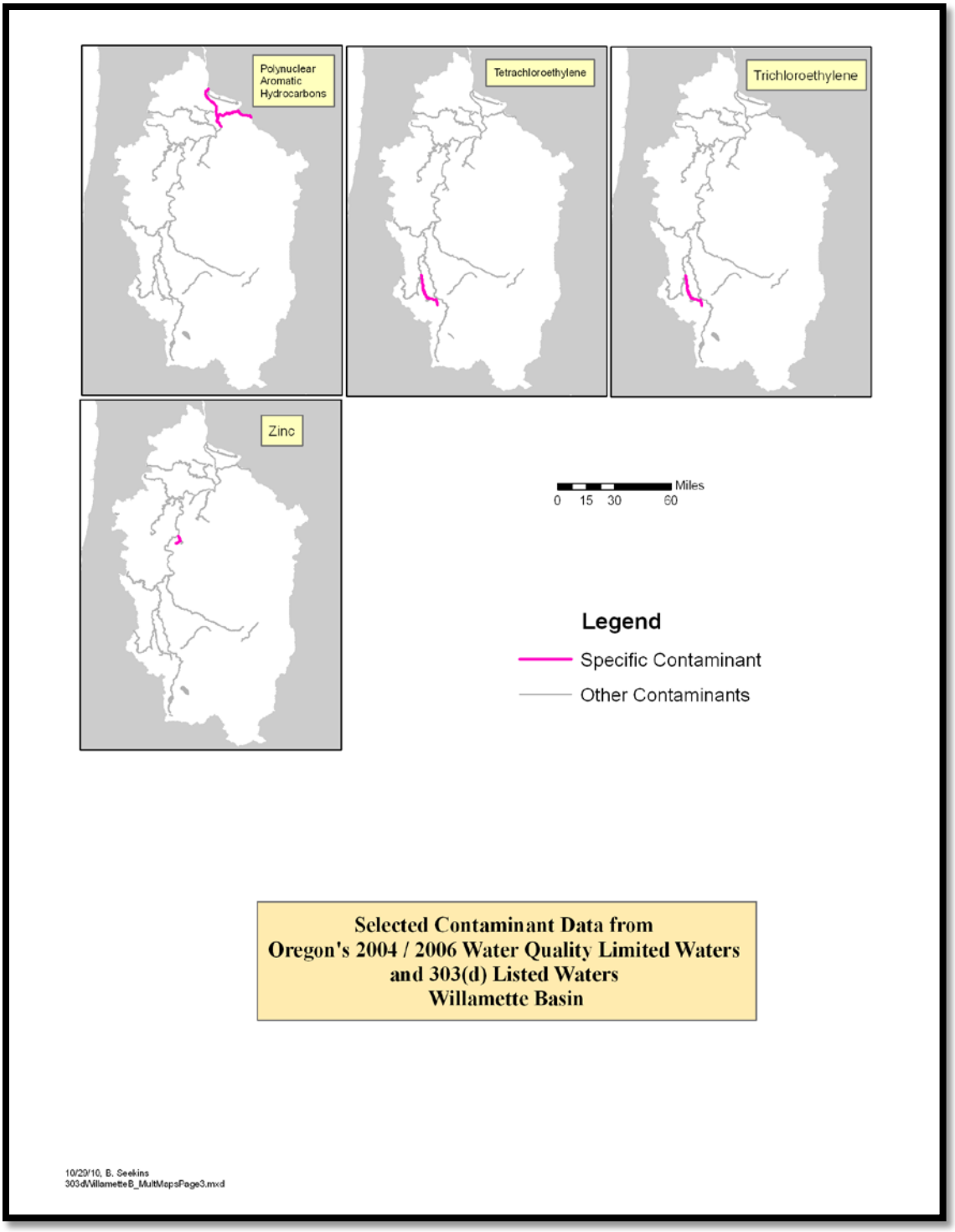

Figure 2.5.1.1.5 303(d) listed waters in the Willamette River Basin, Oregon for specified toxins.

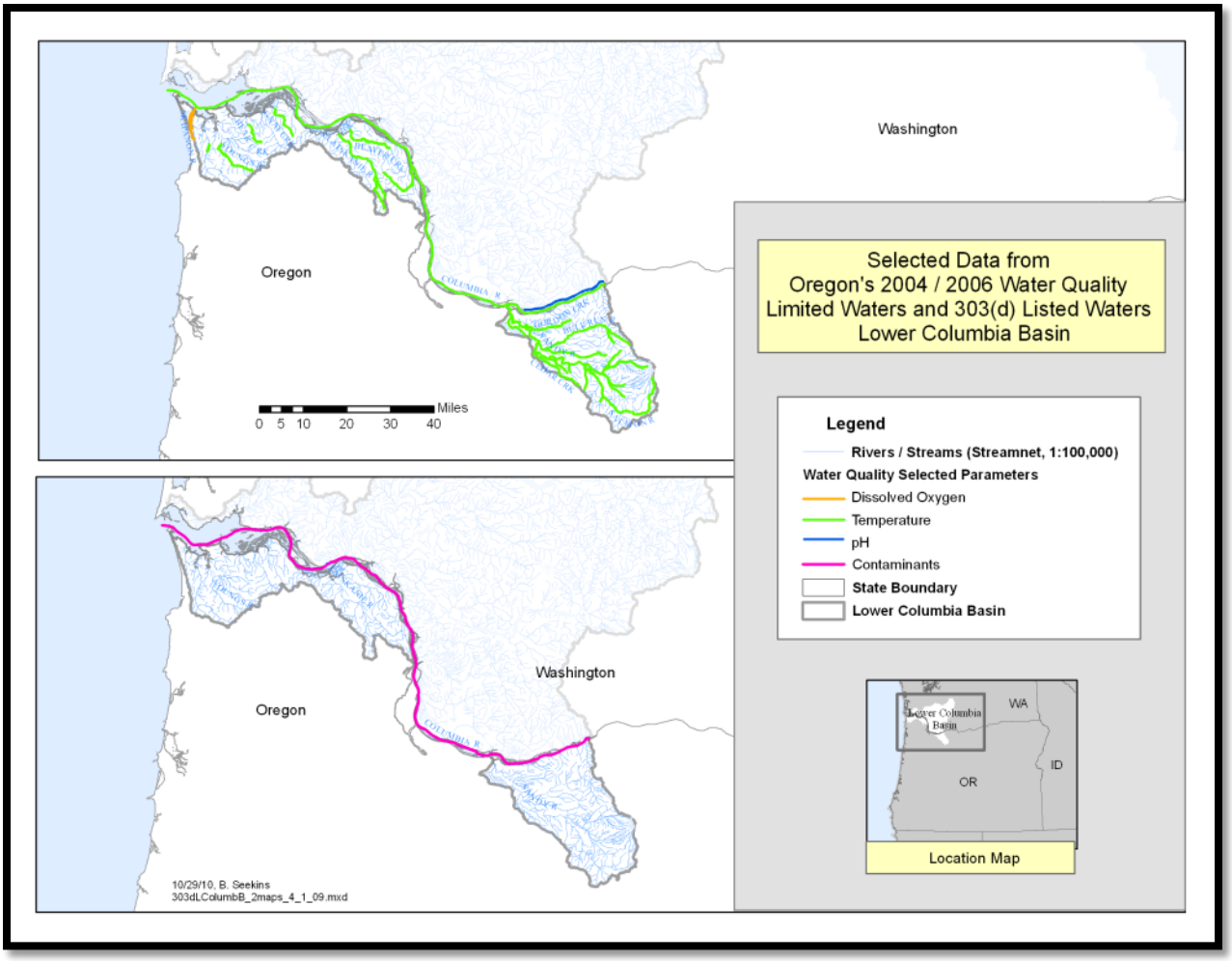

Figure 2.5.1.1.6 303(d) listed waters in the lower Columbia River and associated tributariy rivers in Oregon for dissolved oxygen, pH, temperature, and non-specified toxins.

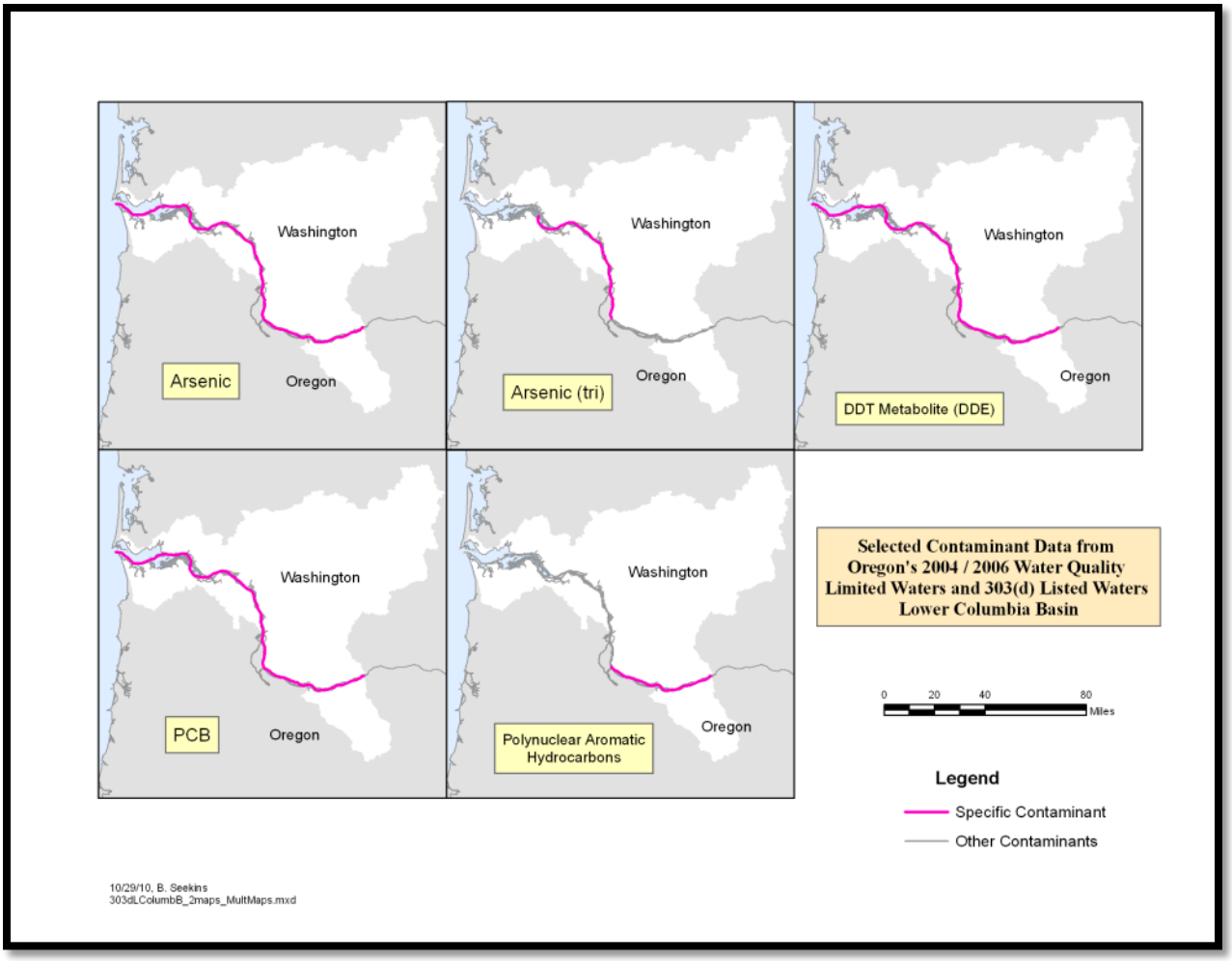

Figure 2.5.1.1.7 303(d) listed waters in the lower Columbia River in Oregon for specified toxins.

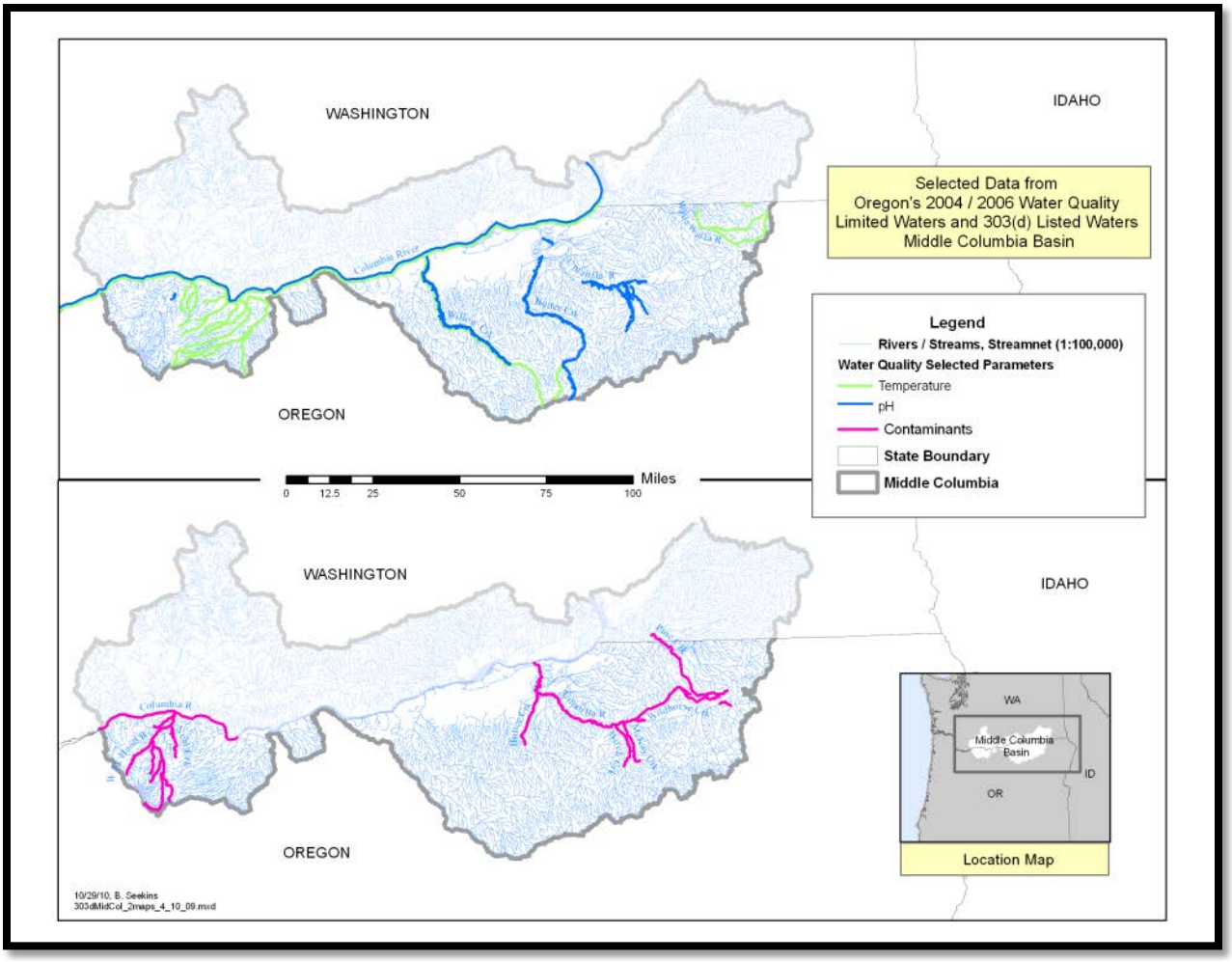

Figure 2.5.1.1.8 303(d) listed waters in the middle Columbia River and associated tributaries in Oregon for dissolved oxygen, pH, temperature, and non-specified toxins.

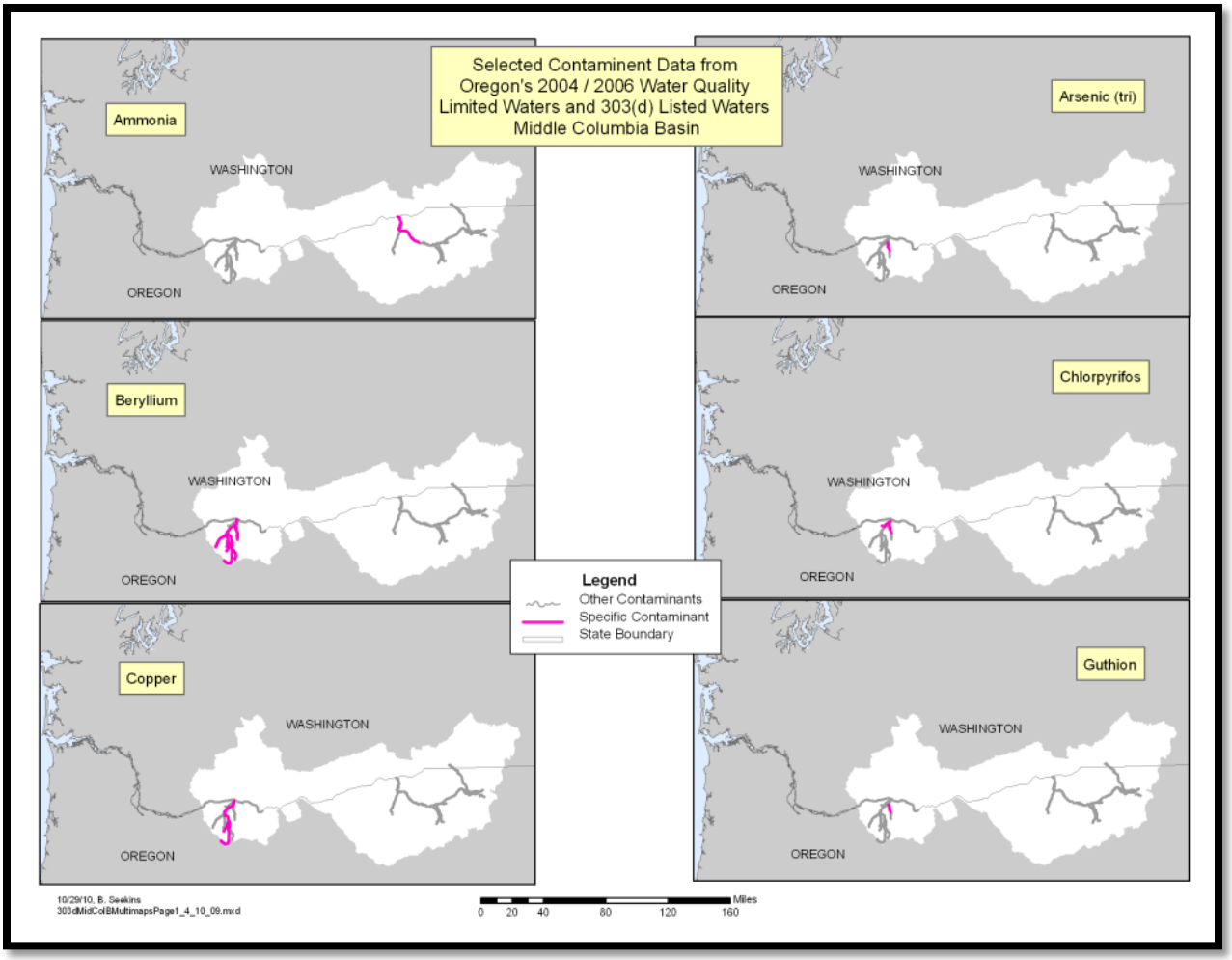

Figure 2.5.1.1.9 303(d) listed waters in the middle Columbia River and associated tributaries in Oregon for specified toxins.

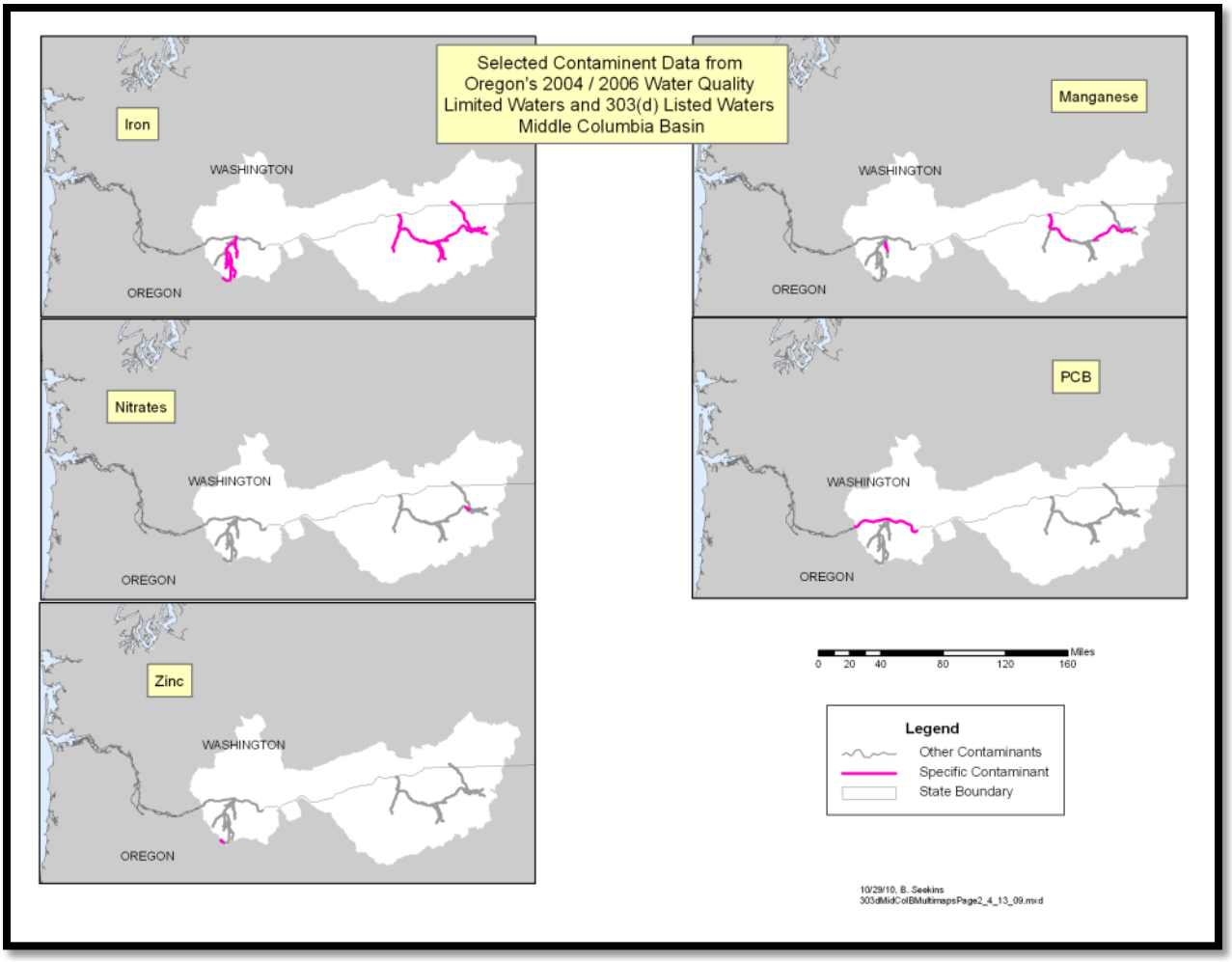

Figure 2.5.1.1.10 303(d) listed waters in the middle Columbia River and associated tributaries in Oregon for specified toxins.

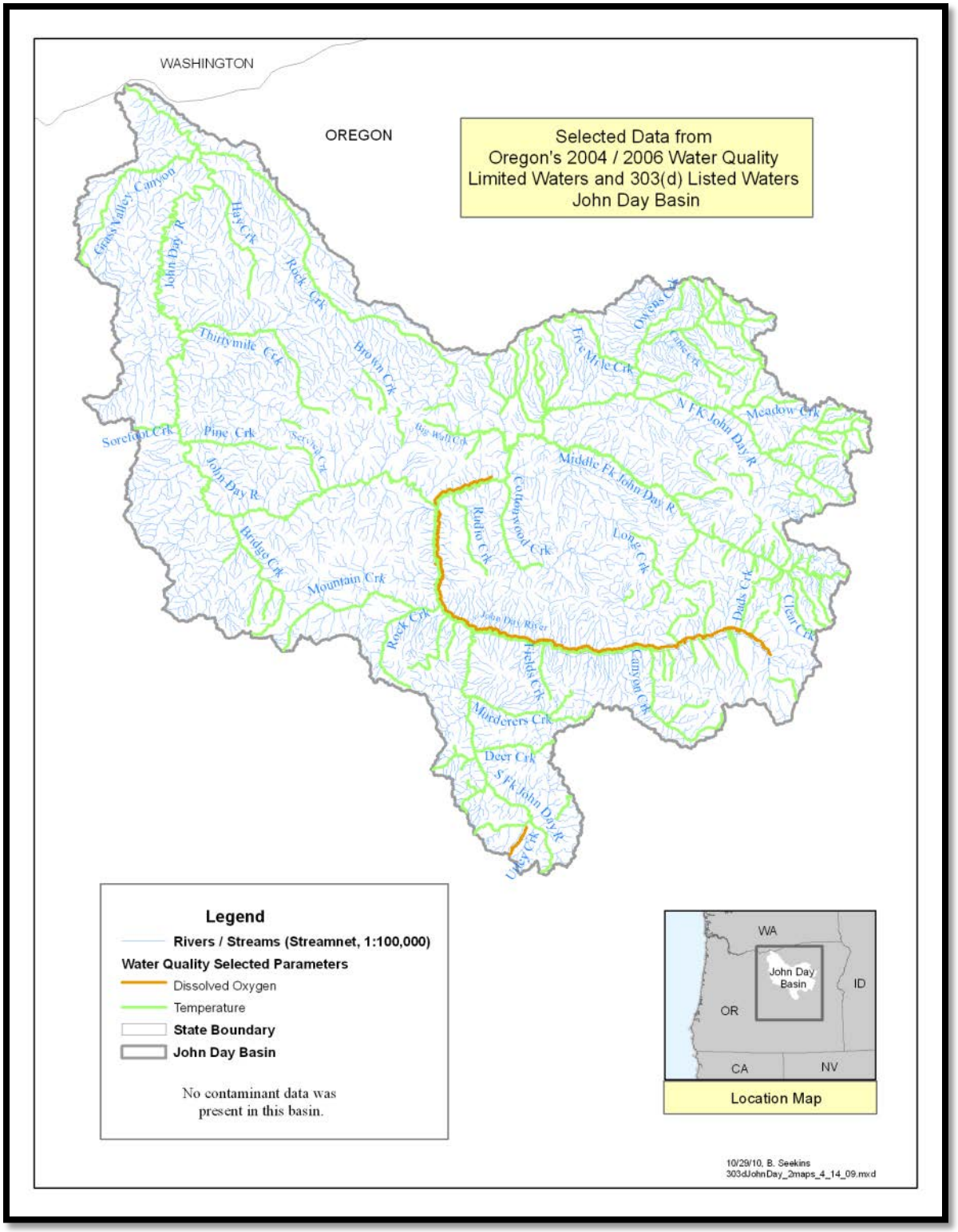

Figure 2.5.1.1.11. 303(d) listed waters in the John Day River Basin, Oregon for dissolved oxygen and temperature. No identified toxins.

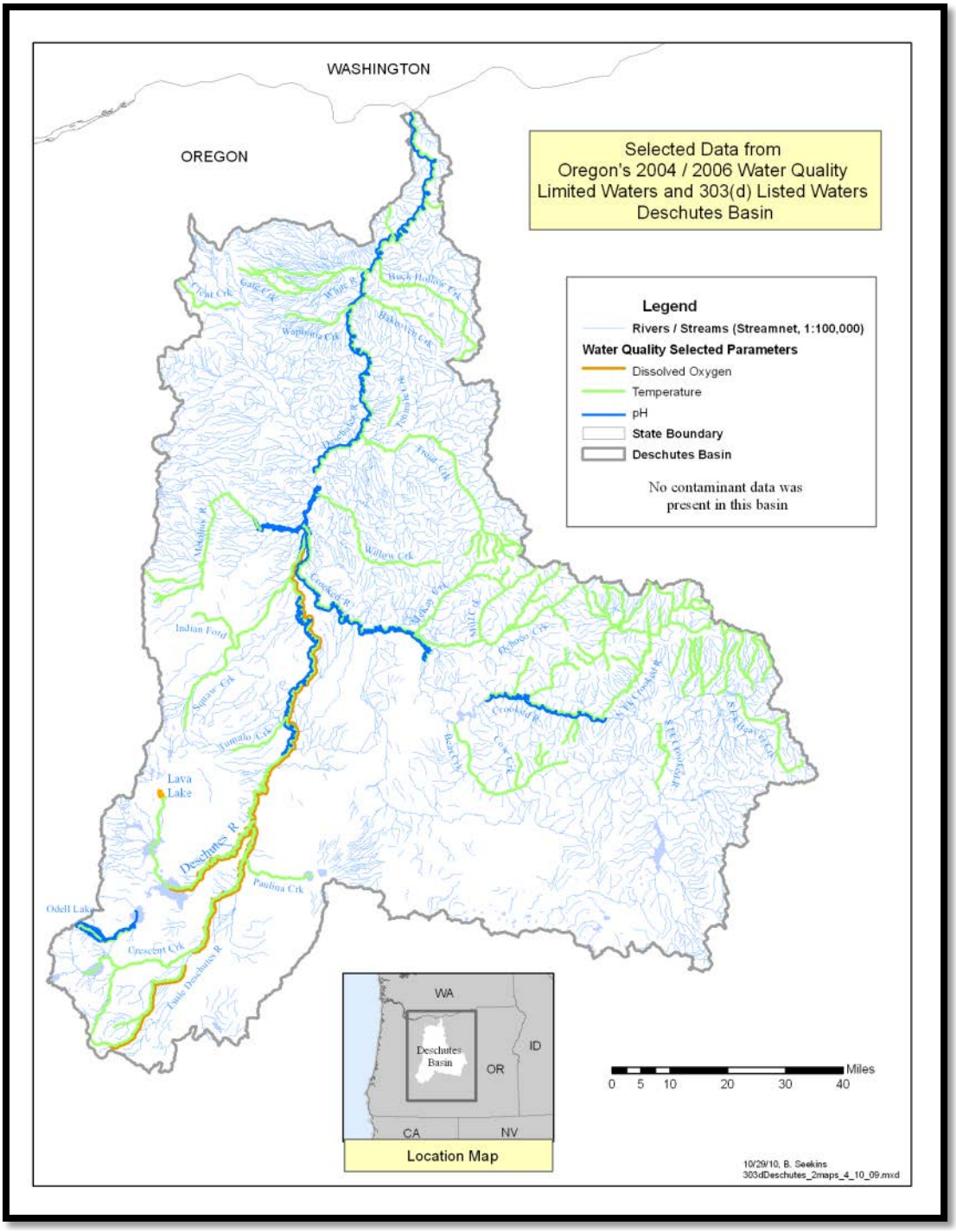

Figure 2.5.1.1.12 303(d) listed waters in the Deschutes River Basin, Oregon for dissolved oxygen, pH, and temperature. No identified toxins.

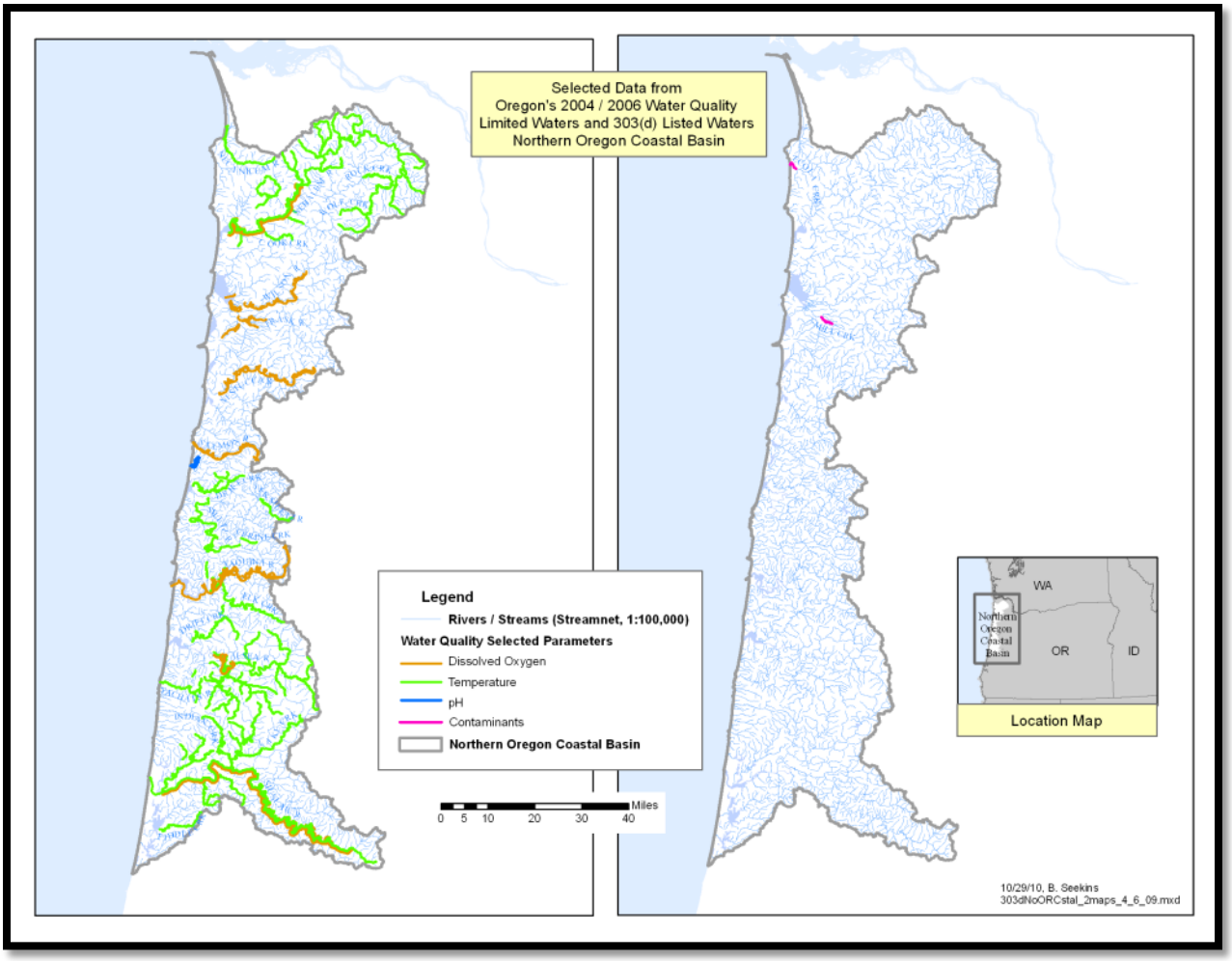

Figure 2.5.1.1.13 303(d) listed waters in the north coast river basins, Oregon for dissolved oxygen, temperature, and non-specified toxins.

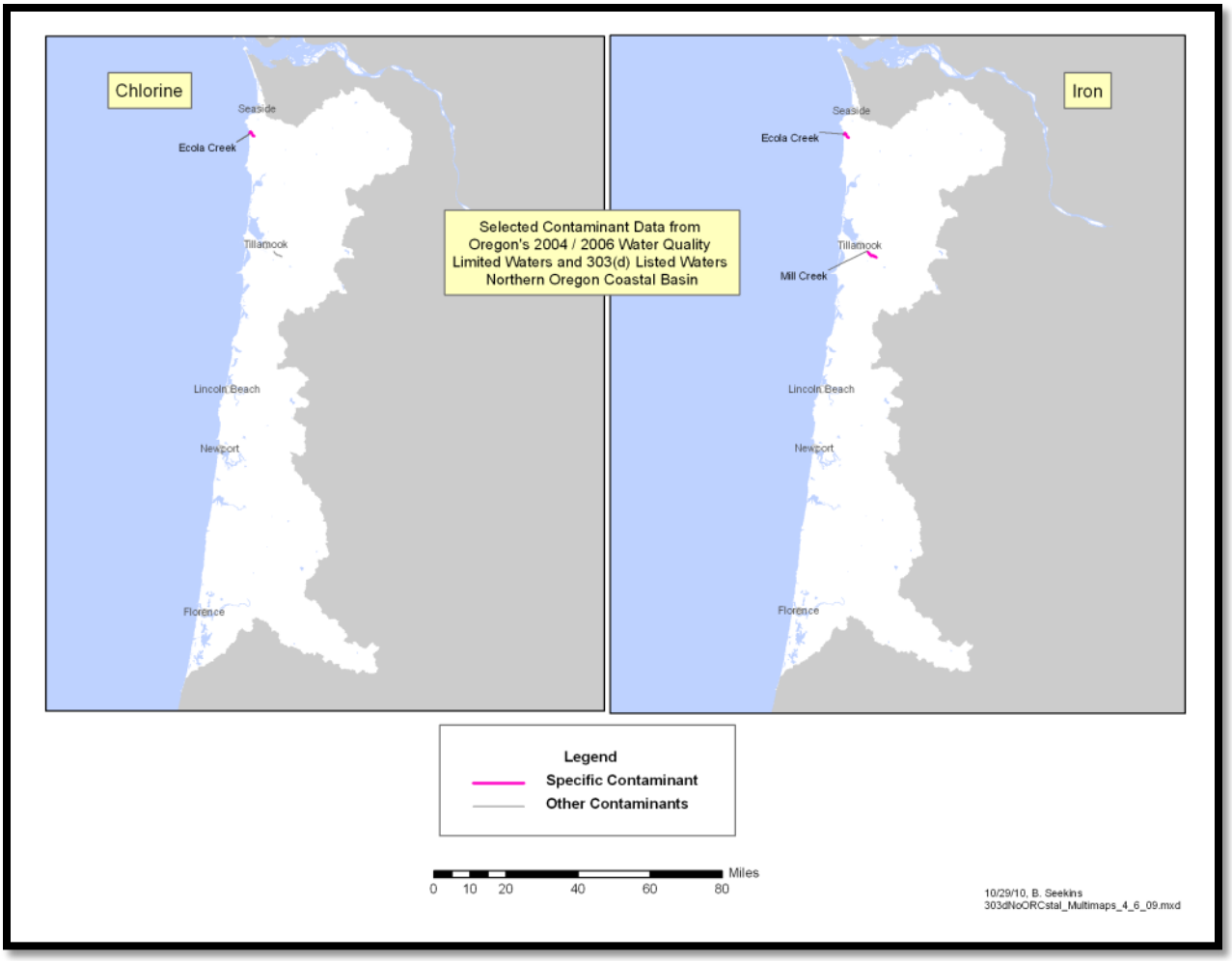

Figure 2.5.1.1.14 303(d) listed waters in the north coast river basins, Oregon for specified toxins.

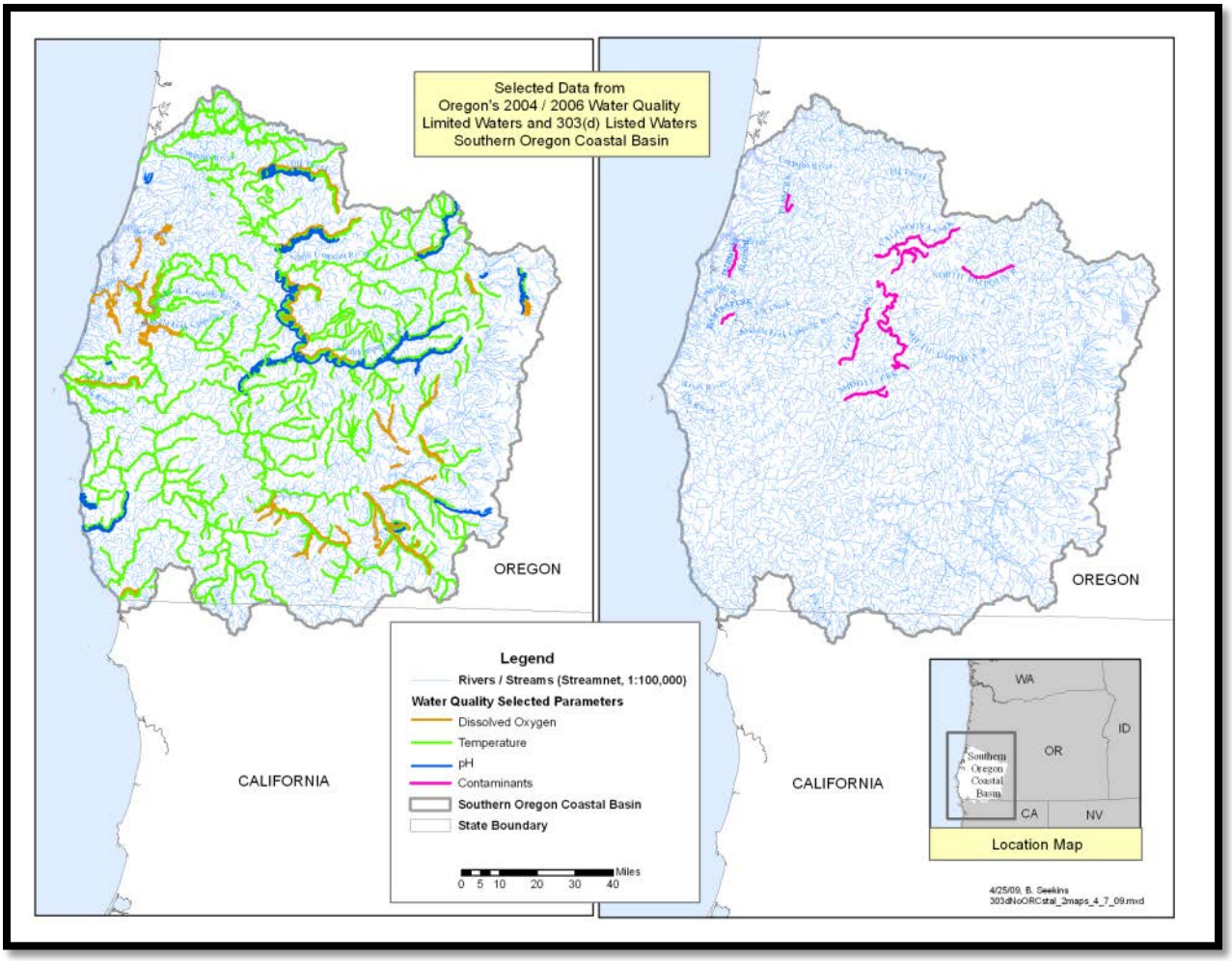

Figure 2.5.1.1.15 303(d) listed waters in the south coastal river basin, Oregon for dissolved oxygen, pH, and temperature, non-and specified toxins.

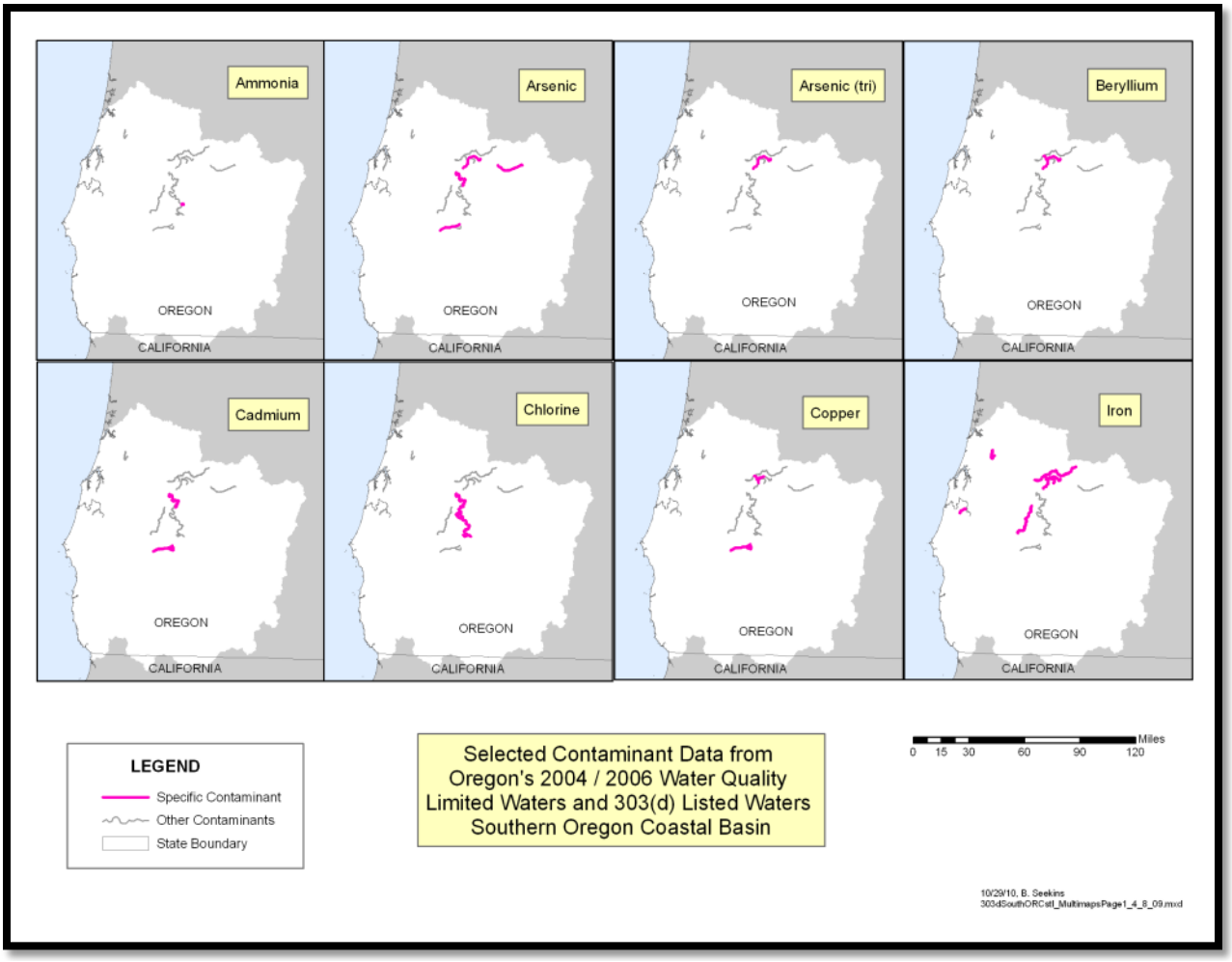

Figure 2.5.1.1.16 303(d) listed waters in the south coast river basins, Oregon specified toxins.

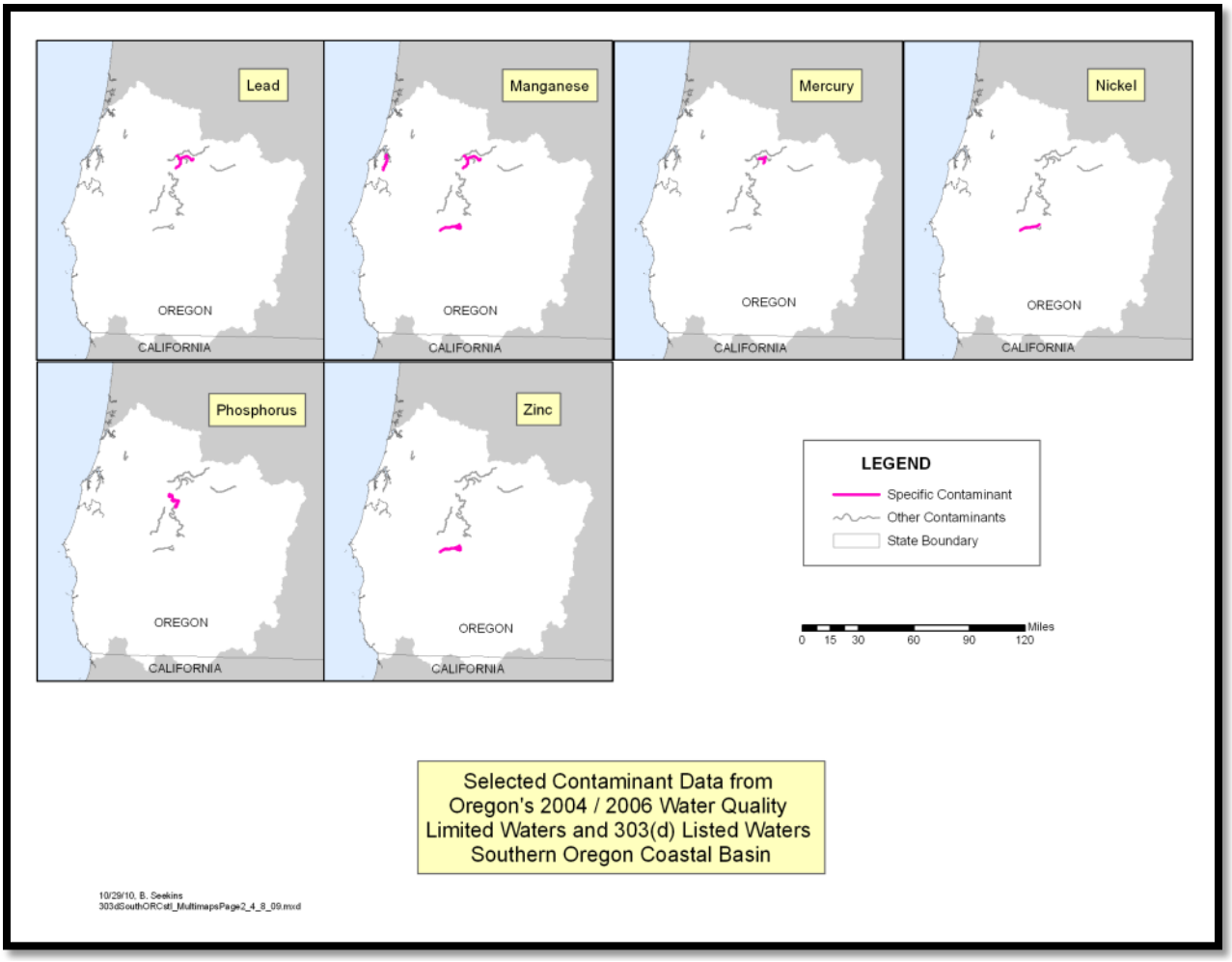

Figure 2.5.1.1.17 303(d) listed waters in the south coast river basins, Oregon for specified toxins.

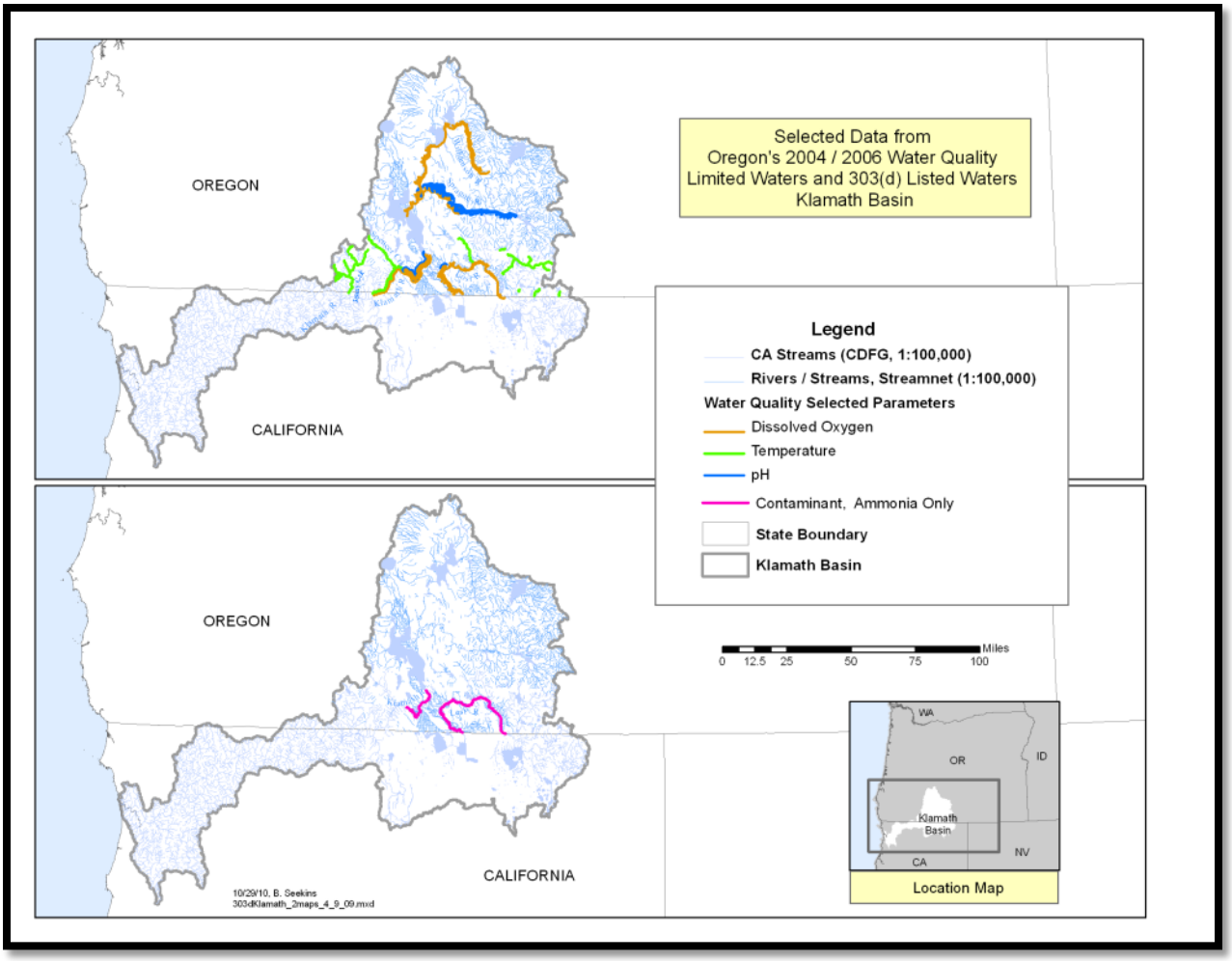

Figure 2.5.1.1.18 303(d) listed waters in the Klamath River Basin, Oregon for dissolved oxygen, pH, and temperature, and non-specified toxins.

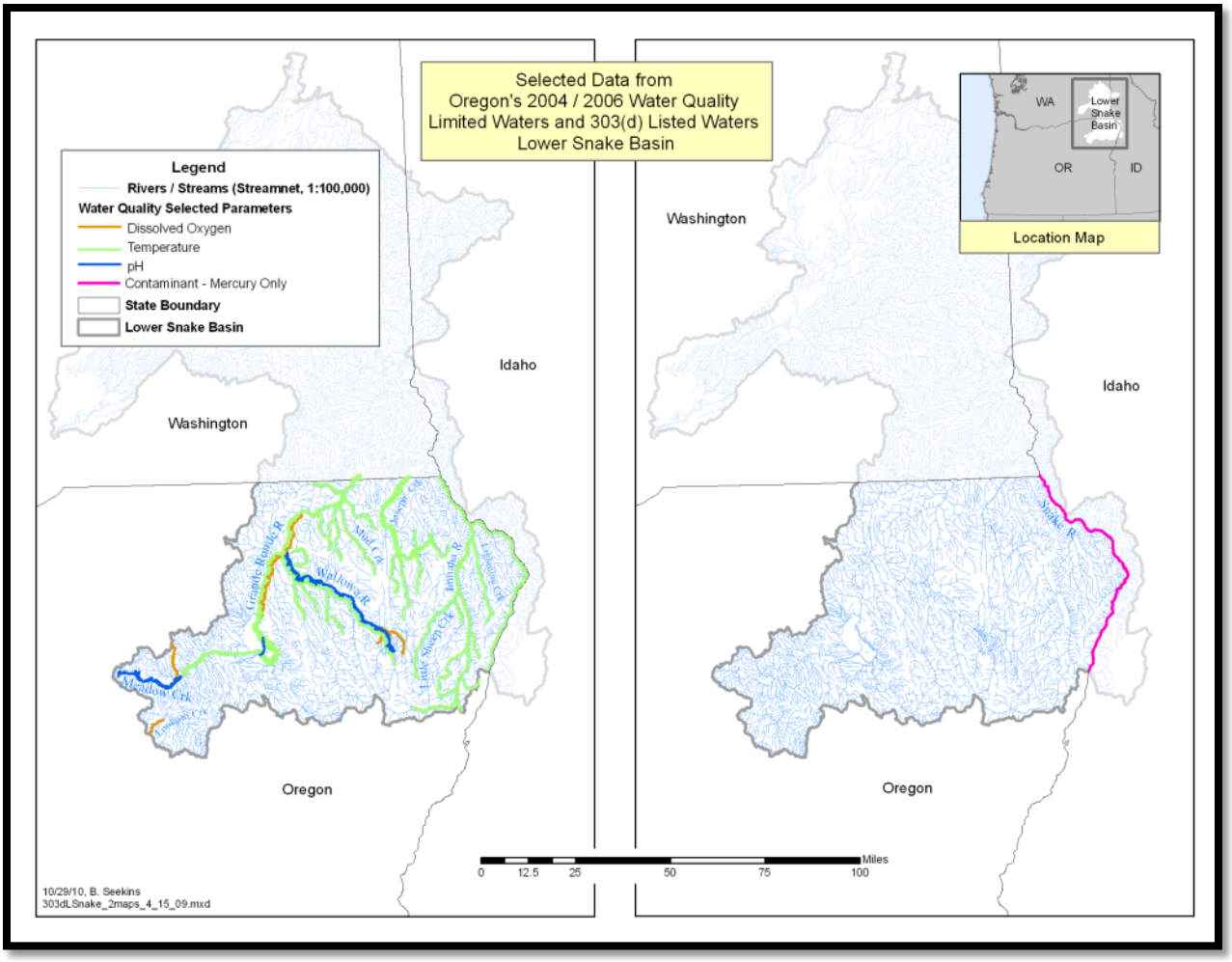

Figure 2.5.1.1.19 303(d) listed waters in the lower Snake River Basin, Oregon for dissolved oxygen, pH, and temperature, and specified toxins.

2.5.2. MS4 and NPDES Permits, Species Distribution, and Exposure Risk Potential

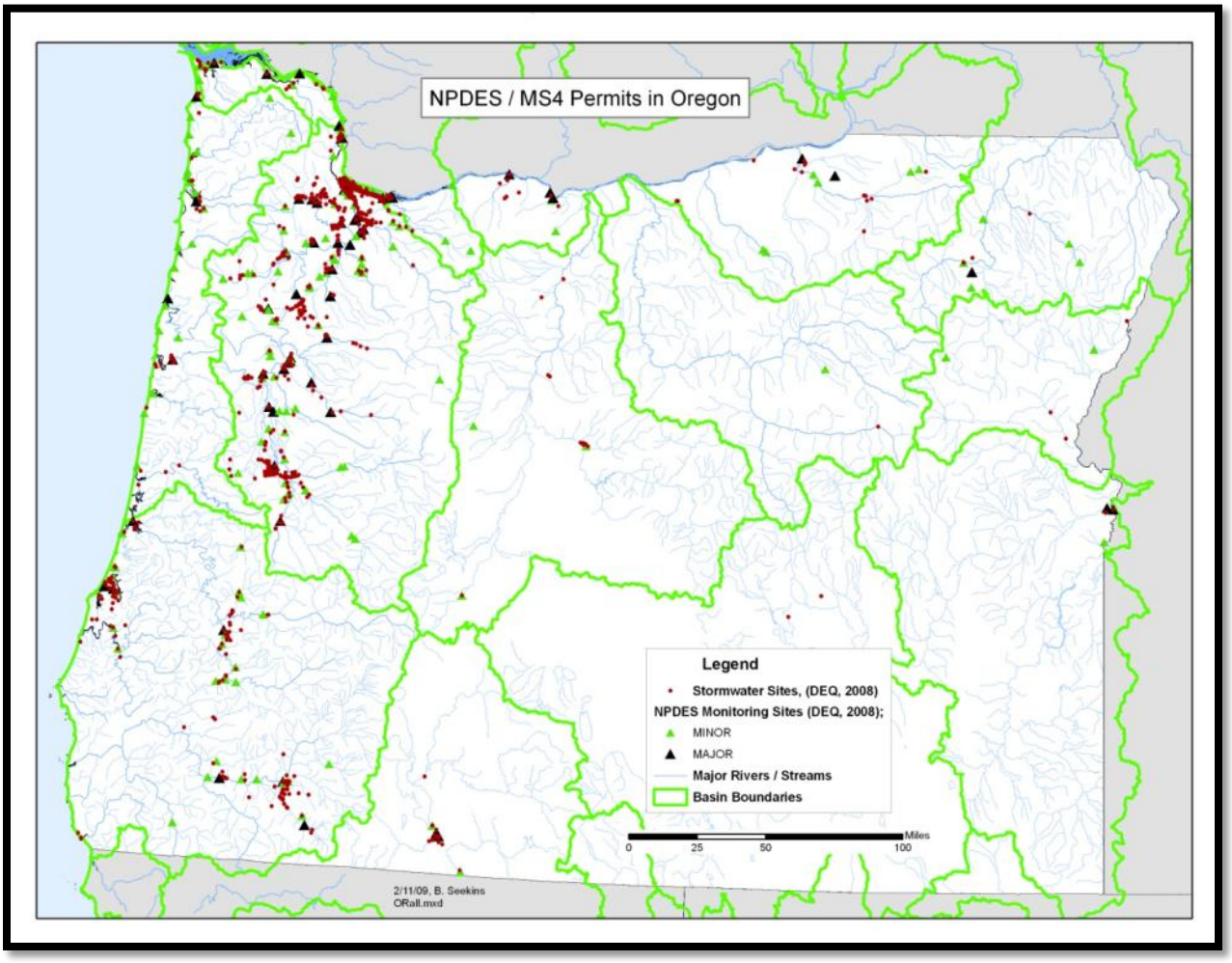

Figure 2.5.2.1 **Overview of the spatial distribution and intensity of point-source discharges in Oregon (MS4 and NPDES permits).**

Table 2.5.2.1.1 and Table 2.5.2.2.2 provide permit-specific information on pollutants for each class of stormwater (MS4) and NPDES permit (*i.e.*, industrial, domestic), where available. For MS4 permits, permit-specific parameters are listed where information was available. For unspecified MS4 permits, NMFS reviewed 91 MS4 permits with specific parameters and identified stormwater parameters common to all reviewed permits, and used this information as a surrogate for the unspecified MS4 permits. Industrial and domestic NPDES permits are categorized as either major (discharge greater than 1 million gallons per day) or minor (discharge less than 1 million gallons per day).

Compounds that are discharged under existing MS4 and/or NPDES permits in Oregon that are listed in Table 1.1:

- Aluminum
- Ammonia
- Arsenic
- Cadmium
- Chromium (III)
- Chromium (VI)
- Copper
- Lead
- Nickel
- Pentachlorophenol
- Selenium
- Silver
- Tributyltin
- Zinc

Compounds listed in Table 1.1 that are associated with 303(d)-listed waters in Oregon:

- Ammonia
- Arsenic
- Cadmium
- Copper
- Dieldrin
- Heptachlor epoxide
- Lead
- Nickel
- Zinc

2.5.2.1 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution.

For SR sockeye salmon, UCR spring Chinook salmon, and UCR steelhead, the ESU/DPS boundaries are outside of the action area, and there are no NPDES or MS4 permits that occur in the action area that overlap with the ESU/DPS boundaries for these species. Therefore, MS4 and NPDES permit, and fish distribution data for these species are not reported in this section. However, smolts and adults will be exposed to stressors of the action as fish pass through the Columbia River, RM zero to RM 297, and in the Pacific Ocean from the mouth of the Columbia River to nautical mile 3.

Table 2.5.2.1.1 through Table 2.5.2.2.4 identify the ESU/DPS, number of populations in Oregon, the number of populations in Oregon without direct exposure to MS4 and/or NPDES point sources, the number of MS4 and/or NPDES point source discharges, and the compounds

associated with each permit type. Figure 2.5.2.1.1 through Figure 2.5.2.1.17 identify the approximate location of each MS4 and/or NPDES permits in each watershed, fish habitat distribution, fish habitat use, and population.

Table 2.5.2.1.1 SR fall-run Chinook Salmon populations in Oregon. Three of eight spawning populations occur in Oregon.

ESU/DPS	Populations in Oregon
SR fall-run Chinook	Snake River—Major Population Group Grande Ronde Snake River Imnaha

Table 2.5.2.1.2 Type, number, and chemicals discharged for MS4 and NPDES permits within the SR fall-run Chinook salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	None	
NPDES	None	

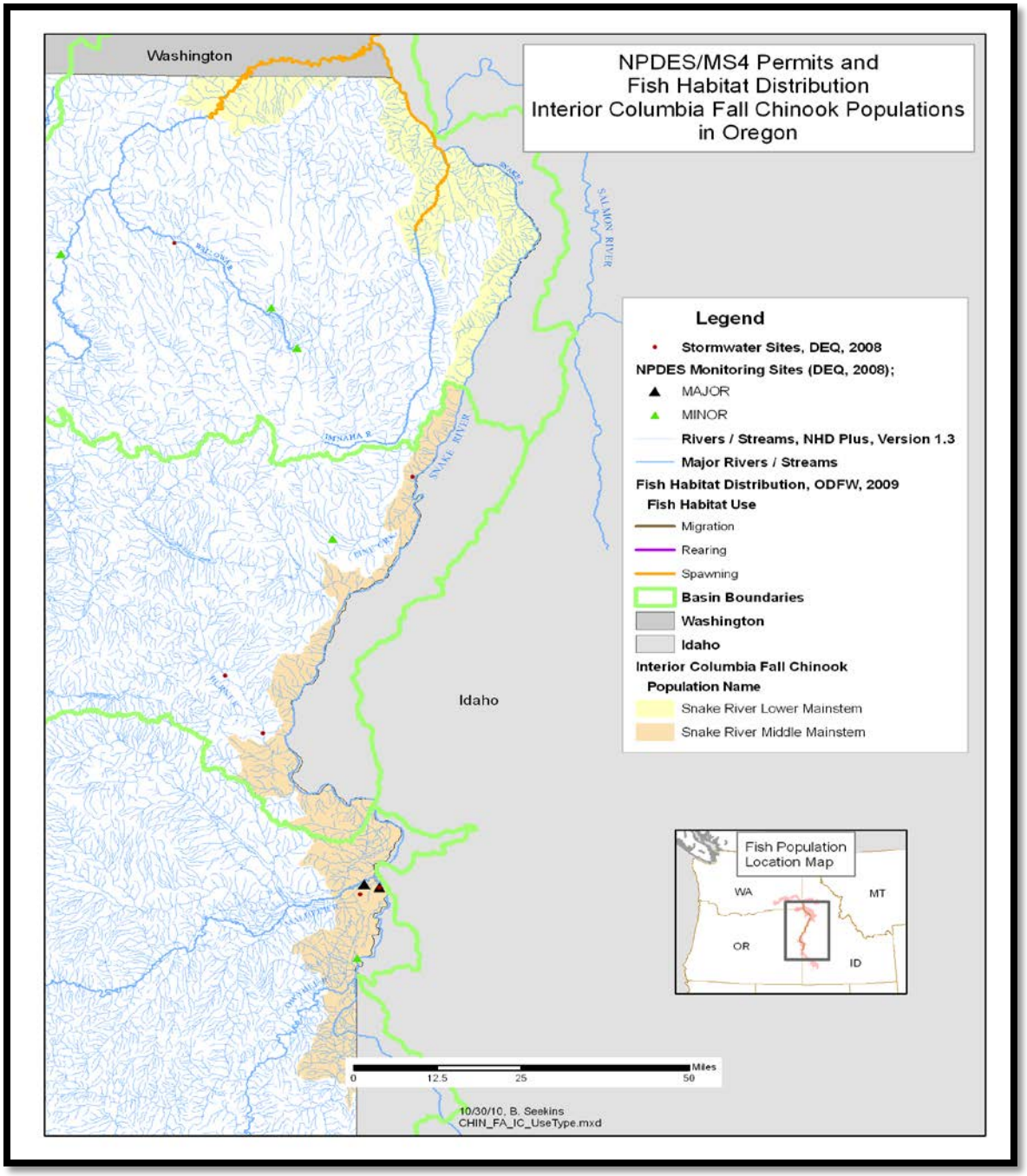

Figure 2.5.2.1.1 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for SR fall-run Chinook salmon.

Table 2.5.2.1.3 SRB steelhead populations in Oregon. Five of 24 populations occur in Oregon.

ESU/DPS	Populations in Oregon
SRB Steelhead	Wallowa River
	Grande Ronde River Upper Mainstem
	Imnaha River
	Joseph Creek
	Grande Ronde River Lower Mainstem

Table 2.5.2.1.4 Type, number, and chemicals discharged for MS4 and NPDES permits within the SRB steelhead DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	2	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	5	Ammonia, Zinc, Lead, Copper

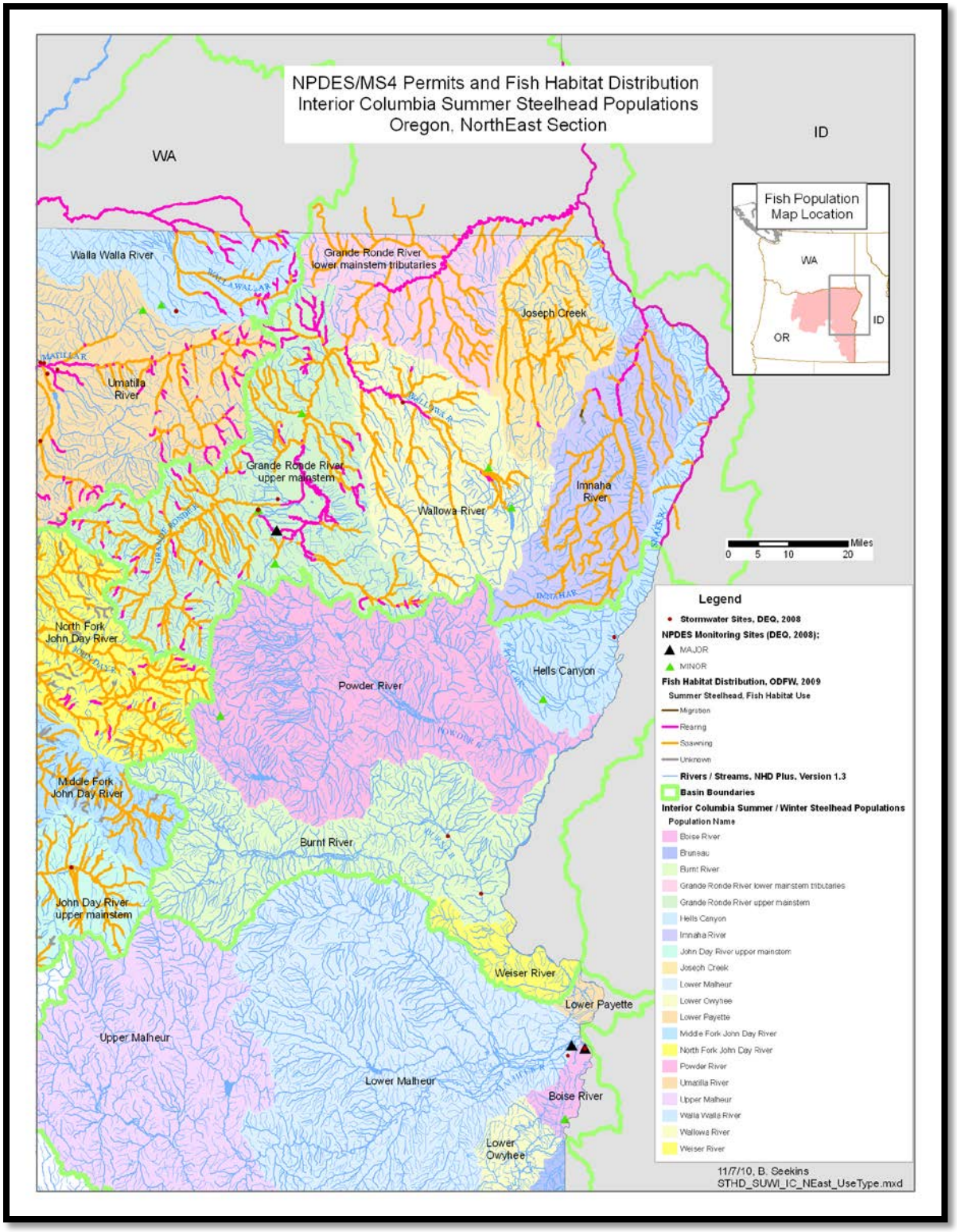

Figure 2.5.2.1.2 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for SRB steelhead.

Table 2.5.2.1.5 SR spring/summer Chinook salmon populations in Oregon. Eight of 27 populations occur in Oregon.

ESU/DPS	Populations In Oregon
SR Spring/Summer-Run Chinook	Grande Ronde UM
	Catherine Creek
	Lostine River
	Imnaha River
	Big Sheep Creek
	Minam River
	Looking Glass Creek
	Wenaha River

Table 2.5.2.1.6 Type, number, and chemicals discharged for MS4 and NPDES permits within the SR spring/summer Chinook salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	2	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	5	Ammonia, Zinc, Lead, Copper

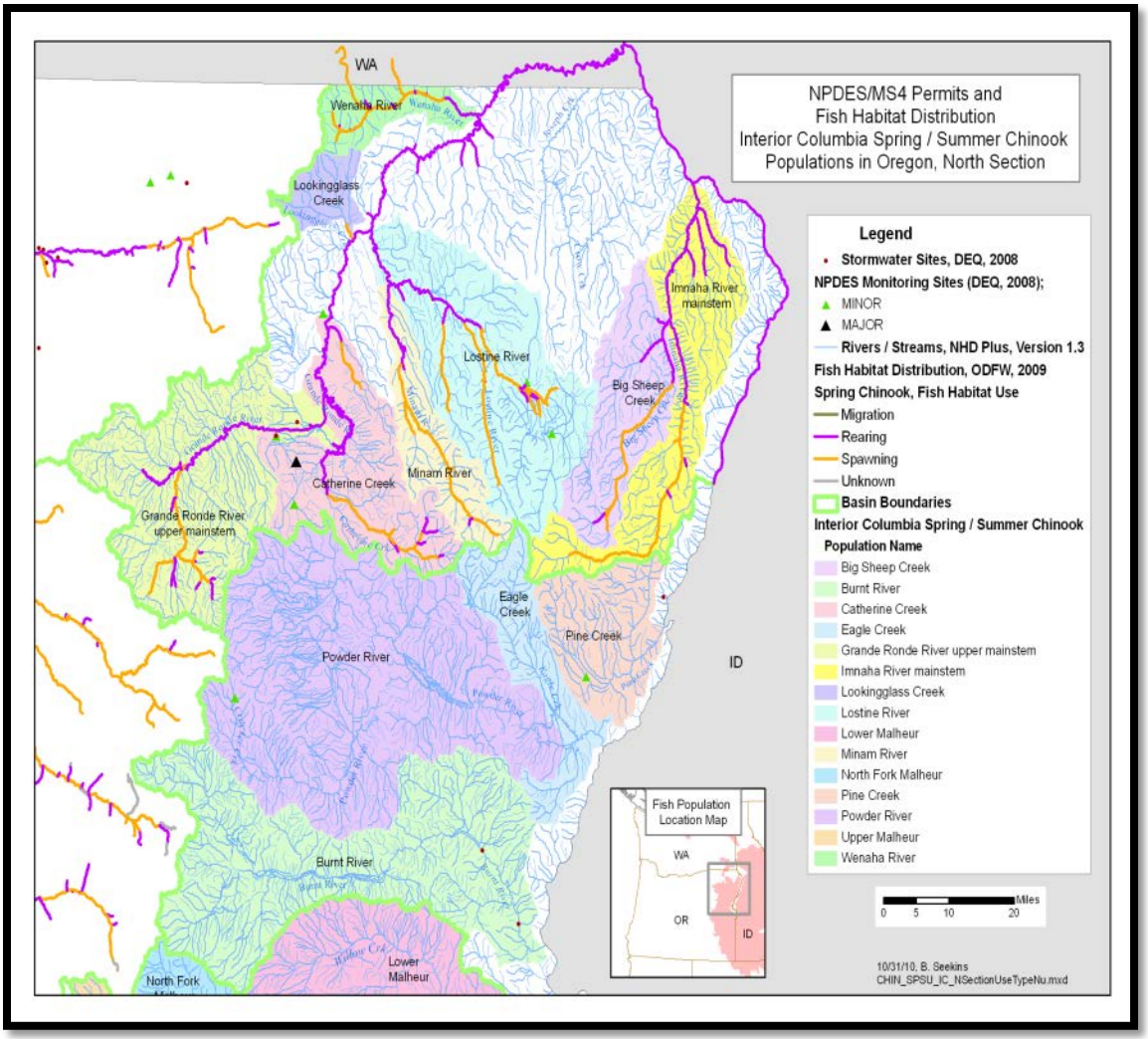

Figure 2.5.2.1.3 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for SR spring/summer-run Chinook salmon.

Table 2.5.2.1.7 MCR steelhead populations in Oregon. Ten of 17 populations occur in Oregon.

ESU/DPS	Populations In Oregon
MCR Steelhead	Walla Walla
	Umatilla River
	John Day Lower Mainstem
	John Day North Fork
	John Day Middle Fork
	John Day Upper Mainstem
	John Day South Fork
	Deschutes Westside
	Deschutes Eastside
	Fifteen Mile Creek

Table 2.5.2.1.8 Type, number, and chemicals discharged for MS4 and NPDES permits within the MCR steelhead DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	21	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	11	Ammonia, Lead, Copper, Zinc

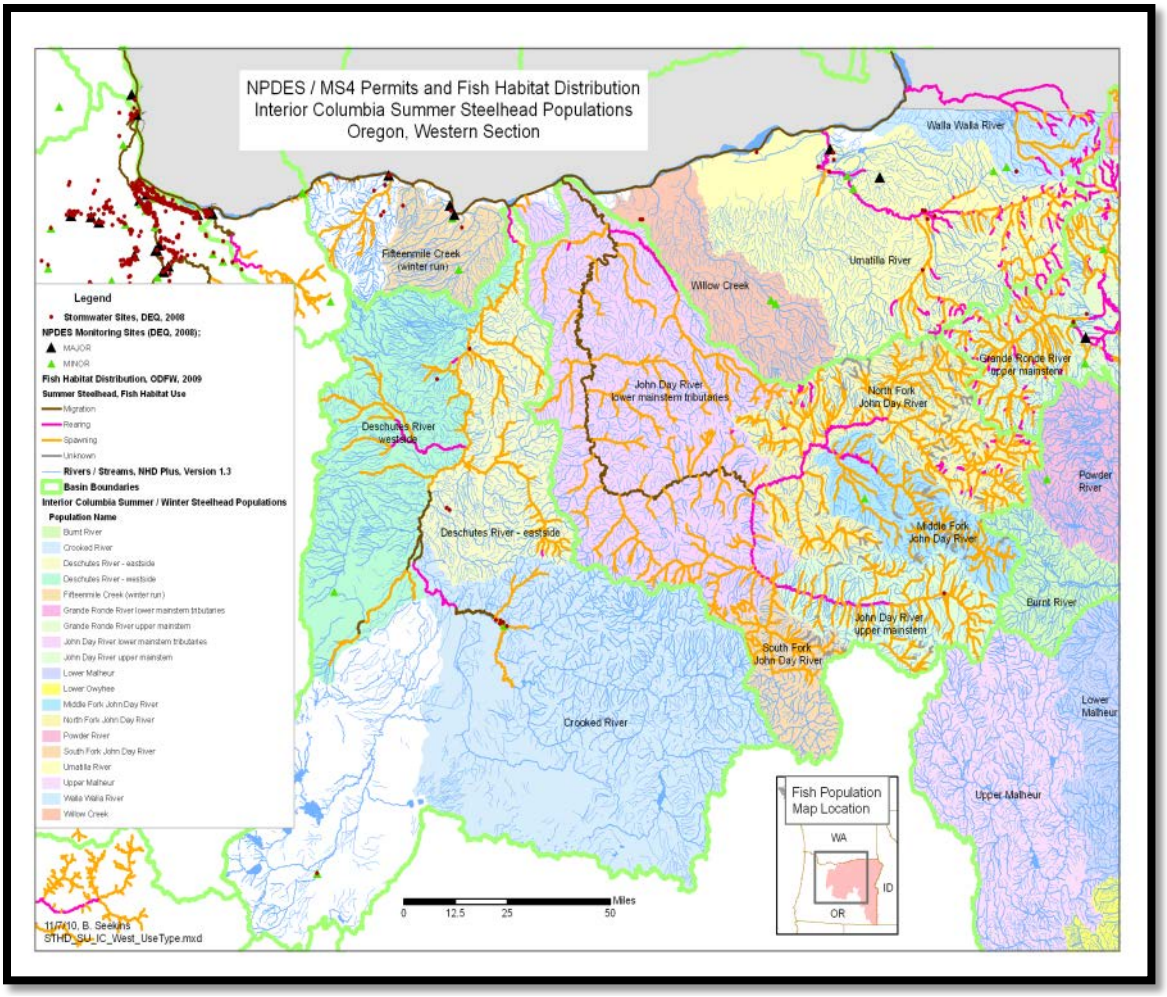

Figure 2.5.2.1.4 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for MCR steelhead.

Table 2.5.2.1.9 LCR Chinook salmon populations in Oregon. Nine of 32 populations occur in Oregon.

ESU/DPS	Populations In Oregon
LCR Chinook Salmon	Hood River (F+S)
	Sandy River (F+S)
	Lower Gorge Tributaries
	Clackamas
	Upper Gorge Tributaries
	Scappoose
	Clatskanine
	Big Creek
	Youngs Bay

Table 2.5.2.1.10 Type, number, and chemicals discharged for MS4 and NPDES permits within the LCR Chinook salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	654	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	48	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

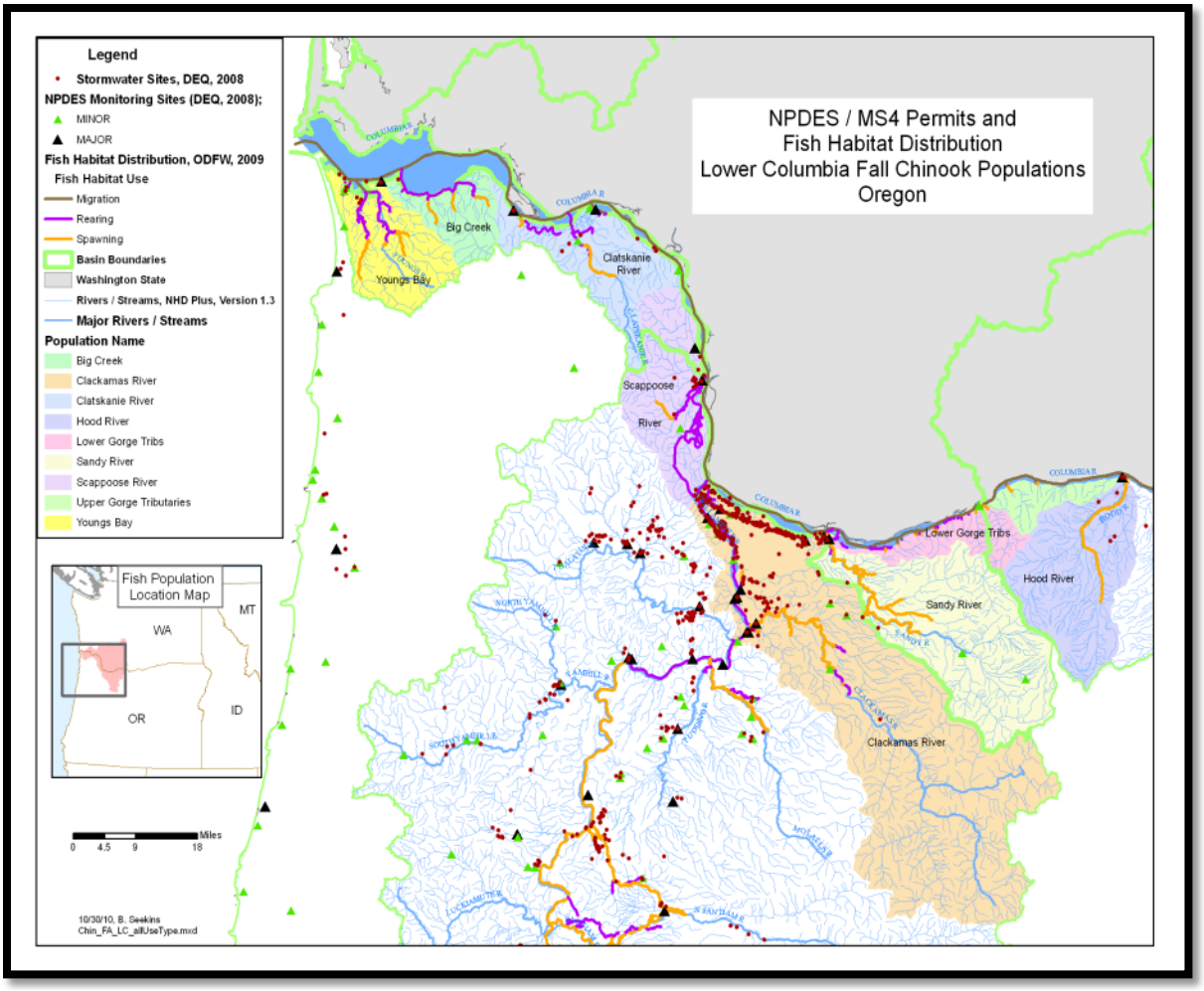

Figure 2.5.2.1.5 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR Chinook salmon.

Table 2.5.2.1.11 CR chum salmon populations in Oregon. One of 17 populations occurs in Oregon (14 of 17 chum populations remain extirpated or nearly so).

ESU/DPS	Populations In Oregon
CR Chum Salmon	Lower Gorge Tributaries/Mainstem
	Big Creek
	Clackamas
	Clatskanine
	Sandy
	Scappose
	Upper Gorge Tributaries
	Youngs Bay

Table 2.5.2.1.12 Type, number, and chemicals discharged for MS4 and NPDES permits within the CR chum salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	654	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	48	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

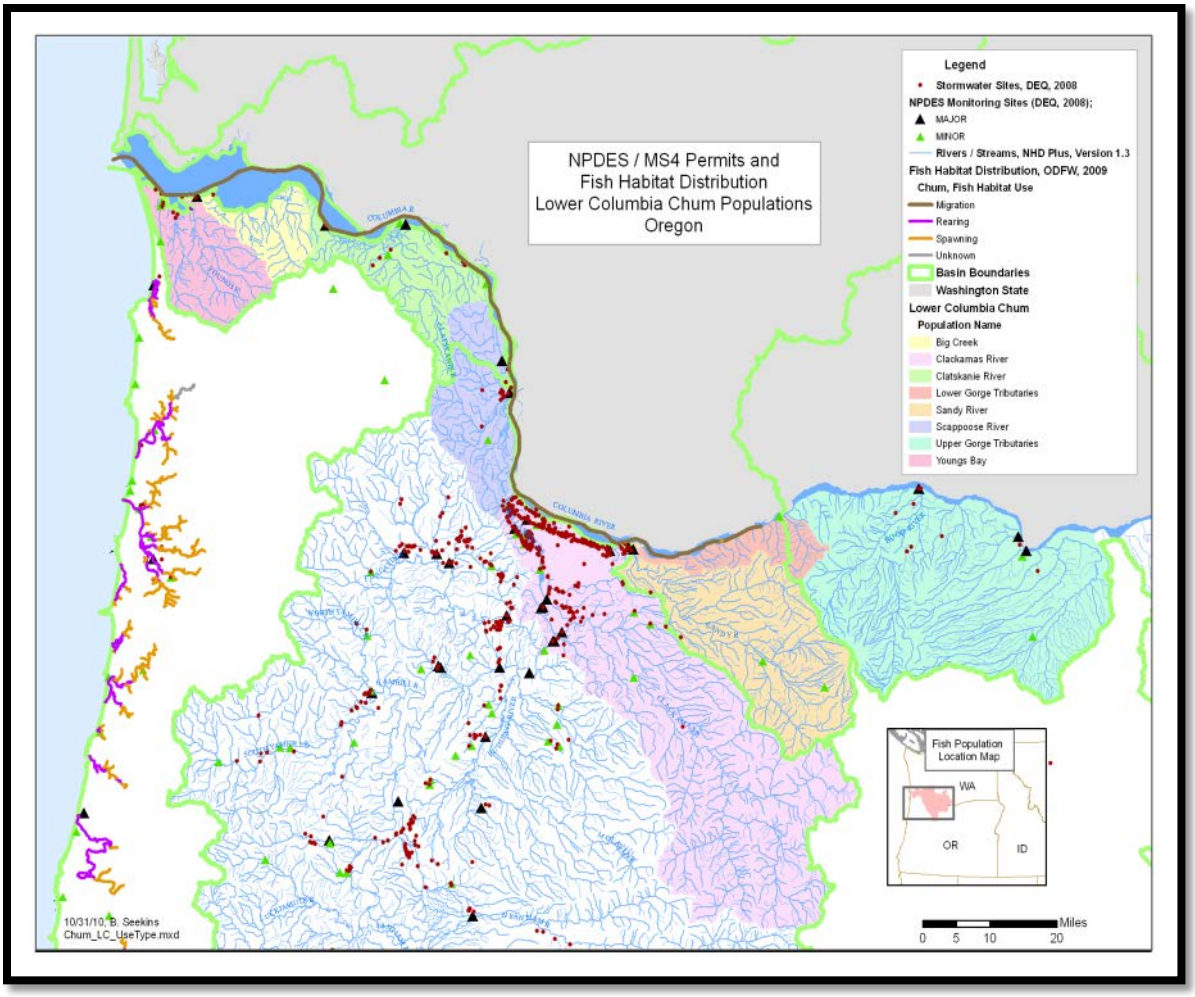

Figure 2.5.2.1.6 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for CR chum salmon.

Table 2.5.2.1.13 LCR coho salmon populations in Oregon. Eight of 27 populations occur in Oregon.

ESU/DPS	Populations In Oregon
LCR Coho Salmon	Big Creek
	Clackamas
	Clatskanie
	Lower Gorge Tributaries
	Upper Gorge and Hood River
	Sandy
	Scappose
	Youngs Bay

Table 2.5.2.1.14 Type, number, and chemicals discharged for MS4 and NPDES permits within the LCR coho salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	654	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	48	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

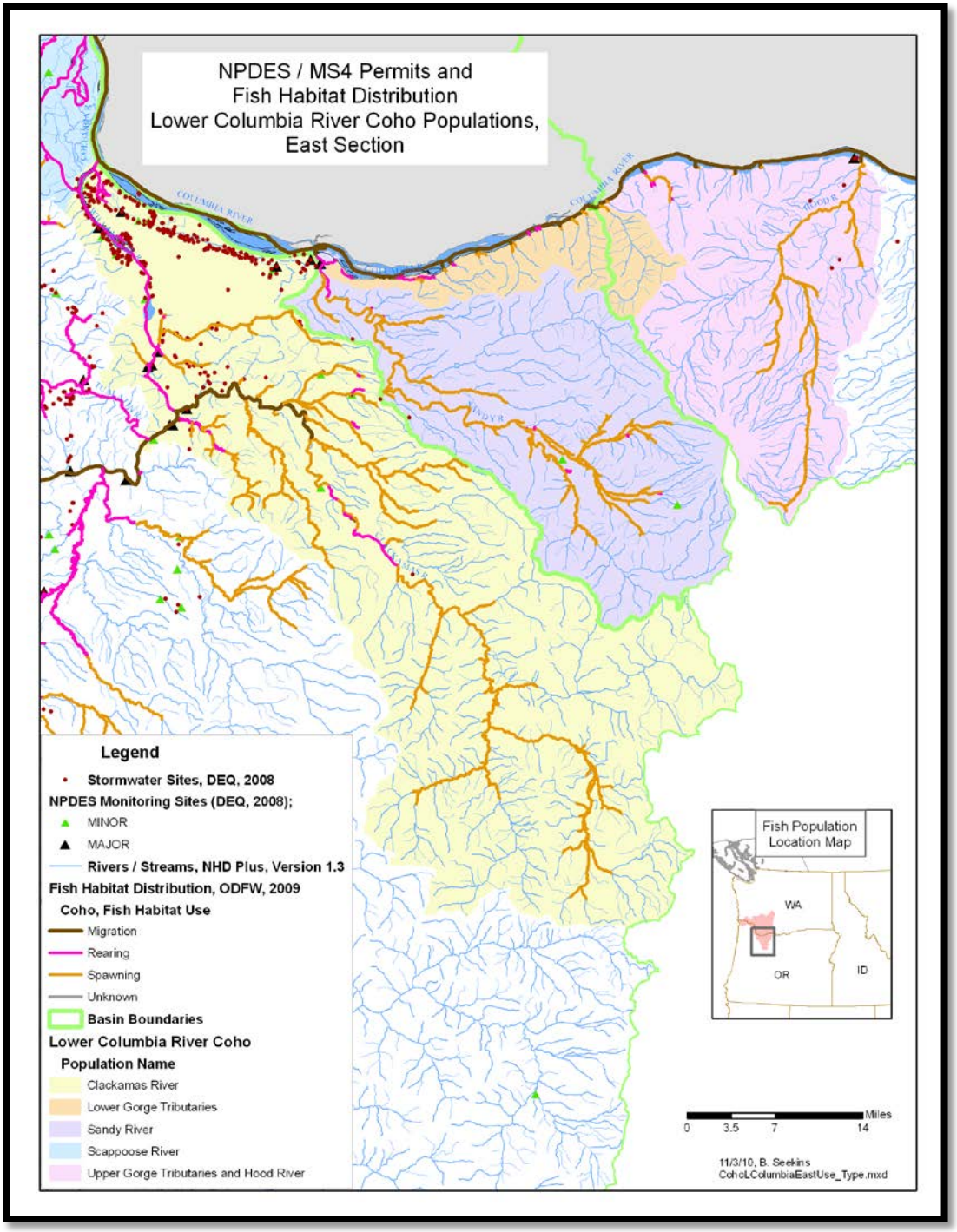

Figure 2.5.2.1.7 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR coho salmon (map 1 of 2).

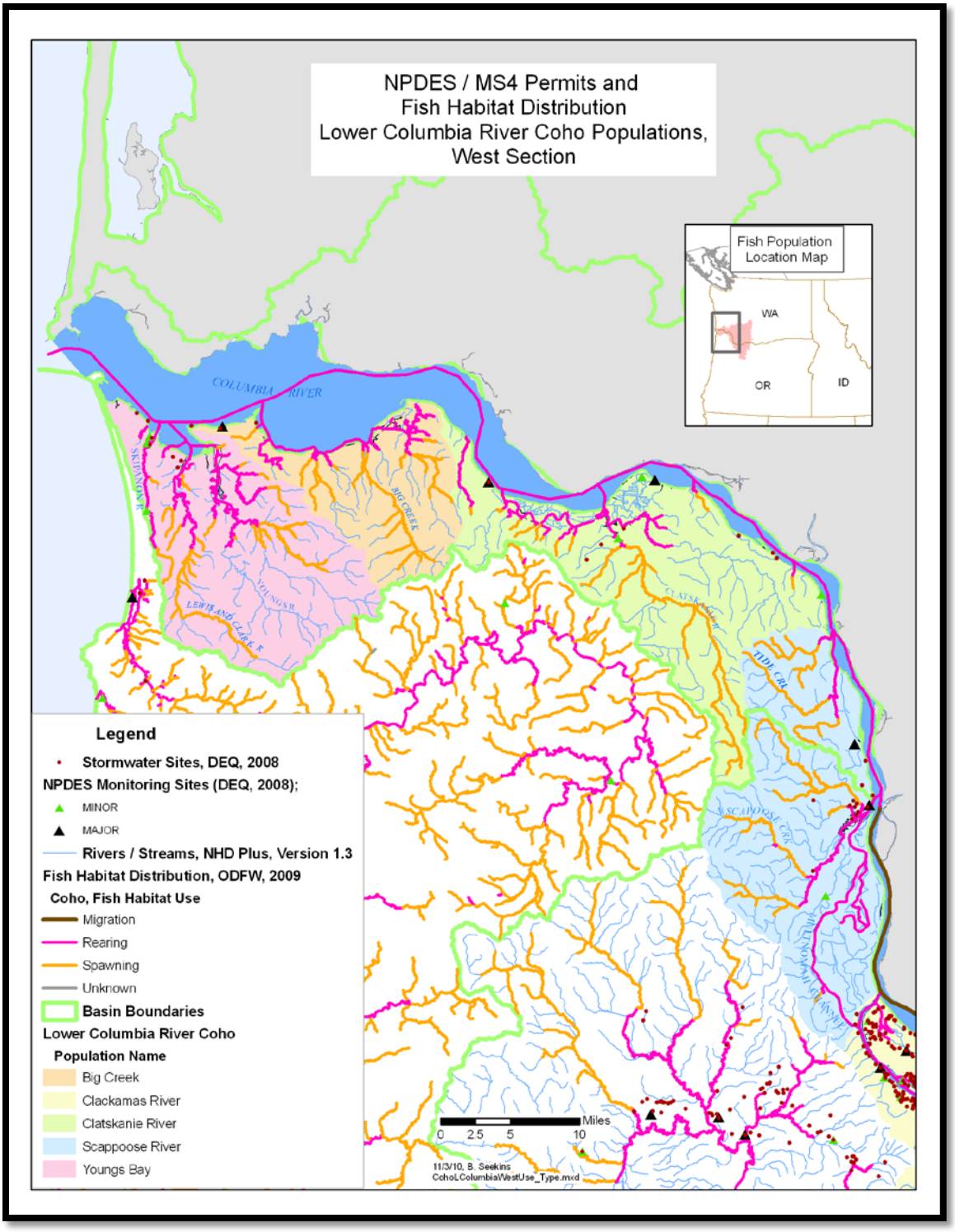

Figure 2.5.2.1.8 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR coho salmon (map 2 of 2).

Table 2.5.2.1.15 UWR steelhead populations in Oregon. All five populations occur in Oregon.

ESU/DPS	Populations In Oregon
UWR Steelhead	Calapooia River
	Molalla River
	North Santiam
	South Santiam
	Westside Tributaries
	Willamette River—Mainstem

Table 2.5.2.1.16 Type, number, and chemicals discharged for MS4 and NPDES permits within the UWR steelhead DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	118	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	50	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

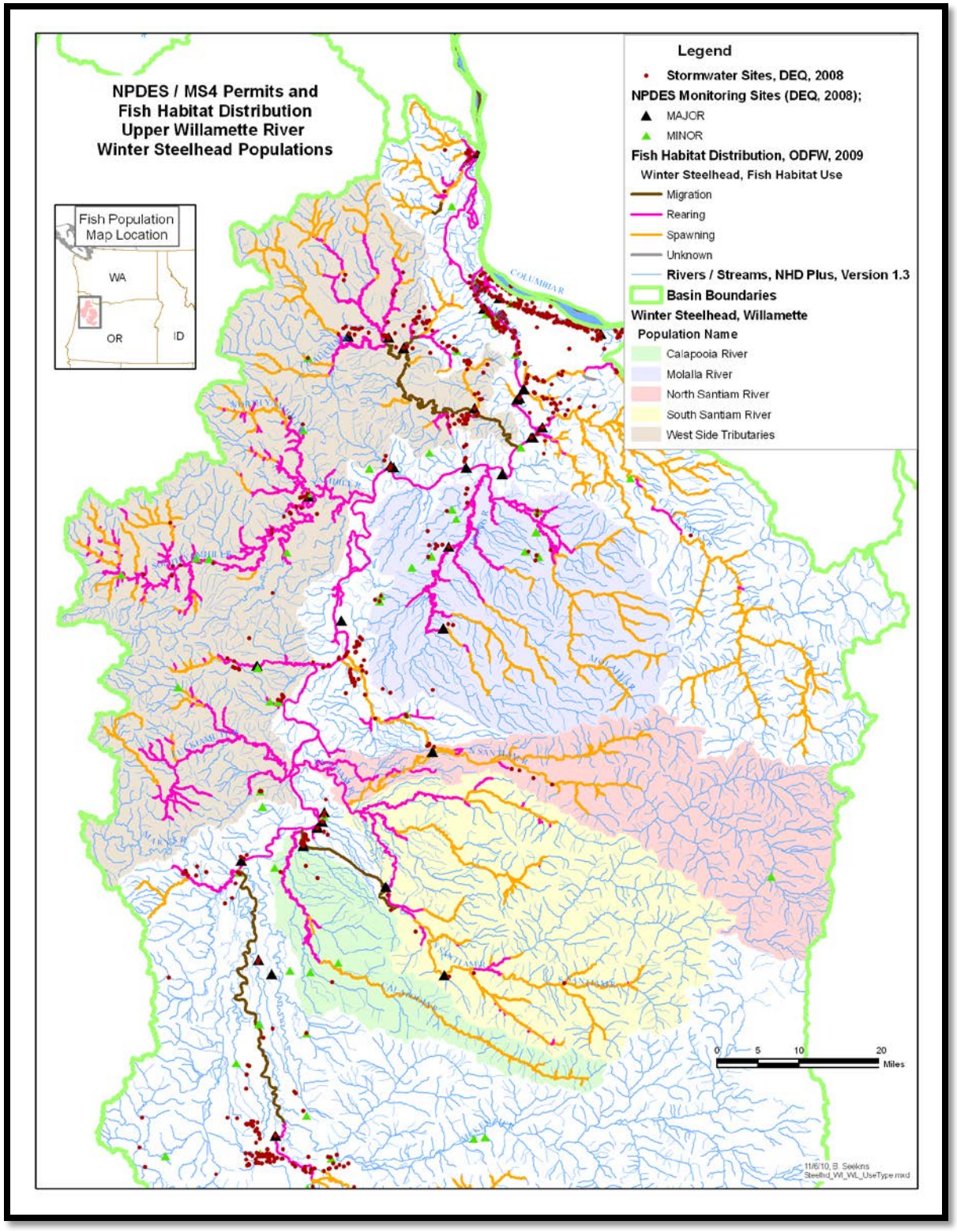

Figure 2.5.2.1.9 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for UWR steelhead.

Table 2.5.2.1.17 UWR Chinook salmon populations in Oregon. All seven populations occur in Oregon.

ESU/DPS	Populations In Oregon
UWR Chinook Salmon	Calapooia
	Clackamas
	McKenzie
	Middle Fork
	Molalla
	North Santiam
	South Santiam
	Willamette River—Mainstem

Table 2.5.2.1.18 Type, number, and chemicals discharged for MS4 and NPDES permits within the UWR Chinook salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	140	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	55	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

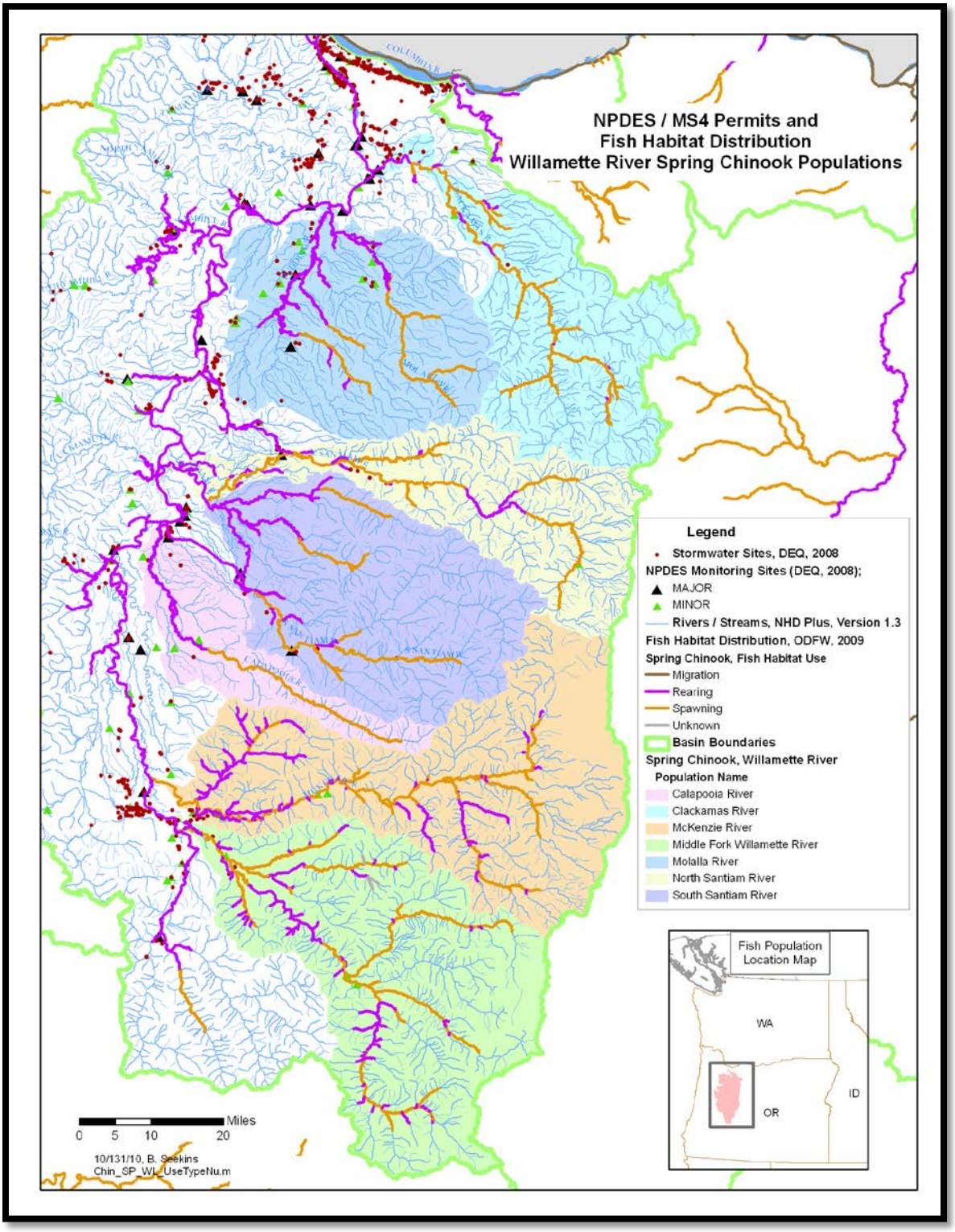

Figure 2.5.2.1.10 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for UWR Chinook salmon (map 1 of 2).

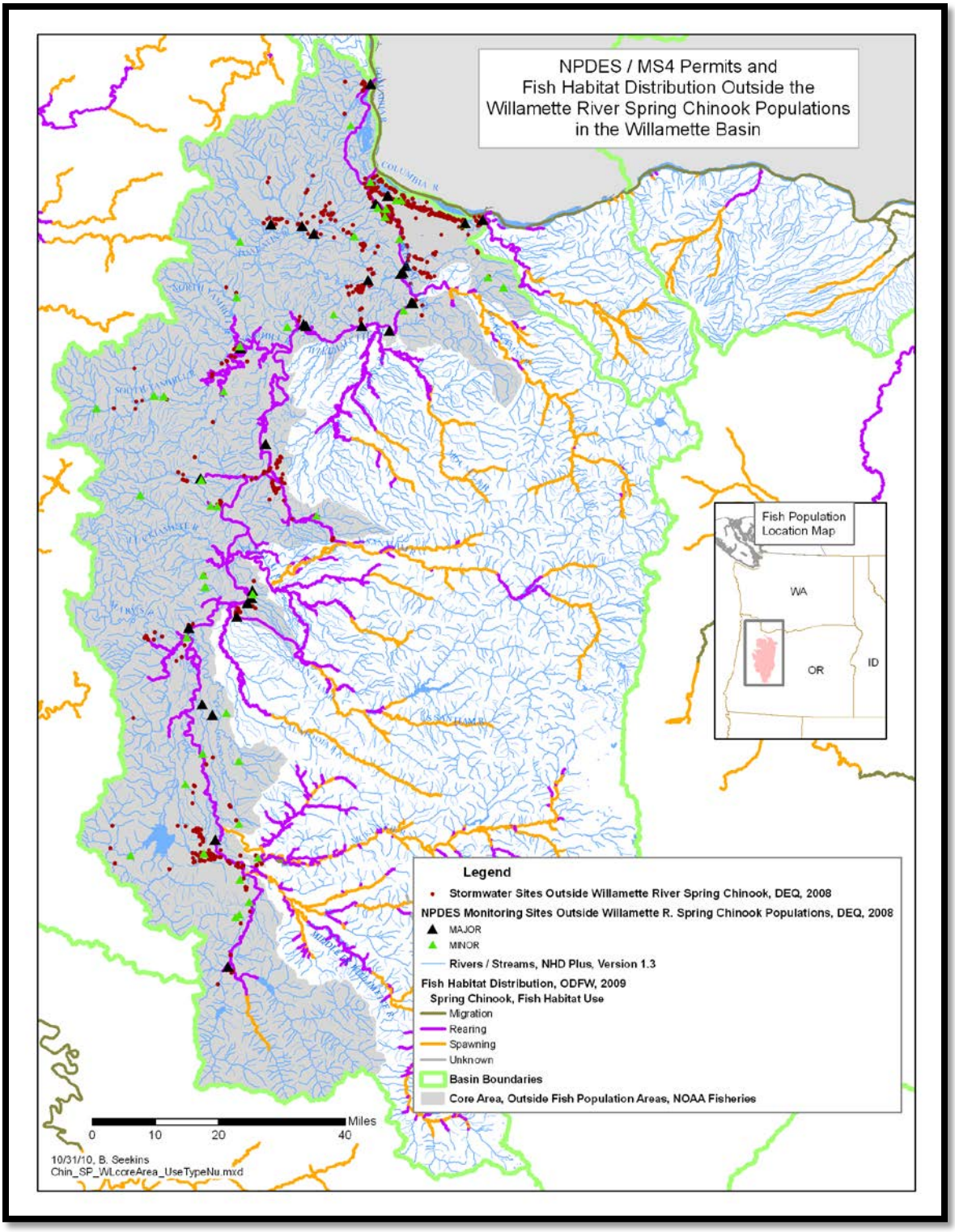

Figure 2.5.2.1.11 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for UWR Chinook salmon, non-core areas (map 2 of 2).

Table 2.5.2.1.19 LCR steelhead populations in Oregon. Five of 26 populations occur in Oregon.

ESU/DPS	Populations In Oregon
LCR Steelhead	Clackamas
	Hood River
	Lower Gorge Tributaries
	Upper Gorge Tributaries
	Sandy River

Table 2.5.2.1.20 Type, number, and chemicals discharged for MS4 and NPDES permits within the LCR steelhead DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	320	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	31	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

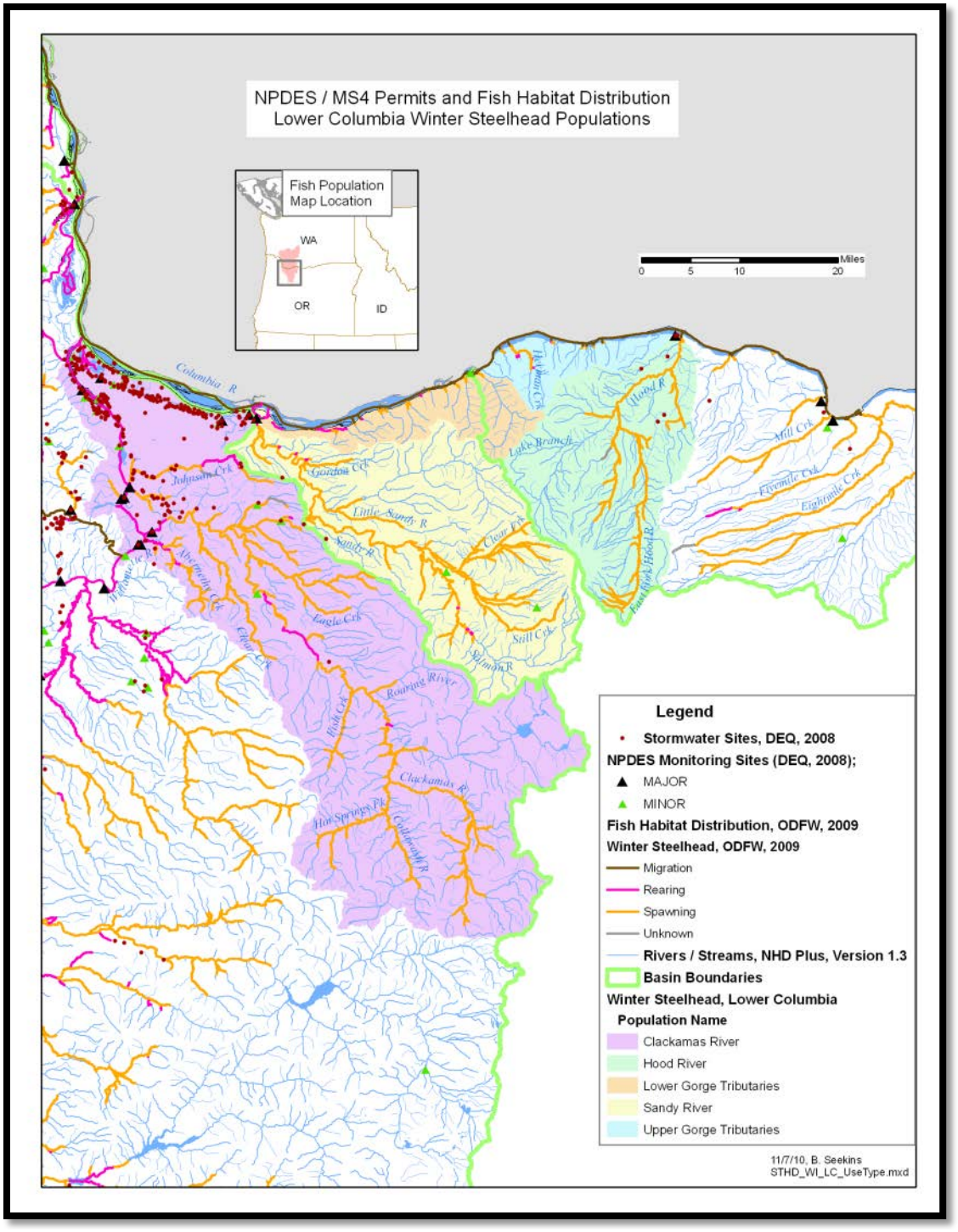

Figure 2.5.2.1.12 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR steelhead (winter).

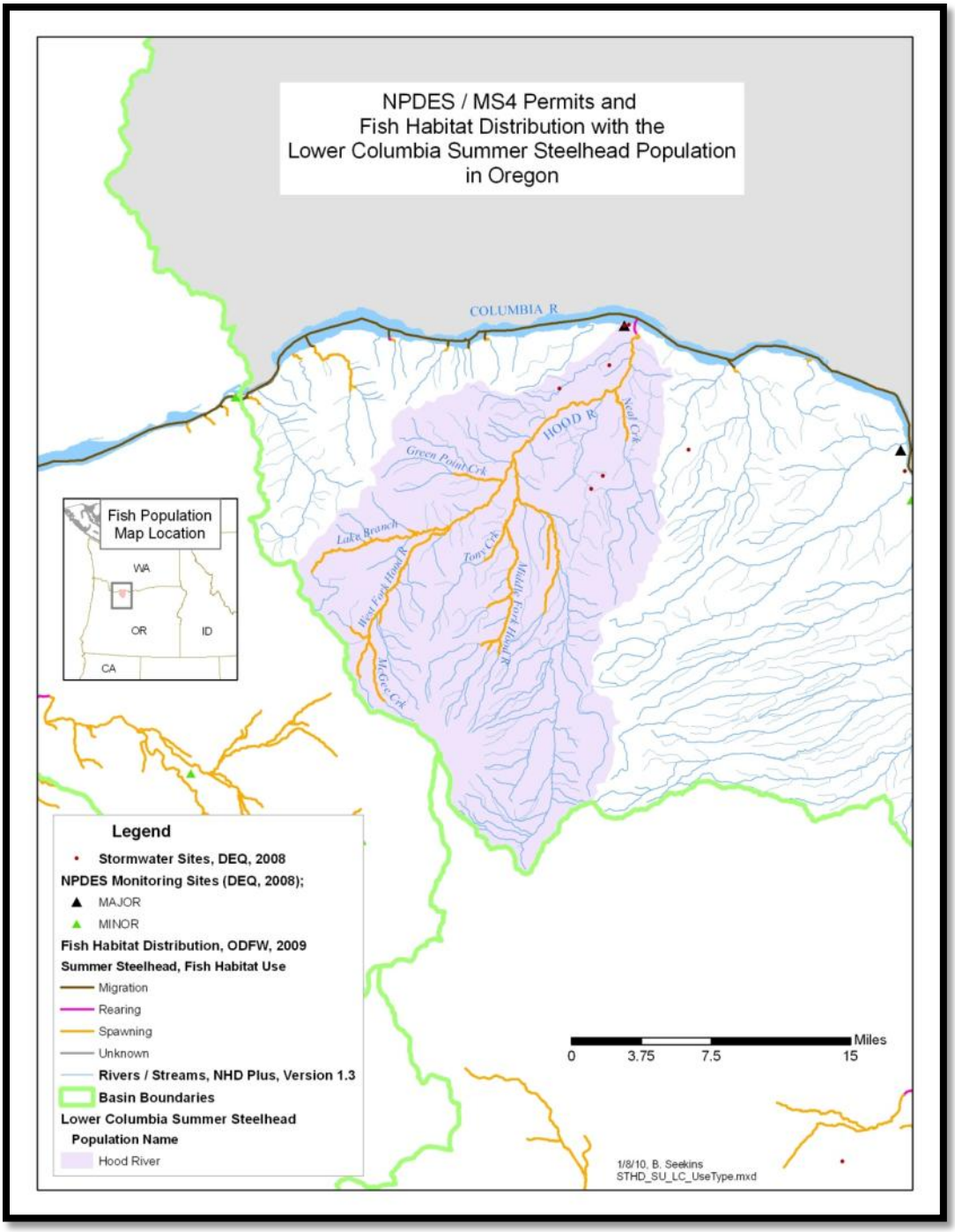

Figure 2.5.2.1.13 LCR Steelhead (summer). MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR steelhead (summer).

Table 2.5.2.1.21 OC coho salmon populations in Oregon. All 56 populations occur in Oregon.

ESU/DPS	Populations In Oregon	
OC Coho Salmon	Necanicum	Devils Lake
	Ecola	Siltcoos
	Arch Cape	Siletz
	Short Sands	Tahkenitch
	Nehalem	Schoolhouse
	Spring	Threemile
	Watseco	Fogarty
	Netarts	Depoe Bay
	Rover	Lower Umpqua
	Sand	Middle Umpqua
	Nestucca	North Umpqua
	Neskowin	South Umpqua
	Alsea	Spencer
	Big (near Alsea)	Wade
	Rocky	Big
	Vingie	Coal
	Yachats	Tenmile
	Cummins	Moolack
	Bob	Coos
	Tenmile Creek	Big (near Yaquina)
	Tillamook Bay	Coquille
	Rock	Yaquina
	China	Johnson
	Cape	Theil
	Berry	Twomile
	Sutton (Mercer Lake)	Beaver
	Salmon	Floras/New
	Siuslaw	Sixes

Table 2.5.2.1.22 Type, number, and chemicals discharged for MS4 and NPDES permits within the OC coho salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	92	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	43	Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Selenium, Silver, Zinc

Figure 2.5.2.1.14 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for OC coho salmon (north coast).

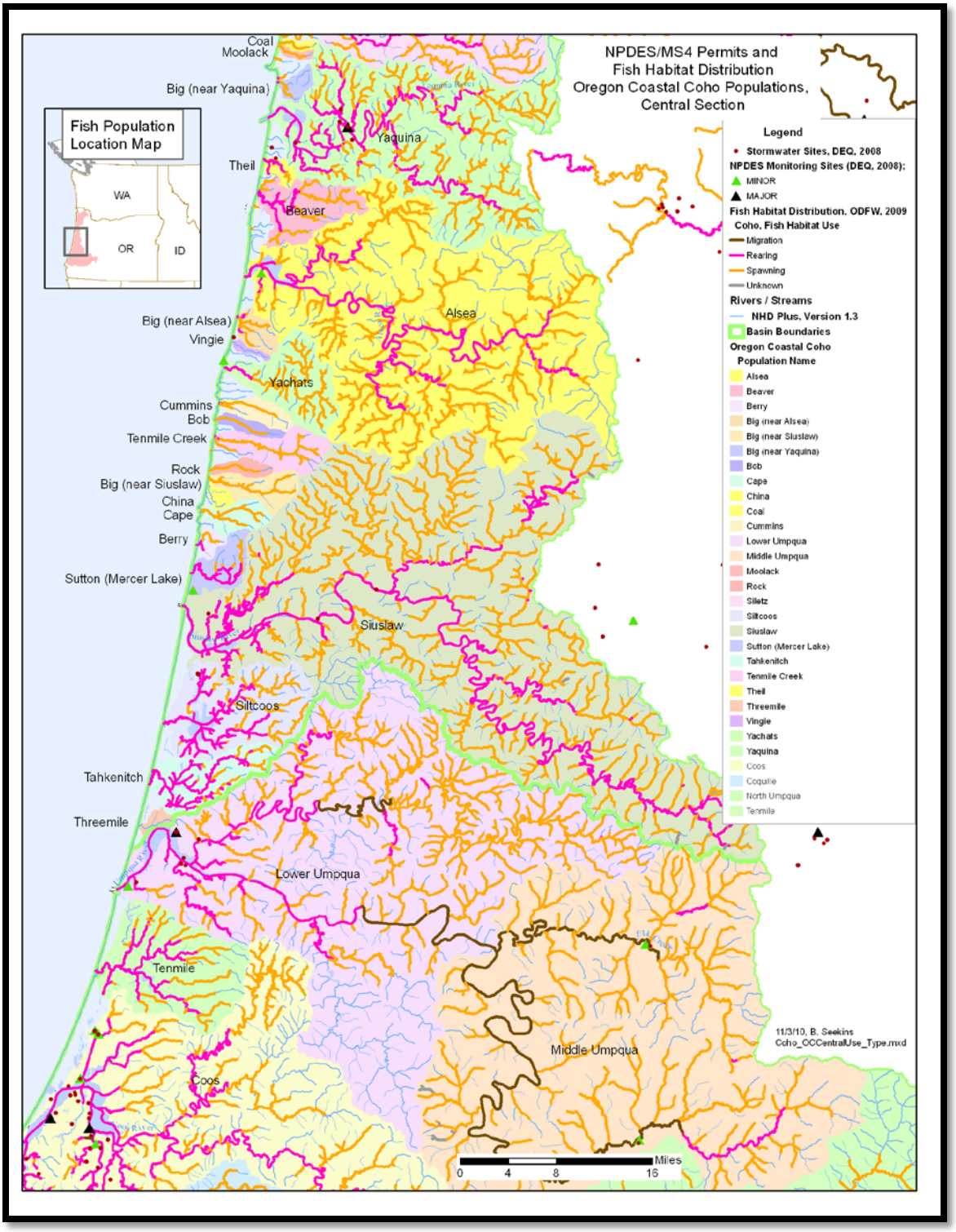

Figure 2.5.2.1.15 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for OC coho salmon (central coast).

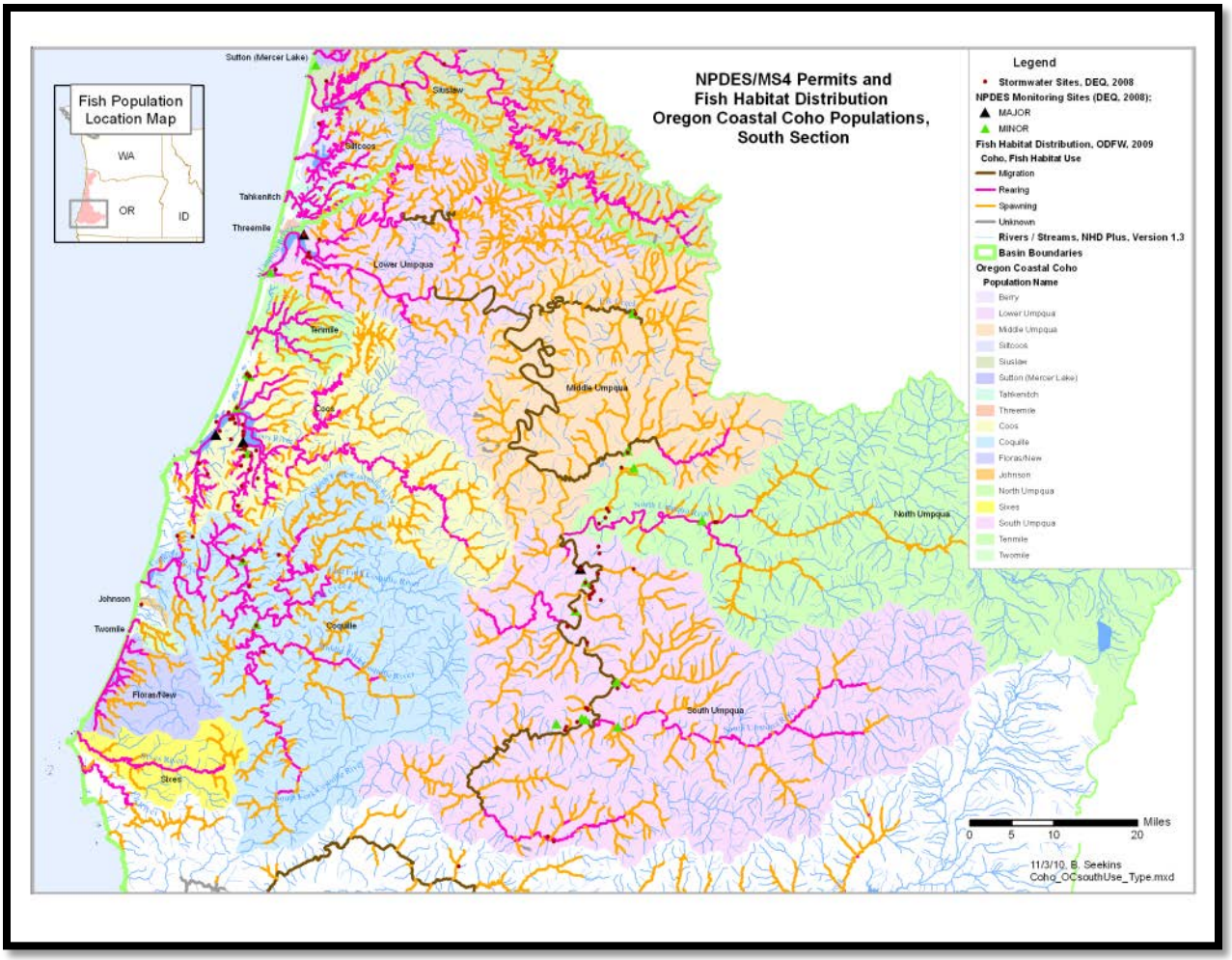

Figure 2.5.2.1.16 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for OC coho salmon (south coast).

Table 2.5.2.1.23 SONCC coho salmon populations in Oregon. Seventeen of 42 populations occur in Oregon.

ESU/DPS	Populations In Oregon
SONCC Coho Salmon	Bush Creek
	Chetco
	Elk
	Euchre
	Hubbard
	Hunter
	Illinois (OR and CA)
	Lower Rouge
	Middle Rouge and Applegate
	Mill Creek
	Mussel Creek
	Pistol
	Smith (OR and CA)
	Upper Klamath (OR and CA)
	Upper Rogue
	Winchuck River
	Brush Creek

Table 2.5.2.1.24 Type, number, and chemicals discharged for MS4 and NPDES permits within the SONCC coho salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	62	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	12	Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Selenium, Silver, Zinc

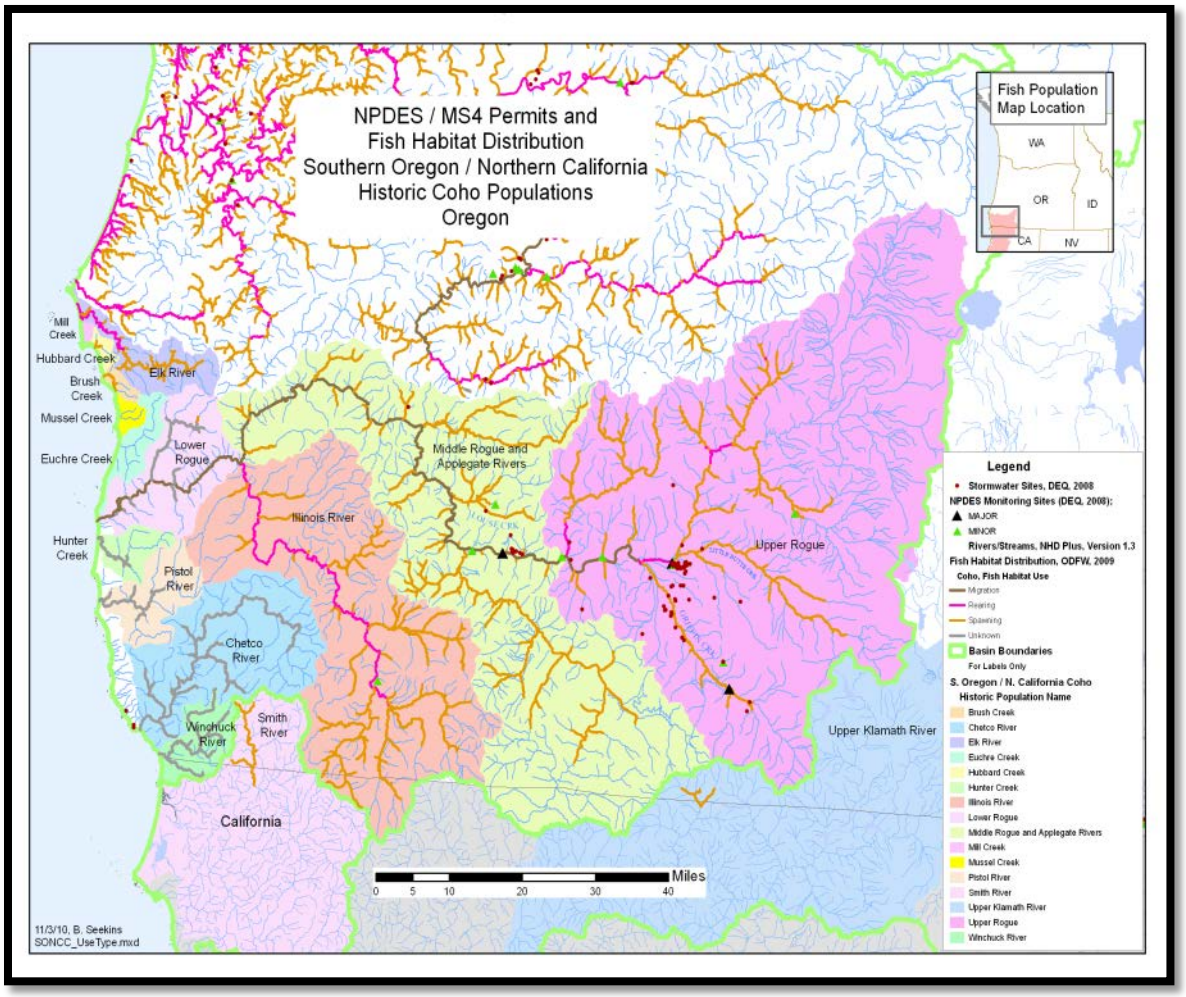

Figure 2.5.2.1.1.17 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for SONCC coho salmon (Oregon populations).

2.5.2.2 Other Anadromous Fishes

2.5.2.2.1. Green Sturgeon

Table 2.5.2.2.1.1 No resident populations occur in Oregon.

ESU/DPS	Populations In Oregon
Green Sturgeon	NA

Table 2.5.2.2.1.2 Type, number, and chemicals discharged for MS4 and NPDES permits in Oregon that overlap with green sturgeon distribution (migratory).

Type of Permit	Number	Chemical(s)
MS4	324	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	23	Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Selenium, Silver, Tributyltin, Zinc

2.5.2.2.2. Eulachon

Table 2.5.2.2.2.1 Type, number, and chemicals discharged for MS4 and NPDES permits within the eulachon DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	327	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	26	Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Selenium, Silver, Tributyltin, Zinc

Table 2.5.2.2.2.2. Eulachon populations in Oregon. Six of 24 populations occur in Oregon.

ESU/DPS	Populations In Oregon
Eulachon	Chetco
	Umpqua
	Ten Mile Creek
	Hood River
	Sandy River
	Columbia River

Table 2.5.2.2.2.3 Regulated and unregulated toxics in the State of Oregon (ODEQ 2003). Compounds considered in this opinion for approval by EPA are shaded.

Aquatic Life Criteria				
	Freshwater	**Freshwater**	**Marine**	**Marine**
	Acute Criteria	**Chronic Criteria**	**Acute Criteria**	**Chronic Criteria**
Compound (µg/L)				
Antimony				
Arsenic *	360	190	69	36
Cadmium ***	3.9	1.1	43	9.3
Chromium III ***	1700	210		
Chromium VI *	16	11	1100	50
Copper ***	18	12	2.9	2.9
Lead ***	82	3.2	241	5.6
Mercury	2.4	0.012	2.1	0.025
Nickel ***	1400	160	75	8.3
Selenium *	260	35	410	54
Silver **	4.1	0.12	2.3	
Thallium				
Zinc ***	120	110	95	86
Cyanide	22	5.2	1	1
Asbestos				
Dioxin (2,3,7,8-TCDD)				
Acrolein				
Acrylonitrile				
Benzene				
Bromoform				
Carbon Tetrachloride				
Chlorobenzene				
Chlorodibromomethane				
Chloroform				
Dichlorobromomethane				
Dichloroethane 1,2-				
Dichloroethylene 1,1-				
Dichloropropane 1,2-				
Dichloropropene 1,3-				
Ethylbenzene				
Methyl Bromide				
Methylene Chloride				
Tetrachloroethane 1,1,2,2-				
Tetrachloroethylene				
Toluene				
Dichloroethylene 1,2-Trans-				
Trichloroethane 1,1,2-				
Trichloroethylene				
Vinyl Chloride				
Chlorophenol 2-				
Dichlorophenol 2,4-				

Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
Dimethylphenol 2,4-				
Methyl-4,6-Dinitrophenol 2-				
Dinitrophenol 2,4-				
Pentachlorophenol	20	13	13	7.9
Phenol				
Trichlorophenol 2,4,6-				
Acenaphthene				
Anthracene				
Benzidine				
BenzoaAnthracene				
BenzoaPyrene				
BenzobFluoranthene				
BenzokFluoranthene				
ChloroethylEther, Bis2-				
ChloroisopropylEther, Bis2-				
EthylhexylPhthalate, Bis2-				
Butylbenzyl Phthalate				
Chloronaphthalene 2-				
Chrysene				
Dibenzoa,hAnthracene				
Dichlorobenzene 1,2-				
Dichlorobenzene 1,3-				
Dichlorobenzene 1,4-				
Dichlorobenzidine 3,3'-				
DiethylPhthalate				
Dimethyl Phthalate				
Di-n-Butyl Phthalate				
Dinitrotoluene 2,4-				
Diphenylhydrazine 1,2-				
Fluoranthene				
Fluorene				
Hexachlorobenzene				
Hexachlorobutadiene				
Hexachlorocyclopentadiene				
Hexachloroethane				
Ideno1,2,3-cdPyrene				
Isophorone				
Nitrobenzene				
Nitrosodimethylamine, N-				
Nitrosodi-n-Propylamine, N-				
Nitrosodiphenylamine, N-				
Pyrene				
Trichlorobenzene 1,2,4-				
Aldrin	3.0		1.3	

Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
BHC, alpha-				
BHC, beta-				
BHC, gamma- (Lindane)	2	0.08	0.16	
Chlordane	2.4	0.0043	0.09	0.004
DDT 4,4'-	1.1	0.001	0.13	0.001
DDE 4,4'-				
DDD 4,4'-				
Dieldrin	2.5	0.0019	0.71	0.0019
Alpha-Endosulfan				
Beta-Endosulfan				
Endosulfan Sulfate				
Endrin	0.18	0.0023	0.037	0.0023
Endrin Aldehyde				
Heptachlor	0.52	0.0038	0.053	0.0036
Heptachlor Epoxide				
Polychlorinated biphenyls PCBs:	2	0.014	10	0.03
Toxaphene	0.73	0.0002	0.21	0.0002
Aluminum				
Ammonia (mg/L)	6	0.76		
Barium				
Chloride	860000	230000		
Chlorine	19	11	13	7.5
Chlorophenoxy Herbicide 2,4,5,-TP				
Chlorophenoxy Herbicide 2,4-D				
Chloropyrifos	0.083	0.041	0.011	0.0056
Demeton		0.1		0.1
Ether, Bis Chloromethyl				
Guthion		0.01		0.01
Hexachlorocyclo-hexane-Technical				
Iron		1000		
Malathion		0.1		0.1
Manganese				
Methoxychlor		0.03		0.03
Mirex		0.001		0.001
Nitrates				
Nitrosamines				
Dinitrophenols				
Nitrosodibutylamine,N				
Nitrosodiethylamine,N				
Nitrosopyrrolidine,N				
Parathion	0.065	0.013		
Pentachlorobenzene				
Phosphorus Elemental				0.1
Sulfide-Hydrogen Sulfide		2.0		2.0

Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
Tetrachlorobenzene,1,2,4,5				
Tributyltin TBT				
Trichlorophenol 2,4,5				
* all criteria expressed as dissolved metal ** all criteria expressed as dissolved metal. FW acute criteria are hardness dependent (concentration shown is hardness = 100 mg/L $CaCO_3$) *** all criteria expressed as dissolved metal. FW criteria are hardness dependent (concentration shown is hardness = 100 mg/L $CaCO_3$)				

The compounds listed in Table 2.5.2.3 that are not directly part of the proposed action (unshaded) are, however, part of EPA's overall approval of Oregon's water quality standards, and are compounds that are part of the environmental baseline. These compounds, either individually or in combination, are likely to adversely affect listed species considered in this opinion where exposure occurs. For example, concurrent exposure to cyanide and ammonia is likely to produce greater than additive effects to acute lethality in rainbow trout, salmon, and chub (Smith *et al.* 1979, Alabaster *et al*, 1983, and Douderoff 1976), and to sublethal effects to growth in rainbow trout (Smith *et al.* 1979). In rainbow trout and salmon, effects to acute lethality were 1.2 and 1.63 times greater than would be expected by additivity. Concurrent exposure to cyanide and zinc also resulted in synergistic effects to acute lethality in fathead minnows, where toxicity was 1.4 times that predicted by additivity (Smith *et al.* 1979).

Furthermore, Glubokoy (1990) reported increased mortality (0.7% to 10% above baseline) of coho salmon during early ontogeny when exposed to dichloro-diphenyl-trichloroethane (DDT) over the range of 0.1 µg/L to 10 µg/L, Niimi (1996) determined that 48 hour to 96 hour exposure to Polychlorinated biphenyls (PCB) concentrations on the order of 1 µg/L or more resulted in fish mortality, and Macek *et al.* (1969) reported a 96 hour LC_{50} value of 2.2 µg/L for rainbow trout exposed at 12.7EC, pH 7.1 in a static experiment with a 95% aldrin concentration.

2.5.2.2.3 Marine Mammals

Marine mammals are unlikely to be directly exposed to the subject pollutants, with the exception of Steller sea lions.

2.5.2.2.4 Sea Turtles

Sea turtles are unlikely to be directly exposed to the subject pollutants.

2.5.2.3 General Environmental Baseline Conditions

Columbia River Basin. Major tributaries to the Columbia River include the Snake, Willamette, Salmon, Flathead, and Yakima Rivers; smaller rivers include the Owyhee, Grande Ronde, Clearwater, Spokane, Methow, Cowlitz, and the John Day Rivers. The Snake River is the largest tributary at more than 1,000 miles long; its headwaters originate in Yellowstone National Park, Wyoming. The second largest tributary is the Willamette River in Oregon (Kammerer 1990, Hinck *et al.* 2004). The average annual discharge at the mouth of the Columbia River is 265,000 cubic feet per second (Kammerer 1990). A saltwater wedge extends 23 miles upstream of the mouth, with tidal influences extending up to 146 miles up river (Hinck *et al.* 2004). Table 2.5.2.3.1 provides information on selected tributaries to the Columbia River.

Table 2.5.2.3.1. Select tributaries of the Columbia River

Watershed	Approx Length (mi)	Basin Size (mi^2)	Physiographic Provinces*	Mean Annual Precip. (in)	Mean Discharge (cfs)
Snake/Salmon Rivers	870	108,495	CU, NR, MR, B/R	14	55,267
Willamette River	143	11,478	CS, PB	60	32,384

Data from Carter and Resh 2005

*Physiographic Provinces: CU = Columbia-Snake River Plateaus, NR = Northern Rocky Mountains, MR = Middle Rocky Mountains, B/R = Basin & Range, CS = Cascade-Sierra Mountains, PB = Pacific Border

Human Activities and Their Impacts.

Land Use. More than 50% of the United States portion of the Columbia River Basin is in Federal ownership (most of which occurs in high desert and mountain areas), 39% is in private land ownership (most of which occurs in river valleys and plateaus), and the remainder is divided among tribes, state, and local governments (Hinck *et al.* 2004) (Table 2.5.2.3.2).

Table 2.5.2.3.2. Land uses and population density in select tributaries of the Columbia River Basin.

Watershed	Land Use Categories (%)				Density (people/mi^2)
	Agriculture	Forest	Urban	Other	
Snake/Salmon Rivers	30	10-15	1	54 scrub/rangeland/barren	39
Willamette River	19	68	5	--	171

Data from Stanford *et al.* 2005

The interior Columbia River basin has been altered substantially by humans, causing dramatic changes and declines in native fish populations. In general the basin supports a variety of mixed uses. Predominant human uses include logging, agriculture, ranching, hydroelectric power generation, mining, fishing, a variety of recreational activities, and urban uses. The decline of salmon runs in the Columbia River is attributed to loss of habitat, blocked migratory corridors, altered river flows, pollution, overharvest, and competition from hatchery fish. Critical ecological connectivity (mainstem to tributaries and riparian floodplains) has been disconnected by dams and associated activities such as floodplain deforestation and urbanization. The most

productive floodplains of the watershed are either flooded by hydropower dams or dewatered by irrigation diversions. Portions of the basin are also subject to impacts from cattle grazing and irrigation withdrawals. In the Willamette River, riparian vegetation was greatly reduced by land conversion. By 1990, only 37 % of the riparian area within 120 meters was forested, 30% was agricultural fields and 16 % was urban or suburban lands.

Agriculture and Ranching. Roughly 6% of the annual flow from the Columbia River is diverted for the irrigation of 7.3 million acres of croplands within the basin. The vast majority of these agricultural lands are located along the lower Columbia River, the Willamette, Hood, and Snake rivers, and the Columbia Plateau (Hinck *et al.* 2004).

Agriculture and ranching increased steadily within the Columbia River basin from the mid to late 1800. By the early 1900s, agricultural opportunities began increasing at a much more rapid pace with the creation of more irrigation canals and the passage of the Reclamation Act of 1902 (NRC 2004). Today, agriculture represents the largest water user within the basin (>90%). Agriculture, ranching, and related services employ more than nine times the national average (19% of the households within the basin; NRC 2004).

Ranching practices have increased soil erosion and sediment loads within the Columbia' River's tributaries, the worst of these effects may have occurred in the late 1800s and early 1900s from deliberate burning to increase grass production (NRC 2004). Several measures are in use to reduce the impacts of grazing, including restricting grazing in degraded areas, reduced grazing allotments, and lower stocking rates. Today, agricultural impacts to water quality within the basin are second to large-scale influences of hydromodification projects for both power generation and irrigation. Water quality impacts from agricultural activities include alteration of the natural temperature regime, insecticide and herbicide contamination, and increased suspended sediments.

The USGS has a number of fixed water quality sampling sites throughout various tributaries of the Columbia River, many of which have been in place for decades. Water volumes, crop rotation patterns, crop type, and basin location are some of the variables that influence the distribution and frequency of pesticides within a tributary. Detection frequencies for a particular pesticide can vary widely. One study conducted by the USGS between May 1999 and January 2000 detected 25 pesticide compounds (Ebbert and Embrey 2001). Another study detected at least two pesticides or their breakdown products in 91% of the samples collected, with the median number of chemicals being eight, and a maximum of 26. The herbicide 2,4-D occurred most often in the mixtures, along with azinphos-methyl, the most heavily applied pesticide, and atrazine, one of the most mobile aquatic pesticides (Fuhrer *et al.* 2004). In addition to current-use chemicals, these legacy chemicals continue to pose a serious problem to water quality and fish communities despite their ban in the 1970s and 1980s (Hinck *et al.* 2004).

Fish and macroinvertebrate communities exhibit an almost linear decline in condition as the level of agriculture intensity increases within a basin (Cuffney *et al.* 1997, Fuhrer *et al.* 2004). A study conducted in the late 1990s examined 11 species of fish, including anadromous and resident fish collected throughout the Columbia River basin for a suite of 132 contaminants, including 51 semi-volatile chemicals, 26 pesticides, 18 metals, seven PCBs, 20 dioxins, and 10 furans. The

study revealed PCBs, metals, chlorinated dioxins and furans (products of wood pulp bleaching operations) and other contaminants within fish tissues; white sturgeon tissues contained the greatest concentrations of chlorinated dioxins and furans (Hinck *et al.* 2004).

Urban and Industrial Development. The largest urban area in the basin is the greater Portland metropolitan area. Portland's population exceeds 500,000, and the next largest cities Salem and Eugene, OR have over 100,000 people (Hinck *et al.* 2004). Overall, the basin's population density is one-third the national average, and while the basin covers about 8% of United States land, only about 1.2% of the United States population lives within the basin (Hinck *et al.* 2004).

Discharges from sewage treatment plants, paper manufacturing, and chemical and metal production represent the top three permitted sources of contaminants within the lower basin according to discharge volumes and concentrations (Rosetta and Borys 1996). Rosetta and Borys (1996) review of 1993 data indicate that 52% of the point source waste water discharge volume is from sewage treatment plants, 39% from paper and allied products, 5% from chemical and allied products, and 3% from primary metals. However, the paper and allied products industry are the primary sources of the suspended sediment load (71%). Additionally, 26% of the point source waste water discharge volume comes from sewage treatment plants and 1% is from the chemical and allied products industry. Nonpoint source discharges (urban stormwater runoff) account for significant pollutant loading to the lower basin, including most organics and over half of the metals. Although rural nonpoint sources contributions were not calculated, Rosetta and Borys (1996) surmised that in some areas and for some contaminants, rural areas may contribute a large portion of the nonpoint source discharge. This is particularly true for pesticide contamination in the upper river basin where agriculture is the predominant land use. Water quality has been reduced by phosphorus loads and decreased water clarity, primarily along the lower and middle sections of the Columbia River Estuary. Although sediment quality is generally very good, benthic indices have not been established within the estuary. Fish tissue contaminant loads (PCBs, DDT, DDD, DDE, and mercury) are high and present a persistent and long lasting effect on estuary biology. Health advisories have been recently issued for people eating fish in the area that contain high levels of dioxins, PCBs, and pesticides. Morace (2012) reported waste water treatment plant samples containing anthropogenic organic compounds, pharmaceuticals, polybrominated diphenyl ether (PBDEs [brominated flame-retardants]), organochlorine or legacy compounds, currently used pesticides, mercury, and estrogenicity.

Habitat Modification. The mainstem habitats of the lower Columbia and Willamette rivers have been reduced primarily to a single channel. As a result, floodplain area is reduced, off-channel habitat features have been eliminated or disconnected from the main channel, and the amount of large woody debris in the mainstem has been reduced. Remaining areas are affected by flow fluctuations associated with reservoir management for power generation, flood control, and irrigation. Overbank flow events, important to habitat diversity, have become rare as a result of controlling peak flows and associated revetments. Portions of the basin are also subject to impacts from cattle grazing and irrigation withdrawals. Consequently, estuary dynamics have changed substantially.

Habitat loss has fragmented habitat and human density increase has created additional loads of pollutants and contaminants within the Columbia River estuary (Anderson, Dugger, and Burke 2007). About 77 percent of swamps, 57 percent of marshes, and over 20 percent of tree cover have been lost to development and industry. The Willamette Basin Valley has been dramatically changed by modern settlement. The complexity of the mainstem river and extent of riparian forest have both been reduced by 80 percent (PNERC 2002). About 75 percent of what was formerly prairie and 60 percent of what was wetland have been converted to agricultural purposes. These actions, combined with urban development, bank stabilization, and in-river and nearshore gravel mining, have resulted in a loss of floodplain connectivity and off-channel habitat (PNERC 2002).

Hydromodification Projects. More than 400 dams exist in the basin, ranging from mega dams that store large amounts of water to small diversion dams for irrigation. Every major tributary of the Columbia River except the Salmon River is totally or partially regulated by dams and diversions. More than 150 dams are major hydroelectric projects, with 18 dams located on mainstem Columbia River and its major tributary, the Snake River. The Federal Columbia River Power System encompasses the operations of 14 major dams and reservoirs on the Columbia and Snake Rivers. These Federal projects are a major source of power in the region, and provide flood control, navigation, recreation, fish and wildlife, municipal and industrial water supply, and irrigation benefits.

Development of the Pacific Northwest regional hydroelectric power system, dating to the early 20th century, has had profound effects on the ecosystems of the Columbia River Basin (ISG 1996). These effects have been especially adverse to the survival of anadromous salmonids. The construction of the Federal power system modified migratory habitat of adult and juvenile salmonids, and in many cases presented a complete barrier to habitat access. Both upstream and downstream migrating fish are impeded by the dams, and a substantial number of juvenile salmonids are killed and injured during downstream migrations. Physical injuries and deaths occur as juveniles pass through turbines, bypasses, and spillways. Indirect effects of passage through all routes may include disorientation, stress, delays in passage, exposure to high concentrations of dissolved gases, warm water, and increased predation. Dams have also flooded historical spawning and rearing habitat with the creation of massive water storage reservoirs. More than 55 percent of the Columbia River Basin that was accessible to salmon and steelhead before 1939 has been blocked by large dams (NWPPC 1986).

The mainstem habitats of the lower Columbia and Willamette Rivers have been reduced primarily to a single channel. As a result, floodplain area has been reduced, off-channel habitat features have been eliminated or disconnected from the main channel, and the amount of large woody debris in the mainstem has been reduced. Remaining areas are affected by flow fluctuations associated with reservoir management for power generation, flood control and irrigation. Overbank flow events, important to habitat diversity, have become rare as a result of controlling peak flows and associated revetments. Consequently, estuary dynamics have changed substantially.

Artificial Propagation. There are several artificial propagation programs for salmon production within the Columbia River basin, many of which were instituted under Federal law to

ameliorate the effects of lost natural salmon production within the basin from the dams. The hatcheries are operated by Federal, state, and tribal managers. For more than 100 years, hatcheries in the Pacific Northwest have been used to produce fish for harvest and replace natural production lost to dam construction, and have only minimally been used to protect and rebuild naturally produced salmonid population (*e.g.*, Redfish Lake sockeye salmon). In 1987, 95 percent of the coho salmon, 70 percent of the spring Chinook salmon, 80 percent of the summer Chinook salmon, 50 percent of the fall Chinook salmon, and 70 percent of the steelhead returning to the Columbia River Basin originated in hatcheries (CBFWA 1990). More recent estimates suggest that almost half of the total number of smolts produced in the basin come from hatcheries (Mann *et al.* 2005).

The impact of artificial propagation on the total production of Pacific salmon and steelhead has been extensive (Hard *et al.* 1992). Hatchery practices, among other factors, are a contributing factor to the 90 percent reduction in natural coho salmon runs in the lower Columbia River over the past 30 years (Flagg *et al.* 1995). Past hatchery and stocking practices have resulted in the transplantation of salmon and steelhead from nonnative basins, and the impacts of these practices are largely unknown. Adverse effects of these practices likely included loss of genetic variability within and among populations (Busack 1990 as cited in Hard *et al.* 1992, Riggs 1990, Reisenbichler 1997), disease transfer, increased competition for food, habitat, or mates, increased predation, altered migration, and displacement of natural fish (Steward and Bjornn 1990, Fresh 1997). Species with extended freshwater residence are likely to face higher risk of domestication, predation, or altered migration than are species that spend only a brief time in fresh water (Hard *et al.* 1992). Nonetheless, artificial propagation also may contribute to the conservation of listed salmon and steelhead although it is unclear whether or how much artificial propagation during the recovery process will compromise the distinctiveness of natural population (Hard *et al.* 1992).

Currently, NMFS is working on a hatchery reform project in the Columbia River Basin, which will include a collaborative review of how harvest and hatcheries (particularly Federally funded hatcheries) are affecting the recovery of listed salmon and steelhead in the basin. This effort was mandated by Congress in 2005, and is in its early stages. Eventually, the project team would create a management approach that allows tribal, state and Federal managers to effectively manage Columbia River Basin hatcheries to meet conservation and harvest goals consistent with their respective legal responsibilities.

Mining. Most of the mining in the basin is focused on minerals such as phosphate, limestone, dolomite, perlite, or metals such as gold, silver, copper, iron, and zinc. Many of the streams and river reaches in the basin are impaired from mining, and several abandoned, and former mining sites are designated as Superfund cleanup areas (Stanford *et al.* 2005, EPA 2007). According to the United States Bureau of Mines, there are about 14,000 inactive or abandoned mines within the Columbia River Basin of which nearly 200 pose a potential hazard to the environment (Quigley *et al.* 1997 as cited in Hinck *et al.* 2004). Contaminants detected in the water include lead and other trace metals. Mining of copper, cadmium, lead, manganese, and zinc in the upper Clark Fork River have contributed wastes to this basin since 1880 (Woodward *et al.* 1994). Benthic macroinvertebrates and fish within the basin have bioaccumulated metals,

which are suspected of reducing their survival and growth (Farag *et al.* 1994, Woodward *et al.* 1994).

Commercial, Recreational, and Subsistence Fishing. During the mid-1800s, an estimated 10 to 16 million adult salmon and steelhead of all species entered the Columbia River each year. Large harvests of returning adult salmon during the late 1800s (20 to 40 million pounds of annually) significantly reduced population productivity (Mann *et al.* 2005). The largest known harvest of Chinook salmon occurred in 1883 when Columbia River canneries processed 43 million pounds of salmon (Lichatowich 1999). Commercial landings declined steadily from the 1920s to a low in 1993, when just over 1 million pounds were harvested (Mann *et al.* 2005).

Harvested and spawning adults reached 2.8 million in the early 2000s, of which almost half are hatchery produced (Mann *et al.* 2005). Most of the fish caught in the river are steelhead and spring/summer Chinook salmon, while ocean harvest consists largely of coho and fall Chinook salmon. Most ocean catches are made north of Cape Falcon, Oregon. Over the past five years, the number of spring and fall salmon commercially harvested in tribal fisheries has averaged between 25,000 and 110,000 fish (Mann 2004 in Mann *et al.* 2005). Recreational catch in both ocean and in-river fisheries varies from 140,000 to 150,000 individuals (Mann *et al.* 2005).

Interior Columbia River major subbasins: Deschutes, John Day, Umatilla, Walla Walla, Grande Ronde, and Imnaha Rivers. Habitat quality in tributary streams in the interior Columbia River subbasins varies from excellent in wilderness and roadless areas to poor in areas subject to heavy agricultural and urban development (Wissmar *et al.* 1994, Carmichael 2006).

Migratory habitat quality in this area has been severely affected by the development and operation of the FCRPS dams and reservoirs in the mainstem Columbia River, Bureau of Reclamation tributary projects, and privately owned dams in the Snake River. For example, construction of Hells Canyon Dam eliminated access to several likely production areas in Oregon and Idaho including the Burnt, Powder, Weiser, Payette, Malheur, Owyhee, and Boise river basins (Good *et al.* 2005). Hydroelectric development modified natural flow regimes, resulting in higher water temperatures, changes in fish community structure leading to increased rates of piscivorous and avian predation on juvenile salmon and steelhead, and delayed migration for both adult and juveniles. Physical features of dams such as turbines also kill migrating fish. In-river survival is inversely related to the number of hydropower projects encountered by emigrating juveniles.

Similarly, development and operation of extensive irrigation systems and dams for water withdrawal and storage in tributaries have drastically altered hydrological cycles. A series of large regulating dams on the middle and upper Deschutes River affect flow and block access to upstream habitat, and have extirpated one or more populations from the Cascades Eastern Slope major population (IC-TRT 2003). Similarly, operation and maintenance of large water reclamation systems such as the Umatilla Basin and Yakima Projects have significantly reduced flows and degraded water quality and physical habitat in this domain.

Many stream reaches are over-allocated under state water law, with more allocated water rights than existing streamflow conditions can support. Irrigated agriculture is common throughout this

region and withdrawal of water increases summer stream temperatures, blocks fish migration, strands fish, and alters sediment transport (Spence *et al.* 1996). Reduced tributary stream flow has been identified as a major limiting factor for all listed salmon and steelhead species in this area except SR fall-run Chinook salmon (NMFS 2005).

North and Middle Oregon Coast. The historical disturbance regime in the central Oregon Coast Range was dominated by a mixture of high and low-severity fires, with a natural rotation of approximately 271 years. Old-growth forest coverage in the Oregon Coast Range varied from 25 to 75% during the past 3,000 years, with a mean of 47%, and never fell below 5% (Wimberly *et al.* 2000). Currently the Coast Range has approximately 5% old-growth, almost all of it on Federal lands. The dominant disturbance now is logging on a cycle of approximately 30 to 100 years, with fires suppressed.

The State of Oregon (2005) completed an assessment of habitat conditions in the range of OC coho in 2005. Oregon's assessment mapped how streams with high intrinsic potential for coho salmon rearing are distributed by land ownership categories. Agricultural lands and private industrial forests have by far the highest percentage of land ownership in high intrinsic potential areas and along all coho stream miles. Federal lands have only about 20% of coho stream miles and 10% of high intrinsic potential stream reaches. Because of this distribution, activities in lowland agricultural areas are particularly important to the conservation of Oregon coastal coho.

The coho assessment concluded that at the scale of the entire domain, pools are generally abundant, although slow-water and off-channel habitat (which are important refugia for coho during high winter flows) are limited in the majority of streams when compared to reference streams in minimally-disturbed areas. Amounts of large wood in streams are low in all four ODFW monitoring areas and land-use types relative to reference conditions. Amounts of fine sediment are high in three of the four monitoring areas, and were comparable to reference conditions only on public lands. Approximately 62 to 91% of tidal wetland acres (depending on estimation procedures) have been lost for functionally and potentially independent populations of coho.

As part of the coastal coho assessment, the Oregon Department of Environmental Quality (ODEQ) analyzed the status and trends of water quality in the range of OC coho using the Oregon water quality index, which is based on a combination of temperature, dissolved oxygen, biological oxygen demand, pH, total solids, nitrogen, total phosphates, and bacteria. Using the index at the species scale, 42% of monitored sites had excellent to good water quality, and 29% show poor to very poor water quality. Within the four monitoring areas, the North Coast had the best overall conditions (three sites in excellent or good condition out of nine sites), and the Mid-South coast had the poorest conditions (no excellent condition sites, and only two out of eight sites in good condition). For the 10-year period monitored between 1992 and 2002, no sites showed a declining trend in water quality. The area with the most improving trends was the North Coast, where 66% of the sites (six out of nine) had a significant improvement in index scores. The Umpqua River basin, with one out of nine sites (11%) showing an improving trend, had the lowest number of improving sites.

Southern Oregon. Many large and small rivers supporting significant populations of coho salmon flow through this area, including the Elk, Rogue, Chetco, Smith and Klamath. The following summary of critical habitat information in the Elk, Rogue, and Chetco rivers is also applicable to habitat characteristics and limiting factors in other basins in this area. The Elk River flows through Curry County, and drains approximately 92 square miles (or 58,678 acres) (Maguire 2001). Historical logging, mining, and road building have degraded stream and riparian habitats in the Elk River basin. Limiting factors identified for salmon and steelhead production in this basin include sparse riparian cover, especially in the lower reaches, excessive fine sediment, high water temperatures, and noxious weed invasions (Maguire 2001).

The Rogue River drains approximately 5,160 square miles within Curry, Jackson and Josephine counties in southwest Oregon. The mainstem is about 200 miles long and traverses the coastal mountain range into the Cascades. The Rogue River estuary has been modified from its historical condition. Jetties were built by the Corps in 1960, which stabilized and deepened the mouth of the river. A dike that extends from the south shore near Highway 101 to the south jetty was completed in 1973. This dike created a backwater for the large shallow area that existed here, which has been developed into a boat basin and marina, eliminating most of the tidal marsh.

The quantity of estuary habitat is naturally limited in the Rogue River. The Rogue River has a drainage area of 5,160 square miles, but the estuary at 1,880 acres is one of the smallest in Oregon. Between 1960 and 1972, approximately 13 acres of intertidal and 14 acres of subtidal land were filled in to build the boat basin dike, the marina, north shore riprap and the other north shore developments (Hicks 2005). Jetties constructed in 1960 to stabilize the mouth of the river and prevent shoaling have altered the Rogue River, which historically formed a sill during summer months (Hicks 2005).

The Lower Rogue Watershed Council's watershed analysis (Hicks 2005) lists factors limiting fish production in tributaries to Lower Rogue River watershed. The list includes water temperatures, low stream flows, riparian forest conditions, fish passage and over-wintering habitat. Limiting factors identified for the Upper Rogue River basin include fish passage barriers, high water temperatures, insufficient water quantity, lack of large wood, low habitat complexity, and excessive fine sediment (Rogue Basin Coordinating Council 2006).

The Chetco River estuary has been significantly modified from its historical condition. Jetties were constructed by the Corps in 1957, which stabilized and deepened the mouth of the river. These jetties have greatly altered the mouth of the Chetco River and how the estuary functions as habitat for salmon migrating to the ocean. A boat basin and marina were built in the late 1950s and eliminated most of the functional tidal marsh. The structures eliminated shallow water habitats and vegetation in favor of banks stabilized with riprap. Since then, nearly all remaining bank habitat in the estuary has been stabilized with riprap. The factors limiting fish production in the Chetco River appear to be high water temperature caused by lack of shade, especially in tributaries, high rates of sedimentation due to roads, poor over-wintering habitat due to a lack of large wood in tributaries and the mainstem, and poor quality estuary habitat (Maguire 2001).

Summary of Environmental Baseline for Anadromous Fishes. Pacific salmon and steelhead, green sturgeon and eulachon are exposed to the impacts of a wide variety of past and

present state, Federal or private actions and other human activities that comprise the action area, as well as Federal projects in this area that have already undergone formal section 7 consultation, and state or private actions that are contemporaneous with this consultation. Here we provide a review of major ESA section 7(a)(2) consultations where NMFS predicted effects would occur within in the action area.

The NMFS consulted on the effects of EPA's registration of pestidice products for chlorpyrifos, diazinon, and malathion (NMFS 2008); carbaryl, carbofuran, and methomyl (NMFS 2009); azinphos methyl, bensulide, dimethoate, disulfoton, ethoprop, fenamiphos, naled, methamidophos, methidathion, methyl parathion, phorate and phosmet (NMFS 2010); and 2,4-D, triclopyr BEE, diuron, linuron, captan, and chlorothalonil (NMFS 2011). These consultations concluded that registration of these pesticide products would jeopardize the continued existence of Pacific salmon and steelhead and/or result in the destruction or adverse modification of their critical habitats.

The NMFS consulted on the effects of fishery harvest actions, including 10-year terms of the Pacific Salmon Treaty (term of biological opinion from 2009-2018, NMFS 2008e) and the *United States v. Oregon* 2008 Management Agreement (term of biological opinion from 2008-2017; NMFS 2008f), and the Pacific Coast Salmon Plan fisheries (NMFS 2009a). In these past harvest opinions, NMFS characterized the short-term and long-term effects on reductions in Chinook abundance that occur during a specified year, and the long-term effects to whales that could result if harvest affected viability of the salmon stock over time by decreasing the number of fish that escape to spawn. The harvest biological opinions referenced above concluded that the harvest actions were not likely to jeopardize the continued existence of listed Chinook salmon.

The NMFS conducted additional consultations on the effects of hydro-power dams and flood control programs on all Columbia River basin salmon and steelhead, green sturgeon, and eulachon (NMFS 2008g, NMFS 2008h). As part of the proposed action for the Federal Columbia River Power System and the Willamette Flood Control Program, action agencies proposed funding hatchery programs in addition to their proposals for dam operations and maintenance. To mitigate for the harmful effects of hatchery production on long-term salmon and steelhead viability the action agencies committed to a schedule of future hatchery reforms.

2.5.2.4 Southern Resident Killer Whales

Prey Availability. Based on persuasive scientific information that the diet of Southern Residents is predominantly composed of Chinook salmon in inland waters (see further discussion in section 2.4.4), their diet may equally be predominantly composed of Chinook salmon when available in coastal waters of the action area. This analysis focuses on Chinook salmon abundance in coastal waters of the Southern Residents range. Focusing on Chinook salmon provides a conservative estimate of potential effects of the proposed action on Southern Residents because the total abundance of all salmon and other potential prey species is orders of magnitude larger than the total abundance of Chinook salmon.

When prey is scarce, whales likely spend more time foraging than when it is plentiful. Increased energy expenditure and prey limitation can cause nutritional stress. Nutritional stress is the

condition of being unable to acquire adequate energy and nutrients from prey resources and as a chronic condition can lead to reduced body size and condition of individuals and lower birth and survival rates of a population. Ford *et al.* reported correlated declines in both the Southern Resident killer whales and Chinook salmon and suggested the potential for nutritional stress in the whales (Ford *et al.* 2005, Ford *et al.* 2010b). Food scarcity could also cause whales to draw on fat stores, mobilizing contaminants stored in their fat and potentially have the ability to alter thyroid homeostasis, reduce immune function, cause neurotoxicity, reproductive failure, and restrict the development and growth of the individual (see Table 9 in NMFS 2008a for a review of physiological effects resulting from exposure to toxic chemicals in marine mammals). Thus, nutritional stress may act synergistically with high contaminant burdens in the whales and result in contaminant-induced adverse health effects, higher mortality rates, or lower birth rates.

The availability of Chinook salmon to Southern Residents is affected by a number of natural and human actions. Climate effects from Pacific decadal oscillation and the El Nino/Southern oscillation conditions and events cause changes in ocean productivity which can affect natural mortality of salmon. Predation in the ocean also contributes to natural mortality of salmon. Salmonids are prey for pelagic fishes, birds, and marine mammals (including Southern Residents). Section 2.5 describes the baseline concentrations and sources (both natural and through human activities) of metal and elemental pollutants in Oregon waters and the potential adverse health effects to fish. Additional human activities and their impacts to salmon include land use such as logging, agriculture, ranching, hydroelectric power generation, mining, fishing, recreational activities, and urban uses (see section 2.5.2.5 above). Many of these activities have a federal nexus and have undergone section 7 consultation. Those actions have all met the standard of not jeopardizing the continued existence of the listed salmonids or adversely modifying their critical habitat, or if they did not meet that standard, we identified reasonable and prudent alternatives. Since the Southern Residents were listed, federal agencies have also consulted on impacts to the whales, including impacts to available prey. In addition, the environmental baseline is influenced by many actions that pre-date the salmonid listings and that have substantially degraded salmon habitat and lowered natural production of Chinook ESUs contemplated in this consultation.

Here we provide a review of Southern Resident killer whale determinations in previous ESA Section 7(a)(2) consultations where effects occurred in the action area, and where effects resulted in a significant reduction in available prey (*i.e.*, where prey reduction was likely to adversely affect or jeopardize the continued existence of the whales).

The NMFS consulted on the effects of fishery harvest actions on Southern Residents, including 10-year terms of the Pacific Salmon Treaty (term of biological opinion from 2009-2018, NMFS 2008e) and the *United States v. Oregon* 2008 Management Agreement (term of biological opinion from 2008-2017; NMFS 2008f), and the Pacific Coast Salmon Plan fisheries (NMFS 2009a). In these past harvest opinions, NMFS characterized the short-term and long-term effects on Southern Residents from prey reduction caused by harvest. We considered the short-term effects to whales resulting from reductions in Chinook abundance that occur during a specified year, and the long-term effects to whales that could result if harvest affected viability of the salmon stock over time by decreasing the number of fish that escape to spawn. These past analyses suggested that in the short term prey reductions were small relative to remaining prey

available to the whales. In the long term, harvest actions have met the conservation objectives of harvested stocks, were not likely to appreciably reduce the survival or recovery of listed Chinook, and were therefore not likely to jeopardize the continued existence of listed Chinook. The harvest biological opinions referenced above concluded that the harvest actions cause prey reductions in a given year, but were not likely to jeopardize the continued existence of ESA-listed Chinook salmon or Southern Residents. New information about the relationship between Chinook salmon abundance and Southern Resident killer whale population growth is currently under scientific review and will inform future consultations and NMFS consideration of these previous conclusions.

NMFS also consulted on the effects of the long-term operations of the Central Valley Project (CVP) and State Water Project (SWP) (2008/09022). The NMFS found that the long-term operations of the CVP and SWP, as proposed, were likely to jeopardize the continued existence of Sacramento River winter-run Chinook salmon, Central Valley spring-run Chinook salmon, Central Valley steelhead, Southern DPS of North American green sturgeon, and Southern Resident killer whales. The increased risk of extinction of the winter- and spring-run Chinook salmon as a long-term consequence of the proposed action diminished the potential for Southern Residents to survive and recover. The involved action agencies are implementing actions identified as part of the reasonable and prudent alternative over specified time periods starting from issuance of the biological opinion.

NMFS conducted additional consultations on the effects of hydro-power dams and flood control programs on Southern Residents (NMFS 2008g, NMFS 2008h). As part of the proposed action for the Federal Columbia River Power System and the Willamette Flood Control Program, action agencies proposed funding hatchery programs in addition to their proposals for dam operations and maintenance. For both programs, the proposed actions did not result in a net decrease in Chinook salmon prey for Southern Residents in the short term. To mitigate for the harmful effects of hatchery production on long-term Chinook salmon viability (and thus killer whale prey availability) the action agencies committed to a schedule of future hatchery reforms.

Quality of Prey. As introduced in the above sections, contaminants enter marine waters from numerous sources throughout the action area, but are typically concentrated near populated areas of high human activity and industrialization. The majority of growth in salmon occurs while feeding in saltwater (Quinn 2005). Therefore, the majority (> 96 percent) of persistent pollutants in adult salmon are accumulated while feeding in the marine environment (Cullon *et al.* 2009, O'Neill and West 2009). Freshwater contamination is also a concern because it may contaminate salmon that are later consumed by the whales in marine waters. Only limited information is available for contaminant levels of Chinook in Oregon rivers; however, in general Chinook salmon contain higher levels of some contaminants than other salmon species (See Table 2.4.4.5 in the Status of the Species). As discussed in the Status of the Species, the marine distribution is an important factor affecting pollutant accumulation as is evident across the different salmon populations. For example, Chinook populations feeding in close proximity to land-based sources of contaminants have higher concentrations (O'Neill *et al.* 2006).

Vessel Activity and Sound. Commercial, military, recreational and fishing vessels traverse the coastal range of Southern Residents. Vessels may affect foraging efficiency, communication, and/or energy expenditure by their physical presence and by creating

underwater sound (Williams *et al.* 2006, Holt 2008). Collisions of killer whales with vessels are rare, but remain a potential source of serious injury and mortality. Large ships that traverse coastal waters of the whales' range move at relatively slow speeds and are likely detected and avoided by Southern Residents.

Vessel sounds in coastal waters are most likely from large ships, tankers and tugs. Sound generated by large vessels is a source of low frequency (5 to 500 Hz) human-generated sound in the world's oceans (National Research Council 2003). While larger ships generate some broadband noise in the hearing range of whales, the majority of energy is below their peak hearing sensitivity. At close range large vessels can still be a significant source of background noise at frequencies important to the whales (Holt 2008). Commercial sonar systems designed for fish finding, depth sounding, and sub-bottom profiling are widely used on recreational and commercial vessels and are often characterized by high operating frequencies, low power, narrow beam patterns, and short pulse length (National Research Council 2003). Frequencies fall between 1 and 500 kHz, which is within the hearing range of some marine mammals, including killer whales, and may have masking effects.

Non-Vessel Sound. Anthropogenic (human-generated) sound in the range of Southern Residents is generated by other sources besides vessels, including oil and gas exploration, construction activities, and military operations. Natural sounds in the marine environment include wind, waves, surf noise, precipitation, thunder, and biological noise from other marine species. The intensity and persistence of certain sounds (both natural and anthropogenic) in the vicinity of marine mammals vary by time and location and have the potential to interfere with important biological functions (*e.g.*, hearing, echolocation, communication).

In-water construction activities are permitted by the Corps under section 404 of the CWA and section 10 of the Rivers and Harbors Act of 1899 and by the State of Washington under its Hydraulic Project Approval program. Consultations on these permits have been conducted and conservation measures have been included to minimize or eliminate potential effects of in-water activities, such as pile driving, on marine mammals. Military sonar also has the potential to disturb killer whales.

Oil Spills. Oil spills have occurred in the coastal range of Southern Residents in the past, and there is potential for spills in the future. Oil can be discharged into the marine environment in any number of ways, including shipping accidents, at refineries and associated production facilities, and pipelines. The magnitude of risk posed by oil discharges in the action area is difficult to precisely quantify, but improvements in oil spill prevention procedures since the 1980s likely provide some reduced risk of spill. New oil spill prevention procedures in the state of Washington likely positively contribute to the decrease in spill volume (WDOE 2007).

In marine mammals, acute exposure to petroleum products can cause changes in behavior and reduced activity, inflammation of the mucous membranes, lung congestion, pneumonia, liver disorders, neurological damage (Geraci and St. Aubin 1990), potentially death, and long-term effects on population viability (Matkin *et al.* 2008). In addition, oil spills have the potential to adversely impact habitat and prey populations, and, therefore, may adversely affect Southern Residents by reducing food availability.

Scientific Research. Although research activities are typically conducted between May and October in inland waters, some permits include authorization to conduct research in coastal waters. In general, the primary objective of this research is population monitoring or data gathering for behavioral and ecological studies. In 2006, NMFS issued scientific research permits to seven investigators who intend to study Southern Residents (NMFS 2006). Additionally in 2008, NMFS issued another scientific permit to one investigator intending to study Southern Residents (NMFS 2008i). In the biological opinions NMFS prepared to assess the impact of issuing the permits, we determined that the effects of these disturbances on Southern Residents were likely to adversely affect, but not likely to jeopardize the continued existence of, the Southern Residents (NMFS 2006, 2008i). A small portion of the authorized take would occur in the coastal range of Southern Residents.

Summary of Southern Residents Environmental Baseline. Southern Residents are exposed to a wide variety of past and present state, Federal or private actions and other human activities in the coastal waters that comprise the action area, as well as Federal projects in this area that have already undergone formal section 7 consultation, and state or private actions that are contemporaneous with this consultation. All of the activities discussed in the above section are likely to have some level of impact on Southern Residents when they are in the action area.

No single threat has been directly linked to or identified as the cause of the recent decline of the Southern Residents, although the three primary threats are identified as prey availability, environmental contaminants, and vessel effects and sound (Krahn *et al.* 2002). Researchers are unsure about which threats are most significant. There is limited information on how these factors or additional unknown factors may be affecting Southern Residents when in coastal waters. For reasons discussed earlier, it is possible that two or more of these factors may act together to harm the whales. The small size of the population increases the level of concern about all of these risks (NMFS 2008a).

2.6 Effects of the Action

'Effects of the action' means the direct and indirect effects of an action on the species or critical habitat, together with the effects of other activities that are interrelated or interdependent with that action, that will be added to the environmental baseline (50 CFR 402.02).

EPA's approval of Oregon's revised water quality standards would have no direct effects to listed species or their habitat—that is, approving new water quality standards, by itself, will not directly affect listed species or designated critical habitat, or change the environmental baseline. However, there are significant indirect effects of approving the standards, because the approval allows the state to implement the standards. The analysis of effects of the proposed action assumes that the species of interest are exposed to waters meeting the water quality standards; however, there are many waters in Oregon that do not meet the current standards and would not meet the proposed standards. Implementation and attainment of the standards are key to improving the state's water quality, however, the only action under consideration in this consultation is EPA's proposed approval of Oregon's revised standards.

2.6.1 Issues Common to All Criteria

The following discussion on acute and chronic toxicity data focuses on issues applicable to the development of all aquatic life criteria, and provides context for the toxicity data analyses on individual compounds provided in this section of the opinion.

Acute Toxicity Data. The acute criteria for aquatic life have been primarily based on compilations of toxicity study results reported in terms of the concentration resulting in 50 percent mortality over a fixed time period [usually 96 hours: *e.g.*, LC_{50}, effects concentration $(EC)_{50,}$ EPA 1986a] using EPA's *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (Stephan *et al.* 1985) (Guidelines). Although there are a number of reasons why data are not included in the data sets used to develop criteria, some of the more common ones are that one or more pieces of information regarding study methodology or calculation of results needed to assess the reliability of the study is missing; data quality of the study is less than acceptable (*e.g.* unacceptably high control mortality); the test species was exposed to a chemical mixture or was previously exposed to the test chemical; the study reported effects on an endpoint other than survival, reproduction or growth; or the test duration was a non-standard test duration (*e.g.*, fish toxicity test reporting a 24-hr LC_{50} instead of the more standard 96-hr LC_{50}).

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, that indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias the magnitude of acute toxic effects. Theses factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that are protective against acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve, and challenge the notion that LC_{50} data that is above the acute criterion is protective against acute toxic effects based soley on a comparison of concentrations.

Acute water quality criteria are calculated by rank ordering the genus mean acute value (GMAV) values from the lowest LC_{50} to the highest LC_{50}, and using a formula given in Stephan *et al.* (1985) to estimate the 5^{th} percentile of the resulting species sentitive distribution (SSD). This 5^{th} percentile of measured GMAVs is termed the (final acute value) FAV in the EPA criteria development documents. As a criterion based on a concentration causing mortality to 50 percent of a test species would not be a protective criterion, EPA divides the FAV by a safety factor of 2.27 (rounded to a factor of 2 in the below analysis) to convert LC_{50} values into concentrations that EPA projects to be near or below lethality.

The database from which the safety factor was derived was published in the Federal Register in 1978. Table 10 from the Federal Register notice (43 FR 21506-21518) lumps data for freshwater and marine fish and invertebrates. The data are broken out by the chemicals tested. There are 219

data points, but a large proportion of them aren't for a specific chemical, but rather for whole effluents of various sources—115 of the 219 data points used to derive the acute adjustment factor are based on effluent studies where individual pollutants are not measured. Interestingly, effluent studies are one of EPA's "not pertinent" or "reject" categories identified in EPA (2005).

The assumption that dividing an LC_{50} by 2 will result in effect concentrations near or below leathility rests on further assumptions of the steepness of the concentration-response slope. Several examples of tests with metals which had a range of response slopes are shown in Figure 2.6.1.1. These examples were selected from data sets that were relevant to salmonid species in Oregon and for which the necessary data to evaluate the range of responses could be located (Chapman 1975, 1978b, Marr *et al.* 1995, Marr *et al.* 1999, Mebane *et al.* 2010, Windward 2002). The citations given include both reports with detailed original data as well as the summarized, published forms of the same tests. The examples range from tests with some of the shallowest concentration-response slopes located to very steep response slopes. In the shallowest tests (panels A and E), an $LC_{50/2}$ concentration would still result in 15 to 20 percent mortality.

One challenge for deriving acute criteria for short-term exposures is that the great majority of available data is for mortality; that is, a concentration that kills 50 percent of a test population. A fundamental assumption of EPA's criteria derivation is that the FAV, which is the LC_{50} for a hypothetical species with a sensitivity equal to the 5^{th} percentile of the SSD, may be divided by 2 in order to extrapolates from a concentration that would likely be extremely harmful to sensitive species in short-term exposures (*i.e.*, kill 50 percent of the population) to a concentration expected to kill few, if any, individuals. This assumption must be met for acute criteria to be protective of sensitive species. It is difficult to evaluate from published literature if this assumption is met because so few studies report the data behind an LC_{50} test statistic. While LC_{50}s are almost universally used in reporting short-term toxicity testing, they are not something that can be "measured," but are statistical model fits. An acute toxicity test is actually a series of 4 to 6 tests runs in parallel in order to test effects at these (usually) four to six different chemical concentrations. An LC_{50} is estimated by some statistical distribution or regression model, which generates an LC_{50} estimate, and some confidence interval, and then all other information is thrown away. Thus, while the original test data included valuable information on what were no, low and severe effects concentrations, that information is lost to reviewers unless the unpublished, raw, lab data are available. However, a more common pattern with the metals data was that an $LC_{50/2}$ concentration would probably result in about a 5 percent death rate (panels B and F), and in many instances, no deaths at all would be expected (panels C and D).

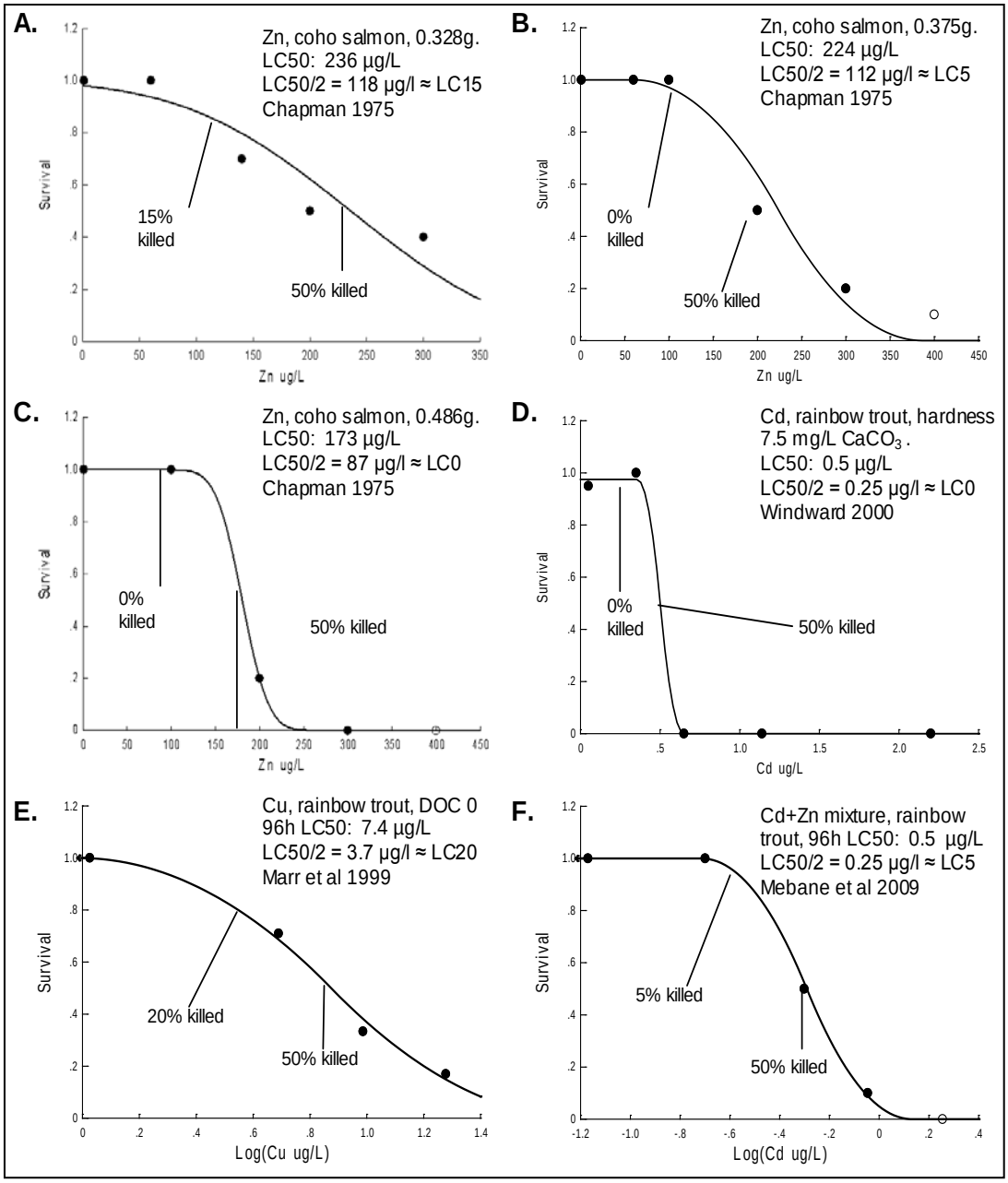

Figure 2.6.1.1 Examples of percentages of coho salmon or rainbow trout killed at one-half their LC_{50} concentrations and at LC_{50} concentrations with cadmium, copper, and zinc.

In one of the few additional published sources that gave relevant information, researchers happened to include effect-by-concentration information on the acute toxicity of chemical mixtures. Rainbow trout and the invertebrate zooplankton *Ceriodaphnia dubia* were exposed for 96 and 48 hours respectively to mixture of six metals, each at their presumptively "safe" acute CMC concentrations. In combination, the CMC concentrations killed 100% of rainbow trout and C. dubia, but 50% of the CMC concentrations killed none (Spehar and Fiandt 1986). This gives some support to the assumption that one-half the FAV divided by 2 is likely to kill a low

percentage of fish, although it raises questions about the overall protectiveness of criteria concentrations in mixtures.

Other relevant reviews include Dwyer *et al.* (2005b), who evaluated the $LC_{50/2}$ assumption with the results of the acute toxicity testing of 20 species with five chemicals representing a broad range of toxic modes of action. In those data, multiplying the LC_{50} by a factor of 0.56 resulted in a low (10%) or no-acute effect concentration. Testing with cutthroat trout and Cd, Pb, and Zn singly and in mixtures, Dillon and Mebane (2002) found that the $LC_{50/2}$ concentration corresponded with death rates of 0 to 15 percent.

Summary: Based on this analysis, acute criteria based on LC_{50} concentrations and the acute adjustment factor, instead of acute criteria that are based on an exposure-response curve, are likely to underestimate the magnitude of effects for field-exposed fishes. Therefore, the shortcomings identified in the above analysis are likely to result in mortality greater than the LC_{50} test predictions and the presumed protection from the acute adjustment factor in deriving acute criteria.

Chronic Toxicity Data. While the Guidelines give a great deal of advice on considerations for evaluating chronic or sublethal data (Stephan *et al.* 1985, at p. 39), those considerations were not usually reflected in the individual national EPA-recommended ambient water quality criteria documents NMFS reviewed. In practice, for most of the criteria documents we reviewed, "chronic values" were simply calculated as the geometric mean of the lowest tested concentration that had a statistically significant adverse effect at the 95 percent confidence level (LOEC), and the next lower tested concentration (NOEC). The "chronic value" as used in individual criteria documents is effectively the same thing as the maximum acceptable toxicant concentration[6] (MATC) used in much environmental toxicology literature, even though the MATC term is never used in the Guidelines. This MATC approach has the potential to seriously underestimate effects because the statistical power in typical toxicity tests is fairly low. A bias in many ecotoxicology papers is to focus on avoiding "false accusations" of a chemical with 95 percent accuracy (*i.e.*, Type I error or false positive, the risk of declaring an effect was present when in fact there was no effect). Often no consideration whatsoever is given to the companion problem, known as Type II error, or false negatives (*i.e.*, declaring no adverse effects occurred when in fact they did occur, but because of the limited sample size or variability, they were not significant with 95 percent confidence).

The magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be large (greater than 30 percent on average for some endpoints), and much higher for individual tests (Crane and Newman 2000). This problem is compounded when the "chronic value" or MATC is calculated in its most common form as the geometric mean of a NOEC and LOEC. For instance, in one study, 100 percent of juvenile brook died after being exposed to 17 µg/L copper for 8 months; this was considered the LOEC for the test. The next lowest concentration tested (9.5 µg/L) had no reduced survival relative to controls. (McKim and Benoit 1971). Therefore, the only thing that can be said about the geometric mean of these two effect concentrations (*i.e.*, the chronic value of 12.8 µg/L that was used in the chronic copper criteria, EPA 1985) is that it represents a concentration that can be expected to kill somewhere between

[6] The MATC is the range between the NOEC and LOEC.

all and no brook trout in the test population. These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that are protective against chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

Suter *et al.* (1987) evaluated published chronic tests with fish for a variety of chemicals and found that, on average, the MATC represented about a 20 percent death rate and a 40% reduction in fecundity. They noted that "although the MATC is often considered to be the threshold for effects on fish populations, it does not constitute a threshold or even a negligible level of effect in most of the published chronic tests. It corresponds to a highly variable level of effect that can only be said to fall between 0 and 90 percent." Barnthouse *et al.* (1989) further extrapolated MATC-level effects to population-level effects using fisheries sustainability models and found that the MATC systematically undervalued test responses such as fecundity, which are both highly sensitive and highly variable.

One implication of this issue is that because the MATC chronic values typically used in the EPA water quality criteria documents for aquatic life criteria may cause a substantial adverse effect for that test species, the criteria on the whole will be less protective than the Guidelines' intended goal of protecting 95 percent of the species. How much less protective is unclear and probably varies among the criteria datasets. One dataset from which a hypothetical NOEC-based chronic criterion could readily be recalculated and compared with the usual MATC criteria was a 2006 cadmium criteria update (Mebane 2006). In this comparison, Mebane determined that the MATC-based chronic criteria would protect about 92 percent of the aquatic species in the dataset at the NOEC level. Because the NOEC statistic also can reflect a fairly sizable effect (Crane and Newman 2000) it may be that at least with cadmium, the true level of protection is closer to about 90 percent than the 95 percent intended by the guidelines.

Summary: Based on this analysis, chronic criteria based on hypothesis tests, instead of acute criteria that are based on an exposure-response curve, are likely to underestimate the magnitude of effects for field-exposed fishes. Therefore, the shortcomings identified in the above analysis are likely to result in sublethal greater than the NOEC/LOEC predictions.

2.6.2 Freshwater Criteria Toxicity Analysis

The ESA directs that section 7 consultations use the best available scientific and commercial data. While EPA conducted an extensive data call and has developed a large database of toxicity (ECOTOX), thousands of toxicity studies were rejected by EPA for use in criteria development and formulation of the BE. A majority of these toxicity studies were rejected because the test duration was non-standard; EPA generally does not consider toxicity tests with non-standard durations (*e.g.*, 4-hr LC_{50} or 192-hr LC_{50}), or endpoint, *e.g.*, behavioral. However, these studies may still meet the standard of the "best available scientific data" as defined by the ESA and, as warranted, were intergrated into the analysis in this opinion.

NMFS also examined EPA's BE effects assessment methodology, but NMFS did not use the EPA effects assessment methodology or the analysis in the BE for its effects analysis as it included too many fundamental problems NMFS identified during preconsultation that EPA did not address in the BE submitted to NMFS. These problems include:

- LC_{50} toxicity data interpretation and application
- NOEC toxicity data interpretation and application
- Exclusion of published toxicity data in the BE analysis
- High uncertainty with use of the acute adjustment factor
- Lack of a sublethal effects analysis
- Lack of a chemical mixture analysis
- Scale of effect determinations—effects of the action as a whole verses effects based on individual criterion

Instead, NMFS used a much more extensive toxicity data set, including toxicity studies from the ECOTOX database that were excluded by EPA, for its analysis, and included an extensive sublethal effects analysis for each compound (where data was available), a chemical mixtures analysis, a direct mortality and population model for the freshwater acute criteria, and a synthesis of effects of the action as a whole.

In this opinion, NMFS also examined EPA's *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (Stephan *et al.* 1985), as it forms the basis for how EPA derives aquatic life criteria. That analysis is provided in Apprendix 1 of this opinion.

The analysis on freshwater criteria starts with a review of the chemical and toxicological concepts, principals, and factors that influence toxicity for each compound, and an assessment of critical exposure-response factors pertinent to the overall analysis. The data analysis in this section has five general components: (1) Available toxicity data presented in table format by endpoint; (2) a summary statistical analysis performed for each endpoint data set consisting of the arithmetic mean, the geometric mean, and the harmonic mean to assess the distribution of the data for each data set, and the statistical analysis is used later in the analysis on chemical mixtures; (3) a relative mortality analysis for the acute criteria; (4) a sublethal effects analysis on the chronic criteria, and (5) an analysis on food items (when data was available).

The toxicity data for salmonid fishes includes data for listed and non-listed salmonid fishes, *e.g.*, rainbow trout are used to directly assess toxicity effects on steelhead as the resident form is indistinguishable from the anadromous form in juvenile life stages. Other salmonid fishes, *e.g.*, brook trout (*Salvelinus fontinalis*) and cutthroat trout (*Oncorhynchus clarki*), are used in addition to the species-specific toxicity data and/or as a surrogate for listed species where toxicity data is not available for listed species to analyze effects on additional endpoints. Our analysis of surrogate species toxicity data showed no difference in the range of concentrations when compared to the toxicity data for listed species. Furthermore, toxicity data for green sturgeon and eulachon was limited or non-existent for most of the compounds in Table 1.1. Therefore, NMFS used the salmonid fishes toxicity data as a surrogate for these two species, as salmonid fishes were the closest taxonomic group for which data were available.

The effects analysis on Southern Resident killer whales follows the analysis on salmon, steelhead, green sturgeon, and eulachon as the Southern Resident killer whale effects analysis is dependent upon the effects analysis and conclusions on salmon and steelhead addressed in this opinion

The summary conclusions provided in this section are based on an analysis of toxicity exposure-response potential for each listed species considered in this opinion and for each freshwater compound listed in Table 1.1. The NMFS based these analyses exclusively on an examination of the available toxicity data from exposure to a single compound. The NMFS also rated the magnitude of effects for each endpoint. The NMFS used a scale of *low intensity* increase in toxicity effects on listed species at the scale of individuals or groups of individuals, *moderate intensity* increase in toxicity effects on listed species at the scale of individuals or groups of individuals, *moderately-high-intensity* increase in toxicity effects on listed species at the scale of individuals or groups of individuals, but not at the scale of any population, and *high-intensity* increase in toxicity effects on listed species that affects one or more population attribute as a means to qualitatively assess the magnitude of acute or chronic toxics effects associated with the toxicity data. The summary conclusions do not take into account effects to the listed species considered in this opinion from exposure to multiple compounds. The issue of chemical mixtures, as well as criteria development issues, direct mortality population modeling, *etc.*, are examined in the *Integration and Synthesis*.

Toxicity Data Sources

The following is a list of data sources used in this opinion.

Data Set ECOTOX — all data are from ECOTOX and were provided to NMFS by EPA. The first data set provided to NMFS by EPA only included the rank ordered LC_{50} data and ranked ordered NOEC data. The NMFS also requested EPA provide the core data files for the compounds subject to this consultation, which were provided to NMFS. The core data files contain all toxicity data available in ECOTOX for the subject conmpounds at the time of the data requests. The EPA only used the rank ordered data for the analysis in their BE. On the other hand, NMFS used the core data files for its analysis in this opinion. Additionally, NMFS made several data requests to EPA for the reference sources listed in the core data files. The EPA only provided NMFS with the reference sources for the rank ordered data and did not provide the

reference sources for the core data files. The NMFS cross-walked the rank ordered data with the references sources for data quality assurance. For the remainder of the core data, NMFS relied on the toxicity data as provided by EPA in the core data files. Reference sources for the ECOTOX data used in this opinion are provided in Appendix 2.

ECOTOX data selection: EPA used the concentration mean values (geometric mean) for the analysis in their BE. The NMFS used either the concentration mean value (geometric mean), the concentration minimum value (lower 95^{th} percentile confidence interval), or the concentration maximum value (upper 95^{th} percentile confidence interval). The NMFS also used statistically determined toxicity data, *e.g.*, LC_{50} values, as many toxicity tests results are based on a regression analysis. When available, NMFS selected the concentration minimum value, *i.e.*, lower 95^{th} percentile confidence interval of the LC_{50}, as it is the best available statistical estimate of the actual reported LC_{50} value (in order to assess the uncertainty of the LC_{50} value as LC_{50} endpoints typically do not indicate the point at which listed fish could be killed or harmed) for a particular chemical-species combination and therefore represents the best available science in evaluating potential effects.

For the ECOTOX data set, the life stage (organism comment) information in each of the criterion-specific tables can be found in the ECOTOX code list document (EPA 2008).

Data Set 2 — all data indentified in tables with “Data Set 2” are from the NMFS’ biological opinion (draft) for the proposed approval of Idaho’s water quality criteria for toxic substances.

Data Set 3 — all data indentified in tables with “Data Set 3” are from NOAA Technical memorandums.

Data Set 4 — all data indentified in tables with “Data Set 4” are from the toxicity data for sturgeon (Section 4, Literature Cited).

Data Set BE — all data indentified in tables with “Data Set BE” are from the BE (saltwater data for cadmium, arsenic, heptachlor epoxide, nickel, pentachlorophenol, and lead).

Other data sources used in the opinion are cited directly in the text (Section 4, Literature Cited). The tables in section 2.6.2 and 2.6.3 provide information on compound concentration, life stage and exposure duration.

2.6.2.1 Organic Pollutants: Analysis of Individual Compounds

In this section, we identify the effects of each compound listed in Table 1.1 , and compare the proposed criteria with available toxicity data. The analysis identifies the potential effects on listed species and their critical habitats of each of the criteria that we would expect to occur if water concentrations were equal to the proposed criteria. Where possible, we also identify sublethal effects, effects related to bioaccumulation, and effects on the food sources of listed species.

Organic Pollutants—Toxicity and Exposure

Eisler's series of synoptic reviews (1970), EPA's criteria documents, and the World Health Organization's environmental health criteria documents (*e.g.*, WHO 1984) were used to provide the following summary of sources, pathways, and toxic effects of organic pollutants. Most of the organic compounds considered in the proposed action are organochlorine pesticides (*e.g.*, dieldrin, lindane, heptachlor), used in the past for a variety of agricultural applications, as well as for controlling insects considered hazardous to human health. The remainder are industrial chemicals (*e.g.*, PCP, TBT) that have been used widely in the past but are now banned or restricted in the United States. Of the organic contaminants included in the proposed action, only lindane, endosulfan, heptachlor, and pentachlorophenol are still used at all United States, and permitted applications for lindane and heptachlor are very limited. They generally enter the aquatic environment attached to organic and inorganic particulate matter. However, because they are not highly water soluble and persistent in the environment, they remain sequestered in sediments and provide a continual source of potential exposure. This is of particular relevance when contaminated streambed sediments are disturbed as part of in-channel work. Organic pollutants may also enter the aquatic environment through non-point surface runoff from contaminated agricultural areas where they have been used in the past. Although the levels of most of these compounds have declined since their use was banned in the 1970s, they are still widely distributed in the environment and found in tissues of aquatic organisms.

Organic contaminants are rarely found alone in discharges or in the environment. Usually, several compounds are found together in areas where there has been extensive agricultural or industrial activity. In industrialized areas, other classes of contaminants (such as metals or aromatic hydrocarbons from petroleum products). For instance, the chemical forms of most organic pesticides and PCBs are mixtures that may contain a large number of isomers and congeners of each compound, of which the toxicity and persistence in the environment can vary considerably.

The most direct exposure pathway for dissolved organic compounds to aquatic organisms is via the gills. Dissolved organic compounds are also taken up directly by bacteria, algae, plants, and planktonic and benthic invertebrates. Organic pollutants can also adsorb to particulate matter in the water column and enter organisms through various routes. Planktonic and benthic invertebrates can ingest particulate-bound organic compounds from the water column and sediments and then be eaten by other organisms. Thus, dietary exposure may be a significant source of organic toxic pollutants for aquatic and aquatic-dependent organisms.

Although organic contaminants bound to sediments are generally less bioavailable to organisms, they are nonetheless present, and changes in the environment (*e.g.*, dredging, storm events, temperature, lower water levels, biotic activity) can significantly alter their bioavailability. Feeding habits of fish can determine the amount of uptake of certain organic contaminants; for example, where piscivorous fish are exposed to different levels of organics than are omnivorous or herbivorous fish.

Organic pollutants can have a wide variety of effects on organisms. Exposure to organochlorines can result in damage to gut tissues, disrupt nervous system operation, and alter liver and kidney

functions, and impair the immune system. Elevated concentrations of many organochlorine compounds can cause growth inhibition, impaired reproduction, and developmental defects that may affect not only the target organisms themselves, but can also impact the growth and survival of predator species farther up the food chain. A number of these compounds are promoters that increase the risk of cancer. They may also disrupt immune function and increase the affected animal's susceptibility to infectious disease. Impacts from organic contamination can shift species composition and abundance towards more pollution-tolerant species. For each of the organic pollutants, we analyze these effects in subsequent sections.

2.6.2.1.1 Dieldrin

Dieldrin Criteria. The proposed acute and chronic criteria for dieldrin are 0.24 µg/L and 0.056 µg/L, respectively.

Tables 2.6.2.1.1.1 through 2.6.2.1.1.6 report toxicity data from the ECOTOX database for freshwater dieldrin, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.1.1 LC_{50} toxicity data for salmonid fishes, eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		**Data Set ECOTOX**
Criterion Concentration Acute 0.24 Micrograms Liter^{-1}	**Temperature 7.4-12° Celsius**	**Arithmetic Mean 635**
Criterion Concentration Chronic 0.056 Micrograms Liter^{-1}	**Hardness 40-272 mg/L $CaCO_3$**	**Geometric Mean 27**
Endpoint/Effect LC_{50}/Mortality	**pH 7.1-7.54**	**Harmonic Mean 5**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.56	UNDERYEARLING	96H
0.9	1.4G	96H
1	0.8G	96H
1.1		
1.4		
1.6	UNDERYEARLING	72H
1.8	0.8G	96H
2	EARLY FRY, 77 D	96H
2.3	UNDERYEARLING	24H
2.4		
4.55	1.1G	96H
4.55	1.1G	96H
5.3	JUVENILE	96H

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **635**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **27**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **7.1-7.54**	**Harmonic Mean** **5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
5.3	JUVENILE	24H
6.1	51-114 MM, 1.45-5 G	96H
9.9	51-79 MM, 3.2 G	72H
9.9		
9.9	51-79 MM, 3.2 G	96H
9.9	51-79 MM, 3.2 G	72H
10	UNDERYEARLING	48H
10.8	57-76 MM, 2.7-4.1 G	96H
10.8		
10.8	57-76 MM, 2.7-4.1 G	96H
11.5	1.1G	96H
13	51-79 MM, 3.2 G	48H
14.4	57-76 MM, 2.7-4.1 G	96H
15.3	57-76 MM, 2.7-4.1 G	96H
15.7	51-79 MM, 3.2 G	24H
17.5	57-76 MM, 2.7-4.1 G	96H
20	FINGERLING, 50.8 MM, 1.71 G	24H
20	FINGERLING, 52.6 MM, 1.87 G	96H
50	FINGERLING, 51.8 MM, 1.85 G	96H
50	FINGERLING, 50.8 MM, 1.71 G	96H
50	FINGERLING, 52.6 MM, 1.87 G	96H
50	FINGERLING, 51.8 MM, 1.85 G	24H
50	FINGERLING, 51.8 MM, 1.85 G	96H
98.4	SPERM	96H
100	FINGERLING, 53.1 MM, 1.86 G	24H
100	FINGERLING, 49.3 MM, 1.52 G	24H
100	FINGERLING, 49.2 MM, 1.55 G	96H
100	FINGERLING, 49.2 MM, 1.55 G	96H
100	FINGERLING, 49.2 MM, 1.55 G	24H
100	FINGERLING, 53.1 MM, 1.86 G	72H
100	FINGERLING, 53.1 MM, 1.86 G	48H
250	FINGERLING, 47.4 MM, 1.31 G	12D
250	FINGERLING, 50.4 MM, 1.64 G	24H

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **635**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **27**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **7.1-7.54**	**Harmonic Mean** **5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
250	FINGERLING, 53.4 MM, 1.94 G	96H
250	FINGERLING, 50.4 MM, 1.64 G	96H
250	FINGERLING, 53.4 MM, 1.94 G	96H
500	FINGERLING, 52.5 MM, 1.91 G	24H
500	FINGERLING, 51.5 MM, 1.87 G	48H
1000	FINGERLING, 54.7 MM, 2.02 G	96H
1000	FINGERLING, 52.7 MM, 1.89 G	24H
10000	5-10 CM	96H
10000	5-10 CM	96H
10000	5-10 CM	96H

Table 2.6.2.1.1.2 Mortality toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **2509**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **54**
Endpoint/Effect **Mortality**	**pH** **7.1-7.54**	**Harmonic Mean** **0.19**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.006	YEARLING, 29.5 G	24H
0.04	NR	24H
0.23	UNDERYEARLING	18D
0.55	NR	90D
0.9	1.4G	4H
0.91	NR	16H
0.97	NR	12H
1.3	0.8G	43D
1.8	0.8G	0.5H
2	EARLY FRY, 77 D	1D
2	6 MO, JUVENILE, 1.8 G	43D
3.3	0.8G	3.5H

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **2509**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **54**
Endpoint/Effect **Mortality**	**pH** **7.1-7.54**	**Harmonic Mean** **0.19**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
6.1	51-114 MM, 1.45-5 G	12H
6.1	51-114 MM, 1.45-5 G	4H
6.4	JUVENILE	100D
6.7	51-114 MM, 1.45-5 G	4H
7.9	51-114 MM, 1.45-5 G	24H
9.4	0.8G	4H
43	ADULT, 175 G	1D
43	ADULT, 175 G	50D
100	JUVENILE, 1-1.5 YR	1D
125	JUVENILE, 1-1.5 YR	2D
250	JUVENILE, 1-1.5 YR	2D
250	JUVENILE, 1-1.5 YR	55D
250	JUVENILE, 1-1.5 YR	42D
250	JUVENILE, 1-1.5 YR	1D
500	FINGERLING, 7.6-10.2 CM	55D
1000	FINGERLING,7.6-10.2 CM	2D
5000	6 WK	30D
5000	6 WK	5D
5000	100-200 G	24H
10000	FERTILIZED EGG, 0 H	45D
10000	FERTILIZED EGG, 24 H	20D
10000	EARLY EYED EGG, 14 D	3D
10000	LATE-EYED EGG, 28 D	5D
10000	SAC FRY, 42 D	5D
10000	5-10 CM	12H
10000	5-10 CM	24H
10000	5-10 CM	4H

Table 2.6.2.1.1.3 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **0.3**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **0.3**
Endpoint/Effect **NOEC/Growth**	**pH** **7.1-7.54**	**Harmonic Mean** **0.3**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.12		
0.55		90D

Table 2.6.2.1.1.4 Growth toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **0.4**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **0.8**
Endpoint/Effect **Growth**	**pH** **7.1-7.54**	**Harmonic Mean** **0.09**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.04	7 MO, JUVENILE, 3.0-5.1 G	12M
0.087	7 MO, JUVENILE, 3.0-5.1 G	16W
0.19	6 MO, JUVENILE, 2.8 G	130D
1.2	1.4G	300D

Table 2.6.2.1.1.5 Physiological toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **1.4**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **0.8**
Endpoint/Effect **Physiological**	**pH** **7.1-7.54**	**Harmonic Mean** **0.2**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.04	7 MO, JUVENILE, 3.0-5.1 G	
1	0.8G	
1.3	0.8G	
2.2	0.8G	
2.3	0.8G	

Table 2.6.2.1.1.6 Reproductive toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **7**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **7**
Endpoint/Effect **Reproductive**	**pH** **7.1-7.54**	**Harmonic Mean** **7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
7	JUVENILE	60MIN

Dieldrin Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and

Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to dieldrin, NMFS added an additional step to its analysis for dieldrin to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.24 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.1.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.1.1, predicts a magnitude of

effect ranging from a low of an LC_{zero} at a concentration of 10,000 µg/L to a high of an LC_{21} at a concentration of 0.56 µg/L. In other words, the acute criterion of 0.24 µg/L has an equivalent toxicity potential predicted to kill zero percent to 21 percent, with a median toxicity potential of an $LC_{0.7}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for dieldrin, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for dieldrin, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Dieldrin is a synthetic cyclic chlorinated hydrocarbons called cyclodienes, and was used extensively in the 1950s and 1960s as a soil insecticide. At that time, dieldrin (and aldrin), were two of the most widely used domestic pesticides in the United States (EPA 1980a). However, the EPA cancelled the registration for both compounds in 1975 (Biddinger and Gloss 1984).

Once aldrin has been applied to any aerobic and biologically active soil, it rapidly undergoes a metabolic epoxidation reaction that converts it to dieldrin (EPA 1980a, and Wolfe and Seiber 1993). In fish, the epoxidation of aldrin to dieldrin occurs via a mixed-function oxidase system, which has been demonstrated in golden shiners, mosquitofish, green sunfish, bluegill sunfish and channel catfish (as cited in Chambers and Yarbrough 1976). Dieldrin can be further modified when exposed to sunlight, via cyclization to photodieldrin (Wolfe and Seiber 1993).

Dieldrin has extremely low volatility and low solubility in water. It is more environmentally stable than aldrin, and is probably the most stable of the cyclodiene insecticides (EPA 1980a, Wolfe and Seiber 1993). For this reason, dieldrin is more frequently observed in the environment than aldrin (Biddinger and Gloss 1984). One study, conducted on the environmental fate and transport of dieldrin in the Coralville Reservoir in eastern Iowa, revealed that 10% of the entire input of dieldrin into the reservoir was taken up by fish, 40% entered the sediment, and 50% was exported from the reservoir in the outflow. Moreover, of the portion of dieldrin that was present specifically in the water column, 74% occurred in fish, 25% was dissolved in water, and less than 1% was adsorbed to suspended solids (Schnoor 1981).

Acute toxicity of dieldrin reported in rainbow trout and other fish includes effects on cardiac muscles, as well as inhibition of oxygen uptake, the central respiratory center, bronchial muscles, and the central nervous system (Lunn *et al.* 1976). Aldrin and dieldrin are similarly toxic to fish, although aldrin is more toxic to cladocerans than dieldrin (EPA 1980a). Additionally, photodieldrin is more toxic than dieldrin (Wolfe and Seiber 1993).

Because it is extremely a-polar, dieldrin that is present in fish has a particularly high affinity for fat. However, although it can be mobilized from tissue when the fish is placed in clean water, the dieldrin that has been eliminated then re-enters the water, making it available for subsequent uptake by other organisms (EPA 1980a). In channel catfish, approximately 50% of the dieldrin that had accumulated in dorsal muscle due to water-born exposure was eliminated after 14 days post-exposure, with total depuration by 28 days post-exposure. However, dieldrin that had accumulated in tissue due to dietary exposure was eliminated more slowly at 28 days post-exposure; approximately one third of the original dieldrin in muscle tissue was still present (Shannon 1977a). For rainbow trout, the predicted time to eliminate 50% of the dieldrin accumulated via dietary exposure is 40 days (Macek *et al.* 1970). In contrast, *Daphnia sp.* required four days to eliminate 50% of the photodieldrin that was accumulated in a water-born exposure study (Khan *et al.* 1975) and goldfish required less than 12 hours (Khan and Khan 1974). For the freshwater mussel *Lampsilis siliquoidea*, the half life of dieldrin was 4.7 days (Bedford and Zabik 1973). Khan and Khan (1974) noted that the initial elimination of dieldrin or photodieldrin from goldfish or *Daphnia* was due to excretion into the surrounding water.

A study by Van Leeuwen *et al.* (1985) examined the effects of water-borne dieldrin on rainbow trout at various early life stages, including fertilized eggs, early and late eye point eggs, sac fry and early fry. In the egg, the yolk acted as a temporary ‘toxicant sink’, but later in development, during the early sac fry stage, dieldrin was delivered from the yolk and began to accumulate in the fish tissue. The highest concentration in tissue was reached at the end of the sac fry stage. The second highest concentration in tissue was reached at the early fry stage, when susceptibility to dieldrin toxicity is most pronounced in early life stages.

The scope of the toxic properties of dieldrin is reinforced by the other studies reported above that involved other salmonid species for which lethality occurred at levels that were also below or slightly above the proposed acute criterion for dieldrin. Two of the trout studies (Van Leeuwen *et al.* 1985, Shubat and Curtis 1986) were more recent than the listed species studies. Also, two trout studies were done in flow-through experiments with measured dieldrin concentrations, which are likely to reflect more accurate estimates of toxicity than static experiments with nominal dieldrin concentrations (Chadwick and Shumway 1969, Shubat and Curtis 1986). The more recent and flow-through studies reported lethality concentrations that were below or near the proposed acute criterion for dieldrin, suggesting that this criterion could kill listed salmonid species.

Phillips and Buhler (1979) exposed fingerling rainbow trout to 0.18 µg/L dieldrin for 61 days under flow-through conditions and measured dieldrin concentrations. This resulted in a reduction in the rate of fat accumulation in fish that were fed a relatively high-fat diet (tubificid worms). Whole wet fish tissue concentration that corresponded to this effect was 0.82 or 1.32 mg/kg dieldrin. The effect of dieldrin exposure on fat accumulation was not apparent when fish were fed a relatively low fat diet (moist pellets), thus demonstrating that dieldrin toxicity can be affected by diet composition.

These limited results suggest that the proposed chronic criterion for dieldrin may avoid harming listed salmon subjected to short-term, water-borne exposure. However, they do not indicate whether the proposed chronic criterion is protective against bioaccumulation-related effects. To

address this, several dietary exposure studies were evaluated that reported dieldrin tissue concentrations and chronic effects. If a specific chronic effect is associated with a specific tissue concentration and the BCF for dieldrin is known, then the tissue concentration and BCF can be used to back-calculate an estimate of the aqueous dieldrin exposure concentration resulting in an equivalent tissue concentration, and thus an equivalent chronic effect.

Two BCF values were identified: 1,700 whole body BCF for early fry rainbow trout (Van Leeuwen *et al.* 1985) and 8,875 whole body BCF for juvenile rainbow trout (calculated from Shubat and Curtis 1986). These BCF values are assumed to represent the low and high range for salmonid BCFs. Using these BCFs and data presented in the following studies, equivalent aqueous (*i.e.*, water-borne only) dieldrin concentrations NMFS estimated to be between 0.89 and 65 times the proposed chronic criterion of 0.056 µg/L for dieldrin.

Hendricks *et al.* (1979) reported repressed growth in juvenile rainbow trout exposed to 5 ppm dieldrin in their diet for 12 months at 12°C, with a corresponding tissue concentration of approximately 1.6 mg dieldrin/kg whole fish. The corresponding concentration for dieldrin in a water-borne-only exposure experiment was estimated here to be between 0.18 µg/L and 0.94 µg/L.

Mehrle *et al.* (1971) reported alteration of the serum concentration of 11 amino acids in rainbow trout exposed to 1 mg dieldrin/kg body weight per week in their diet for 140 days at 16°C, with a corresponding tissue concentration of 1.8 mg dieldrin/kg whole fish. The corresponding concentration for dieldrin in a water-borne-only exposure experiment was estimated here to be between 0.2 µg/L and 1.1 µg/L. The results suggested that the utilization of five of the amino acids was inhibited by dieldrin, possibly due to an effect on enzymes which are responsible for the utilization and energy transformation of these specific amino acids.

Kilbey *et al.* (1972) conducted a 300-day dietary exposure study using rainbow trout held at 17°C. Effects that were observed included increased blood phenylalanine levels, decreased liver phenylalanine hydroxylase activity, and increased concentration of urine phenylpyruvic acid when dieldrin was present in the diet at 14 µg/L to 430 µg/L dieldrin/kg body weight/day (0.36µg/L to 10.8µg/L dieldrin/g of food). The corresponding dieldrin tissue concentration was 0.41 mg/kg to 6.23 mg/kg wet weight. Based on these tissue concentrations, a corresponding concentration for dieldrin in a water-borne only exposure experiment was estimated to be between 0.05 µg/L and 3.66 µg/L. The three effects observed parallel those seen in phenylketonuria, an inherited defect in human phenylalanine metabolism that is also characterized by mental deficiency. Although the study did not address analogous effects, it is possible that fish adaptability, behavior, and survival may be compromised based on biochemical similarities.

There are numerous additional studies on tissue exposure of salmonids to dieldrin. However, they have low utility for the purpose of evaluating the proposed chronic criterion, either because necessary data and findings were not reported, whole body tissue concentration could not be

estimated, or test specimens were exposed to a mixture of compounds (*e.g.*, Macek *et al.* 1970, Mehrle and Bloomfield 1974, Poels *et al.* 1980, Shubat and Curtis 1986).

Salmonid fishes and other freshwater fish species strongly bioaccumulated dieldrin from the water column in laboratory exposure studies. Van Leeuwen *et al.* (1985) exposed early fry rainbow trout to dieldrin for 24 hours and reported a steady state BCF of 1,700. Chadwick and Shumway (1969) reported a whole body BCF equal to approximately 3,200 for newly hatched steelhead trout alevins after 35 days of exposure.

Whole body or lipid BCF calculated from information provided in other studies on exposure concentration, duration, and tissue residue concentration are also indicative of the tendency of dieldrin to bioaccumulate. Shubat and Curtis (1986) exposed juvenile rainbow trout to 0.04 µg/L dieldrin for 16 weeks in a flow-through experiment with a measured dieldrin concentration, and indicated a whole body tissue residue level of 120 to 320 ng dieldrin/g fish tissue, or 7.1 ng to 11 ng dieldrin/mg lipid. This translates into a whole body BCF of approximately 3,000 to 8,000, or a lipid BCF of 178,000 to 275,000. For fish exposed to 0.08 µg/L, the calculated whole body BCF becomes 2,500 to 8,900, and the lipid BCF 225,000, indicating slightly higher bioaccumulation rates at higher water concentrations.

The only other freshwater fish for which laboratory-derived bioaccumulation information was found is the channel catfish *Ictalurus punctatus*. Shannon (1977a) conducted a 28-day exposure to 0.075 µg/L of an 87% dieldrin formulation in a flow-through experiment with measured concentrations of dieldrin. Based on reported tissue concentrations, the calculated dorsal muscle BCF is 2,333 for smaller fish and 3,653 for larger fish. Although Shannon (1977a) suggests that the higher bioaccumulation observed for the larger fish in this study could be due to a higher fat content, this notion was not supported by results from a field study where larger fish did not consistently harbor higher residue concentrations (Kellogg and Bulkley 1976). In another experiment, a 70-day exposure to 0.013 µg/L dieldrin resulted in a calculated dorsal muscle BCF of 2,385, with equilibrium being reached more rapidly at lower level exposures than at higher levels (Shannon 1977b). These laboratory BCF values for catfish are roughly comparable to BCFs determined for salmonids. However, they are approximately 10 fold below the BCF values reported in channel catfish from field studies. Leung *et al.* (1981) sampled fish and water from the Des Moines River in Iowa in June and August 1973, during a time when aldrin was being used on area cropland. The corresponding calculated muscle tissue BCF values range from 2,220 to 22,200. The authors did not discuss the possibility that the tissue residue levels could reflect dieldrin accumulation from food and sediment as well as water. However, Chadwick and Brocksen (1969 as cited in Shannon 1977a) noted that, when selected fish were tested for accumulation of dieldrin from food or water, most of the dieldrin in the tissue came from water. The reported information from additional field studies conducted in the Des Moines River can be used to calculate the BCF values for various other freshwater fish, yielding estimated BCFs of up to 1,600 for carpsucker, 10,200 for sand shiner, 15,500 for spotfin shiner, or 7,500 for bluntnose minnow (Kellogg and Bulkley 1976).

No laboratory derived BCF values were available for any aquatic insect species that are prey for salmonids. Reinert (1972) noted a BCF of approximately 14,000 for *Daphnia magna* exposed to dieldrin for 3 days. Kellog and Bulkley (1986) conducted a field study from which reported

tissue and water concentrations of dieldrin can be used to calculate BCF values for various insect, crustacean, or fish prey species used by salmonids. Water samples contained 0.004 µg/L to 0.012 µg/L dieldrin, and aquatic organisms had tissue levels ranging from 2 ppb to 61 ppb from the Des Moines River in Iowa in 1973. Corresponding calculations result in BCF values that are on the order of 1,500 for the stonefly *Pteronarcys*, 5,100 for the mayfly *Potamanthus*, 3,500 for Chironomidae, 3,600 for Trichoptera, and 1,300 for the crayfish *Oronectes rusticus*.

For photodieldrin, BCF values derived from laboratory studies on various freshwater fish are approximately an order of magnitude lower than laboratory dieldrin BCF values determined for salmonids and catfish. For example, after a one 1-day exposure to 20 µg/L photodieldrin in a static experiment with measured dieldrin concentrations, BCF values were 133 for bluegill (*Lepomis machrochirus*), 150 for minnow (*Lebistes reticulata*), 609 for goldfish (*Carassius auratus*), and 820 for guppy (*Gambia affinis*) (Khan and Khan 1974). The data of Khan and Khan (1974) also indicated a BCF around 1,200 for a Gammarid exposed for four days at 10 µg/L.

Statham and Lech (1975) noted that dieldrin may interact synergistically with carbaryl. In a water-borne exposure study with fingerling rainbow trout, a 4-hour exposure to dieldrin at 1,000 µg/L caused 16% mortality, but when 1 mg/L carbaryl was added to the mixture, the resulting mortality level was 94%, which was greater than the sum of effects for either compound alone. No mechanism for this interaction was determined or suggested. Based on this information, natural freshwater areas that are known to contain both carbaryl (or other carbamate insecticides) and dieldrin may require special consideration with respect to synergistic toxicity to fish.

Interaction between dieldrin and DDT varies depending on the toxicity endpoint considered. Macek *et al.* (1970) conducted an experiment with rainbow trout fed dieldrin and DDT for 140 days. This was sufficient time for equilibrium to be reached with respect to tissue residue accumulation of the two compounds. A significant increase in lipogenesis was seen with either contaminant alone, but, after several months, an additive effect also was apparent in fish that were fed both contaminants. In the pyloric caecae, the accumulation rate of DDT was increased by the presence of dieldrin, while that of dieldrin decreased. Further, elimination of DDT decreased markedly, while elimination of dieldrin remained unchanged. The results from this study suggest the possibility of increased bioaccumulation of DDT when dieldrin and DDT are present together in the environment. In contrast, Mayer *et al.* (1972) noted an antagonistic effect in rainbow trout that were fed dieldrin at non-lethal levels and DDT at lethal levels for 6 days. The fish died at about half the rate as with DDT alone. The mechanism of this interaction was not determined in this study. From an environmental perspective, this observation may be important only when high (lethal) levels of DDT are bioavailable.

An antagonistic interaction also was suggested by Hendricks *et al.* (1979) between dieldrin and aflatoxin B_1. In juvenile rainbow trout fed with both compounds for 12 months, the observed growth inhibition was similar to that caused by dieldrin alone, thus indicating a reduction in the growth inhibitory effect of Aflatoxin B_1.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Acute toxicity data available identified effects of dieldrin on aquatic invertebrates ranging from 0.5 µg/L to 3.7 µg/L:

- Sanders and Cope (1968) reported 96 hour LC_{50} values of 0.5 µg/L for the stonefly naiads *Pteronarcys californica* and *Pteronarcella badia*, and 0.58 µg/L for the stonefly naiad *Claassenia sabulosa*, in static experiments performed at around 15.5°C and pH 7.1.
- Karnak and Collins (1974) reported a 24 hour LC_{50} of 0.7 µg/L for the midge larvae *Chironomus tentans*, using 85% dieldrin at 22°C.
- Bowman *et al.* (1981) reported an 18-hour LD_{50} value of 3.7 µg/L for the glass shrimp *Palaemonetes kadiakensis* at 23°C in a static experiment.

Reports could not be found in the toxicological literature that indicate adverse effects from dieldrin occur to salmonid prey species at levels below the proposed chronic criterion of 0.056 µg/L. Results for three aquatic insects and three crustaceans demonstrate that adverse effects are manifest at the individual or population level only when dieldrin concentrations are much higher, ranging between 9 and 66 times the criterion (Jensen and Gaufin 1966, Adema 1978, Daniels and Allan 1981, Phipps *et al.* 1995).

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Dieldrin. The available evidence for dieldrin indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), reduced growth (moderate intensity), physiological trauma (moderate intensity), and reproduction (low intensity).

2.6.2.1.2 Endosulfan-alpha and Endosulfan-beta

Endosulfan Criteria. The proposed acute and chronic criteria for endosulfan-alpha and endosulfan-beta are 0.22 µg/L and 0.056 µg/L, respectively.

Tables 2.6.2.1.2.1 through 2.6.2.1.2.2 report toxicity data from the ECOTOX database for freshwater endosulfan, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.2.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endosulfan-alpha and endosulfan-beta.

Criterion **Freshwater Endosulfan-alpha and Endosulfan-beta**		**Data Set** **ECOTOX**
Criterion Concentration Acute **0.22 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **0.88**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **30-255 mg/L $CaCO_3$**	**Geometric Mean** **0.66**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **NR**	**Harmonic Mean** **0.51**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.17	NEWBORN	96H
0.24	NEWBORN	96H
0.26	NEWBORN	96H
0.26	NEWBORN	96H
0.27	NEWBORN	96H
0.29	NEWBORN	96H
0.3	NEWBORN	96H
0.3	NEWBORN	96H
0.32	NEWBORN	96H
0.41	NEWBORN	96H
0.42	NEWBORN	96H
0.49	NEWBORN	96H
0.63	NEWBORN	96H
0.69	NEWBORN	96H
0.79	NEWBORN	96H
0.8	NEWBORN	96H
0.8	NEWBORN	96H
0.81	NEWBORN	96H
0.86	NEWBORN	96H
0.94	NEWBORN	96H
1.21	NEWBORN	96H
1.3	NEWBORN	96H
1.34	NEWBORN	96H
1.5	NEWBORN	96H
1.63	NEWBORN	96H
1.69	NEWBORN	96H
1.7	NEWBORN	96H
2.43	NEWBORN	96H
2.6	NEWBORN	96H

Table 2.6.2.1.2.2 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endosulfan-alpha and endosulfan-beta.

Criterion **Freshwater Endosulfan-alpha and Endosulfan-beta**		**Data Set BE**
Criterion Concentration Acute **0.22 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **0.88**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **30-255 mg/L $CaCO_3$**	**Geometric Mean** **0.66**
Endpoint/Effect **NOEC**	**pH** **NR**	**Harmonic Mean** **0.51**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.016		
0.02		
0.063		
0.075		
0.078		
0.17		

Water Quality Parameters as Predictors of Endosulfan Acute Toxicity. Schoettger (1970) tested various water quality parameters to determine their effect on the toxicity of endosulfan to several fish species. Variations in calcium and magnesium salts did not alter the acute toxicity to western white suckers, nor did changes in pH between 6.4 and 8.4. However, experiments with rainbow trout indicated that temperature changes did have an effect on toxicity. In three different studies, endosulfan toxicity increased with increasing temperature. Two other studies using rainbow trout also reported a temperature effect. Sunderam *et al.* (1992) determined that the 96-hour LC_{50} changed from 1.6 µg/L at 4°C to 0.7 µg/L at 12°C, using static conditions, pH 7.5, and measured concentrations of endosulfan. Macek *et al.* (1969) reported 96-hour LC_{50}s of 2.6 µg/L, 1.7 µg/L, and 1.5 µg/L at 1.6°C, 7.2°C, or 12.7°C, respectively, under static conditions at pH 7.1 and nominal endosulfan concentrations.

Endosulfan Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these

studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to endosulfan-alpha and endosulfan-beta, NMFS added an additional step to its analysis for endosulfan-alpha and endosulfan-beta to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.22 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.2.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.2.1, predicts a magnitude of effect ranging from a

low of an $LC_{4.2}$ at a concentration of 2.6 µg/L to a high of an LC_{65} at a concentration of 0.17 µg/L. In other words, the acute criterion of 0.24 µg/L has an equivalent toxicity potential predicted to kill 4.2 percent to 65 percent, with a median toxicity potential of an $LC_{13.9}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for endosulfan-alpha and endosulfan-beta, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for endosulfan-alpha and endosulfan-beta, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Endosulfan is a broad-spectrum polychlorinated cyclodiene insecticide. It is used to control over 100 agricultural pests and 60 food and non-food crops, and does not occur naturally in the environment. It was first developed in Germany by Hoechst in 1954 under the registered trade name Thiodan. In its pure form, endosulfan exists in two different conformations: I (alpha) and II (beta). Technical endosulfan, the form which is most often used in laboratory toxicity studies, is 94% to 96% pure, with an approximate ratio of 7:3 alpha:beta isomers (Naqvi and Vaishnavi 1993).

Endosulfan is virtually insoluble in water, but is readily dissolved in organic solvents before its addition to aqueous formulations (Geobel *et al.* 1982, Naqvi and Vaishnavi 1993). In alkaline water, hydrolysis is the primary process for degradation, with the beta isomer hydrolyzing more rapidly than the alpha isomer (Peterson and Batley 1993). Endosulfan diol is the main product of chemical hydrolysis, but it is also oxidized to endosulfan sulfate (Naqvi and Vaishnavi 1993). In solution, the alpha isomer is more abundant than the beta isomer or endosulfan sulfate. Also, in the aquatic environment, endosulfan beta and endosulfan sulfate are more likely to be bound to sediment and particulates than endosulfan alpha (Peterson and Batley 1993).

Endosulfan acts as a central nervous system poison (Naqvi and Vaishnavi 1993). Of the organochlorine insecticides, it is one of the most toxic to aquatic organisms (EPA 1976; EPA 1980g). In general, freshwater fish are more sensitive to endosulfan than freshwater invertebrates (EPA 1980g), and marine organisms are more sensitive than freshwater ones (Naqvi and Vaishnavi 1993). The toxicities of endosulfan and endosulfan sulfate are roughly equivalent (Naqvi and Vaishnavi 1993). However, comparisons of the toxicity of individual isomers of endosulfan indicate that the alpha form is generally more toxic than the beta. The other biological metabolites of endosulfan that do not contain sulfur, such as endosulfan diol, endosulfan ether, and endosulfan lactone, are considerably less toxic than either the sulfur-containing endosulfan sulfate or alpha or beta isomers.

Most endosulfan toxicity studies on aquatic organisms have evaluated direct water-borne exposure. Studies reported by Barry *et al.* (1995) indicated that, for the cladoceran *Daphnia carinata*, water-borne exposure is the most toxic route. Toxicity towards *D. carinata* also increase at higher food concentrations. This may be due to a higher level of persistence of endosulfan in the water column, or increased uptake of the compound by the test organisms due to elevated metabolism. Similar toxicity studies that assessed food concentration or route of exposure for fish were not found in the literature. However, there are other aspects of study design that can influence toxicity outcome. Static flow or semi-static assay conditions are more likely to underestimate toxicity when compared with the more environmentally relevant constant flow assays. Studies that include nominal, or unmeasured, test compound concentrations during the exposure period also are more likely to underestimate toxicity compared with those with measured concentrations (Naqvi and Vaishnavi 1993). The toxic effects of endosulfan on fish are influenced by water temperature, with increased toxicity generally observed at higher temperatures. The influence of temperature is discussed further below.

The available information on the chronic effects of endosulfan on salmonids or other freshwater fish is limited. Arnold *et al.* (1996) observed sublethal effects at concentrations between 0.2 times and 1.8 times the proposed chronic criterion. Mature male rainbow trout that were exposed for 28 days to 0.01 µg/L endosulfan (measured) in a flow-through assay at 14.5^{o}C developed qualitative hepatic cytological ultrastructural alterations. This dose was the LOEC. At 0.05 µg/L and 0.1 µg/L, degenerative subcellular effects such as dilation of intermembranous spaces in mitochondria and deformation of mitochondria were observed. Other subcellular effects included proliferation of smooth endoplasmic reticulum (SER), circular arrays of rough endoplasmic reticulum (RER), and an increase in lysosomal elements. The SER and RER effects were probably an indication of the activity of mixed-function oxygenases. These type of structural alterations have been shown by many investigators to be highly selective and sensitive biomarkers of chronic toxicity, although specific effects on fish health have not been elucidated.

Toxicity studies on other freshwater fish species have indicated adverse effects when exposure concentrations ranged between 0.8 times and 3.6 times the chronic criterion:

- Verma *et al.* (1981) exposed the freshwater catfish *Mystus vittatus* to 0.045, 0.067, and 0.13 u/L endosulfan for 30 days at 24^{o}C in a nominal, static renewal assay. This treatment caused alterations in acid phosphatase, alkaline phosphatase, and glucose-6-phospatase in liver, kidney, and gills. Although the reason for these alterations is not clear, they may be due to uncoupling of oxidative phosphorylation or structural alterations of lysosomes.
- Sastry and Siddiqui (1982) exposed the freshwater murrel *Channa punctatus* to 0.2 µg/L endosulfan for 15 and 30 days at 20^{o}C, pH 7.4 in a static renewal assay. This resulted in a reduction in the rate of glucose absorption by the intestine, possibly due to structural damage to the intestinal mucosa, or a decrease in the activity of enzymes that are involved in nutrient absorption, such as Na^{+}-K^{+} ATPase and alkaline phosphatase.

The results of several studies indicate adverse effects can occur when concentrations are below or near the proposed chronic criterion after an exposure period less than 96 hours. Effects were evident at concentrations that were between 0.9 times and 1.8 times the proposed chronic criterion, suggesting that chronic toxic effects could occur to salmonids under the proposed

criterion, assuming effects are equal among species. These studies are described below:

- Murty and Devi (1982) exposed the freshwater snakehead fish *Channa punctata* (Bloch) to 0.05 µg/L endosulfan alpha for 4 days at 27^{o}C in a nominal, continuous flow assay. The lipid content and glycogen concentration of liver, muscle, and brain were significantly altered, as was the protein content of muscle and kidney.
- Nowak (1996) exposed the freshwater catfish *Tandanus tandanus* to 0.1 µg/L endosulfan for 24 hours in a nominal, static assay. Effects observed included dark atrophied hepatocytes (usually a sign of cell necrosis resulting from chronic injury); structural (necrotic) changes in liver tissue; proliferation, dilation, and vesiculation of the RER (possibly due to inhibition of protein synthesis); concentric bodies (a possible sign of cytologic regeneration); and residue levels in liver tissue up to 80 ppb.
- Nowak (1992) exposed *Tandanus tandanus* to 0.1 µg/L endosulfan for 24 hours in a measured, static assay. This resulted in edema and lifting and hyperplasia of lamellar epithelium in the gills, and also increased in respiratory diffusion distance. Although this may allow separation of blood from the toxicant, it can also damage gills, having deleterious effects on fish physiology.
- Rao *et al.* (1980) exposed the Indian major carp *Labeo rohita* to 0.1 µg/L endosulfan for 1 hour at 28^{o}C, pH 8.4 in a nominal, static assay. An increase in oxygen consumption was observed.

Information on uptake, metabolism, and elimination of endosulfan was not available for salmonid fishes. However, the following is a brief overview of information available for other freshwater fish species, including the spotted snakehead *Channa punctata* (Devi *et al.* 1981), the rohi *Labeo rohita* (Rao *et al.* 1980), the Indian carp *Catla catla* (Rao 1989), the climbing perch *Anabus testudineus* (Rao and Murty 1980), and goldfish and western white sucker (Schoettger 1970).

The unaltered alpha and beta forms of endosulfan were detected in *Channa punctata*, *Anabus testudineus*, and *Catla catla* in one or more tissues, including brain, gills, kidney, liver, and muscle. In *Catla catla* in particular, muscle was found to be the principle storage site of unaltered endosulfan.

The principal metabolites of endosulfan in *Catla catla*, *Channa punctata*, or *Labeo rohita* were reported to be endosulfan alcohol, endosulfan ether, or endosulfan lactone. Other metabolites that were detected in various fish included endosulfan alpha-hydroxyether and endosulfan sulfate. The liver was cited as either the principal detoxifying organ or the site where uptake appeared to be considerably higher than for other tissues in *Labeo rohita*, the western white sucker *Catostomus commersoni*, and the goldfish *Carassius auratus auratus*. This differed somewhat from the climbing perch, in which both the liver and kidneys were reported as being the principal sites of detoxification.

Both Endosulfan and endosulfan sulfate are known to bioconcentrate, and thought to bioaccumulate (EPA 1999), which is in accord with log K_{ow} values of 4.10, 3.83, and 4.52 for technical endosulfan, isomer I and isomer II, respectively (Karickhoff and Long 1995). Toxicity of endosulfan to aquatic biota is influenced by water temperature (increased toxicity with

increased temperature), and type of isomer (EPA 1999). Of the organochlorine insecticides, it is one of the most toxic to aquatic organisms (EPA 1980f). The primary mode of action of endosulfan is disruption of nerve function in the central nervous system (Casarett and Doull 2001). In general, freshwater fish are more sensitive to endosulfan than freshwater invertebrates (EPA 1980f). Effects of endosulfan toxicity to freshwater organisms include anoxic stress, altered calcium deposition, blood disease, altered gill structure, and reduced survival (EPA 1999).

Reports on the bioconcentration of endosulfan in salmonids were not available, although limited information for other freshwater fish was found, indicating that the BCF can vary greatly between species. Ramaneswari and Rao (2000) exposed *Channa punctata* to 0.141 µg/L endosulfan (alpha or beta isomers) for 1 month and measured a whole body BCF of 13. A similar exposure of *Labeo rohita* yielded a BCF of 37 for alpha endosulfan and 55 for beta endosulfan. The exposure concentration used (0.141 µg/L) was 2.5 times the proposed chronic criterion. These BCF values were much lower than those obtained for yellow tetra (*Hyphessobrycon bifasciatus*), in which the whole body BCF was 11,600 after a 21 day exposure to 0.3 µg/L endosulfan at 22^{o}C, pH 7.1 under static-renewal conditions (Jonsson and Toledo 1993). In this study, the total residues in fish increased with increasing time, and the authors indicated that a steady state had not been reached. The biological half-life was estimated at 1.8 days, which is similar to the half-life in goldfish (Oeser *et al.* 1971 as cited in Geobel *et al.* 1982).

Only two reports of endosulfan bioaccumulation were found for salmonid prey species. Sabaliunas *et al.* (1998) exposed the lake mussel *Anodonta piscinalis* to 1.5 µg/L endosulfan in a continuous flow experiment at 10^{o}C with measured contaminant concentration. They noted a whole BCF of 750 under conditions that may not have reached steady state. Finally, a field study was conducted using paired oyster whole body tissue samples and water samples from the Patuxent River, which discharges into the Chesapeake Bay in Maryland (Lehotay *et al.* 1999). In oyster tissue, more endosulfan sulfate was present compared to the alpha or beta isomers. In the water samples, more of the beta isomer was present than the alpha isomer or endosulfan sulfate (even though beta is less soluble than alpha and constitutes only 30% of the endosulfan mixture that is commonly used). Based on the average concentration of endosulfan alpha, beta, or sulfate in oyster tissue (0.037 ng/g to 0.13 ng/g) or in water samples (0.5 ng/L to1.0 ng/L), one can calculate the BCF range as 37 to 260.

Sublethal Effects Summary. Although the data regarding sublethal effects on fishes exposed to endosulfan-alpha and endosulfan-beta is available, there are no chronic toxicity studies available for juvenile salmonid fishes. If the mechanism and mode of actions are similar for salmonid fishes, salmonid fishes will suffer chronic toxic effects.

Toxicity to Food Organisms. Most toxicity studies indicate lethal effects do not occur on salmonid prey species until concentrations are between 19 and 2,232 times the proposed acute criterion. These species include the freshwater scud *Gammarus lacustris*, with 96-hour LC_{50} values of 4.1 µg/L or 5.8 µg/L (Johnson and Finley 1980; Sanders 1969 as cited in EPA 1980g); the cladoceran *Daphnia magna*, with LC_{50} values of 56 µg/L to 271 µg/L (Schoettger 1970, Nebeker *et al.* 1983, EPA 1976); damselfly naiad 96-hour LC_{50} of 71.8 µg/L to 107 µg/L

(Schoettger 1970); and a 48 hour LC_{50} of 215 µg/L for *Moinodaphnia macleayi* or 491 µg/L for *Ceriodaphnia dubia*.

Chronic exposure studies reported in the scientific literature appear to include only cladocerans, and all of these studies report chronic effects at concentrations well above the proposed chronic criterion. For example, *D. magna* exhibited reduced survival after 22 days of exposure to 7 µg/L endosulfan or reduced reproduction in the second generation at 37.7 µg/L (EPA 1976), the LOEC for decrease in number of young for *C. dubia* was 20 µg/L after 14 days exposure, or 40 µg/L for *M. macleay* (Sunderam *et al.* 1994), and reduction of brood size and body length for *Daphnia carinata* was observed after 6 days at 320 µg/L (Barry *et al.* 1995).

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Endosulfan-alpha and Endosulfan-beta. The available evidence indicates that listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects including mortality (moderate intensity). There are no chronic toxicity studies available for juvenile salmonid fishes. However, the NOEC analysis suggests that salmonid fishes will suffer chronic toxic effects—sublethal effects— (moderate intensity). Furthermore, if the mechanism and/or mode of actions for the fish species with sublethal toxicity data are similar for salmonid fishes, salmonid fishes will suffer sublethal effects (moderate intensity).

2.6.2.1.3 Endrin

Endrin Criteria. The proposed acute and chronic criteria for endrin are 0.086 µg/L and 0.036 µg/L, respectively.

Tables 2.5.2.1.3.1 through 2.5.2.1.3.5 report toxicity data from the ECOTOX database for freshwater endrin, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.3.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion Freshwater Endrin		**Data Set ECOTOX**
Criterion Concentration Acute 0.086 Micrograms Liter^{-1}	**Temperature 1.6-20° Celsius**	**Arithmetic Mean 167**
Criterion Concentration Chronic 0.036 Micrograms Liter^{-1}	**Hardness 44-272 mg/L $CaCO_3$**	**Geometric Mean 1.1**
Endpoint/Effect LC_{50}/Mortality	**pH 6-7.95**	**Harmonic Mean 0.3**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.02	22 D, 32.3 MM, PTERYGIO LARVA	72H
0.02	29 D, 34.1 MM, PTERYGIO LARVA	48H
0.06	29 D, 34.1 MM, PTERYGIO LARVA	72H
0.089	FINGERLING	96H
0.095	.37 G	96H
0.113		
0.117	.37 G	72H
0.12	22 D, 32.3 MM, PTERYGIO LARVA	48H
0.12	71 D, 46.2 MM, JUVENILE	48H
0.12	71 D, 46.2 MM, JUVENILE	72H
0.167	1.30 G	96H
0.192	.37 G	48H
0.192		
0.218	1.30 G	48H
0.25	15 D, 31.0 MM, PROTOPTERYGIO LARVA	48H
0.25	15 D, 31.0 MM, PROTOPTERYGIO LARVA	72H
0.27	1.9 G, 2.5 IN	96H
0.27		
0.3	1.9 G, 2.5 IN	72H
0.3	1.44 G	96H
0.317	1.15 G	96H
0.327	1.24 G	96H
0.343	1.15 G	72H
0.355		
0.4	8 D, 29.2 MM, ELEUTER EMBRYO	72H
0.405		
0.432	1.15 G	48H
0.451	1.24 G	72H
0.464	2.04 G	96H
0.5	22 D, 32.3 MM, PTERYGIO LARVA	24H
0.5	2.04 G	72H
0.51		

Criterion Freshwater Endrin		Data Set ECOTOX
Criterion Concentration Acute **0.086 Micrograms Liter^{-1}**	**Temperature** **1.6-20° Celsius**	**Arithmetic Mean** **167**
Criterion Concentration Chronic **0.036 Micrograms Liter^{-1}**	**Hardness** **44-272 mg/L $CaCO_3$**	**Geometric Mean** **1.1**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6-7.95**	**Harmonic Mean** **0.3**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.52	57-76 MM, 2.7-4.1 G	72H
0.55	29 D, 34.1 MM, PTERYGIO LARVA	24H
0.56	1.9 G, 2.5 IN	48H
0.568	1.24 G	48H
0.58	51-79 MM, 3.2 G	48H
0.58	51-79 MM, 3.2 G	72H
0.58	51-79 MM, 3.2 G	96H
0.58		
0.63	1G	96H
0.64	1G	96H
0.64	1.4G	96H
0.643	1.50 G	96H
0.674	1.50 G	72H
0.7	15 D, 31.0 MM, PROTOPTERYGIO LARVA	24H
0.7	22 D, 32.3 MM, PTERYGIO LARVA	12H
0.7	71 D, 46.2 MM, JUVENILE	24H
0.76	FINGERLING	24H
0.76		
0.79	57-76 MM, 2.7-4.1 G	96H
0.79	51-79 MM, 3.2 G	24H
0.8	57-76 MM, 2.7-4.1 G	48H
0.9	1 G, 1.625-2.25 IN	96H
0.9	1G	24H
0.9		
0.906	2.04 G	48H
0.92	6-8 G	96H
0.92		
0.97	1.4G	96H
1	1G	24H
1	1G	96H
1	1G	24H
1.01	6-8 G	72H
1.02	1.15 G	24H
1.1		
1.116	1.50 G	48H

Criterion Freshwater Endrin		Data Set ECOTOX
Criterion Concentration Acute **0.086 Micrograms Liter^{-1}**	**Temperature** **1.6-20° Celsius**	**Arithmetic Mean** **167**
Criterion Concentration Chronic **0.036 Micrograms Liter^{-1}**	**Hardness** **44-272 mg/L $CaCO_3$**	**Geometric Mean** **1.1**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6-7.95**	**Harmonic Mean** **0.3**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
1.12	1 G, 1.625-2.25 IN	72H
1.2	1.4G	96H
1.2	51-114 MM, 1.45-5 G	48H
1.2	51-114 MM, 1.45-5 G	72H
1.2	51-114 MM, 1.45-5 G	96H
1.2		
1.3	8 D, 29.2 MM, ELEUTER EMBRYO	48H
1.3	15 D, 31.0 MM, PROTOPTERYGIO LARVA	12H
1.3	1.4G	24H
1.3	57-76 MM, 2.7-4.1 G	24H
1.45	1 G, 1.625-2.25 IN	48H
1.5	6-8 G	48H
2	71 D, 46.2 MM, JUVENILE	12H
2	1.4G	96H
2	51-114 MM, 1.45-5 G	24H
2.17	1 G, 1.625-2.25 IN	24H
2.2	0.6-1.5 G	96H
2.355	1.50 G	24H
2.6	1.4G	24H
2.7	29 D, 34.1 MM, PTERYGIO LARVA	12H
2.9	8 D, 29.2 MM, ELEUTER EMBRYO	24H
4.6	1.4G	24H
5.2	2 D, 25.5 MM, ELEUTER EMBRYO	72H
6.3	8 D, 29.2 MM, ELEUTER EMBRYO	12H
7.7	1 D, 25.3 MM, ELEUTER EMBRYO	72H
11.9	1.4G	24H
12	1.9 G, 2.5 IN	24H
14.5	2 D, 25.5 MM, ELEUTER EMBRYO	48H
16.8	1 D, 25.3 MM, ELEUTER EMBRYO	48H
32.7	2 D, 25.5 MM, ELEUTER EMBRYO	24H
36.1	1 D, 25.3 MM, ELEUTER EMBRYO	24H
206	2 D, 25.5 MM, ELEUTER EMBRYO	12H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H

Table 2.6.2.1.3.2 Mortality toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion **Freshwater Endrin**		**Data Set** **ECOTOX**
Criterion Concentration Acute **0.086 Micrograms Liter^{-1}**	**Temperature** **2-20° Celsius**	**Arithmetic Mean** **6364**
Criterion Concentration Chronic **0.036 Micrograms Liter^{-1}**	**Hardness** **44-272 mg/L $CaCO_3$**	**Geometric Mean** **283**
Endpoint/Effect **Mortality**	**pH** **6-7.95**	**Harmonic Mean** **1.4**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.317	1.15 G	96H
0.464	2.04 G	96H
0.7		48H
0.906	2.04 G	48H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H

Table 2.6.2.1.3.3 Physiological toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion Freshwater Endrin		**Data Set ECOTOX**
Criterion Concentration Acute 0.086 Micrograms Liter^{-1}	**Temperature 1.6-20° Celsius**	**Arithmetic Mean**
Criterion Concentration Chronic 0.036 Micrograms Liter^{-1}	**Hardness 44-272 mg/L $CaCO_3$**	**Geometric Mean**
Endpoint/Effect Physiological	**pH 6-7.95**	**Harmonic Mean**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.043	430-520 G	26H
0.12	55-80 G, 12-18 CM	30D
0.12	12-15 CM, 55-80 G	30D
0.343	1.15 G	72H
0.432	1.15 G	48H
0.5	2.04 G	72H
1.02	1.15 G	24H
120	NR	30D

Table 2.6.2.1.3.4 Reproductive toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion Freshwater Endrin		**Data Set ECOTOX**
Criterion Concentration Acute 0.086 Micrograms Liter^{-1}	**Temperature 2-20° Celsius**	**Arithmetic Mean 0.22**
Criterion Concentration Chronic 0.036 Micrograms Liter^{-1}	**Hardness 44-272 mg/L $CaCO_3$**	**Geometric Mean 0.22**
Endpoint/Effect Reproductive	**pH 6-7.95**	**Harmonic Mean 0.22**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.218	1.30 G	48H

Table 2.6.2.1.3.5 Cellular toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion Freshwater Endrin		**Data Set ECOTOX**
Criterion Concentration Acute 0.086 Micrograms Liter^{-1}	**Temperature 1.6-20° Celsius**	**Arithmetic Mean 10**
Criterion Concentration Chronic 0.036 Micrograms Liter^{-1}	**Hardness 44-272 mg/L $CaCO_3$**	**Geometric Mean 4.3**
Endpoint/Effect Cellular	**pH 6-8**	**Harmonic Mean 1.6**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.92	6-8 G	96H
20	FINGERLING, 7 MO, 7.5-8.0 G	0.5H

Endrin Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to endrin, NMFS added an additional step to its analysis for endrin to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.086 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.3.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.3.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 10,000 µg/L to a high of an LC_{100} at a concentration of 0.02 µg/L. In other words, the acute criterion of 0.086 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{5.4}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the criterion concentration for endrin, which implies that listed species exposed to waters equal to criterion concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for endrin, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute toxic effects, but may not suffer chronic toxic effects.

Sublethal Effects. Endrin is a chlorinated pesticide that is a stereoisomer of dieldrin. It is no longer manufactured in the United States. Endrin ketone and endrin aldehyde are variants that occur as impurities or degradation products of endrin in commercial preparations of the insecticide. Endrin was first used in 1951 to control insects and rodents on cotton, apples, sugarcane, tobacco, and grain (IARC 1974, EPA 1980h, HSDB 1995). Its toxicity to migrant populations of migratory birds was the main reason for its cancellation as a pesticide in 1986 (EPA 1992b). It was still used as a toxicant on bird perches for several years, but this use was also banned in 1991 (EPA 1992b). There are no current releases of endrin in the United States

Exposure to endrin has been noted to result in adverse neurologic, liver, kidney, and miscellaneous endocrine and tissue weight effects (Treon *et al.* 1955 as cited in EPA 1980; Deichmann *et al.* 1970 as cited in EPA 1980, NCI 1978 as cited in HHS 1996). There are some indications that endrin may have genotoxic effects, including increased DNA damage in hepatocytes due to oxidative injury (Bagchi *et al.* 1992a, 1993a,1993c as cited in HHS 1996; Hassoun *et al.* 1993 as cited in HHS 1996). However, most studies suggest that endrin is not carcinogenic (NCI 1978 as cited in HHS 1996; EPA 1980h).

There is limited data available regarding chronic effects of water-borne exposure to endrin in salmonids (Tables 2.6.2.1.3.5 to 2.6.2.1.3.9). In other species, adverse effects have not been reported unless water concentrations were more than 10 times the proposed chronic criterion of 0.036 µg/L (*e.g.*, Hansen *et al.* 1977, Jarvenen and Tyo 1978, Jarvenin *et al.* 1988). However, there are some data available on tissue concentrations of endrin associated with a variety of sublethal adverse effects in rainbow trout, which is the non-anadromous form of steelhead trout. Grant and Mehrle (1973) determined that tissue levels associated with effects in rainbow trout included: alteration of plasma parameters, suppression of cortisol secretion and inhibited carbohydrate metabolism after a swim challenge at 0.01 mg/kg to 0.02 mg/kg, hyperexcitability at 0.12 mg/kg, and hyperglycemia and reduction in growth at 0.12 mg/kg to 0.22 mg/kg. No effects were seen at tissue concentrations at or below 0.00025 mg/kg (Grant and Mehrle 1973).

Laboratory exposure studies also suggest that exposure to endrin may affect immune responsiveness in rainbow trout. Bennet and Wolke (1987a,b) exposed rainbow trout for 30 days to sublethal concentrations of endrin (0.12 µg/L to 0.15 µg/L) and found that several immune responses (migration inhibition factor assay (MIF), plaque forming cell assay (PFC), and serum agglutination titres (SAG) were inhibited when fish were exposed to the bacterium *Yersinia ruckeri* O-antigen. Serum cortisol concentrations were found to be significantly elevated in endrin-exposed fish. Fish receiving cortisol in the di*et al.*so showed reduced immune responsiveness, suggesting that elevated serum cortisol concentration obtained in endrin-exposed fish has a central role in repression of the immune response. Fish were exposed to only one dose of endrin in this experiment, however, so there is no information on the threshold endrin concentration for immunosuppressive effects. Exposure to water-borne endrin from agricultural runoff has been associated with an increased prevalence of parasitic infections in cultured sand goby (Supamataya 1988), but the fish were also exposed at the same time to dieldrin, DDTs, and possibly stress due to changes in dissolved oxygen and water temperature.

Singh and Singh (1980) reported total lipid levels in ovary and liver and cholesterol concentrations in ovary, liver and blood serum in the fossil catfish *Heteropneustes fossilis* after 4 weeks exposure to endrin at concentrations of 0.0006 µg/L and 0.008 µg/L during different phases of the annual reproductive cycle. Even the lower concentrations of endrin induced a significant decrease in liver lipid during the preparatory and late post-spawning phases. An appreciable increase in ovarian cholesterol was noticed during the pre-spawning and spawning. Serum cholesterol values demonstrated a significant increase in the preparatory and late post-spawning phases after exposure to endrin at all concentrations. This study suggests that exposure to endrin concentrations below the proposed chronic criterion could affect lipid and cholesterol balance in gravid salmon.

Studies show that endrin is bioaccumulated significantly by fish and other aquatic organisms (ASTDR 1996, EPA 1980h, Metcalf *et al.* 1973). Although specific BCFs are not available for salmonids, for other fish they range from 1,640 to 15,000 (EPA 1980h, Hansen *et al.* 1977). Endrin is also taken up by invertebrate prey species of salmonids, although bioconcentration factors are typically lower than those for fish. Anderson and DeFoe (1980) report pesticide accumulation in stoneflies, an invertebrate prey species, of 350 to 1150 times greater than the water concentrations after a 28-day exposure. However, biomagnification of endrin with increasing trophic level is less than that for some other chlorinated pesticides (Leblanc 1995, Metcalf *et al.* 1973).

Endrin in the diet may be an important source of uptake for fish species. Jarvinen and Tyo (1978) found that endrin in the food at a concentration of 0.63 mg/kg significantly reduced survival of fathead minnows in whole life cycle exposure tests, and residues contributed by food-borne endrin appeared to be additive to those contributed by water. Based on available BCF estimates for endrin, however, prey items would not accumulate endrin at this level under the proposed criterion.

Because endrin is no longer in use in the United States, the major source of this compound will be not through point source discharges into surface water bodies, but from repositories of the contaminant that are persistent in sediments. This means that endrin can occur through the water column, through direct contact with sediments, or through the diet. Thus, studies evaluating the effects of water-borne exposure alone are likely to underestimate actual exposure of organisms in the field.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for endrin is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Invertebrates tend to be more tolerant of endrin than fishes. Anderson and DeFoe (1980) exposed stoneflies, caddis-flies, isopods, and snails to endrin in a flowing-water test system for 28 days, increased mortality was observed at concentration in the 30,000 µg/L to 150,000 µg/L range. These values are at least two orders of magnitude above the acute criterion and at least four orders of magnitude above the chronic criterion. However, the available information is limited and may not account for exposure through other routes of exposure, such as sediments, or other invertebrate taxa.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Endrin. The available evidence for endrin indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), cellular trauma (low intensity), physiological trauma (low intensity), and reproductive failure (low intensity).

2.6.2.1.4 Heptachlor Epoxide

Heptachlor Criteria. The proposed acute and chronic criteria for heptachlor are 0.52 µg/L and 0.0038 µg/L, respectively.

Tables 2.6.2.1.4.1 through 2.6.2.1.4.3 report toxicity data from the ECOTOX database for freshwater heptachlor, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.4.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater heptachlor epoxide.

Criterion Freshwater Heptachlor Epoxide		**Data Set ECOTOX**
Criterion Concentration Acute 0.52 Micrograms Liter^{-1}	**Temperature 13° Celsius**	**Arithmetic Mean 14.7**
Criterion Concentration Chronic 0.0038 Micrograms Liter^{-1}	**Hardness 44 mg/L $CaCO_3$**	**Geometric Mean 13.6**
Endpoint/Effect LC_{50}/Mortality	**pH 7.1**	**Harmonic Mean 12.3**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
6.7	0.8G	96H
16	1.2G	96H
16	1.2G	96H
20	1.2G	96H

Table 2.6.2.1.4.2 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater heptachlor epoxide.

Criterion Freshwater Heptachlor Epoxide		**Data Set BE**
Criterion Concentration Acute 0.52 Micrograms Liter^{-1}	**Temperature 13° Celsius**	**Arithmetic Mean 0.5**
Criterion Concentration Chronic 0.0038 Micrograms Liter^{-1}	**Hardness 44 mg/L $CaCO_3$**	**Geometric Mean 0.47**
Endpoint/Effect NOEC	**pH 7.1**	**Harmonic Mean 0.44**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.25		96H
0.46		96H
0.47		96H
0.53		96H
0.81		96H

Heptachlor Epoxide Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the

criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to heptachlor epoxide, NMFS added an additional step to its analysis for heptachlor epoxide to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.52 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.4.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.4.1, predicts a magnitude of effect ranging from a low of an $LC_{1.3}$ at a concentration of 20 µg/L to a high of an LC_4 at a concentration of 6.7 µg/L. In other words, the acute criterion of 0.52 µg/L has an equivalent toxicity potential predicted to kill 1.3 percent to 4 percent, with a median toxicity potential of an $LC_{1.6}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, the available evidence for heptachlor epoxide indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute toxic effects, but may not suffer chronic toxic effects.

Sublethal Effects. Heptachlor is an organochlorine cyclodiene insecticide first isolated from technical chlordane in 1946 (ATSDR 1993). During the 1960s and 1970s, it was commonly used for crop pest control and by exterminators and home owners to kill termites. In 1976, it was prohibited from home and agricultural use, although commercial applications to control insects continued. In 1988, its use for termite control was banned, and currently its only permitted

commercial use in the United States is fire ant control in power transformers (ATSDR 1993, Leber and Benya 1994 as cited in EPA 2008).

The principal metabolite of heptachlor is heptachlor epoxide, an oxidation product formed by many plant and animal species and through breakdown of heptachlor in the environment. The epoxide degrades more slowly and, as a result, is more persistent than heptachlor. Both heptachlor and heptachlor epoxide adsorb strongly to sediments, and both are bioconcentrated in terrestrial and aquatic organisms (EPA 1980i, ATSDR 1993).

In fishes heptachlor is readily taken up through the skin, lungs or gills, and gastrointestinal tract (ATSDR 1993). Once absorbed, it is distributed systemically and moves into body fat and is readily converted to its most persistent and toxic metabolite, heptachlor epoxide, in mammalian livers (Smith 1991, ATSDR 1993). Heptachlor is also metabolized to some extent by fish, although most evidence points to it being stored in the body predominantly as heptachlor rather than heptachlor epoxide (Feroz and Khan 1979).

Heptachlor and heptachlor epoxide are considered highly to moderately toxic to mammals, birds, and fish. The primary adverse health effects associated with acute exposure are central nervous system and liver effects (Smith 1991, ATSDR 1993, Akay and Alp 1981, Buck *et al.* 1959). Chronic exposure to heptachlor may cause some of the same neurological effects as acute exposure. An increased prevalence of neurological symptoms in humans has been associated with environmental exposure to heptachlor in epidemiological studies (Dayal *et al.* 1995), and in laboratory exposure where effects were noted on functional observational ability and motor activity (Moser *et al.* 1995). There is also evidence from epidemiological and laboratory studies that heptachlor alters the expression and function of dopamine transporters (Miller *et al.* 1999). Heptachlor may also affect immune function by inhibiting normal chemotactic responses of neutrophils and monocytes (Miyagi *et al.* 1998) or promoting necrosis of lymphocytes in the spleen and thymus (Berman *et al.* 1995).

Heptachlor does not appear to be a primary carcinogen, and laboratory tests indicate that neither heptachlor nor heptachlor epoxide are mutagenic (WHO 1984, ATSDR 1993). Heptachlor toxicity can be influenced by the presence of other compounds in the environment, but its interactions with other contaminants have not been well-studied.

As part of our data search, NMFS did not find any chronic toxicity data on salmonid fishes exposed to heptachlor epoxide, therefore we used the available toxicity for fishes as an surrogate for potential adverse effects on listed species considered in this opinion. Carr *et al.* (1999) reported that in channel catfish, heptachlor epoxides, and to a lesser extent heptachlor, bind to the gamma-aminobutyric acid (GABA) receptor and may thus suppress the activity of inhibitory neurons in the central nervous system. However, because this was an in vitro study, the exposure concentrations associated with this effect in live animals are not clear. Hiltibran (1982) investigated the effects heptachlor on the metal-ion-activated hydrolysis of ATP by liver mitochondria in by bluegill (*Lepomis macrochirus*) and found that it significantly inhibited ATP hydrolysis in an in-vitro assay. The lowest effective concentration was 0.00056 g/ml of reaction medium, but how that would compare to water concentrations affecting a live animal is not clear.

Chronic toxicity data are correspondingly lacking for evaluating the protectiveness of the chronic criterion for salmonids. Exposure studies conducted with other species generally report effects at concentrations well above the proposed chronic criterion. For example, a study conducted on fathead minnow (Macek *et al.* 1976) showed 100% mortality after 60 days at 1.84 µg/L, with effects on sublethal endpoints at 0.86 µg/L. Similarly, Goodman *et al.* (1976) found effects of heptachlor on growth and survival of embryos and fry of the saltwater sheepshead minnow to occur when heptachlor concentrations exceeded 1.2 µg/L. Hansen and Parrish (1977) tested the chronic toxicity of heptachlor to sheepshead minnow in an 18-week partial life cycle exposure begun with juveniles, and observed decreased embryo production at 0.71 µg/L, but dose-response relationships were not consistent for this study so the data may not be accurate. The histological studies revealed conspicuous pathological changes in the liver. Other studies with non-salmonids report pathological effects on the liver and kidney, altered enzyme levels, inhibited fin regeneration, and mortality at higher concentrations (3 µg/L to 70 µg/L) with exposures ranging from 5 to 60 days (EPA 1980g, Azharbig *et al.* 1990, Rao *et al.* 1980).

In contrast to studies involving strictly water-borne exposure, other evidence suggests that adverse effects may occur when tissue concentrations are below the 0.34 mg/kg limit used to develop the chronic criterion. For example, Bishop *et al.* (1995) reported increased rearing mortality with heptachlor concentrations of 0.0279 mg/kg in Chinook salmon eggs. However, this was a field study, concentrations were measured in the eggs versus whole body tissues, and other contaminants may have been present. Tests with other species also suggest that some effects could occur at tissue residue levels in the 0.016 mg/kg to 0.3 mg/kg range. In spot (*Leistomus xantharus*), tissue concentrations of 0.654 mg/kg were associated with 25% mortality in test fish, and there are reports of increased long-term mortality at concentrations as low as 0.022 mg/kg in sheepshead minnow and 0.01 mg/kg in spot (Schimmel *et al.* 1976). It should be noted that there are some problems with analyses on which fish tissue heptachlor concentrations associated with the chronic criterion were based, particularly with respect to uncertainty about the applicability of a standardized BCF of 5,220 to salmonids.

Heptachlor is lipophilic, log K_{ow} of 6.26 (Karickhoff and Long 1995 as cited in BE), bioconcentrates and bioaccumulates in fish, animals, and milk (EPA 1999b as cited in BE). Heptachlor epoxide, log K_{ow} of 5.00 (Karickhoff and Long 1995 as cited in BE), would likewise be expected to bioconcentrate and bioaccumulate. Toxicity of heptachlor may be altered by a number of factors including temperature, duration of exposure (Johnson and Finley 1980), and presence of mixtures. Heptachlor is readily taken up in fish through the skin, lungs, gills, and gastrointestinal tract (ATSDR 1993). Heptachlor and its primary metabolite are considered to be moderately to highly toxic to fish (ATSDR 1993). Effects of heptachlor toxicity to freshwater organisms include reduced growth, inhibited ATPase activity, and reduced survival (EPA 1999b as cited in BE).

Both heptachlor and heptachlor epoxide have been shown to bioconcentrate in aquatic organisms such as fish, mollusks, insects, plankton, and algae (ATSDR 1989). They have been found in the fat of fish, mollusks, and other aquatic species at concentrations of 200 to 37,000 times the concentration of heptachlor in the surrounding waters (WHO 1984, ATSDR 1989). A wide range of BCFs have been determined in laboratory studies using fish (EPA 1980i). No BCF values are available for salmonids, but values for fathead minnow range from 9,500 to 14,400 (Veith *et al.*

1979, EPA 1980i), and Goodman *et al.* (1976) reported average bioconcentration factors for heptachlor of 3,600 for sheepshead minnow. Because heptachlor is no longer in use in the United States, except for selected special applications, the major source of this compound will be not through point source discharges into surface water bodies, but from repositories of the contaminant that are persistent in sediments. This means that heptachlor and heptachlor epoxide will be taken up not only through the water column, but also through direct contact with sediments or through the diet. Thus, studies evaluating the effects of water-borne exposure alone are likely to under-estimate actual exposure of organisms in the field.

If it is assumed that sediments are a major source of heptachlor, the sediment-heptachlor concentrations that would result in heptachlor concentrations in the water column at or below the criteria are: For heptachlor, $\log_{10}(K_{ow}) = 6.26$, $\log_{10}(K_{oc}) = 6.15$, and $F_{cv} = 0.0038$, resulting in $SQC_{oc} = 5.37$ mg/kg organic carbon[7]. This would mean that for sediment total organic carbon (TOC) levels of 1% to 5% percent, the sediment heptachlor concentrations would range from 54 ng/g to 269 ng/g sediment. These levels bracket the sediment screening guideline of 10 ng/g dry wet established by the U.S. Corps of Engineers (Corps) for in-water disposal of dredged sediment (Corps 1998), and are above the interim Canadian freshwater sediment guidelines of 0.6 ng/g to 2.74 ng/g dry wet sediment. The higher of these values is a probable effect level, based on spiked sediment toxicity testing and associations between field data and biological effects (CCREM 2001b). This indicates a potential for adverse effects on aquatic life.

Because there has been very little research on the toxicity of sediment-associated heptachlor to salmonids, the sediment concentrations that cause adverse effects are not well defined. The BSAFs have not been determined for salmonids, so it is difficult to estimate the likely tissue concentrations of heptachlor that would be associated with sediment heptachlor concentrations permissible under the proposed criteria.

Sublethal Effects Summary. Although the data regarding sublethal effects on fishes exposed to endosulfan-alpha and endosulfan-beta is available, there are no chronic toxicity studies available for fishes subject to this consultation. If the mechanism and modes of actions are similar for fishes subject to this consultation to those described above, then fishes considered in this opinion may not be protected from chronic toxic effects.

Toxicity to Food Organisms. Heptachlor epoxide is acutely toxic to freshwater aquatic invertebrates at concentrations comparable to those that are lethal to fish (Johnson and Finley 1980). Reported LC_{50} values for freshwater invertebrate species have include 0.9 to 2.8 µg/L for stoneflies (Sanders and Cope 1968), 29 mg/kg to 47 mg/kg for gammarid amphipods (Sanders 1969, 1972), and 42 µg/L to 78 µg/L for *daphnid cladocerans* (Macek *et al.* 1976, Sanders and Cope 1966). These values were derived from static tests in which heptachlor concentrations were unmeasured. Tests using saltwater species using flow-through tests yielded lower LC_{50} values for grass shrimp and pink shrimp (0.03 µg/L to 0.11 µg/L) than static tests for shrimp and crayfish (1.8 µg/L to 7.8 µg/L; Sanders 1972; Schimmel *et al.* 1976), suggesting that the static tests underestimate the toxicity of heptachlor to aquatic invertebrates.

[7] SQCoc SQC stands for sediment quality criteria and oc stands for organic carbon content.

Sublethal effects of acute exposure have also been reported for some invertebrate species at concentrations close to the proposed criteria, although these studies were not conducted in salmonid prey. When the criteria for heptachlor were developed (EPA 1980i), no data were available on chronic effects of this compound on invertebrate species, and little additional information has been generated since that time. Lowest heptachlor concentrations at which effects are reported have been above 0.01 µg/L. For example, a concentration of 0.04 µg/L was associated with increased mortality in the pink shrimp, *Penaeus duoraum* (Schimmel *et al.* 1976), which is well above the proposed chronic criterion.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Heptachlor Epoxide. The available evidence for heptachlor epoxide indicates that listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects including mortality (moderate intensity). As part of our data search, NMFS did not find any chronic toxicity data on salmonid fishes exposed to heptachlor epoxide. However, the NOEC analysis suggests that listed species exposed to waters equal to the chronic criterion concentration will suffer chronic toxic effects (low intensity). Furthermore, if the mechanism and modes of actions are similar for fishes subject to this consultation to those described above in the *Sublethal Effects* analysis, then fishes considered in this opinion will suffer sublethal effects (low intensity).

2.6.2.1.5 Lindane (gamma-BHC)

Lindane Criteria. The proposed acute criterion for lindane is 0.95 µg/L.

Tables 2.6.2.1.5.1 through 2.6.2.1.5.4 report toxicity data from the ECOTOX database for freshwater lindane, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.5.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater lindane.

Criterion Freshwater Lindane		**Data Set ECOTOX**
Criterion Concentration Acute 0.95 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 757**
	Hardness 40-314 mg/L $CaCO_3$	**Geometric Mean 17**
Endpoint/Effect LC_{50}/Mortality	**pH 6.8-8.1**	**Harmonic Mean 0.04**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.0022	312 G	96D
0.0022	175-312 G	96D
0.019	175 G	24D
0.019	183 G	96D
0.019	277 G	24D
0.019	284 G	96D
0.019	262 G	24D
0.019	288 G	48D
1	NR	96H
16	1.1G	96H
16	1G	24H
18	FINGERLING	96H
19	0.6G	96H
20	1.1G	96H
20	1G	24H
20	1G	24H
22	FRY, 3.0 CM	96H
22	0.5G	96H
22	FRY, 3.0 CM	96H
23	FRY,3 CM	96H
23	FRY, 3.0 CM	96H
24	0.7G	96H
24	JUVENILE, 0.69 G	96H
27	1G	96H
27	1G	96H
27	1G	96H
29	1G	96H
30	FRY,3 CM	96H
30	YEARLING,107.8 G,22.4 CM	96H
30	FRY,3 CM	24H
32	1G	96H

Criterion Freshwater Lindane		Data Set ECOTOX
Criterion Concentration Acute **0.95 Micrograms Liter^{-1}**	**Temperature** **12-20° Celsius**	**Arithmetic Mean** **757**
	Hardness **40-314 mg/L $CaCO_3$**	**Geometric Mean** **17**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.8-8.1**	**Harmonic Mean** **0.04**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
32.7	5.2 G	96H
34	1G	96H
34	1G	24H
37	YEARLING,107.8 G,22.4 CM	48H
37	FRY,3 CM	24H
37	FRY, 3.0 CM	96H
38	51-79 MM, 3.2 G	96H
38	51-79 MM, 3.2 G	24H
39	JUVENILE, 0.69 G	48H
39	51-79 MM, 3.2 G	96H
40	51-114 MM, 1.45-5 G	96H
41	51-79 MM, 3.2 G	24H
42	51-79 MM, 3.2 G	96H
42	51-114 MM, 1.45-5 G	96H
42	51-114 MM, 1.45-5 G	24H
42	51-114 MM, 1.45-5 G	48H
44	1G	96H
50	57-76 MM, 2.7-4.1 G	96H
50	ADULT, 175-250 G	48H
56	YEARLING,107.8 G,22.4 CM	24H
56	51-114 MM, 1.45-5 G	72H
56	86 D, 77 MM	48H
500	YOUNG, 9-11 CM	24H
1000	YOLK SAC FRY, STAGE 30-31, 33-34/	11D
1000	ALEVIN	24H
1000	YOLK SAC FRY, STAGE 30-31, 33-34/	96D
1000	8 H POST HATCH,FRY	24D
10000	5-10 CM	72H
10000	5-10 CM	96H
10000	5-10 CM	96H

Table 2.6.2.1.5.2 Mortality toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater lindane.

Criterion Freshwater Lindane		Data Set ECOTOX
Criterion Concentration Acute 0.95 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 19**
	Hardness 40-314 mg/L $CaCO_3$	**Geometric Mean 13**
Endpoint/Effect Mortality	**pH 6.8-8.1**	**Harmonic Mean 5.8**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1	YEARLING	1D
1	YEARLING	24D
4.1	YEARLING	72D
8.8	YEARLING	24D
16	1.1G	NR
16.6	YEARLING	24D
18	FINGERLING	72H
19	5.2 G	24D
19	FINGERLING	2H
20	1.1G	24H
22	0.5G	25H
24	0.7G	25H
26	0.5G	NR
30	1 G, 3.0-4.0 CM, JUVENILE	24H
30	1.1G	24H
30	0.7G	72H
32.7	5.2 G	24H

Table 2.6.2.1.5.3 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater lindane.

Criterion Freshwater Lindane		Data Set ECOTOX
Criterion Concentration Acute 0.95 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 10000**
	Hardness 40-314 mg/L $CaCO_3$	**Geometric Mean 10000**
Endpoint/Effect NOEC/Mortality	**pH 6.8-8.1**	**Harmonic Mean 10000**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
10000	5-10 CM	3H

Table 2.6.2.1.5.4 Physiological toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater lindane.

Criterion Freshwater Lindane		**Data Set ECOTOX**
Criterion Concentration Acute 0.95 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 16**
	Hardness 40-314 mg/L $CaCO_3$	**Geometric Mean 7.9**
Endpoint/Effect Physiological	**pH 6.8-8.1**	**Harmonic Mean 3.9**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
2.1	YEARLING	2D
30	1.1G	NR

Lindane Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to lindane, NMFS added an additional step to its analysis for lindane to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.95 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.5.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.5.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 10,000 µg/L to a high of an LC_{100} at a concentration of 0.0022 µg/L. In other words, the acute criterion of 0.95 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{1.5}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for lindane, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute criterion concentration for lindane, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects.

Sublethal Effects. Lindane is one of the few chlorinated hydrocarbon insecticides considered in the proposed action that is still in use for pharmaceutical products (EPA 2002). It is used primarily for treating wood-inhabiting beetles and seeds, and in a more restricted manner

for soil treatment and as an insecticide on fruit and vegetable crops, timber, and ornamental plants. It is also used as a dip for fleas and lice on pets, and in lotions, creams, and shampoos for the control of lice and mites in humans. It is rated as a "moderately toxic (toxicity class II)" compound by EPA. Labels for products containing it must bear warning labels, and some formulations are classified as RUPs that may only be purchased and used by certified pesticide applicators. Lindane is no longer manufactured, but is still formulated, in the United States, and aerial application of the pesticide has been prohibited. Lindane has been listed as a pollutant of concern to EPA's Great Waters Program due to its persistence in the environment, potential to bioaccumulate, and toxicity to humans and the environment.

Lindane has been sold under a number of trade names, including gamma-Hexachlorocyclohexane, Exagamma, Forlin, Gallogamma, Gammaphex, Inexit, Kwell, Lindagranox, Lindaterra, Lovigram, and Silvanol . Technical-grade lindane is comprised of the gamma-isomer of hexachlorocyclohexane (HCH). Five other isomers (molecules with a unique structural arrangement, but identical chemical formulas) of HCH are commonly found in technical lindane, but the gamma-isomer is the predominant one, comprising at least 99% of the mixture of isomers.

Lindane is moderately water soluble and may accumulate in sediments. It is relatively persistent and experiences significant degradation only under anaerobic conditions. Lindane is readily absorbed into the body, but in mammals is metabolized to some extent through conversion to tri- and tetra-chlorophenols, and conjugation with sulfates or glucuronides. Other pathways involve the ultimate formation of mercapturates. These water soluble end-products are eliminated via the urine (Smith 1991). Of the isomers, g-HCH is stored to the greatest extent in fat (Smith 1991).

Few chronic toxicity data are available for salmonids exposed to lindane in the water column. Macek *et al.* (1976) exposed brook trout for 261 days to 16.6 µg/L lindane. While survival was not affected, a reduction was observed in fish weight and length. Some disruption in reproductive activity was also recorded during the same experiment (Macek *et al.* 1976). Mendiola *et al.* (1981) determined decreased efficiency of protein utilization in rainbow trout exposed to lindane at concentrations of 1 µg/L to 10 µg/L for 21 days.

Some additional information is available on the effects of lindane associated with specific measured tissue residues in test fish. For example, in immature brook trout, Macek *et al.* (1976) found that growth rates were decreased, and observed abnormal spawning behavior in females, when muscle tissue concentrations were 1.2 mg/kg. However, there was no effect on survival. Other fish species also show effects of lindane at relatively low tissue concentrations. For example, in the gudgeon (*Gobio gobio*) the lowest tissue concentration at which a significant increase in mortality could be observed within 96 hours was 0.l9 mg/kg in muscle (Marcelle and Thorne 1983). Similarly, in bluegill, the proposed no observable effect level (NOEL) for growth and mortality was 0.297 mg/kg (Macek *et al.* 1976). For other fish species, adverse biological effects occur at somewhat higher levels. Macek *et al.* (1976) observed decreased growth and increased mortality of fathead minnow at a concentration of 9.53 mg/kg in the carcass. In pinfish, the effective dose $(ED)_{50}$ for growth effects was 5.22 mg/kg (Schimmel *et al.* 1976).

The likely tissue concentrations of lindane in fish exposed to the concentrations of lindane in the water column specified by the criteria can be calculated from EPA's estimated BCFs for lindane. Multiplying the proposed chronic criterion by the geometric mean of BCF values for lindane of 1400 (EPA 1980q) and a percent lipid of 15% (default value for freshwater fish) results in an estimated maximum allowable tissue concentration of 1.68 mg/kg lindane. For lower lipid values (5% to 10%) the values would be on the order of 0.56 mg/kg to 1.12 mg/kg. It should be noted that the normalized BCF value is based primarily on data for fathead and sheepshead minnow, not on studies with salmonids, so it may not reflect uptake in the species of concern. Also, because these BCFs were determined in the laboratory, they may underestimate lindane uptake by animals in the field. Assuming that the BCF values are in a reasonable range, it appears that tissue concentrations of lindane associated with biological effects (Macek *et al.* 1976, Marcelle and Thorne 1983) are relatively close to those predicted based on the proposed chronic criterion (1.68 mg/kg).

Some studies have also been conducted in which lindane was administered through feeding or injection studies. For example, Dunier *et al.* (1994, 1995) report that lindane modified non-specific immune responses in rainbow trout fed lindane for 30 days at a dose of 1 mg/kg. Aldegunde *et al.* (1999) observed lower body weights, increased serum cortisol levels and changes in the serotonergic brain activity after 18 days in rainbow trout implanted with 0.005 mg/kg body weight of lindane in coconut oil. These studies suggest the potential for sublethal effects on growth, metabolism, and immune function at tissue concentrations comparable or lower than those associated with the water quality criteria, but more information on the uptake ratio of lindane would be needed to evaluate these studies.

Lindane will accumulate slightly in fish and shellfish. Uptake of lindane by aquatic organisms is influenced by a number of environmental and water quality factors, including concentrations of organic particulate matter in the water column, turbidity, pH, and season of the year. Residue concentrations may also vary considerably between fish species. However, biological accumulation and persistence of lindane are low when compared to compounds such as DDT or dieldrin (Wilson 1965, Gakstatter and Weiss 1967). Lindane bioconcentrates to some extent in aquatic organisms such as fish, mollusks, insects, plankton, and algae (ATSDR 1989). Lindane has been found in the fat of fish, mollusks, and other aquatic species at concentrations up to 1400 times the concentration in the surrounding waters (WHO 1984, ATSDR 1989, Ulman 1972). Bioconcentration factors determined in laboratory studies with fish have ranged from 35 to 486, with the 486 value determined for rainbow trout (EPA 1980q).

Because lindane use in the United States is limited, one of the sources of this compound will be from repositories of the contaminant that are persistent in sediments. These means that lindane will be taken up not only through the water column, but also through direct contact with sediments or through the diet. Thus, studies evaluating the effects of water-borne exposure alone are likely to under estimate actual exposure of organisms in the field. However, because the value of the octanol/water partitioning coefficient of lindane ($\log_{10}(K_{ow}) = 3.3$) is relatively low in comparison to compounds such as DDTs and PCBs, adsorption and accumulation in sediments is also generally lower.

The quantity and quality of available data raise concerns about the validity of the proposed acute criteria. Based on testing procedures and results from available studies that are not specific to listed species considered in this opinion and their prey, it is possible that mortality could result to both listed species and invertebrate prey under the proposed acute criterion, and adverse effects in listed fish, such as increased long-term mortality, growth reduction, increased cortisol levels, and changes in immune function. There are also a few studies suggesting that increased long-term mortality or sublethal effects could take place at lindane tissue concentrations close to those that might be expected in fish exposed to lindane at levels allowed under the acute aquatic life criteria.

Sublethal Effects Summary. The available evidence indicates that the acute criterion for lindane is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Available data on the acute toxicity of Lindane to aquatic invertebrates suggest that the proposed acute criterion of 0.95 µg/L may be protective of most types of salmonid invertebrate prey. Reported 96-hour LC_{50} values are on the order of approximately 5 to 7 times the criteria, including 4.5 µg/L for stoneflies *Pteronarcys*, and 6.3 µg/L for mysids (*Mysidopsis bahia*; Johnson and Finley 1980). For other prey species, such as Daphnia, LC_{50} values are substantially higher, *e.g.*, 460 µg/L to1460 µg/L (Fernando *et al.* 1995), or as high as 20,000 µg/L for rotifers (Janssen *et al.*1994). For amphipods, reported LC_{50} values have ranged from 5 µg/L to 80 µg/L (*Gammarus pulix*, McLoughlin *et al.* 2000, Abel 1980, Stephenson 1983, Taylor *et al.* 1991; *Gammarus lacutris* and *G. fasciatus*, Sanders 1972, *Hyalella azteca*, Blockwell *et al.* 1998).

Only one study was found that reported effects on aquatic macroinvertebrates at lindane concentrations that were below the chronic criterion; Schulz and Liess (1995) reported reduced emergence of caddisfly larvae after 90 days of exposure to concentrations of lindane as low as 0.0001 µg/L. However, most studies of the chronic effects of lindane exposure on aquatic invertebrates have reported effects occurring at levels that ranged from 2 to 28 times the proposed criterion of 0.95 µg/L. For example, for the amphipod, *Hyalella azteca*, Blockwell *et al.* (1998) reported 240-hour LC_{50}s of 26.9 µg/L and 9.8 µg/L for adults and neonates, respectively. In the amphipod *Gammarus pulix*, growth was reduced after a 14 day exposure to concentrations between 2.7 µg/L and 6.1 µg/L (Blockwell *et al.* 1996). Taylor *et al.* (1998) reported alterations in haeme biosynthesis in *Gammarus pulex* after a 240 hour exposure to lindane at 4.5 µg/L. Similarly, in mesocosm experiments involving exposures of 2 to 4 weeks, some zooplankton species, such as copepod and cyclopod nauplii and midge larvae, experienced significant mortality at lindane concentrations in the 2 µg/L to 12 µg/L range (Fliedner and Klein 1996, Peither *et al.* 1996). In contrast, effects were not observed on survival, reproduction and growth of *Daphnia magna* after 21 days of exposure until concentrations were 250 µg/L or higher (Ferrando *et al.*1995). Available data suggest that the proposed chronic criterion for lindane could adversely affect selected sensitive life stages of certain salmonid prey species.

Summary on Toxicity to Food Organisms. The available evidence indicates that the acute criterion is likely to adversely affect invertebrate productivity and abundance.

Summary of Effects: Lindane. The available evidence for lindane indicates that listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects, *i.e.*, mortality (moderately-high-intensity).

2.6.2.1.6 Pentachlorophenol (PCP)

Pentachlorophenol Criteria. To determine the freshwater criteria as a function of pH the following equation is used:

CMC = exp (1.005 x pH – 4.83 (µg/L)
CCC = exp (1.005 x pH – 5.29 (µg/L)

At a pH of 7.8, the corresponding proposed criteria are 19 µg/L and 15 µg/L for acute and chronic criteria, respectively.

Tables 2.6.2.1.6.1 through 2.6.2.1.6.3 report toxicity data from the ECOTOX database for freshwater pentachlorophenol, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.6.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater pentachlorophenol.

Criterion Freshwater Pentachlorophenol		**Data Set ECOTOX pH-adjusted**
Criterion Concentration Acute 19 Micrograms Liter^{-1}	**Temperature 6-16.5° Celsius**	**Arithmetic Mean 103**
Criterion Concentration Chronic 15 Micrograms Liter^{-1}	**Hardness 5-272 mg/L $CaCO_3$**	**Geometric Mean 87**
Endpoint/Effect LC_{50}/Mortality	**pH 5.7-8.19**	**Harmonic Mean 64**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
10	0.31 G	96H
11	1.3G	96H
11	1.3G	96H
11	1.3G	96H
32	YOLK-SAC FRY, 0.3G	96H
33	0.3G	96H
35	2.14 G, 5.80 CM	96H
36	1G	96H
41	2.14 G, 5.80 CM	96H
49	1 g	96H

Criterion Freshwater Pentachlorophenol		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute 19 Micrograms Liter^{-1}	Temperature 6-16.5° Celsius	Arithmetic Mean 103
Criterion Concentration Chronic 15 Micrograms Liter^{-1}	Hardness 5-272 mg/L $CaCO_3$	Geometric Mean 87
Endpoint/Effect LC_{50}/Mortality	pH 5.7-8.19	Harmonic Mean 64
Concentration Micrograms Liter^{-1}	Life-Stage	Duration
49	.81 g	96H
53	1 g	96H
54	0.68 G	96H
54	0.68 G	96H
55	1G	96H
56	1.90 G, 5.80 CM	96H
56	1.90 G, 5.80 CM	96H
58	1G	96H
60	1 g	96H
61	1G	96H
64	1.39 G, 4.84 CM	96H
66	1.39 G, 4.84 CM	96H
67	0+ PARR	96H
68	0+ PARR	96H
68	0+ PARR	96H
69	1 g	96H
70	FRY, 10 WK, 264 MG, 33 MM	96H
70	JUVENILE, 2.7 G	96H
71	FINGERLING, 1G	96H
72	1G	96H
72	YEARLING, UNDER YEARLING	96H
75	0+ PARR	96H
83	1.0 G, 32 MM	96H
84	1.31 G	96H
87	0+ PARR	96H
93	0+ PARR	96H
95	1.0 G, 32 MM	96H
102	4.61 G, 7.40 CM	96H
103	2.84 G, 5.98 CM	96H
103	0+ PARR	96H
107	4.61 G, 7.40 CM	96H

Criterion Freshwater Pentachlorophenol		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **19 Micrograms Liter^{-1}**	**Temperature** **6-16.5° Celsius**	**Arithmetic Mean** **103**
Criterion Concentration Chronic **15 Micrograms Liter^{-1}**	**Hardness** **5-272 mg/L $CaCO_3$**	**Geometric Mean** **87**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **5.7-8.19**	**Harmonic Mean** **64**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
107	0.87 G, 4.28 CM	96H
107	0.87 G, 4.28 CM	96H
107	0.62 G	96H
108	0+ PARR	96H
108	0+ PARR	96H
108	0.3-0.4 G FINGERLING	96H
110	2.84 G, 5.98 CM	96H
111	1.52 G, 5.24 CM	96H
114	2.48 G	96H
118	1.52 G, 5.24 CM	96H
118	0+ PARR	96H
118	0+ PARR	96H
118	0+ PARR	96H
121	2.2G	96H
122	0+ PARR	96H
124	0+ PARR	96H
124	0+ PARR	96H
127	ADULT, 18 MO, 218.0 MM, 101.0 G	152H
128	YOLK-SAC FRY	96H
129	0+ PARR	96H
132	1.38 G, 5.05 CM	96H
132	1.38 G, 5.05 CM	96H
132	0+ PARR	96H
132	0+ PARR	96H
133	0+ PARR	96H
135	1.9G	96H
136	ADULT, 18 MO, 218.0 MM, 101.0 G	96H
139	0+ PARR	96H
139	0+ PARR	96H
141	0+ PARR	96H
146	0+ PARR	96H

Criterion Freshwater Pentachlorophenol		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute 19 Micrograms Liter^{-1}	**Temperature 6-16.5° Celsius**	**Arithmetic Mean 103**
Criterion Concentration Chronic 15 Micrograms Liter^{-1}	**Hardness 5-272 mg/L $CaCO_3$**	**Geometric Mean 87**
Endpoint/Effect LC_{50}/Mortality	**pH 5.7-8.19**	**Harmonic Mean 64**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
156	0.71 G	96H
158	1.2-3.8 G, 4.6-6.4 CM, STD LENGTH	96H
161	0+ PARR	96H
166	0.46 G	96H
169	YOLK-SAC FRY	96H
174	SWIMUP FRY	96H
174	0.3G	96H
179	9G	96H
192	3.09 G, 6.3 CM	96H
220	1.2-7.9 G	96H
264	SWIMUP FRY, 0.5G	96H
316	EYED EGG	96H

Table 2.6.2.1.6.2 LC_{50} toxicity data for green sturgeon for freshwater pentachlorophenol.

Criterion Freshwater Pentachlorophenol		Data Set 4 pH-adjusted
Criterion Concentration Acute 19 Micrograms Liter^{-1}	**Temperature 22° Celsius**	**Arithmetic Mean 135**
Criterion Concentration Chronic 15 Micrograms Liter^{-1}	**Hardness 160-180 mg/L $CaCO_3$**	**Geometric Mean 134**
Endpoint/Effect LC_{50}	**pH 8.4**	**Harmonic Mean 134**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
149	JUVENILE	12H
121	JUVENILE	24H

Table 2.6.2.1.6.3 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater pentachlorophenol.

Criterion **Freshwater Pentachlorophenol**		**Data Set BE** **pH-adjusted**
Criterion Concentration Acute **19 Micrograms Liter^{-1}**	**Temperature** **6-16.5° Celsius**	**Arithmetic Mean** **26**
Criterion Concentration Chronic **15 Micrograms Liter^{-1}**	**Hardness** **5-272 mg/L $CaCO_3$**	**Geometric Mean** **21**
Endpoint/Effect **NOEC/Growth**	**pH** **7.22-7.54**	**Harmonic Mean** **16**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
6.27		
11.6	EGG	72D
12.8		
24		
25		
31		
31		
67		

Pentachlorophenol Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50}

data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to pentachlorophenol, NMFS added an additional step to its analysis for pentachlorophenol to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 19 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.6.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.6.1, predicts a magnitude of effect ranging from a low of an LC_3 at a concentration of 319 µg/L to a high of an LC_{95} at a concentration of 10 µg/L. In other words, the acute criterion of 19 µg/L has an equivalent toxicity potential predicted to kill 3 percent to 95 percent, with a median toxicity potential of an $LC_{0.09}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for pentachlorophenol, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for pentachlorophenol, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available

information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Pentachlorophenol (PCP) is a chlorinated hydrocarbon that is used primarily as an insecticide and fungicide, but also secondarily as an herbicide, molluscicide, and bactericide (Eisler 1989 as cited in EPA 2008). Technical grade PCP is approximately 86% pure and historically has been contaminated with dioxins and hexachlorobenzene. Pentachlorophenol does not occur naturally in the environment. It is produced by the chlorination of phenol. In pure form, it exists as colorless crystals and has a very sharp characteristic odor when hot. Impure pentachlorophenol is a dark gray to brown dust, beads, or flakes.

Pentachlorophenol rapidly degrades in air, on land, and in water. Pentachlorophenol is teratogenic but evidence of its mutagenic or carcinogenic properties is incomplete (Williams 1982 as cited in EPA 2008). It bioconcentrates, and bioaccumulates in predatory species (Eisler 1989 as cited in EPA 2008). Toxicity of PCP may be altered by a number of factors including pH, temperature, chemical composition (which congeners are present), organic matter, and presence of mixtures (Eisler 1989 as cited in EPA 2008). Toxicity of pure, reagent grade PCP is less than that of commercial PCP due to toxicity of some of the impurities present in commercial formulations (Cleveland *et al.* 1982). Many of the available toxicity tests have been conducted with reagent grade PCP and may thus underestimate toxic effects of commercial PCP releases into the environment. In general, fish are more sensitive to PCP than are other aquatic organisms (FWS 2000 as cited in EPA 2008). Coldwater species are generally more sensitive than warmwater species in acute lethal toxicity tests (EPA 1995 as cited in EPA 2008). Effects of PCP toxicity to algae include chlorosis inhibition, reduced cell numbers, reduced or inhibited growth, and reduced survival (Eisler 1989). Effects of PCP toxicity to freshwater invertebrates include reduced populations, reduced locomotion or immobilization, abnormal larvae development, reduced reproduction (decreased production of eggs or young), decrease in periphyton biomass, larval drift, and suppression of community metabolism in invertebrates (Eisler 1989 as cited in EPA 2008). Effects of PCP toxicity to freshwater fish include reduced growth, increased alevin mortality, reduced food conversion efficiency, reduced ability to capture and consume prey, fin erosion, cranial malformations, reduced activity, reduced egg survival, rapid swimming at the water surface and increased opercular movements, loss of balance, and reduced survival (Eisler 1989 as cited in EPA 2008).

Like other organic pollutants, PCP exhibits a tendency to be bioaccumulated by fish. Van den Heuvel *et al.* (1991) reported BCFs for rainbow trout exposed to PCP (pH 7.6) to be between 411 and 482. Similar values (350 to 764) were reported by Servizi *et al.* (1988) for pink salmon (*Oncorhynchus gorbuscha*) exposed to PCP at pH 7.75. Metabolism of PCP is relatively rapid in rainbow trout (McKim *et al.* 1986; Glickman *et al.* 1977), and this is likely true in other salmonids as well. Nevertheless, the elimination rate of this compound is sufficiently slow that it takes 11.7 days for tissue concentrations to reach 95% steady state (McKim *et al.* 1986). According to the data provided in McKim *et al.* (1986) a 96-hour exposure will produce tissue concentrations that are only 63% of steady state. Therefore, any assessment of the maximum

attainable tissue concentration and resulting biological response for a given exposure concentration must consider a longer time period (*e.g.*, 12 days) to reach that level. An estimate of the steady-state wet-weight BCF for salmonids is 4,600 using the octanol-water partition coefficient for PCP ($\log_{10}$ (K_{ow}) = 5). Bioaccumulation of PCP is pH dependant, because pH determines the proportions of ionized and unionized PCP, which is directly related to bioaccumulation potential. The ionic form of PCP is less likely to bioaccumulate in organisms in large part because it is less likely to be taken up in the first place (Spehar *et al.* 1985).

PCP has a strong propensity to associate with the organic carbon of sediment and the lipids of organisms, as represented by a relatively high value octanol-water partition coefficient ($\log_{10}$ (K_{ow}) = 5; Eisler 1989). One of the primary toxicity mechanisms of PCP is inhibition of oxidative phosphorylation, which causes a decrease in the production of ATP in plants and animals. One consequence of this impairment is increased basal metabolism, resulting in increased oxygen consumption and high fat utilization. The effects of PCP may reduce the availability of energy for maintenance and growth, thus reducing survival of larval fish and ability of prey to escape from a predator (Brown *et al.* 1985, Johansen *et al.* 1985, Eisler 1989). PCP is known to cause several types of adverse effects in animals including dysfunction of the reproductive, nervous, and immune systems, hormone alterations, and impaired growth. In general, fish growth and behavioral endpoints have been shown to be sensitive indicators of PCP exposure (Webb and Brett 1973, Hodson and Blunt 1981, Dominquez and Chapman 1984, Brown *et al.* 1985).

The criteria for pentachlorophenol established by the EPA are pH dependent. In general, the toxicity of PCP increases with decreasing pH. At pH 4.74, half of PCP molecules are ionized (anions) and half are non-ionized. At pH 6, the ratio between the ionic and non-ionized forms is 18 (*i.e.*, the concentration of the ionized form is 18 times greater than the non-ionized form), and at pH 7 the ratio is 182. Studies have concluded that the ionic form of PCP is less toxic, primarily because it is less likely to cross membranes (Spehar *et al.* 1985). A correction factor is therefore needed for assessing bioaccumulation and toxicity to account for the effect of pH on the speciation of PCP.

Iwama *et al.* (1986) exposed juvenile Chinook salmon to 3.9 µg/L of PCP and found altered blood urea and glucose levels. Nagler *et al.* (1986) found oocyte impairment at 22 µg/L (pH 7.5). There is also evidence of sublethal effects occurring during relatively long-term exposures to PCP concentrations that are below the chronic criterion. Webb and Brett (1973) determined that juvenile sockeye salmon experienced decreased growth rates and food conversion efficiencies at PCP EC_{50}s of approximately 1.8 µg/L at pH 6.8 when exposed for 2 to 8 weeks. Hodson and Blunt (1981) also observed reduced weight, growth rate, and biomass in rainbow trout exposed over 4 weeks from embryo to fry stages. Mortality of rainbow trout eggs has also been observed at levels below the PCP chronic criterion when dissolved oxygen fell to low levels of 3 mg/L to 5 mg/L (Chapman and Shumway 1978).

Little *et al.* (1990) examined post-exposure behavioral effects in rainbow trout at exposure concentrations that were from 10 to 100 times less than the acute criterion of 19 µg/L. A statistically significant reduction in the percent survival of trout that were preyed on by largemouth bass occurred at an exposure concentration of 0.2 µg/L. A similar response may be

expected for salmon if the mode of action is similar between species. Survival of trout was 32% to 55% in these predation studies compared to the control at 72%. This equals reductions in fish numbers of 28% to 55% in treatments compared to the control condition. Statistically significant reductions were also observed in the number of *Daphnia sp*. consumed and swimming activity when fish were exposed to a PCP concentration of 2 µg/L and a significant decrease in the strike frequency by trout on *Daphnia sp.* occurred at 20 µg/L. The exposures in Little *et al.* (1990) were conducted for 96 hours under static test conditions, and were based on nominal concentrations. The authors also expressed some concern about contaminants in the formulation used (technical grade PCP). Acetone was used as a carrier for PCP exposure in treatments and controls, which is very common in such experiments, but it is not likely to have contributed to toxicity; the concentration of acetone was 41 µg/L, which is very low. Acetone produces very low toxicity in salmonids (Majewski *et al.* 1978) and it is volatized or biodegraded in a matter of hours (Rathbun *et al.* 1982), implying that acetone was not likely a factor in the observed results.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for pentachlorophenol is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Eisler (1989) reviewed the effects of PCP on invertebrate growth, survival, and reproduction and reported adverse effects in the range of 3µg/L to 100 µg/L. It appears that most invertebrates are less sensitive than fish to PCP concentrations in water. There are, however, studies showing adverse effects to invertebrates exposed to water concentrations below the chronic criterion. Hedtke *et al.* (1985) determined reproductive impairment in a daphnid at 4 µg/L and pH 7.3. Tagatz *et al.* (1981) found a reduction in the number of species and organism abundance at PCP concentrations of 16 µg/L. The pH was not stated for this study but was likely between 7.5 and 8 because seawater was used.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is likely to adversely affect invertebrate productivity and abundance.

Summary of Effects: Pentachlorophenol. The available evidence for pentachlorophenol indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderatel-high-intensity) and reduced growth (moderate intensity).

2.6.2.1.7 Ammonia

Ammonia Criteria. At a pH of 8.0, the corresponding proposed criteria are 5.6 mg/L and 1.7 mg/L as N (NH_3-nitrogen) for acute and chronic criteria, respectively.

Tables 2.6.2.1.7.1 through 2.6.2.1.7.14 report toxicity data from the ECOTOX database for freshwater ammonia, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.7.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **2.1-18.7° Celsius**	**Arithmetic Mean** **34**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **32**
Endpoint/Effect **LC_{50}**	**pH** **6.00-9.46**	**Harmonic Mean** **29**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	**Duration**
7.3	40.0 G; SWIMMING FISH	NR
12.6	22.4 G	NR
14.0	LARVAE	NR
18.4	1.42 G	NR
22.4	10.9 G	NR
22.4	JUVENILE (4.8-9.2 CM)	NR
22.7	3.3 G	NR
23.0	JUVENILE (40 D)	NR
23.6	JUVENILE	NR
23.7	LARVAE	NR
24.4	1.30 G	NR
25.0	10.3 G	NR
25.6	1.30 G	NR
26.0	JUVENILE	NR
27.0	1 D OLD SAC FRY	NR
27.0	1 D OLD SAC FRY	NR
27.0	JUVENILE	NR
27.2	1.01 G	NR
27.7	JUVENILE	NR
27.8	1.11 G	NR
27.9	1.26 G	NR
28.7	0.90 G	NR
28.8	1.13 G	NR
30.6	1.44 G	NR
31.6	0.40 G	NR
32.1	14.0 G	NR
32.2	0.78 G	NR
32.6	JUVENILE (4.8-9.2 CM)	NR
32.7	0.60 G	NR

Criterion Freshwater Ammonia		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **2.1-18.7° Celsius**	**Arithmetic Mean** **34**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **32**
Endpoint/Effect **LC_{50}**	**pH** **6.00-9.46**	**Harmonic Mean** **29**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	**Duration**
33.7	1.50 G	NR
33.7	1.40 G	NR
33.8	1.64 G	NR
33.8	0.90 G	NR
34.0	1.00 G	NR
34.8	0.63 G	NR
35.5	LARVAE	NR
36.1	1.38 G	NR
36.5	0.80 G	NR
37.0	1.60 G	NR
37.4	0.80 G	NR
37.7	0.80 G	NR
37.8	JUVENILE	NR
39.4	0.90 G	NR
39.4	1.30 G	NR
40.5	JUVENILE (4.8-9.2 CM)	NR
41.0	2.01 G	NR
42.6	1.26 G	NR
43.3	LARVAE	NR
46.4	JUVENILE	NR
47.0	40.0 G; RESTING FISH	NR
48.8	JUVENILE (4.8-9.2 CM)	NR
49.5	JUVENILE	NR
56.1	JUVENILE (4.8-9.2 CM)	NR
65.8	JUVENILE (4.8-9.2 CM)	NR
68.6	JUVENILE (4.8-9.2 CM)	NR
89.3	JUVENILE (4.8-9.2 CM)	NR

For Tables 2.6.2.1.7.2 through 2.6.2.1.7.10 NMFS only selected toxicity data in the core data file with a reported concentration type of total ammonia. Since total ammonia is the sum of the two forms of ammonia (NH_4^+ and NH_3), NMFS assumes that the data with a reported concentration type of total ammonia were normalized by EPA. For these toxicity studies, temperature and pH were not reported in the core data files; therefore verification regarding normalization was not possible (note: the acute criterion is not temperature-dependent). In Tables 2.6.2.1.7.5 through 2.6.2.1.7.9 NMFS reported the toxicity data as no other toxicity data was available for an analysis of chronic endpoints for ammonia, and therefore serves as the best available data. Table 2.6.2.1.7.10 through Table 2.6.1.7.13 are the ACR-NOEC analysis for the chronic criterion.

Table 2.6.2.1.7.2 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		**Data Set ECOTOX**
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 0.55**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 0.53**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 0.51**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.380		8H
0.460		8H
0.560		8H
0.790		8H

Table 2.6.2.1.7.3 LD_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		**Data Set ECOTOX**
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 22**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 22**
Endpoint/Effect LD_{50}	**pH NR**	**Harmonic Mean 22**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
22		2D

Table 2.6.2.1.7.4 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **3.3**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **1.2**
Endpoint/Effect **Mortality**	**pH** **NR**	**Harmonic Mean** **0.3**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	**Duration**
0.05		21D
0.2		2.5D
0.3		120D
0.4		2.4H
1.6		289D
4.9		2D
6		4D
6.3		1D
10		90D

Table 2.6.2.1.7.5 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **1.5**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **1.2**
Endpoint/Effect **Growth**	**pH** **NR**	**Harmonic Mean** **0.9**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	**Duration**
0.3		120D
0.9		365D
1.2		365D
1.3		365D
1.6		365D
3.5		85D

Table 2.6.2.1.7.6 Biochemical toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 0.6**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 0.1**
Endpoint/Effect Biochemical	**pH NR**	**Harmonic Mean 0.004**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.001		1D
0.22		84D
0.7		4H
1.6		4H

Table 2.6.2.1.7.7 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 27.1**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 8.4**
Endpoint/Effect Behavioral	**pH NR**	**Harmonic Mean 1.7**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.4		4.8H
4.5		2.4H
6		2D
62.3		NR
62.3		NR

Table 2.6.2.1.7.8 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	Temperature NR	Arithmetic Mean 0.3
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	Hardness NR	Geometric Mean 0.3
Endpoint/Effect Cellular	pH NR	Harmonic Mean 0.3
Concentration Milligrams Liter^{-1}	Life-Stage	Duration
0.3		120D

Table 2.6.2.1.7.9 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	Temperature NR	Arithmetic Mean 0.23
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	Hardness NR	Geometric Mean 0.23
Endpoint/Effect Physiological	pH NR	Harmonic Mean 0.23
Concentration Milligrams Liter^{-1}	Life-Stage	Duration
0.23		42D
0.23		42D

As mentioned above, NMFS only selected chronic toxicity data in the core data file with a reported concentration type of total ammonia. Since total ammonia is the sum of the two forms of ammonia (NH_4^+ and NH_3), NMFS assumes that the data with a reported concentration type of total ammonia were normalized by EPA. For these toxicity studies, temperature and pH were not reported in the core data files; therefore verification regarding normalization was not possible and creates uncertainty. Therefore, as an additional step to address this uncertainty and to assess the potential for chronic toxic effects of ammonia to the listed species considered in this opinion using an additional line of evidence, NMFS used four ACRs to estimate a NOEC for ammonia:

(1) The rank ordered ACR of 3.26 for ammonia used in EPA's BE, Table 2.6.2.1.7.10.

Based on the ACR used in EPA's BE, and using the minimum species mean salmonid fish LC_{50} test concentration for ammonia in Table 2.6.2.1.7.1 and divided that concentration to derive an estimated NOEC concentration to

assess the potential for chronic toxic effects, NMFS calculated an estimated NOEC of 2.2 mg/L.

(2) The EPA reassessment of the 3.26 ACR used in the BE of 4.26 for ammonia, Table 2.6.2.1.7.11.

Based on the EPA reassessment ACR of 4.26, and using minimum species mean salmonid fish LC_{50} test concentration for ammonia in Table 2.6.2.1.7.1 and divided that concentration to derive an estimated NOEC concentration to assess the potential for chronic toxic effects, NMFS calculated an estimated NOEC of 1.7 mg/L.

(3) The ranked ordered data only for fishes—instead of the fish and invertebrate rank ordered data EPA used to calculate the ammonia ACR of 3.26 in the BE as NMFS considers a fish-based ACR the best scientific surrogate to estimate a NOEC for fishes for ammonia, Table 2.6.2.1.7.12.

Based on the adjusted ACR calculation, NMFS calculated an ACR of 5.8. The NMFS then selected minimum species mean salmonid fish LC_{50} test concentration for ammonia in Table 2.6.2.1.7.1 and divided that concentration by the adjusted ACR to derive an estimated NOEC concentration to assess the potential for chronic toxic effects, NMFS calculated an estimated NOEC of 1.3 mg/L.

(4) The ranked ordered data for fishes, without the catfish ACR value, instead of the fish and invertebrate rank ordered data EPA used to calculate the ammonia ACR of 3.26 in the BE as NMFS considers a fish-based ACR the best scientific surrogate to estimate a NOEC for fishes for ammonia, Table 2.6.2.1.7.13.

Based on the adjusted ACR calculation, without the catfish ACR value, NMFS calculated an ACR of 3.6. The NMFS then selected minimum species mean salmonid fish LC_{50} test concentration for ammonia in Table 2.6.2.1.7.1 and divided that concentration by the adjusted ACR to derive an estimated NOEC concentration to assess the potential for chronic toxic effects, NMFS calculated an estimated NOEC of 1.3 mg/L.

NMFS selected the minimum species mean value from the salmonid fishes LC_{50} test concentration for ammonia as it represents the lowest acute toxicity concentration that predicts the greatest risk of adverse toxic effects to field-exposed fishes, predicted at 38.4 percent (Table 2.6.2.1.7.14),), and therefore permits an assessment that considers the “worst case” exposure scenario.

The results of the ACR-NOEC analysis produced one NOEC below the chronic criterion, one NOEC equal to the chronic criterion, and two NOECs above the chronic chronic criterion.

Table 2.6.2.1.7.10 ACR-NOEC toxicity analysis for salmonid fishes, eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set BE pH-adjusted
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	Temperature 16.6	ACR 3.26
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	Hardness NR	Salmonid LC_{50} 7.3 Milligrams Liter^{-1}
Endpoint/Effect ACR-NOEC	pH 6.97	ACR EPA BE
Concentration Milligrams Liter^{-1}	Life-Stage	
2.2	40.0 G; SWIMMING FISH	

Table 2.6.2.1.7.11 ACR-NOEC toxicity analysis for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set BE pH-adjusted
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	Temperature 16.6	ACR 4.26
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	Hardness NR	Salmonid LC_{50} 7.3 Milligrams Liter^{-1}
Endpoint/Effect ACR-NOEC	pH 6.97	ACR EPA Reassessment
Concentration Milligrams Liter^{-1}	Life-Stage	
1.7	40.0 G; SWIMMING FISH	

Table 2.6.2.1.7.12 ACR-NOEC toxicity analysis for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set BE pH-adjusted
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	Temperature 16.6	ACR 5.8
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	Hardness NR	Salmonid LC_{50} 7.3 Milligrams Liter^{-1}
Endpoint/Effect ACR-NOEC	pH 6.97	ACR Fish Only
Concentration Milligrams Liter^{-1}	Life-Stage	
1.3	40.0 G; SWIMMING FISH	

Table 2.6.2.1.7.13 ACR-NOEC toxicity analysis for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set BE pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **16.6**	**ACR** **3.6**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Salmonid LC_{50}** **7.3 Milligrams Liter^{-1}**
Endpoint/Effect **ACR-NOEC**	**pH** **6.97**	**ACR Fish Only (without catfish ACR value)**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	
2	40.0 G; SWIMMING FISH	

Ammonia Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, at face value, none of toxicity studies reported LC_{50} concentrations that are less than the acute criterion concentration for ammonia, which implies that listed species exposed to waters equal to criterion concentrations may not suffer acute toxic effects. However, since some of the LC_{50} data had concentrations near the acute criterion concentration, NMFS added an additional step to its analysis for ammonia to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality (Table 2.6.2.1.7.14). This assessment involved taking the acute criterion of 5.6 mg/L and dividing it by each LC_{50} concentration in Table 2.6.2.1.7.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.7.1, predicts a magnitude of effect ranging from a low of an $LC_{3.2}$ at a concentration of 89.3 mg/L to a high of an $LC_{38.4}$ at a concentration of 7.3 mg/L. In other words, the acute criterion of 5.6 mg/L has an equivalent toxicity potential predicted to kill 3.2 percent to 38.4 percent, with a median toxicity potential of an $LC_{8.6,}$ of the exposed test population, and therefore by inference, field-exposed individuals.

Table 2.6.2.1.7.14 Relative percent mortality analysis for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **2.1-18.7° Celsius**	
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	
Endpoint/Effect **LC_{50}**	**pH** **6.00-9.46**	
Concentration **Milligrams Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
7.3	38.4	
12.6	22.5	
14.0	20.0	
18.4	15.2	
22.4	12.5	
22.4	12.5	
22.7	12.3	
23.0	12.2	
23.6	11.9	
23.7	11.8	
24.4	11.5	
25.0	11.2	
25.6	11.0	
26.0	10.8	
27.0	10.4	
27.0	10.4	
27.0	10.4	
27.2	10.3	
27.7	10.1	
27.8	10.1	
27.9	10.1	
28.7	9.8	
28.8	9.7	
30.6	9.2	
31.6	8.9	
32.1	8.7	
32.2	8.7	
32.6	8.6	
32.7	8.6	

Criterion Freshwater Ammonia		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **2.1-18.7° Celsius**	
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	
Endpoint/Effect **LC_{50}**	**pH** **6.00-9.46**	
Concentration **Milligrams Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
33.7	8.3	
33.7	8.3	
33.8	8.3	
33.8	8.3	
34.0	8.3	
34.8	8.1	
35.5	7.9	
36.1	7.8	
36.5	7.7	
37.0	7.6	
37.4	7.5	
37.7	7.5	
37.8	7.4	
39.4	7.1	
39.4	7.1	
40.5	6.9	
41.0	6.9	
42.6	6.6	
43.3	6.5	
46.4	6.1	
47.0	6.0	
48.8	5.8	
49.5	5.7	
56.1	5.0	
65.8	4.3	
68.6	4.1	
89.3	3.2	

For the chronic criterion assessment, a number of chronic toxicity studies reported concentrations that are less than the chronic criterion concentration for ammonia, which implies that listed species exposed to waters equal to criteria concentrations will suffer chronic toxic effects. The NMFS only selected chronic toxicity data in the core data file with a reported

concentration type of total ammonia. For these toxicity studies, temperature and pH were not reported in the core data file, therefore verification regarding normalization was not possible and creates uncertainty. Nonetheless, the toxicity assessments in Table 2.6.2.1.7.10, which produced a concentration less than the chronic criterion concentration, through Table 2.6.2.1.7.13, with one NOEC equal to the chronic criterion, and two NOECs above the chronic criterion, indicates that listed species exposed to waters equal to chronic criterion concentrations will suffer chronic toxic effects.

When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle, the considerations of the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes, the relative percent mortality analysis, and the chronic toxicity assessment, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. The chemical form of ammonia in water consists of two species, a larger component which is the ammonium ion (NH_4^+) and a smaller component which is the non-dissociated or un-ionized ammonia (NH_3) molecule. The sum of the two forms is usually expressed as total ammonia-nitrogen. The ratio of un-ionized ammonia to ammonium ion, dependent upon both pH and temperature, generally increases 10-fold for each rise of a single pH unit, and approximately 2-fold for each 10°C rise in temperature over the 0 to 30°C range (Erickson 1985 as cited in EPA 2008). Toxicity of ammonia to aquatic life was initially thought to arise largely from the small uncharged NH_3 molecule (Wuhrmann and Woker 1948, Downing and Merkens 1955 as cited in EPA 2008), however more recent information indicates that ammonia is more toxic as the hydrogen ion concentration [H^+] increases (pH decreases), at least below a pH of 7.3 (Armstrong *et al.* 1978, Tomasso *et al.* 1980 as cited in EPA 2008).

Acute effects likely are primarily neurological in origin resulting from severe metabolic alterations of the central nervous system (Smart 1978, Levi *et al.* 1974 as cited in EPA 2008). The toxic symptoms observed in fish acutely exposed to ammonia include hyper-excitability, coma, convulsions and hyperventilation. Sublethal effects can be quite extensive, and include reduced food uptake and growth inhibition, diuresis and ion imbalance, inflammation and degeneration of the gills and other tissues, changes in the oxygen-carrying capacity of the blood, and increased susceptibility to disease (Russo 1985 as cited in EPA 2008).

Physiological effects on salmonid fishes has been reported to occur at concentrations as low as 0.005 mg/L (42-day exposure) (Burrows 1964), but other studies on mortality recorded thresholds as varied as 0.03 mg/L (2-day exposure) (Herbert 1956) and 5 mg/L (3-day exposure) (Holland *et al.* 1960). The physiological harm recorded in Burrows' study (1964) was gill hyperplasia that may additionally result in bacterial gill disease. Gill hyperplasia is a response by epithelial cells and lamellae in the gills of fishes to irritations that may include uncontrolled cell growth, thinning, and fusion of lamellae (Burrows 1964, Post 1971, Dauba *et al.* 1992).

Reductions in growth on rainbow trout may occur as low as 0.0023 mg/L (120-day exposure) (Soderberg *et al.* 1983) or as high as 1.3 mg/L (365-day exposure) (Smith 1972). The NMFS assumes that growth reductions occurred throughout the exposure during the Soderberg *et al.*

study (1983) and that gill hyperplasia occurred throughout the exposure in Burrows' study (1964).

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for ammonia is likely to result in sublethal effects to listed species considered in this opinion.

Summary of Effects: Ammonia. The available evidence for indicates that listed species exposed to water equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (high-intensity), reduced growth (high-intensity), impairment of essential behaviors related to successful rearing and migration (moderately-high-intensity), cellular trauma (high-intensity), physiological trauma (high-intensity), impairment of biochemical processes (high-intensity), and sublethal effects—ACR-NOEC analysis—(moderately-high-intensity to high-intensity).

2.6.2.2 Metal and Elemental Pollutants: Analysis of Individual Compounds

In this section, the effects of each metal and elemental toxic substance listed in Table 1.1 are identified, and the proposed criteria are compared with available toxicity data that describe the results of toxicity tests. The analysis identifies potential effects on listed species and their critical habitat of each of the criteria that would be expected to occur if water concentrations were equal to or less than the proposed criteria. Where possible, effects on the food sources of listed species, and effects related to bioaccumulation, are also identified. The following analysis focuses on each parameter individually.

2.6.2.2.1 Aluminum[8]

Aluminum Criteria. The proposed criteria concentrations of aluminum are 750 µg/L and 87 µg/L for acute and chronic criteria, respectively.

Tables 2.6.2.2.1.1 through 2.6.2.2.1.9 report toxicity data from the ECOTOX database for freshwater aluminum, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

[8] On August 9, 2012, EPA sent NMFS a letter withdrawing their request for consultation on Oregon's acute and chronic aluminum criteria as "EPA has determined that the BE submitted to NMFS in January 2008 incorrectly described the proposed federal action under consultation for aluminum (*i.e*., CW A § 303(c)(3) approval of Oregon's submission of aluminum criteria). Specifically, Oregon's submitted description of the pollutant refers to aluminum in waters with a pH of 6.5- 9.0, but a footnote in the criterion itself indicates that the criterion is meant to apply to waters with pH less than 6.6 and hardness less than 12 mg/L (as $CaCO_3$)." Due to the court-ordered deadline of August 14, 2012, NMFS did not have time to modify its opinion to exclude acute and chronic aluminum from the document. The NMFS acknowledges EPA's revision to the proposed action, however, and notes it does not anticipate EPA will carry out the RPA for aluminum in light of this change. The NMFS will await a further request from EPA relating to EPA's potential future actions regarding Oregon's aluminum criteria.

Table 2.6.2.2.1.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		**Data Set ECOTOX**
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 12-15.7° Celsius**	**Arithmetic Mean 4684**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 6.6-115.8 mg/L $CaCO_3$**	**Geometric Mean 2247**
Endpoint/Effect LC_{50}	**pH 6.5-8.58**	**Harmonic Mean 867**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
170	FERTILIZATION THROUGH 4 DAY POST/	28D
400	EGGS	28D
400	EGGS	28D
445	ALEVINS	96H
510	EGG	28D
1620	JUVENILE, 1-3 G	96H
2860	JUVENILE, 1-3 G	96H
3600	JUVENILE	
5310	JUVENILE, 1-3 G	96H
5330	JUVENILE, 1-3 G	96H
6220	JUVENILE, 1-3 G	96H
7400		24H
7900		
9600	5.52 CM, 33 G	24H
18500	NR	48H

Table 2.6.2.2.1.2 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		**Data Set ECOTOX**
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 1-15° Celsius**	**Arithmetic Mean 2870**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 17-280 mg/L $CaCO_3$**	**Geometric Mean 408**
Endpoint/Effect Mortality	**pH 6.5-8.7**	**Harmonic Mean 134**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
20	EYED EGG STAGE	8D
20	CLEAVAGE EMBRYO, EYED	8D
50	CLEAVAGE EMBRYO, EYED	8D
57	EYED EGG	15D
57	EYED EMBRYO - LARVAE	30D
57	FRY	45D
57	FRY	60D
88	EYED EMBRYO - LARVAE	15D
90	118-355 G, 22-31 CM FORK LENGTH	96H
100	CLEAVAGE EMBRYO, EYED	8D
100	CLEAVAGE EMBRYO, EYED	8D
100	SMOLT, 1 YR, 65 G, 195 MM	23D
169	EYED EMBRYO - LARVAE	15D
169	EYED EMBRYO - LARVAE	30D
169	FRY	45D
242	EYED EGG	15D
242	EYED EGG	15D
242	EYED EGG	15D
242	EYED EGG	30D
242	37 D, JUVENILE	15D
268	0.2 G, 30 D	56H
283	EYED EMBRYO - LARVAE	60D
330	ADULT, 1518 G, 51.5 CM TL	48H
350	EYED EGG	15D
350	EYED EMBRYO - LARVAE	30D
350	FRY	45D
350	FRY	60D
500	CLEAVAGE EMBRYO, EYED	8D
720	JUVENILE, 1-3 G	16D
910	118-355 G, 22-31 CM FORK LENGTH	24H
910	118-355 G, 22-31 CM FORK LENGTH	48H
910	118-355 G, 22-31 CM FORK LENGTH	72H

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 1-15° Celsius**	**Arithmetic Mean 2870**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 17-280 mg/L $CaCO_3$**	**Geometric Mean 408**
Endpoint/Effect Mortality	**pH 6.5-8.7**	**Harmonic Mean 134**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
910	118-355 G, 22-31 CM FORK LENGTH	96H
1000	CLEAVAGE EMBRYO, EYED	8D
1680	JUVENILE, 1-3 G	16D
9100	118-355 G, 22-31 CM FORK LENGTH	24H
9100	118-355 G, 22-31 CM FORK LENGTH	48H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
50000	50-80 MM	96H

Table 2.6.2.2.1.3 LT_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 4245**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness NR**	**Geometric Mean 3261**
Endpoint/Effect LT_{50}	**pH 6.52-8.99**	**Harmonic Mean 1837**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration/Days**
513	11 WK	43.9
5140	FINGERLINGS, 6 WK	7.5
5140	11 WK	38.9
5200	FINGERLINGS, 6 WK	2.98
5230	6 MO	31.96

Table 2.6.2.2.1.4 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		**Data Set ECOTOX**
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 182**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 245-255 mg/L $CaCO_3$**	**Geometric Mean 148**
Endpoint/Effect NOEC/Growth/Behavioral	**pH 6.5-6.6**	**Harmonic Mean 121**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
57	EYED EMBRYO - LARVAE	30D
88	FRY	45D
88	FRY	60D
169	FRY EYED EMBRYO - LARVAE	30D
169	FRY	60D
350	EYED EMBRYO - LARVAE	30D
350	FRY	60D

Table 2.6.2.2.1.5 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		**Data Set ECOTOX**
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 11-19° Celsius**	**Arithmetic Mean 191**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 15-280 mg/L $CaCO_3$**	**Geometric Mean 103**
Endpoint/Effect Growth	**pH 6.52-8.99**	**Harmonic Mean 1.1**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.05	FINGERLINGS, 6-24 WK	222H
38.1	JUVENILE, 7.5-8.5 G	34D
52	6 WK-6 MO	113D
57	FRY	30D
57	FRY	45D
57	FRY	60D
88	FRY	30D
88	FRY	45D
88	FRY	60D
100	SMOLT, 1 YR, 65 G, 195 MM	16D
169	FRY	30D
169	FRY	45D
169	FRY	60D
242	EYED EGG	15D
242	EYED EGG	30D
242	37 D, JUVENILE	15D
268	0.2 G, 30 D	3D
283	EYED EMBRYO - LARVAE	45D
350	FRY	30D
350	FRY	45D
350	FRY	60D
740	JUVENILE, 1-3 G	16D

Table 2.6.2.2.1.6 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		**Data Set ECOTOX**
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 11-13° Celsius**	**Arithmetic Mean 270**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 15-103.5 mg/L $CaCO_3$**	**Geometric Mean 200**
Endpoint/Effect Behavioral	**pH 6.5-8.14**	**Harmonic Mean 148**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
57	FRY	60D
88	FRY	60D
169	FRY	60D
242	EYED EGG	30D
242	37 D, JUVENILE	15D
350	FRY	60D
740	JUVENILE, 1-3 G	16D

Table 2.6.2.2.1.7 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		**Data Set ECOTOX**
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 11.5-19° Celsius**	**Arithmetic Mean 100**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness NR**	**Geometric Mean 100**
Endpoint/Effect Cellular	**pH 7.2**	**Harmonic Mean 100**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
100	SMOLT, 1 YR, 65 G, 195 MM	16D
100	SMOLT, 1 YR, 65 G, 195 MM	16D
100	SMOLT, 1 YR, 65 G, 195 MM	16D

Table 2.6.2.2.1.8 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		**Data Set ECOTOX**
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 1-19° Celsius**	**Arithmetic Mean 149**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness NR**	**Geometric Mean 105**
Endpoint/Effect Physiological	**pH 6.5-7.1**	**Harmonic Mean 81**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
59	SMOLT, 30 G	48H
59	SMOLT, 30 G	2D
330	ADULT, 1518 G, 51.5 CM TL	48H

Aluminum Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to aluminum, NMFS added an additional step to its analysis for aluminum to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality (Table 2.6.2.2.1.9). This assessment involved taking the acute criterion of 750 µg/L and dividing it by each 24H, 48H, and 96H duration LC_{50} concentrations in Table 2.6.2.2.1.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.1.1, predicts a magnitude of effect ranging from a low of an LC_2 at a concentration of 18,500 µg/L to a high of an LC_{84} at a concentration of 445 µg/L. In other words, the acute criterion of 750 µg/L has an equivalent toxicity potential predicted to kill 2 percent to 84 percent, with a median toxicity potential of an $LC_{15,}$ of the exposed test population, and therefore by inference, field-exposed individuals.

Table 2.6.2.2.1.9 Relative percent mortality analysis for salmonid fishes, eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 12° Celsius**	
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 6.6-115.8 mg/L $CaCO_3$**	
Endpoint/Effect LC_{50}	**pH 6.5-8.58**	
Concentration Micrograms Liter^{-1}	**Relative Percent Mortality (acute criterion/LC_{50})**	
445	84	
1620	23	
2860	26	
5310	7	
5330	7	
6220	6	
7400	5	
9600	4	
18500	2	

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for aluminum, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for aluminum, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Aluminum is one of the most abundant elements in the earth's crust and occurs in many rocks and ores, but never as a pure metal. The presence of aluminum ions in streams may result from industrial wastes but is more likely to come from the wash water of drinking water treatment plants. Many aluminum salts are readily soluble; however, there are some that are very insoluble. Those that are insoluble will not exist long in surface water, but will precipitate and settle. Waters containing high concentrations of aluminum can become toxic to aquatic life if the pH is lowered (as in acid rain).

Aluminum, like other metals, generally acts as a surface active toxicant, exerting its damage by binding to anionic sites on respiratory surfaces of aquatic animals, such as a fish gill (Wood *et al.* 1997 as cited in EPA 2008). The physiological manifestation of these deleterious surface effects at the gill include both ionoregulatory and respiratory effects. Ionoregulatory effects of

aluminum predominate at low pH (*e.g.*, less than pH 5.0) and include a mechanism similar to hydrogen ion toxicity alone, *i.e.*, sodium uptake blockade (Playle *et al.* 1989 as cited in EPA 2008). In moderately acidic water, it is generally the respiratory effects of aluminum that predominate. Respiratory effects are likely the result of the physical coating of the gills which occurs when aluminum-rich water passes into the more basic gill microenvironment (Gensemer and Playle 1999 as cited in EPA 2008). Overall, chronic aluminum toxicity to fish species is substantially greater at low pH, particularly for salmonids. For many fish, aluminum toxicity increases with early life stage such that eggs and endogenously-feeding alevins are generally less sensitive than exogenous-feeding swim-up larvae (Buckler *et al.* 1985, DeLonay *et al.* 1993 as cited in EPA 2008). Holtze (1984) concluded that rainbow trout were most sensitive to aluminum during the yolk sac and swim-up fry stages and least sensitive to aluminum during the cleavage stage. Holtze (1984) also concluded that aluminum was beneficial to the survival of cleavage embryos at pH 4.5. Therefore, aluminum at extreme low pH (pH <5) can protect against the direct toxic effects, and aluminum criteria based on higher pH values may undermine embryo survival. Several factors ameliorate aluminum toxicity at low pH, including, but probably not limited to: calcium ion (Brown 1983, Ingersoll *et al.* 1990 as cited in EPA 2008), silicic acid (Birchall *et al.* 1989 as cited in EPA 2008), fluoride (Wilkinson *et al.* 1990 as cited in EPA 2008), and dissolved and natural organic matter (Parkhurst *et al.* 1990; Roy and Campbell 1997 as cited in EPA 2008).

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for aluminum is likely to result in sublethal effects to listed species considered in this opinion.

Summary of Effects: Aluminum. The available evidence for indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (high-intensity), reduced growth (high-intensity), impairment of essential behaviors related to successful rearing and migration (moderately-high-intensity), cellular trauma (moderate intensity), and physiological trauma (moderately-high-intensity).

2.6.2.2.2 Arsenic

Arsenic Criteria. The proposed criteria for dissolved concentrations of trivalent arsenic equal 340 µg/L and 150 µg/L for acute and chronic criteria, respectively.

Tables 2.6.2.2.2.1 through 2.6.2.2.2.5 report toxicity data from the ECOTOX database for freshwater arsenic, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.2.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion Freshwater Arsenic		**Data Set ECOTOX**
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 57845**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 16698**
Endpoint/Effect LC_{50}	**pH 7.4-10.2**	**Harmonic Mean 342**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
10	NR	96H
25	NR	24H
25	57 G	24H
170	FERTILIZATION THROUGH 4 DAY POST	28H
420	EGGS	28H
420	EGGS	144H
490	NR	24H
490	EGG	4H
1400	FINGERLING, 5.7 G	22H
3510	FRY	96H
3830	JUVENILE, 7-8 WK, 0.20 G	96H
4050	JUVENILE, 7-8 WK, 0.34 G	96H
5000	EGG	96H
7500	NR	96H
8200	FINGERLING, 5.7 G	96H
8200	FINGERLING, 5.7 G	30H
10800	YY, 2 mo, 51-76 MM TL	96H
10800	YY, 2 mo, 51-76 MM TL	96H
11600	JUVENILE, 45.5 MM, 0.51 G	96H
12200	3.5 G	144H
12200	3.5 G	96H
12700	JUVENILE, 64.3 MM, 2.49 G	96H
12700	JUVENILE, 64.3 MM, 2.49 G	28H
13500	2.6G	96H
14500	JUVENILE, 39.0 MM, 0.41 G	96H
14500	JUVENILE, 39.0 MM, 0.41 G	24H
17700	FINGERLING, 5.7 G	24H
18100	FRY, 1.99 G	96H
18100	FRY, 1.99 G	96H
19300	FRY, 0.50 G	96H
19300	FRY, 0.50 G	96H

Criterion Freshwater Arsenic		Data Set ECOTOX
Criterion Concentration Acute **340 Micrograms Liter^{-1}**	**Temperature** **5.4-15.1° Celsius**	**Arithmetic Mean** **57845**
Criterion Concentration Chronic **150 Micrograms Liter^{-1}**	**Hardness** **44-343 mg/L $CaCO_3$**	**Geometric Mean** **16698**
Endpoint/Effect **LC_{50}**	**pH** **7.4-10.2**	**Harmonic Mean** **342**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
21900	JUVENILE, 7-11 WK, 1.85 G	96H
23700	ADULT, 18 MO, 200.0 MM, 84.7 G	96H
25300	JUVENILE, 7-11 WK, 0.97 G	96H
25600	3.5 G	144H
25600	3.5 G	144H
32500	JUVENILE, 10-12 WK, 0.41 G	96H
32500	JUVENILE, 10-12 WK, 0.41 G	28H
34000	YOUNG OF YR, 0.5-3.0 G	24H
35000	JUVENILE, 5-6 WK, 0.85 G	96H
42100	ALEVIN, 29.8 MM, 0.24 G	96H
46000	FRY, 1.99 G	24H
47000	FRY, 1.03 G	24H
49400	JUVENILE, 18-22 WK, 0.47 G	96H
49400	JUVENILE, 18-22 WK, 0.47 G	24H
50300	FRY, 0.50 G	24H
55400	FRY, 0.50 G	96H
55400	FRY, 0.50 G	96H
56000	JUVENILE, 18-22 WK, 0.47 G	96H
56100	JUVENILE, 7-10 WK, 1.04 G	96H
56100	JUVENILE, 7-10 WK, 1.04 G	24H
62900	FRY	24H
69900	ALEVIN, 20.8 MM, 0.10 G	96H
70000	FRY, 0.50 G	96H
70000	FRY, 0.50 G	96H
70600	2.6G	96H
74000	JUVENILE, 10-12 WK, 0.41 G	96H
118000	JUVENILE, 7-10 WK, 1.04 G	96H
120000	FRY, 1.03 G	96H
120000	FRY	96H
120000	FRY, 1.03 G	96H
120000	FRY	96H
130000	FRY, 0.50 G	24H
216000	ALEVIN	24H
224000	FRY	24H

Criterion Freshwater Arsenic		Data Set ECOTOX
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 57845**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 16698**
Endpoint/Effect LC_{50}	**pH 7.4-10.2**	**Harmonic Mean 342**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
360000	ALEVIN	96H
360000	ALEVIN	24H
547000	ALEVIN	96H

Table 2.6.2.2.2.2 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion Freshwater Arsenic		Data Set ECOTOX
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 69883**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 62625**
Endpoint/Effect Mortality	**pH 7.4-10.2**	**Harmonic Mean 57167**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
35000	JUVENILE, 5-6 WK, 0.85 G	11W
43300	JUVENILE, 7-11 WK, 0.97 G	4D
60000	ALEVIN	11W
61000	JUVENILE, 5-6 WK, 0.85 G	40D
75000	ALEVIN	10D
145000	ALEVIN	4D

Table 2.6.2.2.2.3 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion Freshwater Arsenic		**Data Set ECOTOX**
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 31332**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 14894**
Endpoint/Effect Growth	**pH 7.4-10.2**	**Harmonic Mean 9305**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3510	FRY	11W
3830	JUVENILE, 7-8 WK, 0.20 G	12W
4050	JUVENILE, 7-8 WK, 0.34 G	12W
6630	JUVENILE, 7-8 WK, 0.34 G	8W
9200	JUVENILE, 7-8 WK, 0.20 G	12W
11600	JUVENILE, 45.5 MM, 0.51 G	8W
17100	ADULT, 18 MO, 200.0 MM, 84.7 G	8W
21100	FRY	11W
23500	ALEVIN, 15.0 MM, 0.02 G	2W
23900	ADULT, 18 MO, 200.0 MM, 84.7 G	4D
25300	JUVENILE, 7-11 WK, 0.97 G	2W
41600	JUVENILE, 7-11 WK, 1.85 G	8W
216000	ALEVIN	8W

Table 2.6.2.2.2.4 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion Freshwater Arsenic		**Data Set ECOTOX**
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 19933**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 19764**
Endpoint/Effect Behavioral	**pH 7.4-10.2**	**Harmonic Mean 19605**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
17800	ADULT, 18 MO, 200.0 MM, 84.7 G	8W
18300	ADULT, 18 MO, 200.0 MM, 84.7 G	8W
23700	ADULT, 18 MO, 200.0 MM, 84.7 G	12W

Table 2.6.2.2.2.5 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion **Freshwater Arsenic**		**Data Set** **ECOTOX**
Criterion Concentration Acute **340 Micrograms Liter^{-1}**	**Temperature** **5.4-15.1° Celsius**	**Arithmetic Mean** **21900**
Criterion Concentration Chronic **150 Micrograms Liter^{-1}**	**Hardness** **44-343 mg/L $CaCO_3$**	**Geometric Mean** **21900**
Endpoint/Effect **Physiological**	**pH** **7.4-10.2**	**Harmonic Mean** **21900**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
21900	JUVENILE, 7-11 WK, 1.85 G	1D

Arsenic Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to arsenic, NMFS added an additional step to its analysis for arsenic to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 340 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.2.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.2.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 547,000 µg/L to a high of an LC_{100} at a concentration of 10 µg/L. In other words, the acute criterion of 340 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{0.7}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for arsenic, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for arsenic, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute toxic effects, but may not suffer chronic toxic effects.

Sublethal Effects. Arsenic occurs naturally in aquatic environments in trace amounts. Background concentrations in freshwater streams are usually less than 1 µg/L (Moore and

Ramamoorthy 1984). Mining, smelting, manufacturing, electric power plants, pesticides, agricultural defoliants, and battery manufacturing and reclamation plants are all significant anthropogenic sources of arsenic (Sorensen 1991).

Arsenic is a suspected carcinogen in fish. It is associated with necrotic and fibrous tissues and cell damage, especially in the liver. Arsenic can result in immediate death through increased mucus production and suffocation. Other effects include anemia and gallbladder inflammation. The toxicity of arsenic is influenced by a number of factors including fish size, water temperature, pH, redox potential, organic matter, phosphate content, suspended solids, presence of other toxicants, speciation of the chemical itself, and the duration of exposure (Dabrowski 1976, Eisler 1988a, McGeachy and Dixon 1989, Sorensen 1991, Cockell *et al.* 1992, Rankin and Dixon 1994, Woodward *et al.* 1994). Juvenile salmonids have been determined to be more sensitive to arsenic toxicity than alevins (Buhl and Hamilton 1990, 1991). Trivalent arsenic (arsenite) tends to be more toxic than other forms of arsenic, and inorganic forms of arsenic (including pentavalent) are typically more toxic than organic forms (EPA 1985b, Eisler 1988a, Sorensen 1991). Chronic toxicity in fish appears to be inversely proportional to water temperature under certain experimental conditions (McGeachy and Dixon 1990). Relatively little data exists that would allow establishment of separate standards for the multiple forms of arsenic that can occur in the aquatic environment.

Arsenic is bioconcentrated by organisms but is not biomagnified through the food chain (Eisler 1988a). Toxic effects of arsenic to aquatic life are significantly modified by numerous biological and abiotic factors (EPA 1985b as cited in EPA 2008) such as water temperature, hardness, pH, organic content, phosphate concentration, suspended solids, *etc.* (Eisler 1988a as cited in EPA 2008). In general, inorganic forms of arsenic are more toxic than organic forms to aquatic biota (EPA 1999). Early life stages are most sensitive, and large interspecies differences are recorded, even among those closely related taxonomically (Eisler 1988a as cited in EPA 2008). In fish, tolerance of arsenic appears to increase with temperature (McGeachy and Dixon 1990 as cited in EPA 2008), whereas in invertebrates the opposite is true (Bryant *et al.* 1985 as cited in EPA 2008). Effects of arsenic toxicity to aquatic biota include: avoidance and immobility in freshwater snails; and anemia, gall bladder inflammation, liver degeneration, reduced hemoglobin, and reduced success in seaward migration of fish.

Birge *et al.* (1981) reported an LC_{10} of 134 µg/L for rainbow trout embryos after a 28-day exposure (Birge *et al.* 1981). However, it is likely that the corresponding 4-day (the longest duration that a concentration can be between the acute and chronic criteria) LC_{10} would be higher, because in general test organisms mortality increases with exposure duration. Also, those results could have been influenced by bioaccumulation, such that the toxicity response was chronic rather than acute in nature. The studies reviewed indicate that acute toxicity, including to alevins, occurs at concentrations that are significantly higher than the proposed acute criterion (*e.g.*, Buhl and Hamilton 1990).

The results of Birge *et al.* (1978, 1981) suggests that chronic arsenic toxicity occurs to developing embryos of salmonids at concentrations below the proposed chronic criterion. For example, rainbow trout embryos exposed to arsenic for 28 days (4 days post-hatching) at 12°C to13°C and a hardness of 93 mg/L to 0.5 mg/L $CaCO_3$ in static tests (Birge *et al.* 1978, 1981) at

concentrations of 40 µg/L to 42 µg/L were associated with the onset of embryo mortality. Acclimation appears to enhance resistance to chronic arsenic toxicity (Dixon and Sprague 1981, EPA 1985b), which may explain in part why no studies were found by NMFS that indicate chronic toxicity occurs to juvenile and adult salmonids at concentrations near or below the proposed chronic criterion. Studies reviewed in Eisler (1988) and EPA (1985a) indicate that chronic effects do not occur in other life stages until concentrations are at least about an order of magnitude higher than the levels determined by Birge *et al.* (1978, 1981) to be detrimental to developing embryos.

Chronic exposure results in bioaccumulation of arsenic to toxic levels in fish, with most accumulating in the liver, pancreas, spleen, and kidneys, and relatively little in muscle tissues. Trivalent arsenic appears to bioaccumulate more readily than pentavalent, but there is no consistent relation with fish size or condition (EPA 1985b, Sorensen 1991). The inorganic pentavalent form appears to be the most stable in aquatic systems (Eisler 1988a). Bioaccumulation rates vary with fish species, where planktivorous fish are more likely to concentrate arsenic than omnivorous or piscivorous fishes (Hunter *et al.* 1981, Sorensen 1991). Diet appears to be a significant pathway for arsenic accumulation in salmonids (Oladimeji *et al.* 1984), although developing embryos have also been documented to uptake arsenic (Dabrowski 1976). Spehar *et al.* (1980) determined that rainbow trout did not accumulate arsenic significantly at concentrations above the proposed criteria. Similarly, Robinson *et al.* (1995) found no evidence of arsenic uptake or accumulation from water in rainbow and brown trout.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for arsenic is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Data on arsenic toxicity to aquatic macroinvertebrates are limited. What information does exist (EPA 1980b, 1985b; Eisler 1988a; Canivet *et al.* 2001) suggests that the proposed criterion should not result in acute or chronic toxicity to most aquatic macroinvertebrate taxa. Results reported in Eisler (1988a) suggest that gammarid amphipods may experience acute toxicity at concentrations of trivalent arsenic that are below the chronic criterion. Canivet *et al.* (2001) similarly determined greater sensitivity of a gammarid amphipod compared with other taxa tested, with a 240-hour LC_{50} of 200 µg/L, which is higher than the proposed chronic criterion. There is evidence that benthic invertebrate communities respond to elevated chronic arsenic levels by shifting community composition to pollution-tolerant taxa, while overall biomass does not change significantly (Canfield *et al.* 1994; Beltman *et al.* 1999). A shift to pollution tolerant taxa could change the availability of forage items. Primary aquatic invertebrate taxa used for food by rearing juvenile Chinook and steelhead (*e.g.*, stoneflies, mayflies, and caddisflies; EPA 1980b, 1985b; Canivet *et al.* 2001) do not appear to exhibit chronic effects at concentrations below the proposed chronic criterion. Irving *et al.* (2008) exposed mayfly nymphs to tri- and pentavalent arsenic in water-only exposures for 12 days. For trivalent arsenic, the threshold of growth effects was about 100 µg/L. However, arsenic levels accumulated by the mayfly nymphs in their study (1.2 to 4.6 µg/g dry wt) were far lower than those reported from stream locations with far lower water concentrations of arsenic but that had elevated arsenic in diet or sediments, suggesting that the water-only exposures may have underrepresented likely environmental exposures.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Arsenic. The available evidence for arsenic indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), interference in physiochemical processes (moderate intensity), interruption of ecological interactions (low intensity), and changes in pathological stress (low intensity).

2.6.2.2.3 Cadmium

Cadmium Criteria. The proposed acute and chronic criteria for cadmium are 2.0 µg/L and 0.25 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.3.1 through 2.6.2.2.3.7 report toxicity data from the ECOTOX database for freshwater cadmium, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.3.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 9.6-17.3° Celsius**	**Arithmetic Mean 18**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 9.2-410.5 mg/L $CaCO_3$**	**Geometric Mean 9**
Endpoint/Effect LC_{50}	**pH 6.84-7.63**	**Harmonic Mean 5.5**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.16	45 MM, 36 G	96H
1.32	3 MO, 0.21 G	96H
1.62	3 MO, 0.21 G	96H
1.64	50 MM	96H
1.77	50 MM	96H
1.84	3 MO, 0.21 G	72H
2.2	45 MM, 36 G	96H
2.29	45 MM, 36 G	96H
2.31	45 MM, 36 G	96H
2.51	3 MO, 0.21 G	72H
2.69	3 MO, 0.21 G	72H
2.71	3 MO, 0.21 G	24H
2.78	JUVENILE, 5 MO, 3.0 G, 7.0 CM	120H
2.81	1-2 G, JUVENILE	96H
2.89	50 MM	96H
3.08	PARR, 6.96 G, 8.6 CM	200H
3.16	ALEVIN, 20.8 MM, 0.10 G	96H
3.3	3 MO, 0.21 G	48H
3.35	50 MM	96H
3.68	2.36-3.01 G	96H
3.68	2.36-3.01 G	168H
4.06	3.9-6.8 CM FORK LENGTH	96H
4.45	SWIM-UP, 0.17 G	96H
4.45	SWIM-UP, 0.17 G	200H
4.62	0.5 G, JUVENILE	96H
4.66	130 MM	96H
4.77	3 MO, 0.21 G	96H
4.97	45 MM, 36 G	96H
5.06	45 MM, 36 G	96H

Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	**Arithmetic Mean** **18**
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	**Geometric Mean** **9**
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	**Harmonic Mean** **5.5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
5.17	3 MO, 0.21 G	48H
5.36	50 MM	96H
5.47	SWIM-UP, 0.23 G	200H
5.47	SMOLT, 68.19 G, 18.8 CM	200H
5.54	3 MO, 0.21 G	48H
5.59	50 MM	96H
5.92	8.8 G	96H
5.92	8.8 G	72H
5.96	3 MO, 0.21 G	72H
6.16	SWIM-UP, 0.23 G	96H
6.84	PARR, 11.58 G, 9.6 CM	200H
7.1	ALEVINS-BUTTONED-UP FRY	96H
7.17	JUVENILE, 41.6-45.8 MM/	96H
7.87	SMOLT, 32.46 G, 14.4 CM	200H
7.89	8.8 G	48H
7.99	136 MM	96H
8.21	135 MM	96H
8.43	JUVENILE, 6.42-6.66 MM/	96H
8.71	NR	408H
9.2	2.36-3.01 G	96H
9.92	SMOLT, 68.19 G, 18.8 CM	96H
9.92	SMOLT, 32.46 G, 14.4 CM	96H
10.46	NR	96H
11.97	PARR, 11.58 G, 9.6 CM	96H
12.12	ALEVIN, 14.3 MM, 0.01 G	96H
12.65	ALEVIN 29.8 MM, 0.24 G	96H
13.13	NR	215H
14.26	0.5 G, JUVENILE	96H
15.5	3 MO, 0.21 G	24H
15.54	40 MM	96H
16.85	1.0 G, 32 MM	96H

Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	**Arithmetic Mean** **18**
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	**Geometric Mean** **9**
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	**Harmonic Mean** **5.5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
21	3.9-6.8 CM FORK LENGTH	96H
23	PARR, 6.96 G, 8.6 CM	96H
23	SWIM-UP, 0.17 G	96H
23	SWIM-UP, 0.23 G	96H
23	PARR	96H
23	SMOLT, 32.46 G,	96H
23	PARR, 11.58 G, 9.6 CM	96H
23		96H
23	ADULT	96H
23	ALEVIN, 0.05 G	96H
23	ALEVIN	96H
25		96H
25.84	3 MO, 0.21 G	48H
31	130 MM	96H
41	ALEVIN, 20.8 MM, 0.10 G	96H
41	JUVENILE,	96H
41	ALEVIN 29.8 MM, 0.24 G	96H
43.5	1-2 G, JUVENILE	96H
43.5	0.5 G, JUVENILE	96H
44	3 MO, 0.21 G	96H
44	ALEVIN, 14.3 MM, 0.01 G	96H
44.4	8.8 G	96H
83.1	FRY, 0.14 G	7D
90	YEARLING	96H
140	JUVENILE	96H
211	FRY, 1.03 G	96H

Table 2.6.2.2.3.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 9.6-17.3° Celsius**	**Arithmetic Mean 5**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 29-410.5 mg/L $CaCO_3$**	**Geometric Mean 3**
Endpoint/Effect NOEC/Mortality/Growth/Reproduction	**pH 6.84-7.63**	**Harmonic Mean 2**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.80	50 MM	100D
1.25	JUVENILE	100D
1.29	50 MM	100D
2.10	JUVENILE	100D
2.15	50 MM	100D
2.34	L. Superior	
2.74	JUVENILE	100D
3.06	YEARLING, 50-70 G	
4.29	2 YR, FEMALE ADULT	60W
6.83	2 YR, FEMALE ADULT	
7.37	West Coast	100D
26.66	NR	10D

Table 2.6.2.2.3.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 5-20° Celsius**	**Arithmetic Mean 27**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 9.2-427 mg/L $CaCO_3$**	**Geometric Mean 4**
Endpoint/Effect NOEC/Mortality	**pH 6.6-8.28**	**Harmonic Mean 2**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.58	50 MM	100D
0.94	JUVENILE	100D
1.14	50 MM	100D
1.55	JUVENILE	100D
2.29	136 MM	1M
2.29	130 MM	96H
2.37	NR	1M
2.75	50 MM	100D
2.95	136 MM	1M
3.63	130 MM	96H
3.69	EGG	2M
3.83	YEARLING, 50-70 G	33M
3.86	JUVENILE	100D
5.17	1.0 G, 32 MM	96H
5.43	1.0 G, 32 MM	96H
11.5	EGGS	19M
12.8	EGGS	1M
41.55	NR	10D
407.7	NR	10D

Table 2.6.2.2.3.4 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 5-20° Celsius**	**Arithmetic Mean 21**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 20-390 mg/L $CaCO_3$**	**Geometric Mean 1.8**
Endpoint/Effect Growth	**pH 6.6-8.28**	**Harmonic Mean 0.3**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.02	EMBRYO	
0.10	NR	84D
0.47	ALEVIN	46D
0.59	18.2-23.5 CM, 51.2-114.9 G	112D
0.71	JUVENILE, 59 G	30D
0.71	JUVENILE, 59 G	30D
0.98	NR	84D
1	24 H, ALEVIN	13W
1.38	ALEVIN	46D
1.98	JUVENILE	30D
2.82	EGG-FRY	12W
3.59	EGG-FRY	12W
4	FINGERLING, 7.8 G	10W
4	FINGERLING, 7.8 G	10W
6.16	ALEVIN, 21 D	21D
6.4	ADULT, 375 G, 31.0 CM	178D
7.15	ADULT, 582 G	30D
7.15	ADULT, 582 G	30D
341	80 G	1W

Table 2.6.2.2.3.5 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 5-20° Celsius**	**Arithmetic Mean 79**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 10.1-320 mg/L $CaCO_3$**	**Geometric Mean 24**
Endpoint/Effect Physiological	**pH 6.6-8.28**	**Harmonic Mean 2**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.27	200-250 G	120D
1.98	JUVENILE	30D
12.7	NR	24H
67	20.01 CM FL, 101.54 G	48H
77.9	3-4 YR	7D
77.9	3-4 YR	24H
128	15-20 CM	24H
267	56 G	24H

Table 2.6.2.2.3.6 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 5-20° Celsius**	**Arithmetic Mean 1**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 44-250 mg/L $CaCO_3$**	**Geometric Mean 0.9**
Endpoint/Effect Reproductive	**pH 6.6-8.28**	**Harmonic Mean 0.8**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.56	270 D, ADULT, FEMALE	65W
0.63	270 D, ADULT, FEMALE	65W
1.13	YEARLING, 50-70 G	33M
1.96	270 D, ADULT, FEMALE	80W

Cadmium Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to cadmium, NMFS added an additional step to its analysis for cadmium to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 2 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.3.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.3.1, predicts a magnitude of effect ranging from a low of an $LC_{0.5}$ at a concentration of 211 µg/L to a high of an LC_{86} at a concentration of 1.16 µg/L (Table 2.6.2.2.3.7). In other words, the acute criterion of 2 µg/L has an equivalent toxicity potential predicted to kill 0.5 percent to 86 percent, with a median toxicity potential of an $LC_{12.7}$, of the exposed test population, and therefore by inference, field-exposed individuals.

Table 2.6.2.2.3.7 Relative percent mortality analysis for salmonid fishes, eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 9.6-17.3° Celsius**	
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 9.2-410.5 mg/L $CaCO_3$**	
Endpoint/Effect LC_{50}	**pH 6.84-7.63**	
Concentration Micrograms Liter^{-1}	**Relative Percent Mortality (acute criterion/LC_{50})**	
1.16	86.2	
1.32	75.8	
1.62	61.7	
1.64	61.0	
1.77	56.5	
1.84	54.3	
2.2	45.5	
2.29	43.7	
2.31	43.3	
2.51	39.8	
2.69	37.2	
2.71	36.9	
2.78	36.0	
2.81	35.6	

Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
2.89	34.6	
3.08	32.5	
3.16	31.6	
3.3	30.3	
3.35	29.9	
3.68	27.2	
3.68	27.2	
4.06	24.6	
4.45	22.5	
4.45	22.5	
4.62	21.6	
4.66	21.5	
4.77	21.0	
4.97	20.1	
5.06	19.8	
5.17	19.3	
5.36	18.7	
5.47	18.3	
5.47	18.3	
5.54	18.1	
5.59	17.9	
5.92	16.9	
5.92	16.9	
5.96	16.8	
6.16	16.2	
6.84	14.6	
7.1	14.1	
7.17	13.9	
7.87	12.7	
7.89	12.7	
7.99	12.5	

Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
8.21	12.2	
8.43	11.9	
8.71	11.5	
9.2	10.9	
9.92	10.1	
9.92	10.1	
10.46	9.6	
11.97	8.4	
12.12	8.3	
12.65	7.9	
13.13	7.6	
14.26	7.0	
15.5	6.5	
15.54	6.4	
16.85	5.9	
21	4.8	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
25	4.0	
25.84	3.9	
31	3.2	
41	2.4	
41	2.4	

Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
41	2.4	
43.5	2.3	
43.5	2.3	
44	2.3	
44	2.3	
44.4	2.3	
83.1	1.2	
90	1.1	
140	0.7	
211	0.5	

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for cadmium, which implies that listed species exposed to waters equal to criteria concentrations will not be protected from acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for cadmium, which implies that listed species exposed to waters equal to criteria concentrations will be protected from acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Cadmium occurs naturally in the aquatic environment, and is considered one of the most toxic of metals to fish (Sorensen 1991). Uses of cadmium include electroplating, pigments, plastic stabilizers, batteries, and electronic components. In aquatic systems, cadmium is taken up quickly by sediments but is readily remobilized through a variety of physical, chemical, and biological processes, and can even be transported from aquatic to terrestrial food webs by emerging insects (Currie *et al.* 1997). Cadmium is a known teratogen, carcinogen and a probable mutagen to which freshwater organisms are considered the most sensitive. Effects of cadmium toxicity on freshwater organisms include spinal deformities; inhibited respiration; blood plasma and other hematological changes, decreased growth, inhibited reproduction and immune response; temporary immobility; and population alterations. Salmonid species are particularly sensitive to cadmium compared to other fish species (Sorensen 1991,

Brent and Herricks 1998, Sanchez-Dardon *et al.* 1999). Chronic sublethal exposure to cadmium does not appear to significantly influence growth in juvenile salmonids (Hollis *et al.* 2000b).

Toxicity of cadmium to aquatic organisms varies with the type and life stage of organisms, presence of other toxicants, duration of exposure, and hardness. Acute mechanisms of cadmium toxicity to fish do not appear to be the same as chronic mechanisms. In acute tests cadmium accumulates in gill tissue to a greater extent than elsewhere, whereas in chronic tests at lower concentrations, cadmium accumulates more in liver and kidney tissue. The principal acute effect is gill toxicity leading to an aquatic organism's inability to breathe. Cadmium toxicity increases with water temperature (Moore and Ramamoorthy 1985, Eisler 1985a, EPA 1985c, Sorensen 1991), which is known to also stress listed species in many parts of Oregon. The presence of zinc, which has similar chemical properties, and selenium have been shown to antagonize cadmium toxicity, whereas other metals do not appear to compete with cadmium for enzyme receptors in aquatic organisms.

Stubblefield *et al.* (1999) determined that adult rainbow trout that were acclimated to elevated cadmium levels would survive sudden increases to higher concentrations at a higher rate than fish that were not acclimated. The non-acclimated fish exhibited an incipient lethal level (ILL: threshold level of exposure to toxic substances beyond which 50% of a test population of organisms cannot survive) of 6.1µg/L at a hardness of 280 mg/L, which is below the proposed acute criterion. However, the ILL was determined to occur after 187 hours of exposure, which is more than the maximum permitted under the proposed criterion (96 hours under the chronic criterion). On the basis of this study, therefore, an adverse effect would be expected at the proposed concentration if the concentrations occurred unmonitored or uncorrected for more than 7 days in waters where background concentrations are well below the chronic criterion. Young-of-year rainbow trout fared better and were determined to be less sensitive than adults (Stubblefield *et al.* 1999). Older (age 1+) fish were not tested, but could exhibit a response between that of the young of year and adult test fish, and thus also be susceptible to acute toxicity at cadmium levels below the proposed acute criterion when they are not suitably acclimated to background levels.

Birge *et al.* (1981) determined reduced survival (52% vs. 90% for control) of 4 day old larvae of rainbow trout after their parents were exposed to a concentration of 0.2 µg/L at 102 mg/L hardness for 18 months, which is well below the proposed chronic criterion. The exposed parents had tissue concentrations that were roughly seven times that of the control fish, indicating the potential for bioaccumulative effects on subsequent reproductive success.

Cadmium has been shown to cause neurotoxic effects in fish. These neurotoxic effects may manifest themselves through altered behavior, which in turn may predict more serious effects including reduced growth, reproductive failure, and death. Hyperactivity probably is the most widely observed maladaptive behavior reported from cadmium exposed fish, with several reports involving a variety of fish species during long-term cadmium exposures. Most fish that exhibited hyperactive behavior in long-term exposures ultimately died. Hyperactivity is detrimental to small fish because it makes them more likely to be seen and attacked by predatory fish. Similarly, hyperactive predatory fish have lower success rates in detecting, orienting to, attacking, and swallowing prey.

Cadmium is bioconcentrated by organisms but is not biomagnified through the food chain (Eisler 1985a as cited in EPA 2008). Toxicity of cadmium to aquatic organisms varies with water hardness, alkalinity, the type and life stage of organisms, presence of organic matter, presence of other toxicants, and the duration of exposure (EPA 1999 as cited in EPA 2008). Cadmium is a known teratogen, carcinogen, and a probable mutagen to freshwater organisms (Eisler 1985a as cited in EPA 2008). Effects of cadmium toxicity to freshwater organisms include spinal deformities, inhibited respiration, immune response, temporary immobility, decreased growth, inhibited reproduction, decreased survival, and population alterations (Sorensen 1991, Eisler 1985a, Brent and Herricks 1998, Sanchez-Dardon *et al.* 1999 as cited in EPA 2008). A known mechanism of cadmium toxicity to fish is suppression of calcium uptake (Verbost *et al.* 1987 as cited in EPA 2008). Calcium is vital for growth in fish (Pelgrom *et al.* 1997) as cited in EPA 2008, and bone repair mechanisms are probably inhibited due to the hypocalcemic effect of cadmium (DWAF, 1996 as cited in EPA 2008).

Cadmium bioaccumulates in numerous fish species including salmonids, where tissue concentrations reflect exposure levels and duration, hardness, and presence of other ions (*e.g.*, zinc). Besser *et al.* (2001) determined a mean bioaccumulation factor of 3.4 from aquatic macroinvertebrates to trout. Omnivorous fish tend to accumulate higher levels of cadmium than carnivorous fish, such as salmonid fishes, and bottom-feeding fish tend to accumulate more cadmium than free-swimming fish feeding in the water column. Evidence suggests that significant biomagnification is exhibited predominantly by species at lower trophic levels in aquatic ecosystems, whereas fish are able to depurate cadmium rapidly (Eisler 1985a, Sorensen 1991). Uptake occurs through both dissolved and particulate forms (Enk and Mathis 1977, Sorensen 1991). Cadmium tends to form stable complexes with metallothionein that have long half-lives and a tendency to accumulate with age in exposed organisms. Accumulation appears to occur primarily in the gills, liver, kidneys, and gastrointestinal tract (Sorenson 1991, Besser *et al.* 2001. Hollis *et al.* 2001). As such, long lived species tend to be at a higher risk from chronic low-level dietary cadmium exposure. Rainbow trout exposed to cadmium have been determined to contain residues in kidney, spleen, gill, muscle, and bone tissues that increase in concentration with duration of exposure (Camusso and Balestrini 1995). In contrast, Saiki *et al.* (1995) found no evidence of cadmium biomagnification in steelhead on the Upper Sacramento River. McGeer *et al.* (2000) reported evidence that cadmium accumulates inside rainbow trout continuously over time with continued exposure, because it not as actively regulated as copper and zinc are by the organism. McGeer used concentrations below the proposed criteria. It is unknown whether bioaccumulation also occurs when concentrations are below the proposed criteria for extended periods, but the possibility appears to exist.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for cadmium is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Amphipods are sometimes abundant in lakes and slow-moving rivers. Amphipods are benthic crustaceans that occupy an intermediate position in aquatic food webs between detritus and predators, such as salamanders and salmonids (Mathias 1971). Aquatic macroinvertebrates, which serve as significant food sources for early life stages of listed species as well as for other aquatic organisms that are in turn prey items, are sensitive to both dissolved and particulate cadmium. Invertebrate communities in rivers appear to respond to

elevated cadmium levels in sediments and water by changing composition to pollution-tolerant taxa, rather than by reducing overall biomass (Canfield *et al.* 1994, Clements and Kiffney 1994). Hare and Shooner (1995) determined that population densities of the two most abundant colonizing insects (chironomidae) in a small lake were unrelated to cadmium gradients in sediments, even though they accumulated the metal in proportion to its concentration in the sediment. Interstitial water cadmium concentrations ranged up to 17 µg/L, suggesting that the two taxa were relatively insensitive to exposure to cadmium levels less than that. Larvae of another chironomid were negatively correlated with cadmium gradient. These tests suggest that the lower abundance at high concentrations is more likely due to toxicity effects than avoidance of cadmium-rich sediments. It is not clear if these effects also occur at water-borne cadmium levels that are below the proposed chronic criterion, although this possibility should not be discounted because of the potential for bioaccumulation.

Cadmium contained in bed sediments appears to be bioavailable to benthic invertebrates, was found to be elevated in benthic invertebrates in field studies conducted in metals-contaminated streams (*e.g.*, Enk and Mathis 1977, Woodward *et al.* 1994). Kiffney and Clements (1996) determined an inverse relation existed between aquatic macroinvertebrate body size and survival at water-borne cadmium levels in excess of the proposed acute criterion, which could partially counter the effects of bioaccumulation when invertebrates are exposed to contaminated sediments. Indirect effects of elevated cadmium levels to listed species therefore include reduced production of larger invertebrate taxa that could influence the availability of food for larger juvenile salmonids, and ingestion of bioconcentrated cadmium by fry and juveniles of all sizes. It is unknown if similar effects occur at concentrations below the proposed chronic criterion.

Salmonids and other fish readily prey upon amphipods, probably consuming them in rough proportion to their abundance relative to other vulnerable invertebrates. For example, in the lower Snake River in Washington and Idaho, amphipods contributed 2.7 and 7.9 percent of identifiable prey categories found in the stomachs of juvenile Chinook salmon and steelhead, respectively from Lower Granite Reservoir, (7^{th} and 5^{th} most important prey categories, respectively) (Karchesky and Bennett 1999).

One invertebrate, the amphipod *Hyalella azteca*, seems particularly sensitive to cadmium. It is the only species with a species mean chronic value that is lower than the NTR of 2.2 µg/L. Six chronic tests with *Hyalella* were analyzed by Mebane (2006). In all six tests, adverse effects would be expected at a concentration of 1 µg/L. Mebane (2006) attempted to evaluate several lines of evidence to evaluate if the predicted effects to this species would have appreciable adverse effects on fish populations or other indirect effects on aquatic ecosystems in the Pacific Northwest. These efforts included (1) reviews of role of *Hyalella azteca* in aquatic food chains, (2) occurrences of *Hyalella azteca* in waters with elevated cadmium concentrations, and (3) simulating effects of cadmium to a natural, coldwater *Hyalella azteca* population.

Potential effects of cadmium at chronic criteria concentrations on wild populations of *Hyalella azteca* were also estimated using mathematical population models that integrate toxicity testing results with ecological theory. The modeling predicted that at the NTR chronic criteria (2.2 µg/L at the scenario hardness of 280 mg/L), quasi-extinction of the population was highly likely, with >80% probability of a >98% population decline occurring during the 6-year modeling scenario.

Applying these modeling results to the Oregon chronic criterion (0.25 µg/L) results in a marginal increased extinction risk.

Toxicity to Food Organisms Summary. The available evidence indicates that the chronic criterion for cadmium is likely to result in sublethal effects to listed species considered in this opinion.

Summary of Effects: Cadmium. The available evidence for indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (high intensity), reduced growth (moderately-high-intensity), impairment of essential behaviors related to successful rearing and migration (moderate intensity), physiological trauma (moderate intensity), and reproductive failure (moderate intensity).

2.6.2.2.4. Chromium (III)

Chromium (III) Criteria. The proposed acute and chronic criteria for chromium (III) are 570 µg/L and 74 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.4.1 through 2.6.2.2.4.2 report toxicity data from the ECOTOX database for freshwater CR (III), except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.4.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater chromium III.

Criterion Freshwater Chromium III		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 570 Micrograms Liter^{-1}	**Temperature 11.9-14.5° Celsius**	**Arithmetic Mean 10099**
Criterion Concentration Chronic 74 Micrograms Liter^{-1}	**Hardness 25-44 mg/L $CaCO_3$**	**Geometric Mean 9825**
Endpoint/Effect LC_{50}	**pH 5.45-7.33**	**Harmonic Mean 9558**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
7762	NR	96H
12436	NR	96H

Table 2.6.2.2.4.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater chromium III.

Criterion Freshwater Chromium III		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **570 Micrograms Liter^{-1}**	**Temperature** **11.9-14.5° Celsius**	**Arithmetic Mean** **53**
Criterion Concentration Chronic **74 Micrograms Liter^{-1}**	**Hardness** **25 mg/L $CaCO_3$**	**Geometric Mean** **53**
Endpoint/Effect **NOEC/Growth/Mortality**	**pH** **5.45-7.33**	**Harmonic Mean** **53**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
53	NR	72H

2.6.2.2.5 Chromium (VI)

Chromium (VI) Criteria. The proposed acute and chronic criteria for chromium (VI) are 570 µg/L and 74 µg/L, respectively.

Tables 2.6.2.2.5.1 through 2.6.2.2.5.2 report toxicity data from the ECOTOX database for freshwater CR (VI), except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.5.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater chromium VI.

Criterion Freshwater Chromium VI		Data Set ECOTOX
Criterion Concentration Acute **16 Micrograms Liter^{-1}**	**Temperature** **3.5-19° Celsius**	**Arithmetic Mean** **98129**
Criterion Concentration Chronic **11 Micrograms Liter^{-1}**	**Hardness** **34-46 mg/L $CaCO_3$**	**Geometric Mean** **68333**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **7-8**	**Harmonic Mean** **44884**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
12079	NR	96H
27201	NR	96H
27496	NR	96H
37905	NR	96H
69722	NR	96H
74239	NR	96H

Criterion Freshwater Chromium VI		Data Set ECOTOX
Criterion Concentration Acute 16 Micrograms Liter^{-1}	**Temperature 3.5-19° Celsius**	**Arithmetic Mean 98129**
Criterion Concentration Chronic 11 Micrograms Liter^{-1}	**Hardness 34-46 mg/L $CaCO_3$**	**Geometric Mean 68333**
Endpoint/Effect LC_{50}/Mortality	**pH 7-8**	**Harmonic Mean 44884**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
98200	NR	96H
109002	NR	96H
141408	NR	96H
201310	NR	96H
280852	NR	96H

Table 2.6.2.2.5.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater chromium VI.

Criterion Freshwater Chromium VI		Data Set ECOTOX
Criterion Concentration Acute 16 Micrograms Liter^{-1}	**Temperature 3.5-19° Celsius**	**Arithmetic Mean 100**
Criterion Concentration Chronic 11 Micrograms Liter^{-1}	**Hardness 34-46 mg/L $CaCO_3$**	**Geometric Mean 52**
Endpoint/Effect NOEC/Growth	**pH 7-8**	**Harmonic Mean 24**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
9.6	EG-JV	7M
10	EG-JV	7M
10	EG-JV	7M
13	LV-JV	110D
13	LV-JV	110D
49	NR	
49	NR	
192	NR	
192	NR	
192	NR	
192	NR	
192	NR	
192	NR	

Chromium III and Chromium VI Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to chromium (III) and chromium (VI), NMFS added an additional step to its analysis for chromium (III) and chromium (VI) to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 570 µg/L for chromium (III) and 16 µg/L for chromium (VI) and dividing it by each LC_{50} concentrations in Table 2.6.2.2.4.1 and Table 2.6.2.2.5.1, respectively, to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.4.1 and Table 2.6.2.2.5.1, respectively, predicts a magnitude of effect ranging from a low of an $LC_{2.3}$ at a concentration of 12,436 µg/L to a high of an $LC_{3.7}$ at a concentration of 7,762 µg/L for chromium (III), and a magnitude of effect of an LC_{zero} at a concentration of 12,074 µg/L and 280,852 µg/L for chromium (VI). In other words, the acute criterion of 570 µg/L for chromium (III) has an equivalent toxicity potential predicted to kill 2.3 percent to 3.7 percent, with a median toxicity potential of an LC_3, of the exposed test population, and therefore by inference, field-exposed individuals. The acute criterion of 16 µg/L for chromium (VI) has an equivalent toxicity potential predicted to kill zero percent.

In summary, none of toxicity studies reported concentrations that are less than the acute criterion concentration for chromium (III), which implies that listed species exposed to waters equal to criterion concentrations may not suffer acute toxic effects. Conversely, the single toxicity data reported for chronic effects is less than the chronic criterion concentration for chromium (III), which implies that listed species exposed to waters equal to criteria concentrations will suffer chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute and chronic toxic effects.

None of the toxicity studies reported concentrations that are less than the acute criterion for chromium (VI), which implies that listed species exposed to waters equal to the acute criterion concentration may not suffer acute toxic effects. A number of toxicity studies reported concentrations that are less than the chronic criteria for chromium (VI), and a number of toxicity studies reported concentrations that are greater than the chronic criterion for chromium (VI), which implies that listed species exposed to waters equal to the chronic criterion concentration will suffer chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration may not suffer acute toxic effects, but will suffer chronic toxic effects.

Sublethal Effects (Chromium III and Chromium VI). Chromium (III) (the trivalent form) is much less toxic than chromium (VI) (the hexavalent form), which is a strong oxidizing agent and reduces readily to the former. Younger life stages of aquatic biota tend to be more sensitive to the toxic effects of chromium (VI). Effects of toxicity include abnormal enzyme activities, altered blood chemistry, lowered resistance to disease, reduced growth, behavioral modifications, disrupted feeding, cell damage in the gills and other tissues, and osmoregulatory upset in outmigrating smolts. The toxicity of chromium is influenced by pH, water temperature, concentrations of other contaminants, and fish age and sex (EPA 1980d, Eisler 1986).

chromium (III) toxicity is influenced by water hardness. It is unclear is the same if true for chromium (VI), which is significantly more toxic. Hexavalent chromium exists in solution in an anionic rather than cationic form, and therefore does not precipitate in an alkaline solution.

The acute standards for chromium (III) are unique from analogous standards for the other metals of concern because the total recoverable to dissolved conversion factor (0.316) is substantially smaller. Depending on the sampling location and the receiving water characteristics (that may promote dissolution of particulate chromium), this means that the proposed criterion could permit discharge of total recoverable chromium (III) at levels that result in higher than assumed, and potentially toxic, dissolved levels downstream.

Chromium may be present in the environment in both inorganic and organic forms. Inorganic forms do not biomagnify; it is unknown whether organic forms of chromium biomagnify (Eisler 1986). Chromium toxicity to aquatic biota is significantly influenced by abiotic variables such as water hardness, temperature, pH, salinity, species, life stage, and presence of mixtures (Eisler 1986). Sensitivity to chromium varies widely, even among closely related species (Eisler 1986). Effects of chromium toxicity to freshwater organisms include reduced survival in freshwater invertebrates (including molluscs), and reduced growth, reduced disease resistance, behavioral modifications, disrupted feeding, cell damage in the gills, osmoregulatory upset in outmigrating smolts, and reduced reproduction and survival in freshwater fish (Anestis and Neufeld 1986, Eisler 1986 and EPA 1999).

Hexavalent chromium is more toxic than the trivalent form because its oxidizing potential is high and it easily penetrates biological membranes (Steven *et al.* 1976, Taylor and Parr 1978 as cited in EPA 2008). At high concentrations, both forms of chromium can be a mutagen, teratogen, and carcinogen (Eisler 1986b as cited in EPA 2008). Although CrIII is the most common form found in nature, the known harmful effects of chromium is speculated to be related to the reduction of hexavalent chromium (chromium VI) to chromium III intracellularly as it crosses the cell membrane and forms complexes with intracellular macromolecules (Danielsson *et al.* 1982, R.O.W. Sciences, 1997 as cited in EPA 2008).

There are more toxicity test data available for the hexavalent form of chromium (VI), probably reflecting its greater toxicity. Insufficient data are available to evaluate the potential harm of the chromium (III) criterion for salmonids specifically. Toxicity data for salmonid fishes indicate that acute and chronic toxicity of chromium (VI) is likely to occur to juvenile salmonids when dissolved concentrations are at or below the chromium (VI) numeric criteria.

Billard and Roubaud (1985) determined that the viability of rainbow trout sperm (but not ova) were adversely affected when exposed directly to a chromium (VI) concentration equal to 5 µg/L, which is well below the chronic criterion of 11 µg/L. Reproductive effectiveness is likely to be reduced if this water concentration occurs during spawning.

There is evidence that invertebrates and fishes bioaccumulate hexavalent chromium when exposed to ambient water concentrations that are above the chronic criterion. Uptake is influenced by water temperature, pH, other contaminant concentrations, fish age and sex, and tissue type (EIFAC 1983, Eisler 1986). Calamari *et al.* (1982) determined that liver, kidney, and muscle tissue concentrations of chromium were elevated in rainbow trout after 30, 90, and 180 days of exposure to 200 µg/L. The fish subsequently were able to depurate some, but not all, of the accumulated chromium within 90 days after exposure ended. At higher concentrations (>2000 µg/L), chromium is known to also accumulate in gill and digestive tract tissues of rainbow trout (Eisler 1986). Gill accumulation appears to continue with exposure, whereas the other tissues may achieve equilibrium in 2 to 4 days. Residues tend to remain high in the liver and kidneys in test fish during post-exposure periods. Eisler (1986) reported that tissue concentrations in excess of 4 mg/kg dry weight were presumptive evidence of chromium contamination, but the biological significance was not clear.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for chromium (III) and chromium (VI) is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Aquatic invertebrates other than cladocerans have been determined in a limited number of studies to experience acute and chronic effects at concentrations below the acute and chronic criterion, respectively, for both chromium (III) and (VI). Data in EPA (1980d) indicate reduced survival and reproductive impairment of daphnids at chromium (III) and (VI) concentrations as low as 4 and 10 µg/L, respectively. These concentrations are less than the proposed chronic criterion for each respective valency. Most studies have determined toxicity to daphnids occurs at higher concentrations than the criterion, however. Data summarized in EPA (1980d), EIFAC (1983), and Eisler (1986) suggest that other invertebrate taxa that juvenile fishes may feed on generally died at chromium (III) and (VI) concentrations that are well above the acute criterion. More recently, Canivet *et al.* (2001) determined 240-hour chromium (VI) LC_{50}s for larvae of a trichopteran and an ephemeropteran that were well above the proposed acute and chronic criteria.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for chromium (III) and chromium (VI) are unlikely to appreciably affect invertebrate productivity and abundance.

S*ummary of Effects: Chromium (III) and Chromium (VI).* The available evidence for chromium (III) and chromium (VI), respectively, indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity, for chromium III, and low intensity for chromium VI) and reduced growth (moderately-high-intensity, for chromium III and chromium VI).

2.6.2.2.6 Copper

Copper Criteria. The proposed acute and chronic criteria for copper are 13 µg/L and 9 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.6.1 through 2.6.2.2.6.11 report toxicity data from the ECOTOX database for freshwater copper, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters, the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.6.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-16° Celsius**	**Arithmetic Mean 145**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 8-495 mg/L $CaCO_3$**	**Geometric Mean 96**
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.0**	**Harmonic Mean 59**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
5.70	4.2 G, 7.4 CM	96H
5.96	4.2 G, 7.4 CM	96H
9.14	YEARLING, 10-18 MO	96H
9.14	LARVAE	96H
11.56	PA	4D
12.85	10 G	96H
18.03	2.6 G	96H
19.32	1.7 G	96H
20.62	YEARLING, 10-18 MO	96H
21.20	LARVAE	96H
23.90	4.3 G	96H
25.45	PA	4D
25.49	3 MO, 1.35 G	96H
25.65	25.6 G, 13.4 CM	96H
27.55	FRY, 0.139 G, 2.87 CM	96H
30.13	2-3 YR	96H
30.48	176 MM	96H
31.26	FRY, 0.66 G	96H
31.61	2.2 G	96H
32.86	ALEVIN	96H

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
33.35	2.7 G	96H
33.41	2.5 G, 6.1 CM	96H
34.31	1.0 G	96H
35.15	ALEVIN	96H
36.39	FRY, 0.138 G, 2.96 CM	96H
37.88	4.4 G, 7.7 CM	96H
38.18	YEARLING, 10-18 MO	96H
38.58	160 MM	96H
39.63	3.1 G	96H
40.66	FRY, 0.87 G	96H
42.63	1.4 G	96H
42.83	1.0 G	96H
43.86	FY	4D
43.88	SMOLT, 5.5 G	96H
44.23	0.71 G	96H
45.86	9.7 G, 8.8 CM	96H
45.87	5.2 G, 8.5 CM	96H
46.38	3 MO, 1.35 G	96H
47.01	AD, MALE	96H
48.10	EM	96H
48.36	SMOLT, 4.69 G, 8.35 CM	96H
50.59	9.4 G, 9.2 CM	96H
51.40	9.4 G, 9.2 CM	96H
52.79	3 MO, 1.35 G	96H
52.79	24.9 G, 13.5 CM	96H
52.86	FRY, 1 G	96H
52.96	ALEVIN	96H
53.76	3.9-6.8 CM FORK LENGTH	96H
56.10	SWIM-UP, 0.17 G	96H
56.39	FRY, 1 G	96H
59.23	SMOLT, 4.8 G	96H

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
59.70	FRY, 0.132 G, 2.95 CM	96H
59.89	FRY, 0.136 G, 2.97 CM	96H
61.06	ALEVIN	96H
61.68	ALEVIN	96H
61.87	PA	4D
63.79	4.4 G, 8.1 CM	96H
64.68	3.2 G, 7.0 CM	96H
65.18	FY	4D
65.54	PA	4D
65.81	PA	4D
66.26	1.8 G	96H
67.63	YEARLING, 10-18 MO	96H
68.31	22.6 G, 11.8 CM	96H
69.01	4.0 G, 7.3 CM	96H
70.11	AD, MALE, ~2.7 KG	96H
70.46	JUVENILE, 5-6 WK, 0.85 G	96H
70.53	5.7 G, 8.9 CM	96H
71.12	SU, <3 mo, 32.1 MM, 0.23 G	96H
71.23	2.2 G	96H
71.38	JUVENILE, 7-8 WK, 0.20 G	96H
72.13	FRY, 1 G	96H
72.85	SMOLT, 4.63 G, 8.07 CM	96H
73.87	SU, <3 mo, 29.1 MM, 0.23 G	96H
73.96	167 MM	96H
74.56	1.1 G	96H
75.30	SMOLT, 68.19 G, 18.8 CM	96H
79.51	FINGERLING, 2.31 G, 6.61 CM	96H
81.10	JV, 14 mo	96H
84.84	PA	4D
86.51	YEARLING, 10-18 MO	96H
86.89	SMT	4D

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
87.12	3 MO, 1.35 G	96H
87.55	ALEVIN	96H
88.37	11.3 G, 9.7 CM	96H
88.91	ALEVIN	96H
90.44	3 MO, 1.35 G	96H
92.43	4.3 G	96H
92.74	4.4 G, 7.7 CM	96H
93.28	ALEVIN	96H
95.28	9.7 G, 8.8 CM	96H
99.44	PARR, 6.96 G, 8.6 CM	96H
99.68	2.7 G, 6.8 CM	96H
99.68	FINGERLING, 3.90 G, 7.17 CM	96H
99.68	25.6 G, 13.4 CM	96H
101.29	PA	4D
107.35	SMT	4D
108.15	0.80 G	96H
108.89	24.9 G, 13.5 CM	96H
111.19	FY, 2.36-3.01 G	96H
112.21	PARR, 11.58 G, 9.6 CM	96H
113.63	JV, 14 mo	96H
113.77	SU, <3 mo, 30.4 MM, 0.26 G	96H
114.29	11.5 G, 9.9 CM	96H
122.21	3.2 G	96H
123.91	4.9 CM	96H
124.94	2.1 G, 6.0 CM	96H
128.87	1.5 G	96H
130.72	JUVENILE, 18-22 WK, 0.87 G	96H
133.67	4.4 G, 8.1 CM	96H
138.04	1.6 G	96H
138.78	FRY, 1 G	96H
140.88	5.2 G, 8.5 CM	96H

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
145.69	11 CM, 13 G	96H
147.81	FRY	96H
148.58	1 G	96H
149.08	100.4(90-115)MM TL,10.6(7.5-14.5) G	96H
150.03	ALEVIN, NEWLY HATCHED	96H
150.52	ALEVINS-BUTTONED-UP FRY	96H
155.59	3 MO, 1.35 G	96H
163.37	16.47 CM FL, 53.85 G	96H
163.44	SU, <3 mo, 30.1 MM, 0.25 G	96H
171.44	2.7 G, 6.8 CM	96H
174.10	3.2 G, 7.0 CM	96H
174.36	JUVENILE	96H
177.75	JUVENILE, 7-10 WK, 0.60 G	96H
179.14	SU, <3 mo, 34.4 MM, 0.29 G	96H
179.91	3 MO, 1.35 G	96H
181.82	6.6 G	96H
183.34	FRY, 1 G	96H
184.58	JUVENILE, 6 G	96H
185.37	SU, <3 mo, 28.4 MM, 0.23 G	96H
189.35	ALEVIN	96H
194.30	3.2 G, 6.9 CM	96H
194.76	SU, <3 mo, 33.4 MM, 0.25 G	96H
199.96	JUVENILE, 7-8 WK, 0.34 G	96H
201.19	SMOLT, 32.46 G, 14.4 CM	96H
210.45	JUVENILE, 10-12 WK, 0.41 G	96H
212.83	FRY	96H
217.16	JUVENILE,29.1G WET WT,6.76 G DRY WT	96H
217.16	SMOLT, 5.5 G	96H
222.22	0.90 G	96H
227.44	SWIM-UP, 0.23 G	96H
228.59	ALEVIN, NEWLY HATCHED	96H

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
229.06	FRY	96H
233.38	FINGERLING, 2.13 G, 6.67 CM	96H
240.00	ADULT, 16-18 CM	96H
240.02	18.7 G, 11.8 CM	96H
244.76	2.36-3.01 G	96H
250.22	5.7 G, 8.9 CM	96H
254.62	ALEVIN	200H
255.80	3 MO, 1.35 G	96H
264.28	PA	4D
266.36	FY, 2.36-3.01 G	96H
271.32	2.1 G, 6.0 CM	96H
274.31	3.2 G, 6.9 CM	96H
288.82	SU, <3 mo, 30.0 MM, 0.25 G	96H
289.33	12-16 CM	96H
301.90	3.2 G	96H
310.51	JUVENILE, 18-22 WK, 0.47 G	96H
313.32	FINGERLING, 3.28 G, 7.26 CM	96H
322.75	3 MO, 1.35 G	96H
326.37	3300 MG	96H
333.58	11.5 G, 9.9 CM	96H
346.63	JUVENILE, 10-12 WK, 0.81 G	96H
355.82	1.4 G	96H
376.54	YEARLING, 10-18 MO	96H
404.21	ALEVIN	96H
447.01	1.5 G	96H
447.48	ALEVIN, 0.05 G	96H
467.01	JUVENILE,3.9 G WET WT,0.94 G DRY WT	96H
475.90	1 G	96H
489.25	ALEVIN	96H
533.72	3 MO, 1.35 G	96H
533.72	JUVENILE,176 G WET WT,46.0 G DRY WT	96H

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
599.98	FRY, 1.60 G	96H
600.44	SMOLT, 5.5 G	96H
1160.10	2.6 G	96H

Table 2.6.2.2.6.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **58**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **16-405 mg/L $CaCO_3$**	**Geometric Mean** **35**
Endpoint/Effect **NOEC/Growth**	**pH** **4.7-8.0**	**Harmonic Mean** **25**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
6.57		
8		
9.5	MX, EG-YE, EXPOSED OR UNEXPOSED PAR	8M
11.4		
12	EGGS	6M
12	SACFRY, 9-11 D, 102.4-110.3 MG WT	15D
12	NR	24M
13.14		
14	FY OR SMT	30D
16	FY OR SMT	10D
16		
17	PA	29D
17.91	MX, EG-YE, EXPOSED OR UNEXPOSED PAR	8M
18	PA	8D
18	PA	29D

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **58**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **16-405 mg/L $CaCO_3$**	**Geometric Mean** **35**
Endpoint/Effect **NOEC/Growth**	**pH** **4.7-8.0**	**Harmonic Mean** **25**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
18	SWIM-UP, 0.23 G	96H
20	3 MO, 1.35 G	96H
20	3 MO, 1.35 G	96H
20.8		
21	PA	8D
21	PA	29D
21	PA	30D
21	FY OR SMT	60D
21.49		
22	PA	60D
22		
22.3		
23	SMOLT, 32.46 G, 14.4 CM	96H
24	ALEVIN, 0.05 G	96H
25	SACFRY, 9-11 D, 102.4-110.3 MG WT	15D
28	PA	60D
30	3 MO, 1.35 G	96H
30	3 MO, 1.35 G	96H
35	PARR, 11.58 G, 9.6 CM	96H
38	PA	9D
39.21		
40	PA	8D
40	FRY, 0.87 G	96H
41	FRY, 0.66 G	96H
41.47		
42.04		
50	3 MO, 1.35 G	96H
50	3 MO, 1.35 G	96H
54.69	FY OR SMT	60D
70.5	PA	60D
75	8 mo	10D
75	8 mo	10D

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **58**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **16-405 mg/L $CaCO_3$**	**Geometric Mean** **35**
Endpoint/Effect **NOEC/Growth**	**pH** **4.7-8.0**	**Harmonic Mean** **25**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
78.1	PA	60D
79	8 mo	10D
95	SMOLT, 4.69 G, 8.35 CM	96H
100	3 MO, 1.35 G	96H
100	3 MO, 1.35 G	96H
150	3 MO, 1.35 G	96H
200	FRY, 0.136 G, 2.97 CM	96H
200	3 MO, 1.35 G	96H
202	FINGERLING, 3.90 G, 7.17 CM	96H
213	FRY, 0.132 G, 2.95 CM	96H
216	SMOLT, 4.63 G, 8.07 CM	96H
240	SMOLT, 4.8 G	96H
312	8 mo	10D

Table 2.6.2.2.6.3 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-18° Celsius**	**Arithmetic Mean** **91**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **135 mg/L $CaCO_3$**	**Geometric Mean** **91**
Endpoint/Effect **Behavioral**	**pH** **4.7-8.54**	**Harmonic Mean** **91**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
65.8	SACFRY,9-11 D,102.4-110.3 MG WET WT	15D

Table 2.6.2.2.6.4 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set 3**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 6.9-16.5° Celsius**	**Arithmetic Mean 6**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 20-240 mg/L $CaCO_3$**	**Geometric Mean 2**
Endpoint/Effect Behavioral/Olfaction	**pH 7.2-7.6**	**Harmonic Mean 0.98**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.18	JUVENILE	3H
0.59	JUVENILE	3H
0.75	JUVENILE	20MIN
0.79	JUVENILE	3H
1.6	JUVENILE	20MIN
2	JUVENILE	21D
2.1	JUVENILE	3H
2.4	JUVENILE	20MIN
5	JUVENILE	6D
10	ADULT	INDEFINITE
20	ADULT	INDEFINITE
25	ADULT	INDEFINITE

Table 2.6.2.2.6.5 Sublethal toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set 2**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4-21° Celsius**	**Arithmetic Mean 4**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 20-120 mg/L $CaCO_3$**	**Geometric Mean 2**
Endpoint/Effect Sublethal/Olfaction	**pH 6.9-8.0**	**Harmonic Mean 1**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.18	JUVENILE	3H
0.59	JUVENILE	
0.6	JUVENILE	3H
0.75	JUVENILE	20 MIN
0.79	JUVENILE	
1.1	JUVENILE	60D
1.6	JUVENILE	20 MIN
1.9	JUVENILE	120D
2	JUVENILE	21D
2	JUVENILE	
2.1	JUVENILE	3H
2.8	JUVENILE	60D
3.1	JUVENILE	23W
5	JUVENILE	6D
8.5	JUVENILE	3M
17	JUVENILE	3M
17	JUVENILE	22M

Table 2.6.2.2.6.6 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-18° Celsius**	**Arithmetic Mean** **136**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **20-306 mg/L $CaCO_3$**	**Geometric Mean** **58**
Endpoint/Effect **Cellular**	**pH** **4.7-8.54**	**Harmonic Mean** **21**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
29.2	YEARLING	15D
30.6	YEARLING, 140 MM	5W
32.2	ALEVIN	37W
32.2	EMBRYO, 14 D POST-FERTILIZATION	41W
45	17.8 CM TL, 65.0 G	96H
60.4	16.47 CM FL, 53.85 G	24H
167.3	FINGERLING, 4.1 G, 6.2 CM	2H
171.8	YEARLING	25H
217	15.5-20.0 CM	24H
1492.4	21.5 CM, 126 G	1H

Table 2.6.2.2.6.7 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-18° Celsius**	**Arithmetic Mean** **110**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **16-380 mg/L $CaCO_3$**	**Geometric Mean** **18**
Endpoint/Effect **Growth**	**pH** **4.7-8.54**	**Harmonic Mean** **6**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
1.1	EM	96H
2.2	FRY, 83.3-91.5 MG WET WT	10D
3.3	SWIM UP FRY, 0.120 G, 25.7 MM	20D
3.5	JUVENILE, 8 G	42D
3.6	YE, YEAR-CLASS I, 15 CM, 27 G MALE	8M
3.6	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
3.6	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-18° Celsius**	**Arithmetic Mean** **110**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **16-380 mg/L $CaCO_3$**	**Geometric Mean** **18**
Endpoint/Effect **Growth**	**pH** **4.7-8.54**	**Harmonic Mean** **6**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
3.6	EG, FROM 8 MO COPPER EXPOSED PARENT	100D
3.6	EG, FROM 8 MO COPPER EXPOSED PARENT	100D
3.6	YE, YEAR-CLASS I, 15 CM, 27 G	8M
3.6	EG, UNEXPOSED PARENTS	1W
5.1	YEARLING, 10-18 MO	37D
8.3	1.7-3.3 G	21D
12.1	EGG, 0-1 D	95D
16.1	1.7-3.3 G	21D
19.6	YEARLING, 14-16 CM, 30-42 G/	720D
25.5	5.6 G, 7.8 CM	100D
25.8	EGG-FRY	14W
25.8	MX, EGG-FRY	14W
30.6	YEARLING, 140 MM	40W
37.2	EMBRYO, 6 H POST-FER	85D
40	ALEVINS-BUTTONED-UP FRY	96H
45	5.74 G, 8.4 CM	30D
63.8	55.5 G	40D
217	15.5-20.0 CM	20.5H
356.8	8 mo	10D
476.7	8 mo	10D
818	8 mo	10D
930	8 mo	10D

Table 2.6.2.2.6.8 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set 3**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 6.9-16.5° Celsius**	**Arithmetic Mean 18**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 20-240 mg/L $CaCO_3$**	**Geometric Mean 8**
Endpoint/Effect Growth	**pH 7.2-7.6**	**Harmonic Mean 4**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.9	NR	120D
2.8	NR	120D
21	NR	60D
45	NR	60D

Table 2.6.2.2.6.9 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-18° Celsius**	**Arithmetic Mean 114**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 10.1-320 mg/L $CaCO_3$**	**Geometric Mean 36**
Endpoint/Effect Physiological	**pH 4.7-8.54**	**Harmonic Mean 9**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.3	200-250 G	120D
11.2	17 G	42D
33.1	NR	24H
36.4	8 MO, 3-8 G	7D
44.9	5.74 G, 8.4 CM	30D
60.4	20.01 CM FL, 101.54 G	96H
65.8	SACFRY, 9-11 D, 102.4-110.3 MG WT	15D
94.1	YEARLING	2H
99.8	YEARLING	78H
100	8 MO, 3-8 G	7D
313.6	75-100 G	8H
500	56 G	24H

Table 2.6.2.2.6.10 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-18° Celsius**	**Arithmetic Mean 1724**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 40-48 mg/L $CaCO_3$**	**Geometric Mean 57**
Endpoint/Effect Reproductive	**pH 4.7-8.54**	**Harmonic Mean 4**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3.5	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
3.5	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
3.5	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
3.5	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
8.8	YEARLING, 14-16 CM, 30-42 G/	720D

Copper Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the

criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to copper, NMFS added an additional step to its analysis for copper to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 13 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.6.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.6.1, predicts a magnitude of effect ranging from a low of an $LC_{0.6}$ at a concentration of 1160 µg/L to a high of an LC_{100} at a concentration of 5.7 µg/L (Table 2.6.2.2.6.11). In other words, the acute criterion of 13 µg/L has an equivalent toxicity potential predicted to kill 0.6 percent to 100 percent, with a median toxicity potential of an LC_{7}, of the exposed test population, and therefore by inference, field-exposed individuals.

Table 2.6.2.2.6.11 Relative percent mortality analysis for salmonid fishes, eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-16° Celsius**	
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 8-495 mg/L $CaCO_3$**	
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.0**	
Concentration Micrograms Liter^{-1}	**Relative Percent Mortality (acute criterion/LC_{50})**	
5.70	114.0	
5.96	109.1	
9.14	71.1	
9.14	71.1	
11.56	56.2	
12.85	50.6	
18.03	36.1	
19.32	33.6	
20.62	31.5	
21.20	30.7	
23.90	27.2	
25.45	25.5	
25.49	25.5	
25.65	25.3	
27.55	23.6	
30.13	21.6	
30.48	21.3	
31.26	20.8	
31.61	20.6	
32.86	19.8	
33.35	19.5	
33.41	19.5	
34.31	18.9	
35.15	18.5	
36.39	17.9	
37.88	17.2	
38.18	17.0	
38.58	16.8	
39.63	16.4	

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
40.66	16.0	
42.63	15.2	
42.83	15.2	
43.86	14.8	
43.88	14.8	
44.23	14.7	
45.86	14.2	
45.87	14.2	
46.38	14.0	
47.01	13.8	
48.10	13.5	
48.36	13.4	
50.59	12.8	
51.40	12.6	
52.79	12.3	
52.79	12.3	
52.86	12.3	
52.96	12.3	
53.76	12.1	
56.10	11.6	
56.39	11.5	
59.23	11.0	
59.70	10.9	
59.89	10.9	
61.06	10.6	
61.68	10.5	
61.87	10.5	
63.79	10.2	
64.68	10.0	
65.18	10.0	
65.54	9.9	

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
65.81	9.9	
66.26	9.8	
67.63	9.6	
68.31	9.5	
69.01	9.4	
70.11	9.3	
70.46	9.2	
70.53	9.2	
71.12	9.1	
71.23	9.1	
71.38	9.1	
72.13	9.0	
72.85	8.9	
73.87	8.8	
73.96	8.8	
74.56	8.7	
75.30	8.6	
79.51	8.2	
81.10	8.0	
84.84	7.7	
86.51	7.5	
86.89	7.5	
87.12	7.5	
87.55	7.4	
88.37	7.4	
88.91	7.3	
90.44	7.2	
92.43	7.0	
92.74	7.0	
93.28	7.0	
95.28	6.8	

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
99.44	6.5	
99.68	6.5	
99.68	6.5	
99.68	6.5	
101.29	6.4	
107.35	6.1	
108.15	6.0	
108.89	6.0	
111.19	5.8	
112.21	5.8	
113.63	5.7	
113.77	5.7	
114.29	5.7	
122.21	5.3	
123.91	5.2	
124.94	5.2	
128.87	5.0	
130.72	5.0	
133.67	4.9	
138.04	4.7	
138.78	4.7	
140.88	4.6	
145.69	4.5	
147.81	4.4	
148.58	4.4	
149.08	4.4	
150.03	4.3	
150.52	4.3	
155.59	4.2	
163.37	4.0	
163.44	4.0	

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
171.44	3.8	
174.10	3.7	
174.36	3.7	
177.75	3.7	
179.14	3.6	
179.91	3.6	
181.82	3.6	
183.34	3.5	
184.58	3.5	
185.37	3.5	
189.35	3.4	
194.30	3.3	
194.76	3.3	
199.96	3.3	
201.19	3.2	
210.45	3.1	
212.83	3.1	
217.16	3.0	
217.16	3.0	
222.22	2.9	
227.44	2.9	
228.59	2.8	
229.06	2.8	
233.38	2.8	
240.00	2.7	
240.02	2.7	
244.76	2.7	
250.22	2.6	
254.62	2.6	
255.80	2.5	
264.28	2.5	

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
266.36	2.4	
271.32	2.4	
274.31	2.4	
288.82	2.3	
289.33	2.2	
301.90	2.2	
310.51	2.1	
313.32	2.1	
322.75	2.0	
326.37	2.0	
333.58	1.9	
346.63	1.9	
355.82	1.8	
376.54	1.7	
404.21	1.6	
447.01	1.5	
447.48	1.5	
467.01	1.4	
475.90	1.4	
489.25	1.3	
533.72	1.2	
533.72	1.2	
599.98	1.1	
600.44	1.1	
1160.10	0.6	

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for copper, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for copper, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Copper toxicity is influenced by chemical speciation, hardness, pH, alkalinity, total and dissolved organic content in the water, previous exposure and acclimation, fish species and life stage, water temperature, and presence of other metals and organic compounds that may interfere with or increase copper toxicity. Synergistic toxicity is suggested for mixtures of copper and aluminum, iron, zinc, mercury, anionic detergents, or various organophosphorus insecticides (Eisler 1998a).

The distinction between copper deficiency and toxicity is small in organisms such as algae and invertebrates that lack effective mechanisms to control absorption (EPA 1999 as cited in EPA 2008). Copper is not strongly bioconcentrated in vertebrates but is more strongly bioconcentrated in invertebrates (EPA 1999 as cited in EPA 2008). Toxicity of copper to aquatic organisms is dependent on pH, temperature, alkalinity, hardness, and concentrations of bicarbonate, sulfide, and organic ligands (EPA 1980b as cited in EPA 2008), as well as the type and life stage of exposed organism (EPA 1999 as cited in EPA 2008). Copper is among the most toxic of the heavy metals to freshwater biota (Schroeder *et al.* 1966, Betzer and Yevich 1975 as cited in EPA 2008). In general, mortality of tested aquatic species is greatest under conditions of low water hardness, starvation, elevated water temperatures, and among early developmental stages (Eisler 1998a as cited in EPA 2008). Effects of copper toxicity to freshwater organisms include valve closure, reduction in filtration rates, impaired structure and function of cellular membranes, and cardiac inhibition in mussels. Impaired disease resistance, disrupted migration (via avoidance behavior of copper-contaminated areas), hyperactivity, impaired respiration, disrupted osmoregulation, pathology of kidneys, liver, and gills, impaired function of olfactory organs and brain, altered blood chemistry, and enzyme activity have been documented in fish (Eisler 1998a as cited in EPA 2008).

Biological copper toxicity has a diversity of systemic effects including reduced growth and survival rates and altered hematology, respiratory, and cardiac physiology. Reproductive effects, including reduced frequency of spawning, reduced egg production, reduced survival of young, and increased deformity of fry, have been reported (Sorensen 1991, Eisler 1998a). Elevated copper levels also influence the immune system and vulnerability to disease. For example, Carballo *et al.* (1995) determined that rainbow trout were more susceptible to the microbial parasite, *Saprolegnia parasitica*, and Dethloff and Bailey (1998) determined physiological changes in immune system characteristics at elevated copper concentrations . Hansen *et al.*

(1999b) determined that cellular damage occurred to the olfactory system of juvenile Chinook salmon and rainbow trout that were exposed to high concentrations of copper.

Copper toxicity appears to be inversely related to the tendency of the metal to bind with the external gill surface via ionic interactions. In other words, a lower affinity of the gill surface to copper leads to a greater likelihood of disruption of intracellular processes, which may lead to gill dysfunction (Reid and McDonald 1991). Some studies have examined the disruption of gill processes by copper. For example, gill Na^+, K^+- ATPase activity in Chinook salmon parr was unaffected after an 18-hour exposure to stream water with elevated copper levels of 48 µg/L (hardness = 13.3 mg/L as $CaCO_3$). With the same exposure, significant inhibition of gill Na^+, K^+-ATPase activity was observed in smolts. Significant increases in hematocrit and plasma glucose were also observed in both parr and smolts resulting from the same 18-hour exposure (Beckman and Zaugg 1988). Sola *et al.* (1995) determined that divalent copper (Cu^{2+}) totally suppressed gill Na^+, K^+- ATPase activity and produced significant cell damage, edema, mucus production, smoothing of apical membranes, swelling of tubular system and destruction of mitochondria in rainbow trout at high concentrations of $CuCl_2$ (3.5 and 134.5 mg/L). They concluded that bioavailable copper, such as divalent copper, immediately damages the hydromineral balance of rainbow trout and causes morphological modifications that are irreversible.

Sauter *et al.* (1976) determined reduced growth in brook trout fry occurred between 3 µg/L and 5 µg/L, at a hardness of approximately 38 mg/L. The resulting chronic value from that study was 3.9 µg/L, which is below the proposed chronic criterion (4.9 µg/L). At a hardness of 187 mg/L, the effect occurred between 5 µg/L and 8 µg/L with a resulting chronic value of 6.3 µg/L, which is well below the proposed chronic criterion of 19 µg/L.

Munoz *et al.* (1991) observed rapid elevations of plasma cortisol, an indicator of stress, in rainbow trout after a 1-hour exposure to approximately 0.2 µg/L of copper at a hardness of 12 mg/L. The elevated plasma cortisol levels were maintained throughout the experiment's duration of 21 days. This concentration is 45 times the chronic criterion, with no corresponding adverse physiological effects detected in association with the elevated cortisol levels. However, elevated plasma cortisol levels are indicative of stress, and potentially represent a diversion of energy from normal physiological processes that may render salmonids more vulnerable to disease. Dethloff *et al.* (2001) also determined that exposure to copper concentrations below the proposed chronic criterion was associated with decreased levels of hematocrit, leukocrit, and lymphocyte percentage in the blood in wild rainbow trout, but condition factors and other biochemical parameters tested did not show a significant difference compared with fish from reference sites.

There is tremendous variation between fish species in the amount of copper that is accumulated for a given exposure. Copper is more strongly bioconcentrated in invertebrates than in fish, and is more commonly found in tissues of herbivorous fish than in carnivorous fish from the same location. In salmonids, copper has been determined to accumulate in liver, gill, muscle, kidney, pyloric caecae, and spleen tissues and the concentrations of copper in fish tissues reflect the amount of bioavailable copper in the environment (Peterson *et al.* 1991, Farag *et al.* 1994, Camusso and Balestrini 1995, Saiki *et al.* 1995, Sorensen 1991). The kidneys and gills are not thought to play a significant role in copper detoxification (Sorensen 1991). Both dissolved and dietary pathways have been associated with bioaccumulation in salmonids, whereas the case for

particulate copper pathways is less clear. However, rainbow trout appear to be able to ingest more copper than cadmium, lead, or zinc without significant effects to survival or growth, and elevated copper levels in their gills and livers have been found to be measures of chronic exposure but not of significant toxic effects (Mount *et al.* 1994, Dethloff and Bailey 1998, Taylor *et al.* 2000).

Chemosensory and Behavioral Effects. In aquatic systems, chemoreception is one of oldest and most important sensory systems used by animals to collect information on their environment and generate behaviors involved in growth, reproduction, and survival (Pyle and Mirza 2007). These behaviors include recognition of conspecifics, mates and predators, food search, defense, schooling, spawning and migration. Stimuli are perceived by sensory structures and converted to electrical signals that are conducted to the central nervous system where the information is integrated and appropriate behavioral responses are generated (Baatrup 1991). Detection of chemical signals involves not only recognition of a spectrum of unique compounds or mixtures but also their spatial and temporal distribution in the medium (Atema 1995). Sensory receptors are in direct contact with the environment, and therefore pollutants may disrupt normal chemosensory function by masking or counteracting biologically relevant chemical signals or by causing direct morphological and physiological damage to the receptors (Baatrup 1991).

Impairment of olfaction can be measured by electrophysiological techniques called electroolfactograms (EOGs) (*e.g.*, Evans and Hara 1985, Baldwin *et al.* 2003) or electroencephalograms (EEGs) (*e.g.*, Hansen *et al.* 1999a, Sandahl *et al.* 2004). In fish, EOGs measure the response along the midline of a rosette within the fish's olfactory chamber (nose), EEGs record the response from the olfactory bulb (forebrain) (Sandahl *et al.* 2004, p. 406). Each rosette contains ciliated olfactory receptor neurons (ORNs) that respond to stimuli as water passes through the olfactory chamber and over the rosette. The EOG measures responses of an assemblage of ORNs. Reductions in or elimination of the EOG and EEG amplitude of exposed fish compared to unexposed fish reflect the in sensory ability.

Copper has been known to disrupt the normal function of the olfactory system in salmonids for over 45 years (Sprauge *et al.* 1965, Hara *et al.* 1976). More recent studies using EOGs and EEGs have shown disruption at concentrations of dissolved copper at or slightly above background concentrations (Baldwin *et al.* 2003, Sandahl *et al.* 2004). Hecht *et al.* (2007) defines background as surface waters equal to 3 µg/L dissolved copper, since experimental waters had background concentrations as high as 3 µg/L dissolved copper. There have been mixed results as to whether certain fish species are more sensitive than others to the olfactory neurotoxicity of copper. In experiments using EEG recordings, Hansen *et al.* (1999a) found that rainbow trout (*O. mykiss*) were more vulnerable than juvenile Chinook salmon (*O. tshawytscha*). Thus, while there may be modest differences in sensitivity for some species, the available evidence suggests that copper is a general olfactory toxicant for all freshwater fish. Although chemoreception is probably a fundamental function in most, if not all, fishes (Tierney *et al.* 2010), many of these studies evaluated copper avoidance or copper-induced olfactory impairment in salmonid fishes (*e.g.*, Hansen *et al.* 1999a,b; Baldwin *et al.* 2003, 2011; Sandahl *et al.* 2007; McIntyre *et al.* 2008a).

Most behavioral studies on toxicity to chemoreception (*i.e.*, avoidance, food attraction, and alarm response) are problematic because it is difficult to separate olfactory toxicity from other forms of toxicity (Tierney *et al.* 2010). Behavioral responses can integrate many inputs, which may introduce uncertainty when attributing olfactory impairment to altered behavioral responses (Tierney *et al.* 2010). A few olfactory toxicological studies have related effects across organizational levels and these can be divided into two categories: 1) those that relate changes in electrochemical responses to physiological responses or to behavioral responses; and 2) those that relate olfactory-mediated physiologic responses to behavioral responses (Tierney *et al.* 2010). For copper, Sandahl *et al.* (2007) demonstrated that the relationship between loss of sensory function (EOG) and behavioral impairment was highly correlated. Alarm pheromone (a substance released during fish injuries) triggered an average reduction in swimming speed of 74% and elicited a mean EOG response of 1.2 mV in unexposed salmon. Salmon exposed to 2 to 20 µg/L copper exhibited reductions in both EOG (50-92%) and in alarm response (Hecht *et al.* 2007, Sandahl *et al.* 2007). Statistically significant reductions in EOG response to skin extract occurred at all concentrations tested (2, 5, 10, and 20 µg/L copper), while no significant reductions in swimming speed (majority of fish did not become motionless) occurred at higher copper concentrations (5, 10, and 20 µg/L; Sandahl *et al.* 2007). In fish, direct exposure to dissolved copper can impair and destroy ORNs, although the precise mechanism remains unknown (Hecht *et al.* 2007).

Given the importance of sensory perception, impaired olfaction may in many cases be of more immediate survival concern than other physiological impairments (Tierney *et al.* 2010). The studies reviewed in this section illustrate several important aspects of copper toxicity to the olfactory system: 1) neurotoxic effects of copper can occur within minutes of exposure; 2) low concentrations can elicit responses; 3) at low concentrations, inhibition is transient and recovery can be seen within hours or when the toxicant is removed; and 4) incomplete or time-sensitive recovery of olfactory system to food-based, conspecific and predator-related odors, and reproductive pheromones.

Several studies indicate that thresholds exist between neurological, physiological and behavioral responses, and more than sufficient information exists to indicate that for fishes, olfaction is indispensible and sensitive to contaminants. Tierney *et al.* (2010) reviewed the ramifications for extrapolating neurological and physiological data to behavioral and ecological impacts as straightforward: lower order measures (*e.g.*, EOG) may underestimate the impact of toxicity to higher order biological responses (*e.g.*, mating). Tierney *et al.* (2010) report that setting regulations below where negative responses are observed in olfactory-based systems is not warranted until effects relevant to populations are better established.

Acute copper toxicity is known to disrupt osmoregulation in fishes by interfering with sodium uptake in the gill. Metal toxicity varies due to various physicochemical characteristics of the exposure water (*e.g.*, either laboratory or field), namely hardness, alkalinity, pH, and dissolved organic matter (Niyogi and Wood 2004). These constituents can protect against toxicity either by competing at the binding sites of the sodium transporter or by reducing the bioavailability of copper by complexation (McIntyre *et al.* 2008a). In 2007, the EPA updated the ambient water quality criteria for copper and employed a biotic ligand model (BLM) to derive copper criteria (EPA 2007). The BLM differs from the previous hardness-based criterion by incorporating the

water chemistry parameters (*e.g.*, pH, temperature, cations, and dissolved organic carbon) to predict lethality caused by copper binding to the gill (EPA 2007).

Due to the differences in structure and physiological function between the gill and olfactory epithelium, the extent to which the BLM can be used to estimate sublethal, neurobehavioral toxicity is unclear (McIntyre *et al.* 2008a). McIntyre *et al.* (2008a) used electrophysiological recordings from juvenile coho salmon to investigate the impacts of copper on the olfactory epithelium in freshwater with different chemical properties. Results showed olfactory function was 1) not affected by change in pH (8.6-7.6), 2) slightly protected by increasing water hardness (0.2-1.6 mM Ca) and alkalinity (0.2-3.2 mM HCO_3^-), and 3) partially restored by increasing dissolved organic carbon (0.1-6 mg/L; McIntyre *et al.* 2008a).

Since olfactory and behavioral endpoints were not used while deriving either the BLM- or hardness-based criteria, concerns have arisen that existing state water quality criteria for copper may not be protective of olfactory impairment especially in the western U.S. (McIntyre *et al.* 2008a). Using data from McIntyre *et al.* (2008a,b), Meyer and Adams (2010) parameterized an olfactory-based BLM and calculated IC_{20}s to evaluate whether the USEPA's BLM-based criteria for copper would be protective of neurological impairment in juvenile salmon. Of the 16 different laboratory test waters (data from Green *et al.* 2010; Hansen *et al.*, 1999a,b; and McIntyre *et al.* 2008a,b), the acute and chronic BLM-based copper criteria protected against at least 20% avoidance of copper and 20% olfactory impairment while the hardness-based criteria were considerably under protective in many of the same exposure waters (Meyer and Adams 2010).

McIntyre *et al.* (2012) calculated survival probabilities for copper exposures relative to controls for coho salmon that ranged from 10 percent at 20 µg/L to 17 percent at 5 µg/L. McIntyre *et al.* (2012) also determined that relatively brief (3 hours) exposures to copper ranging from 5 to 20 µg/L eliminated the behavioral alarm response in coho salmon prey, leading in turn to increased detection, reduced evasion, and reduced survival during predation trials.

Experimental data suggests that significant amelioration of olfactory toxicity due to hardness is unlikely in typical Pacific salmonid freshwater habitats (Hecht *et al.* 2007). The experiment showed that hardness at 20, 120, and 240 mg/L Ca (experimentally introduced as $CaCl_2$) did not significantly protect juvenile coho salmon from olfactory toxicity following 30 minute laboratory exposures to 10 µg dCu/L above an experimental background of 3 µg/L (Baldwin *et al.* 2003).

Hecht *et al.* (2007) calculated an acute CMC using the Biotic Ligand Model (BLM) (EPA 2007). Interestingly, the estimated acute CMC based on the BLM using measured and estimated water quality parameters from Sandahl *et al.* (2007) was 0.63 µg/L with a range from 0.34 to 3.2 µg/L, while the EPA hardness-based acute CMC (EPA 2002) was 6.7 µg/L. Because the BLM-based acute criterion is sensitive to pH and DOC, the range of measured test pH values (6.5–7.1) and the range of estimated DOC values (0.3–1.5 mg/L) produced this range of BLM-based acute criterion values. It is also interesting that the acute CMC range (0.34–3.2 µg/L) overlapped with the olfactory-based BMC range (0.18–2.1 µg/L).

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for copper is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Copper is highly toxic to most freshwater invertebrates (Moore and Ramamoorthy 1984). Aquatic macroinvertebrates are sensitive to both dissolved and particulate copper, and some taxa can be more sensitive than salmonids (*e.g.*, Kemble *et al.* 1994). Data in EPA (1985d) indicate that the proposed criteria are usually protective of invertebrates that juvenile listed species feed on, although in one case (Dave 1984 as cited in EPA 1985d) a cladoceran exhibited an LC_{50} that was lower than the acute and chronic criteria at high hardness. Invertebrate communities in rivers appear to respond to elevated copper in the sediments by changing composition to pollution-tolerant taxa, rather than by reducing overall biomass (Canfield *et al.* 1994, Clements and Kiffney 1994, Beltman *et al.* 1999). The biological significance of such species change to listed species is unknown.

Copper contained in bed sediments was elevated in benthic invertebrates in field studies conducted in metals-contaminated streams (*e.g.*, Ingersoll *et al.* 1994, Woodward *et al.* 1994, Beltman *et al.* 1999, Besser *et al.* 2001). Uptake by invertebrates is strongly influence by the presence of acid-volatile sulfide in the sediments (Besser *et al.* 1995). However, Kiffney and Clements (1996) determined an inverse relationship existed between aquatic macroinvertebrate body size and survival at copper levels in excess of the proposed chronic criterion, which may partially counter the effects of bioaccumulation. Indirect effects of elevated copper levels on listed species therefore likely include reductions in the availability of larger invertebrates as food for larger juvenile fishes, and ingestion of bioconcentrated copper by fry and juveniles of all sizes.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for copper is likely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Copper. The available evidence for copper indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (high-intensity), impairment of essential behaviors related to successful rearing and migration (high-intensity), cellular trauma (moderate intensity), physiological trauma (moderately-high-intensity), reproductive failure (high-intensity), and sublethal effects (high-intensity).

2.6.2.2.7 Lead

Lead Criteria. The proposed acute and chronic criteria for lead are 65 µg/L and 2.5 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.7.1 through 2.6.2.2.7.8 report toxicity data from the ECOTOX database for freshwater lead, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.7.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 78742**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 40-314 mg/L $CaCO_3$**	**Geometric Mean 14675**
Endpoint/Effect LC_{50}/Mortality	**pH 6.8-8.1**	**Harmonic Mean 2277**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
320	ALEVIN	96H
1000	FRY	96H
1700	JUVENILE, 7-11 WK, 0.97 G	96H
2100	JUVENILE, 18-22 WK, 0.94 G	24H
2670	72 WK, 102 G	96H
4100	JUVENILE, 7-10 WK, 0.60 G	96H
4500	145 MM	96H
12000	JUVENILE, 7-8 WK, 0.34 G	96H
170000	JUVENILE, 18-22 WK, 0.94 G	96H
170000	ALEVIN	96H
170000	ALEVIN	96H
170000	JUVENILE, 10-12 WK, 0.41 G	96H
170000	ALEVIN	96H
224000	JUVENILE, 5-6 WK, 0.85 G	96H

Table 2.6.2.2.7.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 2-20.5° Celsius**	**Arithmetic Mean 113**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 23.95-385 mg/L $CaCO_3$**	**Geometric Mean 29**
Endpoint/Effect Growth	**pH 6.5-8.1**	**Harmonic Mean 9**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1	NR	19M
6	SEXUALLY MATURING MALES 2 YR	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
13	NR	141D
14	JUVENILE, 0.38 G WET WT/	29D
16	NR	19M
16	NR	19M
18	EGGS	19M
21	EYED EGGS	19M
36	FRY, 25 MM	19MIN
38	EGGS	7M
39	EMBRYO-ADULT, SPAWNING, F1, 2, 3	38W
77	EGGS/	7M
134	ALEVIN, 21 D	21D
149	F2, EMBRYO-12 WK JUVENILE	6M
154	EMBRYO-ADULT, SPAWNING, F1, 2, 3	38W
213	EMBRYO-ADULT, SPAWNING, F1, 2, 3	38W
305	F2, EMBRYO-12 WK JUVENILE	6M
1216	F1, EMBRYO-ADULT SPAWNING	2.25Y

Table 2.6.2.2.7.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **65 Micrograms Liter^{-1}**	**Temperature** **2-20.5° Celsius**	**Arithmetic Mean** **14011**
Criterion Concentration Chronic **2.5 Micrograms Liter^{-1}**	**Hardness** **16-350 mg/L $CaCO_3$**	**Geometric Mean** **1575**
Endpoint/Effect **NOEC/Mortality/Growth**	**pH** **6.5-8.1**	**Harmonic Mean** **75**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
18	EGGS	19M
32	NR	19M
150	NR	19M
13526	NR	10D
21811	NR	10D
25461	NR	10D
37079	NR	10D

Table 2.6.2.2.7.4 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **65 Micrograms Liter^{-1}**	**Temperature** **2-20.5° Celsius**	**Arithmetic Mean** **4**
Criterion Concentration Chronic **2.5 Micrograms Liter^{-1}**	**Hardness** **50-135 mg/L $CaCO_3$**	**Geometric Mean** **4**
Endpoint/Effect **Behavioral**	**pH** **6.5-8.1**	**Harmonic Mean** **3**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
3	NR	1200S
3	NR	1200S
3	NR	1200S
6	EGG	210D

Table 2.6.2.2.7.5 Biochemical toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 2-20.5° Celsius**	**Arithmetic Mean 501**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 42.3-95 mg/L $CaCO_3$**	**Geometric Mean 190**
Endpoint/Effect Biochemical	**pH 6.5-8.1**	**Harmonic Mean 45**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
9	6-18 MO	2W
12	NR	28D
25	JUVENILE, 0.38 G WET WT/	1D
157	YEARLING	14D
157	YEARLING	56D
83	6-18 MO	2W
367	ALEVIN, 21 D	21D
1438	ALEVIN, 21 D	21D
762	6-8 MO	20D
1000	240 G	3D
1000	240 G	6D
1000	240 G	11H

Table 2.6.2.2.7.6 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 2-20.5° Celsius**	**Arithmetic Mean 414**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 121-150 mg/L $CaCO_3$**	**Geometric Mean 65**
Endpoint/Effect Cellular	**pH 6.5-8.1**	**Harmonic Mean 17**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
6	SEXUALLY MATURING MALES 2 YR	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
454	28 CM, 240 G, FEMALE	26D

Table 2.6.2.2.7.7 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 38**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 40-314 mg/L $CaCO_3$**	**Geometric Mean 15**
Endpoint/Effect Physiological	**pH 6.8-8.1**	**Harmonic Mean 6**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3	NR	191D
72	NR	191D

Table 2.6.2.2.7.8 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 2-20.5° Celsius**	**Arithmetic Mean 395**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 17-314 mg/L $CaCO_3$**	**Geometric Mean 375**
Endpoint/Effect Reproductive	**pH 6.5-8.1**	**Harmonic Mean 354**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
751	F1, EMBRYO-ADULT SPAWNING	2.25Y
1514	F1, EMBRYO-ADULT SPAWNING	2.25Y
1517	YEARLING, 50-70 G, ADULT SPAWNING	38W

Lead Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and

Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to lead, NMFS added an additional step to its analysis for lead to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 65 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.7.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.7.1, predicts a magnitude of effect

ranging from a low of an LC_{zero} at a concentration of 224,000 µg/L to a high of an LC_{10} at a concentration of 320 µg/L. In other words, the acute criterion of 65 µg/L has an equivalent toxicity potential predicted to kill zero percent to 10 percent, with a median toxicity potential of an $LC_{0.5}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, none of the toxicity studies reported concentrations that are less than the acute criterion for lead, which implies that listed species exposed to waters equal to the acute criterion concentration may not suffer acute toxic effects. A number of toxicity studies reported concentrations that are less than the chronic criteria for lead, and a number of toxicity studies reported concentrations that are greater than the chronic criterion for lead, which implies that listed species exposed to waters equal to the chronic criterion concentration will suffer chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects, but will suffer chronic toxic effects.

Sublethal Effects. Lead toxicity is influenced by species and life stage, metal speciation including whether in organic or inorganic form, hardness, pH, water temperature, and the presence of other metals that act either synergistically or antagonistically depending on the element. Elevated lead concentrations are associated with long-term effects including: spinal curvature and other deformities; anemia; caudal chromatophore degeneration (black tail); caudal fin degeneration; destruction of spinal neurons; aminolevulinic acid dehydratase (ALAD) inhibition in blood cells, spleen, liver, and renal tissues; reduced swimming ability; increased mucus formation and coagulation over body and gills and destruction of respiratory epithelium; scale loss; elevated lead in blood, bone and kidney; muscular atrophy and paralysis; teratogenic effects; inhibition of growth; retardation of maturity; changes in blood chemistry; testicular and ovarian histopathology; and death. Fish embryos appear to be more sensitive to lead than older fry and juvenile stages (Hodson *et al.* 1982, EPA 1985f, Eisler 1988b, Sorensen 1991; Farag *et al.* 1994). Organic lead compounds are generally more toxic than inorganic. Aquatic organisms are influenced more by dissolved than by total lead, because lead characteristically precipitates out to bed sediments in aqueous environments (Eisler 1988b, Sorensen 1991).

Although some of the available data suggest that toxic effects of inorganic lead on salmonids occurs above the proposed chronic criterion, the data exhibit wide variation, and there are limited lead toxicity test data available for salmonids, particularly for sublethal or indirect effects. Results for the early life stage are less conclusive than for adults, and there is conflicting evidence regarding the effects. Fish embryos and fry are more sensitive to lead in terms of effects to development than older life stages (Sorenson 1991). The results of Birge *et al.* (1978, 1981) indicate that salmonid embryos exposed for more than 4 days can begin to die when inorganic lead concentrations are between 2.5 µg/L and 10.3 µg/L, and hardness is 100 mg/L as $CaCO_3$.

Other studies were identified in this analysis that indicate the chronic criterion is at or below the NOEC level for the early life stage, as suggested by available data. For example, Sauter *et al.*

(1976) determined that the threshold for adverse chronic effects to rainbow trout eggs and fry occurred at a lead concentration between 71 µg/L and 146 µg/L, both of which are above the chronic criterion. Davies *et al.* (1976) determined that in soft water (hardness ~30 mg/L), adverse developmental effects occurred to eggs and sac-fry when exposure concentrations were between 4.1 µg/L and 7.6 µg/L, which are below the proposed chronic criterion. When the eggs were not exposed, effects to sac-fry were determined to occur when exposure concentrations were between 7.2 µg/L and 14 µg/L in soft water, and between 190 µg/L and 380 µg/L in hard water (300 mg/L). Other bioassays involving adult trout and their offspring in soft water indicated that there were no adverse reproductive effects occurring when lead concentrations were around 6 µg/L (Davies *et al.* 1976); this level is also above the proposed chronic criterion.

The bioavailability of lead increases in environments with low pH, low organic content, and low metal salt content (Eisler 1988b as cited in EPA 2008). Toxicity of lead to aquatic organisms varies with water temperature, pH, water hardness, metal salt concentrations, organic matter, and suspended solid concentration (EPA 1999 as cited in EPA 2008). Invertebrates tend to have higher bioconcentration factors than vertebrates (EPA 1999 as cited in EPA 2008). Effects of lead toxicity to freshwater organisms include reduced growth, spinal curvature and other deformities, anemia, caudal fin degeneration, destruction of spinal neurons, enzyme inhibition, reduced swimming ability, increased mucus formation and coagulation over body and gills and destruction of respiratory epithelium, scale loss, muscular atrophy and paralysis, impaired reproduction, and reduced survival (Hodson *et al.* 1982, Eisler 1988b, Sorensen 1991, Farag *et al.* 1994 as cited in EPA 2008). Organic lead compounds are generally more toxic than inorganic (Eisler 1988b as cited in EPA 2008).

Fish do not accumulate lead extensively and the results and interpretations of lead accumulation studies vary. Farag *et al.* (1994) determined that adult and juvenile rainbow trout accumulated lead in their gut through their diet, and in gill and kidney tissues, when exposed to dissolved lead at concentrations slightly in excess of the proposed chronic criteria. In contrast, Mount *et al.* (1994) determined that much higher levels of dietary lead exposure than that tested by Farag *et al.* (1994) did not result in reduced survival or growth of rainbow trout fry. Fish excrete lead rapidly, and depuration generally reduces levels in tissues and organs (Sorensen 1991).

Lead accumulation is influenced by age, diet, particle size ingested, hardness, pH, water temperature, metal speciation, and presence of other compounds in the water (Eisler 1988b; Sorensen 1991). Bioavailability of lead increases with decreasing pH, organic content, hardness, and metal salt content (Eisler 1988b). Lead precipitation with increasing hardness leads to decreased bioavailability, although the potential for accumulation from precipitated lead still exists (Sorensen 1991). Fish do not accumulate lead extensively, and the results and interpretations of lead accumulation studies consequently vary. Farag *et al.* (1994) determined that adult and juvenile rainbow trout accumulated lead in their gut through their diet, and in gill and kidney tissues when exposed to dissolved lead at concentrations slightly in excess of the chronic criterion. In contrast, Mount *et al.* (1994) determined that much higher levels of dietary lead exposure than that tested by Farag *et al.* (1994) did not result in reduced survival or growth of rainbow trout fry. Fish excrete lead rapidly and depuration generally reduces levels in tissues and organs (Sorensen 1991).

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for lead is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Lead toxicity varies considerably among aquatic macroinvertebrates (EPA 1985f, Eisler 1988b). Results reviewed in EPA (1985f) and Eisler (1988b) indicate that amphipods are more sensitive than other taxa, and that some freshwater isopods are tolerant of elevated lead levels. However, the data indicate that mortality of the more sensitive taxa occurs at concentrations that are well above the acute criterion.

Invertebrates generally have higher bioconcentration factors than vertebrates (Enk and Mathis 1977; Eisler 1988b). Ingersoll *et al.* (1994) determined that while the amphipod *Hyalella azteca* accumulated lead from bed sediments, the level of accumulation was not related to concentration gradient in the riverbed. Because lead occurs in association with copper, cadmium, and zinc in the field studies reviewed, it is difficult to ascribe a direct adverse chronic effect of lead to aquatic invertebrates at exposure concentrations that are below the chronic criterion.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for lead is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Lead. The available evidence for lead indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), reduced growth (moderate) intensity, impairment of essential behaviors related to successful rearing and migration (moderately-high-intensity), cellular trauma (moderately-high-intensity), physiological trauma (moderate intensity), impairment of biochemical processes (moderate intensity), and reproductive failure (low intensity).

2.6.2.2.8 Nickel

Nickel Criteria. The proposed acute and chronic criteria for nickel are 470 µg/L and 52 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.8.1 through 2.6.2.2.8.5 report toxicity data from the ECOTOX database for freshwater nickel, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.8.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater nickel.

Criterion Freshwater Nickel		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 470 Micrograms Liter^{-1}	**Temperature 8-13.3° Celsius**	**Arithmetic Mean 92062**
Criterion Concentration Chronic 52 Micrograms Liter^{-1}	**Hardness 27-39 mg/L $CaCO_3$**	**Geometric Mean 18793**
Endpoint/Effect LC_{50}/Mortality	**pH 6.1-8.3**	**Harmonic Mean 1146**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
107	4 H POST-FER	85D
244	4 H POST-FER	85D
588	LARVAE	96H
8826	ADULT, 16-18 CM	96H
15571	JUVENILE, 43.4 MM, 0.60 G	96H
16390	ALEVIN, 14.3 MM, 0.01 G	96H
17390	JUVENILE, 62.4 MM, 1.44 G	96H
20652	15.4 G, 116 MM, 12 MO	96H
22691	16.4 G, 119 MM, 12 MO	96H
25496	0.37 G, 36 MM, 3 MO	96H
27790	0.58 G, 40 MM, 3 MO	96H
33380	ALEVIN, 29.8 MM, 0.24 G	96H
35978	JUVENILE, 45.8 MM, 0.63 G	96H
50170	ALEVIN, 20.8 MM, 0.10 G	96H
155928	NR	48H
161455	8 MO	4D
503126	NR	48H
561339	NR	48H

Table 2.6.2.2.8.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater nickel.

Criterion Freshwater Nickel		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **470 Micrograms Liter^{-1}**	**Temperature** **4-20° Celsius**	**Arithmetic Mean** **4824**
Criterion Concentration Chronic **52 Micrograms Liter^{-1}**	**Hardness** **11-52 mg/L $CaCO_3$**	**Geometric Mean** **631**
Endpoint/Effect **Growth**	**pH** **6.1-8.3**	**Harmonic Mean** **183**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
60	4 H POST-FER	85D
61	4 H POST-FER	75D
108	4 H POST-FER	75D
413	4 H POST-FER	75D
672	8 MO	75D
672	EGGS	75D
748	4 H POST-FER	75D
9041	EYED EGGS-SWIM UP FRY	75H
31645	EGGS-SACK FRY	75D

Nickel Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on

fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to nickel, NMFS added an additional step to its analysis for nickel to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 470 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.8.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.8.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 561,339 µg/L to a high of an LC_{100} at a concentration of 107 µg/L. In other words, the acute criterion of 470 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an LC_1, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for nickel, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for nickel, which implies that listed species exposed to waters equal to criteria concentrations may

not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute toxic effects, but may not suffer chronic toxic effects.

Sublethal Effects. Nickel poisoning in fish can cause respiratory stress, convulsions, and loss of equilibrium prior to death. In fishes, adverse respiratory effects occur through destruction of gill tissues by ionic nickel and subsequent blood hypoxia. Other effects include decreased concentrations of glycogen in muscle and liver tissues and simultaneous increases in lactic acid and glucose in the blood, and interference with metabolic oxidation-reduction processes (Eisler 1998b). In general, the egg and embryo stages of salmonids are the most, and older stages the least, sensitive to nickel toxicity (Nebeker *et al.* 1985 as cited in Eisler 1998b). In contrast with other metals, alevins and juveniles appear to have a similar sensitivity to nickel (Buhl and Hamilton 1991).

Salmonid fishes accumulate nickel through both dietary and water-borne exposure routes (EIFAC 1984, Eisler 1998b). Bioconcentration factors vary substantially both within and between species, with age of organism, and with exposure concentration, and have been determined to range between 2 inch and 52 inch fish. Bioconcentration has been noted to occur in kidney, liver, and muscle tissues of rainbow trout exposed to ambient water concentrations of nickel equal to 1000 µg/L for 6 months, but the test fish were able to depurate much of the accumulated nickel within 3 months after exposure was terminated and were not visibly affected during the experiment (Calamari *et al.* 1982). Studies of saltwater and freshwater fish species have determined that piscivorous fish bioaccumulate greater levels of nickel in muscle tissues than other fish, indicating the potential for biomagnification to occur (albeit to a limited extent according to most studies; EIFAC 1984, Eisler 1998b). There is evidently a risk of bioaccumulation from chronic nickel exposure, but it remains to be determined to what extent this is a significant hazard for listed species.

Nickel can be carcinogenic, may be mutagenic, and is not teratogenic. It is bioconcentrated and bioaccumulated by aquatic organisms (Eisler 1998b). Toxicity of nickel to aquatic organisms is dependent on water hardness, pH, ionic composition, chemical form, type and concentration of ligands, presence of mixtures, and availability of solid surfaces for adsorption (Eisler 1998b). Nickel interacts with many compounds to produce altered patterns of accumulation, metabolism, and toxicity (Eisler 1998b). Mixtures of metals containing nickel salts are more toxic to daphnids and fishes than are predicted on the basis of individual components (Enserink *et al.* 1991). Effects of nickel toxicity to freshwater invertebrates include reduced growth, impaired reproduction, reduced population biomass, increased respiration rate, and reduced survival (see Eisler 1998b). Effects of nickel toxicity to freshwater fish include delayed hatching time, reduced swimming activity, behavioral alterations (avoidance), disrupted protein metabolism in gills and kidneys, loss of equilibrium, destruction of gill lamellae resulting in decreased ventilation rate, decreased concentrations of glycogen in muscle and liver, and reduced survival in fish (Eisler 1998b).

Several studies have determined that mortality of salmonid embryos occurs over longer-term exposures to concentrations that are below the chronic criterion. For example, Birge *et al.* (1978) determined a 30-day LC_{50} for rainbow trout embryos of 50 µg/L at a water hardness between 93 mg/L and 105 mg/L. The corresponding lethal threshold (LC_1) was estimated to be approximately 0.6 µg/L. Birge and Black (1980; as cited in Eisler 1998, hardness not reported) determined an LC_{10} of 11 µg/L for rainbow trout embryos exposed from fertilization through hatching. In Eisler's (1998b) review, LC_{50}s were reported of 60 µg/L and 90 µg/L at water hardness of 125 and 174 mg/L, respectively, for rainbow trout embryos that were exposed from fertilization through hatching. These results and the review by Birge *et al.* (1981) suggest that adverse effects are likely to occur to embryos exposed to nickel concentrations that are lower than the proposed chronic criterion.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for nickel is likely to result in sublethal effects to listed species considered in this opinion.

Summary of Effects: Nickel. The available evidence for nickel indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity) and reduced growth (moderately-high-intensity).

2.6.2.2.9 Selenium

Selenium Criteria. The proposed acute and chronic criteria for selenium (VI) are 190 µg/L and 5.0 µg/L, and for selenium (IV), 12.8 µg/L and 5.0 µg/L, respectively.

Tables 2.6.2.2.9.1 through 2.6.2.2.9.5 report toxicity data from the ECOTOX database for freshwater selenium, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.9.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium.

Criterion Freshwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 51334**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 2850**
Endpoint/Effect LC_{50}/Mortality	**pH 6.1-9.6**	**Harmonic Mean 7**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.4	NR	96H
0.4	NR	96H

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute **190 Micrograms Liter^{-1}**	**Temperature** **5-30° Celsius**	**Arithmetic Mean** **51334**
Criterion Concentration Chronic **5 Micrograms Liter^{-1}**	**Hardness** **17-340 mg/L $CaCO_3$**	**Geometric Mean** **2850**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.1-9.6**	**Harmonic Mean** **7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.4	NR	96H
0.4	NR	96H
0.4	NR	96H
0.4	NR	24H
0.4	NR	96H
0.4	NR	24H
0.4	NR	96H
1	NR	96D
3.78	4.40 CM, 0.69 G	96H
3.98	4.40 CM, 0.69 G	96H
5	60 MM	96H
7	60 MM	96H
40	EGGS	96M
40	EGG	96M
40	EGG	96M
40	EGG-FRY	96H
45.6	NR	24H
45.6	NR	96H
45.6	NR	24H
45.6	NR	96H
45.6	NR	48H
45.6	NR	96H
45.6	NR	6H
45.6	NR	7H
45.6	NR	24H
50	2.78(2.4-3.0) CM	96D
50	2.78(2.4-3.0) CM	120D
100	EGG, LATE-EYED STAGE	96D
100	EGG, LATE-EYED STAGE	96D
150	3.10(2.4-3.7) CM	43D
170	FERTILIZATION THROUGH 4 DAY POST	28D
170	FERTILIZATION THROUGH 4 DAY POST	28D

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute **190 Micrograms Liter^{-1}**	**Temperature** **5-30° Celsius**	**Arithmetic Mean** **51334**
Criterion Concentration Chronic **5 Micrograms Liter^{-1}**	**Hardness** **17-340 mg/L $CaCO_3$**	**Geometric Mean** **2850**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.1-9.6**	**Harmonic Mean** **7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
220	6.57(5.1-10.1) CM	120D
260	6.57(5.1-10.1) CM	96D
260	6.57(5.1-10.1) CM	96D
300	EGG, LATE-EYED STAGE	96D
300	EGG, LATE-EYED STAGE	96D
310	NR	24D
310	NR	24D
310	NR	96D
310	NR	96D
310	NR	96D
310	NR	96D
430	2.78(2.4-3.0) CM	21D
430	2.78(2.4-3.0) CM	120D
470	6.57(5.1-10.1) CM	48D
470	6.57(5.1-10.1) CM	96D
1000	EGG, LATE-EYED STAGE	96D
1000	EGG, LATE-EYED STAGE	96D
1000	EGG, LATE-EYED STAGE	96D
1100	60 MM	24D
1290	NR	96H
1800	NR	96H
1800	NR	24H
2200	NEWLY FERTILIZED EGG, <48 H	24D
2200	NEWLY FERTILIZED EGG, <48 H	24D
2200	NEWLY FERTILIZED EGG, <48 H	24D
2350	4.40 CM, 0.69 G	96H
2350	4.40 CM, 0.69 G	120H
2350	4.40 CM, 0.69 G	16H
2350	4.40 CM, 0.69 G	96H
2570	4.40 CM, 0.69 G	96H
2570	4.40 CM, 0.69 G	120H
2570	4.40 CM, 0.69 G	96H
2570	4.40 CM, 0.69 G	384H

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute **190 Micrograms Liter^{-1}**	**Temperature** **5-30° Celsius**	**Arithmetic Mean** **51334**
Criterion Concentration Chronic **5 Micrograms Liter^{-1}**	**Hardness** **17-340 mg/L $CaCO_3$**	**Geometric Mean** **2850**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.1-9.6**	**Harmonic Mean** **7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
2820	EGGS	28D
2820	EGGS	21D
3000	EGG, LATE-EYED STAGE	96D
3680	0.8 G	28D
3680	0.8 G	28D
3780	4.40 CM, 0.69 G	96H
3780	4.40 CM, 0.69 G	120H
3780	4.40 CM, 0.69 G	96H
3980	4.40 CM, 0.69 G	96H
3980	4.40 CM, 0.69 G	120H
3980	4.40 CM, 0.69 G	24H
4150	NR	4D
4150	EGG	28D
4150	NR	96D
4990	0.8 G	9D
4990	0.8 G	9D
5000	60 MM	16D
5000	60 MM	384H
5000	60 MM	24D
5170	EGG	28D
5330	0.8 G	9D
5330	0.8 G	9D
6280	JUVENILE, 41.6 MM, 0.47 G	96H
6280	JUVENILE, 41.6 MM, 0.47 G	96H
6300	NEWLY FERTILIZED EGG, <48 H	96D
6700	FRY, 0.5 G	96H
7000	JUVENILE, 49.6 MM, 1.04 G	96H
7200	0.8 G	96H
7200	0.8 G	96H
8200	0.8 G	96H
8200	0.8 G	96H
8600	FRY, 0.5 G	96H

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute **190 Micrograms Liter^{-1}**	**Temperature** **5-30° Celsius**	**Arithmetic Mean** **51334**
Criterion Concentration Chronic **5 Micrograms Liter^{-1}**	**Hardness** **17-340 mg/L $CaCO_3$**	**Geometric Mean** **2850**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.1-9.6**	**Harmonic Mean** **7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
8800	0.8 G	96H
8800	0.8 G	9H
10000	EGG, LATE-EYED STAGE	96D
10000	EGG, LATE-EYED STAGE	96D
10400	60 MM	96D
10600	125 MM	96H
10600	125 MM	24H
10800	FRY, 0.46 G	96H
11500	60 MM	96H
11500	60 MM	96H
11500	60 MM	96H
11500	60 MM	96H
11600	FRY, 2.6 G	96H
12500	125 MM	96H
12500	125 MM	96H
13100	ADULT, 1.8 MO, 210.8 MM, 99.6 G	96H
13400	FRY, 0.7 G	96H
14800	FRY, 0.7 G	96H
17000	FRY, 0.5 G	96H
18300	FRY, 2.6 G	24H
18500	FRY, 0.5 G	96H
18600	FRY, 0.5 G	96H
19200	FRY, 0.31 G	96H
19600	FRY, 2.6 G	96H
23000	FRY, 0.5 G	24H
23800	ADULT, 1.8 MO, 210.8 MM, 99.6 G	48H
23900	FRY, 2.6 G	24H
25000	JUVENILE, 49.6 MM, 1.04 G	96H
25300	FRY, 0.5 G	96H
28200	FRY, 2.6 G	24H
29000	JUVENILE, 51.5 MM, 0.81 G	96H
29000	FRY, 1.7 G	96H

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute **190 Micrograms Liter^{-1}**	**Temperature** **5-30° Celsius**	**Arithmetic Mean** **51334**
Criterion Concentration Chronic **5 Micrograms Liter^{-1}**	**Hardness** **17-340 mg/L $CaCO_3$**	**Geometric Mean** **2850**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.1-9.6**	**Harmonic Mean** **7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
35800	FRY, 0.5 G	96H
36100	FRY, 2.6 G	24H
36300	ADULT, 1.8 MO, 210.8 MM, 99.6 G	24H
38000	NR	96H
38000	NR	24H
38200	FRY, 0.7 G	24H
39000	NR	96H
39000	NR	96H
39300	FRY, 0.5 G	96H
48300	FRY, 0.5 G	24H
50500	FRY, 0.46 G	24H
53000	JUVENILE, 41.6 MM, 0.47 G	96H
53000	JUVENILE, 41.6 MM, 0.47 G	96H
56000	ALEVIN, 15.0 MM, 0.02 G	96H
57100	FRY, 0.6 G	96H
61000	ALEVIN, 29.8 MM, 0.24 G	96H
61000	ALEVIN, 29.8 MM, 0.24 G	96H
63700	ADULT, 1.8 MO, 210.8 MM, 99.6 G	7H
66500	FRY, 0.5 G	96H
74000	FRY, 0.5 G	96H
74200	ADULT, 1.8 MO, 210.8 MM, 99.6 G	6H
78000	ALEVIN, 14.3 MM, 0.01 G	96H
79000	ALEVIN, 20.8 MM, 0.10 G	96H
84000	FRY, 0.31 G	24H
85000	FRY, 0.31 G	96H
85000	FRY, 0.31 G	43H
86000	FRY, 0.7 G	96H
87000	ALEVIN	96H
138000	JUVENILE, 62.4 MM, 1.44 G	96H
151000	ALEVIN	24H
171000	FRY, 0.5 G	24H
274000	ALEVINE, 29.8 MM, 0.24 G	96H

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute **190 Micrograms Liter^{-1}**	**Temperature** **5-30° Celsius**	**Arithmetic Mean** **51334**
Criterion Concentration Chronic **5 Micrograms Liter^{-1}**	**Hardness** **17-340 mg/L $CaCO_3$**	**Geometric Mean** **2850**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.1-9.6**	**Harmonic Mean** **7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
274000	ALEVINE, 29.8 MM, 0.24 G	96H
320000	ALEVIN	24H
320000	ALEVIN	96H
360000	FRY, 0.7 G	24H
361000	FRY, 0.5 G	24H
369000	FRY, 1.7 G	96H
374000	ALEVIN, 20.8 MM, 0.10 G	96H
381000	FRY, 0.31 G	24H
560000	EYED EGG	24H
560000	EYED EGG	96H
1000000	EYED EGG	24H
1000000	EYED EGG	96H

Table 2.6.2.2.9.2 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium

Criterion Freshwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 68398**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 10953**
Endpoint/Effect Mortality	**pH 6.1-9.6**	**Harmonic Mean 417**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
40	EGG	12M
40	EGG	12M
47.2	SAC FRY, 21.7 MM, 0.075 G	5D
100	EGG, LATE-EYED STAGE	5D
300	EGG, LATE-EYED STAGE	24D
300	EGG, LATE-EYED STAGE	5D
1000	EGG, LATE-EYED STAGE	20D
1000	EGG, LATE-EYED STAGE	5D
1000	EGG, LATE-EYED STAGE	5D
1100	60 MM	16D
2200	NEWLY FERTILIZED EGG, <48 H	5D
3000	EGG, LATE-EYED STAGE	70D
6300	NEWLY FERTILIZED EGG, <48 H	90D
8600	FRY, 0.5 G	24H
10000	EGG, LATE-EYED STAGE	42D
10400	60 MM	16D
13100	ADULT, 1.8 MO, 210.8 MM, 99.6 G	16H
16600	1.6 G, FRY	7.6H
17200	1.6 G, FRY	49H
23800	ADULT, 1.8 MO, 210.8 MM, 99.6 G	120H
36300	ADULT, 1.8 MO, 210.8 MM, 99.6 G	12H
38200	FRY, 0.7 G	70H
39600	1.6 G, FRY	7.6H
43200	FRY, 2.4 G	5H
50100	FRY, 2.4 G	5H
50500	FRY, 0.46 G	20H
63700	ADULT, 1.8 MO, 210.8 MM, 99.6 G	16H
63800	1.6 G, FRY	7.6H
65400	FRY, 2.4 G	5H
74000	FRY, 0.5 G	5H

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 68398**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 10953**
Endpoint/Effect Mortality	**pH 6.1-9.6**	**Harmonic Mean 417**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
74200	ADULT, 1.8 MO, 210.8 MM, 99.6 G	90H
79400	FRY, 1.8 G	7.6H
86000	FRY, 0.7 G	5H
94000	FRY, 1.6 G	90H
136000	FRY, 1.6 G	24H
236000	FRY, 1.6 G	90H
360000	FRY, 0.7 G	42H
361000	FRY, 0.5 G	5H
600000	FRY, 1.6 G	30H

Table 2.6.2.2.9.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium.

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 619**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-334 mg/L $CaCO_3$**	**Geometric Mean 167**
Endpoint/Effect NOEC/Mortality	**pH 6.1-9.6**	**Harmonic Mean 73**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
40	EGGS	12M
40	EGG-FRY	1Y
2200	NEWLY FERTILIZED EGG, <48 H	90D
47.2	SAC FRY, 21.7 MM, 0.075 G	1D
99.5	SAC FRY, 21.7 MM, 0.075 G	90D
1290	NR	12H

Table 2.6.2.2.9.4 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium.

Criterion Freshwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 34707**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 1513**
Endpoint/Effect Growth	**pH 6.1-9.6**	**Harmonic Mean 16**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1	NR	21D
40	EGG	4M
47.2	SAC FRY, 21.7 MM, 0.075 G	30D
50	2.78(2.4-3.0) CM/	42D
50	2.78(2.4-3.0) CM/	120D
99.5	SAC FRY, 21.7 MM, 0.075 G	90D
220	6.57(5.1-10.1) CM/	30D
310	NR	12D
2200	NEWLY FERTILIZED EGG, <48 H	30D
7000	60 MM	30H
7000	JUVENILE, 49.6 MM, 1.04 G	12H
10000	5-10 CM	42H
25000	JUVENILE, 49.6 MM, 1.04 G	21H
35800	FRY, 0.5 G	90H
39300	FRY, 0.5 G	30H
57100	FRY, 0.6 G	30H
66500	FRY, 0.5 G	90H
374000	ALEVIN, 20.8 MM, 0.10 G	4H

Table 2.6.2.2.9.5 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium.

Criterion Freshwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 17450**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-334 mg/L $CaCO_3$**	**Geometric Mean 4844**
Endpoint/Effect Cellular	**pH 6.1-9.6**	**Harmonic Mean 392**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
100	EGG, LATE-EYED STAGE	21D
10000	EGG, LATE-EYED STAGE	20D
11400	FRY, 0.7 G	21H
48300	FRY, 0.5 G	20H

Selenium Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50

percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to selenium, NMFS added an additional step to its analysis for selenium to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 470 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.9.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.9.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 1,000,000 µg/L to a high of an LC_{100} at a concentration of 0.4 µg/L. In other words, the acute criterion of 470 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{1.8}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for selenium, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for selenium, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. The behavior of selenium in biological systems is complex. Selenium is a metalloid that exists in three oxidation states in water: selenide (-2), selenite (+4) and selenate (+6). The toxicity of selenium varies with its chemical species. Inorganic selenium is the predominant form in aquatic environments. Organic and reduced forms of selenium (*e.g.*, seleno-methionine and selenite) are generally more toxic and will bioaccumulate more readily (Kiffney and Knight 1990, Besser *et al.* 1993). Toxicity also varies with the species exposed. Species at higher trophic levels, such as piscivorous fish and birds, are affected by the lowest concentrations of selenium. Long-term, low-level exposures from water or food appear to have the greatest effect on aquatic organisms (Lemly 1985). Like mercury, selenium bioaccumulates in muscle tissue and is associated with reproductive impairment and reduced hatching success. Toxic effects of selenium range from physical malformations during embryonic development to sterility and death. Other effects include reduced smolting success, reduced red blood cell volumes and cellular blood iron content, and impaired immune responses (Eisler 1985b, Hamilton *et al.* 1986, Lemly and Smith 1987, Felton *et al.* 1990, Sorensen 1991).

Of all the priority and non-priority pollutants, selenium has the narrowest range of what is beneficial for biota and what is detrimental. Aquatic and terrestrial organisms require 0.5 µg/g dry weight (dw) of selenium in their diet to sustain metabolic processes, whereas concentrations of selenium that are only an order of magnitude greater than the required level have been shown to be toxic to fish. Acute effects are observed after short exposure durations of typically 96 hours or less. Acute effects from the inorganic forms of selenium, selenite and selenate, require concentrations exceeding 300 µg/L, concentrations rarely reached in the environment. In contrast, toxic effects from long-term chronic exposure via diet and water can result in reduction of species in aquatic systems with aqueous concentrations less than 20 µg/L (Lemly 1985 as cited in EPA 2008). As a result of the greater sensitivity to selenium from chronic exposures, water quality management practices over the last 10-15 years have focused on the control of chronic effects. Studies have shown that diet is the primary route of exposure that controls chronic toxicity to fish, the group considered to be the most sensitive to chronic selenium exposure (Coyle *et al.* 1993, Hamilton *et al.* 1990, Hermanutz *et al.* 1996 as cited in EPA 2008).

Effects of selenium toxicity to freshwater organisms range from physical malformations during embryonic development to sterility and death (Lemly and Smith 1987) and include reduced hatch, reduced growth, behavioral alterations (avoidance), shifts in species composition of freshwater algal communities, loss of equilibrium, lethargy, muscle spasms, protruding eyes, liver degeneration, reduction in blood hemoglobin, chromosomal aberrations, and reduced survival (Eisler 1985b).

Selenium is an essential nutrient for normal cell functions. Inadequate dietary uptake (food and water) of selenium results in selenium deficiency syndromes such as reproductive impairment, poor body condition, and immune system dysfunction (Oldfield 1990, CAST 1994). However, excessive dietary uptake of selenium also results in toxicity syndromes that are similar to the deficiency syndromes (Koller and Exon 1986). Selenium is a "hormetic" chemical, *i.e.*, a chemical for which levels of safe dietary uptake are bounded on both sides by adverse-effects thresholds. Most essential nutrients are hormetic, but what distinguishes selenium from other nutrients is the very narrow range between the deficiency threshold and the toxicity threshold (Wilber 1980, Sorensen 1991, Skorupa *et al.* 1996, USDI-BOR/FWS/GS/BIA 1998). In other

words, the difference between useful amounts of selenium and toxic amounts is small.

Water-borne selenium is depurated in fish via a passive excretion pathway, while dietary selenium is excreted more actively. The half-life of selenium is inversely proportional to dietary loading. Inorganic selenium absorbed from water is stored in fish as inorganic selenium. However, inorganic selenium absorbed from the diet is transformed by the liver to an organic form that is more toxic, but can be excreted easily (Hodson *et al.* 1984). Nevertheless, the transformation of selenium to organoselenium is associated with bioconcentration in fish ovaries, resulting in significant pathology and reproductive failure (Baumann and Gillespie 1986, Srivastava and Srivastava 1994). Selenium taken up from water is absorbed across the gills and taken directly to all tissues. Dietary selenium is taken up through the gut, from which the liver receives its blood supply via a portal system. The tissue distribution of selenium within fish is a function of the loading rate, but not the source of selenium (Hodson and Hilton 1983, Sorensen 1991).

Selenium protects some species from the toxicity of other chemicals. For example, selenium antagonizes mercury toxicity in rainbow trout (Eisler 1985b). Selenium criteria are not hardness dependent. The dose-response curves for selenium are relatively steep, indicating a rapid shift to toxic conditions with small increases in metal concentration (Lemly 1998, Skorupa 1998)

Salmonids are sensitive to chronic selenium contamination (Lemly 1996a,b). Depending on the form of selenium and the life-stage of fish considered, water-borne concentrations of selenium less than 5 µg/L can have direct toxic effects on salmonids (Hodson *et al.* 1980, Moore *et al.* 1990). Lemly (1998) concluded that the larval fish life stage is the most sensitive to exposure to selenium, with adverse effects expressed through teratogeny and mortality. Hodson *et al.* (1980) reported that rainbow trout (*O. mykiss*) eggs respond physiologically (reduced median time to hatch) at selenium (as selenite) concentrations above 4.3 µg/L. Studies have also shown that chronic exposure to selenium can reduce fish growth in terms of weight and to a lesser extent length (Eisler 1985b, Hamilton *et al.* 1986, Hamilton *et al.* 1990). Van Derveer and Canton (1997) concluded, based on a sediment-water transfer model, that a 5 µg/L concentration may not always avoid harm to listed salmonids, depending on the organic carbon content in the sediment. Using their model, Mebane (2000) estimated protective selenium levels ranging between 2 µg/L and 8 µg/L for higher gradient mountain streams in the upper Salmon River basin, effectively demonstrating that the chronic criterion is unlikely to avoid adverse effects under the range of environmental conditions.

Skorupa (1998) noted collapse of natural fish populations chronically exposed to 10 µg/L selenium in selenite-dominated waters. Hodson *et al.* (1980) observed significant mortality in rainbow trout eyed eggs exposed to concentrations greater than or equal to 25 µg/L after 44 weeks, and hatchability of eggs was affected at concentrations as low as 16 µg/L. Hamilton *et al.* (1986) determined that exposures to 17 µg/L (selenate:selenite ratio = 6:1) for 30 days caused a significant increase in mortality of Chinook salmon fry.

Kennedy *et al.* (2000) determined, in the case of eggs taken from wild female cutthroat living in a contaminated river with higher exposure concentrations (13.3 µg/L to14.5 µg/L), that there was no significant effect of the resulting elevated selenium concentrations in the eggs on subsequent

survival to hatch or fry deformities when the eggs and fry were reared in water with concentrations below 1 µg/L. They concluded that their result may reflect an evolved tolerance to higher tissue concentrations of selenium in the test population, although it is possible that the absence of subsequent exposure during development may also have influenced the results.

In the CTR biological opinion (USFWS and NMFS 2010), the NMFS and FWS determined that under most circumstances, a 5 µg/L chronic criterion should be protective of aquatic life with regard to direct contact toxicity. However, based on data collected by the U.S. Department of Interior's National Irrigation Water Quality Program from 26 study areas in 14 western states, the Services determined that a 5 µg/L chronic criterion for selenium is only 50% to 70% protective (Seiler and Skorupa 1999), as opposed to the 95% level of protection that EPA's national water quality criteria are intended to achieve.

The consensus of researchers lately, however, is that water-borne exposure to selenium in any form is much less important than dietary exposure and bioaccumulation in determining the potential for chronic effects (EPA 1998). The Services similarly determined in the CTR biological opinion that the 5 µg/L chronic aquatic life criterion for selenium does not protect listed fish in other respects because of bioaccumulation hazards, which may be a reason for results listed above that reported finding adverse effects at concentrations below the proposed criterion. Determinations of effect using solely studies of water-borne exposure underestimate the danger of selenium exposure to fish through bioaccumulation (Hermanutz *et al.* 1992).

Bioaccumulation. Dietary bioaccumulation of selenium is the most dangerous exposure pathway for salmonids and other fish species (EPA 1998). Bioconcentration of selenium is influenced by exposure concentration, selenium speciation, water temperature, age of receptor organism, organ, tissue specificity, and mode of administration (Eisler 1985b). Lemly and Smith (1987) noted that bioconcentration factors in fish experiencing chronic toxicity have ranged from around 100 to more than 30,000, and that bioconcentration can occur when water-borne selenium concentrations are within the range of 2 µg/L to 5 µg/L. Selenium bioconcentration factors appear to be inversely related to water exposure concentrations (EPA 1998). A concentration as little as 0.1 µg/L of dissolved selenomethionine has been found to be sufficient to cause bioaccumulation of an average concentration of 14.9 mg/kg (dry weight) selenium in zooplankton (Besser *et al.* 1993), a concentration that could cause dietary toxicity to most species of fish (Lemly 1996a). Fish bioconcentrate selenium in higher levels in ovaries than in muscle tissues (Lemly 1985, Hamilton *et al.* 1990) and milt (Hamilton and Waddell 1994).

As for the water-borne case, selenium biomagnification factors similarly appear to be inversely related to dietary exposure concentrations (Hamilton *et al.* 1986). Hamilton *et al.* (1990) determined that Chinook salmon fingerlings fed organic selenium in their study accumulated the metal to whole body concentrations that were not significantly different from that in their artificial diet, suggesting that biomagnification may not be significant in this life stage of listed salmonids. Overall, however, magnitudes of biomagnification appear to range from two to six times between producers and lower consumers including invertebrates and forage fish (Lemly and Smith 1987). Piscivorous fish generally accumulate the highest levels of selenium and are one of the first organisms affected by selenium exposure, followed by planktivores and omnivores (Lemly 1985).

Studies of dietary uptake indicate that selenium can be bioaccumulated through the diet to tissue levels resulting in adverse effects in fish. In a comprehensive review, Lemly (1996b) determined that rainbow trout were sensitive to selenium contamination and exhibited toxic symptoms when their tissue concentrations exceeded 2 mg/kg dry weight in several experiments, and 1 mg/kg in one experiment (note: Lemly (1996b) estimated dry weight concentrations to be four times wet-weight concentrations). Mortality was associated with tissue concentrations greater than 5 mg/kg dry weight (Lemly 1996b). However, Hamilton *et al.* (1986), noted adverse effects on parr-smolt transformation for fall Chinook salmon fed a selenium-contaminated diet when whole-body tissue concentrations were much higher, at 23 mg/kg dry weight (4.9 mg/kg wet weight; conversion factor = 4.63).

Adverse effects have been demonstrated in fish when dietary concentrations exceed approximately 3 mg/kg dry weight (Hamilton *et al.* 1990, Lemly 1996b). However, selenium is also required in the diet as a nutrient at concentrations of about 0.1 to 0.5 mg/kg dry weight (Lemly 1998), so there is a narrow range between healthy and toxic dietary concentrations. Lemly (1996b) noted food chain concentrations on the order of 10 mg/kg to 60 mg/kg were associated with water-borne selenium concentrations in the 2 µg/L to 16 µg/L range. The NMFS and FWS (NMFS 2000) determined in the CTR biological opinion that, assuming a bioaccumulation factor for dry weight concentrations of selenium in aquatic invertebrates (compared to water) of 1,800, a water-borne concentration of as little as 1.8 µg/L selenium could result in food concentrations averaging more than 3 mg/kg selenium, and therefore may be sufficient to result in adverse effects in salmonids.

Variability in experimental and natural conditions influence conclusions regarding safe fish tissue levels, and controlled dietary studies of selenium uptake are subject to questions regarding whether the method through which selenium was administered in the diet reflects natural feeding patterns and food types. Nonetheless, the results of such studies suggest collectively that adverse effects related to bioaccumulation to are likely to occur when water-borne concentrations are below the proposed chronic criterion of 5 µg/L.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for selenium is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. According to Lemly (1996b), the results of field studies generally indicate that benthic invertebrates can accumulate relatively large quantities of selenium (*e.g.*, 20 mg/kg to 370 mg/kg dry weight) and still maintain stable, reproducing populations. Peterson and Nebeker (1992) estimated a dry weight bioaccumulation factor of 1,800 for aquatic insects and invertebrates in the Kesterson National Wildlife Refuge, and noted that Lemly had summarized wet weight factors in a previous review to range between 371 and 5,200. The most significant concern for food organisms from the perspective of listed species is probably bioaccumulation from eating aquatic invertebrates that themselves have elevated selenium levels, rather than changes in aquatic invertebrate production due to selenium toxicity. Hence, the proposed criteria can result in diminished food source quality for listed species through the effects of bioaccumulation.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for selenium is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Selenium. The available evidence for selenium indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), reduced growth (moderate intensity), cellular trauma (low intensity), and bioaccumulation (moderately-high-intensity).

2.6.2.2.10 Silver

Silver Criteria. The proposed acute and chronic criteria for silver are 3.2 µg/L and 0.10 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.10.1 through 2.6.2.2.10.3 report toxicity data from the ECOTOX database for freshwater silver, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.10.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater silver.

Criterion Freshwater Silver		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 3.2 Micrograms Liter^{-1}	**Temperature 9.7-18.4° Celsius**	**Arithmetic Mean 345**
Criterion Concentration Chronic 0.10 Micrograms Liter^{-1}	**Hardness 5-255 mg/L $CaCO_3$**	**Geometric Mean 63**
Endpoint/Effect LC_{50}/Mortality	**pH 6.2-9**	**Harmonic Mean 21**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.28	167 MM	96H
2.71	JUVENILE, 2.2 G	96H
7.32	20 D	96H
9.98	20 D	96H
10.03	1-4 G, JUVENILE	96H
13.52	0.25-1.0G	96H
16.03	0.25-1.0 G	96H
16.32	20 D	96H
20.37	1.2 G	96H
22.22	1.0-1.5 G	96H
22.85	20 D	96H

Criterion Freshwater Silver		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **3.2 Micrograms Liter^{-1}**	**Temperature** **9.7-18.4° Celsius**	**Arithmetic Mean** **345**
Criterion Concentration Chronic **0.10 Micrograms Liter^{-1}**	**Hardness** **5-255 mg/L $CaCO_3$**	**Geometric Mean** **63**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.2-9**	**Harmonic Mean** **21**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
25.38	20 D	96H
27.05	1.0-1.5 G	96H
27.72	JUVENILE, 2.2 G	96H
28.88	NR	96H
31.37	0.25-1.0 G	96H
33.77	69 MM	96H
34.30	0.25-1.0 G	96H
34.34	1-3 G	96H
36.66	Juvenile	
37.56	20 D	96H
38.00	2.5-3.5 G	96H
40.77	NR	96H
40.77	NR	96H
43.73	alevin, 0.24 g	
43.96	Juvenile	
45.33	FORK LENGTH, 0.2 G, 32 MM	96H
47.57	NR	96H
49.20	3-10 G	96H
49.24	Juvenile, 0.41 g	
53.58	Juvenile, 0.1 - 0.2 g	
53.58	Juvenile, 0.51 - 1.44 g	
53.68	3-10 G	96H
59.84	1-3 G	96H
61.46	FORK LENGTH, 0.2 G, 28 MM	96H
63.42	alevin, 0.1 g	
63.79	20 D	96H
69.85	173 MM	96H
75.64	Juvenile, 0.6 g	
83.95	146 MM	96H
93.99	FORK LENGTH, 0.2 G, 28 MM	96H
95.52	1-3 G	96H

Criterion Freshwater Silver		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **3.2 Micrograms Liter^{-1}**	**Temperature** **9.7-18.4° Celsius**	**Arithmetic Mean** **345**
Criterion Concentration Chronic **0.10 Micrograms Liter^{-1}**	**Hardness** **5-255 mg/L $CaCO_3$**	**Geometric Mean** **63**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.2-9**	**Harmonic Mean** **21**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
115.08	20 D	96H
117.75	1-3 G	96H
132.46	1-3 G	96H
191.60	20 D	96H
299.64	Juvenile	
350.66	2.5-3.5 G	96H
396.69	Juvenile	
1102.18	JUVENILE, 2.2 G	96H
1352.01	JUVENILE, 2.2 G	96H
2704.01	JUVENILE, 2.2 G	96H
2718.71	JUVENILE, 2.2 G	96H
3762.10	JUVENILE, 2.2 G	96H
4070.71	JUVENILE, 2.2 G	96H

Table 2.6.2.2.10.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater silver.

Criterion Freshwater Silver		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 3.2 Micrograms Liter^{-1}	**Temperature 5-18.4° Celsius**	**Arithmetic Mean 136**
Criterion Concentration Chronic 0.10 Micrograms Liter^{-1}	**Hardness 12.7-140 mg/L $CaCO_3$**	**Geometric Mean 31**
Endpoint/Effect Growth	**pH 6.1-8.8**	**Harmonic Mean 3**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.96	20 D	28D
1.3	25 (20-30) G, JUVENILE	28D
77	25 (20-30) G, JUVENILE	18M
98.2	20 D	6W
196	20 D	18M
440	20 D	6W

Table 2.6.2.2.10.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater silver.

Criterion Freshwater Silver		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 3.2 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 1.2**
Criterion Concentration Chronic 0.10 Micrograms Liter^{-1}	**Hardness 28-36 mg/L $CaCO_3$**	**Geometric Mean 1.1**
Endpoint/Effect NOEC/Mortality	**pH NR**	**Harmonic Mean 0.98**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.68	NR	NR
1.77	NR	NR

Silver Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to selenium, NMFS added an additional step to its analysis for selenium to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS

calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 3.2 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.10.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.10.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 4,070.71 µg/L to a high of an LC_{100} at a concentration of 1.28 µg/L. In other words, the acute criterion of 3.2 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{3.4}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for silver, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for silver, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects, but may not suffer chronic toxic effects.

Sublethal Effects. Silver is one of the most toxic metals to freshwater organisms and is highly toxic to all life stages of salmonids. Ionic silver is the primary form responsible for causing acute toxicity in freshwater fish (EPA 1980o, 1987b, Eisler 1996, Hogstrand and Wood 1998, Bury *et al.* 1999a). Toxicity varies widely depending on the anion present; silver nitrate has a much higher toxicity than silver chloride or silver thiosulfate, by approximately four orders of magnitude (Hogstrand *et al.* 1996). Documented effects of silver toxicity in fish include interruption of ionoregulation at the gills, cell damage in the gills, altered blood chemistry, interference with zinc metabolism, premature hatching, and reduced growth rates (Hogstrand and Wood 1998, Webb and Wood 1998).

Silver is not known to be mutagenic, teratogenic, or carcinogenic (Eisler 1996). It bioconcentrates and may bioaccumulate (Eisler 1996). Toxicity of Ag may be altered by a number of factors including pH, organic carbon, cation exchange capacity, presence of mixtures (Ratte 1999), sulfides, and duration of exposure. Silver, as ionic Ag+, is one of the most toxic metals known to aquatic organisms in laboratory testing (Nebeker *et al.* 1983). Aquatic insects concentrate silver in relative proportion to environmental levels (Nehring 1976 as cited in EPA 2008), and more efficiently than most fish species (Diamond *et al.* 1990 as cited in EPA 2008). Effects of silver toxicity to freshwater algae and phytoplankton include growth inhibition and altered species composition and species succession (Eisler 1996 as cited in EPA 2008). Effects of silver toxicity to freshwater invertebrates include inhibited feeding and coordination, reduced growth, elevated oxygen consumption, and reduced survival (Eisler 1996 as cited in EPA 2008). Effects of silver toxicity to freshwater fish include inhibited ionic flux across gills, reduced growth, premature hatch, and reduced survival (Eisler 1996 as cited in EPA 2008). Interspecies

differences in the ability to accumulate, retain, and eliminate silver are large (Baudin *et al.* 1994 as cited in EPA 2008).

In the original aquatic life criteria document for silver (EPA 1980o), variation in the results of a limited number of chronic toxicity tests precluded determining a freshwater chronic criterion, but it was also noted that chronic toxicity may occur to selected aquatic organisms at concentrations as low as 0.12 µg/L.

The work of Davies *et al.* (1978) suggests that the maximum acceptable silver concentration to prevent chronic mortality in rainbow trout embryos, fry, and juveniles, and avoid premature hatching, is less than 0.17 µg/L for a water hardness equal to 26 mg/L. Nebeker *et al.* (1983 as cited in Hogstrand and Wood 1998) determined that the maximum acceptable toxicant concentration of silver to prevent inhibition of growth of steelhead embryos was less than 0.1 µg/L for a water hardness equal to 36 mg/L.

The EPA (1987b) reported the results of Davies and Goettl (1978), where chronic limits for silver were listed as between 0.03 µg/L and 0.06 µg/L for a water hardness equal to 28 mg/L, and between 0.03 µg/L and 0.06 µg/L for a water hardness equal to 29 mg/L. Birge *et al.* (1981) estimated an LC_{10} and LC_1 of 0.9 µg/L and 0.1 µg/L, respectively, for rainbow trout embryos and larvae in static renewal tests lasting until 4 days post-hatching.

Accumulation of silver is predominantly associated with exposure to its ionic forms rather than complexes. Bioaccumulation occurs primarily in the liver (Hogstrand *et al.* 1996, Galvez and Wood 1997, 1999). Significant food chain biomagnification by fish has been reported to be unlikely because of the low silver concentrations typically encountered in the aquatic environment (Eisler 1996, Hogstrand and Wood 1998, Ratte 1999).

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for silver is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. The LC_{50}s that have been reported for cladocera species that are below the acute criterion (EPA 1980o). Other invertebrate taxa serving as potential food for juvenile salmonids die only at concentrations that are above the acute criterion. Other observed adverse effects include reductions in growth and inhibition of molting (EPA 1980o, Eisler 1996, Call *et al.* 1999). Chronic effects appear to be documented only for daphnids when silver concentrations are below the proposed chronic criterion. Aquatic invertebrates have been reported to accumulate silver more efficiently than fish, in concentrations that are proportional to exposure levels (Eisler 1996, Hogstrand and Wood 1998). Studies involving silver sulfide bioaccumulation through sediment interactions from an amphipod and an oligochaete indicated low potential for listed species to accumulate harmful silver concentrations through this exposure pathway (Hirsch 1998a,b). Adverse effects of the silver criterion on the food organisms of listed species may be potentially meaningful when cladoceran species are a primary food source.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for silver is likely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Silver. The available evidence for silver indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (moderate intensity), and sublethal effects (moderate intensity).

2.6.2.2.11 Tributyltin

Tributyltin Criteria. At a pH of 7.5 and temperature of 18°C the acute criterion for TBT is 0.46 µg/L, and the chronic criterion is 0.063 µg/L, respectively.

Tables 2.6.2.2.11.1 through 2.6.2.2.11.5 report toxicity data from the ECOTOX database for freshwater tributyltin, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.11.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		**Data Set ECOTOX**
Criterion Concentration Acute 0.46 Micrograms Liter^{-1}	**Temperature 4-15.5° Celsius**	**Arithmetic Mean 8**
Criterion Concentration Chronic 0.063 Micrograms Liter^{-1}	**Hardness 246-280 mg/L $CaCO_3$**	**Geometric Mean 3**
Endpoint/Effect LC_{50}	**pH 6.4-7.95**	**Harmonic Mean 1**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.21	13.8 G	96H
0.54	8.3-8.8 CM, 5.6-6.4 G	6D
0.6	NR	96D
0.6	NR	96D
0.6	NR	24D
1.02	1.47 G	96H
1.16	1.47 G	96H
1.34	1.47 G	96H
3.5	8.8 CM, 6.4 G	96D
4.6	0.77 g	96H
4.84	5.94 G	96H
5.5	1.4 g	96H
6.2	0.68(0.17-1.2) G, 45(39-53) MM	96H
6.6	0.68(0.17-1.2) G, 45(39-53) MM	48H
7.9	0.68(0.17-1.2) G, 45(39-53) MM	72H
11.2	JUVENILE	96H

Criterion Freshwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.46 Micrograms Liter^{-1}	**Temperature 4-15.5° Celsius**	**Arithmetic Mean 8**
Criterion Concentration Chronic 0.063 Micrograms Liter^{-1}	**Hardness 246-280 mg/L $CaCO_3$**	**Geometric Mean 3**
Endpoint/Effect LC_{50}	**pH 6.4-7.95**	**Harmonic Mean 1**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
11.2	JUVENILE	96H
15	0.68(0.17-1.2) G, 45(39-53) MM	48H
21	UNDER-YEARLING	96H
50	NR	96MIN

Table 2.6.2.2.11.2 LC_{100} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.46 Micrograms Liter^{-1}	**Temperature 4-15.5° Celsius**	**Arithmetic Mean 28**
Criterion Concentration Chronic 0.063 Micrograms Liter^{-1}	**Hardness 246-280 mg/L $CaCO_3$**	**Geometric Mean 28**
Endpoint/Effect LC_{100}	**pH 6.4-7.95**	**Harmonic Mean 28**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
28	UNDER-YEARLING	14H

Table 2.6.2.2.11.3 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute **0.46 Micrograms Liter^{-1}**	**Temperature** **4-15.5° Celsius**	**Arithmetic Mean** **7.3**
Criterion Concentration Chronic **0.063 Micrograms Liter^{-1}**	**Hardness** **246-280 mg/L $CaCO_3$**	**Geometric Mean** **2.4**
Endpoint/Effect **Growth**	**pH** **6.4-7.95**	**Harmonic Mean** **1.1**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.5	3 WK	21D
1.46	24.5 G, 25.1 CM FORK LENGTH	NR
20	24.5 G, 25.1 CM FORK LENGTH	21H

Table 2.6.2.2.11.4 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute **0.46 Micrograms Liter^{-1}**	**Temperature** **4-15.5° Celsius**	**Arithmetic Mean** **1**
Criterion Concentration Chronic **0.063 Micrograms Liter^{-1}**	**Hardness** **246-280 mg/L $CaCO_3$**	**Geometric Mean** **0.95**
Endpoint/Effect **Physiological**	**pH** **6.4-7.95**	**Harmonic Mean** **0.86**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.6	4-24 MO, 8.5-20.7 CM, 6.0-94.5 G	65D
1.49	24.5 G, 25.1 CM FORK LENGTH	28H

Table 2.6.2.2.11.5 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute **0.46 Micrograms Liter^{-1}**	**Temperature** **4-15.5° Celsius**	**Arithmetic Mean** **0.77**
Criterion Concentration Chronic **0.063 Micrograms Liter^{-1}**	**Hardness** **246-280 mg/L $CaCO_3$**	**Geometric Mean** **0.69**
Endpoint/Effect **Cellular**	**pH** **6.4-7.95**	**Harmonic Mean** **0.63**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.6	4-24 MO, 8.5-20.7 CM, 6.0-94.5 G	28D
0.5	3 WK	28D
0.5	3 WK	28D
1.49	24.5 G, 25.1 CM FORK LENGTH	72H

TributyltinToxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50

percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to tributyltin, NMFS added an additional step to its analysis for tributyltin to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.46 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.11.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.11.1, predicts a magnitude of effect ranging from a low of an $LC_{0.5}$ at a concentration of 50 µg/L to a high of an LC_{100} at a concentration of 0.21 µg/L. In other words, the acute criterion of 0.46 µg/L has an equivalent toxicity potential predicted to kill 0.5 percent to 100 percent, with a median toxicity potential of an $LC_{4.9}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for tributyltin, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute criterion concentration for tributyltin, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects.

None of the toxicity studies reported concentrations that are less than the chronic criterion for tributyltin, which implies that listed species exposed to waters equal to the chronic criterion concentration may not suffer chronic toxic effects. Based on the available toxicity data and the considerations of the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the chronic criterion concentration may not suffer chronic toxic effects.

Summary of Effects: TBT. The available evidence for TBT indicates that listed species exposed to waters equal to the acute criterion concentration will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (moderate intensity), physiological trauma (moderate intensity), and cellular trauma (moderate intensity).

2.6.2.2.12 Zinc

Zinc Criteria. At hardness of 100 mg/L, the acute criterion is 120 µg/L, and the chronic criterion is 120 µg/L, respectively.

Tables 2.6.2.2.12.1 through 2.6.2.2.12.7 report toxicity data from the ECOTOX database for freshwater zinc, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters, the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.12.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 5-18° Celsius**	**Arithmetic Mean 1172**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 5-350 mg/L $CaCO_3$**	**Geometric Mean 1190**
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.3**	**Harmonic Mean 818**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
238	7 MO, 4.95 G, 8.6 CM, JUVENILE	96H
265	LARVAE	96H
268	7 MO, 4.95 G, 8.6 CM, JUVENILE	96H
308	3.9-6.8 CM FORK LENGTH	96H
316	SWIM-UP, 0.17 G	96H
330	SWIM-UP, 0.23 G	96H
330	7 MO, 4.95 G, 8.6 CM, JUVENILE	96H
353	7 MO, 4.95 G, 8.6 CM, JUVENILE	96H
412	FINGERLING, 2-4 G	96H
425	JUVENILE, 5 MO, 3.0 G, 7.0 CM	120H
444	55 MM	96H
453	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
462	PARR, 6.96 G, 8.6 CM	96H
478	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
487	2.36-3.01 G	96H
487	2.36-3.01 G	96H
487	2.36-3.01 G	168H
510	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
530	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
565	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
616	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
620	NR	96H
628	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
678	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
689	JUVENILE, 3.9 G	96H
709	JUVENILE, 3-10 G	96H
716	FY, 2.36-3.01 G	96H
716	FY, 2.36-3.01 G	168H
720	EYED STAGE	96H

Criterion Freshwater Zinc		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **120 Micrograms Liter^{-1}**	**Temperature** **5-18° Celsius**	**Arithmetic Mean** **1172**
Criterion Concentration Chronic **120 Micrograms Liter^{-1}**	**Hardness** **5-350 mg/L $CaCO_3$**	**Geometric Mean** **1190**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.3**	**Harmonic Mean** **818**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
728	NR	96H
743	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
847	70 MM	96H
861	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
959	JUVENILE, 4.9 G	96H
962	190 MM	96H
1166	30.5 g	
1173	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
1193	JUVENILE, 28.4 G	96H
1361	JUVENILE	96H
1471	JUVENILE, 28.4 G	96H
1509	JUVENILE, 3.9 G	96H
1573	PARR, 11.58 G, 9.6 CM	96H
1577	ALEVIN, 1 MO	115H
1686	120 MM	96H
1768	JUVENILE, 4.9 G	96H
1903	140 MM	96H
2010	NR	96H
2191	3-5 G	96H
2197	22.6 g	
2212	SMOLT, 68.19 G, 18.8 CM	96H
2246	ALEVIN, 0.05 G	96H
2251	179 MM	96H
2382	SMOLT, 32.46 G, 14.4 CM	96H
2385	ADULT, 16-18 CM	96H
2564	JUVENILE	96H
2642	PARRI, 9 MO	96H
2674	110 MM	96H
2769	ALEVIN	96H
2865	NR	96H
2885	JUVENILE, 3.0 G	96H

Criterion Freshwater Zinc		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **120 Micrograms Liter^{-1}**	**Temperature** **5-18° Celsius**	**Arithmetic Mean** **1172**
Criterion Concentration Chronic **120 Micrograms Liter^{-1}**	**Hardness** **5-350 mg/L $CaCO_3$**	**Geometric Mean** **1190**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.3**	**Harmonic Mean** **818**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
2906	ALEVINS, 2-D POSTHATCH	144H
3111	JUVENILE, 19.0 G	96H
3466	Juvenile	
3691	JUVENILE, 3.0 G	96H
3700	FY, 2.36-3.01 G	168H
3829	parr	
4168	JUVENILE, 3.9 G	96H
4699	YEARLING, 10-18 MO	96H
4709	JUVENILE, 19.0 G	96H
4741	YEARLING, 10-18 MO	96H
4955	FY, 2.36-3.01 G	96H
5623	FINGERLING	96H
9784	FINGERLING	96H

Table 2.6.2.2.12.2 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 5-18° Celsius**	**Arithmetic Mean 1642**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 5-350 mg/L $CaCO_3$**	**Geometric Mean 1020**
Endpoint/Effect Mortality	**pH 4.7-8.3**	**Harmonic Mean 173**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
11	EGG	18M
320	FINGERLING, 2 G	21M
320	NR	27M
680	ADULT, 66.3 G	120H
695	ADULT, 66.3 G	131H
724	4 WK, LARVAE, SWIM-UP	56D
724	4 WK LARVAE, SWIM-UP	56D
724	EGG	84D
1368	4 WK LARVAE, SWIM-UP	56D
1368	4 WK, LARVAE, SWIM-UP	56D
1368	NEWLY HATCHED LARVAE	84D
1368	EGG	84D
2058	NEWLY HATCHED LARVAE	84D
2476	JUVENILE, 0.316 G	114H
2818	JUVENILE, 0.316 G	117H
3004	JUVENILE, 0.316 G	156H
3077	JUVENILE, 0.316 G	141H
3090	JUVENILE, 0.316 G	141H
5000	JUVENILE, 0.316 G	120H

Table 2.6.2.2.12.3 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 3-20° Celsius**	**Arithmetic Mean 193**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 20-374 mg/L $CaCO_3$**	**Geometric Mean 174**
Endpoint/Effect Growth	**pH 4.7-8.64**	**Harmonic Mean 161**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
104	NR	4D
104	NR	85D
104	NR	85D
104	NR	40W
104	NR	40W
132	NR	180D
132	NR	191D
132	NR	50D
132	NR	40W
172	NR	191D
172	NR	191D
172	NR	180D
172	NR	30D
172	NR	30D
172	NR	40W
172	NR	40W
172	NR	40W
172	NR	21M
172	NR	13W
172	NR	2M
172	NR	13W
358	45 G, YEARLING	13W
384	NR	30D
384	NR	40W
384	NR	1H
384	NR	55D

Table 2.6.2.2.12.4 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 5-18° Celsius**	**Arithmetic Mean 615**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 20-374 mg/L $CaCO_3$**	**Geometric Mean 436**
Endpoint/Effect NOEC/Mortality/Reproduction	**pH 4.7-8.3**	**Harmonic Mean 277**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
98	NR	1M
108	NR	27M
380	EGG	18M
432	JUVENILE	NR
595	ADULT-SMOLT	NR
862	ADULT-SMOLT	NR
1028	YEARLING, 70 G, 3RD GENERATION	82D
1417	EGG	72D

Table 2.6.2.2.12.5 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 3-20° Celsius**	**Arithmetic Mean 38541**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 45-374 mg/L $CaCO_3$**	**Geometric Mean 3075**
Endpoint/Effect Cellular	**pH 4.7-8.64**	**Harmonic Mean 235**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
91	6-18 MO	3.15H
166	45 G, YEARLING	96H
76954	8-12 G, 9-11 CM	0.5H
76954	8-12 G, 9-11 CM	4H

Table 2.6.2.2.12.6 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 3-20° Celsius**	**Arithmetic Mean 2753**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 22-90 mg/L $CaCO_3$**	**Geometric Mean 2427**
Endpoint/Effect Physiological	**pH 4.7-8.64**	**Harmonic Mean 2199**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1360	YEARLING, 70 G, 3RD GENERATION	96H
1370	4 WK, LARVAE, SWIM-UP	4M
1370	EGG	1H
1370	4 WK LARVAE, SWIM-UP	1H
1984	NR	30D
2025	14.4 CM	17H
2074	14.4 CM	16W
2387	13.5 CM	2H
2588	NEWLY HATCHED LARVAE	4H
2588	4 WK, LARVAE, SWIM-UP	4H
2588	EGG	3.15H
2729	13.5 CM	43MIN
3212	14.4 CM	72H
3528	14.4 CM	2H
4857	13.5 CM	6H
8020	NR	30D

Table 2.6.2.2.12.7 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 3-20° Celsius**	**Arithmetic Mean 224**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 30-350 mg/L $CaCO_3$**	**Geometric Mean 147**
Endpoint/Effect Reproductive	**pH 4.7-8.64**	**Harmonic Mean 84**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
30	NR	0.67H
108	NR	0.67H
379	8.3 CM	21M
379	FINGERLING, 2 G	10D

Zinc Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50

percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to zinc, NMFS added an additional step to its analysis for zinc to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 120 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.12.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.12.1, predicts a magnitude of effect ranging from a low of an $LC_{0.6}$ at a concentration of 9,784 µg/L to a high of an $LC_{25.2}$ at a concentration of 238 µg/L. In other words, the acute criterion of 120 µg/L has an equivalent toxicity potential predicted to kill 0.6 percent to 25.2 percent, with a median toxicity potential of an $LC_{5.1}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for zinc, which implies that listed species exposed to waters equal to criteria concentrations will not be protected from acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for zinc, which implies that listed species exposed to waters equal to criteria concentrations will be protected from acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Zinc is an essential element required for healthy fish, and is present in healthy fish tissues in greater concentrations than other heavy metals. However, increased levels of zinc over natural body concentrations can result in mortality, growth retardation, histopathological alterations, respiratory and cardiac changes, and inhibition of spawning and many other elements critical to fish survival. Exposure to high zinc concentrations can result in damage to the gills, liver, kidney and skeletal muscle and cause a physiological shift to occur, making gas exchange more difficult. Toxicity varies with hardness, pH, alkalinity, dissolved oxygen, water temperature, species and life stage, acclimation, and ambient concentrations of other chemicals in the water (EPA 1987c, Sorensen 1991, Eisler 1993). For example, the toxicity of zinc is influenced by antagonistic interactions with cadmium, copper, iron, and molybdenum (Hammond and Beliles 1980). There is evidence that fish acclimated to elevated temperature are more tolerant of zinc toxicity (Hodson and Sprague 1975).

Behavioral avoidance reactions have been noted in three trout species at zinc concentrations that were below the proposed chronic criterion. Juvenile rainbow trout avoidance was documented at zinc concentrations of 5.6 µg/L at a hardness of 13 mg/L (Sprague 1968) and 47 µg/L at a hardness of 112 mg/L (Birge and Black 1980 as cited in EPA 1987c). Juvenile brown trout avoidance was documented at 25 µg/L at a hardness of 100 mg/L (Woodward *et al.* 1995). Juvenile cutthroat trout avoidance was documented at 28 µg/L at a hardness of 50 mg/L (Woodward *et al.* 1997). Avoidance behavior by adult salmonids has not been studied as extensively. As with copper, there are insufficient data available to identify whether these behavioral effects translate into adverse effects in the field because of the confounding influence of acclimation, complexing organic material in natural waters, uncontrolled variables, presence of other metals, and field observations that found fish in "impacted" streams when "un-impacted" streams were also available.

Zinc bioconcentrates but does not biomagnify (EPA 1999). Zinc may be mutagenic and teratogenic (Eisler 1993). Toxicity of zinc to aquatic organisms is dependent on water hardness, pH, DO, presence of mixtures, and trophic level (Sorensen 1991, Eisler 1993). Zinc interacts with many chemicals to produce altered patterns of accumulation, metabolism, and toxicity; some interactions reduce toxicity and others increase toxicity (Eisler 1993). Most of the zinc introduced into aquatic environments is eventually partitioned into sediments (Eisler 1993). Zinc bioavailability from sediment is increased under conditions of high DO, low salinity, low pH, and high levels of inorganic oxides and humic substances. Effects of zinc toxicity to freshwater organisms include reduced growth, reduced populations, and reduced survival in algae species; reduced growth, activity, larval settlement, and reproduction, osmoregulatory impairment and reduced survival in freshwater invertebrates (including molluscs); and reduced growth, behavioral alteration (avoidance), reproduction impairment, increased respiration, decreased swimming ability, increased jaw and branchial abnormalities, hyperactivity, hyperglycemia, and reduced survival in freshwater fish (Eisler 1993).

In Farag *et al.* (1994), they determined that continuous exposure to zinc at the proposed chronic criterion concentration was associated with bioaccumulation of the metal by juvenile and adult rainbow trout. In Mount *et al.* (1994), they determined that tissue concentrations increased in rainbow trout fry fed a diet containing enriched levels of zinc. However, the issue of zinc bioaccumulation in salmonids is confounded by naturally high tissue concentrations and the

ability of fish to regulate internal concentrations. In Alsop *et al.* (1999), they determined that tissue concentrations of zinc in fish exposed to approximately one to two times the acute criterion were not a good indicator of non-lethal, chronic zinc exposure. Physiological costs related to zinc acclimation were determined to be few. The work by Mount *et al.* (1994) did not detect significant effects on survival or growth in rainbow trout fry fed quantities of zinc that were 10 times or greater in concentration than other metals. These studies suggest collectively that the ability of salmonids to regulate internal zinc concentrations may minimize adverse effects of bioaccumulation when the fish are exposed to zinc concentrations near the proposed chronic criterion.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for zinc is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Many freshwater insects and crustaceans appear to be tolerant of zinc concentrations that are similar to the acute criterion (Eisler 1993), although some taxa can be more sensitive to chronic effects than salmonids (Kemble *et al.* 1994). Aquatic invertebrates bioaccumulate zinc to a greater degree than salmonids (EPA 1987c, Eisler 1993). Kiffney and Clements (1994) determined that mayflies were sensitive to zinc, and that the response varied with stream size or location in the stream network. Data in EPA (1987c) indicate that the zinc criteria are usually non-lethal to invertebrates that juvenile listed species feed on, although in two cases in EPA (1987c), cladoceran species exhibited LC_{50}s that were lower than the acute and chronic criteria at a hardness of 45 mg/L. Invertebrate communities in rivers appear to respond to elevated zinc levels in the sediments by changing composition to pollution-tolerant taxa, rather than by reducing overall biomass (Canfield *et al.* 1994, Clements and Kiffney 1994). It is not clear if this adversely affects foraging ability of juvenile salmon.

Zinc contained in bed sediments has been found to be elevated in benthic invertebrates in field studies conducted in metals-contaminated streams (Ingersoll *et al.* 1994; Woodward *et al.* 1994). However, Kiffney and Clements (1996) determined an inverse relation existed between aquatic macroinvertebrate body size and survival at zinc levels in excess of the proposed chronic criterion, which partially counters the effects of bioaccumulation, as organisms die before they are large enough to bioaccumulate high concentrations of zinc. Indirect effects of elevated zinc levels to listed species include reductions in production of larger bodied invertebrate taxa that could influence the availability of food for larger juvenile salmonids, and ingestion of bioconcentrated zinc by fry and juveniles of all sizes.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for zinc is likely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Zinc. The available evidence for zinc indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (moderately-high-intensity), cellular trauma (moderate intensity), physiological trauma (moderate intensity), and reproductive failure (moderately-high-intensity).

2.6.3 Saltwater Criteria Toxicity Analysis

The ESA directs that section 7 consultations use the best available scientific and commercial data. While EPA conducted an extensive data call and has developed a large database of toxicity (ECOTOX), thousands of toxicity studies were rejected by EPA for use in criteria development and formulation of the BE. A majority of these toxicity studies were rejected because the test duration was non-standard; EPA generally does not consider toxicity tests with non-standard durations (*e.g.*, 4-hr LC_{50} or 144-hr LC_{50}). However, these studies mat still meet the standard of the "best available scientific data" as defined by the ESA. For this consultation, NMFS used a much more extensive toxicity data set, including toxicity studies from the ECOTOX database that were excluded by EPA, for its analysis.

The analysis on saltwater criteria starts with a review of the chemical and toxicological concepts, principals, and factors that influence toxicity for each compound, and an assessment of critical exposure-response factors pertinent to the overall analysis. The data analysis in this section has four general components: (1) Available toxicity data presented in table format by endpoint; (2) a summary statistical analysis performed for each endpoint data set consisting of the arithmetic mean, the geometric mean, and the harmonic mean to assess the distribution of the data for each data set, and the statistical analysis is used later in the analysis on chemical mixtures; (3) a sublethal effects analysis on the chronic criteria, and (4) an analysis on food items (when data was available). Due to the paucity of acute saltwater data, NMFS did nor calculate a relative percent mortality for each acute saltwater criterion.

The toxicity data for salmonid fishes includes data for listed and non-listed salmonid fishes, *e.g.*, rainbow trout are used to directly assess toxicity effects on steelhead as the resident form is indistinguishable from the anadromous form in juvenile life stages. Other salmonid fishes, *e.g.*, brook trout (*Salvelinus fontinalis*) and cutthroat trout (*Oncorhynchus clarki*), are used in addition to the species-specific toxicity data and/or as a surrogate for listed species where toxicity data is not available for listed species to analyze effects on additional endpoints. Our analysis of surrogate species toxicity data showed no difference in the range of concentrations when compared to the toxicity data for listed species. Furthermore, toxicity data for green sturgeon and Eulachon was limited or non-existent for most of the compounds in Table 1.1. Therefore, NMFS used the salmonid fishes toxicity data as a surrogate for these two species as these toxicity data sets for salmonid fishes were the closest taxonomic data available. The summary conclusions provided in this section are based on a toxicity exposure-response potential to listed species considered in this opinion for each freshwater compound listed in Table 1.1, based exclusively on an examination of the available toxicity data from exposure to a single compound. The summary conclusions do not take into account effects to listed species considered in this opinion from exposure to multiple compounds. The issue of chemical mixtures, as well as criteria development and implementation issues, direct mortality population modeling, *etc.*, are examined in the *Integration and Synthesis*.

2.6.3.1 Arsenic

Saltwater Arsenic Criteria. The proposed acute and chronic criteria for saltwater arsenic are 69 µg/L and 36 µg/L, respectively.

Tables 2.6.3.1.1 and 2.6.3.1.2 report toxicity data from the ECOTOX database for saltwater arsenic, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.1.1 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater arsenic.

Criterion Saltwater Arsenic		**Data Set BE**
Criterion Concentration Acute 69 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 6658**
Criterion Concentration Chronic 36 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 6658**
Endpoint/Effect Mortality	**pH NR**	**Harmonic Mean 6658**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
6658	NR	NR

Table 2.6.3.1.2. NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater arsenic.

Criterion Saltwater Arsenic		**Data Set BE**
Criterion Concentration Acute 69 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 3974**
Criterion Concentration Chronic 36 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 3974**
Endpoint/Effect NOEC	**pH NR**	**Harmonic Mean 3974**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3974	NR	NR

Summary of Effects: Arsenic. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater arsenic indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and sublethal effects (moderate intensity).

2.6.3.2 Cadmium

Cadmium Criteria. The proposed acute and chronic criteria for saltwater cadmium are 40 µg/L and 8.8 µg/L, respectively.

Tables 2.6.3.2.1 through 2.6.3.2.3 report toxicity data from the ECOTOX database for saltwater cadmium, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.2.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater cadmium.

Criterion Saltwater Cadmium		**Data Set ECOTOX**
Criterion Concentration Acute 40 Micrograms Liter^{-1}	**Temperature 11.2° Celsius**	**Arithmetic Mean 1200**
Criterion Concentration Chronic 8.8 Micrograms Liter^{-1}	**Salinity 28.3 ppt**	**Geometric Mean 1200**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 1200**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1200	SMOLTS, 128 MM	96H

Table 2.6.3.2.2 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater cadmium.

Criterion Saltwater Cadmium		**Data Set ECOTOX**
Criterion Concentration Acute 40 Micrograms Liter^{-1}	**Temperature 11.2° Celsius**	**Arithmetic Mean 1200**
Criterion Concentration Chronic 8.8 Micrograms Liter^{-1}	**Salinity 28.3 ppt**	**Geometric Mean 1200**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 1200**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1200	SMOLTS, 128 MM	96H

Table 2.6.3.2.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater cadmium.

Criterion Saltwater Cadmium		Data Set BE
Criterion Concentration Acute 40 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 163.7**
Criterion Concentration Chronic 8.8 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 163.7**
Endpoint/Effect NOEC	**pH NR**	**Harmonic Mean 163.7**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
163.7	Smolts	

Summary of Effects: Cadmium. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for cadmium indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and sublethal effects (moderate intensity).

2.6.3.3 Chromium VI

CR (VI) Criteria. The proposed acute and chronic criteria for chromium (VI) are 1100 µg/L and 50 µg/L, respectively.

Tables 2.6.3.3.1 through 2.6.3.3.4 report toxicity data from the ECOTOX database for saltwater chromium (VI), except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.3.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater chromium VI.

Criterion Saltwater Chromium VI		**Data Set ECOTOX**
Criterion Concentration Acute 1100 Micrograms Liter^{-1}	**Temperature 3.5-19° Celsius**	**Arithmetic Mean 98129**
Criterion Concentration Chronic 50 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 68333**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 44884**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
12079	NR	96H
27201	NR	96H
27496	NR	96H
37905	NR	96H
69722	NR	96H
74239	NR	96H
98200	NR	96H
109002	NR	96H
141408	NR	96H
201310	NR	96H
280852	NR	96H

Table 2.6.3.3.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater chromium VI.

Criterion Saltwater Chromium VI		**Data Set ECOTOX**
Criterion Concentration Acute 1100 Micrograms Liter^{-1}	**Temperature 3.5-19° Celsius**	**Arithmetic Mean 91**
Criterion Concentration Chronic 50 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 47**
Endpoint/Effect Growth	**pH NR**	**Harmonic Mean 24**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
10	NR	7M
13	NR	110D
49	NR	
192	NR	
192	NR	

Summary of Effects: Chromium VI. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater chromium (VI) indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and sublethal effects (moderately-high-intensity).

2.6.3.4 Copper

Copper Criteria. The proposed acute and chronic criteria for saltwater copper are 4.8 µg/L and 3.1 µg/L, respectively.

Tables 2.6.3.4.1 through 2.6.3.4.3 report toxicity data from the ECOTOX database for saltwater copper, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.4.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater copper.

Criterion Saltwater Copper		**Data Set ECOTOX**
Criterion Concentration Acute 4.8 Micrograms Liter^{-1}	**Temperature 13° Celsius**	**Arithmetic Mean 329**
Criterion Concentration Chronic 3.1 Micrograms Liter^{-1}	**Salinity 28.6 ppt**	**Geometric Mean 329**
Endpoint/Effect LC_{50}/Mortality	**pH 8.1**	**Harmonic Mean 329**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
329	SMOLTS, 132 MM	96H

Table 2.6.3.4.2 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater copper.

Criterion Saltwater Copper		**Data Set ECOTOX**
Criterion Concentration Acute 4.8 Micrograms Liter^{-1}	**Temperature 10.3-13Celsius**	**Arithmetic Mean 329**
Criterion Concentration Chronic 3.1 Micrograms Liter^{-1}	**Salinity 12-35 ppt**	**Geometric Mean 329**
Endpoint/Effect LC_{50}/Mortality	**pH 7.8-8.1**	**Harmonic Mean 329**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
329	SMOLT, 132 MM	96H
329	SMOLTS, 132 MM	96H

Table 2.6.3.4.3 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater copper.

Criterion Saltwater Copper		**Data Set ECOTOX**
Criterion Concentration Acute 4.8 Micrograms Liter^{-1}	**Temperature 10.3-13Celsius**	**Arithmetic Mean 31**
Criterion Concentration Chronic 3.1 Micrograms Liter^{-1}	**Salinity 12-35 ppt**	**Geometric Mean 31**
Endpoint/Effect Reproductive	**pH 7.8-8.1**	**Harmonic Mean 31**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
31	Gamete	60MIN

Summary of Effects: Copper. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity

tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater copper indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and reproductive failure (moderate intensity).

2.6.3.5 Endosulfan (Endosulfan-alpha and Endosulfan-beta)

Endosulfan-a and Endosulfan-b Criteria. The proposed acute and chronic criteria for saltwater endosulfan-a and endosulfan-b are 0.034 µg/L and 0.0087 µg/L, respectively.

Tables 2.6.3.5.1 and 2.6.3.5.2 report toxicity data from the ECOTOX database for saltwater endosulfan, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.5.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater endosulfan-alpha and endosulfan-beta.

Criterion **Saltwater Endosulfan-alpha and Endosulfan-beta**		**Data Set** **ECOTOX**
Criterion Concentration Acute **0.034 Micrograms Liter^{-1}**	**Temperature** **11.4° Celsius**	**Arithmetic Mean** **1.7**
Criterion Concentration Chronic **0.0087 Micrograms Liter^{-1}**	**Salinity** **NR**	**Geometric Mean** **1.7**
Endpoint/Effect **LC_{50}**	**pH** **8.1**	**Harmonic Mean** **1.7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
1.69	SMOLT, 127 MM	96H

Table 2.6.3.5.2 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater endosulfan-alpha and endosulfan-beta.

Criterion **Saltwater Endosulfan-alpha and Endosulfan-beta**		**Data Set** **ECOTOX**
Criterion Concentration Acute **0.034 Micrograms Liter^{-1}**	**Temperature** **11.4-12° Celsius**	**Arithmetic Mean** **765.5**
Criterion Concentration Chronic **0.0087 Micrograms Liter^{-1}**	**Salinity** **NR**	**Geometric Mean** **765.5**
Endpoint/Effect **Reproductive**	**pH** **7.8-8.2**	**Harmonic Mean** **765.5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
765.5	GAMETE	60MIN

Summary of Effects: Endosulfan-a and Endosulfan-b. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater endosulfan-alpha and endosulfan-beta indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and reproductive failure (low intensity).

2.6.3.6 Heptachlor Epoxide

Heptachlor Epoxide Criteria. The proposed acute and chronic criteria for saltwater heptachlor epoxide are 0.053 µg/L and 0.0036 µg/L, respectively.

Tables 2.6.3.6.1 and 2.6.3.6.2 report toxicity data from the ECOTOX database for saltwater heptachlor epoxide, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.6.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater heptachlor epoxide.

Criterion **Saltwater Heptachlor**		**Data Set BE**
Criterion Concentration Acute **0.053 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **0.37**
Criterion Concentration Chronic **0.0036 Micrograms Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **0.37**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **NR**	**Harmonic Mean** **0.37**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.367		

Table 2.6.3.6.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater heptachlor epoxide.

Criterion Saltwater Heptachlor		Data Set BE
Criterion Concentration Acute **0.053 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **0.2**
Criterion Concentration Chronic **0.0036 Micrograms Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **0.2**
Endpoint/Effect **NOEC**	**pH** **NR**	**Harmonic Mean** **0.2**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.214		

Summary of Effects: Heptachlor Epoxide. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater heptachlor epoxide indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity) and sublethal effects (low intensity).

2.6.3.7 Lead

Lead Criteria. The proposed acute and chronic criteria for lead are 210 µg/L and 8.1 µg/L, respectively.

Tables 2.6.3.7.1 through 2.6.3.7.3 report toxicity data from the ECOTOX database for saltwater lead, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.7.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater lead.

Criterion Saltwater Lead		Data Set BE
Criterion Concentration Acute 210 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 805**
Criterion Concentration Chronic 8.1 Micrograms Liter^{-1}	**Hardness NR**	**Geometric Mean 805**
Endpoint/Effect LC_{50}/Mortality	**pH NR**	**Harmonic Mean 805**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
805		

Table 2.6.3.7.2 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater lead.

Criterion Saltwater Lead		Data Set ECOTOX
Criterion Concentration Acute 210 Micrograms Liter^{-1}	**Temperature 12-13.7° Celsius**	**Arithmetic Mean 150**
Criterion Concentration Chronic 8.1 Micrograms Liter^{-1}	**Salinity 27-30 ppt**	**Geometric Mean 150**
Endpoint/Effect Physiological	**pH 7.8-8.2**	**Harmonic Mean 150**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
150	200 G, SALTWATER ADAPTED	2W

Table 2.6.3.7.3 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater lead.

Criterion Saltwater Lead		Data Set ECOTOX
Criterion Concentration Acute 210 Micrograms Liter^{-1}	**Temperature 12-13.7° Celsius**	**Arithmetic Mean 24000**
Criterion Concentration Chronic 8.1 Micrograms Liter^{-1}	**Salinity 27-30 ppt**	**Geometric Mean 24000**
Endpoint/Effect Reproductive	**pH 7.8-8.2**	**Harmonic Mean 24000**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
24000	GAMETE	2W

Summary of Effects: Lead. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater lead indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity), physiological trauma (moderate intensity), and reproductive failure (low intensity).

2.6.3.8 Nickel

Nickel Criteria. The proposed acute and chronic criteria for saltwater nickel are 74 µg/L and 8.2 µg/L, respectively.

Tables 2.6.3.8.1 and 2.6.3.8.2 report toxicity data from the ECOTOX database for saltwater nickel, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.8.1. LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater nickel.

Criterion Saltwater Nickel		**Data Set BE**
Criterion Concentration Acute 74 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 4893**
Criterion Concentration Chronic 8.2 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 4893**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 4893**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
4893		

Table 2.6.3.8.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater nickel.

Criterion Saltwater Nickel		Data Set BE
Criterion Concentration Acute 74 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 1793**
Criterion Concentration Chronic 8.2 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 1793**
Endpoint/Effect NOEC	**pH NR**	**Harmonic Mean 1793**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1793		

Summary of Effects: Nickel. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater nickel indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity) and sublethal effects (low intensity).

2.6.3.9 Pentachlorophenol

Pentachlorophenol Criteria. The proposed chronic criterion for saltwater PCP is 7.9 µg/L, respectively.

Table 2.6.3.9.1 reports toxicity data from the ECOTOX database for saltwater pentachlorophenol, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.9.1 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater pentachlorophenol.

Criterion Saltwater Pentachlorophenol		Data Set BE
	Temperature **NR**	**Arithmetic Mean** **10.5**
Criterion Concentration Chronic **7.9 Micrograms Liter^{-1}**	**Salinity** **NR**	**Geometric Mean** **10.5**
Endpoint/Effect **NOEC**	**pH** **NR**	**Harmonic Mean** **10.5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
10.5		

Summary of Effects: Pentachlorophenol. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater PCP indicates that listed species exposed to waters equal to the chronic criterion concentrations will suffer chronic toxic effects including sublethal effects (moderately-high-intensity).

2.6.3.10 Selenium

Selenium Criteria. The proposed acute and chronic criteria for saltwater selenium are 290 µg/L and 71 µg/L, respectively.

Tables 2.6.3.10.1 and 2.6.3.10.2 report toxicity data from the ECOTOX database for saltwater selenium, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.10.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater selenium.

Criterion Saltwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 290 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 76750**
Criterion Concentration Chronic 71 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 43547**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 30929**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
11600	FRY, 2.6 G	96H
11600	FRY, 2.6 G	96H
16600	1.6 G, FRY	96H
16600	1.6 G, FRY	96H
17200	1.6 G, FRY	96H
17200	1.6 G, FRY	96H
18300	FRY, 2.6 G	96H
18300	FRY, 2.6 G	96H
19600	FRY, 2.6 G	96H
19600	FRY, 2.6 G	96H
23900	FRY, 2.6 G	96H
23900	FRY, 2.6 G	96H
28200	FRY, 2.6 G	96H
28200	FRY, 2.6 G	96H
29000	FRY, 1.7 G	96H
29000	FRY, 1.7 G	96H
36100	FRY, 2.6 G	24H
39600	1.6 G, FRY	24H
43200	FRY, 2.4 G	96H
43200	FRY, 2.4 G	96H

Criterion Saltwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 290 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 76750**
Criterion Concentration Chronic 71 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 43547**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 30929**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
50100	FRY, 2.4 G	96H
50100	FRY, 2.4 G	96H
63800	1.6 G, FRY	24H
65400	FRY, 2.4 G	96H
65400	FRY, 2.4 G	96H
79400	FRY, 1.8 G	96H
79400	FRY, 1.8 G	96H
94000	FRY, 1.6 G	96H
94000	FRY, 1.6 G	96H
136000	FRY, 1.6 G	96H
136000	FRY, 1.6 G	96H
236000	FRY, 1.6 G	24H
369000	FRY, 1.7 G	24H
600000	FRY, 1.6 G	24H

Table 2.6.3.10.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater selenium.

Criterion Saltwater Selenium		Data Set BE
Criterion Concentration Acute 290 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 5551**
Criterion Concentration Chronic 71 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 5048**
Endpoint/Effect NOEC	**pH NR**	**Harmonic Mean 4591**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3243		
7859		

Summary of Effects: Selenium. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species

exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater selenium indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity) and sublethal effects (low intensity).

2.6.3.11 Silver

Silver Criteria. The proposed acute criterion for saltwater silver is 1.9 µg/L.

Tables 2.6.3.11.1 reports toxicity data from the ECOTOX database for saltwater silver, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.11.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater silver.

Criterion Saltwater Silver		**Data Set ECOTOX**
Criterion Concentration Acute 1.9 Micrograms Liter^{-1}	**Temperature 11.5-14° Celsius**	**Arithmetic Mean 195**
	Salinity 25-28.6 ppt	**Geometric Mean 194**
Endpoint/Effect LC_{50}	**pH 7.8-8.2**	**Harmonic Mean 193**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
176	25 G	96H
214	SMOLT, 131 MM	96H

Summary of Effects: Silver. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some

compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

In summary, the available evidence for saltwater silver indicates that listed species exposed to waters equal to the acute criterion concentrations will suffer chronic toxic effects including sublethal effects (low intensity).

2.6.3.12 Tributyltin

Tributyltin Criteria. The proposed acute and chronic criteria for saltwater TBT are 0.37 µg/L and 0.01 µg/L, respectively.

Tables 2.6.3.12.1 through 2.6.3.12.4 report toxicity data from the ECOTOX database for saltwater tributyltin, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.12.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater tributyltin.

Criterion Saltwater Tributyltin		**Data Set ECOTOX**
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 12**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 6.7**
Endpoint/Effect LC_{50}/Mortality	**pH 6.4-7.8**	**Harmonic Mean 3.6**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.02	1.47 G	96H
1.16	1.47 G	96H
1.34	1.47 G	96H
1.46	24.5 G, 25.1 CM FORK LENGTH	96H

Criterion Saltwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 12**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 6.7**
Endpoint/Effect LC_{50}/Mortality	**pH 6.4-7.8**	**Harmonic Mean 3.6**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
4.6	0.77 g	96H
4.84	5.94 G	96H
5.5	1.4 g	96H
6.2	0.68(0.17-1.2) G, 45(39-53) MM	96H
6.6	0.68(0.17-1.2) G, 45(39-53) MM	72H
7.9	0.68(0.17-1.2) G, 45(39-53) MM	48H
11	JUVENILE	96H
11	JUVENILE	96H
15	0.68(0.17-1.2) G, 45(39-53) MM	24H
20	24.5 G, 25.1 CM FORK LENGTH	12H
21	UNDER-YEARLING	48H
28	UNDER-YEARLING	24H
54	24.5 G, 25.1 CM FORK LENGTH	6H

Table 2.6.3.12.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater tributyltin.

Criterion Saltwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 0.52**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 0.52**
Endpoint/Effect Growth	**pH 6.4-7.8**	**Harmonic Mean 0.52**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.5	3 WK	21D
0.54	8.3-8.8 CM, 5.6-6.4 G	10D

Table 2.6.3.12.3 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater tributyltin.

Criterion Saltwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 0.58**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 0.58**
Endpoint/Effect Cellular	**pH 6.4-7.8**	**Harmonic Mean 0.58**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.5	3 WK	7D
0.6	NR	28D
0.6	NR	28D
0.6	NR	28D
0.6	4-24 MO, 8.5-20.7 CM, 6.0-94.5 G	10D

Table 2.6.3.12.4 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater tributyltin.

Criterion Saltwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 27**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 13**
Endpoint/Effect Physiological	**pH 6.4-7.8**	**Harmonic Mean 6.5**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3.5	8.8 CM, 6.4 G	28D
50	NR	65MIN

Summary of Effects: Tributyltin. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range

between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater tributyltin indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity), sublethal effects (low intensity), physiological trauma (low intensity), and cellular trauma (low intensity).

2.6.3.13 Zinc

Zinc Criteria. The proposed acute and chronic criteria for saltwater zinc are 90 µg/L and 81 µg/L.

Tables 2.6.3.13.1 through 2.6.3.13.2 report toxicity data from the ECOTOX database for saltwater zinc, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.13.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater zinc.

Criterion Saltwater Zinc		**Data Set ECOTOX**
Criterion Concentration Acute 90 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 3000**
Criterion Concentration Chronic 81 Micrograms Liter^{-1}	**Salinity 27 ppt**	**Geometric Mean 2828**
Endpoint/Effect LC_{50}	**pH 7.8-8.2**	**Harmonic Mean 2667**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
2000	2 YR, PARR, 14.8 CM FL	48H
4000	YEARLING, 14.5 CM FL	48H

Table 2.6.3.13.2 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater zinc.

Criterion Saltwater Zinc		**Data Set ECOTOX**
Criterion Concentration Acute 90 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 819**
Criterion Concentration Chronic 81 Micrograms Liter^{-1}	**Salinity 27 ppt**	**Geometric Mean 819**
Endpoint/Effect Reproductive	**pH 7.8-8.2**	**Harmonic Mean 819**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
819	GAMETE	60MIN

Summary of Effects: Zinc. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species

exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, he available evidence for saltwater zinc indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity) and reproductive failure (low intensity).

2.6.4 Chemical Mixtures

Where multiple toxic effluents are discharged to receiving water, the resultant ambient toxicity is of interest. Since each effluent is composed of individual toxic substances, a mixture of the effluents in receiving water produces a mixture of these individual pollutants. The overall ambient toxicity could be equal to the sum of each discharge's toxicity (additivity), less than the sum (antagonism), or greater than the sum (synergism). Although the technology does exist to conduct site-specific chemical mixtures analysis, neither the data nor the technical capabilities exist to conduct a chemical mixtures analysis for the compounds listed in Table 1.1 at the scale of this consultation. This is because there are more than 3,000 point source discharges in Oregon, and each discharge represents a unique mixture of pollutants that varies considerably seasonally or more frequently. Once in the receiving water bodies, these discharged pollutants mix with pollutants from non-point sources and natural sources, at rates that are influenced by changes in river discharges. The result is an almost unlimited number of combinations of pollutant types and concentrations that varies nearly continuously and makes a quantitative mixture analysis across the State of Oregon impracticable and unrealistic task. Nonetheless, the issue of chemical mixtures is an important line of evidence to consider when assessing the exposure-response effects and risks to the listed species considered in this opinion.

The concept of independent joint action (also commonly termed response addition) was formalized by Loewe and Muischnek (1926 as cited in EPA 2008) and is used to describe the toxicity of a mixture in which the chemical constituents elicit their effects independently via different mechanisms of action. The other commonly used method to assess mixture toxicity is termed concentration addition (Bliss 1939) and assumes a common mechanism of action. Rider and LeBlanc (2005) and Meyer *et al.* (2007) have integrated these models in a manner that allows assessment of mixture toxicity using both concentration addition and independent joint action in which the toxic response associated with each group of compounds that share a common mechanism of action is first calculated using the concentration addition approach. The combined toxic responses associated with all groups of compounds are then calculated by independent joint action to the yield the predicted effect for the entire mixture.

Norwood *et al.* (2003), in a review of the toxicity of metal mixtures to aquatic species derived from a database of information from 68 literature citations, and mixture effects on 77 species, observed that the commonly used concentration addition approaches accurately predicted metal mixture toxicity 27% of the time. Mixture toxicity was less than additive (*i.e.* the concentration response approach overpredicted mixture toxicity) 43% of the time. The remaining 29% of the mixtures were more than additive (*i.e.* the concentration response approach underestimated mixture toxicity). Norwood *et al.* (2003) attributed the underprediction of mixture toxicity largely to interactions between mixture components. The variability in the studies could be due to different mixtures of metals being used and that some metals may share a common mechanism of action while others may not.

The available information in EPA's technical support document for water quality-based toxics control (EPA 1991) indicates that the combined effects of individual acutely toxic pollutants are 0.4 to 2.8 times the effects predicted by adding the individual effects. The median combined effect is approximately additive (EPA 1991). For this reason, EPA recommends in the absence of site-specific data that regulatory authorities consider combined acute toxicity to be additive. In relation to chronic toxicity, for the growth of fish, Alabaster and Lloyd (1965 as cited in EPA 1991) conclude the joint effect of toxicants has been consistently less than additive, which suggests that dose addition is not the appropriate model for that endpoint.

Although each method described above has its pros and cons, NMFS used a concentration addition analysis to assess whether or not the criteria exposed to multiple compounds under the proposed criteria pose a greater risk to listed species considered in this opinion than does exposure to individual compounds. Here the purpose was to predict the cumulative toxicity that is expected for the mixture. For example, if the assessment effect is 50 percent mortality (*i.e.* the assessment effect concentration, the denominator, is LC_{50}), a result of 1 predicts that the mixture would produce 50 percent mortality. A result of < 1 predicts that, based on additivity, the mortality would be less than 50 percent. A result of > 1 predicts more that 50 percent mortality. The concentration addition analysis is based on an assumption of a similar mechanism of action for each set of compounds, *e.g.*, metals or organics (includes ammonia even though it does not have a C-H bond). For the freshwater acute analysis NMFS used the LC_{50} data from Table 2.6.5.1.2. For the freshwater chronic, saltwater acute and chronic analysis, NMFS used the geometric mean of the respective data sets (Tables 2.6.2.1.5 through 2.6.3.13.2), or the BE if no chronic toxicity data (*i.e.*, ACR value) were available. The NMFS used the following equation in this analysis:

$$\sum_{i=1}^{n} \frac{C_i}{EC_{xi}}$$

where n = the number of compounds in the mixture, C_i = assessment exposure concentration (criterion) and EC_{xi} = assessment effects concentration (geometric mean of the criterion-specific toxicity data set).

Assumptions

This analysis is specific to the compounds listed in Table 1.1, assumes that the listed species considered in this opinion are exposed to the compounds in combination that follow concentration addition. For freshwater and saltwater metals, this scenario is highly likely based on the information in section 2.5.2.1 on compounds discharged in MS4 and NPDES permits (12 of 12 metals). For freshwater and saltwater organic compounds, this scenario is less likely based on the information in the environmental baseline (Section 2.5.2.1) on compounds discharged in MS4 and NPDES permits (1 of 8 organic compounds in freshwater and 1 of 4 in saltwater). The results of NMFS' concentration addition analysis are provided in Table 2.6.4.1.

Table 2.6.4.1 Results of the concentration addition analysis.

Metal Compounds	**Criteria**	**Mixture Prediction**
Al, As, Cd, Cr (III), Cr (VI), Cu, Pb, Ni, Se, Ag, Tributyltin, Zn	Freshwater acute	1.2
Al, As, Cd, Cr (III), Cr (VI), Cu, Pb, Ni, Se, Ag, Tributyltin, Zn	Freshwater chronic	4.7
As, Cd, Cr (VI), Cu, Pb, Ni, Se, Ag, Tributyltin, Zn	Saltwater acute	0.4
As, Cd, Cr (VI), Cu, Pb, Ni, Se, Tributyltin, Zn	Saltwater chronic	1.4
Organic Compounds	**Criteria**	**Mixture Prediction**
Ammonia, Lindane, Dieldrin, Endosulfan-alpha, Endosulfan-beta, Endrin, Heptachlor expoxide, Pentachlorophenol	Freshwater acute	1.3
Ammonia, Dieldrin, Endosulfan-alpha, Endosulfan-beta, Endrin, Heptachlor expoxide, Pentachlorophenol	Freshwater chronic	0.8
Endosulfan-alpha, Endosulfan-beta, Heptachlor expoxide	Saltwater acute	0.2
Endosulfan-alpha, Endosulfan-beta, Heptachlor expoxide, Pentachlorophenol	Saltwater chronic	0.001

Summary: The results of the concentration addition analysis infer that for acute and chronic freshwater criteria for metal compounds, acute freshwater criteria for organic compounds, and chronic saltwater criteria for metal compounds, fish exposed to multiple compounds, versus a single compound exposure, are likely to suffer toxicity greater than the assessment effects (*e.g.*, 50 percent mortality) such as mortality, reduced growth, impairment of essential behaviors related to successful rearing and migration, cellular trauma, physiological trauma, and reproductive failure. For example, the toxicity of a mixture at the freshwater acute criterion is predicted to be equivalent to an exposure to a single compound at 1.2 times the compounds' LC_{50} (*e.g.*, an exposure to cadmium at 2.4 µg/L compared to the proposed criterion concentration of 2 µg/L). The mixture toxicity will be greater than 50 percent mortality, but quantifying this prediction is dependent upon knowing the concentration-response curve. On the other hand, the results of the concentration addition analysis infer that for chronic freshwater criteria for organic compounds, acute saltwater criteria for metal compounds, and for acute and chronic saltwater criteria for organic compounds, fish exposed to multiple compounds, versus a single compound criterion exposure, are unlikely to suffer toxicity greater than the assessment effect concentrations.

2.6.5 Direct Mortality Population Modeling

To determine if population productivity would be at risk due to direct mortality resulting from either acute or chronic exposures to the criterion concentrations of the chemicals of concern, a series of modeling applications was undertaken. These assessed whether juvenile salmon during their freshwater residence encountering the established criterion concentrations would be impacted, and if those changes would be sufficient to produce a change in the population growth rate, *i.e.*, lambda (λ). Model Run I examined the potential lethal and sublethal effects of ammonia, cadmium and copper on salmon productivity. These compounds were chosen because they are more data rich for specific life stages of salmonids and could potentially parameterize population models assessing direct mortality and somatic growth. Specific details regarding model design and parameterization are described in detail in Appendix 3. Model Run II assessed direct mortality impacts on population productivity resulting from exposure to the acute criteria for compounds with limited data.

Model Run I uses the direct mortality population model to assess the impact of the acute and chronic freshwater criteria on population productivity using a taxa- and life stage-specific subset of the acute and chronic toxicity data for ammonia, copper, and cadmium, and uses data-specific calculated dose-response slopes for the toxicity model runs (Appendix 3). This included direct mortality from either acute or chronic exposures. The model applied a mortality factor to first-year survival of the respective life-history models to assess changes in λ.

Model Run II uses the direct mortality population model (Appendix 3) to assess the impact of the acute freshwater criteria on population productivity using the acute toxicity data (LC_{50}), and a default dose-response slope. To assess the impact of the acute freshwater criteria on population productivity, we used the direct mortality population models. To do this, the dose-response slope for each LC_{50} toxicity test is needed. The BE does not provide any dose-response information for the data used in the analysis. Many of toxicity studies we reviewed either did not report the slope or did not provide the information required to calculate the dose-response curve. Since the direct mortality population model requires an LC_{50} slope, we used a default slope (probit slope of 4.5 converted to a sigmoid slope of 3.6) as recommended by EPA:

> In the event that dose response information is not available to estimate a slope, a default slope assumption of 4.5 (lower and upper bounds of 2 to 9) (Urban and Cook 1986 as cited in EPA 2007) is used.

In the analysis for Model Run I and Model Run II we assess the potential for effects associated with chemical exposure during subyearling freshwater rearing on Pacific salmon and steelhead populations using quantitative methods; a direct mortality model linked to a life history population model and a somatic growth model linked to the life history population model. Both methods predict changes in the modeled population's intrinsic rate of growth, *i.e.*, λ. General life-history strategies were constructed and analyzed for coho salmon, sockeye salmon and ocean-type and stream-type Chinook salmon. The model assesses direct mortality to subyearling salmon and its impact on population productivity. Data was reviewed in an attempt to paramaterize a somatic growth population model that explicitly links impairments in the somatic growth of individual subyearling salmon to the productivity of salmon populations.

Available data was insufficent to parameterize the somatic growth model. Both models address impacts on first-year survival, and the results are incorporated into one of four life-history strategies in the model to quantify changes in population productivity (for a detailed description, see Appendix 3).

Primary differences between the four modeled life-history strategies are life span of the female, time to reproductive maturity, the number and relative contribution of the reproductive age classes and general demographic rates (Appendix 3). The models depict general populations representing each life-history strategy and were constructed based upon literature data described in Appendix 3. Specific populations were not modeled due to the difficulty in finding sufficient demographic data for single populations. Due to similarities in life-history strategies, the ocean-type Chinook model was used to estimate impacts on chum salmon and the stream-type Chinook model to estimate impacts on steelhead.

The endpoint used to assess population-level impacts for the direct mortality population model was the percent change in the intrinsic population growth rate (lambda, λ) resulting from chemical exposure. Change in λ is an accepted population parameter often used in evaluating population productivity, status, and viability. The NMFS uses changes in λ when estimating the status of species, conducting risk and viability assessments, developing ESA recovery plans, composing opinions, and communicating with other Federal, state and local agencies (McClure *et al.* 2003 as cited in Appendix 3). While values of λ <1.0 indicate a declining population, in cases when an exposure causes the population growth rate to decrease more than natural variability, a loss of productivity will result even if lambda remains above 1.0. Decreases in response to chemical exposures can be a cause for concern since the impact could make a population more susceptible to decline (*i.e.*, λ dropping below 1.0) due to impacts from other stressors.

2.6.5.1 Direct Mortality Population Model Description

A direct mortality population model was constructed that estimated the population-level impacts of first-year mortality resulting from exposure to the criterion concentrations of aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc (Model Run II). For Model Run II, impacts of first-year mortality resulting from exposure to the criterion concentrations of ammonia, copper, and cadmium over various time frames and life stages of data. These models excluded sublethal and indirect effects of the chemical exposures and focused on the population-level outcomes resulting from an annual exposure of young-of-the-year to a chemical at the criterion concentrations. Scenarios were chosen to represent both the acute and chronic criteria. This was done by parameterizing the model with toxicity data (LC_{50}s) derived from short term (<96 hrs) and long term (>28 days, based on the available data, see Table A3 in Appendix 3) experiments. The lethal impact was implemented as a change in first-year survival for each of the salmon life-history strategies. In order to understand the relative impacts of a short-term exposure of a single chemical on exposed vs. unexposed fish, we used parameters for an idealized control population that exhibits an increasing population growth rate. Four life-history strategies were modeled: ocean-type and stream-type Chinook salmon, coho salmon and sockeye salmon. The details for

each general population model are provided in Appendix 3. Due to similarities in life-history strategies, the ocean-type Chinook model was used to estimate impacts on chum and the stream-type Chinook model to estimate impacts on steelhead.

Population model output consists of the percent change in λ from the unexposed control populations derived from the mean of one thousand calculations each of the unexposed control and the chemical exposed populations. The percent change in lambda (with standard deviation), representing alterations to the population productivity, was selected as the primary model output for reasons outlined previously. The percent change in lambda is considered different from the control when the difference is greater than the percent of one standard deviation of the control λ.

Model Run I: Direct mortality, somatic growth, and population modeling— ammonia, cadmium, and copper.

Model Toxicity Scenario Parameterization

Ammonia (acute criterion = 5.6 mg/L; chronic criterion = 1.7 mg/L): The documents identified by the first round of literature review applying to acute toxicity of ammonia to salmonids were further reviewed for data appropriate to parameterize the direct mortality population model. Data needed to conform to 96-hr LC50 values for subyearling salmonids (free-swimming, 1-4g fish preferred, but did include data on fish of less than 10 g when that was all that was available). The range of values identified for Chinook salmon, coho salmon, rainbow trout and cutthroat trout and are shown below in the units of mg NH_3-N/L, as N (total ammonia-nitrogen). All values were normalized to a pH of 8 using an un-ionized ammonia computer worksheet available from the American Fisheries Society, as cited in Appendix 3. Following the practice in the ammonia Ambient Water Quality Criteria documents (1999, 2009, all as cited in Appendix 1), the fish LC50 values were not normalized for temperature. The normalized species mean values were 26.8, 15.1, 26.2 and 29.4 mg NH_3-N/L for Chinook salmon, coho salmon, rainbow trout and cutthroat trout, respectively (Servizi and Gordon 1990; Buckley 1978; Thurston and Russo 1983; Thurston *et al.,* 1981, Table A3, all as cited in Appendix 3). The genus geometric mean from these data was 23.6 mg NH_3-N/L. A sigmoid dose-response slope was calculated as 6.4 (Broderius and Smith 1979; Buckley 1978, as cited in Appendix 3). Both the genus geometric means and minimum species mean values were used to parameterize the model as discussed above. To assess the chronic criterion, a chronic study was found that exposed cutthroat trout to ammonia for 29 days and reported an LC50 of 21.3 mg NH_3-N/L (Thurston *et al.,* 1978, as cited in Appendix 3). No slope was identified, so the 96-hr slope was used in the model.

Documents investigating the effects of ammonia on growth of fish were reviewed for data appropriate as input to the somatic growth model. No studies were found that could provide the appropriate data. Most studies on exposure of juvenile salmonids to ammonia found that any effects on growth or food intake were temporary and compensation occurred before the end of the exposure period (Lang *et al.*, 1987, Linton *et al.*, 1998, Beamish and Tandler 1990, Larmoyeux and Piper 1973 as cited in Appendix 3). Other studies have shown effects on growth, but exposure occurred over early developmental stages and also produced developmental delays and abnormalities, so differences in size may not have been attributable to direct impacts on

metabolism or growth (Brinkman *et al.*. 2009 as cited in Appendix 3). From a 90-day exposure (Brinkman *et al.*. 2009 as cited in Appendix 3) calculated an EC_{20} that includes hatch effects, delayed swimup, and sac-fry growth of 5.56 mg NH_3-N/L normalized to pH 8. In addition, Lazorchak and Smith (2007 as cited in Appendix 1) reported decreases in growth of rainbow trout (size range <0.2 g) after a 7 day exposure to ammonium chloride, but at concentrations that overlapped with those inducing mortality in the test population inhibition concentration (IC) IC_{25} ranged from 104-210 mg/L ammonium chloride and LC_{50} ranged from 163-271 mg/L ammonium chloride). Moreover, the study organisms used by Lazorchak and Smith (2007 as cited in Appendix 3) were too young to fit within the life stage criteria established for this modeling exercise. In addition, pH was not reported in this study, so accurate normalization was not possible. Broderius and Smith (1979 as cited in Appendix 3) also exposed small rainbow trout (0.18 g) to ammonia over a 30-day period. Significant reductions in growth were seen at 0.32 mg NH_3-N/L, but survival was 70% of that observed in the controls (60%), so the quality and usefulness of this data is suspect. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects if studies where significant mortality occurred were included. Since data for the appropriate life stages or time frames were unavailable, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium (acute criterion = 2.0 µg/L; chronic criterion = 0.25 µg/L): Studies identified by the first round of literature review as having data on acute and chronic toxicity for the freshwater phase of salmonids were examined to gather data for parameterizing the population models. All data were hardness adjusted to 100 mg $CaCO_3$/L and reported as dissolved cadmium in µg/L using the hardness equations found in Mebane (2006 as cited in Appendix 3). The acute toxicity focused on 96-h mortality data for swimup fry, parr and subyearling smolt. Species mean values (geometric means of LC_{50} values) were calculated for salmonid fishes, and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species means at 4.53 µg/L (Appendix 3, Table A3). Sigmoid slopes were calculated when dose-response data were available. The resulting geometric mean of the slopes was 6.4 and the range was 4.7-7.8 (Besser *et al.* 2007, Finlayson and Verrue 1982, Davies *et al.* 1993 as cited in Appendix 3). Besser *et al.*. (2007 as cited in Appendix 1) estimated a 28-day LC_{50} for rainbow trout of 5.5 µg/L (Appendix 1, Table A3). The normalized LC_{50} value of 5.36 µg/L and the acute slope of 6.4 were used to parameterize the chronic criteria scenario of the mortality model.

Chronic cadmium studies were examined for applicable input data for the somatic growth model. Studies on the effects of cadmium on the growth of subyearling salmonids supported the statement by Mebane (2006 as cited in Appendix 3) that growth is seldom a sensitive endpoint for cadmium. At concentrations that produced changes in somatic growth, increased mortality was also observed in most studies (Mebane *et al.*. 2008, Brinkman and Hansen 2007, Hansen *et al.*, 2002b). In 24- and 30-day exposures of Atlantic salmon (*Salmo salar*), a reduction in size was seen after alevins were exposed to 6.75-21.8 µg Cd/L but these concentrations also produced 80-90% mortality (Rombough and Garside 1982, Peterson *et al.*, 1983). Bull trout (*Salvelinus confluentus*) fry (0.2 g) exposed to 1.57 µg Cd/L for 55 days (hardness adjusted to 100 mg $CaCO_3$/L) showed a 28% reduction in growth at this single time point, along with a 37% reduction in survival (Hansen *et al.* 2002b as cited in Appendix 3). No dose response curve for

growth was generated by the study, so these data could not be used for extrapolation to other concentrations.

Brinkman and Hansen (2007 as cited in Appendix 3) exposed brown trout fry (*Salmo* trutta) to cadmium for 30 days under different water chemistries and calculated a range of IC_{20}s from 1.7-4.8 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) for reduced growth in the surviving individuals. Mortality chronic values for the same tests ranged from 2.04 to 4.79 µg Cd/L. They also calculated LC_{50} values for the first 96 h of the exposures and these ranged from 3.27 to 6.75 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L). Possible size-selective mortality or growth compensation due to decreased density were not addressed in the study design. Rainbow trout fry exposed to cadmium for 28 days exhibited increased mortality and dry weight at concentrations above a calculated NOEC of 1.3 µg Cd/L (Besser *et al.* 2007 as cited in Appendix 3). This may be attributed to size-selective mortality or an increase in somatic growth. One rainbow trout early-life-stage exposure lasting 62 days determined an EC_{10} for growth of 0.31 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) without the increased mortality (Mebane *et al.* 2008 as cited in Appendix 3). Changes in growth at these life stages (embryos and alevins) are not compatible with the somatic growth model that assesses changes in free-swimming, feeding fry during the linear portion of their growth phase, and could not be used to parameterize the model. Similarly, brook trout (*Salvelinus fontinalis*) exposed to 0.36 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) for 30 days showed reduced prey capture efficiencies and differences in prey selection in artificial stream channels (Riddell *et al.* 2005 as cited in Appendix 3), which may link to changes in somatic growth, but this link could not be translated into appropriate input parameters for the current growth model.

Copper (acute criterion = 13 µg/L; chronic criterion = 9 µg/L)*:* Studies having data on acute and chronic toxicity for the freshwater phase of salmonids were examined to gather data needed to establish values for several parameters of the population models. All data was hardness adjusted to 100 mg $CaCO_3$/L using the acute and chronic hardness equations for copper (EPA 2002 as cited in Appendix 3). For studies with non-laboratory water that reported total instead of dissolved copper, total copper was adjusted by 80% to estimate the dissolved portion of copper in µg/L. The acute toxicity focused on 96-h mortality data for swim-up fry, parr and subyearling fish. Species mean values (geometric means of LC_{50} values) were calculated (Appendix 1, Table A3) and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species. For direct mortality, the genus mean LC_{50} was 86.8 µg/L with species means ranging from 48.3-190.6 µg/L, while for chronic toxicity (exposures of at least 30 days) the genus mean value was 98.9 µg/L with a range of 73.9-132.2 µg/L. Sigmoid slopes were calculated when dose-response data were available (Appendix 3, Table A3). The resulting geometric means (with ranges) of the slopes were 5.2 (4.1-7.6) for the 96-hr exposures and 4.2 (3.1-5.4) for the longer term mortality studies.

Growth studies on fry over 0.2 grams and under 6 grams produced EC_{50} values ranging from 20.33 µg/L to 112.43 µg/L (all values hardness adjusted, Appendix 3, Table A4). Exposures lasted 15 - 98 days. NOEC values ranged from 5.83 - 113.82µg/L. Mortality was often observed in these studies and ranged from none reported to well over 50% at similar concentrations to those that produced growth effects (Appendix 1, Table A4). For example, Besser *et al.*. (2005 as cited in Appendix 3) reported the lowest growth EC_{50} of 20.33µg/L for 0.2 g fry after a 30 day

exposure, but also reported a 30-day LC_{50} of 16.83µg/L with a slope of 5.4 (Appendix 3, Table A4). Therefore, similar to the results with cadmium exposures occurring to subyearling salmonids between 1 and 6g, growth effects often were confounded by mortality since most of the growth studies reported mortality assessment values (LC_{50}s, chronic values, NOECs) that overlapped with or were less than the growth assessment values (EC_{50}s, NOECs; Appendix 1, Table A4). Hansen *et al.*(2002c as cited in Appendix 3) used the IC_{20} as an endpoint for comparison since concentrations producing over 20% growth inhibition were often accompanied by significant mortality. Many other growth studies found in the literature search were excluded for reasons such as using too few exposure concentrations, using exposures beginning before swim-up (usually just after fertilization), or reporting no effect on growth for the concentrations tested. As mentioned above, in the remaining studies concentrations that produced effects on growth often also showed significant decreases in survival. For example, Mudge *et al.*. (1993 as cited in Appendix 3) reported that, for three of their five tests in coho, mortality was more sensitive than growth (Appendix 3, Table A4). Nonetheless, some limited scenarios were run in the somatic growth model that looked at whether growth alone would be affected by exposures at the chronic criteria value for copper. The time-to-effect and time-to-recovery values used for copper were both 0.5 days.

Model Output

Ammonia: Using the genus geometric mean LC_{50} and dose-response slope, with 100% of the population exposed to the criteria concentrations, the direct mortality population model output showed 0% mortality to subyearlings and a zero percent change in the population growth rate (lambda) for all four life-history models (Table 2.6.5.1.47). The lowest species mean value in the *Oncorhynchus* range was also tested at 15.1 mg NH_3-N/L, and resulted in zero percent mortality and zero percent change in λ. When the chronic criterion was assessed with a 29-d exposure, the direct mortality population model predicted no mortality or change in λ.

Studies on chronic exposures of juvenile salmonids to ammonia reported no or very little effects on somatic growth, but these were accompanied by mortality. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium: Direct mortality population model runs were conducted using exposures to the criteria concentrations and the genus mean value calculated for *Oncorhynchus* (Table 2.6.5.1.1). This value produced 1 percent mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC_{50} and slope values as opposed to the minimum end of the range for species mean values (Table 2.6.5.1.1). Only when the minimum species mean value and the minimum slope were used did mortality rise to a level that produced changes in lambda that were greater than the standard deviation of the control models (Table 2.6.5.1.47). Changes in population growth rates for the stream-type Chinook and coho salmon were larger than one standard deviation from the control models. An estimated 28-day exposure to the chronic criterion produced no mortality or change in lambda.

Studies on chronic cadmium toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model was not run for cadmium.

Copper: Direct mortality population model runs were conducted using exposures to the criteria concentrations and both the acute and chronic parameters calculated for *Oncorhynchus* (Table 2.6.5.1). The acute LC_{50} and slope produced 0% mortality and no changes in the population growth rate for any of the four life history population models. The chronic LC_{50} and slope produced 0 percent mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC_{50} and slope values as opposed to the minimum end of the range for species mean values, but no mortality was projected (Table 2.6.5.1.1).

Studies on copper toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. In spite of this, some growth model scenarios were run. When the maximum exposure period was used for the chronic criteria value in the growth model (140, 164 or 184 days depending on the life history), with an EC_{50} of 20.33, slope of 2.7 (Besser 2005 as cited in Appendix 3) and the chronic criterion value of 9 µg/L, the percent change in λ ranged from -1 to -4 percent (depending on life history). None of these reductions exceeded the control standard deviations. A 30-day exposure produced no decline in population growth rates. When a 30-day exposure for direct mortality was modeled using the minimum species values with a LC_{50} of 73.9 µg/L and a slope of 4.2, the chronic criterion (9 µg /L) produced no change in λ for the four life history models.

Table 2.6.5.1.1 Direct mortality population model scenarios for ammonia, cadmium and copper criteria. Standard scenarios used the genus mean values for the criteria. Since no effect resulted, the minimum species mean values were assessed. The numbers in parentheses are the natural variability in λ. Bold indicates a percent change in lambda greater than one standard deviation from the baseline population model. The direct mortality population model scenarios for ammonia, cadmium, and copper do not take into account sublethal responses, indirect effects, mixture toxicity, and baseline stressors.

		Mortality input parameters			**Output**	**Percent change in lambda**			
Chemical	Test length	LC_{50} (mg/L)	Sigmoid slope	Criteria Conc.	Percent mortality	Chinook ocean-type	Chinook stream-type	Sockeye	Coho
Ammonia	96-hr	23.6[1]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	96-hr	15.1[2]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	29-d	21.3	6.4[3]	1.7	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Cadmium	96-hr	4.53[1]	6.4[1]	2.0	1	0(13)	0(4)	0(8)	0(7)
Cadmium	96-hr	4.53[1]	4.7[2]	2.0	2	-1(13)	-1(4)	-1(8)	-1(7)
Cadmium	96-hr	2.67[2]	6.4[1]	2.0	14	-4(12)	-3(4)	-3(8)	-5(7)
Cadmium	96-hr	2.67[2]	4.7[2]	2.0	20	-7(12)	**-5(4)**	-5(8)	-7(7)
Cadmium	28-d	5.36[1]	6.4[3]	0.25	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Copper	96-hr	86.8[1]	5.2[1]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	96-hr	48.3[2]	4.1[2]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	98.9[1]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	73.9[2]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)

[1]Genus geometric mean for *Oncorhynchus* values
[2]Minimum species mean value from the range of *Oncorhynchus* values.
[3]Slope for chronic exposures not identified, used genus mean slope from 96-hr exposures.

Summary: The only scenarios producing direct mortality sufficient to decrease the population growth rates or productivity were those using the lowest species mean values for cadmium. The other scenarios assessing the direct mortality from exposure to the suggested criteria values for ammonia, cadmium and copper did not result in significant changes in population productivity greater than one standard deviation from baseline population model.

Model Run II: Acute toxicity exposure-response analysis and direct mortality population modeling—aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc.

The statistical inputs for the Model Run II are displayed in Table 2.6.5.1.2. Tables 2.6.5.1.3 through 2.6.5.1.243 provide the output of the direct mortality population modeling on the percent mortality and changes in λ for each freshwater compound and for each of the six

salmonid fishes life history strategies. The NMFS only used LC_{50} toxicity data for free-swimming juvenile life stages for the direct mortality population modeling. Each table provides information on the chemical, concentration (criterion), LC_{50}, the geometric mean and the minimum species mean value of the 96-hour LC_{50} for the respective acute toxicity data set; the default dose-response sigmoid slope; species; percent mortality resulting from the LC_{50} and slope; the percent of the population exposed; the percent change in λ and its standard deviation (impacted) measured against the baseline population model; the mean value of lambda and its standard deviation, the first-year survival rate (S1); and the significant change, which is the percent change in lambda that exceeds one standard deviation of the baseline model. The first table is for each life history type and provides the results of the model run based on the geometric mean of the 96-hour LC_{50}. The second table is for each life history type and provides the results of the model run based on the minimum species mean value of the 96-hour LC_{50}. For details regarding the model output information in Tables 2.6.5.3 through 2.6.5.1.243, refer to Appendix 3.

The direct mortality population model scenarios for aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc do not take into account sublethal responses, indirect effects, mixture toxicity, and baseline stressors.

Table 2.6.5.1.2 Freshwater toxicity data statistics used as inputs for the Model Run II.

Compound	Acute Criterion	Acute Data (Geometric Mean)	Acute Data Used in the Direct Mortality Population Model (the geometric mean and the minimum species mean values)
Aluminum	750	2247	2671—445
Ammonia	5.6	32	32—7.3
Arsenic	340	16698	34269—10
Lindane	0.95	22.7	19.7—1
Cadmium	2	9.1	9—1.16
Chromium (III)	570	9825	9825—7762
Chromium (VI)	16	74908	74908—12079
Copper	13	96	96—5.7
Dieldrin	0.24	27	24—0.56
Endosulfan-alpha	0.22	0.66	0.66—0.17
Endosulfan-beta	0.22	0.66	0.66—0.17
Endrin	0.086	1.1	0.6—0.089
Heptachlor Epoxide	0.52	13.6	13.6—6.7
Lead	65	14675	17042—320
Nickel	470	18793	17663—588
Pentachlorophenol	19	86.9	86.1—10
Selenium	190	2850	4268—0.4
Silver	3.2	63	63—1.28
Tributyltin	0.46	3.2	2.6—0.21
Zinc	120	1190	1188—238

Aluminum

Table 2.6.5.1.3 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	12.9
LC50	2671	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.62e-003	5.56e-003
% Mortality	1	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.4 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-43
Concentration	750	% chg l std	-	7.1
LC50	445	lambda mean	1.09	0.62
LC50 slope	3.6	lambda std	0.10	0.05
species	chinook, ot	S1	5.62e-003	7.47e-004
% Mortality	87	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.5 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	4.3
LC50	2671	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	6.37e-002
% Mortality	1	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.6 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-39
Concentration	750	% chg l std	-	2.6
LC50	445	lambda mean	1.00	0.61
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.44e-002	8.53e-003
% Mortality	87	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.7 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	7.9
LC50	2671	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.55e-002
% Mortality	1	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.8 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-38
Concentration	750	% chg l std	-	4.8
LC50	445	lambda mean	1.01	0.63
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.56e-002	3.41e-003
% Mortality	87	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.9 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	7.5
LC50	2671	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.93e-002
% Mortality	1	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.10 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-49
Concentration	750	% chg l std	-	3.8
LC50	445	lambda mean	1.03	0.52
LC50 slope	3.6	lambda std	0.05	0.03
species	coho	S1	2.97e-002	3.93e-003
% Mortality	87	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.11 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	4.3
LC50	2671	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	6.37e-002
% Mortality	1	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.12 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-39
Concentration	750	% chg l std	-	2.6
LC50	445	lambda mean	1.00	0.61
LC50 slope	3.6	lambda std	0.03	0.02
species	steelhead	S1	6.44e-002	8.53e-003
% Mortality	87	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.13 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	12.9
LC50	2671	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.58e-003
% Mortality	1	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.14 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-43
Concentration	750	% chg l std	-	7.1
LC50	445	lambda mean	1.09	0.62
LC50 slope	3.6	lambda std	0.10	0.05
species	chum	S1	5.62e-003	7.47e-004
% Mortality	87	Significant change		9.1
Percent Exposed	100	[]		

Ammonia

Table 2.6.5.1.15 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg l std	-	12.9
LC50	32	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.62e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.16 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-9
Concentration	5.6	% chg l std	-	11.7
LC50	7.3	lambda mean	1.09	0.99
LC50 slope	3.6	lambda std	0.10	0.09
species	chinook, ot	S1	5.64e-003	4.06e-003
% Mortality	28	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.17 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg l std	-	4.4
LC50	32	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	6.42e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.18 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-8
Concentration	5.6	% chg l std	-	4.1
LC50	7.3	lambda mean	1.00	0.92
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	4.65e-002
% Mortality	28	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.19 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg l std	-	8.0
LC50	32	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.20 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-7
Concentration	5.6	% chg l std	-	7.4
LC50	7.3	lambda mean	1.01	0.93
LC50 slope	3.6	lambda std	0.06	0.05
species	sockeye	S1	2.57e-002	1.86e-002
% Mortality	28	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.21 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg l std	-	7.5
LC50	32	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.22 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-10
Concentration	5.6	% chg l std	-	6.7
LC50	7.3	lambda mean	1.03	0.92
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.14e-002
% Mortality	28	Significant change		5.2
Percent Exposed	100	[]		

Table 2.6.5.1.23 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg l std	-	4.4
LC50	32	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	6.42e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.24 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-8
Concentration	5.6	% chg l std	-	4.1
LC50	7.3	lambda mean	1.00	0.92
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	4.65e-002
% Mortality	28	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.25 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg l std	-	12.9
LC50	32	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.62e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.26 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-9
Concentration	5.6	% chg l std	-	11.7
LC50	7.3	lambda mean	1.09	0.99
LC50 slope	3.6	lambda std	0.10	0.09
species	chum	S1	5.64e-003	4.06e-003
% Mortality	28	Significant change		9.1
Percent Exposed	100	[]		

Arsenic

Table 2.6.5.1.27 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg l std	-	12.8
LC50	34269	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.62e-003	5.62e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.28 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-95
Concentration	340	% chg l std	-	0.6
LC50	10	lambda mean	1.09	0.05
LC50 slope	3.6	lambda std	0.10	0.00
species	chinook, ot	S1	5.63e-003	1.73e-008
% Mortality	100	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.29 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg l std	-	4.4
LC50	34269	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.30 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-95
Concentration	340	% chg l std	-	0.2
LC50	10	lambda mean	1.00	0.05
LC50 slope	3.6	lambda std	0.03	0.00
species	chinook, st	S1	6.43e-002	1.97e-007
% Mortality	100	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.31 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg l std	-	7.9
LC50	34269	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.57e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.32 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-94
Concentration	340	% chg l std	-	0.4
LC50	10	lambda mean	1.01	0.06
LC50 slope	3.6	lambda std	0.06	0.00
species	sockeye	S1	2.57e-002	7.86e-008
% Mortality	100	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.33 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg l std	-	7.5
LC50	34269	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.2
Percent Exposed	100	[]		

Table 2.6.5.1.34 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-99
Concentration	340	% chg l std	-	0.1
LC50	10	lambda mean	1.03	0.01
LC50 slope	3.6	lambda std	0.05	0.00
species	coho	S1	2.97e-002	9.09e-008
% Mortality	100	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.35 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg l std	-	4.4
LC50	34269	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.36 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-95
Concentration	340	% chg l std	-	0.2
LC50	10	lambda mean	1.00	0.05
LC50 slope	3.6	lambda std	0.03	0.00
species	steelhead	S1	6.43e-002	1.97e-007
% Mortality	100	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.37 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg l std	-	13.0
LC50	34269	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.38 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-95
Concentration	340	% chg l std	-	0.6
LC50	10	lambda mean	1.09	0.05
LC50 slope	3.6	lambda std	0.10	0.00
species	chum	S1	5.63e-003	1.73e-008
% Mortality	100	Significant change		9.2
Percent Exposed	100	[]		

Lindane

Table 2.6.5.1.39 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg l std	-	12.9
LC50	19.7	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.64e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.40 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-16
Concentration	0.95	% chg l std	-	10.8
LC50	1	lambda mean	1.09	0.91
LC50 slope	3.6	lambda std	0.10	0.08
species	chinook, ot	S1	5.61e-003	3.07e-003
% Mortality	45	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.41 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg l std	-	4.4
LC50	19.7	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.42 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-14
Concentration	0.95	% chg l std	-	3.8
LC50	1	lambda mean	1.00	0.86
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	3.51e-002
% Mortality	45	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.43 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg l std	-	7.9
LC50	19.7	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.44 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-13
Concentration	0.95	% chg l std	-	6.9
LC50	1	lambda mean	1.01	0.87
LC50 slope	3.6	lambda std	0.06	0.05
species	sockeye	S1	2.57e-002	1.41e-002
% Mortality	45	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.45 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg l std	-	7.6
LC50	19.7	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.06	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.4
Percent Exposed	100	[]		

Table 2.6.5.1.46 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-18
Concentration	0.95	% chg l std	-	6.1
LC50	1	lambda mean	1.03	0.84
LC50 slope	3.6	lambda std	0.05	0.04
species	coho	S1	2.97e-002	1.62e-002
% Mortality	45	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.47 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg l std	-	4.4
LC50	19.7	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.48 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-14
Concentration	0.95	% chg l std	-	3.8
LC50	1	lambda mean	1.00	0.86
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	3.51e-002
% Mortality	45	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.49 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg l std	-	12.9
LC50	19.7	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.50 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-16
Concentration	0.95	% chg l std	-	10.8
LC50	1	lambda mean	1.09	0.91
LC50 slope	3.6	lambda std	0.10	0.08
species	chum	S1	5.61e-003	3.07e-003
% Mortality	45	Significant change		9.2
Percent Exposed	100	[]		

Cadmium

Table 2.6.5.1.51 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	12.9
LC50	10.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.52 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-45
Concentration	2	% chg l std	-	7.0
LC50	1.16	lambda mean	1.09	0.60
LC50 slope	3.6	lambda std	0.10	0.05
species	chinook, ot	S1	5.62e-003	6.94e-004
% Mortality	88	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.53 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	4.3
LC50	10.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	6.42e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.54 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-40
Concentration	2	% chg l std	-	2.6
LC50	1.16	lambda mean	1.00	0.60
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	7.94e-003
% Mortality	88	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.55 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	7.9
LC50	10.6	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.56e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.56 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-39
Concentration	2	% chg l std	-	4.8
LC50	1.16	lambda mean	1.01	0.62
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.57e-002	3.17e-003
% Mortality	88	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.57 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	7.5
LC50	10.6	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.58 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-50
Concentration	2	% chg l std	-	3.7
LC50	1.16	lambda mean	1.03	0.51
LC50 slope	3.6	lambda std	0.05	0.03
species	coho	S1	2.97e-002	3.66e-003
% Mortality	88	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.59 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	4.4
LC50	10.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.41e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.60 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-40
Concentration	2	% chg l std	-	2.5
LC50	1.16	lambda mean	1.00	0.60
LC50 slope	3.6	lambda std	0.03	0.02
species	steelhead	S1	6.43e-002	7.93e-003
% Mortality	88	Significant change		3.0
Percent Exposed	100	[]		

Table 2.6.5.1.61 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	12.8
LC50	10.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.61e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.62 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-45
Concentration	2	% chg l std	-	7.0
LC50	1.16	lambda mean	1.09	0.60
LC50 slope	3.6	lambda std	0.10	0.05
species	chum	S1	5.63e-003	6.94e-004
% Mortality	88	Significant change		9.2
Percent Exposed	100	[]		

Chromium (III)

Table 2.6.5.1.63 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	12.8
LC50	9825	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.64 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	12.8
LC50	7762	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.65e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.65 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	4.4
LC50	9825	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.66 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	4.4
LC50	7762	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.67 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	7.9
LC50	9825	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.68 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	8.0
LC50	7762	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.69 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	7.5
LC50	9825	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.70 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	7.5
LC50	7762	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.71 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	4.4
LC50	9825	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.72 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	4.4
LC50	7762	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.73 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	12.8
LC50	9825	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.74 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	12.9
LC50	7762	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.61e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Chromium (VI)

Table 2.6.5.1.75 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	12.8
LC50	74908	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.65e-003	5.64e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.76 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	12.8
LC50	12079	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.62e-003	5.62e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.77 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	4.4
LC50	74908	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.78 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	4.4
LC50	12079	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.79 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	8.0
LC50	74908	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.80 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	8.0
LC50	12079	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.81 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	7.5
LC50	74908	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.82 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	7.6
LC50	12079	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.06	0.06
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.4
Percent Exposed	100	[]		

Table 2.6.5.1.83 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	4.4
LC50	74908	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.84 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	4.4
LC50	12079	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.85 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	12.8
LC50	74908	lambda mean	1.09	1.09
LC50 slope	4.5	lambda std	0.10	0.10
species	chum	S1	5.65e-003	5.64e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.86 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	12.9
LC50	12079	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.64e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Copper

Table 2.6.5.1.87 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	12.9
LC50	96	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.88 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-57
Concentration	13	% chg l std	-	5.5
LC50	5.7	lambda mean	1.09	0.47
LC50 slope	3.6	lambda std	0.10	0.04
species	chinook, ot	S1	5.64e-003	2.75e-004
% Mortality	95	Significant change		9.3
Percent Exposed	100	[]		

Table 2.6.5.1.89 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	4.4
LC50	96	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.42e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.90 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-52
Concentration	13	% chg l std	-	2.0
LC50	5.7	lambda mean	1.00	0.48
LC50 slope	3.6	lambda std	0.03	0.01
species	chinook, st	S1	6.44e-002	3.14e-003
% Mortality	95	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.91 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	7.8
LC50	96	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.92 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-51
Concentration	13	% chg l std	-	3.7
LC50	5.7	lambda mean	1.01	0.50
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.57e-002	1.26e-003
% Mortality	95	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.93 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	7.5
LC50	96	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.94 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-63
Concentration	13	% chg l std	-	2.7
LC50	5.7	lambda mean	1.03	0.38
LC50 slope	3.6	lambda std	0.05	0.02
species	coho	S1	2.97e-002	1.45e-003
% Mortality	95	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.95 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	4.4
LC50	96	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.96 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-52
Concentration	13	% chg l std	-	2.0
LC50	5.7	lambda mean	1.00	0.48
LC50 slope	3.6	lambda std	0.03	0.01
species	steelhead	S1	6.43e-002	3.14e-003
% Mortality	95	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.97 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	13.0
LC50	96	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.98 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-57
Concentration	13	% chg l std	-	5.4
LC50	5.7	lambda mean	1.09	0.47
LC50 slope	3.6	lambda std	0.10	0.04
species	chum	S1	5.64e-003	2.75e-004
% Mortality	95	Significant change		9.1
Percent Exposed	100	[]		

Dieldrin

Table 2.6.5.1.99 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg l std	-	13.0
LC50	24	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.63e-003	5.65e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.100 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg l std	-	12.6
LC50	0.56	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.37e-003
% Mortality	5	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.101 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg l std	-	4.4
LC50	24	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.102 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg l std	-	4.3
LC50	0.56	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.14e-002
% Mortality	5	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.103 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg l std	-	8.0
LC50	24	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.104 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg l std	-	7.9
LC50	0.56	lambda mean	1.01	1.00
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.46e-002
% Mortality	5	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.105 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg l std	-	7.5
LC50	24	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.106 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-2
Concentration	0.24	% chg l std	-	7.4
LC50	0.56	lambda mean	1.03	1.01
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.83e-002
% Mortality	5	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.107 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg l std	-	4.4
LC50	24	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.108 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg l std	-	4.3
LC50	0.56	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.15e-002
% Mortality	5	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.109 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg l std	-	13.0
LC50	24	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.65e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.110 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg l std	-	12.7
LC50	0.56	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.38e-003
% Mortality	5	Significant change		9.1
Percent Exposed	100	[]		

Endosulfan-alpha

Table 2.6.5.1.111 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg l std	-	12.7
LC50	0.66	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.1
species	Chinook, ot	S1	5.63e-003	5.53E-03
% Mortality	2	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.112 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-30
Concentration	0.22	% chg l std	-	8.8
LC50	0.17	lambda mean	1.09	0.76
LC50 slope	3.6	lambda std	0.10	0.07
species	chinook, ot	S1	5.63e-003	1.60e-003
% Mortality	72	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.113 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg l std	-	4.4
LC50	0.66	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.31E-02
% Mortality	2	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.114 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-27
Concentration	0.22	% chg l std	-	3.2
LC50	0.17	lambda mean	1.00	0.73
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	1.82e-002
% Mortality	72	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.115 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg l std	-	7.9
LC50	0.66	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.58e-002	2.52E-02
% Mortality	2	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.116 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-26
Concentration	0.22	% chg l std	-	5.8
LC50	0.17	lambda mean	1.01	0.75
LC50 slope	3.6	lambda std	0.06	0.04
species	sockeye	S1	2.57e-002	7.26e-003
% Mortality	72	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.117 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg l std	-	7.4
LC50	0.66	lambda mean	1.03	1.02
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.91E-02
% Mortality	2	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.118 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-34
Concentration	0.22	% chg l std	-	4.9
LC50	0.17	lambda mean	1.03	0.68
LC50 slope	3.6	lambda std	0.05	0.04
species	coho	S1	2.97e-002	8.41e-003
% Mortality	72	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.119 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
oncentration	0.22	% chg l std	-	4.4
LC50	0.66	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.31E-02
% Mortality	2	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.120 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-27
Concentration	0.22	% chg l std	-	3.2
LC50	0.17	lambda mean	1.00	0.73
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	1.82e-002
% Mortality	72	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.121 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg l std	-	12.7
LC50	0.66	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.1
species	chum	S1	5.63e-003	5.53E-03
% Mortality	1	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.122 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-30
Concentration	0.22	% chg l std	-	8.8
LC50	0.17	lambda mean	1.09	0.76
LC50 slope	3.6	lambda std	0.10	0.07
species	chum	S1	5.65e-003	1.60e-003
% Mortality	72	Significant change		9.1
Percent Exposed	100	[]		

Endosulfan-beta

Table 2.6.5.1.123 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg l std	-	12.7
LC50	0.66	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.1
species	Chinook, ot	S1	5.63e-003	5.53E-03
% Mortality	2	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.124 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-30
Concentration	0.22	% chg l std	-	8.8
LC50	0.17	lambda mean	1.09	0.76
LC50 slope	3.6	lambda std	0.10	0.07
species	chinook, ot	S1	5.63e-003	1.60e-003
% Mortality	72	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.125 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg l std	-	4.4
LC50	0.66	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.31E-02
% Mortality	2	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.126 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-27
Concentration	0.22	% chg l std	-	3.2
LC50	0.17	lambda mean	1.00	0.73
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	1.82e-002
% Mortality	72	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.127 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg l std	-	7.9
LC50	0.66	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.58e-002	2.52E-02
% Mortality	2	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.128 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-26
Concentration	0.22	% chg l std	-	5.8
LC50	0.17	lambda mean	1.01	0.75
LC50 slope	3.6	lambda std	0.06	0.04
species	sockeye	S1	2.57e-002	7.26e-003
% Mortality	72	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.129 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg l std	-	7.4
LC50	0.66	lambda mean	1.03	1.02
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.91E-02
% Mortality	2	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.130 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-34
Concentration	0.22	% chg l std	-	4.9
LC50	0.17	lambda mean	1.03	0.68
LC50 slope	3.6	lambda std	0.05	0.04
species	coho	S1	2.97e-002	8.41e-003
% Mortality	72	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.131 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
oncentration	0.22	% chg l std	-	4.4
LC50	0.66	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.31E-02
% Mortality	2	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.132 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-27
Concentration	0.22	% chg l std	-	3.2
LC50	0.17	lambda mean	1.00	0.73
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	1.82e-002
% Mortality	72	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.133 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg l std	-	12.7
LC50	0.66	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.1
species	chum	S1	5.63e-003	5.53E-03
% Mortality	1	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.134 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-30
Concentration	0.22	% chg l std	-	8.8
LC50	0.17	lambda mean	1.09	0.76
LC50 slope	3.6	lambda std	0.10	0.07
species	chum	S1	5.65e-003	1.60e-003
% Mortality	72	Significant change		9.1
Percent Exposed	100	[]		

Endrin

Table 2.6.5.1.135 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	12.9
LC50	0.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.64e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.136 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-17
Concentration	0.086	% chg l std	-	10.7
LC50	0.089	lambda mean	1.09	0.91
LC50 slope	3.6	lambda std	0.10	0.08
species	chinook, ot	S1	5.64e-003	2.99e-003
% Mortality	47	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.137 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	4.4
LC50	0.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.138 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	4.4
LC50	0.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.139 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-14
Concentration	0.086	% chg l std	-	3.7
LC50	0.089	lambda mean	1.00	0.85
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	3.41e-002
% Mortality	47	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.140 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	8.0
LC50	0.6	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.58e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.141 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-14
Concentration	0.086	% chg l std	-	6.7
LC50	0.089	lambda mean	1.01	0.87
LC50 slope	3.6	lambda std	0.06	0.05
species	sockeye	S1	2.57e-002	1.36e-002
% Mortality	47	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.142 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	7.5
LC50	0.6	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.143 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-19
Concentration	0.086	% chg l std	-	6.1
LC50	0.089	lambda mean	1.03	0.83
LC50 slope	3.6	lambda std	0.05	0.04
species	coho	S1	2.96e-002	1.57e-002
% Mortality	47	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.144 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	4.4
LC50	0.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.145 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-14
Concentration	0.086	% chg l std	-	3.8
LC50	0.089	lambda mean	1.00	0.85
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	3.42e-002
% Mortality	47	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.146 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	12.9
LC50	0.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.64e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.147 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-17
Concentration	0.086	% chg l std	-	10.7
LC50	0.089	lambda mean	1.09	0.91
LC50 slope	3.6	lambda std	0.10	0.08
species	chum	S1	5.63e-003	2.99e-003
% Mortality	47	Significant change		9.1
Percent Exposed	100	[]		

Heptachlor Epoxide

Table 2.6.5.1.148 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	13.0
LC50	13.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.65e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.149 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	12.8
LC50	6.7	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.150 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	4.4
LC50	13.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.44e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.151 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	4.4
LC50	6.7	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.152 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	7.9
LC50	13.6	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.153 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	7.9
LC50	6.7	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.58e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.154 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	7.4
LC50	13.6	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.2
Percent Exposed	100	[]		

Table 2.6.5.1.155 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	7.5
LC50	6.7	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.2
Percent Exposed	100	[]		

Table 2.6.5.1.156 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	4.4
LC50	13.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.157 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	4.4
LC50	6.7	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.158 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	13.0
LC50	13.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.65e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.159 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	12.9
LC50	6.7	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Lead

Table 2.6.5.1.160 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	12.7
LC50	17042	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.161 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	12.9
LC50	320	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

2.6.5.1.162 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	4.4
LC50	17042	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

2.6.5.1.163 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	4.4
LC50	320	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.41e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.164 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	8.0
LC50	17042	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.56e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.165 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	8.0
LC50	320	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.55e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.166 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	7.5
LC50	17042	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.167 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	7.5
LC50	320	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.168 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	4.4
LC50	17042	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.169 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	4.4
LC50	320	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.41e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.170 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	12.7
LC50	17042	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.171 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	12.9
LC50	320	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.61e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Nickel

Table 2.6.5.1.172 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg l std	-	12.9
LC50	17663	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.62e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.173 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-10
Concentration	470	% chg l std	-	11.5
LC50	588	lambda mean	1.09	0.98
LC50 slope	3.6	lambda std	0.10	0.09
species	chinook, ot	S1	5.62e-003	3.92e-003
% Mortality	31	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.174 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg l std	-	4.4
LC50	17663	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.175 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-9
Concentration	470	% chg l std	-	4.0
LC50	588	lambda mean	1.00	0.91
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	4.45e-002
% Mortality	31	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.176 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg l std	-	8.0
LC50	17663	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.58e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.177 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-8
Concentration	470	% chg l std	-	7.2
LC50	588	lambda mean	1.01	0.92
LC50 slope	3.6	lambda std	0.06	0.05
species	sockeye	S1	2.57e-002	1.78e-002
% Mortality	31	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.178 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg l std	-	7.4
LC50	17663	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.179 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-12
Concentration	470	% chg l std	-	6.6
LC50	588	lambda mean	1.03	0.91
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.05e-002
% Mortality	31	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.180 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg l std	-	4.4
LC50	17663	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.181 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-9
Concentration	470	% chg l std	-	4.0
LC50	588	lambda mean	1.00	0.91
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	4.45e-002
% Mortality	31	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.182 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg l std	-	12.9
LC50	17663	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.62e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.183 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-10
Concentration	470	% chg l std	-	11.6
LC50	588	lambda mean	1.09	0.98
LC50 slope	3.6	lambda std	0.10	0.09
species	chum	S1	5.64e-003	3.87e-003
% Mortality	31	Significant change		9.3
Percent Exposed	100	[]		

Pentachlorophenol

Table 2.6.5.1.184 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg l std	-	12.8
LC50	86.1	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.1
species	Chinook, ot	S1	5.63e-003	5.57E-03
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.185 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-49
Concentration	19	% chg l std	-	6.4
LC50	10	lambda mean	1.09	0.55
LC50 slope	3.6	lambda std	0.10	0.05
species	chinook, ot	S1	5.62e-003	5.09e-004
% Mortality	91	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.186 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg l std	-	4.4
LC50	86.1	lambda mean	1.00	1
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.37E-02
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.187 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-45
Concentration	19	% chg l std	-	2.4
LC50	10	lambda mean	1.00	0.55
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.44e-002	5.81e-003
% Mortality	91	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.188 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg l std	-	7.9
LC50	86.1	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.58e-002	2.55E-02
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.189 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-43
Concentration	19	% chg l std	-	4.4
LC50	10	lambda mean	1.01	0.57
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.56e-002	2.32e-003
% Mortality	91	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.190 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg l std	-	7.5
LC50	86.1	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.94E-02
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.191 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-55
Concentration	19	% chg l std	-	3.4
LC50	10	lambda mean	1.03	0.46
LC50 slope	3.6	lambda std	0.05	0.02
species	coho	S1	2.97e-002	2.68e-003
% Mortality	91	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.192 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg l std	-	4.4
LC50	86.1	lambda mean	1.00	1
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.37E-02
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.193 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-45
Concentration	19	% chg l std	-	2.4
LC50	10	lambda mean	1.00	0.55
LC50 slope	3.6	lambda std	0.03	0.02
species	steelhead	S1	6.43e-002	5.80e-003
% Mortality	91	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.194 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg l std	-	12.8
LC50	86.1	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.1
species	chum	S1	5.63e-003	5.57E-03
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.195 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-49
Concentration	19	% chg l std	-	6.4
LC50	10	lambda mean	1.09	0.55
LC50 slope	3.6	lambda std	0.10	0.05
species	chum	S1	5.64e-003	5.07e-004
% Mortality	91	Significant change		9.1
Percent Exposed	100	[]		

Selenium

Table 2.6.5.1.196 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	12.9
LC50	4268	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.197 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg l std	-	0.1
LC50	0.4	lambda mean	1.09	0.01
LC50 slope	3.6	lambda std	0.10	0.00
species	chinook, ot	S1	5.65e-003	1.30e-012
% Mortality	100	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.198 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	4.4
LC50	4268	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.199 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg l std	-	0.0
LC50	0.4	lambda mean	1.00	0.01
LC50 slope	3.6	lambda std	0.03	0.00
species	chinook, st	S1	6.44e-002	1.49e-011
% Mortality	100	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.200 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	8.0
LC50	4268	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.201 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg l std	-	0.1
LC50	0.4	lambda mean	1.01	0.01
LC50 slope	3.6	lambda std	0.06	0.00
species	sockeye	S1	2.57e-002	5.94e-012
% Mortality	100	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.202 Model output data for coho.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	7.5
LC50	4268	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.203 Model output data for coho.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-100
Concentration	190	% chg l std	-	0.0
LC50	0.4	lambda mean	1.03	0.00
LC50 slope	3.6	lambda std	0.05	0.00
species	coho	S1	2.96e-002	6.85e-012
% Mortality	100	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.204 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	4.4
LC50	4268	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.205 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg l std	-	0.0
LC50	0.4	lambda mean	1.00	0.01
LC50 slope	3.6	lambda std	0.03	0.00
species	steelhead	S1	6.43e-002	1.49e-011
% Mortality	100	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.206 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	12.9
LC50	4268	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.207 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg l std	-	0.1
LC50	0.4	lambda mean	1.09	0.01
LC50 slope	3.6	lambda std	0.10	0.00
species	chum	S1	5.64e-003	1.30e-012
% Mortality	100	Significant change		9.0
Percent Exposed	100	[]		

Silver

Table 2.6.5.1.208 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg l std	-	12.9
LC50	63	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.209 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-60
Concentration	3.2	% chg l std	-	5.0
LC50	1.28	lambda mean	1.09	0.43
LC50 slope	3.6	lambda std	0.10	0.04
species	chinook, ot	S1	5.63e-003	2.00e-004
% Mortality	96	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.210 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg l std	-	4.4
LC50	63	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.211 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-56
Concentration	3.2	% chg l std	-	1.9
LC50	1.28	lambda mean	1.00	0.44
LC50 slope	3.6	lambda std	0.03	0.01
species	chinook, st	S1	6.43e-002	2.29e-003
% Mortality	96	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.212 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg l std	-	7.9
LC50	63	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.213 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-54
Concentration	3.2	% chg l std	-	3.5
LC50	1.28	lambda mean	1.01	0.46
LC50 slope	3.6	lambda std	0.06	0.02
species	sockeye	S1	2.58e-002	9.17e-004
% Mortality	96	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.214 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg l std	-	7.5
LC50	63	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.215 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-67
Concentration	3.2	% chg l std	-	2.4
LC50	1.28	lambda mean	1.03	0.34
LC50 slope	3.6	lambda std	0.05	0.02
species	coho	S1	2.97e-002	1.06e-003
% Mortality	96	Significant change		5.2
Percent Exposed	100	[]		

Table 2.6.5.1.216 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg l std	-	4.4
LC50	63	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.217 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-56
Concentration	3.2	% chg l std	-	1.9
LC50	1.28	lambda mean	1.00	0.44
LC50 slope	3.6	lambda std	0.03	0.01
species	steelhead	S1	6.43e-002	2.29e-003
% Mortality	96	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.218 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg l std	-	12.9
LC50	63	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.219 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-60
Concentration	3.2	% chg l std	-	5.0
LC50	1.28	lambda mean	1.09	0.43
LC50 slope	3.6	lambda std	0.10	0.04
species	chum	S1	5.62e-003	2.00e-004
% Mortality	96	Significant change		9.0
Percent Exposed	100	[]		

Tributyltin

Table 2.6.5.1.220 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg l std	-	13.0
LC50	2.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.65e-003	5.64e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.221 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-55
Concentration	0.46	% chg l std	-	5.6
LC50	0.21	lambda mean	1.09	0.49
LC50 slope	3.6	lambda std	0.10	0.04
species	chinook, ot	S1	5.64e-003	3.16e-004
% Mortality	94	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.222 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg l std	-	4.4
LC50	2.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.223 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-51
Concentration	0.46	% chg l std	-	2.1
LC50	0.21	lambda mean	1.00	0.49
LC50 slope	3.6	lambda std	0.03	0.01
species	chinook, st	S1	6.44e-002	3.61e-003
% Mortality	94	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.224 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg l std	-	7.9
LC50	2.6	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.56e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.225 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-49
Concentration	0.46	% chg l std	-	3.9
LC50	0.21	lambda mean	1.01	0.51
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.57e-002	1.44e-003
% Mortality	94	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.226 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg l std	-	7.4
LC50	2.6	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.227 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-62
Concentration	0.46	% chg l std	-	2.9
LC50	0.21	lambda mean	1.03	0.39
LC50 slope	3.6	lambda std	0.05	0.02
species	coho	S1	2.97e-002	1.66e-003
% Mortality	94	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.228 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg l std	-	4.4
LC50	2.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.229 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-51
Concentration	0.46	% chg l std	-	2.1
LC50	0.21	lambda mean	1.00	0.49
LC50 slope	3.6	lambda std	0.03	0.01
species	steelhead	S1	6.44e-002	3.61e-003
% Mortality	94	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.230 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg l std	-	13.0
LC50	2.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.65e-003	5.64e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.231 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-55
Concentration	0.46	% chg l std	-	5.6
LC50	0.21	lambda mean	1.09	0.49
LC50 slope	3.6	lambda std	0.10	0.04
species	chum	S1	5.64e-003	3.16e-004
% Mortality	94	Significant change		9.1
Percent Exposed	100	[]		

Zinc

Table 2.6.5.1.232 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	12.9
LC50	1188	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.233 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-3
Concentration	120	% chg l std	-	12.5
LC50	238	lambda mean	1.09	1.06
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.19e-003
% Mortality	8	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.234 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	4.4
LC50	1188	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.235 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-2
Concentration	120	% chg l std	-	4.3
LC50	238	lambda mean	1.00	0.98
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	5.93e-002
% Mortality	8	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.236 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	7.9
LC50	1188	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.55e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.237 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-2
Concentration	120	% chg l std	-	7.7
LC50	238	lambda mean	1.01	0.99
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.37e-002
% Mortality	8	Significant change		5.5
Percent Exposed	100	[]		

Table 2.6.5.1.238 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	7.6
LC50	1188	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.06	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.4
Percent Exposed	100	[]		

Table 2.6.5.1.239 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-3
Concentration	120	% chg l std	-	7.3
LC50	238	lambda mean	1.03	1.00
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.73e-002
% Mortality	8	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.240 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	4.4
LC50	1188	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.241 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-2
Concentration	120	% chg l std	-	4.3
LC50	238	lambda mean	1.00	0.98
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	5.93e-002
% Mortality	8	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.242 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	4.4
LC50	1188	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chum	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.243 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-3
Concentration	120	% chg l std	-	12.6
LC50	238	lambda mean	1.09	1.06
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.20e-003
% Mortality	8	Significant change		9.2
Percent Exposed	100	[]		

Summary. Based on the direct mortality population modeling results, juvenile salmon and steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc is predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) ranging from zero percent to -100 percent based on the exposure scenario. Direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for both modeling scenarios.

2.6.6. Case Study on Extrapolating Growth Reductions in Fish to Changes in Population Extinction Risks: Copper and Chinook Salmon

This section examines the potential consequences of reduced growth on the survival of juvenile Chinook salmon from exposure to low levels of copper that commence prior to hatching. Toxicological assays generally do not consider or attempt to link effects on growth to changes in population and to long-term extinction risks. However, Mebane and Arthaud (2010) suggested that size reductions from early-life stage chronic sublethal copper exposure could potentially reduce juvenile salmon survival and population recovery trajectories. This study is different from the direct mortality, somatic growth, and population modeling in section 2.6.5 in which the literature found that growth of fry, on the whole, was not a sensitive endpoint for the effect of copper on juvenile salmonids relative to mortality. In the case study by Mebane and Arthaud (2010) they conclude that growth resulting from early life stage exposure is usually a more sensitive endpoint than mortality to copper. This case study modeled responses of juvenile Chinook salmon exposed to sustained exposures of low levels of copper starting during early development and extrapolated growth reductions and changes in survival related to individual size. Most of the literature on copper and juvenile salmonid fry that examines reduced growth shows little mortality in laboratory toxicity tests, which tend to be short in exposure duration and do not look at relationships between reduced growth and size-dependant survival. Chapman (1994 as cited in Mebane and Arthaud 2010) exposed different life stages of steelhead (*Oncorhynchus mykiss*) for the same duration (3 months) to the same concentration of copper (13.4 µg/L at a hardness of 24 mg/L as $CaCO_3$). The survival of steelhead that were initially

exposed as embryos was no different than that of the unexposed control fish, even though the embryos developed into the usually-sensitive swim-up fry stage during the exposure. In contrast, steelhead that were initially exposed as swim-up fry, without the opportunity for acclimation during the embryo state, suffered complete mortality.

At low-level, sustained exposures, copper is one substance that commonly causes reduced growth but little direct mortality in laboratory toxicity tests with early life stage fish. To explore the relevance of growth reductions under laboratory conditions to wild populations, they 1) estimated growth effects of low-level copper exposures to juvenile Chinook salmon, 2) related growth effects to reduced survival in downriver Chinook salmon migrations, 3) estimated population demographics, 4) constructed a demographically structured matrix population model, and 5) projected the influence of copper-reduced growth on population size, extinction risks, and recovery chances. Reduced juvenile growth from copper in the range of 11 μg/L (the proposed chronic criteria for copper in Oregon is 9 μg/L) was projected to cause disproportionate reductions in survival of migrating juveniles, with a 7.5 percent length reduction predicting about a 23 percent to 52 percent reduction in survival from a headwaters trap to the next census point located 640 km downstream. Projecting reduced juvenile growth out through six generations (~30 years) resulted in little increased extinction risk; however, population recovery times were delayed under scenarios where copper-reduced growth was imposed.

Reduced growth is a common stress response in fish. A variety of causes can lead to stress responses and reduced growth in fish, including suboptimal nutrition or temperatures, low ion content of water (soft water), crowding, subordinate social status, and either the direct effects of chemical exposures or the energy costs of detoxifying chemicals (Wendelaar Bonga 1997 as cited in Mebane and Arthaud 2010). In ecotoxicological bioassays that run long enough, growth effects are a readily and routinely measured endpoint. In water-quality criteria derivation in the United States, the only sublethal effects that *a priori* are considered biologically important are growth or reproductive impairment, although on a case-by-case basis, data on a variety of other sublethal effects of chemicals to fish could also be important, such as swimming performance, disease resistance, or behaviors related to chemoreception (Stephan *et al.* 1985, Stephan 1986 as cited in Mebane and Arthaud 2010). However, laboratory bioassays seldom are a means unto themselves, but probably are at least indirectly conducted because societal values such as protecting the abundance and persistence of populations, biodiversity, conservation of threatened species, and recreational aesthetics (Stephan 1986, Barnthouse *et al.* 1989 as cited in Mebane and Arthaud 2010).

This motivation implies some consideration of population-level effects when interpreting toxicity bioassays. Yet, from a population biology perspective, the only endpoints that matter for a closed population are birth and death rates. Growth and any other sublethal endpoints are irrelevant unless they can be related to birth or death rates. The reproductive consequences of profound growth effects are selfevident; an organism that fails to grow is unlikely to reproduce.

However, the consequences of transitory or subtle growth reductions are less obvious. For instance, in lifecycle testing with brook trout (*Salvelinus fontinalis*) and copper, McKim and Benoit (1971 as cited in Mebane and Arthaud 2010) reported that, for their first several months of life, fish that were exposed to low, sublethal copper concentrations lagged behind control fish in their growth. However, after about six months of copper exposure, fish experienced

compensatory growth rates and largely caught up with control fish by the end of the tests (McKim and Benoit 1971 as cited in Mebane and Arthaud 2010). Because the differences were no longer statistically different at the end of their tests, the growth delays were discounted as adverse effects. Similar instances of transitory or subtle growth reductions have been noted for rainbow trout (*Oncorhynchus mykiss*) exposed to copper (Marr *et al.* 1996, Hansen *et al.* 2002 as cited in Mebane and Arthaud 2010). However, delayed growth may not necessarily be a discountable effect in the wild because, if juvenile fish encounter a size-dependent bottleneck in early life, smaller fish may not survive long enough to benefit from compensatory growth. Traits and costs that have been associated with reduced growth in juvenile fish include acquisition of feeding territory or shelter, predation risk, body size at key times, energy reserves at key times, increased thermoregulatory costs, and mortality (Sogard 1997, Metcalfe and Monaghan 2001, Harwood *et al.* 2002, Coleman and Fausch 2007 as cited in Mebane and Arthaud 2010). The magnitudes of size differences that have been important in outcomes of challenges with juvenile fish can be small. For example, torrent sculpin (*Cottus rhotheus*) are a predator of juvenile salmon in streams. Torrent sculpin that were about 60 mm long were no threat to coho salmon (*O. kisutch*) that were also about 60 mm long. However, the 60 mm sculpin can successfully ambush, subdue, and eat 50 mm coho salmon (Patten 1977 as cited in Mebane and Arthaud 2010). Abbott *et al.* (1985 as cited in Mebane and Arthaud 2010) found that bigger fish tend to dominate smaller fish in contests for territory, and a size disparity of only 5 percent in body weight confers significant advantage. However, subtle growth reductions may be discounted as effects in toxicity tests if they are not statistically different from controls in null hypothesis significance testing with less than a 5 percent likelihood of making a Type I error. These purely statistical definitions of significant effects are at best incomplete and at worst misleading, in part because the probability that a given reduction is statistically significant is inversely related to the quality and quantity of the data (*e.g.*, Barnthouse *et al.* 1989 as cited in Mebane and Arthaud 2010).

The case study of growth effects from copper and a Chinook salmon population explored how subtle growth reductions in juvenile fish might affect the abundance and persistence of natural populations of migratory fish. The study objectives included:

1. Estimating the magnitude of growth reductions likely for Chinook salmon resulting from prolonged laboratory test exposure to copper at 11 μ/gL that had been estimated to be safe for most aquatic ecosystems. The chronic criterion for copper in Oregon is 13 μg/L.
2. Estimating potential consequences of reduced growth for the survival of juvenile Chinook salmon during rearing and migration.
3. Quantifying the potential consequences of reduced survival in migrating juvenile salmon as changes in the long-term extinction risk and recovery potential of the salmon populations.

For this exercise, Mebane and Arthaud selected the Marsh Creek Chinook salmon population, located at the headwaters of the Middle Fork of the Salmon River, Idaho, USA (44◦ 27_N, 115◦14_W at its mouth). Marsh Creek is an oligotrophic, forested watershed, with few pollution or human attributable disturbances other than potentially decreased freshwater productivity and correspondingly diminished carrying capacities from the decline of marine derived nutrients (Kohler *et al.* 2008 as cited in Mebane and Arthaud 2010). The lack of pollution sources greatly

simplifies predicting the potential effects of a chemical stressor. Furthermore, by using a headwaters population for this modeling exercise, the baseline model accounts for a myriad of other factors affecting Chinook salmon populations besides the potential stress of copper pollution considered here.

The projections of potential population-level effects of reduced growth from copper were made in five steps:

- Evaluating the effects of chronic copper toxicity on salmon in laboratory tests
- Extrapolating reduced growth in toxicity test results to survival of juvenile migrants
- Analyzing population demographics
- Developing a baseline population model, and
- Linking changed population vital rates from copper-influenced scenarios to population size and extinction risks.

Nonlinear regression was used to interpolate between effects at the control concentration and the lowest effect concentration to estimate effects at the 1992 NTR criteria concentration of 12 μg/L, total recoverable. Because of this uncertainty, we also examined a chronic test of rainbow trout in soft water that tested lower copper concentrations and required less interpolation (Marr *et al.* 1996 as cited in Mebane and Arthaud 2010). Chinook salmon and rainbow/steelhead trout are closely related, and other tests have shown similar sensitivity to copper and other metals (Chapman 1978 as cited in Mebane and Arthaud 2010).

Logistic regression described the relation between length and copper concentrations well, and it provided an estimated length reduction from controls of 7.5 percent and a weight reduction of 20 percent at 3.6 μg/L, the hardness-adjusted 1992 CCC. The estimated length reductions at 3.6 μg/L ranged from 4 percent to 18 percent, obtained using different statistical distributions and curve fits (*e.g.*, linear, piecewise linear, logistic). For weight reductions, the corresponding reductions were greater, 12 to 20 percent, depending on the model used. The rainbow trout growth reductions were very similar to those estimated at similar concentrations with Chinook salmon using the same statistical models, suggesting that the needed interpolations of the Chinook toxicity data were reasonable.

The selection of a regression model to fit these Chinook salmon data involves fundamental, implicit assumptions of the ecotoxicology of chronic copper and fish. The logistic regression curves slope smoothly downward to interpolate from the control concentration to the first treatment. Thus, an implicit assumption of the model shape is that slight increases in copper result in corresponding slight growth reductions, with no threshold of response. In contrast, the piecewise linear regressions implicitly assume a threshold of response, below which copper concentrations have no effect on growth. It may be unrealistic to assume that no threshold exists for copper exposure and the onset of growth effects. Likewise, the abrupt bend in the corners of the piecewise linear regression that indicate the threshold concentration may also be arbitrary and unrealistic. Because neither model had an obviously better theoretical basis and because both models fit the data well, the effects estimates with each are carried forward through the population modeling using both 7.5 percent and 4 percent length reductions at 3.6 μg/L copper

from the logistic and piecewise models, respectively. This provided a range of estimates of growth effects of copper to Chinook salmon at the 1992 CCC of 3.6 μg/l.

The Mebane and Arthaud analysis focuses on EPA's (NTR 1992) copper criteria of 18 μg/L (CMC) and 12 μg/L (CCC) (updates have been published, EPA 2006 and 2007, although at the time of writing, the 1992 values remained effective in some states, including Oregon). The EPA's 2006 recommended criteria were based on the same approach as the 1992 version with minor dataset revisions. In contrast, the 2007 values were derived from a fundamentally different approach that predicted copper bioavailability through geochemical modeling to estimate copper accumulation on gills and subsequent toxicity. For the water chemistry conditions of Chapman's (1982 as cited in Mebane and Arthaud 2010) test, the 2006 and 2007 chronic copper criteria values would be about 2.7 and 2.1 μg/L, respectively. The interpolated length reductions with Chapman's (1982 as cited in Mebane and Arthaud 2010) Chinook salmon test at the 2006 criterion value of 2.7μg/L ranged from about 6 percent to zero using logistic regression and piecewise regression models, respectively. For the 2007 criterion value of about 2.1 μg/L, the corresponding length reduction estimates ranged from about 4.5 percent to zero. Thus the modeled scenarios are also relevant to the more recent copper chronic criteria updates. For the 2006 version, the upper effects estimate (6% length reduction) would be intermediate to the 7.5 percent and 4 percent length reduction scenarios modeled. For the 2007 version, the upper effects estimate (4.5 percent length reduction) is close to the lower effects scenario modeled here (4 percent length reduction).

Risk probability statistics may provide more relevant assessments of thepopulation's relative risks of declines or extinction than do the population trajectory projections (Ferson *et al.* 1989 as cited in Mebane and Arthaud 2010). Rather than plotting abundance predictions over time, as was done with adult salmon in abundance, projections can be expressed as the risk that the population will be less than a given number or that it will decline by more than a given amount from the initial conditions.

If the risks are instead expressed as the probabilities that the projected numbers would drop below a given number of fish (quasi-extinction), then the risk curves have a similar, but mirrored shape. The probabilities of five consecutive severe declines are much lower than the risk of a single, very low spawning run. For example, under the baseline scenario ($\lambda = 1.31$) with density dependence, there is about a 50 percent risk that the population drops below its initial numbers (145 adults) and stays below that value for five years, and there is about a 32 percent risk that the population similarly drops and stays below our assumed quasi-extinction threshold of 25 adults. In contrast to population trajectory projections wherein by the third generation, the density independent or dependent projections differed markedly, when the baseline versus copper-growth reduction scenarios are compared as relative risks of decline or quasi-extinction, the risk values were mostly similar but slightly higher under the density dependent than independent model either assumptions of density independence or dependence.

Mebane and Arthaud (2010) interpreted the population recovery chances in three ways. First, the most lenient and optimistic statistic was the probability that the population would exceed the simulation model recovery threshold of 500 adults at any one time interval during the simulations. When these probabilities are plotted as a cumulative probability distribution, the

cumulative distribution of recovery times increases monotonically. Each point on this cumulative curve can be interpreted as there is a Y percent probability that the population abundance will exceed the 500 adult threshold in or before the year 30. Focusing on the medians of the distributions, the relative times to reaching the recovery abundance threshold can be compared between the scenarios. When the population growth was unconstrained by carrying capacity limitations,median times for the population to reach 500 adults were about 12, 17, and 27 years for the baseline, 4 percent length reduction from copper, and 7.5 percent length reduction from copper scenarios, respectively. When the population was constrained below a carrying capacity ceiling of 518 adults in the density dependent model, this nearly precluded the population from reaching a recovery target that was only slightly lower; median times projected for the population to reach 500 adults ranged from 22 years for the baseline to >30 years for the copper-lower and higher effects scenarios.

Second, when considering recovery as a more persistent increase in adult abundances over for five consecutive years, under the density independent scenarios, there were 50 percent probabilities that at least for one period of five-consecutive years at some time during the 30-year simulations, the adult abundances would reach about 420, 260, and 175 for the baseline, copper-lower effects (4 percent length reduction), and copper-higher effect (7.5 percent length reduction) scenarios, respectively. Under the ceiling density dependent scenarios, the adult abundances were similarly projected, with 50 percent probabilities, to reach about 290, 225, and 150 for the baseline and copper-lower or higher effects scenarios, respectively (Figure 2.6.6.1). When the threshold for recovery was defined as exceeding 500 adults for any one five-year period, attaining this recovery threshold within 30-years was unlikely for any modeled scenario, with chances of reaching that threshold ranging from 41 percent to nearly 0 percent across the scenarios (Figure 2.6.6.1).

Summary. The Chinook salmon length reductions estimated for the 1992 copper criterion concentration of about 4 to 7.5 percent were projected to result in 2 to 10 percent additional risk of quasi-extinction sometime in the next 6-generations, depending on the model. The corresponding estimated length reductions for the 2007 updated-EPA copper criterion concentration would range from about zero to 4 percent and would be projected to result in zero to 5 percent additional risk of quasi-extinction sometime in the next 6-generations. Chances of recovery differed more between the baseline and copper exposed scenarios in the density independent model than in the ceiling density dependence model. For instance, there were about 40 to 60 percent reductions, attributable to length reductions of 4 to 7.5 percent, respectively, of the highest population adult abundances projected with 50 percent likelihood of being reached and maintained for 5-years running in the next 6-generations. With the ceiling density dependent model, the reductions were projected to be about 20 to 50 percent from baseline population model, which indicates that the chronic criterion for copper is not likely to be protective of chronic toxic effects.

Scenario	Density independent projections			Density dependent projections		
	Baseline	4% length reduction scenario	7.5% length reduction scenario	Baseline	4% length reduction scenario	7.5% length reduction scenario
Expected minimum adult abundances (individuals)	46	34	22	36	28	19
Risk of a single severe (90%) decline of adult spawners (CI)	78% (76–81%)	80% (77–83%)	82% (79–85)	79% (76–82%)	80% (77–82%)	82% (79–85%)
Risk of quasi-extinction (<25 adults per year for 5 consecutive years)	30% (25–36%)	35% (30–42%)	40% (34–48%)	33% (27–39)	35% (29–42%)	42% (35–49%)
Probability of recovery to >500 adults for 5 consecutive years	41% (35–49)	14% (11–17%)	4% (3–5%)	6% (5–9%)	3% (2–4%)	0.2% (0.1–0.3%)
Probability of ending abundances >500 adults (CI)[1]	44% (41–46%)	30% (27–33)	21% (18–24%)	21% (18–24%)	15% (12–18%)	8% (5–11%)
Ending abundance, 50% probability of exceeding (CI)[2]	274 (179–357)	112 (74–167)	62 (43–88)	146 (103–190)	83 (59–133)	41 (23–64)

Table notes: Results of 1000 Monte Carlo simulations, simulations were run through 6 generations. CI- 95% Kolmogorov-Smirnov confidence intervals; [1]Probability that the adult abundance will end up greater than the recovery threshold of 500 after six generations; [2]After 6-generations, there are 50% probabilities that the adult abundances will end up greater than these numbers.

Figure 2.6.6.1 Risks of severe population decline or quasi-extinction, probabilities of recovery greater than a given threshold for different copper effects scenarios, using both density dependent and density independent simulation models (Mebane and Arthaud 2010).

2.6.7 Effects on Critical Habitat

The EPA's approval of the proposed criteria has the potential to adversely affect designated critical habitats through direct water-borne toxicity and bioaccumulation, as described below.

Pacific Salmon and Steelhead

1. ***Freshwater Spawning Sites***
   a. Substrate — Sediment contamination by toxic pollutants is likely to adversely affect critical habitat because the particulate forms of toxicants are either immediately bioavailable via discharge, through re-suspension, are a delayed source of toxicity through bioaccumulation, or are available when water quality conditions favor dissolution at a later date. Specifically, contaminated sediments are expected to influence intra-gravel life stages, food sources, and fish through direct ingestion or deposition on the gill surfaces of particulate forms of toxicants.

      Sediments as a source of contaminant exposure were not considered by EPA in the development of the national criteria, which are the same as the criteria proposed by the State of Oregon. The NMFS recognizes that considerable technical and practical problems exist in defining water quality criteria on a sediment basis, and that this is presently the subject of considerable research and debate. Nevertheless, most organic and metal contaminants adsorb to organic particulates and settle out in sediments, so at sites where there have been past discharges, or where there are continuing discharges of contaminants into the water column, they form a long-term repository and a continuing source of exposure that must be addressed if the water quality component of critical habitat is to be protected. Further, although these substances may not readily be transferred into the water column, they may still be available to fish through food chain transfer from their benthic prey, or through ingestion of sediment while feeding. Not having water quality criteria that consider uptake through these routes leaves a route of exposure to fish that the proposed criteria do not address. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *substrate* be adversely affected, and will be degraded at the watershed and designation scales.
   b. Water Quality — Freshwater spawning sites require water quality conditions that support spawning, incubation, and larval development. Based on the distribution and density, the distribution, fate and transport of the compounds listed in Table 1.1, and the distribution of spawning of UWR Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, LCR Chinook salmon, LCR coho salmon, SR SS Chinook salmon, SR fall-run Chinook salmon, SRB steelhead, CR chum salmon, OC coho

salmon, and SONCC coho salmon, we expect degraded water quality to coincide in time and space with spawning events.

The most severe effects to water quality within spawning sites will be those sites that are located in areas in close proximity to multiple point-source dischargers. Although spawning sites for UWR Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, LCR Chinook salmon, LCR coho salmon, SR SS Chinook salmon, SR fall-run Chinook salmon, SRB steelhead, CR chum salmon, OC coho salmon, and SONCC coho salmon are generally above high density point-source discharges, the downstream effects of low-density pollutant discharges upstream of spawning areas can reduce spawning success. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, and will be degraded at the watershed or designation scales.

c. Water Quantity — No effects are likely to occur.

2. ***Freshwater Rearing***

a. Floodplain Connectivity — No effects are likely to occur.

b. Forage — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *forage* will be adversely affected, but will not be degraded at the watershed or designation scales.

c. Natural Cover — No effects are likely to occur.

d. Water Quality — Freshwater rearing sites need to provide good water quality and abundant forage to support juvenile development. Reductions in either, can limit the existing and potential carrying capacity of rearing sites and subsequently reduce their conservation value.

Recovery of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR

chum salmon, OC coho salmon, SONCC coho salmon populations is tied closely to the success of juveniles to fully develop, mature, and grow during freshwater residency periods. Collectively, the toxicity data indicate that concentrations of the compounds listed in Table 1.1 are sufficient to adversely affect water quality in affected watersheds, as they do not support the associated life history events, such as fry/parr growth and development, for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, and will be degraded at the watershed and designation scales.

e. Water Quantity — No effects are likely to occur.

3. ***Freshwater Migration Corridors***

a. Forage — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *forage* will be adversely affected, but will not be degraded at the watershed or designation scales.

b. Free of Artificial Obstruction — No effects are likely to occur.

c. Natural Cover — No effects are likely to occur.

d. Water Quality — Freshwater migration corridors need to provide good water quality and abundant forage to support juvenile development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

Collectively, the toxicity data indicate that concentrations of the compounds listed in Table 1.1 are sufficient to adversely affect water quality in affected watersheds, as they do not support the associated life history events, such as smolt growth and development, for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run

Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, and will be degraded at the watershed and designation scales.

e. Water Quantity — No effects are likely to occur.

4. ***Estuarine Areas***

a. Forage – Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *forage* will be adversely affected, but will not be degraded at the watershed or designation scales.

b. Free of obstruction – No effects are likely to occur.

c. Natural cover –No effects are likely to occur.

d. Water quality — Estuarine areas require good water quality to support juvenile and adult physiological transitions between fresh water and salt water as well as areas to support growth and maturation.

Collectively, the toxicity data indicate that concentrations of the compounds listed in Table 1.1 are sufficient to adversely affect water quality in affected estuarine areas, as they do not support the associated life history events, such as smolt growth and development, for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon. For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water*

quality will be adversely affected, but will not be degraded at the watershed and designation scales.

5. ***Nearshore Marine Areas***
   a. None designated.
6. ***Offshore Marine Areas***
   a. None designated.

Based on the above assessment, the effects of the proposed action, in particular on the freshwater PCEs *water quality and substrate*, will appreciably diminish the conservation value of critical habitat at the designation scale for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon.

Green Sturgeon

1. ***Freshwater Riverine Systems***
   a. Food resources — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile, sub-adult and adult green sturgeon. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

      Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *food resources* will be adversely affected, but will not be degraded at the designation scale.
   b. Migratory corridor — Freshwater migration corridors need to provide good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

      For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *migratory corridor* will be adversely affected, and will be degraded at the designation scale.

c. Sediments as a source of contaminant exposure were not considered by EPA in the development of the national criteria, which are the same as the criteria proposed by the State of Oregon. The NMFS recognizes that considerable technical and practical problems exist in defining water quality criteria on a sediment basis, and that this is presently the subject of considerable research and debate. Nevertheless, most organic and metal contaminants adsorb to organic particulates and settle out in sediments, so at sites where there have been past discharges, or where there are continuing discharges of contaminants into the water column, they form a long-term repository and a continuing source of exposure that must be addressed if the water quality component of critical habitat is to be protected. Further, although these substances may not readily be transferred into the water column, they may still be available to fish through food chain transfer from their benthic prey, or through ingestion of sediment while feeding. Not having water quality criteria that consider uptake through these routes leaves a route of exposure to fish that the proposed criteria do not address. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *substrate* be adversely affected, and will be degraded at the designation scale.

d. Substrate type or size — No effects are likely to occur.

e. Water depth — No effects are likely to occur.

f. Water flow — No effects are likely to occur.

g. Water quality — Freshwater riverine systems need to provide good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, and will be degraded at the designation scale.

2. ***Estuarine Systems***

a. Food resources — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which can be expected to reduce fitness, in estuaries where food is a limiting factor.

Changes in species composition can have the same results in fitness and survival. Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to

juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *food resources* will be adversely affected, but will not be degraded at the designation scale.

b. Migratory corridor — Estuarine migration corridors need to provide good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *migratory corridor* will be adversely affected, but will not be degraded at the designation scale.

c. Sediments as a source of contaminant exposure were not considered by EPA in the development of the national criteria, which are the same as the criteria proposed by the State of Oregon. The NMFS recognizes that considerable technical and practical problems exist in defining water quality criteria on a sediment basis, and that this is presently the subject of considerable research and debate. Nevertheless, most organic and metal contaminants adsorb to organic particulates and settle out in sediments, so at sites where there have been past discharges, or where there are continuing discharges of contaminants into the water column, they form a long-term repository and a continuing source of exposure that must be addressed if the water quality component of critical habitat is to be protected. Further, although these substances may not readily be transferred into the water column, they may still be available to fish through food chain transfer from their benthic prey, or through ingestion of sediment while feeding. Not having water quality criteria that consider uptake through these routes leaves a route of exposure to fish that the proposed criteria do not address. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *substrate* be adversely affected, and will be degraded at the designation scale.

d. Water flow — No effects are likely to occur.

e. Water depth — No effects are likely to occur.

f. Water quality — Estuarine areas need to provide good water quality and abundant forage to support growth and development.

For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds

listed in Table 1.1, the PCE *water quality* will be adversely affected, but will not be degraded at the designation scale.

3. Coastal Marine Areas

a. Food Resources — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which can be expected to reduce fitness, in coastal marine areas where food is a limiting factor.

Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *food resources* will be adversely affected, but will not be degraded at the designation scale.

b. Migratory Corridor — Coastal marine migration corridors need to provide good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *migratory corridor* will be adversely affected, but will not be degraded at the designation scale.

c. Water Quality — Coastal marine areas require good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

Based on the available toxicity data, the distribution and density of point-source discharges in salt water, the limited area of saltwater habitat for green sturgeon within the action area, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, but will not be degraded at the designation scale.

Based on the above assessment, the effects of the proposed action, in particular on the freshwater PCEs *water quality, migratory corridors, and sediment quality* will appreciably diminish the conservation value of critical habitat at the designation scale for green sturgeon.

Eulachon

1. ***Freshwater Spawning***
   a. Water Flow — No effects are expected to occur.
   b. Water Quality — Freshwater spawning sites require water quality conditions that support spawning, incubation, and larval development. The degradation of water quality by exposure to the stressors of the action is indicated via the toxic responses in a variety of aquatic organisms including listed species. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *water quality* will be adversely affected, and will be degraded at the designation scale.
   c. Water Temperature — No effects are expected to occur.
   d. Substrate — Sediment contamination by toxic pollutants is likely to adversely affect critical habitat because the particulate forms of toxicants are either immediately bioavailable via discharge, through re-suspension, are a delayed source of toxicity through bioaccumulation, or are available when water quality conditions favor dissolution at a later date. Specifically, contaminated sediments are expected to influence intragravel life stages, food sources, and fish through direct ingestion or deposition on the gill surfaces of particulate forms of toxicants.

      Sediments as a source of contaminant exposure were not considered by EPA in the development of the national criteria, which are the same as the criteria proposed by the State of Oregon. The NMFS recognizes that considerable technical and practical problems exist in defining water quality criteria on a sediment basis, and that this is presently the subject of considerable research and debate. Nevertheless, most organic and metal contaminants adsorb to organic particulates and settle out in sediments, so at sites where there have been past discharges, or where there are continuing discharges of contaminants into the water column, they form a long-term repository and a continuing source of exposure that must be addressed if the water quality component of critical habitat is to be protected. Further, although these substances may not readily be transferred into the water column, they may still be available to fish through food chain transfer from their benthic prey, or through ingestion of sediment while feeding. Not having water quality criteria that consider uptake through these routes leaves a route of exposure to fish that the proposed criteria do not address. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *substrate* be adversely affected, and will be degraded at the designation scale.

2. Freshwater Migration

a. Migratory Corridor — Freshwater migration corridors need to provide good water quality to support larval development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

   For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *migratory corridor* will be adversely affected, and will be degraded at the designation scale.

b. Water Flow — No effects are expected.

c. Water Quality — For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *water quality* will be adversely affected, and will be degraded at the designation scale.

d. Water Temperature — No effects are expected.

e. Forage — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

   Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *forage* will be adversely affected, but will not be degraded at the designation scale.

Based on the above assessment, the effects of the proposed action, in particular on the freshwater PBFs *water quality, substrate, and migratory corridor* will appreciably diminish the conservation value of critical habitat at the designation scale for eulachon.

2.6.8 Cumulative Effects

"Cumulative effects" are those effects of future State or private activities, not involving Federal activities, that are reasonably certain to occur within the action area of the Federal action subject to consultation (50 CFR 402.02). Future Federal actions that are unrelated to the proposed action are not considered in this section because they require separate consultation pursuant to section 7 of the ESA.

Some types of human activities that contribute to cumulative effects are likely to have adverse effects on listed species and critical habitat PCEs. Many of which are activities occurred in the recent past and had an effect on the environmental baseline. These can be considered reasonably certain to occur in the future because they occurred frequently in the recent past. Within the freshwater portion of the action area, non-Federal actions are likely to include human population growth, water withdrawals (*i.e.*, those pursuant to senior state water rights) and land use practices. In the action area, state, tribal, and local government actions are likely to be in the form of legislation, administrative rules, or policy initiatives, shoreline growth management and resource permitting.

The states of the west coast region, which contribute water to major river systems, are projected to have the most rapid growth of any area in the U.S. within the next few decades. California, Idaho, Oregon, and Washington are forecasted to have double digit increases in population for each decade from 2000 to 2030 (USCB 2005). Overall, the west coast region had a projected population of 72.2 million people in 2010. The U.S. Census Bureau predicts this figure will grow to 76.8 million in 2015 and 81.6 million in 2020.

Although general population growth stems from development of metropolitan areas, growth in the western states is projected from the enlargement of smaller cities rather than from major metropolitan areas. Of the 46 western state metropolitan areas that experienced a 10% growth or greater between 2000 and 2008, only the Portland-Vancouver-Beaverton, OR (1.81% per year) metropolitan area occurs in the action area (USCB 2009).

As these cities border riverine systems, diffuse and extensive growth will increase overall volume of contaminant loading from wastewater treatment plants and sediments from sprawling urban and suburban development into riverine, estuarine, and marine habitats. Urban runoff from impervious surfaces and roadways may also contain oil, heavy metals, PAHs, and other chemical pollutants and flow into state surface waters. Inputs of these point and non-point pollution sources into numerous rivers and their tributaries will affect water quality in available spawning and rearing habitat for salmon. Based on the increase in human population growth, NMFS expects an associated increase in the number of NPDES permits issued and a concomitant increase of pollutant loading.

Mining has historically been a major component of western state economies. With national output for metals projected to increase by 4.3% annually, output of western mines should increase markedly (Figueroa and Woods 2007). Increases in mining activity will add to existing significant levels of mining contaminants entering river basins. Given this trend, we expect existing water degradation in Oregon streams that feed into or provide spawning habitat for threatened and endangered species to be exacerbated.

As the western states have large tracts of irrigated agriculture, a 2.2% rise in agricultural output is anticipated (Figueroa and Woods 2007). Impacts from heightened agricultural production will likely result in two negative impacts on listed species. The first impact is the greater use and application of pesticide, fertilizers, and herbicides and their increased concentrations and entry into freshwater systems. insecticides, and other pollutants from agricultural runoff may further degrade existing fish habitats. Second, increased output and water diversions for agriculture may

also place greater demands upon limited water resources. Water diversions will reduce flow rates and alter habitat throughout freshwater systems. As water is drawn off, contaminants will become more concentrated in these systems, exacerbating contamination issues in habitats for protected species.

The above non-federal actions are likely to pose continuous unquantifiable negative effects on listed species addressed in this opinion. These effects include increases in sedimentation, increased point and non-point pollution discharges, decreased infiltration of rainwater (leading to decreases in shallow groundwater recharge, decreases in hyporheic flow, and decreases in summer low flows).

Non-federal actions likely to occur in or near surface waters in the action area may also have beneficial effects on listed species addressed in this opinion. They include implementation of riparian improvement measures and fish habitat restoration projects, for example. Coupled with EPA's approval of the proposed water quality standards for aquatic life, the effects from anthropogenic growth on the natural environment will continue to allow toxic discharges to affect and influence the overall distribution, survival, and recovery of listed species in the Columbia River basin and Oregon.

NMFS also expects the natural phenomena in the action area (*e.g.*, oceanographic features, ongoing and future climate change, storms, natural mortality) will continue to influence listed species. Climate change effects are expected to be evident as alterations of water yield, peak flows, and stream temperature. Other effects, such as increased vulnerability to catastrophic wildfires, may occur as climate change alters the structure and distribution of forest and aquatic systems.

Although these factors are ongoing to some extent and likely to continue in the future, past occurrence is not a guarantee of a continuing level of activity. That will depend on whether there are economic, administrative, and legal impediments or safeguards in place. Therefore, although NMFS finds it likely that the cumulative effects of these activities will have adverse effects commensurate with or greater than those of similar past activities; it is not possible to quantify these effects.

2.7 Integration and Synthesis

The Integration and Synthesis section is the final step of NMFS' assessment of the risk posed to species and critical habitat as a result of implementing the proposed action. In this section, we add the effects of the action (section 2.6) to the environmental baseline (section 2.5) and the cumulative effects (section 2.6.8) to formulate the agency's biological opinion as to whether the proposed action is likely to: (1) Result in appreciable reductions in the likelihood of both survival and recovery of the species in the wild by reducing its numbers, reproduction, or distribution; or (2) reduce the value of designated or proposed critical habitat for the conservation of the species. These assessments are made in full consideration of the status of the species and critical habitat (section 2.4).

This section is comprised of the following: (1) a description of the multiple lines of evidence and effects decision criteria used by NMFS to assess toxicity and fitness consequences, (2) a synthesis of information regarding likely toxicity and environmental effect pathways, species and critical habitat status, cumulative effects and fitness consequences associated with exposure to Oregon's freshwater and saltwater criteria, and (3) ESU/DPS-specific evaluations. These components are described in detail below.

The analysis on multiple lines of evidence and effects decision criteria provides a breakdown of the significance of the likely effects of each criterion based on the analysis of the freshwater and saltwater toxicity data, an overview of how the toxicity data factor into our effect determinations, and a description of how NMFS applied the results of the direct mortality population modeling. The synthesis of information on acute and chronic endpoints, environmental stressors, species and critical habitat status, cumulative effects, and fitness consequences is a qualitative risk assessment for each criterion that considers endpoint-effects on listed species, risks associated with exposure to chemical mixtures, results of the direct mortality population modeling, and threats associated with interactions of the criteria with environmental baseline stressors. The ESU/DPS-specific evaluations analyze how the proposed action affects population attributes, species viability, and the conservation value of critical habitat.

Legacy Compounds.

In 1987 the EPA banned all uses of dieldrin. In 2010 EPA took action to eliminate all uses of endosulfan in the U.S., with a complete phase-out scheduled by 2016. In 1986 the EPA banned production of endrin in the U.S. In 1988 EPA banned the use of heptachlor epoxide except for limited use for fire ant control in underground transformers. In 2006 EPA issued final orders cancelling pesticide products containing lindane. However, the Food and Drug administration permits the use of lindane in pharmaceutical products to control lice and scabies. The NMFS does not expect population-level adverse effects to listed species considered in this opinion from exposure to any of the six legacy criteria (*i.e.*, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, and lindane,) as their use is either prohibited by law or highly restricted.

(1) Multiple Lines of Evidence and Effects Decision Criteria.

The foremost line of evidence applied in NMFS' effects decision is the criterion-specific toxicity data. The NMFS coupled this toxicity data analysis with the summary analysis, the chemical mixtures analysis, the direct mortality population modeling, and exposure to baseline chemical stressors. The NMFS then used this information used to assess the risk associated with exposure to the compounds in Table 1.1 on each of the affected species considered in this opinion.

To examine the significance of the effects of all freshwater criteria, NMFS ran the acute criteria (for all chemicals) and chronic criteria (for ammonia, cadmium, and copper only) through a direct mortality population model (see section 2.6.5 and Appendix 3) to evaluate the magnitude of the effects of juvenile mortality on productivity for the salmonid fish species considered in this opinion. The NMFS also examined the available toxicity data on ammonia, cadmium, and copper for inclusion in a somatic growth model to assess changes in fry growth that would affect

population growth rates, but the available data for these compounds could not be translated into appropriate input parameters for this model (see Appendix 3). Therefore, NMFS relied on the chronic toxicity data analysis for determining the risks of growth impairment and other sublethal effects associated with the chronic criteria and the significance of those risks to the listed species considered in this opinion.

The NMFS applied the results of the direct mortality population model as secondary line of evidence to assess the potential impact that EPA's approval of the numeric criteria would have on species' productivity. The NMFS applied the modeling results to the effects analysis in the following manner:

1. For compounds where all four modeling scenarios (described above in section 2.6.5.1) predicted a measurable level of mortality with a resulting change in λ (except for the legacy compounds), then NMFS considered these compounds to have a very high probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

2. For compounds where three of the four modeling scenarios predicted a level of mortality with a resulting change in λ (except for the legacy compounds), NMFS considered these compounds to have a high probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

3. For compounds where two of the four modeling scenarios predicted a level of mortality with a resulting change in λ (except for the legacy compounds), NMFS considered these compounds to have a moderate-to-high probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

4. For compounds where one of the four modeling scenarios predicted a level of mortality with a resulting change in λ (except for the legacy compounds), NMFS considered these compounds to have a moderate probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

5. For compounds where none of the four modeling scenarios predict a level of mortality, NMFS considered these compounds to have a low probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

These results of the direct mortality population model were then integrated into the primary lines of evidence in the opinion—the acute toxicity data, chronic toxicity data, the analysis on the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the mixtures analysis—to determine which compounds result in the highest-intensity of acute and/or chronic toxic effects on the listed species considered in this opinion. As part of this integration, NMFS also considered the exposure scenario and the magnitude of the change in λ when assessing which compounds were associated with significant adverse toxicological and biological effects.

Depending upon the modeling scenario for the legacy compounds, the direct mortality modeling predicted a negative percent change in λ. However, since the legacy compounds are either prohibited by law or highly restricted, NMFS considered that these compounds would be unlikely to appreciably reduce productivity and abundance such that the listed species' survival and recovery would not be at increased risk as water surface concentrations of these compounds will continue to decrease in the long term.

NMFS used the salmonid fishes toxicity data as a surrogate for green sturgeon and eulachon, as toxicity data for these two species was limited or non-existent, and because the salmonid fishes toxicity data sets were the best taxonomic data available (green sturgeon, eulachon, and salmonid fishes are in the same superorder: *Protacanthopterygii*). However, differences in the life history strategies and the certainty of similar toxic effects among species for all mechanisms and modes of action is not evident in the literature, so the results of the direct mortality population analysis for the salmonid fishes do not necessarily apply to green sturgeon and eulachon. Nonetheless, NMFS gives the benefit of the doubt to the listed species, and, based on the evidence considered in this opinion, NMFS expects that the stressors of the action to result in mortality (albeit an unquantifiable amount) of green sturgeon and eulachon. We further expect, based on the toxicity data, that the fitness of green sturgeon and eulachon will be reduced via sub-lethal effects (*i.e.*, interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis).

(2) Summary analysis on acute and chronic endpoints, chemical mixtures, population modeling, interactions with baseline environmental stressors, and fitness consequences associated with exposure to the proposed freshwater and saltwater criteria.

The summary analysis is a qualitative assessment of likely fitness consequences due to approval and implementation of each proposed criterion that considers:

- Acute and chronic toxicity data for the criteria compounds to listed species.
- The likelihood that listed species will encounter mixtures of multiple criteria chemicals in mixing zones due to the typical presence of these mixtures in wastewater and stormwater discharges under NPDES permits.
- The likelihood that listed species will encounter chemicals at concentrations greater than criteria concentrations due to overlapping mixing zones in some areas, and to environmental baseline stressors that add to the exposures.
- Results of the direct mortality population model
- The likely effects of interactions of the criteria compounds with other environmental baseline stressors (*e.g.*, high water temperature, other toxic substances)

The results of the summary analysis are given in Tables 2.7.1 and 2.7.2.

The summary analysis assesses the overall effects of approving the compounds listed in Table 1.1, individually and in combination with each other and with environmental baseline stressors, on the listed species considered in this opinion. In the summary analysis, we did not add up or otherwise mathematically combine its components. Rather, we applied best professional

judgment to characterize the intensity of adverse effects on individuals and populations of the listed species. We took this approach in large part because the available toxicity data for each compound varies significantly by quantity, test method, water source, life stage, *etc*. Therefore, we were not able to generate a mathematical expression or hazard quotient in the summary analysis, but did apply the qualitative results in the *Integration and Synthesis*.

Table 2.7.1. Results of the summary analysis on acute and chronic endpoints, chemical mixtures, environmental stressors, and fitness consequences associated with exposure to Oregon's freshwater criteria (empty cells = no data).

Stressor	Mortality	Growth	Behavioral	Cellular	Physiological	Biochemical	Reproductive	Sublethal	Bioaccumulation	Chemical Mixtures	Criteria Interactions with Environmental Baseline Stressors
Compound											
Aluminum	++++	+++	+++	++	+++					+++	+++
Ammonia	++++	++++		++	++++	++++				+++	+++
Arsenic	++	++	+		+					+++	+++
Lindane	++									+	+
Cadmium	++++	+++	++		++		++			+++	+++
Chromium (III)	++	+++								+++	+++
Chromium (VI)	+	+++								+++	+++
Copper	++++	++++	++++	++	+++		++++	++++		+++	+++
Dieldrin	++	++			++		+	+		++	++
Endosulfan-alpha	+++							++		+	+
Endosulfan-beta	+++							++		+	+
Endrin	+++			+	+		+			+	+
Heptachlor Epoxide	++							+		+	+
Lead	++	++	+++	+++	++	++	+			+++	+++
Nickel	++	+++								+++	+++
Pentachlorophenol	++	++								+++	+++
Selenium	++	++		+					+++	+++	+++
Silver	+++	++						++		+++	+++
Tributyltin	+++	++		++	++					+++	+++
Zinc	+++	+++		++	++		+++			+++	+++

\+ Low intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals
++ Moderate intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals
+++ Moderately-high-intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals, but not at the scale of any population
++++ High-intensity increase in toxicity effects on listed species that affects one or more population attributes

Table 2.7.2. Results of the summary analysis on acute and chronic endpoints, chemical mixtures, environmental stressors, and fitness consequences associated with exposure to Oregon's saltwater criteria (empty cells = no data).

Stressor	Mortality	Growth	Behavioral	Cellular	Physiological	Biochemical	Reproductive	Sublethal	Chemical Mixtures	Criteria Interactions with Environmental Baseline Stressors
Compound										
Arsenic	++							++	++	+++
Cadmium	++							++	++	+++
Chromium (VI)	++	+++							++	+++
Copper	++						++		++	+++
Endosulfan-alpha	++						+		+	+
Endosulfan-beta	++						+		+	+
Heptachlor Epoxide	+++							+	+	+
Lead	+++				+++		+		++	+++
Nickel	++							++	++	+++
Pentachlorophenol								+++	+	+++
Selenium	++							++	++	+++
Silver	++								++	+++
Tributyltin	++			++	+			++	++	+++
Zinc	++						++		++	+++

\+ Low intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals
++ Moderate intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals
+++ Moderately-high-intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals, but not at the scale of any population
++++ High-intensity increase in toxicity effects on listed species that affects one or more population attributes

(3) ESU/DPS-Specific Evaluations

The ESU/DPS-specific evaluations are an integration of the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model (when applicable), and the summary analysis. For each ESU or DPS, the evaluations are partitioned into six parts: (1) a summary of the acute and chronic toxicity data analysis on each species considered in this opinion, (2) a summary of the results of the direct mortality population model (when applicable), (3) an explanation of how effects of the proposed action are likely to affect productivity and abundance from multiple stressors, (4) a summary of how reductions in productivity and abundance are likely to affect the population attributes spatial structure and genetic diversity (when applicable), (5) a summary of effects associated with the freshwater and saltwater criteria that are likely to adversely affect critical habitat (when applicable) within the action area, and (6) conclusions on the listed species and critical habitat.

Furthermore, based on the summary analysis that we described earlier, certain compounds proposed by EPA are likely to have significant (high-intensity toxicological effects), long-term negative effects on one or more population attributes for the listed species considered in this opinion (Tables 2.7.1 and 2.7.2).

LCR Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; LCR Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 32 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all four modeling scenarios for each of the 32 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to

baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect LCR Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for LCR Chinook salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity for LCR Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for LCR Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of LCR Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (40.2 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of LCR Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of LCR Chinook salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UWR Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests

(uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; UWR Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 7 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 7 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect UWR Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for UWR Chinook salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UWR Chinook salmon through multiple mechanisms, including including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of UWR Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for UWR Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances,

and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of UWR Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of UWR Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of UWR Chinook salmon critical habitat such that it will not retain the current ability for the PCE water quality to serve the intended conservation role for the species for either survival or recovery.

UCR Spring-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; UCR spring-run Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 4 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 4 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect UCR spring-run Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for UCR spring-run Chinook salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UCR spring-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of UCR spring-run Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for UCR spring-run Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of UCR spring-run Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (30.8 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of UCR spring-run Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of UCR spring-run Chinook salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Spring/Summer-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SR SS-run Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 27 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 27 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SS-run Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for SS-run Chinook salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SS-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SS-run Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR SS-run Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SR SS-run Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE

water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (25.3 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery for SR SS-run Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SR SS-run Chinook salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Fall-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SR fall-run Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for the single SR fall-run Chinook salmon ESU (which consists of eight spawning populations). The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for the single SR fall-run Chinook salmon ESU (which consists of eight spawning populations).

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SR fall-run Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for SR fall-run Chinook salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat

quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SR fall-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SR fall-run Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR fall-run Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SR fall-run Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (25.3 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of SR fall-run Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SR fall-run Chinook salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

CR Chum Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; CR chum salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to

the freshwater acute criteria (one compound at a time). The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 17 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 17 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect CR chum salmon, and is likely to appreciably affect the VSP parameters productivity and abundance diversity of CR chum salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of CR chum salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of CR chum salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for CR chum salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of CR chum salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This

is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (26 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of CR chum salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of CR chum salmon critical habitat such that it will not retain the current ability for the PCE water quality to serve the intended conservation role for the species for either survival or recovery.

LCR Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; LCR coho salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 27 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 27 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect LCR coho salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for LCR coho salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in

abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of LCR coho salmon.

(5) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of LCR coho salmon.

SONCC Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SONCC coho salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 42 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 42 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SONCC coho salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for SONCC coho salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat

quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SONCC coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SONCC coho salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SONCC coho salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SONCC coho salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (37.8 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of SONCC coho salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SONCC coho salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

OC Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; OC coho salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to

the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 56 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 56 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect OC coho salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for OC coho salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of OC coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of OC coho salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for OC coho salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of OC coho salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of OC coho salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of OC coho salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Sockeye Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SR sockeye salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for the single SR sockeye salmon population. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for the single SR sockeye salmon population.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SR sockeye salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for SR sockeye salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SR sockeye salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of

environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SR sockeye salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR sockeye salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SR sockeye salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (34.5 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of SR sockeye salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SR sockeye salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

LCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; LCR steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 26 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 26 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect LCR steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for LCR steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of LCR steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for LCR steelhead. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of LCR steelhead. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (33 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of LCR steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of LCR steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UWR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; UWR steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 5 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 5 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect UWR steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for UWR steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UWR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of UWR steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR fall-run Chinook salmon.

Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of UWR steelhead. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of UWR steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of UWR steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

MCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; MCR steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 17 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 17 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream

temperatures), the proposed action is likely to adversely affect MCR steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for MCR steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of MCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of MCR steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR fall-run Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SR fall-run Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (75.7 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of MCR steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of MCR steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; UCR steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the

concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 4 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 4 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect UCR steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for UCR steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of UCR steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for UCR steelhead. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of UCR steelhead. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in

particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (30.8 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of UCR steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of UCR steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SRB Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SRB steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 24 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 24 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SRB steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for SRB steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in

abundance can negatively affect the spatial distribution and/or the genetic diversity of SRB steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SRB steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SRB steelhead. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SRB steelhead. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (34.5 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of SRB steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SRB steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species' for either survival or recovery.

Green Sturgeon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, and the summary analysis; green sturgeon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect green sturgeon, and is likely to appreciably affect the productivity and abundance for green sturgeon.

(3) The NMFS expects the stressors of the action to result in unquantifiable mortality of green sturgeon, and affect green sturgeon fitness via sub-lethal effects (*i.e.*, interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis).

(4) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for green sturgeon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of green sturgeon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events at the designation level. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (10.4 percent of the total designation).

(5) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of green sturgeon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of green sturgeon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

Eulachon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, and the summary analysis; eulachon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect eulachon, and is likely to appreciably affect the productivity and abundance for Eulachon.

(3) The NMFS expects the stressors of the action to result in unquantifiable mortality of Eulachon, and affect eulachon fitness via sub-lethal effects (*i.e.*, interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis).

(4) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for eulachon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of eulachon. In particular the PBF water quality, is unlikely to remain functional, *i.e.*, support associated life history events, at the designation level. This is based on the magnitude of likely effects on the PBF water quality (high-intensity increase in toxicity that affects one or more PBFs) and the overall percentage of critical habitat for this species that would be adversely affected (53.9 percent of the total designation).

(5) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of eulachon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of Eulachon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

Synthesis

Even though our predicted outcomes regarding the survival and recovery of the listed species considered in this opinion, as well the conservation value of their critical habitats, is based on the effects of the proposed action as a whole, our analysis is structured such that the proposed numeric criteria with the highest-intensity adverse toxicological and adverse biological effects on the listed species can be separated and identified. The multiple lines of evidence used in our analysis to identify the numeric criteria with the highest-intensity adverse toxicological and adverse biological effects include: the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis); the relative percent mortality analysis; the chemical mixtures analysis; the direct mortality population model; and the summary analysis. Table 2.7.3 provides a summary of the relative percent mortality analysis in section 2.6. Table 2.7.4 then provides a list of the proposed criteria that are likely to cause the highest-intensity adverse toxicological and adverse biological effects. Table 2.7.4 also shows which compounds, individually and in combination with other compounds and environmental stressors, are likely to reduce appreciably the likelihood of both the survival and recovery of the listed species, or reduce appreciably the conservation value of their critical habitat.

Table 2.7.3. Relative percent mortality analysis summary for freshwater acute criteria.

Compound	**Median LC_{50}**
Chromium VI	0.01
Pentachlorophenol	0.09
Lead	0.5
*Dieldrin	0.7
Arsenic	0.7
Nickel	1
*Lindane	1.5
*Heptachlor Epoxide	1.6
Selenium	1.8
Chromium III	3
Silver	3.4
Tributyltin	4.9
Zinc	5.1
*Endrin	5.4
Copper	7
Ammonia	8.6
Cadmium	12.7
*Endosulfan-alpha	13.9
*Endosulfan-beta	13.9
Aluminum	15

*Legacy compounds.

Table 2.7.4. Findings as to whether compounds associated with significant adverse toxicological and biological effects on the listed species considered in this opinion that, individually and in combination with exposure to multiple compounds and stressors, are likely to reduce appreciably the likelihood of both the survival and recovery (S/R), and are likely to reduce appreciably the conservation value (CV) of their critical habitat.

Stock	Cadmium (Acute)	Aluminum (Acute and Chronic)	Ammonia (Acute and Chronic)	Copper (Acute and Chronic)
LCR Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
UWR Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
UCR spring-run Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SR spring/summer-run Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SR fall-run Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
CR Chum Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
LCR Coho Salmon	S/R	S/R	S/R	S/R
SONCC Coho Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
OC Coho Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SR Sockeye Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
LCR Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
UWR Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
MCR Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
UCR Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SRB Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
Green Sturgeon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
Eulachon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SR Killer Whales	*S/R determination is based on a long-term, permanent reduction in primary prey—Chinook salmon			

2.8 Southern Resident Killer Whales—Effects Analysis

The best available information indicates that salmon are the primary prey of Southern Residents year round (Section 2.4), including in coastal waters, and that the whales predominantly consume Chinook salmon, likely including Oregon salmon stocks. Based on coded wire tag recoveries, Oregon salmon stocks are available to Southern Residents across their coastal range (Weitkamp 2010). The proposed action has the potential to affect Southern Residents indirectly by reducing prey quality, increasing toxic chemicals in the whales, and reducing availability of Chinook salmon. A decrease in the quality and availability of salmon, and Chinook salmon in particular, and an increase of toxic chemicals in individual whales, may adversely affect the entire DPS of Southern Resident killer whales.

In this analysis, NMFS considers effects of the proposed action on the Southern Residents by qualitatively evaluating the reduction of prey quality caused by the action as well as the potential accumulation of toxic chemicals in the whales, and the reduction of prey availability.

Effects of Reduced Prey Quality and Toxic Chemical Accumulation in the Southern Resident Killer Whales

The NMFS anticipates increased contaminant loading in Chinook salmon, as described above, and therefore also anticipates reduced prey quality and subsequent toxic chemical accumulation in the Southern Residents. First, we briefly review the mechanisms for reduced prey quality and then discuss the anticipated resulting accumulation of toxic chemicals in the whales.

Reduced Prey Quality

The quality of Chinook salmon is likely influenced by a variety of factors including size of the fish and the contaminant load. In addition to the anticipated fish mortality (as described in section 2.6.5), some toxic chemicals can cause sub-lethal effects such as a reduction in growth, a common stress response observed in fish (review in section 2.6.7). Because Southern Residents consume mostly large Chinook salmon (review Status of the Species), a reduction in fish growth could affect the foraging efficiency of Southern Resident killer whales. However, the degree to which reduced fish growth could affect Southern Resident foraging is unknown. When compared to current conditions, approval of the proposed criteria will result in reduced pollutant loading and reduced body burden of contaminants in fishes. Nonetheless, the proposed water quality standards will continue to increase mass loading of toxic substances in the Southern Residents' primary prey with implications for toxic chemical accumulation in the whales, as discussed below.

Toxic Chemical Accumulation in the Southern Residents

The NMFS evaluated the effects of toxic chemical accumulation qualitatively. We reviewed the best available information about the bioaccumulation, biomagnification, concentration levels in the whales, and toxicity of the compounds in Table 1.1 (as introduced earlier), which are: aluminum, ammonia, arsenic, cadmium, chromium (III and VI), copper, dieldrin, endosulfan

(alpha and beta), endrin, heptachlor epoxide, lead, lindane, nickel, PCP, selenium, silver, TBT, and zinc.

In many cases the best available information was limited. For example, there is limited information about the levels of these compounds in the environment or in the whales, and no information about chemical toxicity specifically in Southern Residents. Where there was no data on chemical levels in Southern Residents, we considered levels in other marine mammals to estimate the potential extent of bioaccumulation in the Southern Residents. This literature review helped us put in context the potential killer whale health effects from the proposed water quality criteria. First, we identified the compounds in Table 1.1 that were not anticipated to cause adverse health effects in the Southern Residents. Second, we identified the compounds in Table 1.1 that may cause adverse health effects in the Southern Residents.

Compounds with No Anticipated Health Effects. The available data indicate that Southern Residents are not at risk of health effects from aluminum, ammonia, nickel, selenium, silver, zinc, and PCP. Some of these compounds are essential elements to the nutrition of marine mammals (*e.g.*, aluminum, nickel, selenium, and zinc; Das *et al.* 2003) and are generally found in low levels in marine mammals distributed throughout the world's oceans (see Appendices 10-5 to 10-8 in O'Shea 1999 for summaries of selected surveys of metals and trace element concentrations in tissues of seals, sea lions, toothed whales, baleen whales, sea otters, dugongs, manatees, and polar bears). Therefore, these essential elements found in low concentrations in marine mammals distributed globally are not anticipated to cause adverse health effects for Southern Resident killer whales. Although silver is not considered an essential element for mammals, its toxicity is generally not a concern and it has not been measured often in marine mammals (O'Hara *et al.* 2003). Ammonia does not build up in the food chain, but serves as a nutrient for plants and bacteria (EPA 2003) and is not anticipated to accumulate in the whales. PCP is an organochlorine pesticide that does not readily bioaccumulate. When found in marine mammals, its presence is likely the result of biotransformation of other chemicals and not bioaccumulation (*e.g.*, as observed in bowhead whales, Hoekstra *et al.* 2003). Furthermore, PCP readily degrades in the environment and by all available evidence does not appear to biomagnify (Garrett and Ross 2010). The NMFS does not anticipate that the proposed action will affect accumulation of PCPs in Southern Residents. For these reasons, NMFS does not anticipate that the proposed action will result in any health effects from these compounds and we do not discuss these compounds further.

Compounds that May Cause Adverse Health Effects. In order to evaluate effects of these remaining compounds, we first review the current levels measured in the blubber of Southern Residents (or in surrogate marine mammals if data are unavailable for Southern Residents), and compare levels to health effect thresholds found for surrogate species. We then consider the effects the proposed criteria will have on the whales' levels over time.

Long-lived, upper trophic-level predators, such as the Southern Residents, are susceptible to compounds that biomagnify because even low concentrations in the prey can accumulate and magnify to high concentration levels in the predators. Bioaccumulative compounds that have the potential to biomagnify are likely to pose the greatest health risks to the Southern Residents. Therefore, we evaluate the effects of compounds that may bioaccumulate but are not anticipated

to biomagnify separate from the compounds that may bioaccumulate and biomagnify. These steps are described in more detail below: (1) identify the compounds that may bioaccumulate (or increase in concentration in an individual) but are not anticipated to biomagnify (or not anticipated to increase in concentration up the food chain), (2) identify the compounds that may bioaccumulate and biomagnify, and compare the concentrations of these compounds in the Southern Residents or in surrogate species to known health effects levels in surrogate species, and (3) put the effects of the proposed action in context by comparing the existing numeric criteria with the proposed numeric criteria, and evaluating the anticipated trend in the Southern Residents' long-term bioaccumulation.

Compounds that may bioaccumulate but are not anticipated to biomagnify. Metals can bioaccumulate in the aquatic environment (EPA 2007). However, most metals (with the exception of methylmercury), do not appear to biomagnify and are regulated and excreted (Gray 2002, EPA 2007). As discussed in section 2.6.1., arsenic, cadmium, chromium, copper, and lead do not appear to biomagnify. Therefore, NMFS anticipates that these metals will not biomagnify in the Southern Residents.

Upper trophic-level predators can still accumulate metals even in the absence of biomagnification (Reinfelder *et al.* 1998). However, low levels of arsenic, chromium, copper, and lead have been measured in marine mammal tissues (O'Shea 1999, Grant and Ross 2002, Das *et al.* 2003). Although high cadmium levels are measured in some marine mammals, cadmium is known to combine with metallothionein (a protein molecule) to mitigate the toxic effects (Dietz *et al.* 1998, Klaassen *et al.* 2009). Further, no toxic effects of cadmium have been observed in marine mammals. Although threshold levels at which adverse health effects occur are currently unknown for these metals, the available data indicate that the low levels measured in their tissues do not pose a health risk to marine mammals (O'Shea 1999).

Compounds that may bioaccumulate and biomagnify. The remaining compounds with proposed criteria are the organic pollutants that have the ability to biomagnify up the food chain. These compounds are dieldrin, endosulfan, endrin, heptachlor epoxide, lindane, and TBT. The best available data indicate that Southern Residents (or surrogate species) have relatively low concentration levels of these compounds (see the Status of the Species). In contrast, the Southern Residents have higher levels of the legacy organochlorines, PCBs and DDTs, and the emerging PBDEs[9].

At certain concentrations, dieldrin, endosulfan, endrin, heptachlor epoxide, lindane, and TBT can have a wide variety of toxic effects on organisms including neurotoxicity, reproductive defects, tremors and convulsions, organ tissue damage (*e.g.*, liver or kidney tissue damage), cancer, endocrine disruption, and reduced immune response (see the Status of the Species). Here we compare the concentrations of these compounds in the Southern Residents or in surrogate species to known threat levels found in surrogate species. There are currently no known killer whale-specific health effects thresholds, thereby requiring the use of surrogate species to estimate risks. There are several different types of threat levels or measures of toxicity used in laboratory studies. A median lethal dose, LD_{50}, is the dose required to kill half the tested population in 2 weeks and generally indicates a substance's acute toxicity. In contrast, a Lowest Observable

[9] PCBs, DDTs, and PBDEs are not among the proposed criteria in the current action.

Adverse Effect Level (LOAEL) is the smallest dose that causes a detectable adverse effect typically measured when assessing chronic toxicity. Additionally, a No Observable Adverse Effect Level (NOAEL) is the highest dose at which no adverse effects occur. Dieldrin, endosulfan, endrin, heptachlor epoxide, lindane, and TBT levels in Southern Residents and surrogate marine mammals are below the threat levels (*e.g.*, LD_{50}, NOAEL, LOAEL) in laboratory species from different studies identified in Table 2.8.1. For example, alpha endosulfan levels determined in the blubber of Southern Residents were below the limits of quantification (< 2.2 - < 14 ng/g wet weight). This average level is substantially below the NOAEL found for rats and grey partridge at 2,400 to 40,000 ng/g wet weight, respectively (see Table 2.6.9.1). Therefore, we anticipate that the Southern Residents' current levels of these compounds do not pose a health threat to the whales.

Table 2.8.1 Measured concentration levels in marine mammals compared to threat levels found in laboratory species.

	Current Levels		**Threat Levels**		
Compound	Measured Concentration/Species (ng/g wet weight)	Reference	Concentration (ng/g wet weight)	Species	Reference
Dieldrin	9.2 – 440 / Southern Residents	1	25,000 - 168,000	2 week-old rats	7
Endosulfan	< 2.2 - < 14 / Southern Residents	1	40,000	grey partridge	8; 9
			2,400	rat	10; 9
Endrin	ND - 12.7 (µg/g lipid) / blue and humpback whales	2	25	dog	11
Heptachlor epoxide	5.3 – 660 / Southern Residents	1	195,000-250,000 (ng/g bw)	rat	12
Lindane	< 1.9 – 17 / Southern Residents	1	0.3 ng/g/day	rat	13
TBT	100/killer whales	3	>10,000	Dall's porpoise	14
	180/ killer whales	4	> 120	rat* and rabbit**	15*; 16**
PCB	1,306 -39,420 / Southern Residents	5, 6	100-200 (dietary NOAEL & LOAEL)	seals and dolphins	17
DDT	426 - 35,040 / Southern Residents	5, 6	50,000 ng/g/day	mallard	18
PBDE	199 -2,745/ Southern Residents	5, 6	170-460 ng/g lw in blubber	grey seal	19

ND = non detect, lw = lipid wet References: (1) G. Ylitalo NWFSC, pers. comm.; (2) Metcalfe *et al.* 2004; (3) Kannan *et al.* 1997; (4) Tanabe *et al.* 1998; (5) Krahn *et al.* 2007a; (6) Krahn *et al.* 2009; (7) EPA 2003; (8) Sample *et al.* 1996; (9) Small and Solomon 2005; (10) USEPA 2005, as cited in Small and Solomon 2005; (11) FAO/WHO 1971; (12) Heptachlor epoxide fact sheet CAS Number: 1024-57-3; (13) USEPA 1999; (14) Kim *et al.* 1998; (15) Snoeij *et al.* 1986; (16) Elferink *et al.* 1986; (17) Kannan *et al.* 2000; (18) Tucker and Crabtree 1970) ; (19) Hall *et al.* 2003.

Comparison Between Existing Criteria and Proposed Criteria and the Resulting Trend in Long Term Accumulation in Southern Residents

In this section, we put the effects of the proposed action in context by comparing the existing numeric criteria with the proposed numeric criteria (see Table 2.8.1), and evaluating the resulting trend in long term bioaccumulation in the Southern Residents. As discussed above, several compounds (*i.e.*, arsenic, cadmium, chromium, copper, and lead) are not anticipated to biomagnify, are likely to be low in concentration in the Southern Residents, and are not currently toxic. The proposed numeric criteria for arsenic, cadmium, and chromium (III) are likely to result in less accumulation in the Southern Residents than with the existing numeric criteria (see Table 2.8.2). The proposed numeric criteria for chromium (VI) will not change from the existing criteria, and therefore we assume the accumulation of chromium (VI) in the whales will remain the same. Lastly, the proposed criteria for copper and lead are more strict for freshwater and less strict for saltwater. Given that copper and lead are not likely to biomagnify, we do not anticipate that a small increase of these compounds in saltwater will cause a measurable increase in concentration in the whales. Therefore, we anticipate that approval of the proposed criteria for these compounds will not increase the potential for adverse health effects in the whales.

The proposed numeric criteria for the bioaccumulative compounds that biomagnify (*e.g.*dieldrin, endosulfan, endrin, heptachlor epoxide, lindane, and TBT) are likely to result in less accumulation than with the existing numeric criteria (see Table 2.8.2). For example, several of these compounds (*e.g.*, endosulfan, heptachlor epoxide, and TBT) were previously unregulated. Although dieldrin and endrin have both more strict and less strict proposed criteria, the exposure of dieldrin and endrin will be from past usage since they have been banned for 20 to 30 years. Dieldrin and endrin could theoretically be in surface waters, however, occurrence will be very minimal as these compounds strongly adhere to sediment (as previously discussed). Overall, accumulation of these compounds will be either reduced, or the same, and is not a health concern. Therefore, we anticipate that approval of the proposed criteria for these compounds will either not change accumulation or potential health effects or, in some cases may reduce accumulation and the risk of health effects in the whales.

Table 2.8.2. Resulting accumulation in the Southern Resident killer whales from the proposed changes in the numeric criteria.

Compound	Change in Criteria				Accumulation in Whales
	Freshwater		Salwater		
	Acute	Chronic	Acute	Chronic	
Arsenic	decrease	decrease	same	same	decrease
Cadmium	decrease	decrease	decrease	decrease	decrease
Chromium (III)	decrease	decrease			decrease
Chromium (VI)	same	same	same	same	same
Copper	decrease	decrease	increase	increase	same
Dieldrin	decrease	increase			decrease
Endosulfan (-a,-b)	prev. unreg.	prev. unreg.	prev. unreg.	prev. unreg.	decrease
Endrin	decrease	increase			decrease
Heptachlor epoxide	prev. unreg.	prev. unreg.	prev. unreg.	prev. unreg.	decrease
Lead	decrease	decrease	increase	increase	same
Lindane	decrease				decrease
TBT	prev. unreg.	prev. unreg.	prev. unreg.	prev. unreg.	decrease

In summary, when compared to current conditions, the proposed criteria will result in reduced bioaccumulation and biomagnification in the Southern Residents. Based on the best available information, we anticipate that the currently low concentrations of bioaccumulative compounds in the whales will remain low, and that these levels are substantially lower than threat levels found in surrogate species and are not anticipated to pose a risk to the Southern Residents.

Effects of Reduced Prey Availability

We rely on the salmon determinations to ensure that the proposed action does not appreciably reduce the likelihood of survival and recovery of the Southern Residents in the long term. Later in this opinion, NMFS concludes that the proposed action is likely to appreciably reduce the likelihood of survival and recovery of the UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon, green sturgeon, and Eulachon. In other words, the proposed action appreciably increases the risk of extinction of these listed species.

Our analysis focused on the short- and long-term reductions in Chinook salmon available to the whales as a result of the proposed action. Below we discuss the effects from (1) the short-term or annual reduction in Chinook salmon stocks, and (2) the long-term appreciable reduction in the likelihood of survival and recovery of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon.

Short-term or annual reduction in Chinook stocks

Mortality of Chinook could affect the annual prey availability to the whales where the marine ranges of the affected Chinook stocks and the whales overlap. Mortality of adult Chinook salmon could affect the quantity of prey available to the whales in a given year, whereas mortality of juvenile Chinook salmon could affect prey availability in future years. Juvenile mortality from exposure to the compounds in Table 1.1 translates to the effective loss of only a few adult-equivalent Chinook salmon from a variety of runs three to five years after the juvenile mortality occurred (*i.e.*, by the time these juveniles would have grown to be adults and available prey of killer whales). This reduction would occur each year that the proposed criteria remain in place.

Given the total quantity of prey available to Southern Resident killer whales throughout their range, this annual reduction in prey is extremely small, and although measurable, the percent reduction in prey abundance is not anticipated to be different from zero by multiple decimal places (based on NMFS' previous analyses of the effects of salmon harvest on Southern Residents; *e.g.*, NMFS 2008e, NMFS 2011). Because the annual reduction is so small, there is also a low probability that any of the juvenile Chinook salmon killed from implementation of the proposed action would be intercepted by the killer whales across their vast range in the absence of the proposed action. Therefore, NMFS anticipates that the short-term reduction of Chinook salmon would have an insignificant effect on Southern Resident killer whales.

Long-term appreciable reduction in the likelihood of survival and recovery of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon

NMFS qualitatively evaluated long-term effects on the Southern Residents from the anticipated appreciable reduction in the likelihood of survival and recovery of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon. We assessed the likelihood for localized depletions, and long-term implications for Southern Residents' survival and recovery, resulting from the increased risk of extinction of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon. In this way, NMFS can determine whether the increased likelihood of extinction of prey species is also likely to appreciably reduce the likelihood of survival and recovery of Southern Residents.

A reduction in prey would occur over time as abundance declined for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon. Hatchery programs, which account for a portion of the production of these ESUs, may provide a short-term buffer, but it is uncertain whether hatchery-only stocks could be sustained indefinitely. The total 5-year geometric mean abundance for the 5 ESUs (UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and the SR fall-run Chinook salmon) is 128,534 total spawners. The loss of these ESUs would also preclude the potential for their future recovery to healthy, more substantial numbers. Fewer populations contributing to Southern Residents' prey base will reduce the representation of diversity in life histories, resiliency in withstanding

stochastic events, and redundancy to ensure there is a margin of safety for the salmon and Southern Residents to withstand catastrophic events.

The long-term reduction of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon can lead to nutritional stress in the whales. Nutritional stress can lead to reduced body size and condition of individuals and can also lower reproductive and survival rates. Prey sharing would distribute more evenly the effects of prey limitation across individuals of the population that would otherwise be the case. Therefore, poor nutrition from the reduction of prey could contribute to additional mortality in this population. Food scarcity could also cause whales to draw on fat stores, mobilizing contaminants stored in their fat and affecting reproduction and immune function.

Differences in adult salmon life histories and locations of their natal streams likely affect the distribution of salmon across the Southern Residents' coastal range. The continued decline and potential extinction of the UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon, and consequent interruption in the geographic continuity of salmon-bearing watersheds in the Southern Residents' coastal range, is likely to alter the distribution of migrating salmon and increase the likelihood of localized depletions in prey, with adverse effects on the Southern Residents' ability to meet their energy needs. A fundamental change in the prey base originating from Oregon is likely to result in Southern Residents abandoning areas in search of more abundant prey or expending substantial effort to find depleted prey resources. This potential increase in energy demands should have the same effect on an animal's energy budget as reductions in available energy, such as one would expect from reductions in prey.

In summary, approval of the numeric criteria listed in Table 1.1 in the long term will increase the likelihood of extinction of the Chinook salmon stocks which will appreciably reduce the likelihood of survival and recovery of the Southern Resident killer whales.

2.8.1. Integration and Synthesis: Southern Resident Killer Whales.

Based on the analysis of the acute and chronic toxicity data, the results of the summary analysis, and the predicted long-term effects on UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, and LCR Chinook salmon, the proposed action is likely to affect the productivity and abundance, spatial distribution, and affect the long-term viability of Southern Resident killer whales.

Several factors identified in the final recovery plan for Southern Resident killer whales may be limiting recovery. These are quantity and quality of prey, toxic chemicals that accumulate in top predators, and disturbance from sound and vessels. Oil spills are also a risk factor. It is likely that multiple threats are acting together. For example, reduction in prey availability makes it harder for the whales to locate and capture prey, which can cause them to expend more energy and catch less food. Although it is not clear which threat or threats are most significant to the survival and recovery of Southern Residents, all of the threats are important to address.

The Southern Resident killer whale DPS is composed of one small population (88 whales) which is currently at most half of its likely previous size (140 to as many as 400 whales). The effective population size (based on the number of breeders under ideal genetic conditions) of 26 whales is very small, and this in combination with the absence of gene flow from other populations may elevate the risk from inbreeding and other issues associated with genetic deterioration. This population has a variable growth rate (28-year mean=0.3% ± 3.2% s.d), and risk of quasi extinction that ranges from 1% to as high as 66% over a 100-year horizon, depending on the population's survival rate and the probability and magnitude of catastrophic events. Because of this population's small size, it is susceptible to demographic stochasticity and genetic deterioration, as described in the Status of the Species. The influences of demographic stochasticity and potential genetic issues in combination with other sources of random variation combine to amplify the probability of extinction, known as the extinction vortex.

The larger the population size, the greater the buffer against stochastic events. It also follows that the longer the population stays at a small size, the greater its exposure to demographic stochastic risks and genetic risks. In addition, as described in the Status of the Species section, small populations are inherently at risk because of the unequal reproductive success of individuals within the population. The more individuals added to a population in any generation, the more chances of adding a reproductively successful individual. Random chance can also affect the sex ratio and genetic diversity of a small population, leading to lowered reproductive success of the population as a whole. For these reasons, the failure to add even a few individuals to a small population in the near term can have long-term consequences for that population's ability to survive and recover into the future. A delisting criterion for the Southern Resident killer whale DPS is an average growth rate of 2.3% for 28 years (NMFS 2008a). In light of the current average growth rate of 0.3%, this recovery criterion and the risk of stochastic events and genetic issues described above underscore the importance for the population to grow quickly.

The effects of the proposed action include bioaccumulation, biomagnification, and reduced prey quality and quantity. As explained in the section [*Toxic Chemical Accumulation in the Southern Residents*], compared to current conditions, the proposed criteria will result in the same levels for some compounds and reduced bioaccumulation and reduced biomagnification in the Southern Residents for some compounds. The NMFS anticipates that the relatively low concentrations of the bioaccumulative compounds in the whales will remain low and below health effects thresholds found in surrogate species. For these reasons, NMFS anticipates that the effects of the proposed action on the accumulation of the toxic chemicals in Southern Residents will be insignificant.

As explained in the section *Effects of Prey Reduction*, the anticipated short-term reduction of Chinook salmon associated with the proposed action would result in an insignificant annual reduction in adult equivalent prey resources for Southern Resident killer whales.

Over the long-term, however, the proposed action will increase the risk of extinction of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, and LCR Chinook salmon stocks and could result in a greater reduction in prey quantity and affect availability of prey in other ways (*i.e.*, spatially or temporally). Fewer populations contributing to Southern Residents' prey base will reduce the representation of

diversity in life histories, resiliency in withstanding stochastic events, and redundancy to ensure there is a margin of safety for the salmon and Southern Residents to withstand catastrophic events. These reductions increase the extinction risk of Southern Residents.

The extinction of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, and LCR Chinook salmon would reduce prey availability and increase the likelihood for local depletions of prey in particular locations and times. In response, the Southern Residents would increase foraging effort or abandon areas in search of more abundant prey. Reductions in prey or a resulting requirement of increased foraging efficiency increase the likelihood of physiological effects. The Southern Residents would likely experience nutritional, reproductive, or other health effects (*e.g.*, reduced immune function from drawing on fat stores and mobilizing contaminants in the blubber) from this reduced prey availability. These effects would lead to reduced body size and condition of individuals and can also lower reproductive and survival rates and thereby diminish the potential for Southern Residents to recover.

In summary: (1) The toxic chemicals discussed in this opinion have the ability to accumulate in the Southern Residents, however, bioaccumulation and biomagnification is expected to be relatively low, and levels in the whales are not anticipated to cause health effects. Furthermore, the proposed criteria will result in reduced bioaccumulation and biomagnifications of some compounds and levels will remain low and below health effects thresholds in the Southern Residents. (2) Short-term (or annual) reduction in prey availability associated with the proposed action would result in an insignificant annual reduction in adult equivalent prey resources for Southern Resident killer whales. (3) Increased risk of extinction of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, as a long-term consequence of the proposed action increases the risk of a permanent reduction in prey available to Southern Residents, and increases the likelihood for local depletions of prey in particular locations and times. (4) Losing the potential for future recovery of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, appreciabley diminishes the potential for Southern Residents to recover.

2.9 Conclusion

After reviewing the best available scientific and commercial information regarding the biological requirements and the status of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, Eulachon and Southern Resident killer whales considered in this opinion (section 2.4), the environmental baseline (section 2.5) for the action area, the effects of the proposed action (section 2.6), and the cumulative effects (section 2.6.8), NMFS concludes that the proposed action is likely to jeopardize the continued existence of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon,

SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, Eulachon, and Southern Resident killer whales.

Furthermore, NMFS has determined NMFS has determined that the proposed action will result in the destruction or adverse modification of critical habitat as a result of degraded water quality in Oregon for LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, and Eulachon.

2.10. Reasonable and Prudent Alternative

This opinion has concluded that the proposed action will jeopardize the continued existence of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, eulachon, and Southern Resident killer whales.

This opinion also concluded that the proposed action will destroy or adversely modify critical habitat for LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, and eulachon.

Therefore, NMFS must discuss with EPA the availability of reasonable and prudent alternatives (RPAs) that EPA can take to avoid violation of EPA's ESA section 7(a)(2) responsibilities (50 CFR 402.14(g)(5)). RPAs are alternative actions identified during formal consultation that: (1) can be implemented in a manner consistent with the intended purpose of the action, (2) can be implemented consistent with the scope of the Federal agency's legal authority and jurisdiction, (3) are economically and technologically feasible, and (4) that NMFS believes would avoid the likelihood of jeopardizing the continued existence of listed species or resulting in the destruction or adverse modification of critical habitat (50 CFR 402.02).

This section presents EPA with an RPA that will avoid jeopardy and destruction or adverse modification of critical habitat, while meeting the requirements listed above. Because this opinion has found jeopardy and destruction or adverse modification of critical habitat, the EPA is required to notify NMFS of its final decision on the implementation of the reasonable and prudent alternative.

2.10.1 Proposed RPA

The NMFS identified seven criteria (*i.e.*, copper [acute and chronic], ammonia [acute and chronic], cadmium [acute], and aluminum [acute and chronic])—that would cause significant adverse toxicological and biological effects on the listed species considered in this opinion. Individually and in combination with exposure to multiple compounds and stressors, these

criteria are likely to reduce appreciably the likelihood of both the survival and recovery of the listed species, and are likely to reduce appreciably the conservation value of their critical habitats.

The NMFS and the EPA considered a variety of alternatives to avoid jeopardy and destruction or adverse modification of critical habitat to the listed species considered in this opinion. Based on the best available information, NMFS and EPA were able to identify alternative numeric criteria for three of the seven criteria (acute and chronic copper, chronic ammonia). The alternative criteria are supported by both the best available information considered in this opinion as well as recent reanalysis conducted by EPA under the CWA.[10] These criteria will avoid jeopardy/adverse modification and are also within EPA's authority to implement.

For the remaining four criteria found to result in jeopardy/adverse modification, discussions between NMFS and EPA about the availability of an RPA that meets the regulatory criteria did not result in revised numeric criteria. Instead, the RPA specifies biological requirements to satisfy the conservation needs of the affected species and specific parameters EPA must work within to derive criteria that meet those requirements and avoid jeopardy and adverse modification of critical habitat.

Copper

Acute. The EPA shall disapprove the State of Oregon's acute criterion of 13 µg/L at 100 mg/L $CaCO_3$ for freshwater copper.

The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, a new acute criterion of 2.3 µg/L for freshwater copper using EPA's 2007 BLM-based aquatic life criteria. The EPA will ensure that the new acute copper criterion will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Chronic. The EPA shall disapprove the State of Oregon's chronic criterion of 9 µg/L at 100 mg/L $CaCO_3$ for freshwater copper.

The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, a new chronic criterion of 1.45 µg/L for freshwater copper using EPA's 2007 BLM-based aquatic life criteria. The EPA will ensure that the new chronic copper criterion will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Ammonia

Acute. The EPA shall use the Process for Deriving Criteria, specified below, to derive an acute criterion for freshwater ammonia at pH 8 and 20°C (total ammonia-N). The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, the derived

[10]http://water.epa.gov/scitech/swguidance/standards/criteria/aqlife/pollutants/copper/upload/2009_04_27_criteria_copper_2007_criteria-full.pdf

acute ammonia criteria. The EPA will ensure that the derived acute ammonia criteria will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Chronic. The EPA shall disapprove the State of Oregon's chronic criterion of 1.7 mg/L at pH 8 and 20°C for freshwater ammonia (total ammonia-N).

The EPA shall recommend that the State of Oregon maintain the current chronic criterion of 0.76 mg/L at pH 8 and 20°Cfor freshwater ammonia (total ammonia-N).

Cadmium

Acute.The EPA shall disapprove the State of Oregon's acute criterion of 2.0 µg/L at 100 mg/L $CaCO_3$ for freshwater cadmium.

The EPA shall use the Process for Deriving Criteria, specified below, to derive an acute criterion for the State of Oregon for freshwater cadmium. The EPA shall recommend that the State adopt, and EPA will promulgate if necessary, the derived acute cadmium criteria. The EPA will ensure that the derived acute ammonia criteria will be effective within 24 months after EPA's final to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Aluminum[11]

Acute. The EPA shall disapprove the State of Oregon's acute criterion of 750 µg/L at pH 6.5-9.0for freshwater aluminum.

The EPA shall use the Process for Deriving Criteria, specified below, to derive an acute criterion for the State of Oregon for freshwater aluminum at pH 6.5-9.0. The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, the derived acute aluminum criteria. The EPA will ensure that the derived acute aluminum criteria will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Chronic. The EPA shall disapprove the State of Oregon's chronic criterion of 87 µg/L at pH 6.5-9.0for freshwater aluminum.

[11] On August 9, 2012, EPA sent NMFS a letter withdrawing their request for consultation on Oregon's acute and chronic aluminum criteria as "EPA has determined that the BE submitted to NMFS in January 2008 incorrectly described the proposed federal action under consultation for aluminum (*i.e.*, CW A § 303(c)(3) approval of Oregon's submission of aluminum criteria). Specifically, Oregon's submitted description of the pollutant refers to aluminum in waters with a pH of 6.5- 9.0, but a footnote in the criterion itself indicates that the criterion is meant to apply to waters with pH less than 6.6 and hardness less than 12 mg/L (as $CaCO_3$)." Due to the court-ordered deadline of August 14, 2012, NMFS did not have time to modify its opinion to exclude acute and chronic aluminum from the document. The NMFS acknowledges EPA's revision to the proposed action, however, and notes it does not anticipate EPA will carry out the RPA for aluminum in light of this change. The NMFS will await a further request from EPA relating to EPA's potential future actions regarding Oregon's aluminum criteria.

The EPA shall use the Process for Deriving Criteria, specified below, to derive a chronic criterion for the State of Oregon for freshwater aluminum at pH 6.5-9.0. The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, the derived chronic aluminum criteria. The EPA will ensure that the derived chronic aluminum criteria will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Process for Deriving Criteria

The EPA shall utilize analytical methods that meet specified requirements to derive numeric criteria for aquatic life, taking into account the same factors that NMFS did in completing its analysis for the other criteria in this opinion. The EPA will then evaluate the analytical results with a population model that meets the requirements set out below, and thus is equivalent to that used by NMFS in this opinion, to confirm that the derived criteria will not jeopardize listed fish or adversely modify their critical habitat.

In particular, the EPA shall derive criteria for acute ammonia, acute cadmium, and acute and chronic aluminum in compliance with the following five requirements:

1) Only use toxicity data for ammonia, cadmium, and aluminum that is specific to salmonid fishes (if new information becomes available for these compounds for green sturgeon and eulachon, then EPA shall include this data in its analysis);
2) All toxicity data used to derive the numeric criteria must be curve-fitted, where the literature provides the necessary data to perform this step;
3) When available, the curve-fitted toxicity data must be used to extrapolate threshold acute and chronic toxic effect concentrations;
4) Derived criteria must be model-adjusted to account for chemical mixtures; and,
5) An appropriate population model must be applied to the derived criteria, and must predict no negative change in the intrinsic population growth rate (*e.g.*, lambda, λ).

More specifically, EPA shall ensure that the derived criteria are developed in compliance with the following mandatory sideboards:

- The EPA shall use toxicity data specific to salmonid fishes. The EPA shall use the acute and chronic toxicity data in this opinion as a minimum data set. For green sturgeon and eulachon, EPA shall use the salmonid fishes toxicity data for this analysis, as described in section 2.6.2 in this opinion, in addition to any new data that becomes available for green sturgeon and eulachon.
- The EPA shall use toxicity data based on exposure-response curves and fixed durations toxicity tests to estimate acute and chronic toxic effect thresholds to assess effects on multiple life stages and multiple endpoints, to include at a minimum: mortality, latent mortality, reproduction, growth, physiological, cellular, behavioral, and biochemical effects, where the data exists. The EPA may use existing toxicity data for ammonia, cadmium, and aluminum or generate new data, but the data shall be curve-fitted (see Figure 2.6.1.1) to determine the minimum effect thresholds (*e.g.*, 5%) at which acute and chronic toxic effects are predicted. The minimum effects thresholds shall be used to

derive the criteria instead of using the EPA acute adjustment factor or the acute-to-chronic ratio to derive criteria.

- The EPA shall ensure that each derived criterion for ammonia, cadmium, and aluminum is adjusted to account for chemical mixtures using a concentration–addition model or response-addition model to determine whether or not exposure to multiple compounds will result in additive effects to the listed species considered in this opinion. The concentration–addition model or response-addition model shall include all compounds listed in Table 1.1. If the mixture effects prediction is greater than one, EPA shall adjust the concentrations for ammonia, cadmium, and aluminum until the mixture effects prediction is less than one.
- The EPA shall ensure that the derived criteria for ammonia, cadmium, and aluminum do not result in a negative change in the intrinsic population growth rate based on the geometric mean abundance data for each life history type, *i.e.*, coho salmon (*O. kisutch*), sockeye salmon (*O. nerka*) and ocean-type and stream-type Chinook salmon (*O. tshawytscha*), of salmonid fish considered in this opinion, at the population scale. The EPA shall use stream-type Chinook salmon as a surrogate for steelhead, and ocean-type Chinook salmon as a surrogate for chum salmon in the population model, as described in section 2.6.5.1 of this opinion. Pacific salmon and steelhead abundance data is available from the Northwest Fisheries Science Center Salmon Population Summary Database[12] or from the Columbia Basin Fish and Wildlife Authority Status of the Fish and Wildlife Resources Database[13]. The abundance data used for the population growth rate analysis shall include data from all years with available abundance data. For green sturgeon and eulachon, EPA shall use the salmonid fishes toxicity data and modeling results as surrogate data and outputs for this analysis.
- To ensure that the derived numeric criteria for ammonia, cadmium, and aluminum meet the population growth rate condition of the RPA, EPA shall run the criteria for ammonia, cadmium, and aluminum through a population model (*e.g.*, Leslie Matrix), parameterized for Pacific salmonid fishes. Model requirements include: (1) scenarios based on change in first year survival; (2) an assumption that the populations are density-independent, to reduce the probability of Type II errors; (3) sigmoid slopes are generated from the data used to derive the numeric criteria, and if a slope cannot be generated from the data, EPA shall use the default sigmoid slope of 3.6 used in this opinion; and (4) exposure-response scenarios using the geometric mean of the curve-fitted data, and the minimum species mean value of the curve-fitted data, from the toxicity data used to derive the numeric criteria.

2.10.2 Compliance with RPA Criteria

A reasonable and prudent alternative to the proposed action is one that avoids jeopardy by ensuring that the action's effects do not appreciably increase the risks to the species' potential for survival or to the species' potential for recovery. It also must avoid destruction or adverse modification of designated critical habitat. A detailed analysis of how the RPA avoids jeopardy

[12]https://www.webapps.nwfsc.noaa.gov/sps
[13]http://sotr.cbfwa.org

and destruction or adverse modification of critical habitat is set out in section 2.10.3, below. In summary:

Implementation of the RPA avoids jeopardy to the listed species of fish because:

- We find that, based on the acute and chronic data in this opinion, effects of the revised action will not manifest at the population scale.
- We considered factors such as latent mortality and hypothesis tests in our effects analysis to assess the uncertainty of the revised action.
- The revised action will not result in appreciable population-level effects, (*i.e.*, lethal and sublethal effects do not result in a negative change in the intrinsic population growth rate, *e.g.*, lambda, λ).
- The available evidence indicates that the revised action is unlikely to appreciably affect invertebrate productivity and abundance.
- The requirement to adjust the criteria using a concentration–addition model or response-addition model will ensure that the revised action has a low probability of causing additive effects to the listed species.
- It can reasonably be concluded that the time needed to fully implement the revised action will not measurably impact the listed ESUs/DPSs or their critical habitat affected by this action.

For similar reasons, implementation of the RPA avoids adverse modification of the critical habitats for the listed species fish because:

- The revised action will not adversely modify critical habitats for the listed species considered in this opinion as the data suggests that the criteria concentrations are likely to have low-intensity adverse effects on the PCEs substrate, forage, or water quality at the watershed and designation scales. The available evidence indicates that the revised action is unlikely to appreciably affect invertebrate productivity and abundance.
- The revised action will minimize loading of copper, ammonia, cadmium, and aluminum in the affected watersheds so that habitat functions are maintained consistent with the conservation needs of the species.
- It can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their critical habitat affected by this action.

Implementation of the RPA avoids jeopardy to Southern Resident killer whales because, for those listed fish species that are prey for Southern Resident killer whales and the subject of this opinion, the RPA will ensure the impact on productivity and abundance is at a level where it does not pose an appreciable risk to the listed fish species and their designated critical habitats. Implementation of the RPA will also decrease the accumulation of toxic chemicals in the whales by reducing the bioaccumulation and toxic burdens in their prey to levels consistent with recovery of the listed species. For these reasons, NMFS expects that implementation of the RPA will avoid jeopardy for Southern Resident killer whales.

The reasonable and prudent alternative must also be: (1) consistent with the intended purpose of the action; (2) within the scope of the Federal agency's legal authority and jurisdiction; and

(3) economically and technologically feasible. This RPA is consistent with the purpose of EPA's action, as it will ensure that Oregon's water quality criteria for toxic pollutants will be protective of aquatic species. The EPA has authority, under the Clean Water Act, to ensure that state water quality standards are consistent with the requirements of the Clean Water Act requirements, which include ensuring that aquatic life is adequately protected.

Implementation of the RPA may impose some additional costs on the State of Oregon by requiring the state to meet more stringent numeric criteria than proposed, but neither the State of Oregon nor EPA conducted an economics analysis for the proposed action. With respect to chronic ammonia and acute and chronic copper, the RPA has been demonstrated to be economically and technologically feasible, because the freshwater chronic criterion of 0.76 mg/L for freshwater ammonia (total ammonia-N) at pH 8 and 20°C is currently being implemented in Oregon, and the acute and chronic criteria for copper are EPA's nationally recommended aquatic life criteria. For acute ammonia, acute cadmium, and acute and chronic aluminum, the RPA is economically and technologically feasible for EPA since it requires the agency to conduct an analysis and ensure the derived criteria are implemented in the State of Oregon, both functions that can be readily accommodated within the agency's normal course of business.

2.10.3 RPA Effects Analysis

The RPA Effects analysis is provided with reference to the effects of the action detailed above (section 2.6), which analyses effects of all criteria. This section provides particularized discussion of the seven criteria for which an RPA is provided.

2.10.3.1 Copper – Acute and Chronic

The revised criteria for copper are 1.45 µg/L (chronic) and 2.3 µg/L (acute), using EPA's 2007 BLM-based aquatic life criteria.[14]

The NMFS has determined that these revised criteria satisfy the conservation needs of the species and function of critical habitat PCEs because when we apply the same analysis that we used in the Effects Analysis, as described in section 2.6 of this opinion to the revised copper criteria, we find that the revised acute and chronic criteria for copper are unlikely to cause acute or chronic toxic effects to the listed fishes considered in this opinion that would manifest at the population scale.

More specifically:

- The NMFS compared the acute and chronic toxicity data in section 2.6.2.2.6 of this opinion to the revised criteria. For the acute criterion, none of the LC_{50} data was identified as being

[14]With regard to BLM-derived freshwater criteria, to develop a site-specific criterion for a stream reach, one is faced with determining what single criterion is appropriate even though a BLM criterion calculated for the event corresponding to the input water chemistry conditions will be time-variable. This is not a new problem unique to the BLM—hardness-dependent metals criteria are also time-variable values. Although the variability of hardness over time can be characterized, EPA has not provided guidance on how to calculate site-specific criteria considering this variability.

less than the revised acute criterion, the relative percent mortality analysis predicts a median toxicity potential of an $LC_{1.2}$, and only 11 of the 150 chronic data points were identified as being less than the revised chronic criterion.

- To take into account the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes, we considered factors such as latent mortality and hypothesis tests in our effects analysis to assess the uncertainty of the revised criteria
- The NMFS ran the revised acute criterion for freshwater copper of 2.3 µg/L through the direct mortality $populat_{io}n$ model (Appendix 1) using the geometric mean and the minimum species mean values of the LC_{50} data for copper to assess effects on mortality and lambda. The exposure-response scenario using the minimum species mean value with the revised criterion concentration of 2.3 µg/L predicted 1% mortality for all life history types with a 0% change in λ for all life history types. The exposure-response scenario using the geometric mean value predicted 0% mortality with 0% change in λ for all life history types. The NMFS considers the results of the direct mortality population model using the minimum species mean value to be a very conservative exposure-response scenario. The fact that this conservative exposure-response scenario predicts no change in λ for any of the life history types provides a level of assurance that the revised acute criterion for freshwater copper of 2.3 µg/L is unlikely to cause population-level adverse effects.
- Our analysis of the revised chronic criterion suggests that the revised criterion concentration is likely to avoid adverse chemosensory and behavioral effects to juvenile salmonid fishes (Hecht *et al.* 2007).
- The available evidence indicates that the chronic criterion for copper is unlikely to appreciably affect invertebrate productivity and abundance.
- For similar reasons, the revised criteria for copper will not adversely modify critical habitats for the listed species considered in this opinion as the data suggests that the criteria concentrations are likely to have low-intensity adverse effects on the PCEs substrate, forage, or water quality at the watershed and designation scales.

2.10.3.2 Ammonia – Chronic

The revised chronic criterion for ammonia is 0.76 mg/L as N (NH_3-nitrogen) at pH of 8.0 and 20°C.

The NMFS has determined that these revised criteria satisfy the conservation needs of the species and function of critical habitat PCEs because when we apply the same analysis that we used in the Effects Analysis, as described in section 2.6 of this opinion to the revised ammonia criterion, we find that, the revised chronic criterion for ammonia is unlikely to cause chronic toxic effects to the listed fishes considered in this opinion that would manifest at the population scale.

More specifically:

- The NMFS compared the chronic toxicity data in section 2.6.2.1.7 of this opinion to the revised criterion. For the chronic criterion only 9 of the 19 chronic data points were identified as being less than the revised chronic criterion. As described in the opinion, NMFS only

selected toxicity data in the core data file with a reported concentration type of total ammonia. For these toxicity studies, temperature and pH were not reported in the core data files; therefore verification regarding normalization was not possible and creates uncertainty. Therefore, as an additional step to address this uncertainty and to assess the potential for chronic toxic effects of ammonia to the listed species considered in this opinion using an additional line of evidence, NMFS used four ACRs described in section 2.6.2.1.7 of this opinion to estimate a NOEC for ammonia. These produced no concentrations less than the chronic criterion concentration, which indicates that listed species exposed to waters equal to chronic criterion concentrations may not suffer chronic toxic effects. To take into account the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes, we considered factors such as hypothesis tests in our effects analysis to assess the uncertainty of the revised criteria.

- The NMFS also considered non-lethal effects based on best available information and determined that they would be suffered at low-intensity.
- The revised criterion for ammonia will not adversely modify critical habitats for the listed species considered in this opinion as the data suggests that the criteria concentrations are likely to have low-intensity adverse effects on the PCEs substrate, forage. Ammonia does not bioaccumulate or bind to sediments—therefore effects on the PCEs substrate and forage are unlikely to be affected in a manner inconsistent with the recovery requirements of the listed fishes considered in this opinion. Furthermore, based on the ACR analyses, the revised criterion is likely to have low-intensity adverse effects on the PCEs substrate, forage, or water quality at the watershed and designation scales.

2.10.3.3 Derived Criteria

The EPA will derive criteria for acute ammonia, acute cadmium, and acute and chronic aluminum in accordance with the Process for Deriving Criteria set out above to ensure an adequately protective criterion is established.

The NMFS has determined that the derived criteria will satisfy the conservation needs of the species and function of critical habitat PCEs because the RPA relies on a conservative, well-defined methodology and requires EPA to ensure that the acute criterion for ammonia, the acute criterion for cadmium, and the acute and chronic criteria for aluminum do not cause a change in the intrinsic population growth rate (*e.g.*, λ). More specifically, NMFS developed the following requirements to address the uncertainties associated with the toxicity data, sublethal effects, multiple environmental stressors, and biological requirements consistent with the principles of conservation biology.

Toxicity Data

Because EPA is required to use toxicity data specific to salmonid fishes (and green sturgeon and eulachon, if it becomes available), this will minimize the uncertainties regarding the use of surrogate species and methodologies, *e.g.*, interspecies correlation analyses, to derive criteria that are consistent with the biological requirement of the species considered in this opinion.

Curve-fitted Data to Extrapolate Threshold Concentrations

The EPA is required to use toxicity data based on exposure-response curves and fixed durations toxicity tests to estimate acute and chronic toxic effect thresholds to assess effects on multiple life stages and multiple endpoints, to include at a minimum: mortality, latent mortality, reproduction, growth, physiological, cellular, behavioral, and biochemical effects, where the data exists. This requirement operates to ensure the derived criteria account for effects beyond the standard mortality, growth, and reproduction endpoints, but considers effects on a species life cycle and on sublethal endpoint that can affect the fitness and survival of affected species.

Adjust for Chemical Mixtures

The EPA is required to adjust each derived criterion for chemical mixtures using a concentration–addition model or response-addition model to determine whether or not exposure to multiple compounds will result in additive effects to the listed species. This requirement operates to ensure that environmental exposure conditions are considered in the development of the derived criteria. Fish exposed to multiple compounds, versus a single compound exposure, are likely to suffer toxicity greater than the assessment effects such as mortality, reduced growth, impairment of essential behaviors related to successful rearing and migration, cellular trauma, physiological trauma, and reproductive failure. The requirement to adjust the criteria using a concentration–addition model or response-addition model will ensure that the derived criteria have a low probability of causing additive effects to the listed species.

No Negative Change in Intrinsic Population Growth

Important assurances are provided by the requirement that the derived criteria do not result in a negative change in the intrinsic population growth rate based on the geometric mean abundance data for each life history type (as determined by a population model parameterized for Pacific salmonid fishes and otherwise meeting the RPA requirements). The requirement that the derived criteria are run through a population model is a method to assess population-level effects. A change in the intrinsic population growth rate, *e.g.*, λ, is an accepted population parameter often used in evaluating population productivity, status, and viability. The NMFS uses changes in λ when estimating the status of species, conducting risk and viability assessments, developing recovery plans, ESA consultations, and communicating with other federal, state and local agencies (McClure *et al.*, 2003). While values of $\lambda<1.0$ indicate a declining population, in cases when an exposure causes the population growth rate to decrease more than natural variability, a loss of productivity will result even if lambda remains above 1.0. Decreases in response to chemical exposures can be a cause for concern since the impact could make a population more susceptible to declining (lambda dropping below 1.0) due to impacts from other stressors. Therefore, the no change in the intrinsic population growth rate ensures that effects from the derived criteria will not manifest at the population scale, and are consistent with the recovery of the species considered in this opinion.

2.10.3.4. Mixtures Analysis

Since EPA has not derived specific numeric criteria for acute ammonia, acute cadmium, and acute and chronic aluminum, NMFS cannot run the revised numbers through the concentration-addition model used in this section 2.6.4 of this opinion to generate a revised mixtures effects prediction. Nonetheless, the requirement to adjust the criteria using a concentration–addition model or response-addition model will ensure that the revised criteria have considered environmental exposure conditions of multiple compounds.

2.10.3.5 Implementation Period

The NMFS evaluated the impact of the time lag between completion of the opinion and implementation of the revised action. In the proposed action, EPA assumed that the numeric criteria would be met outside the State's applicable mixing zone boundaries, *i.e.*, that the criteria represent ambient water quality conditions. The NMFS carried the assumption that the criteria concentrations represent the ambient water quality conditions through its analysis of the proposed action and of the RPA. Yet, based on Oregon DEQ's water quality assessment program data,[15] it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before ambient water quality conditions reach criteria concentrations.

To explore this in more detail, NMFS compared the current water quality baseline against the ambient criteria identified in the RPA to determine the likelihood that concentrations of these toxics would exceed the criteria identified in the RPA during the implementation period. The NMFS focused its analysis on the chronic criteria for ammonia. The NMFS determined that ammonia is a reasonable proxy for the remaining criteria because the RPA criteria for chronic ammonia is the same criterion currently in place;[16] thus, ammonia provides a natural reflection of the current distribution of the proposed new criterion, which is conducive to assessing the likelihood that the new criterion will be exceeded in a significant manner across the State during the implementation period. In addition, the other criteria do not so readily lend themselves to analysis.[17]

The data that we used was derived from Oregon Department of Environmental Quality's Water Quality Assessment Database. We extracted all available records associated with lakes and streams that had data for ammonia. The data included 273 records from river reaches in 64 subbasins across Oregon. Only four reaches in four subbasins were identified as sufficiently water quality limited as a result of ammonia to warrant listing on the State's CWA section 303(d) list. Three of these subbasins are above the range of anadromous fish. The remainder of the subbasins had no reaches that had high enough concentrations of ammonia to warrant listing on the 303(d) list. Even in the more densely populated area of the Willamette, approximately 68%

[15] http://www.deq.state.or.us/lab/wqm/watershed.htm

[16] The RPA states that EPA shall approve a new chronic criterion for the State of Oregon by maintaining the current chronic criterion of 0.76 mg/L at pH 8 and 20°C for freshwater ammonia (total ammonia-N).

[17] The derived criteria are not yet available for this type of analysis and because the copper criteria will be developed using the BLM approach it cannot be evaluated independent of other parameters necessary to determine site specific values.

of the reported reaches were fully attaining for ammonia. Extrapolating generally from the ammonia data, which demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations, it can reasonably be concluded that the time needed to implement the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

2.10.4 RPA Integration and Synthesis

For the RPA Effects Analysis, NMFS integrated the effects of the revised and derived criteria for copper, ammonia, cadmium, and aluminum into an overall effects analysis, taking into account the effects of the other criteria proposed by EPA. Similar to the RPA Effects Analysis, the RPA Integration and Synthesis considers the effects of the action as a whole, with additional focus on the seven compounds that NMFS identified with the highest-intensity adverse toxicological and adverse biological effects on the listed species considered in this opinion.

The RPA Integration and Synthesis section fully considers the effects of the action (section 2.6) to the environmental baseline (section 2.5), the cumulative effects (section 2.6.8), and the Integration and Synthesis (section 2.7) to formulate the agency's biological opinion as to whether the revised action is likely to: (1) Result in appreciable reductions in the likelihood of both survival and recovery of the species in the wild by reducing its numbers, reproduction, or distribution; or (2) reduce the value of designated or proposed critical habitat for the conservation of the species. These assessments are made in full consideration of the status of the species and critical habitat (section 2.4).

ESU/DPS-Specific Evaluations

LCR Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; LCR Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for LCR Chinook salmon or the broader watershed scale for their critical habitat.

(2) NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population

model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 32 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect LCR Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for LCR Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for LCR Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of LCR Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of LCR Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of LCR Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely

affected (40.2 percent of the total designation), but will not appreciably reduce the conservation value.

(6) NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSsor their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of LCR Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of LCR Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UWR Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; UWR Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for UWR Chinook salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 7 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the

relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect UWR Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for UWR Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UWR Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UWR Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for UWR Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UWR Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will incrementally improve water quality conditions for UWR Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances, and improve habitat quality that adequately provides for the conservation needs of UWR Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of UWR Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it

may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of UWR Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of UWR Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UCR Spring-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; UCR spring-run Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for UCR spring-run Chinook salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 4 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect UCR spring-run Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for UCR

spring-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UCR spring-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UCR spring-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for UCR spring-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UCR spring-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of UCR spring-run Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of UCR spring-run Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of UCR spring-run Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (30.8 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will

not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of UCR spring-run Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of UCR spring-run Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Spring/Summer-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SS-run Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for SS-run Chinook salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 27 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SS-run Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for SS-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SS-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SS-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SS-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SS-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SS-run Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SS-run Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SS-run Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (25.3 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery for SR SS-run Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to

reduce appreciably the conservation value of SR SS-run Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Fall-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SR fall-run Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for SR fall-run Chinook salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for the single SR fall-run Chinook salmon ESU (which consists of eight spawning populations).

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SR fall-run Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for SR fall-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SR fall-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial

arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SR fall-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SR fall-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SR fall-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SR fall-run Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SR fall-run Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SR fall-run Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (25.3 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of SR fall-run Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of SR fall-run Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

CR Chum Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; CR chum salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for CR chum salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 17 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect CR chum salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for CR chum salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of CR chum salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of CR chum salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced

fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for CR chum salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of CR chum salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of CR chum salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of CR chum salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of CR chum salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (26 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(6) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of CR chum salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of CR chum salmon critical habitat such that it will retain the current ability for the PCE water quality to serve the intended conservation role for the species for either survival or recovery.

LCR Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; LCR coho salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for LCR coho salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 27 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect LCR coho salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for LCR coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced

fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for LCR coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(6) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of LCR coho salmon.

SONCC Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SONCC coho salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for SONCC coho salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 42 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SONCC coho salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for SONCC coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SONCC coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SONCC coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SONCC coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SONCC coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SONCC coho salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SONCC coho salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SONCC coho salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (37.8 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of SONCC coho salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of SONCC coho salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

OC Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; OC coho salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for OC coho salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 56 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum;

and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect OC coho salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for OC coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of OC coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of OC coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for OC coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of OC coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of OC salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of OC coho salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of OC coho salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally

from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of OC coho salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of OC coho salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Sockeye Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SR sockeye salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for SR sockeye salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for the single SR sockeye salmon population.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SR sockeye salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for SR sockeye salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SR sockeye salmon such

that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SR sockeye salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SR sockeye salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SR sockeye salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SR sockeye salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SR sockeye salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SR sockeye salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (34.5 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of SR sockeye salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of SR sockeye salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

LCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; LCR steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for LCR steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 26 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect LCR steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for LCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat

quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for LCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of LCR steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of LCR steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of LCR steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (33 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of LCR steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of LCR steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UWR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; UWR steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for UWR steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 5 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect UWR steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for UWR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UWR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UWR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental

variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for UWR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UWR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of UWR steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of UWR steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of UWR steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of UWR steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of UWR steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

MCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with

consideration of the other proposed numeric criteria; MCR steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for MCR steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 17 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect MCR steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for MCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of MCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of MCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for MCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of MCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of MCR steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of MCR steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of MCR steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (75.7 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of MCR steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of MCR steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; UCR steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for UCR steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to

the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 4 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect UCR steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for UCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for UCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of UCR steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of UCR steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of UCR steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and

the overall percentage of critical habitat for this species that would be adversely affected (30.8 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of UCR steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of UCR steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SRB Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SRB steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for SRB steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 24 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the

relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SRB steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for SRB steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SRB steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SRB steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SRB steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SRB steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SRB steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SRB steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SRB steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (34.5 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an

example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of SRB steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of SRB steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species' for either survival or recovery.

Green Sturgeon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; green sturgeon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for green sturgeon or the broader watershed scale for their critical habitat.

(2) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect green sturgeon, but is not likely to appreciably affect the VSP parameters productivity and abundance for green sturgeon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of green sturgeon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(3) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of green sturgeon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of green sturgeon.

Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of green sturgeon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (10.4 percent of the total designation), but will not appreciably reduce the conservation value.

(4) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(5) After considering all the information in this opinion, NMFS concludes that the revised action is likely not to reduce appreciably the likelihood of both the survival and recovery of green sturgeon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of green sturgeon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

Eulachon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; eulachon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for eulachon or the broader watershed scale for their critical habitat.

(2) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely

affect eulachon, but is not likely to appreciably affect the VSP parameters productivity and abundance for eulachon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of eulachon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(3) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of eulachon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of eulachon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of eulachon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (53.9 percent of the total designation), but will not appreciably reduce the conservation value.

(4) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(5) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of eulachon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of Eulachon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

Southern Resident Killer Whales.

As explained in section 2.8, we previously concluded that in the short-term, annual reductions in salmon prey caused by the proposed action would not have significant effects on Southern Resident killer whales. However, we determined that in the long-term, the continued decline and potential extinction of the UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon, and consequent interruption in the geographic continuity of salmon-bearing watersheds in the Southern Residents' coastal range was likely to alter the distribution of migrating salmon and increase the likelihood of localized depletions in prey, with adverse effects on the Southern Residents' ability to meet their energy needs. We concluded that the proposed action would

appreciably reduce the likelihood of survival and recovery of the Southern Resident killer whales.

Under the RPA, there will remain a reduction in prey in the short-term. However, as discussed in section 2.8, the annual prey reduction will be extremely small, and the probability is low that any of the juvenile Chinook salmon killed from implementation of the RPA would be intercepted by the killer whales across their vast range. Therefore, NMFS anticipates that the short-term reduction of Chinook salmon from the implementation of the RPA will have an insignificant effect on Southern Resident killer whales. The RPA will remove the long-term threat to killer whales by avoiding population-level and ESU/DPS-level effects to salmonids. Because the RPA will avoid ESU/DPS-level effects on abundance and productivity, and because we expect any short-term prey reductions to be insignificant, we also expect long-term effects from the RPA to be insignificant for Southern Resident killer whales. Also as discussed in Section 2.8, the available data indicate that Southern Residents are not at risk of health effects from the toxic criteria considered in this opinion. Because the RPA will further reduce levels of copper, ammonia, cadmium, and aluminum, we expect that any effects from the revised criteria will be insignificant and/or discountable.

In summary, implementation of the RPA avoids jeopardy to Southern Resident killer whales because it will reduce the impact on salmonids productivity and abundance to a level where it will not cause a discernable reduction in prey for Southern Resident killer whales and will also avoid adverse health effects to the whales.

Conclusion

Based on these considerations and the foregoing description of the RPA, NMFS finds that the RPA meets each of the criteria stated at 50 CFR 402.02.

After reviewing the best available scientific and commercial information regarding the biological requirements and the status of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, eulachon and Southern Resident killer whales considered in this opinion (section 2.4), the environmental baseline (section 2.5) for the action area, the effects of the proposed action (section 2.6), the cumulative effects (section 2.6.8), and the RPA (section 2.10), NMFS concludes that the revised action is not likely to jeopardize the continued existence of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, eulachon, and Southern Resident killer whales.

Furthermore, NMFS has determined NMFS has determined that the revised action will not result in the destruction or adverse modification of critical habitat as a result of degraded water quality in Oregon for LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon,

SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, and eulachon.

2.11 Incidental Take Statement

Section 9 of the ESA and Federal regulations pursuant to section 4(d) of the ESA prohibit the take of endangered and threatened species, respectively, without a special exemption. Take is defined as to harass, harm, pursue, hunt, shoot, wound, kill, trap, capture or collect, or to attempt to engage in any such conduct. Harm is further defined by regulation to include significant habitat modification or degradation that results in death or injury to listed species by significantly impairing essential behavioral patterns, including breeding, feeding, or sheltering. Incidental take is defined as take that is incidental to, and not the purpose of, the carrying out of an otherwise lawful activity. For this consultation, we interpret "harass" to mean an intentional or negligent action that has the potential to injure an animal or disrupt its normal behaviors to a point where such behaviors are abandoned or significantly altered.[18] Section 7(b)(4) and section 7(o)(2) provide that taking that is incidental to an otherwise lawful agency action is not considered to be prohibited taking under the ESA if that action is performed in compliance with the terms and conditions of this incidental take statement.

The NMFS has not yet promulgated an ESA section 4(d) rule prohibiting take of threatened eulachon. Anticipating that such a rule may be issued in the future, we have included a prospective incidental take exemption for eulachon. The elements of this ITS that relate to eulachon would take effect on the effective date of any future 4(d) rule prohibiting take of eulachon.

2.11.1 Amount or Extent of Take

All of the species of ESA-listed salmon, steelhead, green sturgeon, and eulachon analyzed in this opinion will be exposed to concentrations of criteria chemicals in the action area that are directly related to the action under the RPA. These concentrations of chemicals are likely to cause deaths and injuries of the listed species. These concentrations are also likely to cause habitat degradation that will result in the death or injury of listed species by reducing the availability of suitable prey organisms and thereby significantly impairing the essential behavioral pattern of feeding. All life stages are likely to be affected due to direct exposure of adults and/or juveniles to the chemicals and to latent effects on gametes following exposure of gravid adults. For the reasons set forth in the RPA section (section 2.10), incidental take of Southern Resident killer whales is not likely and therefore killer whales are not included within this ITS.

[18] NMFS has not adopted a regulatory definition of harassment under the ESA. The World English Dictionary defines harass as "to trouble, torment, or confuse by continual persistent attacks, questions, etc." The U.S. Fish and Wildlife Service defines "harass" in its regulations as "an intentional or negligent act or omission which creates the likelihood of injury to wildlife by annoying it to such an extent as to significantly disrupt normal behavioral patterns which include, but are not limited to, breeding, feeding, or sheltering (50 CFR 17.3). The interpretation we adopt in this consultation is consistent with our understanding of the dictionary definition of harass and is consistent with the Service's interpretation of the term.

Incidental take caused by the habitat-related effects of this action cannot be accurately quantified as a number of fish to be taken, because the number of fish at a given location at a given time are affected by myriad abiotic and biotic factors such as habitat quality and availability, competition, and predation, as well as interactions among these factors. These factors interact in ways that may be random or directional, and may operate across broader temporal and spatial scales that are affected by the proposed action. Thus, the distribution and abundance of fish within the action area cannot be attributed entirely to habitat conditions, nor can NMFS precisely predict the number of fish that are reasonably certain to be injured or killed due to habitat degradation related to the proposed action. Also, there is no feasible way to count, observe, or determine the number of fish that would be injured or killed by exposure to compounds listed in Table 1.1. This is because (1) the effects of the action would take place over a large geographic area (the action area for this consultation covers approximately 90,000 square miles, including the nearshore environment of the Pacific Ocean along the Oregon coast), and most injuries or deaths are likely to occur in areas where fish cannot be observed (*e.g.*, deep water or remote areas); (2) even if injured or dead fish were observed, it would be difficult or impossible in many cases to determine an exact cause of injury or death; and (3) sublethal effects of the proposed action could manifest later in time at locations where they could not readily be observed (*e.g.*, the Pacific Ocean).

In this case, NMFS will use quantitative measurements of ambient concentrations of ammonia and copper as surrogates for the amount of incidental take due to the action under the RPA. Ammonia and copper are suitable surrogates for the amount of incidental take for several reasons. Both chemicals are commonly discharged throughout the action area. These were among the most toxic chemicals analyzed by NMFS, and therefore they are likely to contribute significantly to incidental take. As described in the effects analysis, exposure to these chemicals is likely to cause chronic toxic effects at criterion concentrations that are reasonably certain to result in eventual death or injury of some individuals of the listed species considered in this opinion. There is abundant data about how both chemicals affect fish and invertebrate species that may be prey items. Although many of the criteria chemicals under the RPA action may be discharged at or below levels that can be accurately measured with current analytical methods, ammonia and copper concentrations that are likely to cause sublethal, adverse effects on the ESA-listed species are readily measurable. Because of similar fate and transport pathways (particularly with respect to copper and other metals), concentrations of ammonia and copper are likely to correlate reasonably well with concentrations of other criteria compounds and can thereby serve as surrogates for the overall extent of take indicator.

The NMFS selected the chronic criterion concentrations for ammonia and copper because, as compared to the acute concentrations, they provide a more continuous environmental concentration that could be monitored over the long term at the scale of the stream/river reach or watershed. Acute concentrations are more likely to be exceeded in highly localized areas for short periods of time, and therefore would be difficult to detect by monitoring designed to determine trends at larger scales of time and space that are needed to assess the overall extent of take. Also, exceedences of chronic concentrations in many cases likely will result in exceedences of acute concentrations.

The NMFS proposes to use the ambient water quality monitoring network program of the DEQ to determine whether the extent of take is exceeded. The DEQ monitors a fixed station network of 131 sites on more than 50 large rivers and streams across the state in its ambient program.[19] These sites, shown in Figure 2.11.1.1., cover 4th order and larger rivers in 16 basins delineated by the DEQ. Some of these basins are inhabited by only one ESA-listed species considered in this opinion, some are inhabited by more than one ESA-listed species, and some are not inhabited by ESA-listed species (*e.g.*, the Powder and Malheur basins). The DEQ selected these sites to represent all major rivers in the state and provide statewide geographical representation. The sites are primarily "integrator" sites, meaning they reflect the integrated water quality effects from point and nonpoint source activities as well as the natural geological and hydrological factors for the watershed. Larger river basins have multiple sites, which may be based on tributaries, land use changes, topographical changes, ecoregions, point sources, and nonpoint sources.

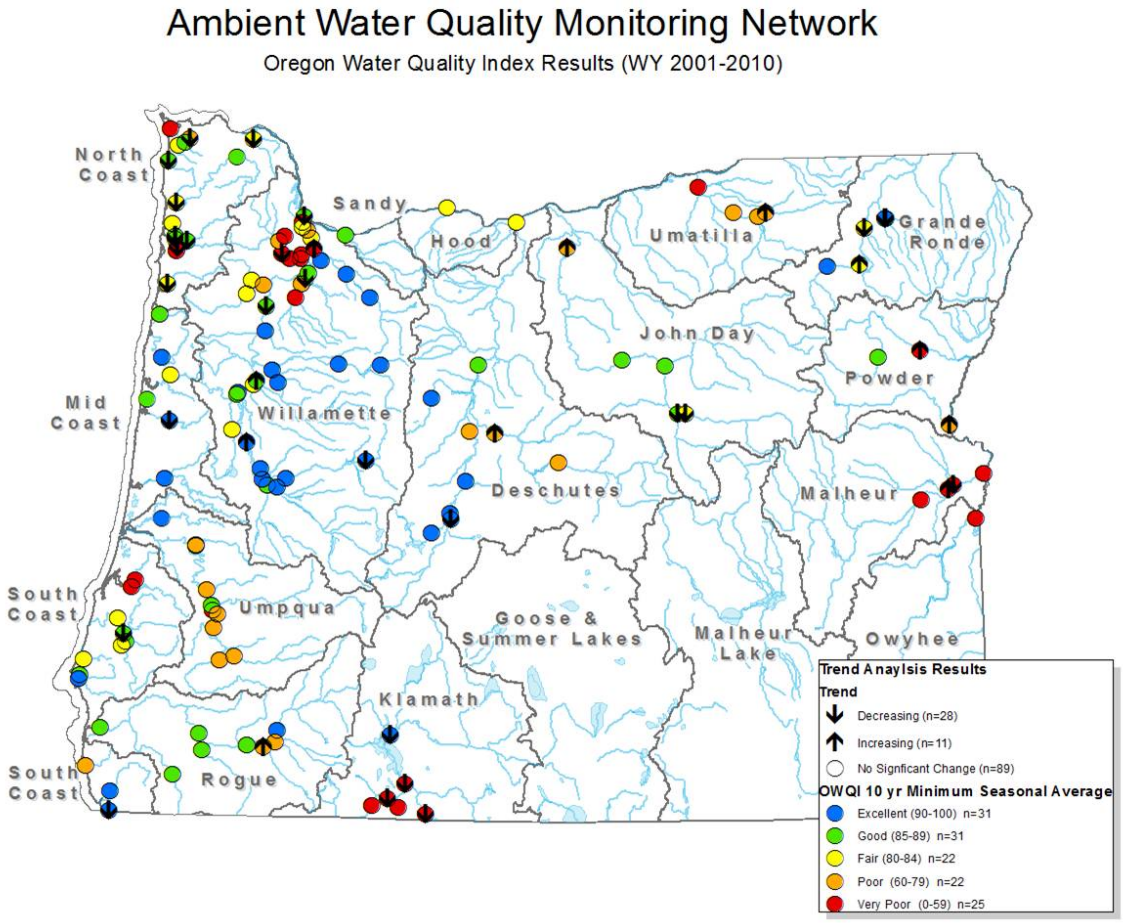

Figure 2.11.1.1. Fixed stations in the ambient water quality monitoring network of the DEQ. Text box in lower right is not relevant to the incidental take statement.

[19]Telephone discussion between Jeff Lockwood, NMFS, and Aaron Borisenko, DEQ, August 7, 2012.

The DEQ uses its ambient monitoring program to understand trends in Oregon's water quality over time, determine whether there is too much pollution in a water body, and set limits of how much pollution a water body can safely receive. The DEQ regularly samples sites within the action area for this consultation. At its ambient monitoring sites, DEQ monitors ammonia concentrations, but it does not currently monitor concentrations of any metals.

In order to comply with this incidental take statement, EPA will need to ensure that monitoring for ambient concentrations of ammonia and copper occurs at DEQ sample sites consistent with the final monitoring plan that will be developed within 12 months of the signing of this opinion. The EPA shall ensure that implementation of the monitoring plan (which will incorporate both the ammonia and the copper criteria) within 6 months of when EPA approves the new criteria for ammonia and copper.

The extent of take for a given ESA-listed species will be exceeded if, in any given DEQ fourth-field or larger USGS hydrologic unit code watershed (as delineated and labeled in Figure 2.11.1.1) that is inhabited by that species, the median value of the valid results for freshwater samples taken in that watershed for ammonia or copper are higher than the threshold values of 0.76 mg/L at pH 8 and 20°C for ammonia, or 1.45 µg/L for copper, respectively, for two consecutive sampling periods. As recognized in the biological opinion, there will be a time lag between establishment of the criteria and incorporation within the terms of all NPDES permits in the state.

To account for this lag period in the event of an exceedence in a given watershed, the extent of take indicator will be triggered only when at least 75 percent of the watershed NPDES permits have been issued under the new criteria. This approach is necessary because it would be unreasonable to assume that all NPDES permits will incorporate the new criteria until existing permits written under the old criteria are renewed over the 5-year permit cycle.

Although the extent of take indicators are the same as the revised criteria for freshwater chronic ammonia and copper, they nevertheless will function as an independent trigger for reinitiation of consultation, because establishing the criteria does not ensure that the criteria always will be met. As the State of Oregon’s current CWA section 303(d) list illustrates, waters within the state for various reasons can exceed established water quality standards. The chosen surrogates of chronic copper and ammonia measured as specified above will function to establish predetermined instances where monitored watersheds exceed established levels of toxic compounds and therefore the effects analysis of this biological opinion.

2.11.2 Effect of the Take

In section 2.10, NMFS determined that the anticipated level of incidental take, coupled with other effects of the proposed action, is not likely to result in jeopardy to the species or destruction or adverse modification of critical habitat when the RPA is implemented.

2.11.3 Reasonable and Prudent Measures

"Reasonable and prudent measures" are nondiscretionary measures to minimize the amount or extent of incidental take (50 CFR 402.02). The following measures are necessary and appropriate to minimize the impact of incidental take of listed species from the proposed action.

1. The EPA shall monitor and report to NMFS on the implementation of the RPA.
2. The EPA shall ensure completion of the monitoring and reporting program to ensure that the extent of take is not exceeded, and to confirm that the terms and conditions in this incidental take statement are effective in avoiding and minimizing incidental take.

2.11.4 Terms and Conditions

The terms and conditions described below are non-discretionary, and the EPA must comply with them in order to implement the reasonable and prudent measures (50 CFR 402.14). The EPA has a continuing duty to monitor the impacts of incidental take and must report the progress of the action and its impact on the species as specified in this incidental take statement (50 CFR 402.14). If the EPA does not comply with the following terms and conditions, the protective coverage of section 7(o)(2) likely will lapse.

1. To implement reasonable and prudent measure # 1 (monitoring the implementation of the RPA) the EPA shall:
    a. Implement oversight of the State of Oregon's NPDES program to ensure that the NPDES permit protocols are implemented in a manner consistent with the EPA technical support document (EPA 1991) and that (a) the State of Oregon is renewing NPDES permits in a manner consistent with the Clean Water Act and its implementing regulations; and (b) the numeric criteria proposed for approval by EPA, as well as any numeric criteria that change when derived by EPA or adopted by the State of Oregon consistent with the RPA, are being implemented in all new and renewed NPDES permits.
    b. Provide NMFS with annual reports on the monitoring requirements by October 31 of each year, for a minimum of 10 years from the date of EPA's final action under the Clean Water Act on Oregon's proposed criteria. Each of these reports shall include:
        i. An assessment of whether or not the State of Oregon is renewing all NPDES permits within the normal 5-year renewal period.
        ii. An assessment of the extent to which the State of Oregon is implementing the numeric criteria proposed for approval by EPA, as well as any numeric criteria that change when derived by EPA or adopted by the State of Oregon consistent with the RPA, in new and renewed NPDES permits.

2. To implement reasonable and prudent measure #2 (monitoring and reporting program) the EPA shall:
    a. Work with NMFS and the DEQ to develop a plan to collect, analyze and summarize the data on ambient concentrations of ammonia and copper in all freshwater monitoring sites in the DEQ's ambient monitoring network that are in

streams or rivers inhabited by ESA-listed species. The monitoring plan shall be finalized no later than 12 months from the date of this opinion.

b. Ensure that sampling, analysis and reporting the monitoring for ambient concentrations of ammonia and copper at the DEQ sample sites begins within 6 months of when EPA approves the new criteria for ammonia and copper.
c. After monitoring and reporting begin, notify NMFS if any of the incidental take thresholds described in this incidental take statement are exceeded within 1 month of receiving the information from the DEQ.
d. Provide NMFS with annual reports on the monitoring requirements by October 31 of each year, for a minimum of 10 years from the date of EPA's final action under the Clean Water Act on Oregon's proposed criteria. Each of these reports shall include a summary of the results of the monitoring of ambient concentrations of ammonia and copper (as described in term/condition 1.b. above).

2.12 Conservation Recommendations

Section 7(a)(1) of the ESA directs Federal agencies to use their authorities to further the purposes of the ESA by carrying out conservation programs for the benefit of the threatened and endangered species. Conservation recommendations are discretionary measures suggested to minimize or avoid adverse effects of a proposed action on listed species or critical habitats, or regarding development of additional information. The following conservation recommendations are discretionary measures that are suggestions regarding discretionary measures to minimize or avoid adverse effects of a proposed action on listed species or critical habitat or regarding the development of information (50 CFR 402.02) consistent with these obligations, and therefore should be carried out by the EPA for the proposed action:

1. To improve the potential for recovery of listed species in the State of Oregon, the EPA should carry out management actions to reverse threats to survival as identified in the Columbia River Basin recovery plans for salmon and steelhead, the SONCC coho salmon recovery plan, and futire recovery plans for green sturgeon and eulachon.

2. The EPA should replace the fixed duration LC_{50} acute toxicity tests used for criteria development with acute toxicity tests based on exposure-response curves to describe the relationship between exposure and toxicological effects, and EPA should replace the current chronic tests, *i.e.*, hypothesis testing, used for criteria development with chronic toxicity tests based on exposure-response curves to describe the relationship between exposure and toxicological effects.

3. The EPA should work with the State of Oregon to develop a monitoring protocol for toxic pollutants that establishes a consistent monitoring program across the state, and is designed to measure, in real-time, whether or not a particular point-source discharger is in compliance with the aquatic life criteria.

4. The EPA should work with the State of Oregon to minimize effects from chemical mixtures and decrease mixing zone dimensions such that no mixing zones overlap in space and time, or impact more than 5 percent of the cross-sectional area of the affected

waterbody , and are calculated using the “one-day, once in ten year low flow” (1Q10) statistic or its equivalent.

2.13 Reinitiation of Consultation

As provided in 50 CFR 402.16, reinitiation of formal consultation is required where discretionary Federal action agency involvement or control over the action has been retained, or is authorized by law, and if: (1) the amount or extent of incidental take is exceeded, (2) new information reveals effects of the agency action on listed species or designated critical habitat in a manner or to an extent not considered in this opinion, (3) the agency action is subsequently modified in a manner that causes an effect on the listed species or critical habitat not considered in this opinion, or (4) a new species is listed or critical habitat designated that may be affected by the action.

To reinitiate consultation, contact the Oregon State Office Habitat Office of NMFS and refer to NMFS Number **2008/00148**.

2.14 Not Likely to Adversely Affect Determinations

In this opinion NMFS concludes that the proposed action is not likely to adversely affect (NLAA) Steller sea lions, humpback whales, blue whales, fin whales, Sei whales, sperm whales, North Pacific Right whales, loggerhead sea turtles, green sea turtles, leatherback sea turtles, or Olive Ridley sea turtles.

The above identified marine mammal and sea turtle species are distributed in coastal areas and may be exposed to effects related to the proposed numeric criteria. Similar to Southern Resident killer whales, effects would be indirect and would include reduced prey availability, reduced prey quality, and potential accumulation in the individuals exposed. However, the occurrence of the subject ESA-listed sea turtles and large whales would be rare, infrequent, and transitory in the action area. For example, the blue whale and Sei whale are likely to have limited exposure to contaminant sources as their migratory patterns are circumglobal with definite seasonal movements to offshore areas outside the likely extent of effects. In the event that the turtles and large whales are present, they would be unlikely to accumulate a significant amount of persistent pollutants because they primarily consume lower trophic-level prey. Thus, sea turtles and large whales are unlikely to accumulate significant levels of contaminants in the action area that would be a cause for concern.

Steller sea lions of the eastern DPS occur in Oregon waters throughout the year, with breeding rookeries on offshore rocks and islands and haulout locations on and offshore along the coast and in the Columbia River (Table 2.14.1). Steller sea lions are not known to predictably occur along coastal reaches, in coastal bays or in river systems of Oregon aside from areas proximate to their haulout and rookery locations and their seasonal occurrence in the lower Columbia River and Rogue River. Steller sea lions are generalist predators that eat a variety of fishes and cephalopods, including salmon (NMFS 2008k). It is likely that Steller sea lions will be exposed to pollutants from the proposed numeric criteria through ingestion of prey; however, the extent of likely exposure is difficult to determine. Unlike Southern Resident killer whales that consume

primarily salmonids (which are highly contaminated. upper-trophic level prey), Steller sea lions have a large foraging base and consume prey at a relatively lower trophic level (*i.e.*, Steller sea lions are likely exposed to less-contaminated prey than the Southern Resident killer whales are). There is limited information on the contaminant levels in Steller sea lions. Heavy metal concentrations in Steller sea lions are generally lower than northern fur seals (Noda *et al.* 1995, Beckmen *et al.* 2002). Overall, studies suggest a decline in contaminant concentrations over time, which is consistent with that reported for other wildlife species (NMFS 2008k). Additionally, comparable levels of zinc, copper, and metallothionein were measured in pups from both the eastern and western Steller sea lion DPSs (Castellini and Cherian 1999). Although these studies are not comprehensive, they indicate that heavy metals were not likely a significant factor in the decline of the Steller sea lions (NMFS 2008k). However, the population has grown steadily for the past 20 to 30 years, with no indication that contaminant-induced health effects are limiting recovery. For these reasons, the potential for exposure to contaminants from ingesting contaminated prey and for any subsequent chance of bioaccumulation of contaminants in Steller sea lions are likely to be insignificant.

The proposed action may reduce the quantity of prey available, due to the incidental take of salmon, green sturgeon, and eulachon. The NMFS anticipates similar effects on non-listed species that may be prey items for the subject listed species. Any salmonid take up to the aforementioned maximum extent and amount would result in an insignificant reduction in prey resources for marine mammals that may intercept these species within their range.

The NMFS finds that all effects of the action are likely to be discountable or insignificant, and therefore concludes that the proposed action is not likely to adversely affect Steller sea lions, humpback whales, blue whales, fin whales, Sei whales, sperm whales, North Pacific Right whales, loggerhead sea turtles, green sea turtles, leatherback sea turtles, or Olive Ridley sea turtles.

Table. 2.14.1 Steller Sea Lion Haulout and Rookery Locations in Oregon Waters (ODFW 2010).

MARINE LOCATION	HAULOUT SITE	COUNT/USE	LATITUDE/LONGITUDE[1]	ROOKERY?
Columbia River	Tip of the South Jetty	>500 Common	46.2338 / -124.0702	
	East Mooring Basin	<10 Rare	46.1963 / -123.8006	
	Phoca Rock	10-<100 Occasional	45.5720 / -122.1820	
	Bonneville Dam, Tailrace	10-<100 Occasional	45.6450 / -121.9480	
Tillamook Head	Tillamook Rock, Offshore from Tillamook Head	500-1,000 Common	45.9368 / -124.0185	
Ecola Point	Ecola Point	<10 Rare	45.9185 / -123.9805	
Three Arch Rocks	Three Arch Rocks	10-<100 Common	45.4637 / -123.9833	Yes
Cascade Head	Sea Lion Cove_2	10-<100 Common	45.0692 / -124.0085	
	Sea Lion Cove_3	100-500 Common	45.0670 / -124.0123	
Seal Rock	North Offshore	10-<100 Occasional	44.5022 / -124.0943	
Cape Arago	Simpsons Reef	10-<100 Common	43.3137 / -124.4082	
	Shell Island Area	100-500 Common	43.3133 / -124.4013	
Blanco Reef	Blanco Reef	100-500 Common	42.8239 / -124.5836	
Orford Reef	Large Brown Rock	10-<100 Common	42.7922 / -124.6008	
	Long Brown Rock	>500 Common	42.79136 / -124.6060	Yes
	Best Rock	100-500 Common	42.7906 / -124.5955	
	Square White Rock	10-<100 Occasional	42.7882 / -124.6048	
	Seal Rock (Orford Reef)	100-500 Common	42.7870 / -124.5946	Yes
	Miscellaneous (Orford Reef)	10-<100 Occasional	42.7825 / -124.6047	
	Arch Rock	100-500 Common	42.7784 / -124.5974	Yes
	West Conical Rock	100-500 Common	42.7774 / -124.6010	Yes
	Steamboat Rocks	10-<100 Common	42.7760 / -124.6041	
Rogue Reef	Double Rock	10-<100 Common	42.4494 / -124.4901	
	Needle Rock	100-500 Common	42.4484 / -124.4837	Yes
	Pyramid Rock- Miscellaneous	10-<100 Common	42.4467 / -124.4695	
	Miscellaneous (Rogue Reef)	10-<100 Common	42.4455 / -124.4793	
	Pyramid Rock	>500 Common	42.4441 / -124.4693	Yes
	Southern Seal Rock (Rogue)	10-<100 Common	42.4365 / -124.4652	
Crook Point	Crook Point	10-<100 Occasional	42.2453 / -124.4141	

[1]Latitude and longitude reported in decimal degrees.
Source: ODFW.

Critical Habitat

Steller Sea Lion and Leatherback Turtle. The NMFS designated critical habitat for the Steller sea lion in certain areas and waters of Alaska, Oregon and California on August 27, 1993 (NMFS 1993). Certain rookeries, haulouts, and associated areas with essential prey resources for at least lactating adult females, young-of-the-year, and juveniles were designated as critical habitat. In Oregon, these areas include Long Brown Rock and Seal Rock at Orford Reef and Pyramid Rock at Rogue Reef. There are no "special aquatic foraging areas" identified as critical habitat in Oregon. Critical habitat includes air zones extending 3,000 feet above the terrestrial and aquatic zones, and aquatic zones extending 3,000 feet seaward from the major rookeries and haul-outs.

Designated critical habitat for leatherback sea turtles in the action area includes one 24,500 square-mile marine area stretching from Cape Flattery, Washington, to the Umpua River, Oregon. The PCEs that NMFS identified as essential for the conservation of leatherback sea turtles when it proposed to revise critical habitat to include marine waters off the U.S. West Coast include: (1) A sufficient quantity and quality of their jellyfish prey; and (2) migratory pathway conditions that allow for safe and timely passage to, from, and within high-use forage areas.

Based on the best scientific and commercial data available, as discussed previously, NMFS does not expect that the proposed action would adversely affect the quantity, quality, or availability of any of the constituent elements of critical habitat, or the physical, chemical, or biotic phenomena that give the designated area value for the conservation of the species when no constituent elements were identified in the designation. Although NMFS would expect critical habitat for Eastern Steller sea lions and proposed critical habitat for the leatherback sea turtle to be exposed to toxic chemicals due to the proposed action, the concentrations would be sufficiently low that the effects would be insignificant. Critical habitat for green sea turtles does not occur in the action area.

The NMFS finds that all effects of the action are likely to be insignificant, and therefore concludes that the proposed action is not likely to adversely affect Steller sea lion and leatherback turtle critical habitat.

3. DATA QUALITY ACT DOCUMENTATION AND PRE-DISSEMINATION REVIEW

Section 515 of the Treasury and General Government Appropriations Act of 2001 (Public Law 106-554) (Data Quality Act) specifies three components contributing to the quality of a document. They are utility, integrity, and objectivity. This section of the opinion addresses these Data Quality Act (DQA) components, documents compliance with the DQA, and certifies that this opinion has undergone pre-dissemination review.

3.1 Utility: Utility principally refers to ensuring that the information contained in this consultation is helpful, serviceable, and beneficial to the intended users. The intended users are EPA and the State of Oregon.

An individual copy was provided to EPA. This consultation will be posted on the NMFS Northwest Region website (http://www.nwr.noaa.gov). The format and naming adheres to conventional standards for style.

3.2 Integrity: This consultation was completed on a computer system managed by NMFS in accordance with relevant information technology security policies and standards set out in Appendix III, 'Security of Automated Information Resources,' Office of Management and Budget Circular A-130; the Computer Security Act; and the Government Information Security Reform Act.

3.3 Objectivity:

Information Product Category: Natural Resource Plan.

Standards: This consultation and supporting documents are clear, concise, complete, and unbiased; and were developed using commonly accepted scientific research methods. They adhere to published standards including the NMFS ESA Consultation Handbook, and the ESA Regulations, 50 CFR 402.01, *et seq*.

Best Available Information: This consultation and supporting documents use the best available information, as referenced in the Literature Cited section. The analysis in this opinion contains more background on information sources and quality.

Referencing: All supporting materials, information, data and analyses are properly referenced, consistent with standard scientific referencing style.

Review Process: This consultation was drafted by NMFS staff with training in ESA implementation, and reviewed in accordance with Northwest Region ESA quality control and assurance processes.

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APPENDIX 1: EPA's Guidelines for Deriving Numerical National Water Quality Criteria and Issues Common to All Criteria

The following discussion and analysis examines the shortcomings of EPA's methodology for deriving the national criteria and is critical to understanding the relationship between the numeric criteria and the exposure-response analysis in this opinion. The discussion and analysis in this Section is separated into two main categories: (1) EPA's methodology for deriving the national aquatic life criteria, and (2) overview of the effects assessment methodology in EPA's BE for the Oregon criteria.

Derivation of EPA Aquatic Life Criteria

The foremost problem with EPA's national aquatic life criteria lies with the derivation methodology, which is set out in EPA's *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (Stephan *et al.* 1985) (Guidelines). The extent of technical issues delineated in this section regarding the Guidelines produces far more uncertainty than predictability regarding the reliability of the criteria to protect aquatic life, and in particular, listed species. This analysis highlights the risks associated with use of the Guidelines and assesses how they are likely to influence the chemical and environmental stressors affecting the listed species evaluated in this opinion.

First, we look at EPA's general approach as described in the Guidelines. Second, we look at the risks or conservatisms associated with EPA's approach. Third, we provide a summary that qualitatively assesses the degree of uncertainty and likely influences on the effects associated with exposure-response risks to the listed species considered in this opinion.

The derivation methodology for EPA's water quality criteria, the basis of Oregon's proposed water quality criteria, is detailed in the Guidelines (Stephan *et al.* 1985). An overview of the Guidelines, as described in EPA's BE, is presented below.

> The first stage in deriving water quality criteria is to compile the available data on the chemical of interest regarding its toxicity to and bioaccumulation by aquatic animals and plants. These data then go through a review process to identify studies that should not be used to derive national criteria. Although there are a number of reasons why data are not included in the data sets used to develop national criteria, some of the more common ones are that one or more pieces of information regarding study methodology or calculation of results needed to assess the reliability of the study is missing; data quality of the study is less than acceptable (*e.g.* unacceptably high control mortality); the tested species does not have a reproducing population in North America; the test species was exposed to a chemical mixture or was previously exposed to the test chemical; the study reported effects on an endpoint other than survival, reproduction of growth; or the test duration was a non-standard test duration (*e.g.* fish toxicity test reporting a 24-hr LC_{50} instead of the more standard 96-hr LC_{50}).

Once the available data have been reviewed and unacceptable or inappropriate study results have been removed from the data set, the data are reviewed to ensure that certain types of data are available. Specifically, for freshwater aquatic biota, the following eight types of toxicity data should be available:

- Data for a fish species in the family *Salmonidae* of the class *Osteichthys*
- Data for a fish species from a second family in the class *Osteichthys*
- Data for a third family in the phylum *Chordata* (may be a third fish species or an amphibian species)
- Data for a planktonic crustacean species
- Data for a benthic crustacean species
- Data for an aquatic insect species
- Data for a species in a phylum other than *Arthropoda* or *Chordata* (*e.g. Rotifera*, *Annelida*, *Mollusca*, etc.)
  Data for a species in any family in any order of insect or any aquatic phylum not already represented.

Additionally, the following three other pieces of information are needed before a national water quality criterion can be developed for a given chemical (required to derive both freshwater and saltwater criteria). Unlike toxicity data, which must be from exposures of species to chemicals in freshwater in order to derive freshwater criteria, the following information can be either for freshwater data only or a specified mixture (Stephan *et al.* 1985) of freshwater and saltwater data.
Acute-chronic ratios (ACRs) for at least three different families of aquatic species. Toxicity data for at least one freshwater plant (can be either algal or a vascular plant)
At least one bioconcentration factor (BCF).

The eight taxa for which saltwater toxicity data are required prior to derivation of a saltwater criterion obviously differ from those for freshwater, and must be from the taxonomic groupings listed below:

- Data from two families in the phylum *Chordata*
- Data from a family in a phylum other than *Arthropoda* or *Chordata*
- Data from a species in either the *Mysidae* or *Penaeidae* family
- Data from three other families not in the phylum *Chordata* (may include data for a species from a phylum or family listed in taxa groups 1 - 3 above but which was not used)
- Data from any other saltwater family

Ideally, the above freshwater and marine species toxicity data have both LC_{50} data of appropriate duration and chronic NOEC data available. In practice, most chemicals with water quality criteria have sufficient LC_{50} data to permit derivation of an acute water quality criterion from measured LC_{50} data, but do not have sufficient measured chronic NOEC to use the above procedure to directly calculate a chronic criterion. Instead, most chronic criterion are calculated by dividing the calculated acute criterion by the available ACR value.

If toxicity data are available from multiple studies (*e.g.* three LC_{50} results are available for rainbow trout), a species mean acute value (SMAV) (or species mean chronic value if one is deriving a chronic criterion, although the rest of this discussion will assume that only measured acute toxicity data are available) is calculated as the geometric mean of the three available LC_{50} values in this example. Similarly, if two or more LC_{50} results are available for different species of the same genus (*e.g.* LC_{50} data are available for rainbow trout and Chinook salmon, both members of the genus *Oncorhynchus*), a genus mean acute value (GMAV) is calculated from the geometric mean of all toxicity data for members of that genus. If only one LC_{50} value is available for a species from a given genus, that single value becomes both the SMAV and GMAV for subsequent criteria calculations.

Geometric means are used to calculate central tendency species mean, genus mean, ACR and BCF values throughout the development of water quality criteria. This is because toxicity data and ratio data (ACRs and BCFs are ratios) tend to be lognormally distributed instead of normally distributed.

Acute water quality criteria are calculated by rank ordering the GMAV values from the lowest LC_{50} to the highest LC_{50}, and using a formula given in Stephan *et al.* (1985) to estimate the 5^{th} percentile of the resulting species sensitivity distribution (SSD). This 5^{th} percentile of measured GMAVs is termed the final acute value (FAV) in the EPA criteria development documents. As a criterion based on a concentration causing mortality to 50 percent of a test species would not be a protective criterion, the FAV is divided by two to convert LC_{50} values to concentrations expected to cause little or no mortality to test species. The FAV divided by two value becomes the EPA acute water quality criterion unless a commercially or recreationally important species, or an ESA listed species has a GMAV lower than the calculated water quality criterion. In these cases, the results of one or more individual species GMAVs is used to directly calculate an acute criterion.

If sufficient chronic NOEC data are available for the freshwater and/or saltwater taxa described earlier, the same approach described above is used with the measured NOEC data to calculate a final chronic value (FCV) from the 5^{th} percentile of the NOEC data. Final chronic values are not divided by two to obtain the chronic criterion, as unlike LC_{50} data, NOEC values are already assumed to be concentrations that have no adverse effects on survival, reproduction and growth of the tested species. Much more common is the situation where the calculated acute criterion is divided by an acute-chronic ratio (ACR) to obtain the chronic criterion.

Additional details of the Guidelines to develop national water quality criteria and the assumptions that go into their derivation are provided in Stephan *et al.* (1985). Of all the assumptions that are made during the derivation of EPA water quality criteria, perhaps the most critical is that the species sensitivity distribution of

measured toxicity data used during the calculation of criteria values is representative of the range of toxicity of a chemical to all aquatic species. There are over 700 species of freshwater fish alone in North America, making it impractical to perform toxicity tests on all species with all chemicals for which criteria exist.

Water quality criteria calculated from the methodology described above have several levels of conservatism built into them, including:

- protection of 95 percent of all aquatic genera
- division of the 5th percentile of all genus mean acute values by two during the derivation of acute criteria
- use of no effect concentrations to derive chronic criteria
- short exposure durations at criteria concentrations relative to the lifespan of many aquatic species

However, water quality criteria are not designed to protect all aquatic species from exposure to chemical concentrations that may adversely affect some of the more sensitive species to a given chemical. Nor are criteria designed to protect all individuals of a given species, whether or not that species is a listed species. Despite these design aspects of the national water quality criteria, many of them are protective of more than 95 percent of aquatic genera from adverse effects, and are protective of all ESA listed species known to occur within many discrete geographical areas. ESA listed aquatic species as a group are generally not believed to be more sensitive to chemicals than aquatic species as a whole (Dwyer *et al.* 2005, Sappington *et al.* 2001, Dwyer *et al.* 1999).

The toxic criteria proposed by the State of Oregon for EPA approval are identical to the corresponding national toxic criteria developed by EPA as guidance for the states.

The following section provides NMFS' analysis on the Guidelines.

Risks from Using Acute Criteria Based on LC_{50} Concentrations and the EPA Acute Adjustment Factor. The acute criteria for aquatic life have been primarily based on compilations of toxicity study results reported in terms of the concentration resulting in 50 percent mortality over a fixed time period [usually 96 hours: *e.g.*, LC_{50}, effects concentration $(EC)_{50,}$ EPA 1986a]. Although there are a number of reasons why data are not included in the data sets used to develop criteria, some of the more common ones are that one or more pieces of information regarding study methodology or calculation of results needed to assess the reliability of the study is missing; data quality of the study is less than acceptable (*e.g.* unacceptably high control mortality); the test species was exposed to a chemical mixture or was previously exposed to the test chemical; the study reported effects on an endpoint other than survival, reproduction or growth; or the test duration was a non-standard test duration (*e.g.*, fish toxicity test reporting a 24-hr LC_{50} instead of the more standard 96-hr LC_{50}).

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, that indicate the concentration at which 50 percent of the test population was killed. However, what is often

not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias the magnitude of acute toxic effects. Theses factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that are protective against acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve, and challenge the notion that LC_{50} data that is above the acute criterion is protective against acute toxic effects based soley on a comparison of concentrations.

Acute water quality criteria are calculated by rank ordering the GMAV values from the lowest LC_{50} to the highest LC_{50}, and using a formula given in Stephan *et al.* (1985) to estimate the 5^{th} percentile of the resulting SSD. This 5^{th} percentile of measured GMAVs is termed the FAV in the EPA criteria development documents. As a criterion based on a concentration causing mortality to 50 percent of a test species would not be a protective criterion, EPA divides the FAV by a safety factor of 2.27 (referred to as a factor of 2 in the below analysis) to convert LC_{50} values into concentrations that EPA projects to be near or below lethality.

The database from which the safety factor was derived (actually the safety factor is 2.27) was published in the Federal Register in 1978. Table 10 from the Federal Register notice (43 FR 21506-21518) lumps data for freshwater and marine fish and invertebrates. The data are broken out by the chemicals tested. There are 219 data points, but a large proportion of them aren't for a specific chemical, but rather for whole effluents of various sources—115 of the 219 data points used to derive the acute adjustment factor are based on effluent studies where individual pollutants are not measured. Interestingly, effluent studies are one of EPA's "not pertinent" or "reject" categories identified in EPA (2005).

The assumption that dividing an LC_{50} by 2 will result in effect concentrations near or below leathility rests on further assumptions of the steepness of the concentration-response slope. Several examples of tests with metals which had a range of response slopes are shown in Figure A1. These examples were selected from data sets that were relevant to salmonid species in Oregon and for which the necessary data to evaluate the range of responses could be located (Chapman 1975, 1978b, Marr *et al.* 1995, Marr *et al.* 1999, Mebane *et al.* 2010, Windward 2002). The citations given include both reports with detailed original data as well as the summarized, published forms of the same tests. The examples range from tests with some of the shallowest concentration-response slopes located to very steep response slopes. In the shallowest tests (panels A and E), an $LC_{50/2}$ concentration would still result in 15 to 20 percent mortality.

One challenge for deriving acute criteria for short-term exposures is that the great majority of available data is for mortality; that is, a concentration that kills 50 percent of a test population. A fundamental assumption of EPA's criteria derivation is that the FAV, which is the LC_{50} for a hypothetical species with a sensitivity equal to the 5^{th} percentile of the SSD, may be divided by 2 in order to extrapolates from a concentration that would likely be extremely harmful to sensitive species in short-term exposures (*i.e.*, kill 50 percent of the population) to a concentration

expected to kill few, if any, individuals. This assumption must be met for acute criteria to be protective of sensitive species. It is difficult to evaluate from published literature if this assumption is met because so few studies report the data behind an LC_{50} test statistic. While LC_{50}s are almost universally used in reporting short-term toxicity testing, they are not something that can be "measured," but are statistical model fits. An acute toxicity test is actually a series of 4 to 6 tests runs in parallel in order to test effects at these (usually) four to six different chemical concentrations. An LC_{50} is estimated by some statistical distribution or regression model, which generates an LC_{50} estimate, and some confidence interval, and then all other information is thrown away. Thus, while the original test data included valuable information on what were no, low and severe effects concentrations, that information is lost to reviewers unless the unpublished, raw, lab data are available. However, a more common pattern with the metals data was that an $LC_{50/2}$ concentration would probably result in about a 5 percent death rate (panels B and F), and in many instances, no deaths at all would be expected (panels C and D).

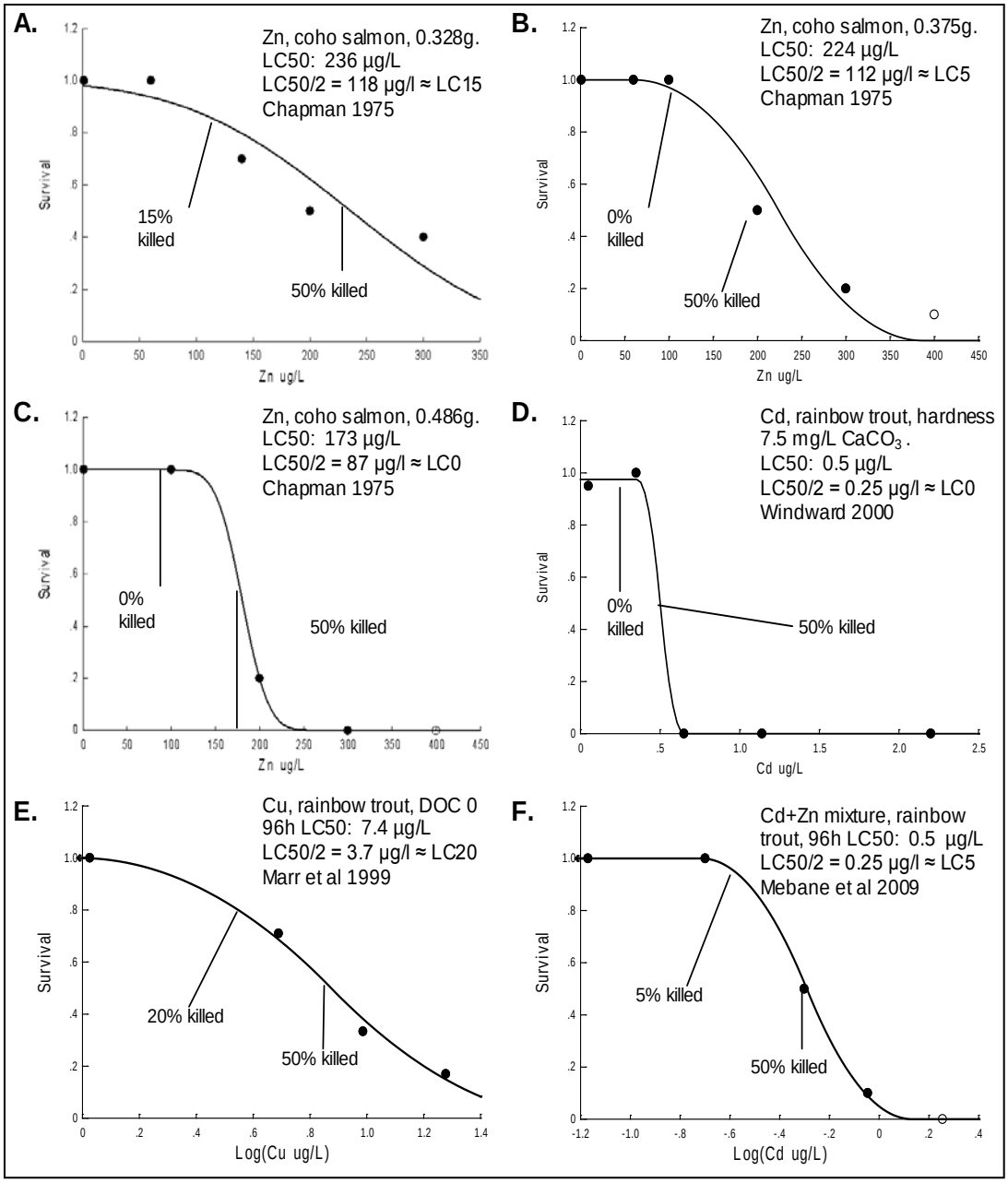

Figure A1. Examples of percentages of coho salmon or rainbow trout killed at one-half their LC_{50} concentrations and at LC_{50} concentrations with cadmium, copper, and zinc.

In one of the few additional published sources that gave relevant information, researchers happened to include effect-by-concentration information on the acute toxicity of chemical mixtures. Rainbow trout and the invertebrate zooplankton *Ceriodaphnia dubia* were exposed for 96 and 48 hours respectively to mixture of six metals, each at their presumptively "safe" acute CMC concentrations. In combination, the CMC concentrations killed 100% of rainbow trout and C. dubia, but 50% of the CMC concentrations killed none (Spehar and Fiandt 1986). This gives some support to the assumption that one-half the FAV divided by 2 is likely to kill a low

percentage of fish, although it raises questions about the overall protectiveness of criteria concentrations in mixtures.

Other relevant reviews include Dwyer *et al.* (2005b), who evaluated the $LC_{50/2}$ assumption with the results of the acute toxicity testing of 20 species with five chemicals representing a broad range of toxic modes of action. In those data, multiplying the LC_{50} by a factor of 0.56 resulted in a low (10%) or no-acute effect concentration. Testing with cutthroat trout and Cd, Pb, and Zn singly and in mixtures, Dillon and Mebane (2002) found that the $LC_{50/2}$ concentration corresponded with death rates of 0 to 15 percent.

Summary: Based on this analysis, there are increased risks to listed species considered in this opinion from using acute criteria based on LC_{50} concentrations and the acute adjustment factor, as acute criteria based on a hazard quotient—the acute adjustment factor, instead of acute toxicity tests that predict in $LC_{near\text{-}zero}$ concentrations, and are based on fixed duration toxicity tests instead of an exposure-response curve, are likely to underestimate the magnitude of effects for field-exposed fishes. Therefore, the risks identified in the above analysis are likely to result in mortality greater than the LC_{50} test predictions and the presumed protection from the acute adjustment factor in deriving acute criteria.

Risks from Using the Chronic Value Statistic in Setting Criteria. An issue of concern with the derivation of the chronic criteria is the test statistic used to summarize chronic test data for species and genus sensitivity rankings. Literature on chronic effects of chemicals often contains a variety of measurement endpoints, different terms, and judgments by the authors of what constitutes an acceptable or negligible effect. While the Guidelines give a great deal of advice on considerations for evaluating chronic or sublethal data (Stephan *et al.* 1985, at p. 39), those considerations were not usually reflected in the individual national EPA-recommended ambient water quality criteria documents NMFS reviewed. In practice, for most of the criteria documents we reviewed, "chronic values" were simply calculated as the geometric mean of the lowest tested concentration that had a statistically significant adverse effect at the 95 percent confidence level (LOEC), and the next lower tested concentration (NOEC). The "chronic value" as used in individual criteria documents is effectively the same thing as the maximum acceptable toxicant concentration[20] (MATC) used in much environmental toxicology literature, even though the MATC term is never used in the Guidelines. This MATC approach has the potential to seriously underestimate effects because the statistical power in typical toxicity tests is fairly low. A bias in many ecotoxicology papers is to focus on avoiding "false accusations" of a chemical with 95 percent accuracy (*i.e.*, Type I error or false positive, the risk of declaring an effect was present when in fact there was no effect). Often no consideration whatsoever is given to the companion problem, known as Type II error, or false negatives (*i.e.*, declaring no adverse effects occurred when in fact they did occur, but because of the limited sample size or variability, they were not significant with 95 percent confidence).

The magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be large (greater than 30 percent on average for some endpoints), and much higher for individual tests (Crane and Newman 2000). This problem is compounded when the "chronic value" or MATC is calculated in its most common form as the geometric mean of a NOEC and

[20] The MATC is the range between the NOEC and LOEC.

LOEC. For instance, in one study, 100 percent of juvenile brook died after being exposed to 17 µg/L copper for 8 months; this was considered the LOEC for the test. The next lowest concentration tested (9.5 µg/L) had no reduced survival relative to controls. (McKim and Benoit 1971). Therefore, the only thing that can be said about the geometric mean of these two effect concentrations (*i.e.*, the chronic value of 12.8 µg/L that was used in the chronic copper criteria, EPA 1985) is that it represents a concentration that can be expected to kill somewhere between all and no brook trout in the test population. These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that are protective against chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

Suter *et al.* (1987) evaluated published chronic tests with fish for a variety of chemicals and found that, on average, the MATC represented about a 20 percent death rate and a 40% reduction in fecundity. They noted that "although the MATC is often considered to be the threshold for effects on fish populations, it does not constitute a threshold or even a negligible level of effect in most of the published chronic tests. It corresponds to a highly variable level of effect that can only be said to fall between 0 and 90 percent." Barnthouse *et al.* (1989) further extrapolated MATC-level effects to population-level effects using fisheries sustainability models and found that the MATC systematically undervalued test responses such as fecundity, which are both highly sensitive and highly variable.

One implication of this issue is that because the MATC chronic values typically used in the EPA water quality criteria documents for aquatic life criteria may cause a substantial adverse effect for that test species, the criteria on the whole will be less protective than the Guidelines' intended goal of protecting 95 percent of the species. How much less protective is unclear and probably varies among the criteria datasets. One dataset from which a hypothetical NOEC-based chronic criterion could readily be recalculated and compared with the usual MATC criteria was a 2006 cadmium criteria update (Mebane 2006). In this comparison, Mebane determined that the MATC-based chronic criteria would protect about 92 percent of the aquatic species in the dataset at the NOEC level. Because the NOEC statistic also can reflect a fairly sizable effect (Crane and Newman 2000) it may be that at least with cadmium, the true level of protection is closer to about 90 percent than the 95 percent intended by the guidelines.

Summary: Based on this analysis, there are increased risks from using the chronic value statistic in setting criteria is high, as it is likely to result in sublethal effects, such as interference

in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Risks from the CMC and CCC Duration and Frequency of Exposure. The CMC and the CCC are just two of six parts of an aquatic life criterion; the other four parts are the acute averaging period, the chronic averaging period, acute frequency of allowed exceedence, and chronic frequency of allowed exceedence (EPA 2006), refered to as the concentration-duration-frequency format (EPA 1991).

Concentration (magnitude) refers to how much of a pollutant, expressed as a concentration, is allowable. Duration refers to the period of time (averaging period) over which the instream concentration is averaged for comparison with criteria concentrations. This specification limits the duration of concentrations above the criteria. And, frequency refers to how often criteria can be exceeded (EPA 1991).

The 1-hour CMC averaging period means that the 1-hour average concentration of the compound does not exceed the CMC more than once every three years on the average. In other words, an organism should not be exposed to a pollutant concentration *greater* than the CMC for more than 1 hour, and an exceedence, *i.e.*, a concentration *greater* than the respective CMC, of the CMC 1-hour average concentration should not occur more than once every three years on the average. The 4-day CCC averaging period means the 4-day average concentration of the compound does not exceed the CCC more than once every three years on the average. In other words, an organism should not be exposed to a pollutant concentration *greater* than the CCC for more than 4 days, and an exceedence, *i.e.*, a concentration *greater* than the respective CCC, of the CCC 4-day average concentrations should not occur more than once every three years on the average.

This means that the averaging periods are average concentrations that are measured against the respective numeric parts of the criterion with the purpose being to minimize the duration of exposure above the CMC and CCC criteria concentrations. Figures A2 and A3 provide conceptual examples of the 1-hour and the 4-day chemical averaging periods for acute and chronic criteria, respectively. These figures show that excursions (short term concentrations above the CMC or CCC) can produce concentration “spikes” that, when compared to the available toxicity data, can result in exposure with lethal and sub-lethal responses in listed species, but that the average concentration is below the respective criterion and thus in compliance.

Figures A2 and A3 conceptually represent respective averaging concentrations for acute and chronic criteria. For example, the 1-hour averaging concentration must be evaluated for each hour of the day. That is, the average concentration in the acute example of 55.2 μg/L is a series of continuous (persistent) receiving water concentrations that occurs each hour on a continuum. The same holds true for the chronic average concentration, where the 4-day average concentration in the chronic example of 23.7 μg/L is a series of continuous (persistent) receiving water concentrations that occurs on a continuum. In these examples, the chronic criterion concentration will eventually determine the concentration outside the regulated mixing zone [defined as an area where an effluent discharge undergoes initial dilution and is…an allocated impact zone where water quality criteria can be exceeded as long as acutely toxic conditions are

prevented (EPA 1991)] boundary, and is a more accurate representation of ambient concentrations outside of regulated mixing zones. Inside regulated mixing zones, water quality criteria are permitted to be higher than criterion concentrations. While a particular toxic criterion must be met at the acute and chronic mixing zone boundaries, mixing zone boundaries vary with flow and discharge. For example, based on publically-available information from ODEQ analyzed by NMFS in this consultation, in the Willamette River mixing zone size varies greatly from a low of 1,089 square feet to a high of 1,000,000 square feet (n=19). So, meeting the aquatic life criteria at the edge of the mixing zone is a misleading protective assumption.

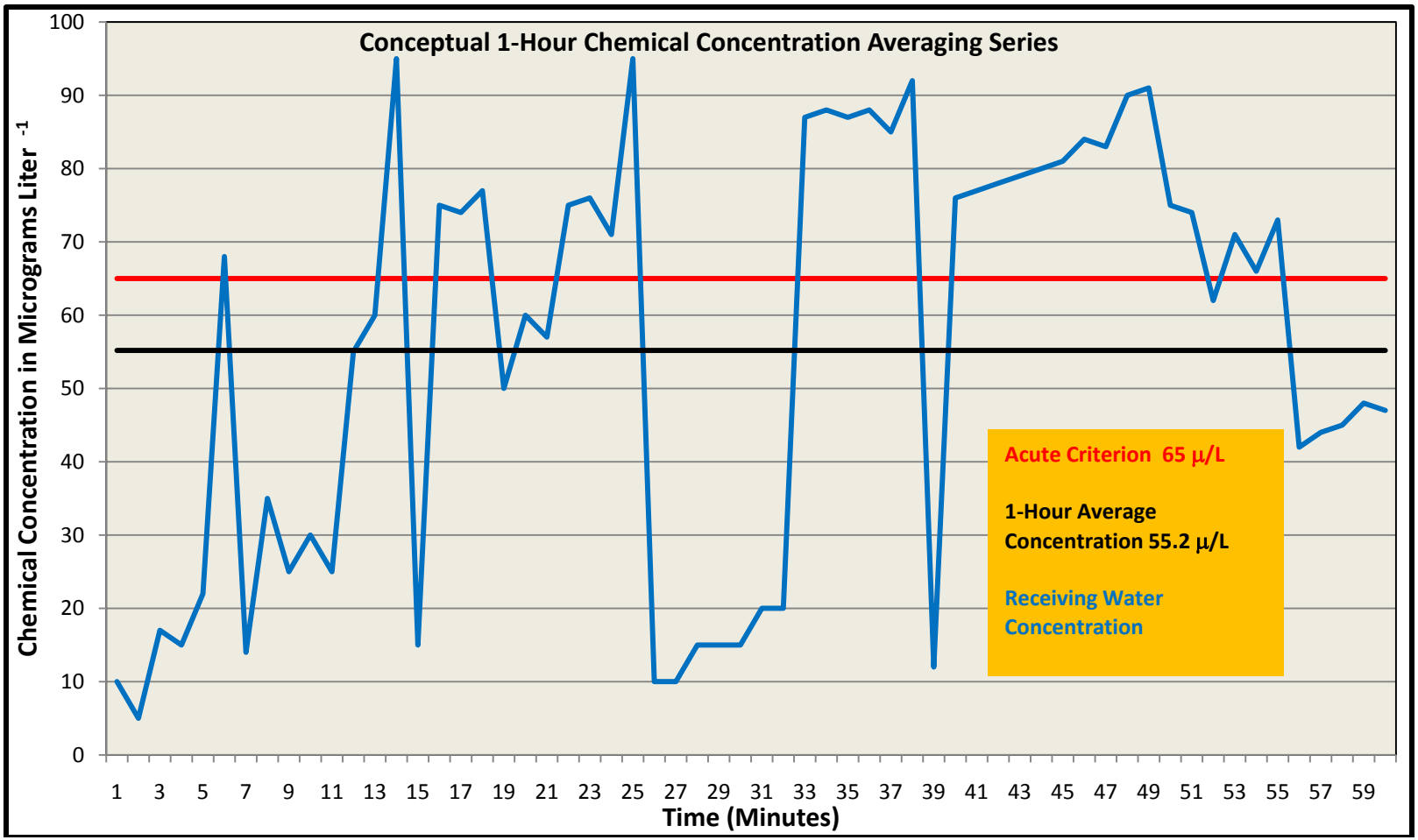

Figure A2. Conceptual concentration averaging series for acute criteria.

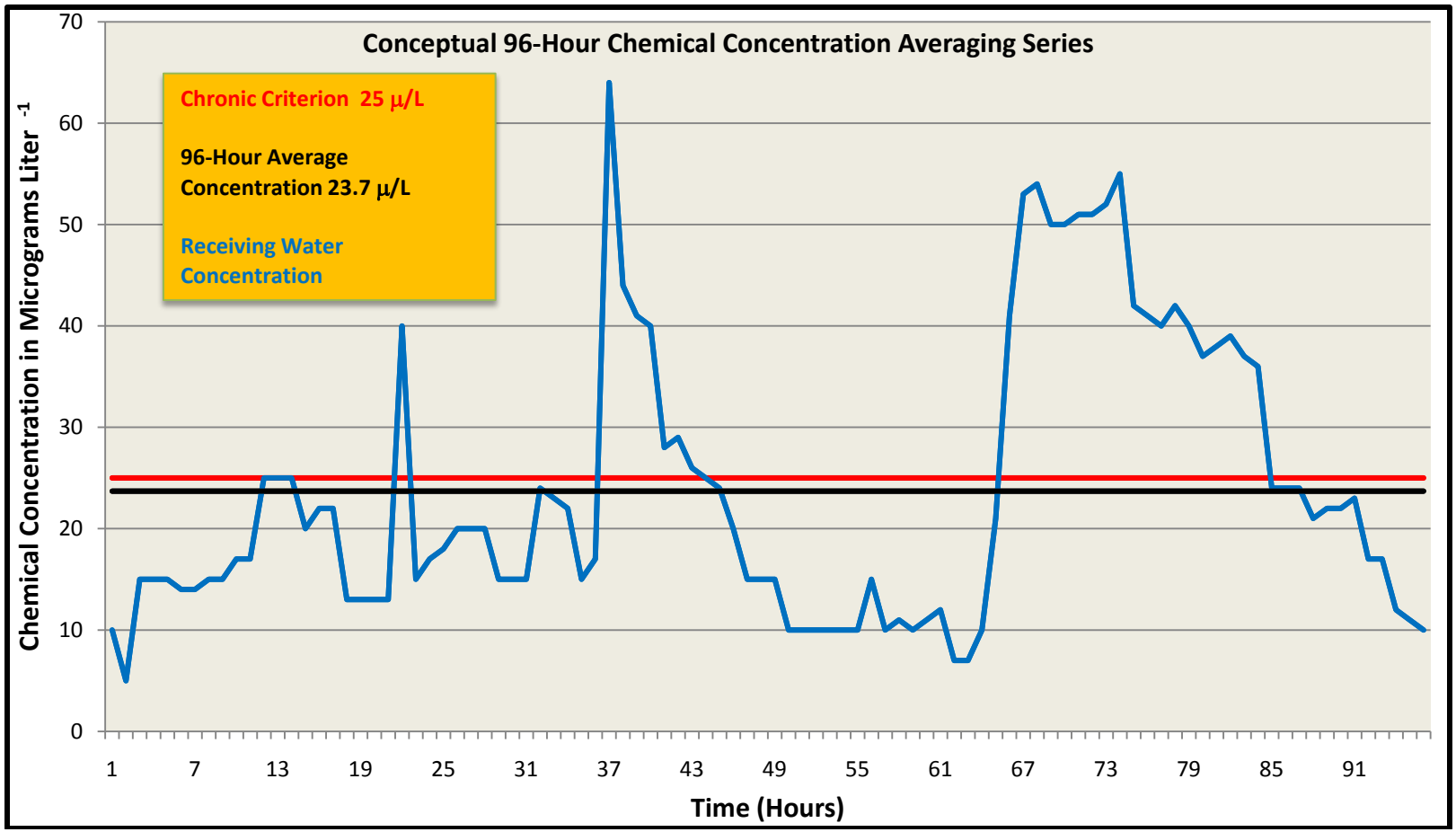

Figure A3. Conceptual concentration averaging series for chronic criteria.

Outside regulated mixing zones, chemical concentrations are theoretically lower than the proposed criteria, especially the acute criteria. However, waters that are 303(d)-listed for toxics do not meet water quality standards for toxics. So the assumption of lower concentrations at the edge of mixing zones is not met. That is, there is no assimulative capacity outside mixing zones.

The 1-hour and 4-day durations and averaging periods for criteria were based upon judgments by EPA authors that included considerations of the relative toxicity of chemicals in fluctuating or constant exposures. EPA's (1985) Guidelines considered an averaging period of one hour most appropriate to use with the criterion maximum concentration or (CMC or acute criterion) because high concentrations of some materials could cause death in one to three hours. Also, even when organisms do not die within the first few hours, few toxicity tests continue to monitor for delayed mortality after the exposure period is over. Thus it was not considered appropriate to allow concentrations *above* the CMC for more than one hour (Stephan *et al.* 1985). Recent criteria documents (*e.g.*, USEPA 2007) have used an averaging period of 24 hours for their CMC, although no explanation could be found for the deviation from the 1985 Guidelines.

A review of more recent information did not contradict these judgments. Some of the more relevant research relates the rapid accumulation of metals on the gill surfaces of fish to their later dying. When fish are exposed to metals such as cadmium, copper, or zinc, a relatively rapid increase occurs above background levels of metal bound to the gill. This rapid increase occurs on the order of <3 to 24 hours, and this brief exposure has been sufficient to predict toxicity at 120 hours (Di Toro *et al.* 2001, MacRae *et al.* 1999, Playle 1998, Playle *et al.* 1993). Acute exposures of 24-hours might not result in immediate toxicity, but deaths could result over the next few days. Simple examination of the time-to-death in 48 or 96 hour exposures would not detect latent toxicity from early in the exposures. Observations or predictions of appreciable mortality resulting from metals exposures on the order of only three to six hours supports the earlier recommendations by Stephan *et al.* (1985) that the appropriate averaging periods for the CMC is on the order of one hour.

The 4-day averaging period for chronic criteria was selected for use with the CCC for two reasons (Stephan *et al.* 1985): First, "chronic" responses with some substances and species may not really be due to long-term stress or accumulation, but rather the test was simply long enough that a briefly occurring sensitive stage of development was included in the exposure (Barata and Baird 2000, Chapman 1978a, De Schamphelaere and Janssen 2004, Grosell *et al.* 2006b, Mebane *et al.* 2008). Second, a much longer averaging period, such as 1 month would allow for substantial fluctuations above the CCC. Substantial fluctuations may result in increased adverse effects from those expected in constant exposures. A comparison of the effects of the same average concentrations of copper on developing steelhead, *Oncorhynchus mykiss*, that were exposed either through constant or fluctuating concentrations found that steelhead were about twice as resistant to the constant exposures as they were to the fluctuating exposures (Seim *et al.* 1984). The literature reviewed by NMFS either supports or at least does not contradict the Guidelines' recommendations on averaging periods.

In addition to the averaging periods, the Guidelines recommend for exceedence of the CMCs and the CCCs once every three years, on average. This recommendation was based on a review case studies of recovery times of aquatic populations and communities from locally severe

disturbances such as spills, fish eradication attempts, or habitat disturbances (Yount and Niemi 1990, Detenbeck *et al.* 1992). In most cases, once the cause of the disturbance was lifted, recovery of populations and communities occurred on a time frame of less than three years. The EPA has subsequently further evaluated the issue of allowable frequency of exceedences through extensive mathematical simulations of chemical exposures and population recovery. Unlike the case studies, these simulations addressed mostly less severe disturbances that were considered more likely to occur without violating criteria (Delos 2008). Unless the magnitude of disturbance was extreme or persistent, this three-year period seemed reasonably supported or at least was not contradicted by the information reviewed by NMFS.

A more difficult evaluation is the allowable exceedence magnitude, which is undefined and unlimited by the proposed criteria. Thus, theoretically, a once-per three year exceedence with no defined limits to its magnitude could be infinitely large, and have adverse effects on listed species. This is because environmental data such as chemical concentrations in water are not unpredictable, but can be described with statistical distributions and statements of exceedence probabilities. Commonly with water chemical data and other environmental data, the statistical distributions do not follow the common bellcurve or normal distribution, but have a skewed distribution with more low than high values. This pattern may be approximated with a log-normal statistical distribution (Blackwood 1992, Delos 2008, Helsel and Hirsch 2002, Limpert *et al.* 2001).

An important consideration that is often not addressed in water quality monitoring is the issue of sampling frequency. In order to accurately compare water quality samples with regulatory criteria, samples need to be collected at least at the same frequency as the criteria (*i.e.*, every hour for CMC and every four days for CCC). Otherwise, an exceedence could occur without detection. Samples, however, are not often taken at the specified frequency, and instead exceedence is detected indirectly through observed fish kills.

Summary: Based on this analysis, the duration and frequency parts of an aquatic life criterion seem like reasonable measures to keep the numeric criteria from exceeding criteria concentrations over long periods. However, the issue of excursions, exceedences with no defined limits on magnitude, and water quality monitoring and sampling sufficient to detect exceedences poses adverse risks likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Metals Toxicity and Risks from Using Formula-based Metal Criteria. Pursuant to EPA policy, states may adopt criteria for metals measured as either the amount of metal dissolved in water or the total recoverable amount of metal. For dissolved criteria, water samples are filtered to remove any suspended solids before analysis, and a conversion factor (CF) is applied to add back a fraction of the suspended metal based on assumptions regarding bioavailability. Total recoverable metals criteria are a measurement of the suspended and dissolved amounts added together. In its National Toxics Rule (NTR) (58 FR 31177), EPA originally promulgated criteria for metals as total recoverable metals. Subsequently, EPA issued a new policy for setting water quality criteria for metals measured as dissolved metals and promulgated revised national metals criteria expressed in terms of dissolved metals (60 FR 22228, May 4, 1995). At the same time,

EPA promulgated recommended conversion factors for converting between dissolved and total recoverable criteria. The metals criteria in Oregon are expressed as dissolved metals, meaning that water samples are filtered to remove suspended solids before analysis.

Metals addressed in this consultation include: As, Cd, Cr(III), Cr(VI), Cu, Pb, Ni, Se, Ag, and Zn. The proposed ambient water quality criteria are formula-based, meaning that the criteria vary based on site-specific conditions, for the following metals: As, Cd, Cr(III), Cr(VI), Cu, Pb, Ni, Ag, and Zn. To determine criteria for these metals that are applicable to a given water body, site-specific hardness data must be obtained, input to a formula, and numeric criteria computed. There are three types of site-specific data that may be necessary to determine and/or modify the criterion for a metal at a site: water hardness, conversion factors (CF) and translators, and water effect ratios (WER). The following is a brief description of these types of data.

The general formula for a hardness-based acute (CMC) or chronic (CCC) criterion with respect to total metal concentration (dissolved and particulate) is:

$$\text{CMC or CCC (total recoverable)} = e^{(m[\ln(\text{hardness})]+b)}$$

Note that this is algebraically equivalent to the simpler expression:

$$\text{CMC or CCC (total recoverable)} = K\,(\text{hardness})^{m}$$

where $K = e^{b}$. When the m-exponent is close to 1.0, the relationship is approximately linear. Dissolved concentrations are evaluated using a total-to-dissolved CF that is based on the fraction of the metal that was in a dissolved form during the laboratory toxicity tests and that was used to develop the original total based criteria. The appropriate formula is:

$$\text{CMC or CCC (dissolved)} = CF \times e^{(m[\ln(\text{hardness})]+b)} = CF \times K \times (\text{hardness})^{m}$$

There is an added level of complexity in the computations of criteria for cadmium and lead because the CFs for these metals also vary with hardness.

If a total maximum daily load (TMDL) is needed to regulate discharges into an impaired water body, the dissolved criterion must be converted or translated back to a total value so that the TMDL calculations can be performed. The translator can simply be the CF (*i.e.*, divide the dissolved criterion by the CF to get back to the total criterion), or site-specific data on total and dissolved metal concentrations in the receiving water are collected and a dissolved-to-total ratio is used as the translator.

Formulae for all the metals listed above also include a WER, a number that acts as a multiplication factor. A WER is intended to account for the difference in toxicity of a metal in a site water relative to the toxicity of the same metal in reconstituted laboratory water. The reason is that natural waters commonly contain constituents which "synthetic" or "reconstituted" laboratory waters lack, such as dissolved organic compounds, that may act to bind metals and reduce their bioavailability. Where such constituents act to modify the toxicity of a metal in a site water compared to the toxicity of the same metal in laboratory water, a "water effect" is

observed. If no site-specific WER is determined, then the WER is presumed to be 1 and would not modify a formula result.

The EPA has provided specifications and guidance regarding procedures and requirements for determining "site-specific" WER values that include extensive comparative toxicity testing with several test organisms and statistical analysis of results. The example provided below only illustrates the basic principle in defining a WER value.

Example WER calculation:

Suppose the LC_{50} of copper in site water is 30 µg/L
Suppose the LC_{50} of copper in laboratory water is 20 µg/L
Assume a site hardness of 100 mg/L
The freshwater CF for copper = 0.96
Acute criteria (CMC) for total recoverable copper without the WER = 18 µg/L

$$WER = \frac{\text{Site } LC_{50}}{\text{Lab } LC_{50}} = \frac{30\ \mu g/L}{20\ \mu g/L} = 1.5$$

$$\text{Copper Site-Specific CMC} = WER \times CF \times e^{(m[\ln(40)]+b)}$$
$$= 1.5 \times 0.96 \times 18$$
$$= 24\ \mu g/L$$

In the NTR, the EPA described and required minimum and maximum hardness values (25 mg/L and 400 mg/L as $CaCO_3$, respectively) to be used when calculating hardness-dependent freshwater metals criteria. Most of the data that the EPA used to develop the hardness formulae were in the hardness range of 25 to 400 mg/L. Therefore, the EPA stated that the formulae were most accurate in that range.

Formula-based metals criteria are discussed as a group here because the key issues of how dissolved metal criteria are derived and the implications of using the present formulae are similar for each of them. Issues include the influence of hardness, site-specific water quality characteristics, and the speciation of metal considered. The present formula-based metal method in the Guidelines does not consider the environmental fate, transport, and transformations of metals in natural environments (specifically for As, Cd, Cr (III), Cr (VI), Cu, Pb, Ni, Ag, and Zn), nor the influence of other water quality constituents on toxicity, and therefore affords incomplete protection for listed species.

A direct pathway for dissolved metals into aquatic organisms is through the gills. Dissolved forms of metals can adsorb to particulate matter in the water column and enter organisms through various routes. Metals adsorbed to particulates can also be transferred across the gill membranes (Lin and Randall 1990, Playle and Wood 1989, Sorensen 1991, Wright *et al.* 1986). Planktonic and benthic invertebrates can ingest particulate metals from the water column and sediments and then be eaten by other organisms. Thus, dietary exposure may be a significant source of metals to aquatic and aquatic dependent organisms.

Although metals bound to sediments are generally less bioavailable to organisms, they are still present, and changes in the environment (*e.g.*, dredging, storm events, temperature, lower water levels, biotic activity) can significantly alter the bioavailability of these metals. The feeding habits of fish can determine the amount of uptake of certain metals. Piscivorous fish are exposed to different levels of metals than omnivorous and herbivorous fish. For example, cadmium is more commonly found in omnivorous fish tissues than in carnivorous fish tissues from the same location (Enk and Mathis 1977).

Listed species are exposed to metals not only through the dissolved fraction in ambient waters, but they are also exposed to toxic effects of particulate metals through the mechanism of respiratory uptake in fish and by ingestion of contaminated particulate material. In addition, Finlayson *et al.* (2000) determined that metal-laden sediments in Keswick Reservoir, California were toxic to rainbow trout when re-suspended in moderately alkaline (pH 7.8) and soft (38 mg/L) water and elutriated. As fish respire, a nearly continuous flow of water passes across their gills (Moyle and Cech 1988) and particulate metals suspended in the water column may become entrapped. At the lowered pHs occurring near gill surfaces associated with gas exchange (Lin and Randall 1990, Playle and Wood 1989, Wright *et al.* 1986), entrapped particulate metals may release soluble metal ions, the form that is most bioavailable and efficiently taken up by aquatic organisms (EPA 1993a, 1997a). Although most research has been done on particulate exposures to gills of fish including salmonids, it is possible that other gill-breathing organisms (*e.g.*, aquatic macroinvertebrates) can be affected in the same way.

Current guidance for waste load allocation calculations (EPA 1996a) consists of simple dilution formulations using effluent metal loads, receiving water flows, and dissolved-to-total metals ratios in the receiving waters. Formula-based metal criteria are not protective of threatened or endangered aquatic species with respect to loading because the criteria development methods do not adequately consider the environmental fate, transport, and transformation of metals in natural environments. This concern is based in part on analyses conducted during the California Toxics Rule (CTR) consultation (USFWS and NMFS 2000), in which NMFS determined that substantial increases in total metals would be permitted in hypothetical discharges under the proposed criteria. The CTR analysis determined that as the fraction of particulate metal in the receiving water increases, the allowable discharge of particulate metals also increases rather than decreases. Such increases would be expected to occur through allowable TMDLs under the proposed ODEQ criteria because a TMDL is is based on the instream total metal concentration (EPA 1996a). Under Oregon's proposed water quality standards, total metal discharges may increase as long as the dissolved criteria are not exceeded.

Further, discharges from agricultural or urban non-point sources are largely uncontrolled through the discharge-permitting process. Metals criteria based only on dissolved concentrations provide little incentive for reducing non-point sources, which involve largely the particulate form. Thus, metals criteria based on dissolved concentrations in the absence of sediment criteria linked to total metals will not effectively prevent sediment contamination by metals and may lead to increased allowable loads of metals to sediments.

Formulae used to compute toxicity criteria for Cd, Cu, Cr(III), Pb, Ni, Ag, and Zn are presently functions of water hardness. By convention, hardness measurements are expressed in terms of

the equivalent concentration of $CaCO_3$ (expressed in mg/L) required to contribute that amount of calcium + magnesium hardness. Under the proposed criteria, hardness is determined for a site (expressed as mg/L of $CaCO_3$), and input to the criteria formulae for each metal. In natural waters considerable variation can occur in the calcium:magnesium ratio, contributing to site-specific water hardness. Studies show significant differences in toxicity for some metals depending on this ratio. In general, calcium provides greater reductions in toxicity. Site-specific hardness values with contributions from other multivalent cations (*e.g.*, iron, aluminum, manganese) that are evaluated using criteria based only on calcium + magnesium hardness result in site criteria that may not be protective. For example, in the case of cadmium, the presence of calcium is protective against toxicity whereas, magnesium, sodium, sulfate ions and the carbonate system appear to give little to no protection (Carroll *et al.* 1979). Welsh *et al.* (2000b) determined that calcium also afforded significantly greater protection against copper toxicity than magnesium.

The calcium:magnesium ratio in natural waters of Oregon varies substantially (Table A1).

Table A1. Total hardness for selected watersheds in Oregon in mg/L CaCO3. Data from USGS (1977).

Watershed	Mean	Standard Deviation	Range
Snake River ID-OR Border	141.3	33.7	97-190
Rogue River (RM 25)	37.5	5.1	30-45
John Day River	88.4	32.8	46-140
Deschutes River	41.5	2.7	37-45
Columbia River (RM 140)	69	11.8	45-94
Tualatin River	38.1	14.2	25-80
Willamette River (RM 10)	24	3.4	19-32
Nehalem River	18.9	6.5	12-32
Umpqua River	28.3	4.3	19-34

The majority of hardness data used to develop the EPA hardness-dependent criteria formulae were in the range of 25 mg/L to 400 mg/L (40 CFR Part 131). Consequently, EPA's regulations (40 CFR 131.36) specify that the minimum hardness that can be used in criteria equations is 25 mg/L. This requirement reflects that toxicity effects at hardness concentrations less than 25 mg/L are not known with a reasonable degree of certainty. Existing criteria formulae can result in toxic concentrations in water with hardness below the 25 mg/L lower threshold. There are some streams in Oregon where hardness concentrations average less than 25 mg/L, for which concentrations of contaminants with hardness ameliorated toxicity should be calculated on actual site conditions.

Comparable toxicity test data for hardness values greater than 400 mg/L appear to exist only for zinc, which precludes direct evaluation of the effects of extrapolating the criteria equations upwards. However, the ameliorating effect of increasing concentration of calcium ions means that the use of a default limiting value of 400 mg/L is protective for listed species in harder water in the case of metals for which toxicities are influenced by hardness.
The value of the site-specific hardness value will depend on where samples are collected. The calculated criteria may be less protective when samples are collected downstream of effluent

sources that may increase hardness locally (it is highly unlikely that discharges decrease downstream hardness). In otherwords, the use of hardness values measured downstream of the effluent source could lead to greater-than-intended site criteria. In some cases, certain effluents may alter ambient hardness, but not other important water quality constituents that influence metal toxicity (*e.g.*, pH, alkalinity, dissolved organic carbon, calcium, sodium, chloride, *etc.*). Alterations in receiving water chemistry by a discharge (*e.g.*, abrupt elevation of hardness, changes in pH, exhaustion of alkalinity, abrupt increases in organic matter *etc.*) could result, depending on the hardness value applied in the criteria formulae, in increased allowable discharges of toxic metals.

Water hardness and the hardness acclimation status of a fish will affect toxicity and toxic response. However the use of hardness alone as a universal surrogate for all water quality parameters that can modify metal toxicity will not always correlate well with the predicted toxic effect on listed species. The importance of water quality parameters other than hardness on metals toxicity has been understood for some time (Howarth and Sprague 1978). Numerous studies have been performed on the toxicity of metals in test waters of various compositions, and the results do not confer a singular role to hardness in ameliorating metals toxicity. Test water characteristics in most studies, including pH, calcium, alkalinity, dissolved organic carbon, chloride, sodium, suspended solids, and other chemical properties, are varied in a controlled manner while observing the responses of test organisms. It is likely that understanding metal toxicity in waters of various chemical makeups is not possible without the use of a geochemical model, and that a univariate regression formula will not suffice. It is also possible that simple toxicity tests (using mortality, growth, or reproductive endpoints) are not capable of discriminating the role of hardness relative to other water chemistry characteristics in modulating metals toxicity (Erickson *et al.* 1996).

Summary**:** Based on this analysis, using formula-based criteria for aquatic life criteria derived following the Guidelines are likely to be underprotective of listed species considered in this opinion. Formula-based metal criteria are discussed as a group here because the key issues of how dissolved metal criteria are derived and the implications of using the present formulae are similar for each of them. Issues include the influence of hardness, site-specific water quality characteristics, and the speciation of metal considered. The present formula-based metal method does not consider the environmental fate, transport, and transformations of metals in natural environments (specifically for arsenic, cadmium, chromium (III), chromium (VI), copper, lead, nickel, silver, and zinc), nor the influence of other water quality constituents on toxicity, and therefore affords incomplete protection for listed species and is likely to result in sublethal effects, such as central nervous system disruption, altered liver and kidney function, impaired reproduction, decreased olfactory response, delayed smoltification, impaired ability to avoid predation and capture prey, growth inhibition, growth stimulation, changes in prey species community composition (which will increase foraging budgets), and death of listed species considered in this opinion.

Additive and Synergistic Toxicity. When two or more toxic pollutants are present, their combined effect may be either additive, synergistic (where the net effect exceeds the sum of effects), or antagonistic. The proposed water quality standards do not take these effects into account. Relatively few toxicity studies have addressed this issue, and some studies have

indicated conflicting results due to complex interactions that vary with the combination(s) and concentrations involved (Sorenson 1991). However, a number of studies have determined conclusively that adverse effects due to additive or synergistic toxicity mechanisms occur when several criteria are near or equal to acute criteria concentrations (*e.g.*, Alabaster and Lloyd 1982, Spehar and Fiandt 1986, EIFAC 1987, Enserink *et al.* 1991, Sorenson 1991). Spehar and Fiandt (1986) determined that rainbow trout embryo survival and growth were not reduced when exposed to combinations of arsenic, cadmium, chromium, copper, and lead at chronic concentrations, but production and growth of *Daphnid sp.* were reduced for the same test mixtures. Combinations of organic pollutants also have been shown to result in different toxic responses, as have combinations of organic and metals contaminants.

Alabaster and Lloyd (1982) observed from their data that the combined acutely lethal toxicity to fish and other aquatic organisms is approximately the simple addition of the proportional contribution from each toxicant. The median value of the effect on fish is 0.95 of that predicted; the collective value for sewage effluents, river waters and a few industrial wastes is 0.85. The range for effluents, river wastes, and industrial wastes is 0.4 to 2.8, which indicates that the combined effects of individual acutely toxic pollutants are from 0.4 to 2.8 times the effects predicted by adding the individual effects. The median combined effect is approximately additive (EPA 1991).

Summary: Based on this analysis, the aquatic life criteria derivied following the Guidelines do not take into account additive or synergistic effects, thus increasing the likelihood of acute toxic effects and sublethals effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Assumption that Effects in Laboratory Tests are Reasonable Predictors of Effects in Field Situations. The preceding discussion concerned whether compilations of laboratorytest values were appropriate to treat as surrogates of the diversity of natural systems. A fundamental question in evaluating the Guidelines and the national criteria is whether tests of chemicals in laboratory aquaria with “domesticated” cultures of test animals are likely to produce similar effects as would exposure to the same substance on the same or closely related species in the wild. If the responses between animals in laboratory aquaria or the wild are different, is there a bias in the sensitivity of responses from either the lab or wild settings? That is, are the effects of chemical contamination likely more or less severe in the laboratory or wild settings? This question is important because water quality criteria are designed to apply to and protect ambient waters (that is, streams, rivers, and lakes), yet the data used to develop them are invariably compiled from laboratory testing under tightly controlled and thus quite artificial environments. There are myriad factors that may influence the effects of a chemical stressor on aquatic organisms, and this complexity makes the question of bias in sensitivity difficult or even impossible to answer with any certainty. The conclusion by Chapman (1983) regarding comparability of laboratory exposure-response effects and field exposure-response effects contributed to one the most fundamental assumptions in the Guidelines, that is, “the Guidelines have been developed on the theory that effects which occur on species in appropriate laboratory tests will generally occur on the same species in comparable field situations.” A number of reasons why the effects of a criteria chemical could be more or less severe on listed species in laboratory or in wild settings are summarized in Table A2.

Table A2. Factors influencing the effects of a chemical stressor in a laboratory setting or in the wild.

FACTOR	ARE EFFECTS LIKELY MORE SEVERE IN TYPICAL LAB SETTINGS OR IN THE WILD?
Environmental Conditions	
Nutritional state - acute test exposures	**In the wild:** In acute toxicity tests with fish fry, fish are selected for uniform size, and unusually underweight fish that might be weakened from being in poor nutritional state are culled from tests. For instance, if <90% of control fish survive the 4 days of starvation in an acute toxicity test, the test may be rejected from inclusion in the criteria dataset. In the wild, not all fish will be in optimal nutritional state. While perhaps counterintuitive, starvation can protect fish against waterborne copper exposure (Kunwar *et al.* 2009). Fish are routinely starved during acute laboratory tests of the type used in criteria development.
Nutritional state – chronic test exposures	**In the wild:** Fish in the wild must compete for prey, and if chemicals impair fish's ability to detect and capture prey because of subtle neurological impairment, this could cause feeding shifts and reduce their competitive fitness (Riddell *et al.* 2005). Fish in chronic lab tests with waterborne chemical exposures are often fed to satiation, and food pellets don't actively evade capture like live prey. Perhaps these factors dampen responses in lab settings.
Temperature	**In the wild:** In lab test protocols, nearly optimal test temperatures are recommended (*e.g.*, 12°C for rainbow trout, the most commonly tested salmonid). Fish may be most resistant to chemical insults when at optimal temperatures. At temperatures well above optimal ranges, increased toxicity from chemicals often results from increased metabolic rates (Sprague 1985); Under colder temperatures, fish have been shown to be more susceptible to at least Cu, Zn, Se and cyanide, although the mechanisms of toxicity are unclear (Dixon and Hilton 1985, Erickson *et al.* 1987, Hansen *et al.* 2002a, Hodson and Sprague 1975, Kovacs and Leduc 1982, Lemly 1993).
Flow	**In the wild:** Fish expend energy to hold their position in streams and to compete for and defend preferred positions that provide optimal feeding opportunity from the drift for the energy expended. Subordinate fish in the wild are forced to less profitable positions and become disadvantaged. Subordinate fish in lab settings still get adequate nutrition from feeding. Chemical exposure can reduce swimming stamina or speeds, as can exposure to soft water. (Adams 1975, De Boeck *et al.* 2006, Kovacs and Leduc 1982, McGeer *et al.* 2000).
Disease and parasites	**In the wild:** Disease and parasite burden are common in wild fish, but toxicity tests that used diseased fish likely were considered compromised and results likely were not used in criteria development. Chemical exposure may weaken immune responses and increase morbidity or deaths (Arkoosh *et al.* 1998, Stevens 1977).
Predation	**In the wild:** Fish use chemical cues to detect and evade predators; these can be compromised by some chemical exposures (Berejikian *et al.* 1999, Labenia *et al.* 2007, Phillips 2003, Scott *et al.* 2003)
Exposure	
Variable exposures	**In the lab:** Most toxicity tests used to develop criteria are conducted at nearly constant exposures. Criteria are expressed not just as a concentration but also with an allowed frequency and duration of allowed exceedences. In field settings, most point or non-point pollution scenarios that rarely if ever exceed the criteria concentration (*i.e.*, no more than for one 4-day interval per 3 yrs), will have an average concentration that is less than the criterion concentration. For some chemicals, such as copper, fish might detect and avoid harmful

FACTOR	ARE EFFECTS LIKELY MORE SEVERE IN TYPICAL LAB SETTINGS OR IN THE WILD?
	concentrations if clean-water refugia were readily available.
Metal form and bioavailability	**Uncertain:** Metals other than Hg and some organics are commonly more bioavailable in the lab because dissolved organic carbon, which reduces the bioavailability and toxicity of several metals, is low in laboratory tests that are eligible for use in criteria. The Guidelines call for <5 mg/L TOC (total organic carbon) in studies to be used in criteria (Stephan *et al.* 1985), but probably more often TOC is <2 mg/L in laboratory studies.
Chemical equilibrium	**Uncertain:** While results conflict, metals are usually considered less toxic when in equilibrium with other constituents in water, such as organic carbon, calcium, carbonates and other minerals. In the wild, daily pH cycles prevent full equilibria from being reached (Meyer *et al.* 2007). Likewise, in conventional laboratory flow-through tests, designs chemicals may not have long enough contact time to reach equilibrium. Static-renewal tests are probably nearly in chemical equilibria, although organic carbon accretion can lessen toxicity which may not reflect natural settings (Santore *et al.* 2001, Welsh *et al.* 2008).
Prior exposure	**Uncertain:** If fish are exposed to sublethal concentration of a chemical they could either become weakened or become more tolerant of future exposures. With some metals, normally sensitive life stages of fish may become acclimated and less sensitive during the course of a chronic test if the exposure was started during the resistant egg stage (Brinkman and Hansen 2007, Chapman 1983, 1985, Sprague 1985).
Life stages exposed	**In the wild:** Most lab studies are short term and realistically testing all life stages of anadromous fish is probably infeasible. Reproduction is often the most sensitive life stage with fish but most "chronic" studies are much shorter and just test early life stage survival and growth (Suter *et al.* 1987). At different life stages and sizes, salmonids can have very different susceptibility to some chemicals; even when limited to a narrow window of young-of-year fry, sensitivity can vary substantially. Unless the most sensitive life stages are tested, lab tests could provide misleadingly high toxicity values for listed species.
Chemical mixtures	**In the wild:** In field conditions, organisms never experience exposure to a single pollutant; rather, ambient waters typically have low concentrations of numerous chemicals. The toxic effects of chemicals in mixture can be less than those of the same chemicals singly, greater than, or have no appreciable difference. The best known case of one toxicant reducing the effects of another is probably Se and Hg (*e.g.*, Belzile *et al.* 2006). However, strongly antagonistic responses are probably uncommon, and much more common are situations where chemical mixtures have greater toxicity than each singly or little obvious interaction (*e.g.*, Borgert 2004, Laetz *et al.* 2009, Norwood *et al.* 2003, Playle 2004, Scholz *et al.* 2006). In general, it seems prudent to assume that if more than one toxicant were elevated, it is likely that lower concentrations of chemicals would be required to produce a given magnitude of effect than would be predicted from their actions separately.
Dietary exposures	**In the wild:** Toxicity test data used in criteria development have been mostly based solely on waterborne exposures, yet in the wild, organisms would be exposed to contaminants both through dietary and water exposures. With at least some organics and metals (*i.e.*, As, Se) dietary exposures are more important than water exposures. For some other metals (*i.e.*, Cd, Cu, Ni, Pb, Zn), at environmentally relevant concentrations that would be expected when waterborne concentrations are close to criteria, dietary exposures have not been shown to directly result in appreciable adverse effects on fish (Hansen *et al.* 2004, Schlekat *et al.* 2005). However, while dietary exposures of some metals have not yet been implicated in adverse effects on fish at or below criteria

FACTOR	ARE EFFECTS LIKELY MORE SEVERE IN TYPICAL LAB SETTINGS OR IN THE WILD?
	concentrations, they may in fact be both the primary route of exposure and an important source of toxicity for benthic invertebrates rather than fish (Buchwalter *et al.* 2008, Irving *et al.* 2003). For instance Besser *et al.* (2005a) found that the effects threshold for Pb to the benthic crustacean *Hyalella sp.* was well above the chronic criterion in water exposures, but when Pb was added to the diet, effects threshold dropped to near criteria concentrations. Ball *et al.* (2006) found that feeding Cd-contaminated green algae to the benthic crustacean *Hyalella* sp. caused a 50% growth reduction at about the NTR chronic criterion.
Population Dynamics	
Density effects	**In the lab:** Salmonid fishes are highly fecund (~500 to 17,000 eggs per spawning female). When abundant, overcrowding, and competition for food and shelter may result in relatively high death rates for some life stages, particularly young-of-year during their first winter. After many fish die in a density-dependent bottleneck, the survivors have greater resources and improved growth and survival. Conceptually, if an acute contamination episode killed off a significant portion of young-of-year fish prior to their entering a resource bottleneck, then assuming no residual contaminant effects, the losses to later life stages and to adult spawners could be buffered.
Meta-population dynamics	**In the lab:** If habitats are interconnected, as is the case in intact stream networks, and if pervasive contamination from discharges to a stream were to impair only some endpoints or life-stages, such as reproductive failure or YOY mortalities, immigration from source populations may make detection of population reductions in the affected sink population difficult (Ball *et al.* 2006, Palace *et al.* 2007). If an episodic contamination pulse were to kill a large proportion of fish in a stream, the proximity of refugia and donors from source populations affect recovery rates (Detenbeck *et al.* 1992).

Summary: Based on this analysis, the assumption that effects in laboratory tests as reasonable predictors of effects to species in the wild is dependent upon the specific factor being considered. Overall NMFS finds that laboratory tests are likely to underpredict effects, as adverse effects are generally likely to be more severe in the wild than under laboratory conditions. Thus aquatic life criteria derivied following the Guidelines are likely to result in sublethals effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Risks of Using Flow-Through, Renewal, or Static Exposure Test Designs. One area of controversy in evaluating toxicity test data or risk assessments, or criteria derived from them, has to do with potential bias in how test organisms are exposed to test solutions. Exposures of test organisms to test solutions are usually conducted using variations on three techniques. In "static" exposures, test solutions and organisms are placed in chambers and kept there for the duration of the test. The "renewal" technique is like the static technique except that test organisms are periodically exposed to fresh test solution of the same composition, usually once every 24 hours or 48 hours, by replacing nearly all the test solution. In the "flow-through" technique, test

solution flows through the test chamber on a once-through basis throughout the test, usually with at least five volume replacements/day (ASTM 1997).

The term “flow-through test” is commonly mistaken for a test with flowing water, *i.e.*, to mimic a lotic environment in an artificial stream channel or flume. This is not the case; rather the term refers to the once-through, continuous delivery of test solutions (or frequent delivery in designs using a metering system that cycles every few minutes). Flows on the order of about five volume replacements per 24 hours are insufficient to cause discernable flow velocities. In contrast, even very slow moving streams have velocities of around 0.04 ft/sec (an inch per second) or more. At that rate, a parcel of water would pass the length of a standard test aquarium (~2 ft) in about 48 seconds, resulting in about 9,000 volume replacements per day. A more typical stream velocity of about 0.5 ft/sec would produce over 100,000 volume replacements per day.

Historically, flow-through toxicity tests were thought to provide a better estimate of toxicity than static or renewal toxicity tests because they provide a greater control of toxicant concentrations, minimize changes in water quality, and reduce accumulation of waste products in test exposure waters (Rand *et al.* 1995). Flow-through exposures have been preferred in the development of standard testing protocols and water quality criteria. The Guidelines first advise that for some highly volatile, hydrolysable, or degradable materials, it is probably appropriate to use only results of flow-through tests. However, this advice is followed by specific instructions that if toxicity test results for a species were available from both flow-through and renewal or static methods, then results from renewal or static tests are to be discounted (Stephan *et al.* 1985). Thus, depending upon data availability, toxicity results in the criteria databases may be a mixture of data from flow-through, renewal or static tests, raising the question of whether this could result in bias. In the Guidelines, the rationale for the general preference for flow-through exposures was not detailed, but it was probably based upon assumptions that static exposures will result in LC_{50}s that are biased high (apparently less toxic) than comparable flow-through tests, or that flow-through tests have more stable exposure chemistries and will result in more precise LC_{50} estimates.

With metals, renewal tests produce higher EC_{50}s (*i.e.*, metals were less toxic), probably because of accretion of dissolved organic carbon (DOC) (Erickson *et al.* 1996, Erickson *et al.* 1998, Welsh *et al.* 2008). However, in contrast to earlier EPA and ASTM recommendations favoring flow-through testing, Santore *et al.* (2001) suggested that flow-through tests were biased low because copper complexation with organic carbon, which reduces acute toxicity, is not instantaneous, and typical flow-through exposure systems allowed insufficient hydraulic residence time for complete copper-organic carbon complexation to occur. Davies and Brinkman (1994) similarly found that cadmium and carbonate complexation was incomplete in typical flow-through designs, although in their study incomplete complete complexation had the opposite effect of the copper studies, with cadmium in the aged, equilibrium waters being more toxic. A further complication is that it is not at all clear that natural flowing waters should be assumed to be in chemical equilibria because of tributary inputs, hyporheic exchanges and daily pH, inorganic carbon, and temperature cycles. Predicting or even evaluating risk of toxicity through these cycles is complex and seldom attempted (Meyer *et al.* 2007), in part because pulse exposures cause latent mortality (*i.e.*, fish die after exposure to the contaminant is removed), a phenomenon that is often overlooked or not even recognized in standard acute toxicity testing.

When comparing data across different tests, it appears that other factors such as testing the most sensitive sized organisms or organism loading may be much more important than if the test was conducted by flow through or renewal techniques. For instance, Pickering's and Gast's (1972) study with fathead minnows and cadmium produced flow-through LC_{50}s that were lower than comparable static LC_{50}s (~ 4,500 to 11,000 µg/L for flow-through tests vs. ~30,000 µg/L for static tests). The fish used in the static tests were described as "immature," weighing about 2 g (2000 mg). The size of the fish used in their flow-through acute tests were not given, but is assumed to have been similar. In contrast, 8 to 9 day old fathead minnow fry usually weigh about 1 mg or less (USEPA 2002b). Using newly hatched fry weighing about $1/1000^{th}$ of the fish used by Pickering and Gast (1972) in the 1960s, and modern protocols, cadmium LC_{50}s for fathead minnows at similar hardnesses tend to be around 50 µg/L, with no obvious bias for test exposure. Similar results have been reported with brook trout. One each flow-through and static acute tests with brook trout were located, both conducted in waters of similar hardness (41 to 47 mg/L). The LC_{50} of the static test which used fry was < 1.5 µg/L whereas the LC_{50} of the flow-through test using yearlings was > 5,000 µg/L (Carroll *et al.* 1979, Holcombe *et al.* 1983).

Many studies on which the proposed criteria are based involve laboratory-based LC_{50} bioassays using static exposure systems and nominal contaminant concentrations. Such studies often yield LC_{50} values substantially higher than values obtained with flow-through tests or tests in which actual concentrations of contaminants in the system during the experiment are measured, with differences in some cases of an order of magnitude lower. For example, LC_{50} values for static tests have been determined to be approximately 20 times higher than those from flow-through tests for DDT (Earnest and Benville 1971). Mercury toxicity testing of trout embryos has indicated that concentration-based endpoints (*e.g.*, EC_{50}) could be as much as one to two orders of magnitude lower in flow-through than static tests (Birge *et al.* 1979, 1981). Static assays were also found to underestimate the toxicity of endosulfan in comparisons with flow-through systems (Naqvi and Vaishnavi 1993). Several additional studies with a variety of compounds report increased toxicity in flow-through compared to static systems (*e.g.*, Erickson *et al.* 1998, Hedtke and Puglisi 1982, Vernberg *et al.* 1977, Randall *et al.* 1983, Burke and Ferguson 1969). Static conditions may underestimate the true exposure concentration because the fish will deplete the concentration in solution over time, causing a lack of steady-state exposure. There may also be important differences in energy expenditure and metabolism of test fish between static and flow-through tests, depending on the experimental setup. In the case of listed salmonids in Oregon, this may be an important source of variation because they typically live in flowing waters. Acute LC_{50}s for salmonids that are based on static tests could therefore underestimate toxicity, and water quality standards based on such tests may consequently not be sufficiently protective against conditions reasonably expected to occur in Oregon waters.

Summary: Based on this analysis, using flow-through, renewal, or static exposure test designs may result in greater than predicted effects.

Effects of Acclimation on Susceptibility to Chemicals. Exposure to sublethal concentrations of organic chemicals and other metals may result in pronounced increases in resistance to later exposures of the organisms. With metals the resistance may be on the order of two to four times greater for acute challenges, but for some organic contaminants may be much higher (Chapman 1985). However, the increased resistance can be temporary and can be lost in

as little as seven days after return to unpolluted waters (Bradley *et al.* 1985, Hollis *et al.* 1999, Sprague 1985, Stubblefield *et al.* 1999). For this reason, the Guidelines specify that test results from organisms that were pre-exposed to toxicants should not be used in criteria derivation (Stephan *et al.* 1985).

Effects from acclimation, however, are not precluded by the Guidelines and influence chronic values and thus chronic criteria. Several tests have shown that at least with fish and metals, if the toxicity tests were initiated during more resistant early life stages (ELS, *e.g.*, embryo stage), acclimation may occur, and later in the test when the more sensitive life stages become exposed (*e.g.*, fry stage), the usually sensitive life stages may be more resistant than the same life stages of fish which had no pre-exposure (Brinkman and Hansen 2004; 2007, Chapman 1978a; 1994, Spehar *et al.* 1978).

Chapman (1994) exposed different life stages of steelhead (*Oncorhynchus mykiss*) for the same duration (three months) to the same concentration of copper (13.4 µg/L at a hardness of 24 mg/L as $CaCO_3$). The survival of steelhead that were initially exposed as embryos was no different than that of the unexposed control fish, even though the embryos developed into the usually-sensitive swim-up fry stage during the exposure. In contrast, steelhead that were initially exposed as swim-up fry, without the opportunity for acclimation during the embryo state, suffered complete mortality. Brinkman and Hansen (2007) compared the responses of brown trout (*Salmo trutta*) to long-term cadmium exposures that were initiated either at the embryo stage (*i.e.*, early-life stage tests) or the swim-up fry stage (*i.e.*, chronic growth and survival tests). In three comparative tests, fish that were initially exposed at the swim-up fry stage were consistently two to three times less resistant than were the fish initially exposed at the embryo stage.

These studies support the counterintuitive conclusion that because of acclimation, longer-term tests or tests that expose fish over their full life cycle are not necessarily more sensitive than shorter-term tests that are initiated at the sensitive fry stage. Conceptually, whether this phenomenon is important depends on the assumed exposure scenario. If it were assumed that spawning habitats would be exposed, then the less-sensitive ELS tests would be relevant. However, for migratory fishes such as listed salmon and steelhead, life histories often involve spawning migrations to headwater reaches of streams, followed by downstream movements of fry shortly after emerging from the substrates, and followed by further seasonal movements to larger, downstream waters to overwinter (Baxter 2002, Quinn 2005, Willson 1997). These life history patterns often correspond to common human development and metals pollution patterns where headwater reaches likely have the lowest metals concentrations, and downstream increases occur due to point source discharges or urbanization.

From the discussion of the types of chronic data with fish that are acceptable for use in criteria development, it is clear that the intent was to capture information on the most sensitive life stage of a fish species. Unfortunately, the wording of the Guidelines could be interpreted to preclude the use of the more-sensitive chronic growth and survival tests that were initiated with salmonid fry stage, and specify the use of the less-sensitive ELS tests (Stephan *et al.* 1985, p. 44).

Summary: Based on this analysis, the risks of acclimation on susceptibility to chemicals are likely to result in sublethal effects, such as interference in physiochemical processes,

interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Toxic Responses of Different Species and Life Stages. The chemical concentrations causing toxic effects differ between taxa, with some species being more sensitive than others. The EPA's national water quality criteria, on which the proposed criteria are based, were developed from toxicity data compiled for a wide range of species and life stages and were determined on the basis of protecting roughly 95% of the species considered. However, because the criteria were not developed specifically to protect the most sensitive species or life stage present, it is possible that the proposed criteria may not be protective when that species and life stage is a listed species, *i.e.*, a species at risk of extinction. This is recognized in the Guidelines which indicate that it is possible to revise the criteria if it is determined that there is a more sensitive species and life stage present (EPA 1994a).

The EPA identified SMAVs in their criteria documents for most of the pollutants subject to this consultation that differ between species of salmon and trout. SMAV's for marine mammals, sea turtles, green sturgeon, and eulachon have not been developed. However, the SMAVs were in most cases based on limited toxicity testing data collected under varying conditions, and therefore may not be indicative of actual species differences. Moreover, SMAVs are not completely protective of listed species because they represent an average condition, where lower concentrations may be toxic to those species under certain test conditions. There is evidence that under similar testing conditions, some trout species have similar toxic responses (*e.g.*, rainbow and brown trout, Cohen *et al.* 1993). There is also evidence of differences in toxicity response between species when exposed to specific metals or organic compounds under similar conditions (*e.g.*, Chinook and coho salmon, Hamilton and Buhl 1990; Chinook salmon, Chapman 1978b; rainbow and brook trout, Holcombe and Andrew 1978; brown trout, Chinook and coho salmon, Macek and Allister 1970, Katz 1961; rainbow trout, and Chinook and coho salmon, Macek *et al.* 1969, Katz 1961), so species differences cannot be completely discounted. Overall, however, experimental evidence (including data presented in the various EPA water quality criteria documents) suggests that there is greater variation in toxic response between life stages than between species within the family Salmonidae.

Since a species can only be considered protected from acute toxicity if all life stages are protected, EPA's Guidelines recommend that if the available data indicate that some life stages are more resistant than other life stages by at least a factor of two, the data for the more resistant life stages should not be used to calculate species mean acute values (Stephan *et al.* 1985). Smaller, juvenile life stages of fish are commonly expected to be more vulnerable to metals toxicity than larger, older life stages of the same species. For instance, a standard guide for testing the acute toxicity of fish (ASTM 1997) recommends that tests should be conducted with juvenile fish (that is, post-larval or older and actively feeding), usually in the size range from 0.1 to 5.0 g in weight.

A review of several data sets in which salmonids of different sizes were similarly tested shows that even among juvenile fish in the 0.1 to 5.0-g size range, differences in sensitivity can approach a factor of 10. This emphasizes the importance of EPA's Guidance not to use the more resistant life stages. However, the data sets analyzed by NMFS indicated that in practice, there

were sometimes greater influences of life stage on the sensitivity of salmonids to some substances than was apparent to the authors of the individual criteria documents using the datasets available to them at the time. Some of the SMAVs and GMAVs which were used to rank species sensitivity and set criteria were considerably higher than EC_{50}s for salmonids that were tested at the most sensitive life stages (Figure A4).

For three Pacific salmonid species for which comparable test data were available for different life stages (coho salmon (*O. kisutch*), rainbow trout (*O. mykiss*) and cutthroat trout (*O. clarki*), the data suggest that swim-up fish weighing around 0.5 g to about 1 g may be the most sensitive life stage. None of the data sets or published studies NMFS examined in detail had sufficient resolution to truly define what weight fish was most sensitive to metals, but along with other data they suggest that larger fish are less sensitive than fish at 0.4 to 0.5 g. For instance, with zinc, rainbow trout in the size range of about 0.1 to about 1.5 g were consistently more sensitive to zinc in two studies with multiple tests in that size range. The paucity of data with salmonids in the size range of about 0.5 to 2 g prevents definitive identification of a most sensitive size across species or even tests. All data located for early swim-up stage *Oncorhynchus* in the 0.1 to 0.5 g range were consistent with increasing sensitivity with size. With Hansen *et al.* (1999b) rainbow trout studies, this relationship continued with fish up to about 1.5 g. However, with cutthroat trout, the few data available suggests that fish larger than about 0.5 g are less sensitive with increasing size.

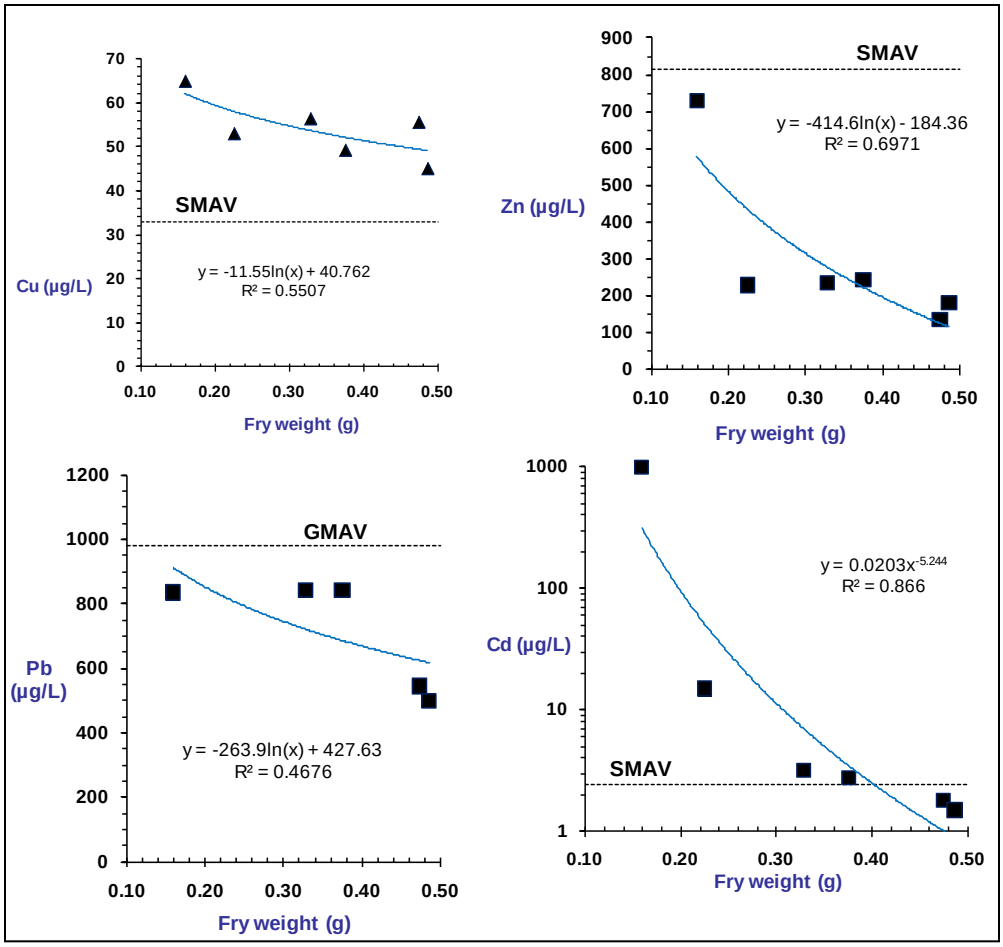

Figure A4. Size-developmental stage patterns SMAVs and GMAVs with coho salmon from 2 to 7 weeks posthatch, with data from Chapman (1975), and EPA (1984a, 1984b, 1985, 1987), adjusted to test water hardness. All tests used Willamette River water, TOC 3.4 mg/L, hardness 22 mg/L.

Some studies with older and larger rainbow trout have found that the fish became more resistant to zinc and copper (Chakoumakos *et al.* 1979, Chapman 1978b, Chapman and Stevens 1978, Howarth and Sprague 1978). Studies with copper all showed this trend, but the strength of size-sensitivity relations varied across studies. Chakoumakos *et al.* (1979) found that fish between about 1 and 25g in weight varied in their sensitivity to copper by about 8 times, but steelhead (*O. mykiss*) that were tested with copper at sizes of 0.2, 7, 70, and 2700 g showed little pattern of sensitivity with size (Chapman and Stevens 1978, Chapman 1978b). However, the large differences in sizes may have missed changes at intermediate sizes in the ranges compared (Figure A4). Similarly, with copper and rainbow trout, Anderson and Spear (1980) found that rainbow trout at sizes of 3.9, 29 to 176 g had similar sensitivities.

The NMFS reviewed several data sets indicated increasing susceptibility of salmonids to at least metals with increasing size and age as fish progressing from the resistant alevin stage. These

patterns indicate caution is needed when using SMAVs or GMAVs as a summary statistics for ranking species sensitivity or setting criteria.

Salmonids can have profound difference in susceptibility to chemicals at different life stages and in some instances SMAVs used in criteria may be skewed high because insensitive life stages were included. Across several good datasets, the most vulnerable life stage and size appeared to be swim-up fry weighing between 0.5 and 1.5g.

Summary: Based on this analysis, the risks from relying on toxicity data from species and life stages that are less sensitive than the most sensitive salmonid life stage is moderate to high, as aquatic life criteria derivied following the Guidelines is likely to result in sublethal effects, such as interference in physiochemical processes, interption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Bioconcentration and Bioaccumulation Factors Used in Determining and Evaluating Proposed Criteria Associated with High Variability and Uncertainty. An important problem with many of EPA's chronic criteria for organic pollutants is that the bioconcentration or bioaccumulation factors used in their determination may not be accurate. The BCFs determined in the laboratory based on water-borne exposure are typically much lower than field-derived values, and so may significantly underestimate uptake in the natural environment. Even among field-derived bioconcentration factors, estimates can vary by several orders of magnitude. Consequently, it is difficult to determine if BCF-based comparisons of water-borne and tissues concentrations are accurate when evaluating the chronic criteria proposed in this action.

The Guidelines include a component designed to assure that the water quality criterion for a substance is sufficiently low that residue accumulations will not impair the use of a waterbody by aquatic organisms, and specify that data from residue studies are to be considered alongside acute and chronic toxicity data in the criteria development process (EPA 1985a). However, metals criteria are presently based solely on results of aquatic toxicity tests (62 FR 42159), where metal exposures occur directly across gills or other respiratory surfaces.

Metals and organic contaminants can bioaccumulate, through either bioconcentration (an increase in concentration of a substance in relation to the concentration in ambient water) or biomagnification (a progressive increase in concentration from one trophic level to the next higher level in the aquatic food chain (Moore and Ramamoorthy 1984, Sorensen 1991).

All of the organic pollutants of concern in this action bioaccumulate. All biomagnify to some extent in the food chain, although this is more of a serious concern for some contaminants than others. The Guidelines include a component designed to address the risks of elevated fish tissue residues of organic compounds to humans and avian and mammalian predators, but not the risk of that residue to fish (EPA 1985a). In fact, this process drives nearly all of the numeric criteria established for organic contaminants. What is not considered in these evaluations, however, is whether these tissue residues would directly affect the health of the aquatic organisms. Similar to metals, the consumption of aquatic invertebrates by fish is never formally considered in the development of the criteria for organic compounds. It is well established that invertebrates may accumulate organic contaminants in aquatic systems, and that these contaminants are passed on

to fish through the diet (*e.g.*, Streit 1998). Consequently, if the water quality criteria do not protect invertebrate prey species from organic residue accumulations, they may not protect listed species from adverse effects associated with dietary exposure.

In particular, measuring compliance with the criteria through ambient water concentrations alone leaves exposure pathways to several organic pollutants un-regulated. For example, dieldrin, lindane, and heptachlor epoxide are not highly water soluble, and are persistent in both food and sediments. A number of the organic compounds reviewed here (*e.g.*, dieldrin, lindane, heptachlor epoxide), have considerable potential to biomagnify in aquatic systems (Suedal *et al.* 1994). The Guidelines for such compounds do not consider food web transfer and bioaccumulation with respect to the target species. Consequently, they may greatly underestimate the toxicity of these chemicals in the environment. This is particularly important for the juvenile life stage of anadromous salmonids while they reside in rearing habitat, if such exposure later influences their downstream migration and subsequent ability to osmoregulate as they enter saltwater. This is an especially significant concern for organic contaminants such as organochlorine pesticides (*e.g.*, dieldrin, lindane, heptachlor epoxide), for which exposure is primarily via sediments and tissues of prey organisms.

A biologically significant pathway for exposures of aquatic organisms to contaminants is through consumption of contaminated aquatic detritus, plants, invertebrates, and other food items (bioaccumulation). Invertebrates that can accumulate metals in aquatic systems are often prey consumed by salmonids and other fish species (*e.g.*, Moore *et al.* 1991, Luoma and Carter 1991, Cain *et al.* 1992, Kiffney and Clements 1993, Rainbow and Dallinger 1993, Timmermans 1993, Ingersoll *et al.* 1994, Dallinger 1994, Cain *et al.* 1995, Gerhardt and Westermann 1995).

In an experiment that shows how readily contaminated food items lead to elevated fish tissue concentrations, Woodward *et al.* (1994) held paired groups of age 0 rainbow trout in clean and contaminated over a range of metal-concentrations. They fed one group a diet of reconstituted, metals contaminated invertebrates, and the other group a comparable diet based on uncontaminated invertebrates. After 91 days, they observed that only fish fed the contaminated diet exhibited reduced survival and growth. These results demonstrate that exposure to a dissolved metal can be a secondary hazard pathway in cases where food is contaminated and fish can bioaccumulate the substance of concern. In cases where fish can bioaccumulate a metal, these results and similar results from other studies of diet-borne metal exposures to salmonids collectively indicate that toxic effects can occur through dietary pathways (*e.g.*, Dallinger and Kautzky 1985, Dallinger *et al.* 1987, Spry *et al.* 1988, Giles 1988, Harrison and Klaverkamp 1989, Harrison and Curtis 1992, Miller *et al.* 1993, Mount *et al.* 1994, Farag *et al.* 1994).

In general, the metals considered in this opinion do not appear to biomagnify in the food chain, with the exception of selenium. The Guidelines include a component designed to assure that the water quality criterion for a substance is sufficiently low that residue accumulations will not impair the use of a waterbody by aquatic organisms, and that data from residue studies are to be considered alongside acute and chronic toxicity data in the criteria development process (EPA 1985a). However, metals criteria are presently based solely on results of aquatic toxicity tests (62 FR 42159), where metal exposures occur directly across gills or other respiratory surfaces.

Risk management via water concentration-based water quality criteria is not protective of listed salmonids for toxic pollutants that strongly bioaccumulate (*e.g.*, selenium, and organic pollutants: Pease *et al.* 1992; Taylor *et al.* 1992, 1993; Canton 1997; EPA 2001). This is because the true potential for toxic hazards to fish and wildlife through bioaccumulation is determined not only by an immediate water-borne exposure and direct toxicity effects, but also by the rate of mass loading into an aquatic ecosystem, the corresponding environmental partitioning of mass loads between the water column, sediments, and biota (food chain), and how the toxic pollutant is assimilated and acts on the organism. A water column concentration of a toxic pollutant may not reflect mass loading or be reflected in food chain bioaccumulation. Therefore, water quality criteria are useful guides for risk management only to the extent that they protect aquatic food chains from bioaccumulation.

This is an especially significant concern for organic contaminants such as organochlorine pesticides, for which exposure is primarily via sediments and tissues of prey organisms. Indeed, environmental agencies in some other countries, including Canada, no longer recommend water quality guidelines for these substances, but regulate them through other media such as sediment, soil, or tissue (CCREM 2001a).

Because hydrophobic compounds are expected to show a similar or proportional affinity for the lipid of an organism as that for octanol (which is used to calculate the partition coefficient[21]), the degree of partitioning exhibited between water and octanol, as characterized by the partition coefficient K_{ow}, can be a useful means for evaluating and predicting bioaccumulation (Mackay 1982, Di Toro *et al.* 1991). For organic compounds that are not metabolized, the relationship between the bioconcentration factor (BCF) and K_{ow} is strong (Mackay 1982). The expected wet-weight BCF for a non-metabolized hydrophobic compound is a function of the lipid content of an organism and the value of K_{ow} for the compound. The standard equation for determining the expected BCF is:

$$BCF = 0.046 \times K_{ow}$$

which is derived from fish studies and is based on an average lipid content of 4.6% wet weight (McCarty 1986). This relationship is used in this opinion for evaluating effects related to exposure and bioconcentration of the toxic organic pollutants addressed by the ODEQ.

Sediment concentrations that would result in organic toxic pollutant concentrations in the water column can be calculated using the equation (Di Toro *et al.* 1991):

$$SQC_{oc} = K_{oc} \times F_{CV}$$

where:

SQC_{oc} = sediment contaminant concentration in mg/kg organic carbon
K_{oc} = partitioning coefficient for sediment organic carbon

[21] A coefficient representing the ratio of the solubility of a compound in octanol (a non-polar solvent) to its solubility in water (a polar solvent). The higher to K_{OW}, the more non-polar the compound. Log K_{OW} is generally used as a relative indicator of the tendency of an organic compound to adsorb to soil. Log K_{OW} values are generally inversely related to aqueous solubility and directly proportional to molecular weight.

F_{cv} = the chronic water quality criterion in µg/L

K_{oc} can be calculated from the octanol/water partitioning coefficient, Kow, using the formula:

$Log_{10}(K_{oc}) = 0.00028 + 0.983 \text{ X } Log_{10}(K_{ow})$

This equation is used in the analysis of effects later in this opinion, provided that the data necessary to conduct the analysis were available, to evaluate the potential for water-borne exposure concentrations of organic pollutants at or below criteria concentrations.

Summary: Based on this analysis, the risks of bioconcentration and biooaccumulation factors are likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Insufficient Information on Behavioral and Other Sublethal Endpoints. In the case of chronic criteria, data are available for a range of sublethal effects such as growth and fecundity or sperm production. However, some important effects reported in mammals, such as immunosuppression and endocrine disruption, are inadequately studied in salmonids therefore were not considered in the development of the national criteria. These sublethal effects cannot be considered trivial, because they are associated with the potential for increased mortality (Arkoosh *et al.* 1998). Sublethal effects involving alterations in behavior can occur during relatively low concentration, short-term exposure, and can have profound biological implications (*e.g.*, chemical migration barrier, interference with spawning behavior). The NMFS recognizes that relevant data may not be available for all toxic substances, and that determination of a repeatable, detectable endpoint may involve a degree of subjectivity. Relatively little data are available to help elucidate these concerns; however, the research that does exist indicates that sublethal effects can be very serious for at least some toxicants.

Summary: Based on this analysis, the risks of sublethal effects will exacerbate adverse effects, and are likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Influence of Temperature, pH, and other Water Quality Stressors on Fish Response to Toxicity. In addition to direct influences on toxic pollutant speciation and chemical toxicity mechanisms, several water quality parameters influence general fish health, and susceptibility and ability to acclimate to and depurate after short-term increases in toxic parameter concentrations. This is generally addressed indirectly (with respect to toxicity) through conventional water quality criteria (*e.g.*, water temperature, pH, dissolved oxygen, dissolved gases, ammonia, *etc.*). However, it is possible for fish to be stressed or become stressed more rapidly when conventional water quality parameters are near or exceed criteria limits. This effect pathway is not addressed by most existing toxic pollutant criteria, and represents a shortcoming of the proposed criteria.

Summary: Based on this analysis, the risk that temperature, pH, and other water quality stressors will exacerbate the effects of the proposed criteria is high, as aquatic life criteria derived following the Guidelines do not take these additional stressors into account and are therefore likely to result in sublethals effects, such as interference in physiochemical processes, interption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Toxicity of Total Recoverable vs. Dissolved Metal Concentrations and the Use of Conversion Factors and Translators. Acute and chronic criteria for metals may be interpreted using either total recoverable or dissolved metal concentrations, depending on the objective of the study. The term "total recoverable" metal refers specifically to metal concentrations determined in unfiltered samples that have been acidified (pH < 2) before analysis. The term "dissolved" metal refers specifically to metal concentrations determined in samples that have been filtered (generally a 0.45 micron pore size) prior to acidification and analysis. Total recoverable metal concentration includes both the dissolved form and the portion either attached to particles in the water or present in suspended insoluble form. Particulate metals can be single atoms or metal complexes adsorbed to or incorporated into silt, clay, algae, detritus, plankton, *etc.*, which can be removed from the test water by filtration through a 0.45 micron filter.

Only dissolved metals are immediately bioavailable and thus immediately toxic to freshwater organisms (however, the particulate form may still affect listed species, as discussed below). The non-dissolved form is generally not directly hazardous to listed salmonids except under certain circumstances were (1) changes in water chemistry conditions lead to increased solubility from particulate forms within the water column, or (2) metal contaminated particulates are ingested or encounter gill surfaces. Factors in addition to hardness that influence solubility, and thus bioavailability and toxicity, include suspended sediment concentration, pH, organic carbon content, and chemical speciation of the metal. Further, some metal compounds are less soluble than others for a given set of water quality conditions.

Studies indicate that particulate metals contribute to organism exposure to metals. Particulates may act as a sink for metals, but they may also act as a source. Through chemical, physical, and biological activity, particulate metals can become bioavailable (Moore and Ramamoorthy 1984). Particulate and dissolved metals that end up in sediments are not rendered entirely nontoxic nor completely immobile, and may still contribute to the toxicity of the metal in natural waters. Of special concern are situations where waters contain both high particulate metal concentrations and dissolved concentrations near the proposed criteria. Additionally, those metals that can bioaccumulate through food-chain organisms and can cause indirect effects through particulate metal contamination.

Particulate metals are removed from the proposed regulatory "equation" through at least two methods: the use of CFs to determine the dissolved metal criteria from total recoverable criteria, and the use of a translator to convert back to a total metal concentration for use in waste load limit calculations. When waste discharge limits are to be developed and TMDLs are determined for a receiving waterbody, the dissolved criterion must be "translated" back to a total concentration because TMDLs are based on total metals.

EPA originally used total metal concentrations to establish national criteria, as provided in the National Toxics Rule published in 1992. The EPA subsequently changed to use of dissolved metal criteria, as explained in a 1993 policy statement:

> [I]t is now the policy of the Office of Water that the use of dissolved metal to is now the policy of the Office of Water that the use of dissolved metal to set and measure compliance with water quality standards is the recommended approach, because dissolved metal more closely approximates the bioavailable fraction of metal in the water column than does total recoverable metal. This conclusion regarding metals bioavailability is supported by a majority of the scientific community within and outside the Agency. One reason is that a primary mechanism for water column toxicity is adsorption at the gill surface which requires metals to be in the dissolved form (Prothro 1993).

Because no supporting references were given in support of the policy, it is hard to evaluate. There is theoretical support for the assumption that metals need to be in dissolved form to adsorb to the gill surface (Wood *et al.* 1997), and it does seem logical to assume that metals bound to particulates would be less toxic. However, two studies that examined the toxicity of particulate metals in controlled experimental studies (Brown *et al.* 1974, Erickson *et al.* 1996) found toxicity associated with particulate bound copper.

Erickson *et al.* (1996) estimated that the adsorbed copper has a relative toxicity of almost half that of dissolved copper, and noted that the assumption that toxicity can be simply related to dissolved copper was questionable, and a contribution of adsorbed copper to toxicity cannot be generally dismissed (Erickson *et al.* 1996). One possible reason for the observed toxicity from particulate-bound copper is that the pH of water changes as it crosses the gills of fish, and at pH of 6 or greater in the water where a fish is living, the pH of water will be lowered as it crosses the gill (Playle and Wood 1989).

Attempting to define, evaluate and manage risks associated with dietary exposures of metals or contaminated sediments by basing criteria on total recoverable metals would likely be so indirect as to be ineffective. However, in the absence of such efforts, the stance that metals sorbed to particles are in effect biologically inert and can safely be ignored is questionable. The effect of this stance is to give up some conservatism in aquatic life criteria for metals.

Conversion Factors. The EPA derived ambient dissolved metals criteria from aquatic toxicity tests that produced dose-response relationships in test organisms under controlled (laboratory) conditions. In most of these studies, organism responses were plotted against nominal test concentrations of metals or concentrations determined by analyzing unfiltered samples to which soluble metal compounds had been added. Thus, until recently, metals criteria have been expressed in terms of total metal concentrations. Current EPA metals policy (EPA 1993a) and the ODEQ stipulate that criteria be expressed on a dissolved basis. The CF used in the EPA formulae for computing criteria represents a corresponding adjustment so that criteria based on total metal concentrations used in laboratory testing can be "converted" to a dissolved basis actually present in the toxicity test solutions. Metals for which a CF has been applied include arsenic, cadmium, chromium, copper, lead, nickel, silver, and zinc.

CF values for the proposed metals criteria are near 1.0 for most metals, because they were determined using laboratory toxicity-test solutions prepared with purified, soluble metal compounds, rather than using natural waters where relative contributions of water-borne particulate metals are much greater. To develop the coversion factors, EPA reviewed test data that reported both total and dissolved concentrations in their test waters and also conducted simulations of earlier experiments to determine the dissolved to total ratios (60 FR 1536, 62 FR 42159). In this way, the historical toxicity database could be utilized and a large number of new toxicity tests would not have to be performed. However, the CFs in many cases (*e.g.*, As, Ni, Cr, Pb) developed based upon a small number of studies and samples compared to the historical database of toxicity tests. Although additional confirmatory studies were performed to develop the CFs, the database available appears to be limited and calls into question the protectiveness of the CFs determined for these metals in cases when site-specific water quality approaches toxic conditions.

Translators. The EPA provides three methods to translate criteria based on dissolved metals to permit-specific criteria based on total recoverable metals. These three methods may result in greatly different outcomes relative to particulate metal loading. These methods are::

1. Determination of a site-specific translator by measuring site specific ratios of dissolved metal to total metal and then dividing the dissolved criterion by this translator. As an example, a site specific ratio of 0.4 (40 percent of the metal in the site water is dissolved) would result in a 2.5-fold allowable increase in the discharge of total metals. The higher the fraction of particulate metal in the site water the greater the allowable discharge of total metal. This is EPA's preferred method.

2. Theoretical partitioning relationship. This method is based on a partitioning coefficient determined empirically for each metal, and (when available), the concentration of total suspended solids in the site-specific receiving water.

3. The translator for a metal is assumed to be equivalent to the Guidance conversion factor for that metal (*i.e.*, use the same value to convert from total to dissolved and back again).

Since translators are needed to calculate discharge limits they become important in determining the total metals allowed to be discharged. In California, economic analyses performed by the EPA and evaluated by the State Water Resources Control Board (SWRCB 1997) indicated that translators based on site-specific data would decrease dischargers costs of implementing the new CTR criteria by an estimated 50%. This cost savings is "directly related to the less stringent effluent limitations that result from the use of site-specific translators," and implies a strong economic incentive for dischargers to reduce costs by developing site-specific translators and ultimately being allowed to discharge more total metals. This conclusion regarding the impact of site specific translators is supported by documents received by the NMFS in the CTR consultation from EPA (*i.e.*, EPA 1997c).

The EPA performed a sensitivity analysis on the effect of the site specific translator, which relies on determining the ratio of metal in water after filtration to metal in water before filtration in downstream waters. The EPA's analysis indicated that use of a site-specific translators to

calculate criteria would result in greater releases of toxic-weighted metals loads above the option where the CFs are used as the translators. The potential difference was estimated to be between 0.4 million and 2.24 million "toxic weighted" pounds of metals discharged to California waterways (USFWS and NMFS 2000). Lastly, the current use of conversion factors and site specific translators in formula-based metal criteria is not sufficiently protective of threatened and endangered aquatic species because:

- Particulate metals are not regulated, yet chemical, physical, and biological activity can subsequently cause these particulate metals to become bioavailable and cause adverse effects.
- Particulate metal concentrations are not always negligible in critical habitat in Oregon.
- The national criteria were developed using toxicity tests that expose test organisms to metal concentrations with very low contributions from particulate metals.
- Toxicity tests do not assess whether the toxic contributions of particulate metals are negligible when particulate concentrations are great and dissolved concentrations are at or near criteria levels.
- This method has the potential to allow point sources to significantly increase the discharge of total metal loads into the environment, even though dissolved metal criteria are being met by a discharger.
- Metal loading occurs from the water column to streambed sediments.

Summary: Based on this analysis, the risks of using conversion factors and translators is likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

The Water-Effect Ratio Provision. The water-quality criteria for metals all include a WER in their formulas. The WER is the ratio of the test LC_{50} in site water divided by the LC_{50} in laboratory water; the ratio is then multiplied by the aquatic life criteria to obtain a WER-adjusted site-specific criteria. The approach has probably been most used with copper because of the profound effect of organic carbon (DOC) to ameliorate toxicity, which is not correlated with hardness. The purpose of WERs is to empirically account for characteristics other than hardness that might affect the bioavailability and thus toxicity of metals on a site-specific basis. Because the WERs are directly incorporated into the criteria equations, no separate action is needed to change the criteria values using a WER. The default WER value is 1.0 unless DEQ determines that a different value should apply.

The concept of adjusting metals criteria to account for differences in their bioavailability in site waters has long been a precept of water quality criteria (Bergman and Dorward-King 1997, Carlson *et al.* 1984, USEPA 1994). The WER approach uses one or more standard-test species (usually *Ceriodaphnia* and/or fathead minnows), which are tested in tandem in dilution waters collected from the site of interest and in standard reconstituted laboratory water. The results in the laboratory water are presumed to represent the types of waters used in tests relied on by EPA in criteria documents.

The main problem with this concept and approach is trying to define a single "typical" laboratory dilution water that reflects that used in criteria documents. Testing laboratories may generate valid results using all sorts of different dilution waters including dechlorinated tap water, natural groundwater (well water), natural surface water such as Lake Superior or Lake Erie, and reconstituted waters made from deionized water with added salts. The widely used "Interim Guidance on Determination and Use of Water-Effect Ratios for Metals" (Stephan *et al.* 1994) specified using recipes from EPA or American Society for Testing and Materials (ASTM) for making standardized test water that results in a water hardness with unusually low calcium relative to magnesium concentrations compared to that of most natural waters. This has the effect of making metals in the reconstituted laboratory water made by standard recipe more toxic than would be expected in water with more natural proportions of Ca and Mg. This is because, at least for fish and some invertebrates and copper, Ca reduces toxicity but Mg affords little or no protection (Borgmann *et al.* 2005, Naddy *et al.* 2002, Welsh *et al.* 2000). Lastly, the water-effect ratio seems to have always been recognized by EPA as an interim, operational substitute to establishing criteria on a more mechanistic basis that could directly account for a lot of the factors that affect toxicity. A major development toward this is the biotic ligand model (BLM) which is supposed to capture the major interactions between metals concentrations, competition, and complexation, which control bioavailability and thus toxicity (Di Toro *et al.* 2001, Niyogi and Wood 2004). For copper, the BLM was used as the basis of EPA's (2007) updated aquatic life criterion, which for copper at least, should negate much of the need for empirical WER testing.

Summary: Based on this analysis, the risks of using water-effect ratios is likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Summary of the Derivation of the EPA Aquatic Life Criteria. Based on the analysis on the derivation of the EPA aquatic life criteria, NMFS concludes that predicted effects associated with the aquatic life criteria are likely to be significantly greater than asserted and are likely to have significant consequences for field-exposed species.

APPENDIX 2: ECOTOX References Sources

Freshwater Criteria

Freshwater dieldrin:

Author	Year	Reference Source
Brooke, L.T.	1993	U.S.EPA Contract No.68-C1-0034, Work Assignment No.5, to R.L.Spehar, U.S.EPA, Duluth, MN :18 p.
Chadwick and Shumway	1969	Ambient Water Quality Criteria for Aldrin/Dieldrin, USEPA, October,1980
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
Douglas, M.T., D.O. Chanter, I.B. Pell, and G.M. Burney	1986	Aquat.Toxicol. 8(4):243-249
Gilroy, D.J., H.M. Carpenter, L.K. Siddens, and L.R. Curtis	1993	Fundam.Appl.Toxicol. 20(3):295-301
Hendricks, J.D., T.P. Putnam, and R.O. Sinnhuber	1979	J.Environ.Pathol.Toxicol. 2(3):719-728
Holden, A.V.	1966	J.Appl.Ecol. 3:45-53
Katz, M.	1961	Trans.Am.Fish.Soc. 90(3):264-268
Katz, M.	1961	Ambient Water Quality Criteria for Aldrin/Dieldrin, USEPA, October,1980
Lunn, C.R., D.P. Toews, and D.J. Pree	1976	Can.J.Zool. 54(2):214-219
Macek, *et al.*	1969	Ambient Water Quality Criteria for Aldrin/Dieldrin, USEPA, October,1980
MacPhee, C., and R. Ruelle	1969	Univ.of Idaho Forest, Wildl.Range Exp.Station Bull.No.3, Moscow, ID :112 p.
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)
Mayhew, J.	1955	Proc.Iowa J.Acad.Sci. 62:599-606
Mehrle, P.M., F.L. Mayer, and W.W. Johnson	1977	In: F.L.Mayer and J.L.Hamelink (Eds.), Aquatic Toxicology and Hazard Evaluation, 1st Symposium, ASTM STP 634, Philadelphia, PA :269-280 (Publ in Part As 6797)
Reinert, R.E., L.J. Stone, and H.L. Bergman	1974	Proc.17th Conf.Great Lakes Res .:52-58
Schoettger, R.A.	1970	U.S.Dep.Interior, Bur.Sport Fish.Wildl.Res., Publ. 106:2-40 (Publ in Part As 6797)
Shubat, P.J., and L.R. Curtis	1986	Environ.Toxicol.Chem. 5(1):69-77
Statham, C.N., and J.J. Lech	1975	Toxicol.Appl.Pharmacol. 34(1):83-87
Swedburg, D.	1969	Prog.Sport Fish Res., Div.Fish.Res., Bureau Sport Fish Wildl. 88:8-9
Van Leeuwen, C.J., P.S. Griffioen, W.H.A. Vergouw, and J.L. Maas-Diepeveen	1985	Aquat.Toxicol. 7(1-2):59-78

Freshwater endosulfan-alpha and endosulfan-beta:

Author	Year	Reference Source
Lemke, A. E.	1980	Ambient Water Quality Criteria for Endosulfan, USEPA , October, 1980.
Macek, K. J., et al	1969	Ambient Water Quality Criteria for Endosulfan, USEPA , October, 1980.
Schoettger, R.A.	1970	Ambient Water Quality Criteria for Endosulfan, USEPA , October, 1980.

Freshwater endrin:

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Bennett, R.O., and R.E. Wolke	1987	J.Fish Biol. 31(3):387-394
Bennett, R.O., and R.E. Wolke	1988	Mar.Environ.Res.24(1-4):351 (ABS)
Dinnel, P.A., J.M. Link, Q.J. Stober, M.W. Letourneau, and W.E. Roberts	1989	Arch.Environ.Contam.Toxicol. 18(5):748-755
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
Eller, L.L.	1971	Am.J.Pathol. 64(2):321-336
Grant, B.F., and P.M. Mehrle	1970	In: Resour.Publ.No.88, Prog.Sport Fish.Res.1969, Div.Fish.Res., Bur.Sport Fish.Wildl., U.S.D.I., Washington, D.C. :13-15
Katz	1961	Ambient Water Quality Criteria for Endrin. USEPA, Oct. 1980
Katz and Chadwick	1961	Ambient Water Quality Criteria for Endrin. USEPA, Oct. 1980
Katz, M.	1961	Trans.Am.Fish.Soc. 90(3):264-268
Katz, M., and G.G. Chadwick	1961	Trans.Am.Fish.Soc. 90(4):394-397
Macek, *et al.*	1969	Ambient Water Quality Criteria for Endrin. USEPA, Oct. 1980
Macek, K.J., C. Hutchinson, and O.B. Cope	1969	Bull.Environ.Contam.Toxicol. 4(3):174-183 (Publ in Part As 6797)
MacPhee, C., and R. Ruelle	1969	Univ.of Idaho Forest, Wildl.Range Exp.Station Bull.No.3, Moscow, ID :112 p.
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)
McKim, J.M., and H.M. Goeden	1982	Comp.Biochem.Physiol.C 72(1):65-74
Post and Schroeder	1971	Ambient Water Quality Criteria for Endrin. USEPA, Oct. 1980
Post, G., and T.R. Schroeder	1971	Bull.Environ.Contam.Toxicol. 6(2):144-155
Thurston, R.V., T.A. Gilfoil, E.L. Meyn, R.K. Zajdel, T.L. Aoki, and G.D. Veith	1985	Water Res. 19(9):1145-1155
Wohlgemuth, E.	1977	Prirodoved.Pr.Ustavu Cesk.Akad.Ved Brne 11(6):1-38 (Author Communication Used); Vertebratologicke Zpravy 1:20-21

Freshwater heptachlor epoxide:

Author	Year	Reference Source
Johnson, W. W. and M. T. Finley	1980	Human health and aquatic life literature search and data base evaluation for Heptachlor Epoxide. USEPA, Office of Water Regulations and Standards, Sept. 30, 1985
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)

Freshwater lindane:

Author	Year	Reference Source
	1960	Wash.Dep.Fish.Res.Bull. 5:1-161
Biagianti-Risbourg, S., C. Pairault, G. Vernet, and H. Boulekbache	1996	Chemosphere 33(10):2065-2079
Boulekbache, H., and C. Spiess	1974	Bull.Soc.Zool.Fr. 99(1):79-85 (FRE) (ENG ABS)
Katz, M.	1961	Trans.Am.Fish.Soc. 90(3):264-268
Macek, K.J., and W.A. McAllister	1970	Trans.Am.Fish.Soc. 99(1):20-27 (Publ in Part As 6797)
Macek, K.J., K.S. Buxton, S.K. Derr, J.W. Dean, and S. Sauter	1976	EPA-600/3-76-046, U.S.EPA, Duluth, MN :50 p.
MacPhee, C., and R. Ruelle	1969	Univ.of Idaho Forest, Wildl.Range Exp.Station Bull.No.3, Moscow, ID :112 p.
Matsuo, K., and T. Tamura	1970	Sci.Pest Control/Botyu-Kagaku 35(4):125-130
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)
McLeay, D.J.	1976	J.Fish.Res.Board Can. 33(6):1303-1311
Oliver, B.G., and A.J. Niimi	1985	Environ.Sci.Technol. 19(9):842-849
Peterson, R.H.	1976	J.Fish.Res.Board Can. 33(8):1722-1730
Rozados, M.V., M.D. Andres, and M.A. Aldegunde	1991	Aquat.Toxicol. 19(1):33-40
Tooby, T.E., and F.J. Durbin	1975	Environ.Pollut. 8(2):79-89
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Freshwater pentachlorophenol:

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Alexander, D.G., and R.M.V. Clarke	1978	Water Res. 12(12):1085-1090
Bentley, R.E., T. Heitmuller, B.H. Sleight III, and P.R. Parrish	1975	U.S.EPA, Criteria Branch, WA-6-99-1414-B, Washington, D.C .:13
Burridge, L.E., and K. Haya	1990	Bull.Environ.Contam.Toxicol. 45(6):888-892
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Chapman, G.A.	1969	Ph.D.Thesis, Oregon State University, Corvallis, OR :87 p.
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Davis, J.C., and R.A.W. Hoos	1975	J.Fish.Res.Board Can. 32(3):411-416
Dominguez, S.E., and G.A. Chapman	1984	Arch.Environ.Contam.Toxicol. 13:739-743
Douglas, M.T., D.O. Chanter, I.B. Pell, and G.M. Burney	1986	Aquat.Toxicol. 8(4):243-249
Fogels, A., and J.B. Sprague	1977	Water Res. 11(9):811-817
Glickman, A.H., C.N. Statham, A. Wu, and J.J. Lech	1977	Toxicol.Appl.Pharmacol. 41(3):649-658
Hattula, M.L., V.M. Wasenius, H. Reunanen, and A.U. Arstila	1981	Bull.Environ.Contam.Toxicol. 26(3):295-298
Hickie, B.E., and D.G. Dixon	1987	Aquat.Toxicol. 9(6):343-353
Hickie, B.E., D.G. Dixon, and J.F. Leatherland	1989	Fish Physiol.Biochem. 6(3):175-185
Hodson, P.V., and B.R. Blunt	1981	Aquat.Toxicol. 1(2):113-127
Hodson, P.V., D.G. Dixon, and K.L.E. Kaiser	1984	Environ.Toxicol.Chem. 3(2):243-254
Iwama, G.K., and G.L. Greer	1980	Trans.Am.Fish.Soc. 109(2):290-292
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Iwama, G.K., and G.L. Greer	1979	Bull.Environ.Contam.Toxicol. 23(4/5):711-716
Johnson and Finley	1980	Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates, Resource Publication 137. U.S. Department of Interior, Fish and Wildlife Service, Washington, DC, 1980.6-56
Kennedy, C.J.	1990	Ph.D.Thesis, Simon Fraser University, Canada:188 p.; Diss.Abstr.Int.B Sci.Eng.53(1):18 (1992)
MacPhee, C., and R. Ruelle	1969	Univ.of Idaho Forest, Wildl.Range Exp.Station Bull.No.3, Moscow, ID :112 p.
Matida, Y., S. Kimura, M. Yokote, H. Kumada, and H. Tanaka	1971	Bull.Freshwater Fish.Res.Lab.(Tokyo) 20(2):127-146
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)
McKim, J., P. Schmieder, and G. Veith	1985	Toxicol.Appl.Pharmacol. 77:1-10
McKim, J.M., P.K. Schmieder, and R.J. Erickson	1986	Aquat.Toxicol. 9(1):59-80
McKim, J.M., P.K. Schmieder, R.W. Carlson, E.P. Hunt, and G.J. Niemi	1987	Environ.Toxicol.Chem. 6:295-312
Negilski, D.S.	1973	M.S.Thesis, Oregon State Univ., Corvallis, OR:80 p.(Author Communication Used)
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Office of Pesticide Programs	2000	Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.
Oikari, A.O.J.	1987	Bull.Environ.Contam.Toxicol. 39(1):23-28
Peterson, R.H.	1976	J.Fish.Res.Board Can. 33(8):1722-1730
Sappington, L.C., F.L. Mayer, F.J. Dwyer, D.R. Buckler, J.R. Jones, and M.R. Ellersieck	2001	Environ.Toxicol.Chem. 20(12):2869-2876

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Statham, C.N., and J.J. Lech	1975	Toxicol.Appl.Pharmacol. 34(1):83-87
Stehly, G.R., and W.L. Hayton	1989	Aquat.Toxicol. 14(2):131-148
Thurston, R.V., T.A. Gilfoil, E.L. Meyn, R.K. Zajdel, T.L. Aoki, and G.D. Veith	1985	Water Res. 19(9):1145-1155
Van den Heuvel, M.R., L.S. McCarty, R.P. Lanno, B.E. Hickie, and D.G. Dixon	1991	Aquat.Toxicol. 20(4):235-252
Vigers, G.A., and A.W. Maynard	1977	Water Res. 11(4):343-346
Webb, P.W., and J.R. Brett	1973	J.Fish.Res.Board Can. 30(4):499-507

Freshwater ammonia:

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Allan, I.R.H.	1955	Int.Assoc.Theor.Appl.Limnol.Proc./Int.Ver.Theor.Angew.Limnol.Verh. 12:804-810
Arillo, A., C. Margiocco, and F. Melodia	1979	J.Fish Biol. 15(4):405-410
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Arillo, A., C. Margiocco, F. Melodia, P. Mensi, and G. Schenone	1981	Environ.Technol.Lett. 2:285-292
Arillo, A., N. Maniscalco, C. Margiocco, F. Melodia, and P. Mensi	1979	Comp.Biochem.Physiol.C 63(2):325-331
Arillo, A., R. Mantovani, C. Margiocco, F. Melodia, and P. Mensi	1979	Mem.Ist.Ital.Idrobiol.Dott Marco Marchi 37:51-61
Arthur *et al.*	1987	Bull. Environ. Contam. Toxicol. 38:324-331
Belding, D.L.	1927	Trans.Am.Fish.Soc. 57:100-119
Buhl and Hamilton	2000	Trans. Am. Fish. Soc., 129:2, 408-418.
Burrows, R.E.	1964	U.S.Fish Wildl.Serv., Res.Rep.No.66, Washington, DC :12 p.

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Calamari *et al.*	1981	Rapp. P.-v. Reun. Cons. int. Explor. Mer. 178:81-86.
Calamari, D., and R. Marchetti	1975	Prog.Water Technol. 7(3/4):569-577
Corti, U.A.	1951	Int.Assoc.Theor.Appl.Limnol.Proc./Int.Ver.Theor.Angew.Limnol.Verh. 11:84-87
Danecker, E.	1964	Osterreichs Fischerei.3/4:55-68 (ENG TRANSL)
Department of Scientific and Industrial Research	1955	Dep.Sci.Ind.Res., Water Pollut.Res.Bd., London :81 p.
Environment Canada	2004	Guideline for the release of ammonia dissolved in water found in wasterwater effluents.
Environment Canada	2004	Ammoniaproject: summary of pure ammonia rainbow trout toxicity testing.
Fedorov, K.Y., and Z.V. Smirnova	1978	Vopr.Ikhtiol. 19(2):320-328
Fisher, C.J., and C.D. Ziebell	1980	Eisenhower Consortium Bull. 7:1-11
Fitzsimons, J.D.	1989	Proc.32nd Conf.Great Lakes Res.:48 (ABS)
Guerra, M., and N. Comodo	1972	Boll.Soc.Ital.Biol.Sper. 48(22):898-901 (ITA)
Herbert, D.W.M.	1956	Bull.Cent.Belge Etud.Documentation Des Eaux 32:115-120
Holland, G.A., J.E. Lasater, E.D. Neumann, and W.E. Eldridge	1960	Res.Bull.No.5, State of Washington Dept.Fish., Seattle, WA :263 p.
Knoph	1992	Parr. 101C:275-282.
Kreutzmann, H.L., and H. Sordyl	1985	Zool.Jahrb.Abt.Allg.Zool.Physiol.Tiere 89(4):427-439 (GER) (ENG ABS)
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Thuvander, A.	1989	J.Fish Biol. 35(4):521-529
Van Leeuwen, C.J., P.S. Griffioen, W.H.A. Vergouw, and J.L. Maas-Diepeveen	1985	Aquat.Toxicol. 7(1-2):59-78

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Viale, G., and D. Calamari	1984	Environ.Pollut.Ser.A Ecol.Biol. 35(3):247-257
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Yamamoto, Y., and M. Inoue	1985	Bull.Jpn.Soc.Sci.Fish.(Nippon Suisan Gakkaishi) 51(10):1733-1735 (JPN) (ENG ABS)
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Author	Year	Reference Source
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Hale, J.G.	1977	Bull.Environ.Contam.Toxicol. 17(1):66-73
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Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)
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Olson, P.A. & H.F. Foster	1956	Hanford Biol. Res. Annual Rep. #HW-41500, p 35-49
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Birge, W.J.	1978	In: J.H.Thorp and J.W.Gibbons (Eds.), Dep.Energy Symp.Ser., Energy and Environmental Stress in Aquatic Systems, Augusta, GA 48:219-240
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Birge, W.J., J.A. Black, A.G. Westerman, and J.E. Hudson	1980	In: C.Gale (Ed.), EPA-600/9-80-022, Oil Shale Symposium: Sampling, Analysis and Quality Assurance, March 1979, U.S.EPA, Cincinnati, OH :519-534 (U.S.NTIS PB80-221435)
Birge, W.J., J.A. Black, and A.G. Westerman	1979	In: S.W.Nielsen, G.Migaki, and D.G.Scarpelli (Eds.), Symp.Animals Monitors Environ.Pollut., 1977, Storrs, CT 12:108-118
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Hodson, P.V., B.R. Blunt, D.J. Spry, and K. Austen	1977	J.Fish.Res.Board Can. 34(4):501-508
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Schreck, C.B., and H.W. Lorz	1978	J.Fish.Res.Board Can. 35(8):1124-1129
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Christensen, G.M.	1975	Toxicol.Appl.Pharmacol. 32:191-197(Used Ref 2022, 9586)
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Hodson, P.V., B.R. Blunt, and D.J. Spry	1978	Water Res. 12(10):869-878
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Hodson, P.V., B.R. Blunt, U. Borgmann, C.K. Minns, and S. Mcgaw	1983	Environ.Toxicol.Chem. 2(2):225-238
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Becker, C.D., and M.G. Wolford	1980	Environ.Pollut. 21(3):181-189
Bentley, R.E., T. Heitmuller, B.H. Sleight III, and P.R. Parrish	1975	U.S.EPA, Criteria Branch, WA-6-99-1414-B, Washington, D.C .:14
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Birge, W.J., J.A. Black, A.G. Westerman, and J.E. Hudson	1980	In: C.Gale (Ed.), EPA-600/9-80-022, Oil Shale Symposium: Sampling, Analysis and Quality Assurance, March 1979, U.S.EPA, Cincinnati, OH :519-534 (U.S.NTIS PB80-221435)
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Palawski, D., J.B. Hunn, and F.J. Dwyer	1985	Trans.Am.Fish.Soc. 114:748-753
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Birge, W.J., J.A. Black, A.G. Westerman, and J.E. Hudson	1980	In: C.Gale (Ed.), EPA-600/9-80-022, Oil Shale Symposium: Sampling, Analysis and Quality Assurance, March 1979, U.S.EPA, Cincinnati, OH :519-534 (U.S.NTIS PB80-221435)
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Buhl, K.J., and S.J. Hamilton	1991	Ecotoxicol.Environ.Saf. 22:184-197
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Hamilton, S.J., and K.J. Buhl	1990	Arch.Environ.Contam.Toxicol. 19(3):366-373
Hodson, P.V., D.J. Spry, and B.R. Blunt	1980	Can.J.Fish.Aquat.Sci. 37(2):233-240
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Galvez, F., C. Hogstrand, and C.M. Wood	1998	Comp.Biochem.Physiol.C 119(2):131-137
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Martin, R.C., D.G. Dixon, R.J. Maguire, P.V. Hodson, and R.J. Tkacz	1989	Aquat.Toxicol. 15(1):37-52
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Martin, R.C., D.G. Dixon, R.J. Maguire, P.V. Hodson, and R.J. Tkacz	1989	Aquat.Toxicol. 15(1):37-52
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Bradley, R.W., C. Duquesnay, and J.B. Sprague	1985	J.Fish Biol. 27(4):367-369
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Brown, V.M., and R.A. Dalton	1970	J.Fish Biol. 2(3):211-216
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Skidmore, J.F., and I.C. Firth	1983	Tech.Pap.No.81, Australian Water Resour.Council, Dep.Resour.Energy, Australian Gov.Publ.Serv., Canberra, Australi a:129
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Sprague, J.B.	1964	J.Fish.Res.Board Can. 21(1):17-26
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Spry, D.J., and C.M. Wood	1984	J.Comp.Physiol.B Biochem.Syst.Environ.Physiol. 154(2):149-158
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Author	Year	Reference Source
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Office of Pesticide Programs	2000	Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.
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Saltwater copper:

Author	Year	Reference Source
Dinnel, P.A., J.M. Link, Q.J. Stober, M.W. Letourneau, and W.E. Roberts	1989	Arch.Environ.Contam.Toxicol. 18(5):748-755
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208

Saltwater endosulfan-alpha and endosulfan-beta:

Author	Year	Reference Source
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
Dinnel, P.A., J.M. Link, Q.J. Stober, M.W. Letourneau, and W.E. Roberts	1989	Arch.Environ.Contam.Toxicol. 18(5):748-755

Saltwater lead:

Author	Year	Reference Source
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
Varanasi, U.	1978	In: D.A.Wolfe (Ed.), Marine Biological Effects of OCS Petroleum Development, NOAA ERL, Boulder, CO :41-53

Saltwater selenium:

Author	Year	Reference Source
Hamilton, S.J., and K.J. Buhl	1990	Arch.Environ.Contam.Toxicol. 19(3):366-373

Saltwater silver:

Author	Year	Reference Source
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
Ferguson, E.A., and C. Hogstrand	1998	Environ.Toxicol.Chem. 17(4):589-593

Saltwater tributyltin:

Author	Year	Reference Source
Alabaster, J.S.	1969	Int.Pest Control 11(2):29-35 (Author Communication Used)
Baldwin, I.G., M.M.I. Harman, and D.A. Neville	1994	Water Res. 28(10):2191-2199
Bruggemann, R., J. Schwaiger, and R.D. Negele	1995	Chemosphere 30(9):1767-1780
Buccafusco, R., C. Stiefel, D. Sullivan, B. Robinson, and J. Maloney Jr.	1978	U.S.EPA-OPP Registration Standard
Douglas, M.T., D.O. Chanter, I.B. Pell, and G.M. Burney	1986	Aquat.Toxicol. 8(4):243-249
Martin, R.C., D.G. Dixon, R.J. Maguire, P.V. Hodson, and R.J. Tkacz	1989	Aquat.Toxicol. 15(1):37-52
Office of Pesticide Programs	2000	Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.

Orthuber, G.	1991	Ph.D.Thesis, Ludwig-Maximilians Univ., Muenchen, Germany:194 p.(GER) (ENG ABS)
Schwaiger, J., F. Bucher, H. Ferling, W. Kalbfus, and R.D. Negele	1992	Aquat.Toxicol. 23(1):31-48
Short, J.W., and F.P. Thrower	1987	Aquaculture 61(3-4):193-200
Triebskorn, R., H.R. Kohler, J. Flemming, T. Braunbeck, R.D. Negele, and H. Rahmann	1994	Aquat.Toxicol. 30(3):189-197
Triebskorn, R., H.R. Kohler, K.H. Kortje, R.D. Negele, H. Rahmann, and T. Braunbeck	1994	Aquat.Toxicol. 30(3):199-213

Saltwater zinc:

Author	Year	Reference Source
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
Herbert, D.W.M., and A.C. Wakeford	1964	Int.J.Air Water Pollut. 8(3/4):251-256

APPENDIX 3: Direct Mortality Population Modeling

Introduction

To assess the potential for adverse impacts of chemical exposures during subyearling freshwater post-swimup rearing on Pacific salmon populations, two models were developed. One model assesses direct mortality and its impact on population productivity and another model explicitly links impairments in the somatic growth of individual subyearling salmon to the productivity of salmon populations. Both models address impacts on first-year survival, and the results are incorporated into one of four life-history models to quantify changes in population productivity. General life-history models were constructed and analyzed for coho salmon (*Oncorhynchus kisutch*), sockeye salmon (*O. nerka*) and ocean-type and stream-type Chinook salmon (*O. tshawytscha*). For this exercise a population is defined following Ricker's (1972) definition of a "stock" as "a group of fish of the same species that spawns in a particular lake or stream (or portion thereof) at a particular season and which, to a substantial degree, does not interbreed with fish from any other group spawning in a different place or in the same place at a different season." The investigation of population-level responses to chemical exposures uses life-history transition matrix models. Individuals within a population exhibit various growth, reproduction, and survivorship rates depending on their developmental or life-history stage or age. The life-history strategy and demographic rates defining the survival and reproductive contribution of the various age classes determine the population productivity and determine the model transition matrix. Alterations of the demographic rates can impact a population's intrinsic growth rate which is calculated directly from the transition matrix as described below.

The basic salmonid life history consists of hatching and rearing in freshwater, smoltification in estuaries, migration to the ocean, growth to maturation at sea, and returning to the natal freshwater stream for spawning followed shortly by death. Differences between the four modeled life-history strategies are lifespan of the female, time to reproductive maturity, and the number and relative contribution of the reproductive age classes (Figure A1). The coho females modeled reach reproductive maturity at age 3 and provide all of the reproductive contribution at this time. Sockeye females in the modeled life history reach maturity at age 4 or 5, but the majority of reproductive contributions are provided by age 4 females. Chinook females can mature at age 3, 4 or 5, with the majority of the reproductive contribution from ages 4 and 5. The primary difference between the ocean-type and stream-type Chinook is juvenile freshwater residence time, with ocean-type juveniles migrating to the ocean as subyearlings and stream-type Juveniles overwintering in freshwater and migrating to the ocean as yearling smolts. The models depicted general populations representing each life-history strategy and were constructed based upon literature data described below. Specific populations were not modeled due to the difficulty in finding sufficient demographic and reproductive data for single populations.

The endpoint used to assess population-level impacts for both the somatic growth model and the direct mortality population model was the percent change in the intrinsic population growth rate (lambda, λ) resulting from the chemical exposure. Change in λ is an accepted population parameter often used in evaluating population productivity, status, and viability. The National Marine Fisheries Service uses changes in λ when estimating the status of species, conducting risk and viability assessments, developing Endangered Species Recovery Plans, composing

Biological Opinions, and communicating with other federal, state and local agencies (McClure *et al.*, 2003). While values of $\lambda<1.0$ indicate a declining population, in cases when an exposure causes the population growth rate to decrease more than natural variability, a loss of productivity will result even if lambda remains above 1.0. Decreases in response to chemical exposures can be a cause for concern since the impact could make a population more susceptible to declining (lambda dropping below 1.0) due to impacts from other stressors.

To determine if population productivity would be at risk due to direct mortality resulting from either acute or chronic exposures to the criterion concentrations of the chemicals of concern, a direct mortality population model was constructed. This model assessed whether juvenile salmon during their freshwater residence encountering the established criterion concentrations would experience individual mortality, and if that mortality would be sufficient to produce a change in the population growth rate. This included direct mortality from either acute or chronic exposures. The model applied a mortality factor to first-year survival of the respective life-history models to assess changes in lambda.

In the freshwater portion of their life, Pacific salmon are exposed to chemicals that also may act in a sublethal manner by inhibiting somatic growth. Juvenile growth is a critical determinant of freshwater and marine survival for Chinook salmon (Higgs *et al.* 1995). Reductions in the somatic growth rate of salmon fry and smolts are believed to result in increased size-dependent mortality (Healey 1982, West and Larkin 1987, Zabel and Achord 2004). Zabel and Achord (2004) and Mebane and Arthaud (2010) observed size-dependent survival for Juvenile salmon during the freshwater phase of their outmigration. Mortality is also higher among smaller and slower growing salmon because they are more susceptible to predation during their first winter (Healey 1982, Holtby *et al.* 1990, Beamish and Mahnken 2001). These studies suggest that factors affecting the organism and reducing somatic growth could result in decreased first-year survival and, thus, reduce population productivity. Using a modeling approach, Mebane and Arthaud (2010) suggested that size reductions from early-life stage chronic sublethal copper exposure could potentially reduce Juvenile salmon survival and population recovery trajectories.

Changes in juvenile salmon size due to exposure to the chemicals of concern were linked to size-dependent survival of Juveniles during their first year. Exposures and somatic growth were determined from the free-swimming and feeding fry stage (1.0g fish) to either outmigration, for ocean-type stocks, or to the fall when parr prepare for overwintering, in the case of stream-type stocks. Somatic growth models were constructed for coho, sockeye, ocean-type and stream-type Chinook. A steelhead (*O. mykiss*) life-history model was not constructed due to the lack of demographic information relating to the proportions of resident and anadromous individuals, the freshwater residence time of steelhead, and rates of repeated spawning. Models for chum (*O. keta*) and pink salmon (*O. gorbuscha*) were not constructed due to their short freshwater residence which would not allow sufficient rearing time to alter somatic growth rate and size to the point of altering survival rates. The somatic growth model used here is an extension of one developed for investigating the effects of pesticides on the biochemistry, behavior and growth of ocean-type Chinook salmon (Baldwin *et al.*, 2009).

The following descriptions detail how the direct mortality and somatic growth models were developed to serve as a means to assess the potential effects on ESA-listed salmon populations

from exposure to chemicals that cause direct mortality and reductions in somatic growth. Comparing the results from different chemical exposure scenarios to a control (*i.e.* unexposed) scenario can indicate the potential for chemical exposures to lead to changes in either mortality or somatic growth and size-dependent survival of individual subyearling salmon. Subsequent changes in salmon population dynamics as indicated by percent change in a population's intrinsic rate of increase assist us in estimating the potential population-level impacts to listed populations.

Methods

Model Life-history Strategies

Both models investigated the population-level responses to chemical exposures using life-history projection matrix models. Individuals within a population exhibit various growth, reproduction, and survivorship rates depending on their developmental or life-history stage or age. These age specific characteristics are depicted in the life-history graph (Figure A1A-C) in which transitions are depicted as arrows. The nonzero matrix elements represent transitions corresponding to reproductive contribution or survival, located in the top row and the subdiagonal of the matrix, respectively (Figure A1C). The survival transitions in the life-history graph are incorporated into the n x n square matrix (A) by assigning each age a number (1 through n) and each transition from age i to age j becomes the element a_{ij} of matrix A (i = row, j = column) and represents the proportion of the individuals in each age passing to the next age as a result of survival. The reproductive element (a_{1j}) gives the number of offspring that hatch per individual in the contributing age, j. The reproductive element value incorporates the proportion of females in each age, the proportion of females in the age that are sexually mature, fecundity, fertilization success, and hatch success.

A prospective analysis of the transition matrix, A, (Caswell 2001) explored the intrinsic population growth rate as a function of the vital rates (survival and reproduction). The intrinsic population growth rate, λ, equals the dominant eigenvalue of A and was calculated using matrix analysis software (MATLAB version 2010b by The Math Works Inc., Natick, MA). Therefore λ is calculated directly from the matrix. Variability was integrated by repeating the calculation of λ 2000 times selecting the values in the transition matrix from their normal distribution defined by their mean and standard deviation. The mean value of λ for control and exposed scenarios were determined. From these values the percent change in λ (and standard deviation) was calculated. The influence of each matrix element, a_{ij}, on λ was assessed by calculating the sensitivity values for A. The sensitivity of matrix element a_{ij} equals the rate of change in λ with respect to a_{ij}, defined by $\delta\lambda/\delta a_{ij}$. Higher sensitivity values indicate greater influence on λ. The elasticity of matrix element a_{ij} is defined as the proportional change in λ relative to the proportional change in a_{ij}, and equals (a_{ij}/λ) times the sensitivity of a_{ij}. One characteristic of elasticity analysis is that the elasticity values for a transition matrix sum to unity (one). The unity characteristic also allows comparison of the influence of transition elements and comparison across matrices.

Due to differences in the life-history strategies, specifically lifespan, age at reproduction and first year residence and migration habits, four separate life-history models were constructed representing coho, sockeye, ocean-type Chinook and stream-type Chinook. This was done to

encompass the different responses of these species to freshwater chemical exposures and assess potentially different population-level responses. In all cases, transition values were determined from literature data on survival and reproductive characteristics of each species. All characteristics exhibit density independent dynamics. The models assume closed systems, allowing no migration impact on population size. No stochastic impacts are included beyond natural variability as represented by selecting parameter values from a normal distribution about a mean value for each model iteration (year). Ocean conditions, freshwater habitat, fishing pressure, and marine resource availability were assumed constant and density independent.

A life-history model was constructed for coho salmon (*O. kisutch*) with a maximum age of 3 years. Spawning occurs in late fall and early winter with emergence from March to May. Fry spend 14-18 months in freshwater, smolt and spend 16-20 months in the saltwater before returning to spawn (Pess *et al.* 2002). Survival numbers were summarized in Knudsen *et al.* (2002) as follows. The average fecundity of each female is 4500 with a standard deviation of 500. The observed number of males:females was 1:1. Mean survival rate (standard deviation) from spawning to emergence is 0.3 (0.07). Survival from emergence to smolt is 0.0296 (0.00029) and marine survival is 0.05 (0.01). All parameters followed a normal distribution (Knudson *et al.* 2002). The calculated values used in the matrix are listed in Table A1. The growth period for first year coho was set at 184 days to represent the time from mid-spring to mid-fall when the temperatures and resources drop and somatic growth slows (Knudson *et al.* 2002, Table A2).

The life-history model for sockeye salmon (*O. nerka*) was based upon the lake wintering populations of Lake Washington, Washington, USA. These female sockeye salmon spend one winter in freshwater, then migrate to the ocean to spend three to four winters before returning to spawn at ages 4 or 5. Jacks return at age 2 after only one winter in the ocean. The age proportion of returning adults is 0.03, 0.82, and 0.15 for ages 3, 4 and 5, respectively (Gustafson *et al.*1997). All age 3 returning adults are males. Hatch rate and first year survival were calculated from brood year data on escapement, resulting presmolts and returning adults (Pauley *et al.* 1989) and fecundity (McGurk 2000). Fecundity values for age 4 females were 3374 (473) and for age 5 females were 4058 (557) (McGurk 2000). First year survival rates were 0.737/month (Gustafson *et al.* 1997). Ocean survival rates were calculated based upon brood data and the findings that approximately 90% of ocean mortality occurs during the first 4 months of ocean residence (Pauley *et al.* 1989). Matrix values used in the sockeye baseline model are listed in Table A1. The 168 day growth period represents the time from lake entry in mid-spring to early fall when the temperature drops and somatic growth slows (Gustafson *et al.* 1997, Table A2).

A life-history model was constructed for ocean-type Chinook salmon (*O. tshawytscha*) with a maximum female age of 5 and reproductive maturity at ages 3, 4 or 5. Ocean-type Chinook migrate from their natal stream within a couple months of hatching and spend several months rearing in estuary and nearshore habitats before continuing on to the open ocean. Transition values were determined from literature data on survival and reproductive characteristics from several ocean-type Chinook populations in the Columbia River system (Healey and Heard 1984, Howell *et al.* 1985, Roni and Quinn 1995, Ratner *et al.* 1997, PSCCTC 2002, Green and Beechie 2004). The sex ratio of spawners was approximately 1:1. Estimated size-based fecundity of 4511 (65), 5184 (89), and 5812 (102) was calculated based on data from Howell *et al.*, 1985, using length-fecundity relationships from Healy and Heard (1984). Control matrix values are listed in

Table A1. The growth period of 140 days encompasses the time the fish rear in freshwater prior to entering the estuary and open ocean (Table A2). The first three months of estuary/ocean survival are the size-dependent stage. Size data for determining subyearling Chinook condition indices came from data collected in the lower Columbia River and estuary (Johnson *et al.* 2007).

An age-structured life-history matrix model for stream-type Chinook salmon with a maximum age of 5 was defined based upon literature data on Yakima River spring Chinook from Knudsen *et al.* (2006) and Fast *et al.* (1988), with sex ratios of 0.035, 0.62 and 0.62 for females spawning at ages 3, 4, and 5, respectively. Length data from Fast *et al.* (1988) was used to calculate fecundity from the length-fecundity relationships in Healy and Heard (1984). The 184-day growth period produces control fish with a mean size of 96mm, within the observed range documented in the fall prior to the first winter (Beckman *et al.* 2000). The size-dependent survival encompasses the 4 early winter months, up until the fish are 12 months old.

Direct Mortality Population Model

A direct mortality population model was constructed that estimated the population-level impacts of first-year mortality resulting from exposure to the criterion concentrations of ammonia, copper and cadmium. These models excluded sublethal and indirect effects of the chemical exposures and focused on the population-level outcomes resulting from an annual exposure of young-of-the-year to a chemical at the criterion concentrations. Scenarios were chosen to represent both the acute and chronic criteria. This was done by parameterizing the model with toxicity data (LC_{50}s) derived from short term (<96hrs) and long term (>28day, based on the available data, see Table A3) experiments. The lethal impact was implemented as a change in first year survival for each of the salmon life-history strategies. In order to understand the relative impacts of a short-term exposure of a single chemical on exposed vs. unexposed fish, we used parameters for an idealized control population that exhibits an increasing population growth rate. Four life-history strategies were modeled, ocean-type and stream-type Chinook salmon, coho salmon and sockeye salmon. The details for each general population model are provided above in the *Model life-History Strategies* section.

The mortality responses are modeled as direct reduction in the first-year survival rate (S1 in Table A1 and Figure A1D). Exposures are assumed to result in a cumulative reduction in survival as defined by the concentration and the dose-response curve as defined by the LC_{50} and slope for each chemical. A sigmoid dose-response relationship is used to model the mortality dose-response to be consistent with other dose-response relationships. The model inputs for each scenario are the exposure concentration and fish LC_{50}, as well as the sigmoid slope for the LC_{50}. For a given concentration a chemical survival rate is calculated and is multiplied by the control first-year survival rate, producing an exposed scenario first-year survival for the life-history matrix. Variability is incorporated using means and standard deviations to select from normally distributed survival and reproductive rates and repeating the calculation of lambda 2000 times as described above.

Population model output consists of the percent change in lambda from the unexposed control populations derived from the mean of one thousand calculations each of the unexposed control and the chemical exposed populations. The percent change in lambda (with standard deviation),

representing alterations to the population productivity, was selected as the primary model output for reasons outlined previously. The percent change in lambda is considered different from control when the difference is greater than the percent of one standard deviation of the control lambda.

Somatic Growth Model

Toxic impacts on somatic growth to individual juvenile salmon were modeled as a change in daily growth rate resulting from an exposure concentration occurring during the growth phase of first year freshwater residence. Toxicity parameters relied on experiments producing EC50 values (effect concentration producing 50% change in growth) and slopes for chronic exposures. Sigmoidal dose-response relationships, at steady-state, between each exposure and somatic daily growth rate were modeled using growth EC50s and slopes. The timecourse for each exposure was built into the model as a pulse with a defined start and end during which the exposure remained constant (Figure A2B). The timecourse for daily growth rate was modeled using two single-order exponential functions, one for the time required for the exposure to reach full effect and the other for time required for complete recovery following the end of the exposure (time-to-effect and time-to-recovery, respectively). For all compounds, both timecourses were assumed to be within a day, so a value of 0.5 was used for the half-lives of effect and recovery. Incorporating dynamic effects and recovery variables does allow the model to simulate differences in the pharmacokinetics (*e.g.* the rates of uptake from the environment and of detoxification) of various chemicals, but this requires additional, compound-specific, data.

The growth models were replicated for 1000 individual fish to capture the variability of possible output. The initial weight of each replicate was selected from a normal distribution with a mean of 1.0 g and standard deviation of 0.1 g. The size of 1.0 g was chosen to represent subyearling size in the mid-spring at the onset of the stable growth trajectory (*i.e.* the growth rate is not changing). For each iteration (day) of the model, the somatic growth rate is calculated for each fish by selecting the parameter values from normal distributions with specified means and standard deviations (Table A2). The weight for each fish is then adjusted based on the calculated daily growth rate to generate a new weight for the next iteration. The length (days) to run the growth portion of the model was selected to represent the time from when the fish enter the linear portion of their growth trajectory in the mid to late spring until they change their growth pattern in the fall due to reductions in temperature and resources or until they migrate out of the system. The mean weights (with standard deviations) after the species-appropriate growth period (Table A2) were used to calculate the size-dependent survival as described below. A sensitivity analysis was run to determine the influence of the parameter values on the size distribution output of the somatic growth model.

The species-specific parameter values defining control conditions, such as the length of the growth period and control daily growth rate are listed in Table A2. Each exposure scenario was defined by a concentration and exposure time for each chemical.

Below are the mathematical equations used to derive Figure A2.

Figure A2A uses a sigmoid function:

y= bottom + (top – bottom)/(1 + (exposure concentration/EC50)^slope).

Figure A2B uses a step function:

time < start; exposure = 0

start ≤ time ≤ end; exposure = exposure concentration(s)

time > end; exposure = 0.

Figure A2C uses a series of exponential functions:

time < start; y = c

start ≤ time ≤ end; y = c – (c – i)*(1 – exp(-ke*(time – start)))

time > end; ye = c – (c – i)*(1 – exp(-ke*(end – start)))

y = ye + (c – ye)*(1 – exp(-kr*(time – end))).

For Figure A2A, y = Daily Growth Rate, top = Gc, bottom = 0. For Figure A2C, c = Gc, i = Gi, ke = ln(2)/Growth effect half-life, kr = ln(2)/Growth recovery half-life. For Figure A2C the value of ye is calculated to determine the amount of inhibition that is reached during the exposure time, which may not be long enough to reach the maximum level of inhibition.

Linking to Survival in Population Model

The weight distributions from the somatic growth portion of the model are used to calculate size-dependent first-year survival for a life-history matrix population model for each species and life-history type. This incorporates the impact that reductions in size could have on population growth rate and abundance. The first-year survival element of the transition matrix incorporates a size-dependent survival rate for a three- or four-month interval (depending upon the species) which takes the Juveniles up to 12 months of age. This time represents the 4-month early winter survival in freshwater for stream-type Chinook, coho, and sockeye models. For ocean-type Chinook, it is the 3-month period the subyearling smolt spend in the estuary and nearshore habitats (*i.e.* estuary survival). The weight distributions from the organismal model are converted to length distributions by applying condition factors from data for each modeled species (cf; 0.0095 for sockeye and 0.0115 for all others) as shown in Equation L.

Equation L: length(mm) = ((fish weight(g)/cf)^(1/3))*10

The relationship between length and early winter or estuary survival rate was adapted from Zabel and Achord (2004) to match the survival rate for each control model population (Howell *et al.* 1985, Kostow 1995, Myers *et al.* 2006, Figure A3). The relationship is based on the length of a subyearling salmon relative to the mean length of other competing subyearling salmon of the same species in the system, Equation D, and relates that relative difference to size-dependent survival based upon Equation S. The values for α and resulting size-dependent survival (survival ϕ) for control runs for each species are listed in Table A2. The constant α is a species-specific parameter defined such that it produces the correct control survival ϕ value when Δlength equals zero.

Equation D: Δlength = fish length(mm) – mean length(mm)

Equation S: Survival $\phi = (e^{(\alpha+(0.0329*\Delta\text{length}))}) / (1 + e^{(\alpha+(0.0329*\Delta\text{length}))})$

Randomly selecting length values from the normal distribution calculated from the organismal model output size and applying equations 1 and 2 generates a size-dependent survival probability for each fish. This process was replicated 1000 times for each exposure scenario and simultaneously 1000 times for the paired control scenario and results in a distribution with a mean size-dependent survival rate for each population. The resulting size-dependent survival rates are inserted in the calculation of first-year survival in the respective control and chemical-exposed transition matrices of the life-history population models described above.

In the population model an individual fish experiences an exposure once as a subyearling (during its first spring) and never again. The chemical exposure is assumed to occur each year to the subyearling age class. All subyearlings within a given population are assumed to be exposed to the chemical. No other age classes experience the exposure. The model integrates this as every brood class being exposed as subyearlings and thus the vital demographic rates of the transition matrix are continually impacted in the same manner. Regardless of other effects due to the direct exposure, only growth effects are incorporated in the model.

The population model recalculates first-year survival for each run using a size-dependent survival value selected from a normal distribution with the mean and standard deviation produced by Equation S. Population model output consists of the percent change in lambda from the unexposed control populations derived from the mean of two thousand calculations of both the unexposed control population and the chemical exposed population. Change in lambda (with standard deviation), representing alterations to the population productivity, was selected as the primary model output for reasons outlined previously.

Model Toxicity Scenario Parameterization

Literature Review. Data for parameterizing the toxicity scenarios for the direct mortality and somatic growth models were identified by conducting extensive literature searches. The first round of searches broadly gathered papers and reports that had toxicological information on the effects of ammonia, cadmium, and copper on mortality and growth in Juvenile salmonids. Several different online databases and print sources were used in the literature search that was conducted to identify appropriate data:

1. The Thomson Reuters online academic citation index, Web of Science, was used. Search terms included the name of the contaminant: (ammonia), (copper OR cu), (cadmium OR cd); types of effects: (LC50 OR acute OR lethal* OR growth*); and order, family, genus, main species names, and main common names of salmonids: (acantholingua OR amago OR arctic char OR arctic cisco OR baikal omul OR bloater OR brachymystax OR char OR Chinook OR chum OR cisco OR coho OR coregoninae OR coregonus OR dolly varden OR grayling OR hucho OR inconnu OR keta OR kisutch OR kiyi OR lake herring OR nerka OR oncorhynchus OR parahucho OR prosopium OR salmo OR salmon OR salmonid* OR salmonidae OR

salmoniformes OR salmoninae OR salvelinus OR salvethymus OR sockeye OR steelhead OR stenodus OR taimen OR thymallinae OR thymallus OR trout OR tshawytscha OR whitefish).

2. The U.S. The EPA online ECOTOX database was used. This database includes single chemical toxicity information and citations for aquatic life. The query included genus and species names, common names, chemical names, and growth or mortality as effects endpoints (similar to above).

3. The online database Aquatic Sciences and Fisheries Abstracts (AFSA), a component of the international Aquatic Sciences and Fisheries Information System (ASFIS), was used. Input search terms were ammonia and salmon or salmonids.

4. The bibliography of the EPA Draft 2009 Update Aquatic Life Ambient Water Quality Criteria for Ammonia - Freshwater; the EPA Aquatic Life Ambient Freshwater Quality Criteria – Copper 2007 Revision; and the Draft Idaho Water Quality Standards Biological Opinion (section on copper).

5. Citations from relevant research articles and reports that were obtained as part of the above searches, and citations from published literature reviews, were also used.

Toxicity Value selection for Exposure Scenarios

The publications identified by the broad literature search were reviewed for appropriate methodologies, replication, measurement endpoints, and life stages exposed. Those studies with insufficient replication or single exposure concentrations were omitted. The review of studies focused on those conducted with Juvenile salmonids exposed during the life stages between swim-up to parr or subyearling smolt to match with the exposure regimes of the models. When multiple toxicity values or slopes were found, the genus geometric mean was used as the initial model input value. In addition, the minimum species mean values were used to parameterize the model to examine the range of potential impacts and avoid overlooking impacts to sensitive listed species. Direct mortality endpoints were collected from 96-h continuous exposure studies for modeling acute exposures and >28 day exposure studies to model chronic exposures.

Studies critically assessed for growth reported endpoints including changes in weight (wet or dry), length, or biomass resulting from water exposures lasting at least 28 days. The assumptions regarding initial fish size in the somatic growth model are very sensitive to the study data used for parameterization. The model simulates the stable portion of the growth phase during which the growth rate is relatively constant that occurs in Juvenile salmonids from about 1g to the their first fall or until outmigration to ocean habitats (Weatherley and Gill 1995). Younger fry (*e.g.* 0.2g) have very different rates and efficiencies of food conversion than 1g and larger fry and parr (Weatherly and Gill 1995). Fry that still are absorbing their yolk sac may have this reabsorption affected by contaminants. In addition, somatic growth rate responses across temperatures for younger fry differ from those of larger fish (Weatherly and Gill 1995). Therefore, smaller fry commonly found in these studies could respond very differently to contaminant exposures than those at greater than 1g, and studies on these sizes were excluded from consideration. Similarly,

data from studies initiated with Juveniles greater than 10g were not considered since this is past the majority of growth during the first summer (*e.g.* Thedinga *et al.* 1998, Johnson *et al.* 2007). The specific review and value selection procedures used for ammonia, cadmium and copper are discussed below.

Ammonia: The documents identified by the first round of literature review applying to acute toxicity of ammonia to salmonids were further reviewed for data appropriate to parameterize the direct mortality population model. Data needed to conform to 96-hr LC50 values for subyearling salmonids (free-swimming, 1-4g fish preferred, but did include data on fish of less than 10 g when that was all that was available). The range of values identified for Chinook salmon, coho salmon, rainbow trout and cutthroat trout and are shown below in the units of mg NH_3-N/L, as N. All values were normalized to a pH of 8 using an un-ionized ammonia computer worksheet available from the American Fisheries Society (http://www.fisheries.org/afs/hatchery.html, Table 9 Ammonia Calculator (Freshwater) Excel spread sheet from the web site). Following the practice in the ammonia Ambient Water Quality Criteria documents (1999, 2009), the fish LC50 values were not normalized for temperature. The normalized species mean values were 26.8, 15.1, 26.2 and 29.4 mg NH_3-N/L for Chinook salmon, coho salmon, rainbow trout and cutthroat trout, respectively (Servizi and Gordon 1990; Buckley 1978; Thurston and Russo 1983; Thurston *et al..*, 1981, Table A3). The genus geometric mean from these data was 23.6 mg NH_3-N/L. A sigmoid dose-response slope was calculated as 6.4 (Broderius and Smith 1979; Buckley 1978). Both the genus geometric means and minimum species mean values were used to parameterize the model as discussed above. To assess the chronic criterion, a chronic study was found that exposed cutthroat trout to ammonia for 29 days and reported an LC50 of 21.3 mg NH_3-N/L (Thurston *et al.*, 1978). No slope was identified, so the 96-hr slope was used in the model.

Documents investigating the effects of ammonia on growth of fish were reviewed for data appropriate as input to the somatic growth model. No studies were found that could provide the appropriate data. Most studies on exposure of Juvenile salmonids to ammonia found that any effects on growth or food intake were temporary and compensation occurred before the end of the exposure period (Lang *et al.*, 1987; Linton *et al.*, 1998; Beamish and Tandler 1990; Larmoyeux and Piper 1973). Other studies have shown effects on growth, but exposure occurred over early developmental stages and also produced developmental delays and abnormalities, so differences in size may not have been attributable to direct impacts on metabolism or growth (Brinkman *et al.*, 2009). From a 90-day exposure Brinkman *et al.*, (2009) calculated an EC20 that includes hatch effects, delayed swimup, and sac-fry growth of 5.56 mg NH_3-N/L normalized to pH 8. In addition, Lazorchak and Smith (2007) reported decreases in growth of rainbow trout (size range <0.2g) after a 7 day exposure to ammonium chloride, but at concentrations that overlapped with those inducing mortality in the test population (IC25 ranged from 104-210 mg/L ammonium chloride and LC50 ranged from 163-271 mg/L ammonium chloride). Moreover, the study organisms used by Lazorchak and Smith (2007) were too young to fit within the life stage criteria established for this modeling exercise. In addition, pH was not reported in this study, so accurate normalization was not possible. Broderius and Smith (1979) also exposed small rainbow trout (0.18g) to ammonia over a 30 day period. Significant reductions in growth were seen at 0.32mg NH_3-N/L, but survival was 70% of that observed in the controls (60%), so the quality and usefulness of this data is suspect. The somatic growth model does not incorporate

direct mortality and would greatly underestimate population-level effects if studies where significant mortality occurred were included. Since data for the appropriate life stages or time frames were unavailable, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium: Studies identified by the first round of literature review as having data on acute and chronic toxicity for the freshwater phase of salmonids was examined to gather data for parameterizing the population models. All data were hardness adjusted to 100 mg $CaCO_3$/L and reported as dissolved cadmium in µg/L using the hardness equations found in Mebane (2006). The acute toxicity focused on 96-h mortality data for swimup fry, parr and subyearling smolt. Species mean values (geometric means of LC50 values) were calculated for *Oncorhynchus tshawytscha, O. kisutch, O. mykiss, and O. clarki lewisi* and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species means at 4.53 µg/L (Table A3). Sigmoid slopes were calculated when dose-response data were available. The resulting geometric mean of the slopes was 6.4 and the range was 4.7-7.8 (Besser *et al.,* 2007, Finlayson and Verrue 1982, Davies *et al.,* 1993). Besser *et al.,* 2007 estimated a 28-day LC50 for rainbow trout of 5.5 µg/L (Table A3). The normalized LC50 value of 5.36 µg/L, and the acute slope of 6.4 were used to parameterize the chronic criteria scenario of the mortality model.

Chronic cadmium studies were examined for applicable input data for the somatic growth model. Studies on the effects of cadmium on the growth of subyearling salmonids supported the statement by Mebane (2006) that growth is seldom a sensitive endpoint for cadmium. At concentrations that produced changes in somatic growth, increased mortality was also observed in most studies (Mebane *et al.,* 2008, Brinkman and Hansen 2007, Hansen *et al.,* 2002b). In 24 and 30 day exposures of Atlantic salmon (*Salmo salar*) a reduction in size was seen after alevins were exposed to 6.75-21.8 µg Cd/L but these concentrations also produced 80-90% mortality (Rombough and Garside 1982, Peterson *et al.,* 1983). Bull trout (*Salvelinus confluentus*) fry (0.2g) exposed to 1.57 µg Cd/L for 55 days (hardness adjusted to 100 mg $CaCO_3$/L) showed a 28% reduction in growth at this single time point, along with a 37% reduction in survival (Hansen *et al.* 2002b). No dose response curve for growth was generated by the study, so these data could not be used for extrapolation to other concentrations. Brinkman and Hansen (2007) exposed brown trout fry (*Salmo* trutta) to cadmium for 30 days under different water chemistries and calculated a range of IC20s from 1.7-4.8 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) for reduced growth in the surviving individuals. Mortality chronic values for the same tests ranged from 2.04 to 4.79 µg Cd/L. They also calculated LC50 values for the first 96h of the exposures and these ranged from 3.27 to 6.75 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L). Possible size-selective mortality or growth compensation due to decreased density were not addressed in the study design. Rainbow trout fry exposed to cadmium for 28 days exhibited increased mortality and dry weight at concentrations above a calculated NOEC of 1.3 µg Cd/L (Besser *et al.,* 2007). This may be attributed to size-selective mortality or an increase in somatic growth. One rainbow trout early-life-stage exposure lasting 62 days determined an EC10 for growth of 0.31 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) without the increased mortality (Mebane *et al.,* 2008). Changes in growth at these life stages (Embryos and alevins) are not compatible with the somatic growth model that assesses changes in free-swimming, feeding fry during the linear portion of their growth phase, and could not be used to parameterize the model. Similarly, brook trout (*Salvelinus fontinalis*) exposed to 0.36 µg Cd/L (hardness adjusted to 100

mg $CaCO_3$/L) for 30 days showed reduced prey capture efficiencies and differences in prey selection in artificial stream channels (Riddell *et al.*, 2005a, b), which may link to changes in somatic growth, but this link could not be translated into appropriate input parameters for the current growth model.

Copper: Studies identified by the first round of literature review as having data on acute and chronic toxicity for the freshwater phase of salmonids were examined to gather data needed to establish values for several parameters of the population models. All data was hardness adjusted to 100 mg $CaCO_3$/L using the acute and chronic hardness equations for copper (EPA 2002). For studies with non-laboratory water that reported total instead of dissolved copper, total copper was adjusted by 80% to estimate the dissolved portion of copper in µg/L. The acute toxicity focused on 96-h mortality data for swim-up fry, parr and subyearling fish. Species mean values (geometric means of LC50 values) were calculated (Table A3) and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species. For direct mortality, the genus mean LC50 was 86.8 µg/L with species means ranging from 48.3-190.6 µg/L, while for chronic toxicity (exposures of at least 30 days) the genus mean value was 98.9 µg/L with a range of 73.9-132.2 µg/L. Sigmoid slopes were calculated when dose-response data were available (Table A3). The resulting geometric means (with ranges) of the slopes were 5.2 (4.1-7.6) for the 96-hr exposures and 4.2 (3.1-5.4) for the longer term mortality studies.

Growth studies on fry over 0.2 grams and under 6 grams produced EC50 values ranging from 20.33 µg/L to 112.43 µg/L (all values hardness adjusted, see Table A4 below). Exposures lasted from 15 to 98 days. NOEC values ranged from 5.83 to 113.82µg/L. Mortality was often observed in these studies and ranged from none reported to well over 50% at similar concentrations to those that produced growth effects (Table A4). For example, Besser *et al.* (2005) reported the lowest growth EC50 of 20.33µg/L for 0.2g fry after a 30 day exposure, but also reported a 30 day LC50 of 16.83µg/L with a slope of 5.4 (Table A4). Therefore, similar to the results with cadmium, an analysis of the available literature found that for exposures occurring to subyearling salmonids between 1 and 6g, growth effects often were confounded by mortality since most of the growth studies reported mortality assessment values (LC50s, chronic values, NOECs) that overlapped with or were less than the growth assessment values (EC50s, NOECs; Table A4). Hansen *et al.* 2002c used the IC20 as an endpoint for comparison since concentrations producing over 20% growth inhibition were often accompanied by significant mortality. Many other growth studies found in the literature search were excluded for reasons such as using too few exposure concentrations, using exposures beginning before swim-up (usually just after fertilization), or reporting no effect on growth for the concentrations tested. As mentioned above, in the remaining studies concentrations that produced effects on growth often also showed significant decreases in survival. For example, Mudge *et al.* (1993) reported that, for 3 of their 5 tests in coho, mortality was more sensitive than growth (Table A4). Nonetheless, some limited scenarios were run in the somatic growth model that looked at whether growth alone would be impacted by exposures at the chronic criteria value for copper. The time-to-effect and time-to-recovery values used for copper were both 0.5 days.

Results

Sensitivity Analysis

The sensitivity analysis of all four of the control population matrices predicted the greatest changes in population growth rate (λ) result from changes in first-year survival. Parameter values and their corresponding sensitivity values are listed in Table A1. The elasticity values for the transition matrices also corresponded to the driving influence of first-year survival, with contributions to lambda of 0.33 for coho, 0.29 for ocean-type Chinook, 0.25 for stream-type Chinook and 0.24 for sockeye.

Model Output

Ammonia: Using the genus geometric mean LC_{50} and dose-response slope, with 100% of the population exposed to the criteria concentrations, the direct mortality model output showed 0% mortality to subyearlings and a zero percent change in the population growth rate (lambda) for all four life-history models (Table 2.6.5.47). The lowest species mean value in the *Oncorhynchus* range was also tested at 15.1 mg NH_3-N/L, and resulted in 0% mortality and 0% change in λ. When the chronic criterion was assessed with a 29-d exposure, the direct mortality model predicted no mortality or change in λ.

Studies on chronic exposures of juvenile salmonids to ammonia reported no or very little impacts on somatic growth, but these were accompanied by mortality. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium: Direct mortality population model runs were conducted using exposures to the criteria concentrations and the genus mean value calculated for *Oncorhynchus* (Table A5). This value produced 1% mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC50 and slope values as opposed to the minimum end of the range for species mean values (Table A5). Only when the minimum species mean value and the minimum slope were used, did mortality rise to a level that produced changes in lambda that were greater than the standard deviation of the control models (Table A5). Changes in population growth rates for the stream-type Chinook and coho were larger than one standard deviation from the control models. An estimated 28-day exposure to the chronic criterion produced no mortality or change in lambda.

Studies on chronic cadmium toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model was not run for cadmium.

Copper: Direct mortality population model runs were conducted using exposures to the criteria concentrations and both the acute and chronic parameters calculated for *Oncorhynchus* (Table A5). The acute LC50 and slope produced 0% mortality and no changes in the population growth rate for any of the four life history population models. The chronic LC50 and slope produced 0% mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC50 and slope values as opposed to the minimum end of the range for species mean values but no mortality was projected (Table A5).

Studies on copper toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. In spite of this, some growth model scenarios were run. When the maximum exposure period was used for the chronic criteria value in the growth model (140, 164 or 184 days depending on the life history), with an EC50 of 20.33, slope of 2.7 (Besser 2005) and the chronic criteria value of 9 µg/L, the percent change in lambda ranged from -1 to -4% (depending on life history). None of these reductions exceeded the control standard deviations. A 30-day exposure produced no decline in population growth rates. When a 30 day exposure for direct mortality was modeled using the minimum species values with a LC50 of 73.9µg/L and a slope of 4.2, the chronic criteria (9 µg /L) produced no change in lambda for the four life history models.

Summary

The only scenarios producing direct mortality sufficient to decrease the population growth rates were those using the lowest species mean values for cadmium. The other scenarios assessing the direct mortality from exposure to the suggested criteria values did not result in any changes in the population productivity.

Somatic growth during the freshwater subyearling stage of salmon has been shown to directly influence first year survival, so it was the focus of a literature review and modeling exercise to examine population-level impacts that may result from chemical exposures. In studies assessing growth endpoints of subyearling salmonids greater than 1g exposed to ammonia, cadmium or copper, mortality often confounded any growth effects identified since most studies that reported significant impacts on growth also reported significant simultaneous mortality. The somatic growth models do not include other stressors, such as direct mortality and could underestimate impacts for compounds which have overlapping dose response curves for mortality and somatic growth. In addition, the direct mortality population model inherently requires fewer assumptions regarding exposure and physiology than does the somatic growth population model. Overall, for the two impacts modeled here (direct mortality and somatic growth) we feel it is more appropriate when assessing potential risk to populations from exposures to these compounds during the free-swimming to rearing period of Juvenile salmonids to focus on the direct mortality population model output.

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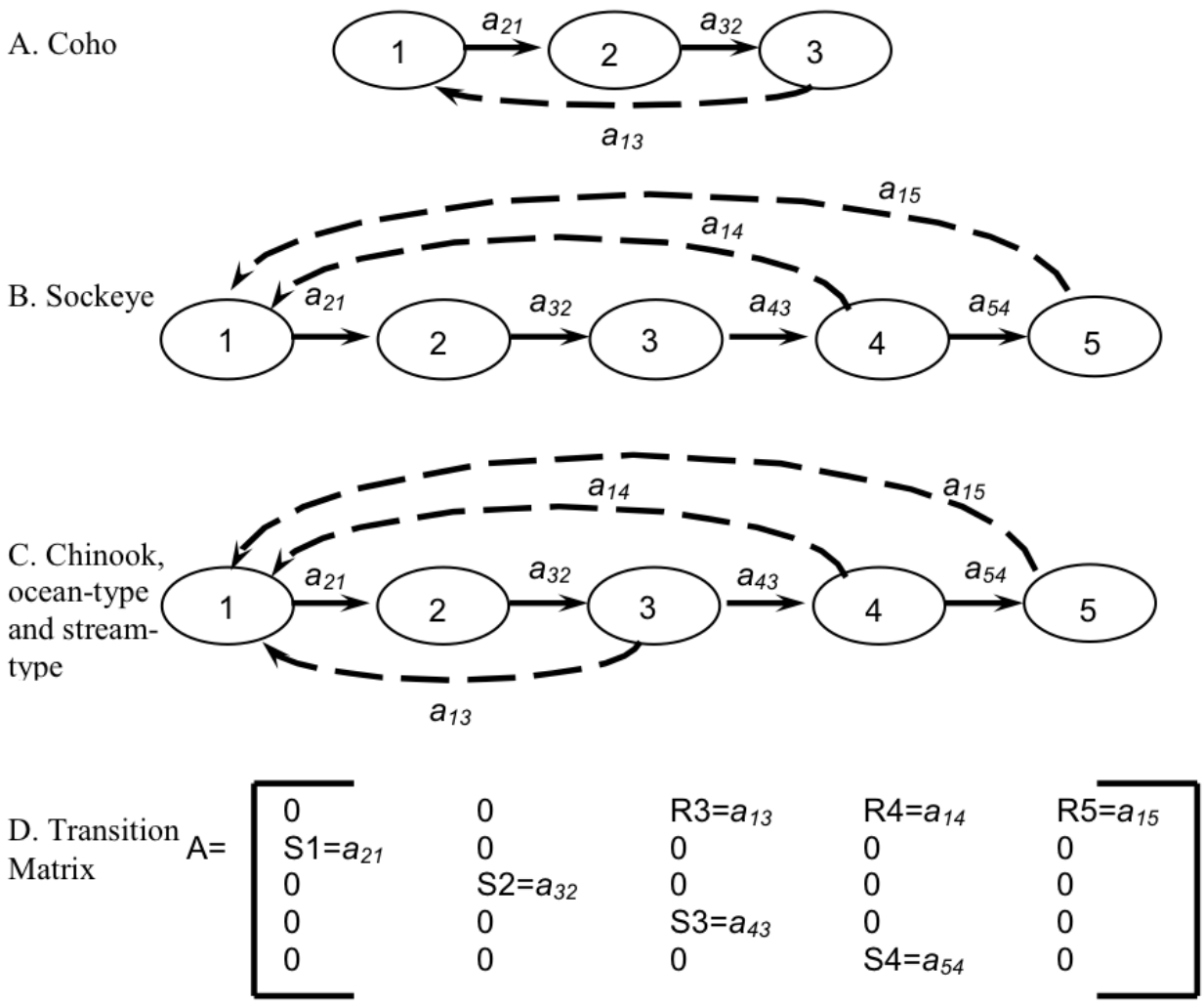


Figure A1. Life-History Graphs and Transition Matrix for coho (A), sockeye (B) and Chinook (C) salmon. The life-history graph for a population labeled by age, with each transition element labeled according to the matrix position, a_{ij}, i row and j column. Dashed lines represent reproductive contribution and solid lines represent survival transitions. D) The transition matrix for the life-history graph depicted in C.

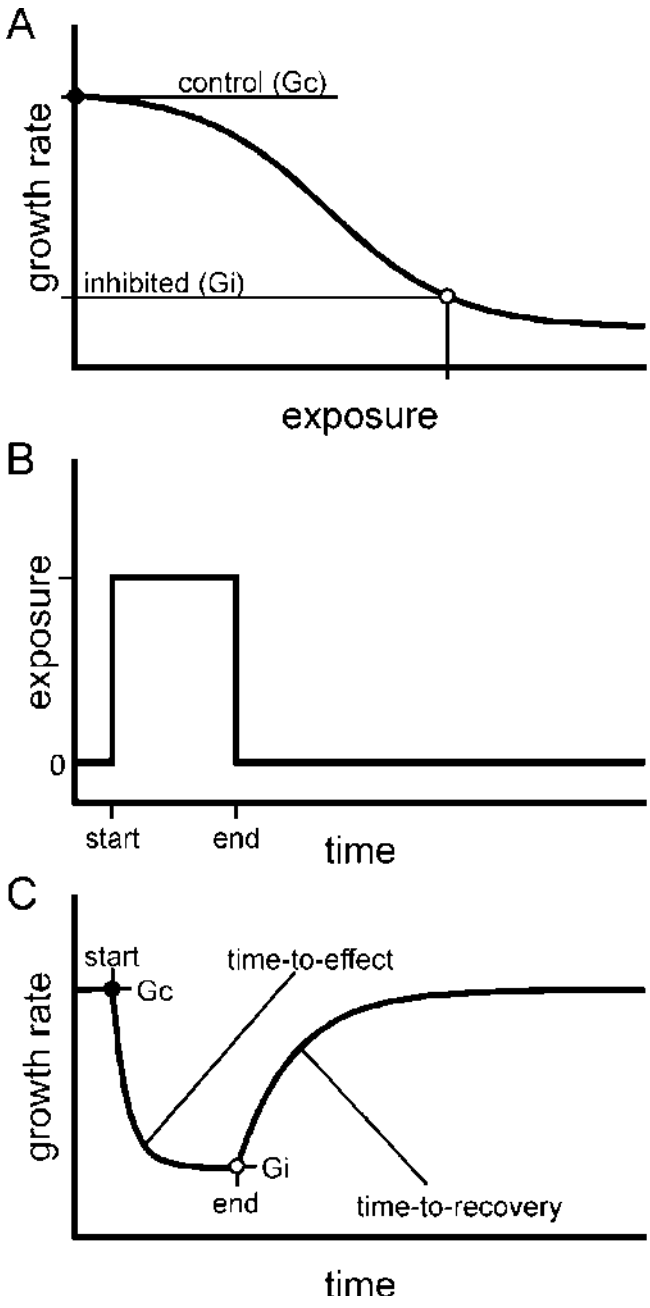

A
control (Gc)
growth rate
inhibited (Gi)
exposure
B
exposure
0
start
end
time
C
start
Gc
time-to-effect
growth rate
Gi
end
time-to-recovery
time

Figure A2.

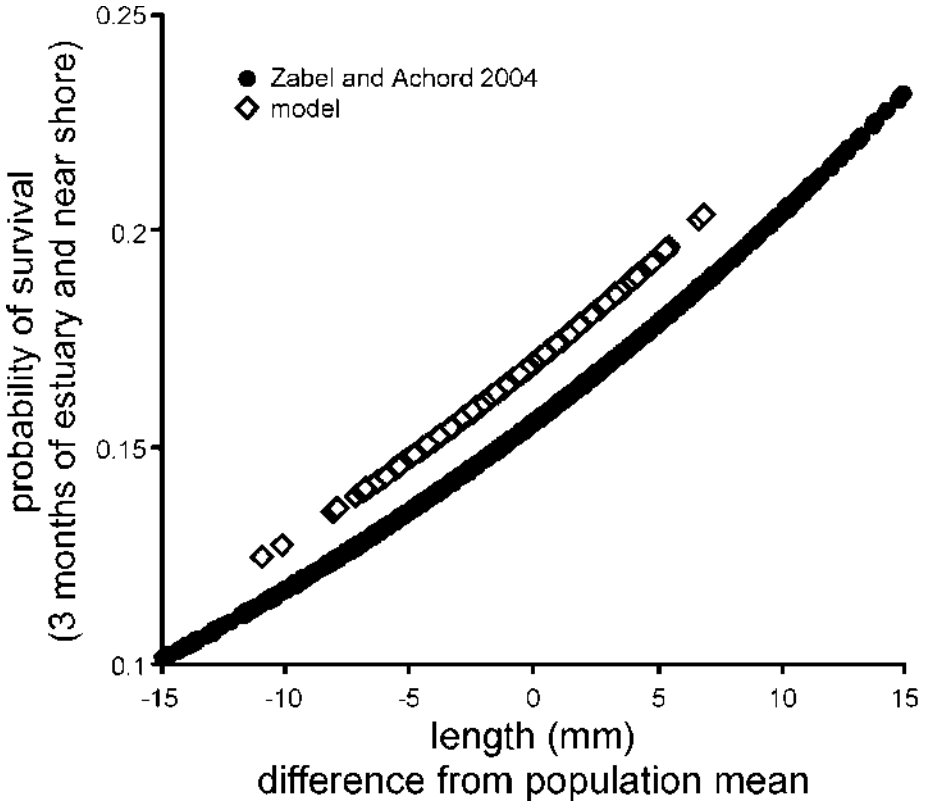


Figure A3. Relationships between difference in length from population mean and probability of survival for three-month period. Values shown are output based upon the original size and survival equations derived by Zabel and Achord (2004) and equations adapted for the model population used in the ocean-type Chinook model. Figure from Baldwin *et al.*, 2009.

Table A1. Matrix transition element and sensitivity (S) and elasticity (E) values for each model species. These control values are listed by the transition element taken from the life-history graphs as depicted in Figure A1 and the literature data described in the method text. Blank cells indicate elements that are not in the transition matrix for a particular species. The influence of each matrix element on λ was assessed by calculating the sensitivity (S) and elasticity (E) values for A. The sensitivity of matrix element a_{ij} equals the rate of change in λ with respect to the transition element, defined by $\delta\lambda/\delta a$. The elasticity of transition element a_{ij} is defined as the proportional change in λ relative to the proportional change in a_{ij}, and equals (a_{ij}/λ) times the sensitivity of a_{ij}. Elasticity values allow comparison of the influence of individual transition elements and comparison across matrices.

Transition Element	Chinook Stream-type			Chinook Ocean-type			Coho			Sockeye		
	Value[1]	S	E	Value[2]	S	E	Value[3]	S	E	Value[4]	S	E
S1	0.0643	3.844	0.247	0.0056	57.13	0.292	0.0296	11.59	0.333	0.0257	9.441	0.239
S2	0.1160	2.132	0.247	0.48	0.670	0.292	0.0505	6.809	0.333	0.183	1.326	0.239
S3	0.17005	1.448	0.246	0.246	0.476	0.106				0.499	0.486	0.239
S4	0.04	0.319	0.0127	0.136	0.136	0.0168				0.1377	0.322	0.0437
R3	0.5807	0.00184	0.0011	313.8	0.0006	0.186	732.8	0.000469	0.333			
R4	746.73	0.000313	0.233	677.1	0.000146	0.0896				379.57	0.000537	0.195
R5	1020.36	1.25E-05	0.0127	1028	1.80E-05	0.0168				608.7	7.28E-05	0.0437

[1] Value calculated from data in Healy and Heard 1984, Fast *et al.* 1988, Beckman *et al.* 2000, Knudsen *et al.* 2006

[2] Value calculated from data in Healey and Heard 1984, Howell *et al.* 1985, Roni and Quinn 1995, Ratner *et al.* 1997, PSCCTC 2002, Green and Beechie 2004, Johnson *et al.* 2007

[3] Value calculated from data in Pess *et al.* 2002, Knudsen *et al.* 2002

[4] Value calculated from data in Pauley *et al.* 1989, Gustafson *et al.* 1997, McGurk 2000

Table A2. Species specific control parameters to model organismal growth and survival rates. Growth period and survival rate are determined from the literature data listed for each species. Gc and α were calculated to make the basic model produce the appropriate size and survival values from the literature.

	Chinook Stream-type[1]	Chinook Ocean-type[2]	Coho[3]	Sockeye[4]
days to run organismal growth model	184	140	184	168
growth rate % body wt/day (Gc)	1.28	1.30	0.90	1.183
α from equation S	-0.33	-1.99	-0.802	-0.871
Control Survival ϕ	0.418	0.169	0.310	0.295

[1] Values from data in Healy and Heard 1984, Fast *et al.* 1988, Beckman *et al.* 2000, Knudsen *et al.* 2006
[2] Values from data in Healey and Heard 1984, Howell *et al.* 1985, Roni and Quinn 1995, Ratner *et al.* 1997, PSCCTC 2002, Green and Beechie 2004, Johnson *et al.* 2007
[3] Values from data in Pess *et al.* 2002, Knudsen *et al.* 2002
[4] Values from data in Pauley *et al.* 1989, Gustafson *et al.* 1997, McGurk 2000

Table A3. Acute and chronic exposure studies providing LC_{50} data used in the direct population mortality model. When multiple experiments were summarized in one paper, the geometric mean is reported here (*). All values were incorporated individually in calculating the species and genus geometric means.

		Exposure Information			LC50			Slope		Geometric Mean	
Species	**Age**	**Days**	**pH**	**Temp (C)**	**reported**	**pH adj**			**Reference**	**LC50**	**slope**
Acute and Chronic Exposure Ammonia											
Chinook	fingerling	4	7.8	7	29.3	26.8			Servizi and Gordon 1990	26.8	
coho	fingerling	4	8.1	17.2	12.1	15.1			Buckley 1978	15.1	
rainbow trout	fingerling	4	7.4	14.5	70.1	18.0			Calamari *et al.*1981		
rainbow trout	fry	4	7.86*	12.9*	35.8*	26.7			Thurston and Russo 1983 (8 tests,1-4g fry)		
rainbow trout	fry	4	7.95	10	36.6	32.7		6.4	Broderius and Smith 1979	26.2	6.40
cutthroat trout	fry	4	7.7	10	29.1	27.0			Thurston *et al.* 1981		
cutthroat trout		fry	4	7.8*	12.6*	47.7*	30.1		Thurston *et al.* 1978 (4 tests)		29.4
Genus mean - acute										**23.6**	**6.40**
cutthroat trout		fry	29	7.8*	12.6*	33.6*	21.3		Thurston *et al.* 1978 (4 tests)		
Genus mean - chronic											**21.3**
Acute and Chronic Exposure Cadmium			**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Chinook	swimup	4	24	total	1.8	5.94	5.61		Chapman 1978		
Chinook	fingerling	4	25	total	1.41	4.50	4.25		Chapman 1978		
Chinook	fingerling	4	21	total	1.1	4.06	3.83		Finlayson and Verrue 1982		
Chinook	parr	4	24	total	3.5	11.55	10.91		Chapman 1978	5.62	6.90
coho	fry	4	22	total	3.66	12.99	12.27		Chapman 1975		
coho	fry	4	22	total	2.76	9.80	9.25		Chapman 1975		
coho	fry	4	22	total	1.73	6.14	5.80		Chapman 1975		
coho	fry	4	22	total	1.4	4.97	4.69		Chapman 1975		

coho	fry	4	22	total	2.7	9.59	9.05		Chapman 1975		
rainbow trout	swimup	4	23	total	1.3	4.45	4.20		Chapman 1978	7.75	
rainbow trout	swimup	4	7.5	dissolved	0.48	4.19	3.96		Windward 2002		
rainbow trout	swimup	4	14	dissolved	0.97	5.03	5.03		Windward 2002		
		Exposure Information			**LC50**			**Slope**		**Geometric Mean**	
Species	**Age**	**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Acute and Chronic Exposure Cadmium – cont.											
rainbow trout	swimup	4	21	dissolved	0.84	3.10	3.10		Windward 2002		
rainbow trout	swimup	4	24	dissolved	1.3	4.29	4.29		Windward 2002		
rainbow trout	swimup	4	26	dissolved	1.58	4.88	4.88		Windward 2002		
rainbow trout	swimup	4	26	dissolved	1.61	4.97	4.97		Windward 2002		
rainbow trout	swimup	4	29	dissolved	0.83	2.34	2.34		Windward 2002		
rainbow trout	swimup	4	30	dissolved	0.99	2.71	2.71		Windward 2002		
rainbow trout	swimup	4	32	dissolved	0.89	2.31	2.31		Windward 2002		
rainbow trout	fry	4	103	total	3.7	3.61	3.61		Besser et al 2007		6.57
rainbow trout	fry	4	103	total	5.2	5.07	5.07		Besser et al 2007		7.78
rainbow trout	fry	4	103	total	5.4	5.27	5.27		Besser et al 2007		
rainbow trout	fry	4	400	total	5.92	1.86	1.75		Davies et al 1993		
rainbow trout	fry	4	200	total	6.57	3.68	3.47		Davies et al 1993		
rainbow trout	fry	4	50	total	3.08	5.50	5.19		Davies et al 1993		4.70
rainbow trout	fry	4	140	total	22	16.60	15.67		Hollis et al 1999		
rainbow trout	fry	4	9.2	total	0.5	3.68	3.48		Cusimano et al 1986		
rainbow trout	fry	4	28	total	0.47	1.36	1.29		Hansen et al 2002a		
rainbow trout	fry	4	30	total	0.51	1.40	1.32		Hansen et al 2002a		
rainbow trout	fingerling	4	44	total	3	5.96	5.63		Phipps and Holcombe 1985		
rainbow trout	parr	4	23	total	1	3.42	3.23		Chapman 1978	3.63	
West Slope cutthroat trout	fry	4	21	dissolved	0.35	1.29	1.29		EVS 1996		
West Slope cutthroat trout	fry	4	21	dissolved	0.93	3.43	3.43		Windward 2002		
West Slope cutthroat trout	fry	4	32	dissolved	1.41	3.66	3.66		Windward 2002		

West Slope cutthroat trout	fry	4	31	dissolved	1.18	3.14	3.14		Windward 2002	2.67	
Genus mean -acute										**4.53**	**6.38**
rainbow trout	swimup	28	103	dissolved	5.50	5.36	5.36		Besser et al 2007	5.36	
Genus mean - chronic										**5.36**	
		Exposure Information			**LC50**			**Slope**		**Geometric Mean**	
Species	**Age**	**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Acute Exposure Copper											
Chinook	alevin	4	23	dissolved	26	103.84	103.84		Chapman 1978		
Chinook	fry	4	23	dissolved	19	75.88	75.88		Chapman 1978		
Chinook	fry	4	21	total	32	139.24	111.39	4.2	Finlayson 1982		
Chinook	fry	4	35	dissolved	12.5	33.61	33.61	2.7	Welsh 2000		
Chinook	fry	4	38	dissolved	14.3	35.58	35.58	4.2	Welsh 2000		
Chinook	fry	4	36	dissolved	18.3	47.92	47.92	3.8	Welsh 2000		
Chinook	fry	4	36	dissolved	7.4	19.38	19.38	9	Welsh 2000		
Chinook	fry	4	25	dissolved	33.1	122.20	122.20		Chapman 1982		
Chinook	fry	4	211	dissolved	54	26.72	26.72		Hamilton 1990		
Chinook	fry	4	211	dissolved	58	28.70	28.70		Hamilton 1990		
Chinook	juvenile	4	100	dissolved	50	50.00	50.00		Chapman 1977		
Chinook	juvenile	4		total	180			4.6	Holland 1960		
Chinook	parr	4	23	dissolved	38	151.76	151.76		Chapman 1978		
Chinook	smolt	4	23	dissolved	26	103.84	103.84		Chapman 1978	57.31	4.42
coho	alevin	1	41	dissolved	67	155.21	155.21		Buhl 1990		
coho	alevin	4	41		20	46.33	46.33		Buhl 1990		
coho	fry	4	31	total	44	132.65	106.12		Mudge 1993		
coho	juvenile	1	41	dissolved	23.4	54.21	54.21		Buhl 1990		
coho	juvenile	1	41	dissolved	42.2	97.76	97.76		Buhl 1990		
coho	juvenile	1	41	dissolved	62.3	144.32	144.32		Buhl 1990		
coho	juvenile	4	33	dissolved	17	48.32	48.32		Buckley 1983		
coho	juvenile	4	41	dissolved	15.1	34.98	34.98		Buhl 1990		
coho	juvenile	4	41	dissolved	23.9	55.36	55.36		Buhl 1990		
coho	juvenile	4	41	dissolved	31.9	73.90	73.90		Buhl 1990		
coho	juvenile	4	128	total	60	47.55	38.04		Hedtke 1982		

coho	juvenile	4	128	total	81	64.19	51.35		Hedtke 1982		
coho	juvenile	4	128	total	150	118.87	95.10		Hedtke 1982		
coho	juvenile	4	128	total	166	131.55	105.24		Hedtke 1982		
coho	juvenile	4	128	total	212	168.01	134.40		Hedtke 1982		
coho	juvenile	4	128	total	192	152.16	121.72		Hedtke 1982		
		Exposure Information			**LC50**			**Slope**		**Geometric Mean**	
Species	**Age**	**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Acute Exposure Copper – Cont.											
coho	juvenile	4	95	total	60	62.97	50.38		Lorz 1976		
coho	juvenile	4	95	total	72	75.57	60.45		Lorz 1976		
coho	juvenile	4	94	total	61	64.66	51.73	5.3	Lorz 1977		
coho	juvenile	4	94	total	71	75.26	60.21	9.6	Lorz 1977		
coho	juvenile	4	94	total	73	77.38	61.91	9.7	Lorz 1977		
coho	juvenile	4	94	total	55	58.30	46.64	6.7	Lorz 1977		
coho	parr	4	31	total	67	201.98	161.59		Mudge 1993		
coho	smolt	4	31	total	44	132.65	106.12		Mudge 1993	73.44	7.58
pink	alevin	4	83	total	143	170.44	136.35		Servizi 1978		
pink	alevin	4	83	total	83	98.93	79.14		Servizi 1978		
pink	fry	4	83	total	199	237.19	189.75		Servizi 1978	126.99	
sockeye	alevin	4	83	total	190	226.46	181.17		Servizi 1978		
sockeye	alevin	4	83	total	120	143.03	114.42		Servizi 1978		
sockeye	fry	4	83	total	150	178.79	143.03		Servizi 1978		
sockeye	parr	4	41	total	240	555.96	444.77		Davis 1978		
sockeye	smolt	4	83	total	200	238.38	190.71		Servizi 1978	190.59	
rainbow trout	alevin	1	41	dissolved	46.4	107.49	107.49		Buhl 1990		
rainbow trout	alevin	4	41	dissolved	36	83.39	83.39		Buhl 1990		
rainbow trout	fry	4	103	dissolved	48	46.68	46.68	4.8	Besser 2007		
rainbow trout	fry	4	90	dissolved	17.2	19.00	19.00	4.4	Welsh 2000		
rainbow trout	fry	4	42	dissolved	3.4	7.70	7.70	3	Welsh 2000		
rainbow trout	fry	4	90	dissolved	32	35.34	35.34	6.7	Welsh 2000		
rainbow trout	fry	4	39	dissolved	8.1	19.67	19.67	2.8	Welsh 2000		
rainbow trout	juvenile	1	41	dissolved	18.9	43.78	43.78		Buhl 1990		

rainbow trout	juvenile	4	100	dissolved	22	22.00	22.00		Gish 1971		
rainbow trout	juvenile	4	100	dissolved	30	30.00	30.00		Taylor 2000		
steelhead	alevin	4	23	dissolved	28	111.82	111.82		Chapman 1978		
steelhead	fry	4	23	dissolved	17	67.89	67.89		Chapman 1978		
steelhead	juvenile	4	22	dissolved	20	83.29	83.29		Chapman 1973		
		Exposure Information			**LC50**			**Slope**		**Geometric Mean**	
Species	**Age**	**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Acute Exposure Copper – Cont.											
steelhead	parr	4	23	dissolved	18	71.89	71.89		Chapman 1978		
steelhead	parr	4	31	total	57	171.84	137.47		Mudge 1993		
steelhead	smolt	4	23	dissolved	29	115.82	115.82		Chapman 1978	48.34	4.12
Genus mean - acute										**86.79**	**5.17**
Chronic exposure Copper		**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
coho	fry	120	31	total	60	163.22	130.58		Mudge 1993		
coho	fry	120	31	total	80	217.63	174.11		Mudge 1993		
coho	fry	120	31	total	39	106.10	84.88		Mudge 1993		
coho	parr	120	31	total	69	187.71	150.17		Mudge 1993		
coho	parr	120	31	total	52	141.46	113.17		Mudge 1993		
coho	parr	120	31	total	70	190.43	152.34		Mudge 1993		
coho	parr	120	31	total	65	176.83	141.46		Mudge 1993	132.23	
rainbow trout	fry	30	170	total	33.1	21.03	16.83	5.4	Besser 2005		
rainbow trout	fry	56	100	dissolved	55.1	55.10	55.10	4.7	Hansen 2002c		
rainbow trout	fry	28	103	dissolved	56	54.60	54.60	3.1	Besser 2007		
steelhead	parr	120	31	total	84	228.51	182.81		Mudge 1993		
steelhead	parr	120	31	total	70	190.43	152.34		Mudge 1993		
steelhead	parr	120	31	total	53	144.18	115.34		Mudge 1993	73.88	4.29
Genus mean - chronic										**98.84**	**4.29**

Table A4. Copper studies identified that investigated the impacts of copper exposure on juvenile growth.

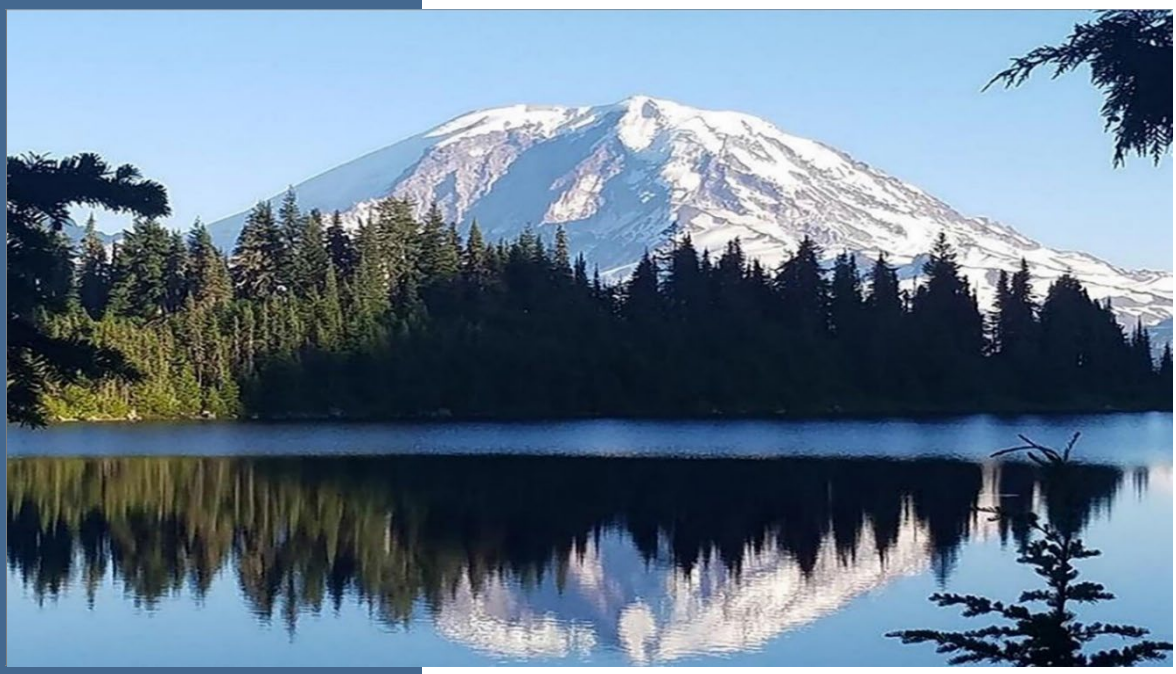
		Exposure Information									
Species	**Age (size)**	**Days**	**Hardness**	**Measurement**	**Uncorrected Value µg/L**	**hardness adj**	**dissolved adj**	**Notes**	**slope**	**Reference**	**Mortality reported with correction**
rt	fry (swim-up)	30	170	total	40	25.42	**20.33**	EC50, size not specified, fed ad libitum	2.7	Besser 2005	16.78 µg/l LC50, 5.4 slope
rt	fry (0.2 g)	28	103	dissolved	59	57.53	57.53	28% dec in biomass		Besser 2007	50% at 57.53 µg/l
coho	juv (6 g)	98	280	dissolved	271	112.43	**112.43**	EC50	1.28	Buckley 1982	
rt	parr (1.7 - 3.3 g)	21	374	total	194	62.85	50.28	~50% dec in growth, ration based on init biomass		Dixon 1981	
rt	fry (0.2 g)	56	105	dissolved	54	51.79	**51.79**	EC50, fed fixed ration (3.5%)	1.4	Hansen 2002c	52.75 µg/l LC50, 4.7 slope
rt	juv (20 g)	28	120	total	52	44.50	35.60	56% dec in growth, fixed ration		Kamunde 2005	26% at 35.60 µg/l
rt	fry (0.12 g)	60	25	total	13	42.50	**34.00**	EC50, fed fixed ration (4.5%)	1.5	Marr 1996	
rt	juv (3.2 g)	35	140	total	75	56.26	45.01	no effect reported, only conc tested		McGeer 2000	
coho	fry (na)	60	26	total	21	66.39	53.11	NOEC		Mudge 1993	45.53 µg/l NOEC
steelhead	parr (na)	60	26	total	45	142.27	113.82	NOEC		Mudge 1993	60.70 µg/l NOEC
rt	fry (0.1 g)	15	135	total	5	3.87	**3.10**	EC50, fed excess of satiation	1.8	Neville 1995	3.40 µg/l LC50, 2.6 slope
rt	juv (18-20 g)	28	120	total	52	44.50	**35.60**	49% dec in wt, only conc tested, consumption meas		Nyogi 2006	
rt	juv (1-2 g)	30	120	total	62	53.06	42.44	NOEC (highest tested), fed fixed ration		Taylor 2000	
rt	juv (1-2 g)	30	20	total	1.7	6.73	5.38	NOEC (highest tested), fed fixed ration		Taylor 2000	
rt	juv (5-6 g)	20	100	total	77	77.00	**61.60**	EC50 from eq 1 (@pH 7.5, 5.5 g)		Waiwood 1978	

rt = rainbow trout

Table A5. Direct mortality population model scenarios for ammonia, cadmium and copper criteria. Standard scenarios used the genus mean values for the criteria. Since no effect resulted, the minimum species mean values were assessed. * indicates a percent change in lambda of greater than one standard deviation from the baseline population model (Chinook ocean-type 9, Chinook stream-type 3, Sockeye 6, Coho 5).

		Mortality input parameters			Output	% change in lambda			
Chemical	Test length	LC_{50}	Sigmoid slope	Criteria Conc.	Percent mortality	Chinook ocean-type	Chinook stream-type	Sockeye	Coho
		(mg/L)							
Ammonia	96-hr	23.6[1]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	96-hr	15.1[2]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	29-d	21.3	6.4[3]	1.7	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Cadmium	96-hr	4.53[1]	6.4[1]	2.0	1	0(13)	0(4)	0(8)	0(7)
Cadmium	96-hr	4.53[1]	4.7[2]	2.0	2	-1(13)	-1(4)	-1(8)	-1(7)
Cadmium	96-hr	2.67[2]	6.4[1]	2.0	14	-4(12)	-3(4)	-3(8)	-5(7)
Cadmium	96-hr	2.67[2]	4.7[2]	2.0	20	-7(12)	-5*(4)	-5(8)	-7(7)
Cadmium	28-d	5.36[1]	6.4[3]	0.25	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Copper	96-hr	86.8[1]	5.2[1]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	96-hr	48.3[2]	4.1[2]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	98.9[1]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	73.9[2]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)

[1]Genus Geometric Mean for *Oncorhynchus* values
[2]Minimum Species Mean value from the range of *Oncorhynchus* values
[3]Slope for chronic exposures not identified, used Genus Mean slope from 96-hr exposures



Proposed updates to Aquatic Life Toxics Criteria, WAC 173-201A-240

Technical Support Document

Water Quality Program

Washington State Department of Ecology

Olympia, Washington

February 2024, Publication 24-10-007

DEPARTMENT OF ECOLOGY
State of Washington

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Publication 23-10-040: Focus on: Aquatic Life and Toxic Substances
Publication 24-10-008: Draft Rule Implementation Plan
Publication 24-10-009: Preliminary Regulatory Analyses

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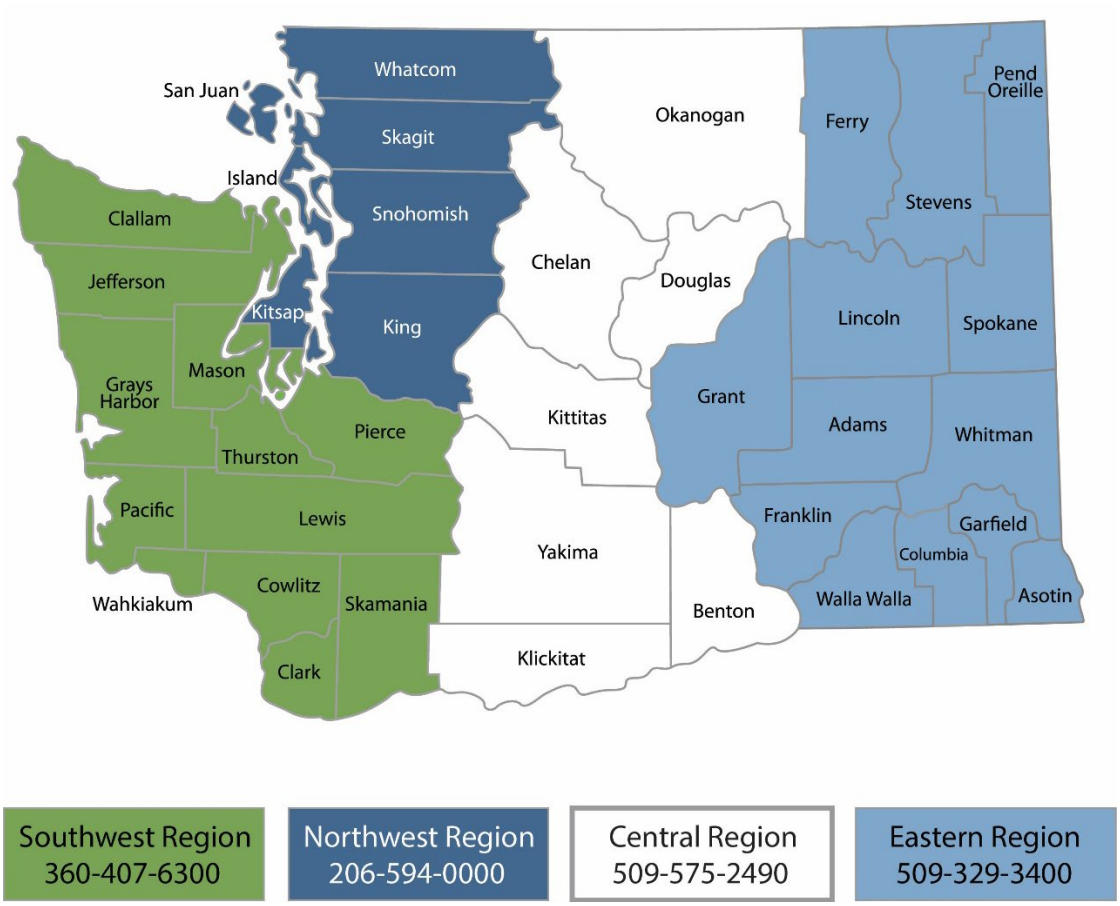
Department of Ecology's Regional Offices

Map of Counties Served



Southwest Region 360-407-6300	Northwest Region 206-594-0000	Central Region 509-575-2490	Eastern Region 509-329-3400

Region	Counties served	Mailing Address	Phone
Southwest	Clallam, Clark, Cowlitz, Grays Harbor, Jefferson, Mason, Lewis, Pacific, Pierce, Skamania, Thurston, Wahkiakum	PO Box 47775 Olympia, WA 98504	360-407-6300
Northwest	Island, King, Kitsap, San Juan, Skagit, Snohomish, Whatcom	PO Box 330316 Shoreline, WA 98133	206-594-0000
Central	Benton, Chelan, Douglas, Kittitas, Klickitat, Okanogan, Yakima	1250 W Alder St Union Gap, WA 98903	509-575-2490
Eastern	Adams, Asotin, Columbia, Ferry, Franklin, Garfield, Grant, Lincoln, Pend Oreille, Spokane, Stevens, Walla Walla, Whitman	4601 N Monroe Spokane, WA 99205	509-329-3400
Headquarters	Across Washington	PO Box 46700 Olympia, WA 98504	360-407-6000

Proposed updates to Aquatic Life Toxics Criteria, WAC 173-201A-240

Technical Support Document

Water Quality Program
Washington State Department of Ecology
Headquarters

Olympia, WA

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Acknowledgements

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Abbreviations and Acronyms

µg/L	micrograms per liter
ACR	Acute to chronic ratio
BCF	Bioconcentration factor
BE	Biological evaluation
BiOp	Biological opinion
BLM	Biotic ligand model
CaCO3	Calcium carbonate
CCC	Criterion continuous concentration
CF	Conversion factor
CFR	Code of Federal Regulations
CMC	Criterion maximum concentration
CWA	Clean Water Act
DOC	Dissolved organic carbon
EIM	Environmental Information Management
EPA	Environmental Protection Agency
ESA	Endangered Species Act
FACR	Final acute to chronic ratio
FAV	Final acute value
FW	Freshwater
GMAV	Genus mean acute value
GSD	Genus Sensitivity Distribution
LAA	Likely to adversely affect
LC50	Lethal Concentration 50

LOER	Lowest observed effect residue
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MLR	Multiple linear regression model
NLAA	Not likely to adversely affect
NMFS	National Marine Fisheries Service
NOER	No observed effect residue
ODEQ	Oregon Department of Environmental Quality
PPA	Performance Partnership Agreement
SMAC	Species mean acute value
SW	Saltwater
TOC	Total organic carbon
USFWS	United States Fish and Wildlife Services
WAC	Washington Administrative Code

Executive Summary

The Department of Ecology is proposing to amend chapter 173-201A Washington Administrative Code (WAC) Water Quality Standards for Surface Waters of the State of Washington. These proposed changes include revising the aquatic life toxics criteria in WAC 173-201A-240. The purpose of this document is to provide background and technical analysis for the proposed aquatic life toxics criteria.

We compared Environmental Protection Agency (EPA) nationally recommended aquatic life toxics criteria against Washington's current criteria to determine if updates are needed. If updates were deemed necessary, we evaluated previous Endangered Species Act (ESA) consultations from the National Marine Fisheries Service (NMFS) and United States Fish and Wildlife Service (USFWS) Biological Opinions (BiOps) from Idaho and Oregon to determine whether additional considerations are needed to protect ESA-listed species in Washington. We used information from Oregon and Idaho BiOps for similarly listed species in Washington to determine if Washington's endangered species and their populations need additional protection. We also used the Swinomish Tribe Biological Evaluation by EPA to inform decisions to update criteria.

We considered available ESA consultation information for this rule update because the process and goals for evaluating species protection is different for NMFS and USFWS compared to EPA. The aim of EPA's aquatic life criteria is to protect 95% of genera. The ESA consultation process evaluates protection of endangered species populations by evaluating impacts to individual species of a population. If population modeling indicates that the proposal could lead to harm of a species population (referred to as "jeopardy"), then the criteria will be disapproved.

Previous ESA consultations in Oregon and Idaho have indicated that EPA's recommendations for some aquatic life toxics may not adequately protect ESA listed species in Washington. If select toxics were not deemed "approvable" through ESA consultation, we evaluated new scientific data, alternative methods to calculate criteria, and new modeling tools as remedies to providing additional protection to aquatic life species. In instances where EPA recommendations are not likely to provide protection for endangered species populations, we used an alternative method to derive more protective criteria.

EPA recommends deriving criteria using the 5th percentile of the toxicity data distribution. We derived the 1st percentile of the toxicity data distribution to provide additional protection that equates to protection of 99% of genera 99% of the time. More stringent protection levels were applied when previous BiOps indicated endangered species vulnerability to extinction at toxic concentrations equal to EPA's national recommendations and when new science alone did not provide adequate protection. While EPA's national recommendations are generally protective and endangered species are usually not more chemically sensitive, there are instances where a higher protection level is needed to prevent populations from extinction.

Decisions for each toxic are provided in this document alongside information on previous ESA consultations in Region 10 states, criteria calculations, new science, and proposed numeric values.

BACKGROUND

Updating the aquatic life toxics criteria is a high priority for Ecology and was included in the Five-Year Work Plan developed as part of the 2010 triennial review. Ecology decided it would be most beneficial for our state to wait until final Endangered Species Act (ESA) consultations and subsequent EPA approvals had been completed for adjacent states before moving forward with adopting aquatic life toxics criteria in order to increase the likelihood they would meet ESA considerations and be approved by EPA. Ecology decided to move forward with developing human health toxics criteria as a higher priority, to be followed by aquatic life toxics criteria when there was more certainty which EPA-recommended criteria would be approvable through ESA consultation. The decision to prioritize human health criteria updates ahead of aquatic life toxics criteria was made, in part, because of significant delays in the several ESA consultations for EPA's nationally recommended aquatic life toxics criteria in other states.

More recently, updates to aquatic life toxics criteria were outlined in our performance partnership agreement (PPA) with EPA in 2021 and in our most recent triennial review report[2] submitted to EPA in April 2022. During the triennial review, we received overwhelming public support for updating rules for aquatic life toxics criteria based on new information and approaches to aquatic life protection. As part of this process, we considered and received feedback on several approaches to a rulemaking. Based on feedback, we decided to proceed with updating all necessary aquatic life toxics criteria in a single rulemaking. This decision is influenced in part by ongoing litigation for EPA to evaluate and potentially promulgate aquatic life toxics criteria for Washington.

We anticipated that a single rulemaking of all aquatic life toxics criteria will be more efficient than multiple rulemakings. Stakeholders, Tribes, and other interested parties will be able to engage in the full scope of aquatic life toxic criteria considerations within one rulemaking, without Ecology placing one toxic substance or group of substances on an earlier rule schedule than others.

[2] https://apps.ecology.wa.gov/publications/summarypages/2210002.html

INTRODUCTION

Overview

Under Clean Water Act (CWA) regulations, any revisions to a state's surface water quality standards must be approved by EPA and may be subject to review of potential impacts to endangered species. The last major update to Washington's aquatic life toxics criteria was in 1992 in response to impending federal promulgation, called the National Toxics Rule, for states that had insufficient protections for certain toxic substances. Ecology chose to adopt most aquatic life toxics criteria that were recommended by EPA prior to this promulgation, and EPA approved updates to some of Washington's aquatic life toxics criteria in 1993. Washington has made minor updates to their aquatic life criteria as recently as 2007. Since the National Toxics Rule of 1992, EPA has added additional toxic substances to their list of recommended criteria and provided several updates to previously established criteria.

In this rulemaking, we compared EPA's nationally recommended aquatic life toxics criteria against Washington's current criteria to determine if updates are needed. We also considered any draft EPA criteria that may not be finalized before the rule proposal phase of this rulemaking. Furthermore, we evaluated previous ESA consultations from the NMFS' and USFWS' Biological Opinions (BiOps) from other Pacific Northwest states (i.e., Idaho and Oregon) to determine whether additional considerations are needed to protect ESA-listed species in Washington. We also used the Swinomish Tribe Biological Evaluation by EPA to inform our decisions.

EPA Region 10 states have submitted updates to their aquatic life toxics criteria over the past few decades, but EPAs required ESA Section 7 consultations with the National Oceanographic and Atmospheric Administration National Marine Fisheries Service (NMFS) and the U.S. Fish and Wildlife Service (USFWS) have been significantly delayed for several states (such as Oregon and Idaho). EPA's consideration of Oregon's aquatic life toxics criteria adopted in 2004 was significantly delayed as the federal agencies worked through ESA consultation. In 2013, EPA disapproved several aquatic life criteria that the Oregon Environmental Quality Commission (ODEQ) adopted in 2004. Since 2013, ODEQ adopted and EPA approved revisions to several of the disapproved criteria. EPA's approvals of Idaho's aquatic life criteria likewise were stalled, leaving the state-adopted aquatic life criteria unusable for CWA actions for several years.

Previous ESA consultations for EPA nationally recommended criteria in Idaho and Oregon have indicated some aquatic life toxics may not adequately protect ESA listed species in Washington. If select toxics were not deemed "approvable" through ESA consultation in Idaho and Oregon for similarly listed species in Washington, then we evaluated new scientific data, alternative methods to calculate criteria, and the new modeling tools as remedies to provide full protection to endangered species and their populations.

Clean Water Act – Water Quality Standards

The CWA was established to regulate discharges of pollutants into water of the United States and regulate quality standards for surface waters. Under Section 303(c) of the CWA and federal

implementing regulations at 40 Code of Federal Regulations (CFR) § 131.4, states and authorized Tribes have the primary responsibility for reviewing, establishing, and revising water quality standards. Water quality standards consist primarily of the designated uses of a waterbody or waterbody segment, the water quality criteria that protect those designated uses, and an antidegradation policy to protect high quality waters.

EPA has compiled a list of nationally recommended water quality criteria for the protection of aquatic life and human health in surface waters. These criteria are published pursuant to Section 304(a) of the CWA and provide guidance for states and Tribes to establish water quality standards and provide the foundation for controlling the release of pollutants and identifying impaired waters. The state water quality standards are federally approved by EPA and describe the level of protection for Waters of the State.

All state-adopted water quality standards are required to be submitted to EPA for review and approval (or disapproval). If EPA does not approve state water quality standards, then they are required to promulgate federal water quality standards for states that do not adopt standards (unless the state resubmits a revised rule package to EPA). The following outlines the steps and timing of the federal action:

1. Ecology submits the adopted rule to EPA.
2. EPA reviews the submittal for acceptability under the CWA.
3. EPA has 60 days to approve or 90 days to disapprove the State's rule.

EPA is required to evaluate potential impacts of the state-adopted aquatic life criteria to endangered species. EPA writes a Biological Evaluation (BE) that describes effects that the rule package (i.e., the "action") may have on endangered species. If EPA's approval of the rule is likely to adversely affect endangered species (LAA), EPA will request ESA Section 7(a)(2) consultation with NMFS and USFWS to determine if the action would jeopardize those species. Alternatively, EPA can designate the proposal as not likely to adversely affect (NLAA) endangered species. If a LAA determination is made, USFWS and NMFS write BiOps that analyze the effects of the rule to ESA listed species. The conclusion of the BiOps will state if any part of the rule is likely to jeopardize the continued existence of a listed species or harm critical habitat. A jeopardy call can lead to a disapproval of a rule or portion of a rule if EPA cannot conclude that the rule is protective of the applicable designated uses, which include consideration of ESA-listed species. BiOps can include conservation recommendations or reasonable and prudent actions to minimize any "take" of listed species. A likely to adversely affect determination with no jeopardy means that effects to endangered species are measurable, observable, and likely to occur, but will not affect the continued existence of the species at the population level or landscape scale (i.e., critical habitat).

Endangered Species Act Consultation

Background

The Endangered Species Act (ESA) of 1973 (16 U.S.C. 1531 *et seq*.), as amended, establishes a national program for conserving threatened and endangered species of fish, wildlife, plants, and the habitat on which they depend. Section 7(a)(2) of the ESA requires federal agencies to

ensure, in consultation with the USFWS and the NMFS, as appropriate, that their actions are not likely to jeopardize the continued existence of endangered or threatened species or adversely modify or destroy their designated critical habitats. This is called "jeopardy." Section 7(a)(4) of the ESA requires federal agencies to confer with USFWS and NMFS, as appropriate, in cases where the agency or the Services have determined that a proposed or ongoing Federal action is likely to jeopardize the continued existence of species proposed to be listed under section 4 of the ESA or result in the destruction or adverse modification of critical habitat proposed to be designated for such species.

The USFWS also encourages federal agencies to confer on actions that may affect a proposed species or proposed critical habitat. In such cases, conference concurrence determinations or conference opinions can be adopted as formal concurrences or biological opinions, respectively, after a proposed species is listed or the critical habitat is designated.

In accordance with policy and regulation, the jeopardy analysis relies on four components:

1. The *Status of the Species*, which evaluates the species' rangewide condition, the factors responsible for that condition, and its survival and recovery needs.
2. The *Environmental Baseline*, which evaluates the condition of the species in the action area, the factors responsible for that condition, and the relationship of the action area to the survival and recovery of the species.
3. The *Effects of the Action*, which determines the direct and indirect impacts of the proposed Federal action and the effects of any interrelated or interdependent activities on the species.
4. *Cumulative Effects*, which evaluates the effects of future, non-Federal activities in the action area on the species.

The jeopardy call is made by evaluating the effects of the proposed federal action in the context of the species' current status, taking into account any cumulative effects, to determine if implementation of the proposed action is likely to cause an appreciable reduction in the likelihood of both the survival and recovery of the species in the wild.

Both the BE (written by EPA) and the BiOps (written by USFWS and NMFS) contain a discussion of the effects of each water quality standard adopted by the state and submitted to EPA. These analyses could result in three potential effect outcomes for each standard: (1) no effect; (2) not likely to adversely affect (NLAA); or (3) likely to adversely affect (LAA).

The following sections provide information on the outcomes of ESA consultation for Oregon, Idaho, and information from EPA's BE of the Swinomish Indian Tribal Community following their submittal of aquatic life toxics criteria.

Oregon

Oregon Department of Environmental Quality (ODEQ) submitted revised water quality standards for aquatic life toxics criteria on July 8, 2004. The updated criteria incorporated EPA recommended criteria for toxic pollutants that were current at the time. USFWS received a letter from EPA requesting formal consultation on January 16, 2008. The BiOp for Oregon's 2004 submittal was completed in 2012. Table 1 and Table 2 provides a summary of the results

of Oregon's ESA consultation for the adoption of EPA recommended criteria in 2012 and the toxics criteria that had jeopardy calls (or likely to adversely affect endangered species; USFWS, 2012; NMFS, 2012). Oregon's endangered species list is different from Washington, but the two states do share common endangered species such as the Chinook salmon. Thus, we only used ESA consultation information for similarly listed species in Washington.

Table 1. Oregon aquatic life toxics criteria submitted in 2004.

Substance	Freshwater Acute Criteria (µg/L)		Freshwater Chronic Criteria (µg/L)		Saltwater Acute Criteria (µg/L)		Saltwater Chronic Criteria (µg/L)	
	Previous	Proposed	Previous	Proposed	Previous	Proposed	Previous	Proposed
Aluminum	N/A	750	N/A	87	-	-	-	-
Ammonia (@pH 8 & 20C)	6	5.6 (salmonids) 8.4 (no salmonids)	0.76 (salmonids) 1.08 (no salmonids)	1.7	-	-	-	-
Lindane	2	0.95		-		-		-
Cadmium*	3.9	2.0	1.1	0.25	43	40	9.3	8.8
Chromium III*	17000	570	210	74		-		-
Chromium VI*	16	16	11	11	1100	1100	50	50
Copper*	18	13	12	9	2.9	4.8	2.9	3.1
Dieldrin	2.5	0.24	0.0019	0.056		-		-
Endosulfan (alpha)	N/A	0.22	N/A	0.056	N/A	0.034	N/A	0.0087
Endosulfan (beta)	N/A	0.22	N/A	0.0056	N/A	0.034	N/A	0.0087
Endrin	0.18	0.086	0.0023	0.036		-		-
Heptachlor epoxide	N/A	0.52	N/A	0.0038	N/A	0.053	N/A	0.0036
Lead*	82	65	3.2	2.5	140	210	5.6	8.1
Nickel*		470		52		74		8.2
Pentachlorophenol (@pH 7.8)	20	19	13	15	13	13	N/A	7.9
Selenium	260	12.82 (selenate) 185.9 (selenite)	35	5.0	410	290	54	71
Silver*	4.1	3.2	0.12	0.10	2.3	1.9		-
Tributyltin	N/A	0.46	N/A	0.063	N/A	0.37	N/A	0.01
Zinc*	120	120	110	120	95	90	86	81

* Hardness of 100 mg/L

Table 2. Summary of the ESA consultation results for Oregon's 2004 submittal of aquatic life criteria (LAA = likely to adversely affect; NLAA = not likely to adversely affect; USFWS, 2012; NMFS, 2012). Some criteria have been updated since Oregon last submitted aquatic life criteria updates (i.e., aluminum, cadmium, copper, selenium, ammonia).

Chemical	Freshwater Acute	Freshwater Chronic	Saltwater Acute	Saltwater Chronic
Aluminum	LAA*	LAA*	N/A	N/A
Arsenic	NLAA	LAA	N/A	N/A
Cadmium	LAA*	LAA	NLAA	NLAA
Chromium III	LAA	LAA	N/A	N/A
Chromium VI	LAA	LAA	NLAA	NLAA
Copper	LAA*	LAA*	NLAA	NLAA
Lead	LAA	LAA	NLAA	NLAA
Nickel	LAA	LAA	NLAA	NLAA
Selenium	LAA	LAA	NLAA	LAA
Silver	LAA	N/A	NLAA	N/A
Zinc	LAA	LAA	NLAA	NLAA
Ammonia	LAA	LAA	N/A	N/A
Dieldrin	NLAA	NLAA	N/A	N/A
Endosulfan (alpha)	NLAA	NLAA	NLAA	NLAA
Endosulfan (beta)	NLAA	NLAA	NLAA	NLAA
Endrin	NLAA	NLAA	N/A	N/A
Heptachlor Epoxide	NLAA	NLAA	NLAA	NLAA
Lindane (gamma-BHC)	NLAA	NLAA	N/A	N/A
Pentachlorophenol	LAA	LAA	NLAA	NLAA
Tributyltin	NLAA	NLAA	NLAA	NLAA

* Criterion also received subsequent Jeopardy call by USFWS or NMFS

Idaho

Idaho submitted revised aquatic life toxics criteria on April 11, 2006. These criteria were approved by EPA in 2007, subject to ESA consultation. The BiOp from NMFS and USFWS were completed in 2014 and 2015, respectively. Tables 3 and 4 provide the revised aquatic life toxics criteria submitted by Idaho and the results of ESA consultation, indicating which criteria received a likely to adversely affect endangered species determination or jeopardy calls (NMFS, 2014; USFWS, 2015). Idaho's endangered species list is different from Washington, but the two states do share common endangered species such as the bull trout. Thus, we only used ESA consultation information for similarly listed species in Washington.

Table 3. Ambient water quality criteria for toxic pollutants submitted for consultation in EPA's 1999 Assessment and revisions by the State of Idaho (NMFS, 2014; USFWS, 2015).

Substance	Freshwater Acute Criteria (µg/L)		Freshwater Chronic Criteria (µg/L)	
	Previous	Proposed	Previous	Proposed
Arsenic	360	340	190	150
Cadmium*	-	-	-	-
Copper	17	17	11	11
Cyanide	22	22	5.2	5.2
Lead	65	65	2.5	2.5
Mercury	2.1	2.1	0.012	0.012
Selenium	20	20	5	5
Zinc	114	120	105	120
Chromium III	550	570	180	74
Chromium VI	15	16	10	11
Nickel	1400	470	160	52
Silver	3.4	3.4	N/A	N/A
Endosulfan (alpha and beta)	0.22	2.0	0.056	89
Aldrin	3	0.00014	-	0.000050
Chlordane	2.4	0.00057	0.0043	0.00081
4,4-DDT	1.1	0.00059	0.001	0.00022
Dieldrin	2.5	0.00014	0.0019	0.000054
Endrin	0.18	0.81	0.0023	0.060
Heptachlor	0.52	0.00021	0.0038	0.000079
Lindane (gamma-BHC)	2	0.063	0.08	1.8
Polychlorinated biphenyls (PCBs)	N/A	0.000045	0.014	0.000064
Pentachlorophenol	20	6.2	13	3.0
Toxaphene	0.73	0.00075	0.0002	0.00028

*Consultation completed in 2011

Table 4. Summary of the Endangered Species Act consultation results for Idaho's aquatic life criteria (LAA = likely to adversely affect; NLAA = not likely to adversely affect; NMFS, 2014; USFWS, 2015).

Chemical	Freshwater Acute	Freshwater Chronic
Arsenic	NLAA	LAA*
Chromium III	NLAA	NLAA
Chromium VI	NLAA	LAA
Copper	LAA*	LAA*
Lead	NLAA	LAA*
Mercury	NLAA	LAA*
Nickel	LAA*	LAA*
Selenium	NLAA	LAA*
Silver	LAA	N/A
Zinc	LAA*	LAA*
Aldrin	NLAA	NLAA
Chlordane	NLAA	NLAA
Cyanide	LAA*	LAA*
4,4-DDT	NLAA	NLAA
Dieldrin	NLAA	NLAA
Endosulfan (alpha)	NLAA	NLAA
Endosulfan (beta)	NLAA	NLAA
Heptachlor	NLAA	NLAA
Lindane (ɣ-BHC)	NLAA	NLAA
Pentachlorophenol	NLAA	NLAA
Polychlorinated biphenyls	N/A	NLAA
Toxaphene	NLAA	NLAA

* Criterion also received subsequent Jeopardy call by USFWS or NMFS

Swinomish Indian Tribal Community

The Swinomish Indian Tribal Community (Swinomish Tribe) submitted aquatic life toxics criteria to EPA for review and approval under the CWA on February 8, 2017. The Swinomish Tribe revised the aquatic life toxics criteria submittal, and the Swinomish Senate adopted the revisions into their water quality standards on April 8, 2019. The revised water quality standards were submitted to EPA on April 30, 2019. EPA's biological evaluation of the Swinomish Tribe aquatic life toxics criteria was completed on June 22, 2022 (USEPA, 2022a). EPA has subsequently submitted the biological evaluation of the Swinomish Tribe's updates to USFWS and NMFS for ESA consultation. Table 5 summarizes EPA's BE.

EPA did not evaluate some of the Swinomish Tribe aquatic life toxics criteria, including freshwater chronic arsenic, freshwater acute and chronic chloride, freshwater acute and chronic cyanide, and freshwater and saltwater acute and chronic mercury. The criteria that

were not consulted on were found by NMFS and/or USFWS to likely adversely affect salmonid species in Idaho or Oregon or were predicted to cause effects based on new science.

Table 5. Biological evaluation results for the Swinomish Tribe (LAA = likely to adversely affect; NLAA = not likely to adversely affect; USEPA, 2022a).

Chemical	Freshwater Acute	Freshwater Chronic	Saltwater Acute	Saltwater Chronic
Arsenic	NLAA	Not evaluated	NLAA	LAA
Chromium III	NLAA	NLAA	-	-
Chromium VI	NLAA	LAA	LAA	LAA
Copper	NLAA	NLAA	NLAA	NLAA
Iron	-	LAA	-	-
Lead	NLAA	NLAA	NLAA	NLAA
Mercury	Not evaluated	Not evaluated	Not evaluated	Not evaluated
Nickel	LAA	NLAA	NLAA	NLAA
Selenium	NLAA	NLAA	NLAA	LAA
Silver	NLAA	-	NLAA	-
Zinc	LAA	LAA	NLAA	NLAA
Acrolein	NLAA	NLAA	-	-
Aldrin	NLAA	-	NLAA	-
Carbaryl	NLAA	NLAA	NLAA	-
Chlordane	NLAA	NLAA	NLAA	NLAA
Chloride	Not evaluated	Not evaluated	-	-
Chlorine	NLAA	NLAA	LAA	NLAA
Chlorpyrifos	NLAA	NLAA	NLAA	NLAA
Cyanide	Not evaluated	Not evaluated	NLAA	NLAA
Demeton	-	NLAA	-	NLAA
Diazinon	NLAA	NLAA	NLAA	NLAA
Dieldrin	NLAA	NLAA	NLAA	NLAA
Endosulfan (alpha & beta)	NLAA	NLAA	NLAA	NLAA
gamma-BHC (Lindane)	NLAA	-	NLAA	-
Guthion	-	NLAA	-	NLAA
Heptachlor	NLAA	NLAA	NLAA	NLAA
Heptachlor epoxide	NLAA	NLAA	NLAA	NLAA
Hydrogen sulfide	-	LAA	-	LAA
Malathion	-	NLAA	-	NLAA
Methoxychlor	-	NLAA	-	NLAA
Mirex	-	NLAA	-	NLAA
Nonylphenol	LAA	NLAA	LAA	LAA
4,4-DDT	NLAA	NLAA	NLAA	NLAA
Parathion	NLAA	NLAA	-	-
Pentachlorophenol	NLAA	NLAA	LAA	NLAA

Chemical	Freshwater Acute	Freshwater Chronic	Saltwater Acute	Saltwater Chronic
Polychlorinated biphenyls	-	NLAA	-	NLAA
Toxaphene	NLAA	NLAA	NLAA	NLAA
Tributyltin	NLAA	NLAA	NLAA	NLAA

The Swinomish Tribe water quality submission was approved by EPA on August 4, 2023, with the exceptions noted above that EPA did not act upon (USEPA, 2023). However, formal ESA consultation was not completed by NMFS and USFWS. Rather, Section 7(d) of the Endangered Species Act and Habitat Conservation Plans was used to allow for implementation of the Swinomish Tribe water quality criteria. The USFWS specifically states the following regarding section 7(d):

> "The Services' Interagency Consultation Handbook provides limited guidance regarding the application of section 7(d) during the consultation process other than to state that the section 7(d) restriction is triggered by the determination of "may affect." The Consultation Handbook also states that "Not all irreversible and irretrievable commitments of resources are prohibited. The formulation or implementation of any reasonable and prudent alternative must be foreclosed by the resource commitment to violate section 7(d). Thus, resource commitments may occur as long as the action agency retains sufficient discretion and flexibility to modify its action to allow formulation and implementation of an appropriate reasonable and prudent alternative." Destroying potential alternative habitat within the project area, for example, could violate section 7(d)."

Because formal ESA consultation was not completed, we will continue to use EPA's 2022 BE for the Swinomish Tribe to provide ancillary support for decision-making in this rulemaking.

Litigation

Determination of Consistency with Clean Water Act

In October 2013, Northwest Environmental Advocates (NWEA) petitioned EPA to use its CWA authority to determine that Washington needed new or revised aquatic life toxics criteria and to promulgate such criteria for Washington. EPA denied this petition in 2017, and in September 2020, NWEA filed a lawsuit in federal court challenging EPA's denial. On December 29, 2021, the U.S. District Court ruled that EPA's denial of the rulemaking petition was unreasonable and ordered EPA to determine whether Washington's aquatic life criteria are consistent with the CWA or if they need to be revised (NWEA vs. EPA, 2021, Case No. C20-1362 MJP).

Following issuance of the order, EPA and NWEA negotiated a proposed modification to the order which the Court granted in August 2022. The modified order required EPA to evaluate the following nine pollutants by June 2023: arsenic, cadmium, copper, cyanide, mercury, selenium, nickel, acrolein, and aluminum, and determine whether they are consistent with CWA requirements and protect the applicable designated uses of Washington's surface waters. The modified order further directed EPA to evaluate the following additional eight pollutants by June 2026: chromium III, DDT and metabolites, endosulfan, endrin, tributyltin, zinc, lead, and

nonylphenol. If any of Washington's criteria for these 17 toxics are determined to be inconsistent with CWA requirements, the CWA requires EPA to promulgate new or revised criteria for Washington that meets such requirements, unless the state adopts and submits new or revised criteria that EPA approves first.

In May 2023, EPA determined that Washington's existing criteria for arsenic, cadmium, copper, cyanide, mercury, nickel, and selenium are not protective of the applicable designated use and that Washington lacks aquatic life criteria for acrolein and aluminum where information indicates that Washington needs criteria for those pollutants to protect applicable designated uses.

Endangered Species Act Consultation on Cyanide

The Center for Biological Diversity filed a lawsuit in federal court alleging that EPA failed to ensure its approval of Washington's cyanide criteria will not jeopardize the survival and recovery of endangered and threatened species or adversely modify habitat (Center for Biological Diversity vs. EPA, Case 1:22-cv-00486-BAH, 8/08/23). The litigation is ongoing and its outcome uncertain. However, if the court reaches the merits of the case or the parties settle, EPA may be required to consult on Washington's existing cyanide criteria under the Endangered Species Act.

Rulemaking Strategy

We are updating our aquatic life toxics criteria to ensure consistency with CWA recommendations, protect endangered species, and avoid federal promulgation stemming from litigation. In this rulemaking, we are using information from previous ESA consultations in Oregon and Idaho to determine whether to adopt EPA CWA recommendations or adopt state-specific criteria that will be protective of Washington's listed endangered species. The biological opinions from Oregon and Idaho provided information on protection levels needed for full protection for similarly listed endangered species in Washington. In addition, we used a recently completed EPA biological evaluation for aquatic life toxics criteria for the Swinomish Tribe to inform endangered species protection levels. The methods section below describes the decision-making process for developing criteria and the specific approach for protecting endangered species and their populations.

Endangered and Threatened Species in Washington

The following aquatic species are federally listed endangered and threatened in Washington:

- Chinook salmon and critical habitat
- Sockeye salmon
- Coho salmon
- Steelhead
- Chum salmon
- Bocaccio and critical habitat
- Yelloweye rockfish

- Humpback whale
- Southern resident killer whale and critical habitat
- Bull trout and critical habitat
- Marbled murrelet
- Green sturgeon
- Eulachon smelt

METHODS

Standard EPA Derivation Methods

EPA is tasked with developing aquatic life toxics criteria that protect aquatic life from the harmful effects of toxic chemicals. EPA uses derivation methods that can be broken down into four steps:

1. Calculate species mean acute/chronic values,
2. Calculate genus mean acute/chronic values,
3. Rank the genus mean acute/chronic values, and
4. Determine the 5th percentile of the genus sensitivity distribution (GSD) and divide by a factor of two to yield protective acute criteria, while chronic criteria are based directly on the 5th percentile of the GSD.

A more detailed procedure can be found in EPA 1985 guidance on developing aquatic life toxics criteria (Stephan et al. 1985). These EPA standard derivation methods aim to protect 95% of aquatic genera 99% of the time. In the 1985 EPA guidance document, EPA states that because aquatic ecosystems can tolerate some stress and occasional adverse effects, protection of all species at all times and places is not deemed necessary. If data are available for a large and diverse number of taxa, a reasonable level of protection will be provided if all except a small fraction of taxa are protected.

One notable issue with EPA methods is when endangered species and their populations are especially sensitive and fall outside national protection levels or new toxicity data has been generated and not yet incorporated into EPA national criteria. In other instances, studies with endangered species have examined toxicity using surrogates or endpoints that are not considered using standard EPA derivation methods (such as indirect effects on prey items of endangered species) and are the cause of jeopardy calls during ESA consultation.

During ESA consultation, EPA's BE considers all toxicity data and indirect effects of toxic chemicals to endangered species at the individual level. EPA's BEs consider direct effects to growth, survival, and reproduction, but can also consider endpoints other than growth, survival, or reproduction (non-apical endpoints) that can be quantitatively linked to population-level effects. A BE can also assess impacts to the prey of a listed species to determine potential affects to listed species. The BE can consider tissue data, bioaccumulation potential, and ambient water concentrations to predict toxicity to prey. NMFS and USFWS consider if and how effects documented in EPA's BE results in population-level effects to inform Jeopardy and Non-Jeopardy calls. The difference in approach between EPA methods for developing aquatic life toxics criteria and ESA consultation methods has led to several issues in adopting EPA 304(a) recommendations in Pacific Northwest states.

Alternative Aquatic Life Toxics Derivation Method

If Washington adopts EPA 304(a) recommendations for aquatic life toxics criteria that through the ESA Consultation process are not shown to be protective of endangered and threatened species and their populations, we anticipate that we will not receive federal approval as

demonstrated in other Pacific Northwest states with similarly listed species (such as Oregon and Idaho). EPA's nationally recommended aquatic life criteria for some toxics have been determined in previous federal BiOps by NMFS and USFWS to jeopardize or adversely affect certain ESA-listed species that exist in Washington (NMFS, 2012; NMFS, 2014; USFWS 2012; USFWS, 2015).

We evaluated alternative methods to develop criteria, in addition to using new scientific data since the last EPA updates, to calculate more stringent criteria than EPA's national recommendations for some criteria to ensure that the criteria would be protective of endangered species and their populations. The alternative method (i.e., 1st percentile derivation procedure) described is used to address extinction susceptibility of Washington's endangered species populations and are not a result of a particular species chemical sensitivity. However, the outcome of using this method is improved protection for all aquatic species.

We decided to set state-specific criteria for certain pollutants where Oregon and Idaho BiOps concluded that EPA recommendations for those pollutants would likely adversely affect or jeopardize ESA-listed species and their populations that also exist in Washington. The first step in developing state-specific criteria for select pollutants in the proposed rule was to evaluate the new science since EPA last updated the national criteria to determine if incorporating new science into the criteria derivation would adequately protect endangered species in Washington. When developing state-specific criteria using new science only, we used standard EPA methods (Stephan et al. 1985) to incorporate new science and calculate the new criteria. The newly calculated criteria based on new science alone was compared to the information in the Idaho and Oregon BiOps for similarly listed endangered species in Washington to determine if new science alone provided adequate protection.

When new science did not provide adequate protection for endangered species, we applied a more conservative derivation process than EPA methods recommend in their 1985 guidance document for criteria development. We used the 1st percentile of the toxicity data distribution to derive a more conservative criterion value that will protect a greater proportion of species. Deriving the 1st percentile of the toxicity data distribution results in a protection level of 99% of genera 99% of the time, which translates to greater overall protection to all aquatic species, including susceptible populations of endangered species. The general procedure for evaluating pollutants in this rule was as follows:

1. Match EPA recommendations if there were no LAA determinations or jeopardy calls for similarly listed species in Idaho and Oregon.
2. If there were LAA determinations or jeopardy calls in Idaho and Oregon for similarly listed species in Washington, then evaluate the new science since EPA last updated national recommendations.
3. If new science met protection levels described in the Idaho and Oregon BiOps, then use the new science to derive the criteria.
4. If criteria based on new science did not provide adequate protection, then derive the 1st percentile of the toxicity data distribution.

We reviewed EPA national recommendations for aquatic life toxics and identified several of Washington's aquatic life toxics criteria that need to be updated. Table 6 shown below

compares the year numeric aquatic life toxics were last updated by Washington and when EPA last updated their CWA recommendations. Table 7 below lists criteria that are not included in Washington's water quality standards for aquatic life toxics but are recommended by EPA. Updates to Washington's aquatic life toxics criteria were placed in six different categories:

1. We are proposing taking no action ("No change"). No action means that Washington aquatic life criteria are identical to EPA CWA recommendations and there are no ESA consultation jeopardy calls.
2. We are proposing adopting EPA CWA recommendations ("EPA recommendation").
3. We are proposing not adopting criteria with EPA CWA recommendations into Washington's standards ("Do not adopt").
4. We are proposing new criterion specific to Washington with no EPA CWA recommendations ("New state-specific criteria") or we are proposing criteria with EPA recommendations but have used a state-specific approach ("State-specific criteria").
5. We are proposing updated criteria for select toxics with ESA jeopardy calls or likely to adversely affect determinations that incorporate new science since EPA last updated the criteria ("New science").
6. We are proposing updated criteria for select toxics with ESA jeopardy calls that incorporate new science since EPA last updated the criteria and uses the 1st percentile of the toxicity data distribution to derive the protective value ("New science and 1st percentile"). In instances where likely to adversely affect determinations were made for a pollutant and the new science was incorporated into the new criteria but resulted in a greater criterion, the 1st percentile was applied to increase protection levels.

These different strategies are outlined for each toxic chemical in the Strategy for Aquatic Life Toxics section below.

Table 6. Washington's current list and adoption year of aquatic life toxics criteria compared with EPA's last update.

Toxic Substance	Year WA Last Updated	Year EPA Last Updated
4,4'-DDT (and metabolites)	1988*	1980
Aldrin	1988*	1980
Ammonia	2003	2013
Arsenic	1992	1995
Cadmium	1997	2016
Chlordane	1988*	1980
Chloride (dissolved)	1992	1988
Chlorine (total)	1988	1986
Chlorpyrifos	1988*	1986
Chromium III	1992	1995
Chromium VI	1992	1995
Copper	1997	2007
Cyanide	2003*	1985
Dieldrin	1988*	1995

Toxic Substance	Year WA Last Updated	Year EPA Last Updated
Endosulfan	1988*	1980
Endrin	1988*	1995
Heptachlor	1988*	1980
Hexachlorocyclohexane (gamma-BHC; Lindane)	1988*	1995
Lead	1992	1984
Mercury	1997	1995
Nickel	1997	1995
Parathion	1988*	1995
Pentachlorophenol (PCP)	1992	1995
Polychlorinated Biphenyls (PCBs)	1988*	1986
Selenium	1997	2016
Silver	1992	1980
Toxaphene	1988*	1986
Zinc	1992	1995

*Record of identical criteria in 1988 standards but not in 1981. Criteria may have been incorporated between 1982 and 1988.

Table 7. Toxic substances listed in EPA national recommended 304(a) criteria and year last updated for which Washington has no numeric criteria.

Toxic Substance	Year EPA Last Updated
Acrolein	2009
Aluminum	2018
Boron	1986
Carbaryl	2012
Demeton	1985
Diazinon	2005
Guthion	1986
Heptachlor Epoxide	1981
Iron	1986
Malathion	1986
Methoxychlor	1986
Mirex	1986
Nonylphenol	2005
Perfluorooctanoic Acid (PFOA)	2022 (draft)
Perfluorooctane Sulfonate (PFOS)	2022 (draft)
Sulfide-hydrogen sulfide	1986
Tributyltin	2004

Evaluating Scientific Articles for Criteria Derivation

Databases

We evaluated new science in calculating state-specific criteria. We used the EPA ECOTOX database[3] to obtain new scientific articles for incorporation into criteria development. We restricted the ECOTOX database to look at new science from the year before EPA published their last update for a toxic to present day. We searched for articles from the year before EPA last updated criteria because of delays in publishing and time taken to complete updates. During this process we discovered that the ECOTOX database is not updated to present day for most toxics. We therefore requested information from the ECOTOX database coordinator on when the ECOTOX database was last updated for the toxics with state-specific criteria (see Table 8).

We used this information to evaluate the open literature, primarily using Google Scholar, for additional scientific articles from the time ECOTOX was last updated to March 2023. Search terms for individual toxics in the open literature included "<insert chemical name> LC50", "<insert chemical name> EC50", "<insert chemical name> NOEC", "<insert chemical name> LOEC", and "<insert chemical name> EC20."

Table 8. ECOTOX database latest updates for chemicals selected for state-specific criteria.

Chemical	Most Recent Literature Search
Arsenic	January 2020
Cadmium	January 2013
Chromium VI	February 2013
Lead	July 2010
Nickel	June 2013
Silver	October 2008
Zinc	November 2014
Chlorine	June 2012
Cyanide	November 2013
Nonylphenol	February 2016
Pentachlorophenol	February 2016

Study Acceptability

After obtaining a list of potential articles that could be used to update select aquatic life toxics criteria, each one had to be individually evaluated for data quality and assurance. EPA does not have clear guidelines for the inclusion of scientific articles into criteria derivation but does have some general guidance that can be used from their 1985 guidelines. We used the 1985 EPA guidance in addition to standard method test acceptability requirements. Below are the criteria used to evaluate scientific studies for the inclusion into criteria development. Articles that did

[3] https://cfpub.epa.gov/ecotox/

not meet these requirements were disqualified and removed from consideration. The test acceptability and data requirements were as follows:

- Study must include control treatment(s)
- Control survival should meet standard methods (generally greater than 90%)
- Water quality of dilution water and/or test conditions must be reported
- If chemical toxicity is based on water quality (e.g., hardness), then that parameter must be reported
- Appropriate dilution water was used for test species
- Study should use replicates of test concentrations (at least two)
- Technical grade chemicals were used and reported
- Formulated mixtures and emulsifiable concentrations cannot be used
- For volatile, hydrolysable, and degradable chemicals, only flow through tests are acceptable unless initial test concentrations were used to calculate threshold values
- Feeding should not occur during acute studies (few exceptions)
- Studies should not use brine shrimp as test species
- Test species must be a non-invasive North American species (**invasive species with established populations were not considered in this rule because they do not represent native fauna of Washington, there is a significant amount of time and resources used to eradicate these species, and they are generally less sensitive than native species thereby precluding their use as a surrogate**)
- Test organisms must not be previously exposed to a test chemical
- Do not use a study if total organic carbon or particulate matter exceeded 5 milligrams per liter (mg/L) in dilution waters
- Test with cladocerans should use organisms less than 24 hours old
- Tests with single celled organisms should not be used
- Acute values reported as “greater than” should not be used when they represent one of the four lowest genus mean acute values
- Toxicity values should not be averaged for same species if studies used different life stages with the most sensitive species used for criteria calculations
- Toxicity values from species were rejected when other species within a genus were approximately 10X more sensitive (i.e., 10-fold difference in toxicity values resulted in rejection of the less sensitive species)
- Chronic studies must use a flow-through test design and measured chemical concentrations using analytical methods (exception for cladocerans)
- Acute studies can use static, static-renewal, or flow through test designs, and measuring chemical concentrations is optional
- Hierarchy of studies were given for test design: flow through > static renewal > static (if multiple studies existed for same species, studies were rejected if the more representative test design was used)
- Hierarchy of studies were given for studies measuring chemical concentrations versus unmeasured concentrations

Appendix A of this document includes the studies considered in this rulemaking and reasons for removing studies from consideration for criteria derivation. References for studies that were obtained from Google Scholar are reported in the reference section.

Metal Reporting

For metals where new scientific information was used, we reported all metal concentrations as total recoverable as per EPA guidelines for consistency in calculating the criterion maximum concentration (CMC) or acute criterion and criteria continuous concentration (CCC) or chronic criterion. When a toxicity value such as median lethal concentration (LC50), which describes the amount of a toxic chemical that kills 50% of organisms, was reported as a dissolved metal, the dissolved concentration was back-calculated to total metal concentrations using EPA's metal conversion factors (Table 9). If a study reported both dissolved and total metal concentrations, total metal concentrations were used for this analysis. The CMC and CCC based on total metal concentrations were translated to dissolved metal concentrations using EPA's conversion factors. The final criteria values were reported as dissolved metal concentrations.

Table 9. EPA acute and chronic conversion factors (CF) for metals (Kinerson et al. 1996).

Metal	Acute CF	Chronic CF
Arsenic	1.000	1.000
Cadmium*	0.944	0.909
Chromium III	0.316	0.860
Chromium VI	0.982	0.962
Copper	0.960	0.960
Lead*	0.791	0.791
Mercury	0.85	-
Nickel	0.998	0.997
Silver	0.85	-
Zinc	0.978	0.986

*Conversion factors for cadmium and lead are hardness dependent. The values shown are with a hardness of 100 mg/L as calcium carbonate ($CaCO_3$).

RESULTS

Summary Table of Proposal

Table 10 provides a summary of our proposed freshwater acute, freshwater chronic, saltwater acute, and saltwater chronic aquatic life toxics criteria. For each criterion, we have also provided a comparison to EPA national recommended criteria when applicable.

Table 10. Proposed acute and chronic aquatic life toxics criteria for freshwater (FW) and saltwater (SW) and EPA recommendations. MLR = multiple linear regression.

Chemical	FW Acute (µg/L)		FW Chronic (µg/L)		SW Acute (µg/L)		SW Chronic (µg/L)	
	WA	EPA	WA	EPA	WA	EPA	WA	EPA
Aluminum	MLR Model (West: 510[#]) (East: 820[#])	MLR Model	MLR model (West: 270[#]) (East: 480[#])	MLR Model	-	-	-	-
Arsenic	300	340	130	150	27	69	12	36
Cadmium	1.3*	1.8*	0.41*	0.72*	33	33	7.9	7.9
Chromium III	570*	570*	74*	74*	-	-	-	-
Chromium VI	18	16	4.5	11	1100	1100	50	50
Copper	MLR model (West: 2.0[#]) (East: 2.5[#])	BLM model	MLR Model (West: 1.6[#]) (East: 1.8[#])	BLM Model	4.8	4.8	3.1	3.1
Iron	-	-	-	1000	-	-	-	-
Lead	65*	65*	2.5*	2.5*	210	210	8.1	8.1
Mercury	1.4	1.4	0.012	0.77	1.8	1.8	0.025	0.94
Nickel	34*	470*	5.6*	52*	74	74	8.2	8.2
Selenium	EPA's tissue & water criteria	EPA's tissue & water criteria	EPA's tissue & water criteria	EPA's tissue & water criteria	290	290	71	71
Silver	0.52*	3.2*	0.21	-	2.2	1.9	0.87	-
Zinc	57*	120*	39*	120*	90	90	81	81

Chemical	FW Acute (µg/L)		FW Chronic (µg/L)		SW Acute (µg/L)		SW Chronic (µg/L)	
	WA	EPA	WA	EPA	WA	EPA	WA	EPA
4,4”-DDT (and metabolites)	1.1	1.1	0.001	0.001	0.13	0.13	0.001	0.001
6PPD-quinone (N-(1,3-Dimethylbutyl)-N’-phenyl-p-phenylenediamine-quinone)	0.008	-	-	-	-	-	-	-
Acrolein	3	3	3	3	-	-	-	-
Aldrin	3	3	0.0019	-	1.3	1.3	0.0019	-
Carbaryl	2.1	2.1	2.1	2.1	1.6	1.6	-	
Chlordane	2.4	2.4	0.0043	0.0043	0.09	0.09	0.004	0.004
Chloride	860000	860000	230000	23000	-	-	-	-
Chlorine	19	19	11	11	13	13	7.5	7.5
Chlorpyrifos	0.083	0.083	0.041	0.041	0.011	0.011	0.0056	0.0056
Cyanide	12	22	2.7	5.2	1	1	1	1
Demeton	-	-	0.1	0.1	-	-	0.1	0.1
Diazinon	0.17	0.17	0.17	0.17	0.82	0.82	0.82	0.82
Dieldrin	0.24	0.24	0.056	0.056	0.71	0.71	0.0019	0.0019
Endosulfan (alpha)	0.22	0.22	0.056	0.056	0.034	0.034	0.0087	0.0087
Endosulfan (beta)	0.22	0.22	0.056	0.056	0.034	0.034	0.0087	0.0087
Endrin	0.086	0.086	0.036	0.036	0.037	0.037	0.0023	0.0023
gamma-BHC	0.95	0.95	0.08	-	0.16	0.16	-	-
Guthion	-	-	0.01	0.01	-	-	0.01	0.01
Heptachlor	0.52	0.52	0.0038	0.0038	0.053	0.053	0.0036	0.0036
Heptachlor epoxide	-	0.52	-	0.0038	-	0.053	-	0.0036
Malathion	-	-	0.1	0.1	-	-	0.1	0.1
Methoxychlor	-	-	0.3	0.3	-	-	0.3	0.3
Mirex	-	-	0.001	0.001	-	-	0.001	0.001
Nonylphenol	28	28	6.6	6.6	7	7	1.7	1.7
Parathion	0.065	0.065	0.013	0.013	-	-	-	-

Chemical	FW Acute (µg/L)		FW Chronic (µg/L)		SW Acute (µg/L)		SW Chronic (µg/L)	
	WA	EPA	WA	EPA	WA	EPA	WA	EPA
Pentachlorophenol	9.4^	19^	4.7^	15^	13	13	6.7	7.9
Polychlorinated biphenyls	2	-	0.014	0.014	10	-	0.03	0.03
PFOS	EPA's water & tissue criteria	EPA's water & tissue criteria	EPA's water & tissue criteria	EPA's water & tissue criteria	550	550	-	-
PFOA	EPA's water & tissue criteria	EPA's water & tissue criteria	EPA's water & tissue criteria	EPA's water & tissue criteria	7000	7000	-	-
Sulfide-hydrogen sulfide	-	-	-	2	-	-	-	2
Toxaphene	0.73	0.73	0.002	0.002	0.21	0.21	0.002	0.002
Tributyltin	0.46	0.46	0.072	0.072	0.42	0.42	0.0074	0.0074

* Based on hardness of 100 mg/L

5th percentile default criteria from statewide dataset

^ Based on a pH of 7.8

Strategy for Aquatic Life Toxics Criteria

Table 11. Strategy for each freshwater (FW) and saltwater (SW) aquatic life toxics criterion considered in this rulemaking. Detail on each strategy can be found in the Alternative Aquatic Life Toxics Method section described above.

Chemical	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Aluminum	EPA recommendation	EPA recommendation	-	-
Arsenic	New science & 1st percentile	New science & 1st percentile	New science & 1st percentile	New science & 1st percentile
Cadmium	EPA recommendation with modification	1st percentile	EPA recommendation	EPA recommendation
Chromium III	EPA recommendation	EPA recommendation	-	-
Chromium VI	New science	New science	No change	No change

Chemical	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Copper	State-specific criteria	State-specific criteria	No change	No change
Iron	-	Do not adopt	-	-
Lead	No change	No change	EPA recommendation	EPA recommendation
Mercury	EPA recommendation	No change	No change	No change
Nickel	New science	New science	No change	No change
Selenium	EPA recommendation	EPA recommendation	No change	No change
Silver	New science	New state-specific criteria	New science	New state-specific criteria
Zinc	New science	New science	No change	No change
4,4'-DDT (and metabolites)	No change	No change	No change	No change
6PPD-quinone (N-(1,3-Dimethylbutyl)-N'-phenyl-p-phenylenediamine-quinone)	New state-specific criteria	-	-	-
Acrolein	EPA recommendation	EPA recommendation	-	-
Aldrin	EPA recommendation	No change	EPA recommendation	No change
Carbaryl	EPA recommendation	EPA recommendation	EPA recommendation	-
Chlordane	No change	No change	No change	No change
Chloride	No change	No change	-	-
Chlorine	No change	No change	No change	No change
Chlorpyrifos	No change	No change	No change	No change
Cyanide	New science & 1st percentile	New science & 1st percentile	No change	No change
Demeton	-	EPA recommendation	-	EPA recommendation
Diazinon	EPA recommendation	EPA recommendation	EPA recommendation	EPA recommendation
Dieldrin	EPA recommendation	EPA recommendation	No change	No change
Endosulfan (alpha & beta)	No change	No change	No change	No change
Endrin	EPA recommendation	EPA recommendation	No change	No change
gamma-BHC	EPA recommendation	No change	No change	-

Chemical	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Guthion	-	EPA recommendation	-	EPA recommendation
Heptachlor	No change	No change	No change	No change
Heptachlor epoxide	Do not adopt	Do not adopt	Do not adopt	Do not adopt
Malathion	-	EPA recommendation	-	EPA recommendation
Methoxychlor	-	EPA recommendation	-	EPA recommendation
Mirex	-	EPA recommendation	-	EPA recommendation
Nonylphenol	EPA recommendation	EPA recommendation	EPA recommendation	EPA recommendation
Parathion	No change	No change	-	-
Pentachlorophenol	New science	New science	New science	New science
Polychlorinated biphenyls	No change	No change	No change	No change
PFOA	EPA recommendation	EPA recommendation	EPA recommendation	-
PFOS	EPA recommendation	EPA recommendation	EPA recommendation	-
Sulfide-hydrogen sulfide	-	Do not adopt	-	Do not adopt
Toxaphene	No change	No change	No change	No change
Tributyltin	EPA recommendation	EPA recommendation	EPA recommendation	EPA recommendation

Metals

This section provides a summary of recommended criteria for metals, which we have listed in alphabetical order. The frequency of exceedance for acute criteria is a 1-hour average concentration not to be exceeded more than once every three years on average. The frequency of exceedance for chronic criteria is a 4-day average concentration not to be exceeded more than once every three years on average. Exceptions to these frequencies of exceedances are otherwise noted in table footnotes (such as selenium).

Some metal's criteria are based on hardness. EPA presents the metals that are dependent on hardness at 100 mg/L on their recommended aquatic life toxics criteria webpage[4]. We are presenting Washington's current criteria and the proposed criteria at 100 mg/L as well. However, most datasets that EPA used to calculate criteria are based on 50 mg/L. Therefore, the tables containing species mean acute values (SMAVs) and genus mean acute values (GMAVs) presented throughout this document are normalized for 50 mg/L (except for cadmium), similar to EPA documents, and converted using the hardness dependent equation to criteria based on 100 mg/L. Any criteria that are dependent on hardness or pH and were updated in this proposed rulemaking have an accompanying equation that was updated as well.

Aluminum

Summary of Criteria Recommendations and Changes

Washington does not have aluminum criteria for aquatic life (Table 12). EPA first recommended aluminum criteria in 1988 and finalized the multiple linear regression (MLR)-based criteria for aluminum in 2018 (USEPA, 2018). EPA recommendations for aluminum consists of a model-based approach for criteria based on water chemistry data (i.e., pH, dissolved organic carbon, hardness). The MLR model is presented as a regression equation that uses water body specific inputs to calculate criteria. We recommend adopting EPA recommendations for aluminum using the MLR model. We have calculated default criteria using state-specific data that can be used when site-specific water chemistry data are not available. The default freshwater acute criterion is 510 µg/L for western Washington and 820 µg/L for eastern Washington (boundaries for eastern and western are defined in the methodology below and in WAC 222-16-010). The freshwater chronic default criterion is 270 µg/L for western Washington and 480 µg/L for eastern Washington. Criteria calculated using concurrently sampled pH, hardness, and DOC for a specific water body supersede the default criteria, regardless of whether the default criteria are higher or lower.

[4] https://www.epa.gov/wqc/national-recommended-water-quality-criteria-aquatic-life-criteria-table

Table 12. Comparison of Washington's current freshwater (FW) and saltwater (SW) aluminum acute and chronic criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	Multiple Linear Regression Model	Multiple Linear Regression Model	-	-
Proposed	West: 510[#] East: 820[#] (Multiple Linear Regression Model; 1-hour)	West: 270[#] East: 480[#] (Multiple Linear Regression Model; 4-day)	-	-

[#] Represents the 5th percentile default criteria. The boundary between east and west designations is found in WAC 222-16-010.

Endangered Species Consultation

The previous 2012 Oregon and 2014/2015 Idaho Biological Opinions (BiOps) were completed prior to EPA's recommendation of the aluminum MLR model. However, more recently EPA promulgated the aluminum MLR model in Oregon (USEPA, 2022b), and both NMFS and USFWS concluded that the aluminum MLR model did not result in jeopardy to Oregon's endangered species (NMFS, 2020).

Criteria Calculations

Methodology for Default Criteria

The default criteria were calculated using concurrently sampled pH, hardness, and dissolved organic carbon data from Washington's EIM database and the Federal Water Quality (WQ) Portal. Data from EIM and the federal WQ Portal was downloaded in March 2023. We also examined concurrently sampled total organic carbon (TOC), hardness, and pH and conductivity, pH, and DOC. We calculated conversion factors to translate TOC to DOC and conductivity to hardness as detailed below.

The data qualifiers and management decisions are presented in Appendix B of this document. Data was reviewed for quality with respects to the intended use of the aquatic life toxics rulemaking. We reviewed sampling locations, the study's purpose, outlier values and units, reported QA levels, and field collection comments. Records not meeting the intended use of the aquatic life toxics rulemaking were removed (see Appendix B).

The final count of concurrent samples is 3,337 events across 646 unique locations (Figure 1). Each of the 3,337 concurrent samples were entered into the EPA Aluminum MLR calculator. We then compiled the 3,337 calculated criteria values for waterbodies throughout the state and calculated the 5th percentile of those 3,337 different criteria to be representative of the default criteria. The 5th percentile of the criteria distribution represents a conservative criteria value that is intended to protect the majority of waters with regulated discharge of aluminum. We

considered ecoregional default values (e.g., EPA level III ecoregions), but we had limited geospatial representation in some ecoregions and therefore developed default values for western and eastern Washington. Eastern and western Washington is defined by definitions in WAC 222-16-010 (Figure 2). More specifically, **"Eastern Washington"** means the geographic area in Washington east of the crest of the Cascade Mountains from the international border to the top of Mt. Adams, then east of the ridge line dividing the White Salmon River drainage from the Lewis River drainage and east of the ridge line dividing the Little White Salmon River drainage from the Wind River drainage to the Washington-Oregon state line. **"Western Washington"** means the geographic area of Washington west of the Cascade crest and the drainages defined in Eastern Washington. We had 367 unique sample locations with 2,210 samples in western Washington and 279 unique locations with 1,127 samples in eastern Washington.

A 5th percentile default criteria was used to provide protection of all aquatic species. In EPA's Biological Evaluation of Oregon's freshwater aluminum water quality criteria that was promulgated by EPA, EPA states that the 10th percentile of outputs should be protective in the majority of cases but circumstances may warrant use of a more stringent model output such as consideration of an endangered species (USEPA, 2019). EPA found that a 10th percentile default ecoregional aluminum criterion yielded <90% protection for some ecoregions and that the 5th percentile of measured numeric values in Oregon will be protective of the vast majority of cases in Oregon (USEPA, 2019).

Oregon had adequate data to develop ecoregional default values whereas Washington developed an east and west default value due to limited dispersion of concurrent sampling sites throughout the state. Thus, a higher level of protection at the 5th percentile default criteria is appropriate because individual ecoregions and watershed water chemistry is not accounted for using a default value but rather becomes integrated into the dataset. The 5th percentile default value is more protective of waters with higher bioavailability of aluminum and endangered species.

Permittees will have the opportunity to collect their own site-specific chemistry data to calculate site-specific criteria that may afford a higher criteria value than the 5th percentile default criteria. If site-specific criteria are less than the 5th percentile default criteria, permittees will need to use the site-specific information to determine effluent limits.

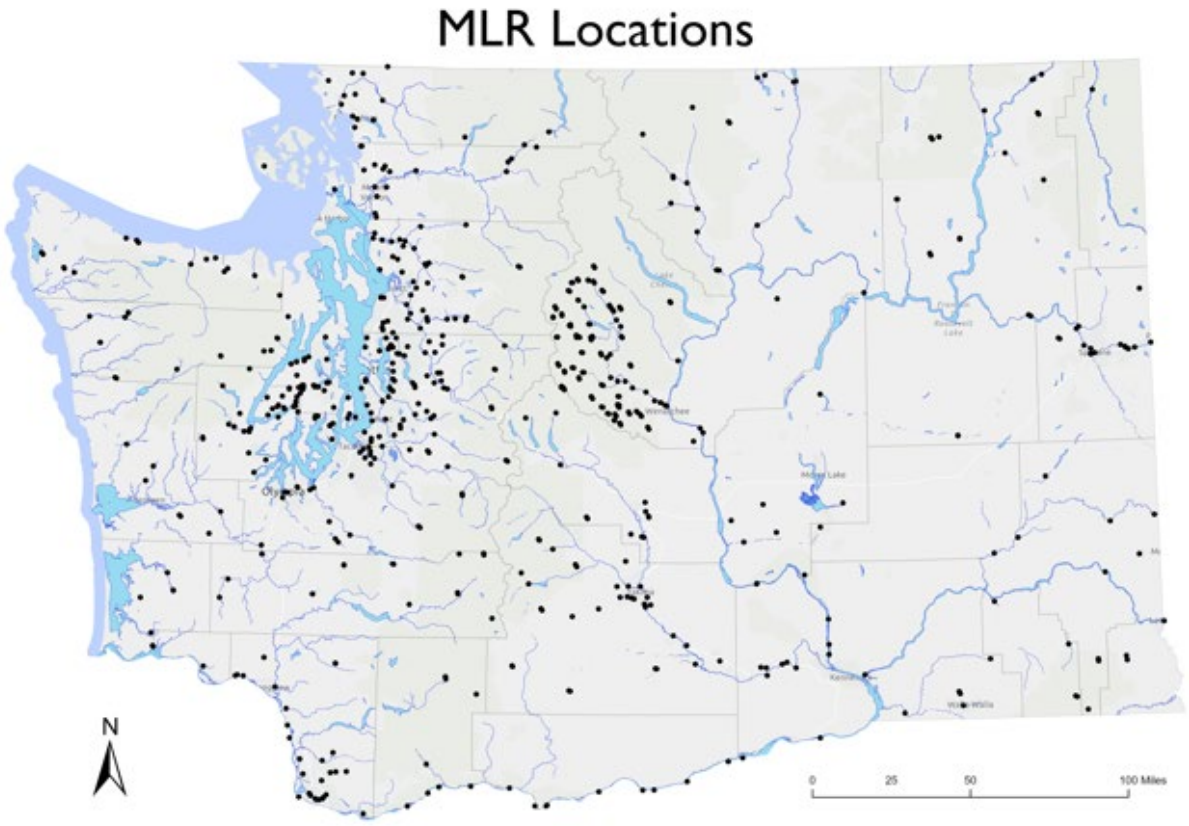

Figure 1. Locations in Washington with concurrently sampled pH, hardness, and dissolved organic carbon. Some hardness samples were calculated from conductivity and some dissolved organic carbon samples were calculated for total organic carbon.

Figure 2. Boundary defined between eastern and western Washington in WAC 222-16-010.

Conversion Factors

Total Organic Carbon to Dissolved Organic Carbon

We also examined instances where we had concurrently sampled TOC, hardness, and pH since 2000 to add additional sampling events and increase representation of waterbodies throughout the state. We developed a conversion factor to translate TOC to DOC. We downloaded all the concurrently sampled TOC and DOC data in May 2023 and calculated the ratio of DOC to TOC, or the proportion of TOC that is DOC. For the TOC conversion factor, we used the 10th percentile of all the different ratios for statewide data. We used a conservative value (i.e., 10th percentile) aimed to protect all state aquatic life (i.e., the lower the DOC value the lower the criteria value), to account for uncertainty in the conversion, and to be protective of the majority of state waters.

After converting TOC to DOC, 105 sampling events were added to our MLR dataset (105 sample events out of the 3,337 total sampling events). The statewide conversion factor, based on the 10th percentile of the ratio of DOC to TOC, is 0.81 (see example below). The TOC to DOC conversion factor is comparable to Oregon's conversion factor of 0.83 (ODEQ, 2021), EPA's reported conversion value in the copper criteria document of 0.86 (USEPA, 2007), and Massachusetts' value of 0.86 (MassDEP, 2021).

Example:
TOC = 10 mg/L
DOC = 10 mg/L (TOC) x 0.81 (conversion factor) = 8.1 mg/L

Conductivity to Hardness

We also examined instances where we had concurrently sampled conductivity, hardness, and pH since 2000 to add additional sampling events and increase representation of waterbodies throughout the state. We developed a conversion factor to translate conductivity to hardness (Figure 3). We downloaded all the concurrently sampled conductivity and hardness measurements data in August 2023. For the specific conductance versus hardness dataset, we first took the natural log of the values before running a linear regression between the two variables to improve model fit. The natural-log transformed data were used to establish the conversion equation used to estimate total hardness from conductivity. When we converted conductivity to hardness, 910 sampling events were added to our MLR dataset (910 sample events out of the 3,337 total sampling events). The linear regression equation that was used to convert conductivity to hardness is as follows:

LN(Hardness) = 1.0108*LN(conductivity) - 0.9233

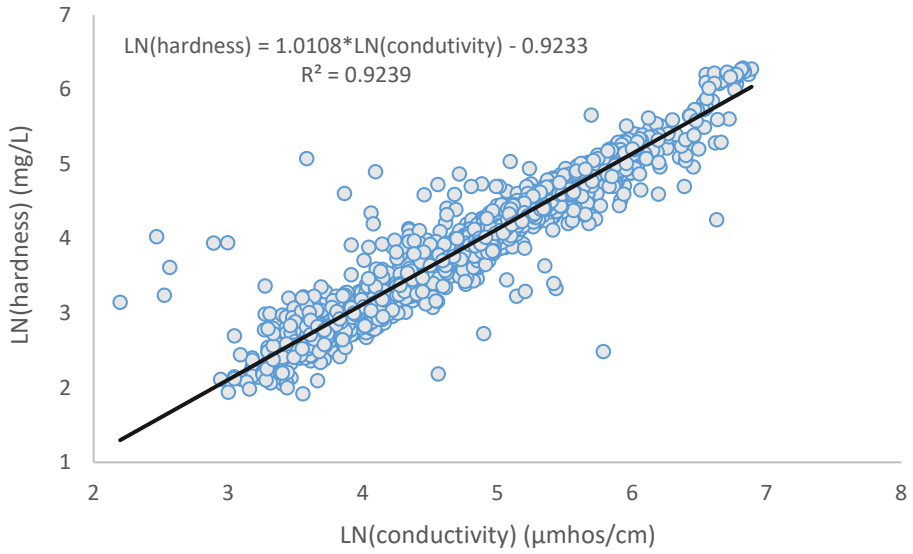

Figure 3. Relationship between hardness and conductivity (in micromhos per centimeter (µmhos/cm) for concurrent sampling throughout Washington.

Freshwater Acute and Chronic Criteria

The default freshwater acute aluminum criterion of 510 µg/L applies to western Washington and 820 µg/L is applicable to eastern Washington. The default freshwater chronic aluminum criterion is 270 µg/L for western Washington and 480 µg/L for eastern Washington are based on concurrent sampling from Ecology's EIM database and the federal WQ Portal.

If site-specific water quality information exists for a water body, that information must be used to develop site-specific aluminum criteria. A permittee is expected to work with the permit writer to determine adequate sampling data. In the absence of site-specific water chemistry data, the aluminum default criteria apply.

Arsenic

Summary of Criteria Recommendations and Changes

The proposed arsenic criteria (based on arsenic III) for freshwater and saltwater are more stringent than EPA recommendations to account for endangered species protection concerns (Table 13). New science since EPA last updated the arsenic freshwater criteria in 1995 (USEPA, 1996) and the saltwater criteria in 1984 (USEPA, 1985) was incorporated into the proposed criteria. Additionally, the 1st percentile of the toxicity data distribution was used to calculate the proposed freshwater and saltwater criteria for arsenic to ensure protection of endangered species in Washington. The EPA recommended freshwater chronic arsenic criterion was implicated in previous BiOps for causing indirect effects to freshwater endangered species (i.e., bull trout and sturgeon).

The revised arsenic criteria are aimed at improving protection for endangered species. However, BiOps and toxicity data indicate that some freshwater prey species (i.e., gammarid and mayflies) of endangered species may be negatively affected over chronic durations at 100 µg/L arsenic. We support the derived chronic criteria of 130 µg/L as protective of endangered species for the reasons described within this section and additional analyses provided in the Endangered Species Act Consultation section for Idaho. Fish species have diversity in their range of diet and are not strictly dependent on gammarid or mayfly populations for their food source. Other environmental factors, organism life history, and water quality play a role in realistic exposure scenarios that may mediate toxicity compared with controlled laboratory studies.

An important point in setting arsenic criteria is that it is based on arsenic III toxicity data which is one inorganic form of arsenic (USEPA, 1985). The EPA approved analytical method for arsenic is based on total recoverable inorganic arsenic, which includes both arsenic III and arsenic V. Arsenic III is known to be more acutely toxic than arsenic V (USEPA, 1985; Spehar et al. 1980; Suhendrayatna and Maeda, 1999; Jeyasingham and Ling, 2000; Hughes, 2002; Suhendrayatna et al. 2002; Iriving et al. 2008). The analytical method cannot distinguish between different oxidation states (USEPA, 1985). This means the criteria may be overly protective when based on the total recoverable method because we are measuring both arsenic III and arsenic V in the environment, but only arsenic III is used to derive the criteria. Therefore, any compliance monitoring for permitting purposes may be overestimating arsenic levels because of the inclusion of both inorganic species, arsenic III and arsenic V. When based on the total recoverable method, the criteria may be overly protective (USEPA, 1986). Given these factors combined, we support a freshwater chronic criterion value of 130 µg/L for arsenic because of the conservatism built into the criteria.

The proposed saltwater arsenic criteria are intended to protect endangered species and are more conservative than EPA recommendations. The Swinomish Tribe BE suggested that the EPA recommended saltwater chronic arsenic criteria may not be protective of individuals of endangered species in Washington (USEPA, 2022a). The Swinomish BE analysis was based on existing data and results compiled by EPA and may be subject to change if re-evaluated with updated datasets. The Swinomish Tribe BE back-calculated tissue residue concentrations from

the chronic criterion using a bioconcentration factor (BCF) that resulted in a tissue concentration of 1.6 mg/kg ww. They used this criteria-based value and compared it to bioaccumulation studies that reported no observed effect residues of 0.07 to 0.20 mg/kg. The newly proposed saltwater chronic criterion of 12 µg/L translates to a tissue residue of 0.53 mg/kg.

While we contend that translating water concentration thresholds to tissue residue is a useful exercise, there is a very high degree of uncertainty. Back-calculating tissue residue concentrations from a water quality criterion has high uncertainty because BCFs are site and species specific, and the chronic based criterion is based on several different species with different physiologies. The BCF used for back-calculation was not specific to the endangered species listed in Washington and may need updated using more relevant aquatic species compared with the BCF used in the Swinomish BE analysis. Furthermore, the toxicity studies used threshold tissue concentrations representative of no observed effect residues (NOERs). Typically, threshold values are calculated by taking the mean value of NOERs and the lowest observed effect residue (LOER). By using the NOER, the threshold value is being overestimated because no observed effects may occur at higher residue levels. Most often the NOERs are a product of the toxicity test design and not true threshold values. Given all these factors combined, we support a saltwater chronic criterion value of 12 µg/L for arsenic as protective of endangered species in Washington.

Table 13. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic arsenic criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	360^ (1-hour)	190^ (4-day)	69^ (1-hour)	36^ (4-day)
EPA	340^ (1-hour)	150^ (4-day)	69^ (1-hour)	36^ (4-day)
Proposed	300^ (1-hour)	130^ (4-day)	27^ (1-hour)	12^ (4-day)

^ Presented as the dissolved fraction

Endangered Species Act Consultation

Idaho

A jeopardy call was listed for arsenic freshwater chronic criterion of 150 µg/L in Idaho BiOps (NMFS, 2014; USFWS, 2015). The Idaho USFWS BiOp implicates indirect effects of arsenic on sturgeon, bull trout, and other salmonids through the bioaccumulation of arsenic from invertebrate prey species. Washington has bull trout and green sturgeon listed on their endangered and threatened species list. Thus, the effects described in the Idaho BiOp are relevant to Washington. The Idaho USFWS BiOp specifically states:

> "Bioaccumulation of arsenic in invertebrate organisms (that serve as prey for salmonids like the bull trout) to concentrations harmful to salmonids is likely to occur in streams with dissolved arsenic concentrations below the proposed chronic criterion; inorganic arsenic in the diet of rainbow trout is associated with reduced growth, organ damage and other adverse physiological effects (Cockell et al. 1991, p. 518; Hansen et al. 2004, pp. 1902-1910; Erickson et al. 2010, pp. 122,123). For those reasons, we expect that arsenic concentrations below the proposed chronic criteria are likely to contaminant the prey base within bull trout critical habitat to an extent that precludes it from being adequate to support normal growth and reproduction in the bull trout. For that reason, the proposed chronic criterion for arsenic is likely to significantly impair the capability of bull trout critical habitat to provide an abundant food base (PCE 3) for the bull trout over a significant portion of the range of designated critical habitat."
>
> "We also assume that sturgeon sensitivity to arsenic is at least as sensitive as for the rainbow trout. With rainbow trout, dietary arsenic has been linked to reduced growth at about 20 mg/kg dw and higher (see *Dietary Toxicity*, section 2.5.2.2 above), and these concentrations in benthic invertebrates have been measured in field conditions with water concentrations much lower than the proposed 150 µg/L chronic criterion for arsenic (Table 5). The observed effects of arsenic contamination in salmonids include altered feeding behavior, and reduced body weight, reproductive success, and survival. Absent information specific to the effects of the proposed arsenic criteria on white sturgeon prey species, we are assuming that information on the effects of the proposed arsenic criteria on bull trout prey species also applies to white sturgeon prey species."

These claims are further substantiated in the Idaho BiOp from Irving et al. (2008) and Canivet et al. (2001) that found arsenic III thresholds for growth effects at 100 µg/L and mortality of gammarid amphipods and mayflies at 100 µg/L. They conclude that because invertebrates accumulate arsenic from sediments and biofilms, arsenic accumulations in aquatic invertebrates have been implicated in reduced growth and tissue damage in salmonids and are likely to cause adverse effects to bull trout. However, Maeda et al. (1990) concluded that methylated arsenic in organisms increase in higher trophic levels, while total arsenic bioaccumulation decreases with an order of magnitude with each trophic level. The work by Maeda et al. (1990) suggests that threshold effects using inorganic or total arsenic should not be evaluated in terms of arsenic accumulation to higher trophic levels as was done in the USFWS BiOp for the chronic arsenic criterion. The threshold effects cited in the USFWS for gammarids and mayflies at 100 ug/L should not be extrapolated to higher trophic organisms (i.e., salmonids) that prey on these invertebrates.

Idaho's USFWS jeopardy call for the freshwater arsenic chronic criterion of 150 µg/L uses studies from Cockell et al. (1991), Hansen et al. (2004), and Erickson et al. (2010) as a basis for their determination. These articles have several uncertainties and should be reconsidered in the assessment of endangered species protection compared with surface water quality criteria. Cockell et al. (1991) directly spiked fish diets to determine effect levels. The translation between spiked diet and water column concentrations are unknown for this study, rendering it difficult to conclude whether a diet-based study is relevant to evaluating surface water quality standards based on water column concentrations. Furthermore, the Hansen et al. (2004) study

used field-collected sediments that contained several different metals, rendering it difficult to discern between effects related to arsenic versus other metals. Finally, Erickson et al. (2010) exposed earthworms to very high arsenic concentrations that would rarely be found in the environment and it is unclear if the effects would be evident at concentrations similar to the freshwater chronic arsenic criteria of 130 ug/L (an order of magnitude lower than test concentrations).

Swinomish Tribe Biological Evaluation

The Swinomish BE represents EPA's evaluation of proposed actions and does not represent NMFS/USFWS positions or conclusions of formal ESA consultation (USEPA, 2022a). However, the results of EPA's BE can be used to inform potential adverse effects that would be recognized in formal ESA consultation. In the Swinomish BE, the arsenic marine chronic criterion resulted in a likely to adversely affect (LAA) determination. The Swinomish BE specifically states:

> "The marine chronic arsenic criterion of 36 µg/L multiplied by the bioconcentration factor from the criteria document of 44 L/kg yields a tissue screening concentration (TSC) of 1.6 mg/kg wb/ww. Two NOERs were found and compared to the TSC. The first is 0.14 mg/kg from a brook trout exposure that assessed physiological effects (Harper, Farag, Hogstrand, & MacConnell, 2009); the second study EPA reviewed provides a range of 0.07 to 0.20 mg/kg based on mortality in lake trout swim up fry (Fitzsimons, Huestis, & Williston, 1995). The available residue-effects data indicates exposure to arsenic at chronic criteria levels appears likely to result in bioaccumulation of arsenic to levels associated with toxicity to aquatic species."

The BCF of 44 L/kg used in the Swinomish BE was developed using existing data and results compiled by EPA and may be subject to change if re-evaluated with updated datasets. We do not wish to update the Swinomish BE but other datasets suggest that a BCF of 44 may be an overestimate and that aquatic life based BCFs presented in USEPA (1985) arsenic criteria document may be more appropriate for comparative purposes. The results of using a lower BCF value in this assessment will likely yield a lower magnitude of effects to endangered species.

Criteria Calculations

Freshwater Acute Arsenic Criterion

The data used to derive the freshwater acute arsenic criterion is presented in Table 14. New studies that met data acceptability requirements are presented in Table 15. Studies used in previous EPA derivations but not used in this derivation are found in Table 16. The proposed freshwater acute criterion for arsenic was derived using 17 GMAVs and the 1st percentile of the toxicity data distribution. Calculation results are as follows:

Final acute value (FAV) = 596.2

CMC = 298.1

Acute criterion (total) = 300 µg/L (rounded to two significant digits)

Conversion factor (total to dissolved fraction) = 1.00

Acute criterion (dissolved) = 300 x 1.00 = 300 µg/L (rounded to two significant digits)

Table 14. Freshwater acute toxicity data used for criteria derivation.

Rank	GMAV (µg/L)	Species	SMAV (µg/L)
1	874	Gammarus pseudolimnaeus	874
2	1175	Simocephalus vetulus	1700
		Simocephalus serrulatus	812
3	1600	Hyalella azteca	1600
4	1634	Ceriodaphnia reticulata Ceriodaphnia dubia	1511 1768
5	2533	Daphnia magna Daphnia pulex	3841 1670
6	7100	Chironomus dilutus	7100
7	13700	Thymallus arcticus	13700
8	14065	Pimephales promelas	14065
9	14960	Salvelinus fontinalis	14960
10	18100	Ictalurus punctatus	18100
11	18513	Oncorhynchus mykiss Oncorhynchus kisutch Oncorhynchus tshawytscha	16026 18500 21400
12	20130	Jordanella floridae	20130
13	22040	Plecoptera	22040
14	24500	Aplexa hypnorum	24500
15	28100	Danio rerio	28100
16	41760	Lepomis macrochirus	41760
17	97000	Tanytarus dissimilis	97000

Table 15. New freshwater acute studies that met data acceptability requirements since EPA last updated arsenic criteria (S = static, FT = flow-through, U = unmeasured test concentrations, M = measured test concentrations).

Species	Method	LC50 (µg/L)	Used in Derivation?	Reference
Oncorhynchus mykiss	S, U	16000	Yes.	Buhl 1991
Oncorhynchus mykiss	S, M	15300	Yes.	Tisler & Zagorc-Koncan 2002
Oncorhynchus tshawytscha	S, U	21400	Yes.	Hamilton & Buhl 1990
Oncorhynchus kisutch	S, U	18500	Yes.	Buhl 1991
Chironomus dilutis	S, M	7100	Yes.	Liber et al. 2011
Thymallus arcticus	S, U	13700	Yes.	Buhl 1991
Daphnia pulex	S, M	2566	Yes.	Shaw et al. 2007
Ceriodaphnia dubia	S, U	1768	Yes.	Hocket & Mount 1996
Daphnia magna	S, U	2500	Yes.	Tisler & Zagorc-Koncan 2002
Danio rerio	S, M	28100	Yes.	Tisler & Zagorc-Koncan 2002
Hyalella azteca	S, M	1600	Yes.	Liber et al. 2011
Oncorhynchus mykiss	FT, M	20200	Yes.	Rankin & Dixon 1994

Table 16. Freshwater acute studies not used from previous EPA criteria derivations (FT = flow-through, M = measured test concentrations).

Species	Method	LC50 (µg/L)	Reason	Reference
Carassius auratus	FT, M	26040	Non-north American species	USEPA, 1985

Freshwater Chronic Arsenic Criterion

There was inadequate freshwater chronic arsenic data to calculate criteria using the eight-family method. The FACR (final acute to chronic ratio) of 4.594 was used to calculate the freshwater chronic arsenic criterion. This ACR is the same as the EPA derived ACR from the 1995 updates to aquatic life (USEPA, 1996). Calculation results are as follows:

FAV = 596.2

FACR = 4.594

CCC = 129.9 µg/L

Chronic criterion (total) = 130 µg/L (rounded to two significant digits)

Conversion factor (total to dissolved fraction) = 1.00

Chronic criterion (dissolved) = 130 x 1.00 = 130 µg/L (rounded to two significant digits)

Saltwater Acute Arsenic Criterion

The data used to derive the saltwater arsenic criteria is presented in Table 17. New studies that met data acceptability requirements since EPA's last update in 1984 are found in Table 18. The proposed saltwater acute criterion for arsenic was derived using 12 GMAVs and the 1st percentile of the toxicity data distribution. Calculation results are as follows:

FAV = 54.3

CMC = 27.2 µg/L

Acute criterion (total) = 27 µg/L (rounded to two significant digits)

Conversion factor (total to dissolved fraction) = 1.00

Acute criterion (dissolved) = 27 x 1.00 = 27 µg/L (rounded to two significant digits)

Table 17. Saltwater acute toxicity data used for criteria derivation.

Rank	GMAV (µg/L)	Species	SMAV (µg/L)
1	232	Cancer magister	232
2	508	Acartia clausi	508
3	1564	Crassostrea gigas Crassostrea virginica	326 7500
4	1740	Mysidopsis bahia	1740
5	>3000	Mytilus edulis	>3000

6	3490	Argopecten irradians	3490
7	8227	Ampelisca abdita	8227
8	10120	Neanthes arenaceodentata	10120
9	12700	Cyprinodon variegatus	12700
10	14950	Apeltes quadracus	14950
11	16030	Menidia menidia	16033
12	16737	Fundulus heteroclitus	16737

Table 18. New saltwater acute studies that met data acceptability requirements since EPA last updated arsenic criteria (R = static renewal, U = unmeasured test concentrations).

Species	Method	LC50 (µg/L)	Used in Derivation?	Reference
Fundulus heteroclitus	R, U	16737	Yes.	Shaw et al. 2007

Saltwater Chronic Arsenic Criterion

There was inadequate saltwater chronic arsenic data to calculate criteria using the eight-family method. The ACR of 4.594 was used to calculate the saltwater chronic arsenic criterion. This ACR is the same as the EPA derived ACR from the 1995 updates to aquatic life (USEPA, 1996). Calculation results are as follows:

FAV = 54.3

FACR = 4.594

CCC = 11.8 µg/L

Chronic criterion (total) = 12 µg/L (rounded to two significant digits)

Conversion factor (total to dissolved fraction) = 1.00

Chronic criterion (dissolved) = 12 x 1.00 = 12 µg/L (rounded to two significant digits)

Cadmium

Summary of Criteria Recommendations and Changes

The proposed freshwater acute and chronic cadmium criteria are more stringent than EPA recommendations (Table 19). The freshwater cadmium criteria are intended to provide additional protection to endangered species (specifically bull trout). Saltwater cadmium criteria match EPA recommendations, and there are no known endangered species concerns. Recent litigation has vacated EPA's freshwater chronic cadmium criteria and remanded the freshwater acute cadmium criteria (Center for Biological Diversity v. United States Environmental Protection Administration et al, No. 4:2022cv00138 - Document 39 (D. Ariz. 2023).

Table 19. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic cadmium criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	3.7*^ (1-hour)	1.0*^ (4-day)	42^ (1-hour)	9.3^ (4-day)
EPA	1.8*^ (1-hour)	0.72*^ (**vacated**) (4-day)	33^ (1-hour)	7.9^ (4-day)
Proposed	1.3*^ (1-hour)	0.41*^ (4-day)	33^ (1-hour)	7.9^ (4-day)

* Hardness based criteria (numeric value shown based on 100 mg/L)

^ Presented as the dissolved fraction

Endangered Species Act Consultation

Oregon

A jeopardy call was listed for EPA's 2001 cadmium freshwater acute (2.0 µg/L) in the Oregon BiOps, while likely to cause adverse effects were reported for the chronic criteria of 0.25 ug/L (Table 2). The Oregon BiOps (NMFS, 2012; USFWS, 2012) specifically state:

> "The LC10 developed using direct data for bull trout exposure to cadmium is 1.24 µg**/**L (at 100 mg/L CaCO3) for juvenile fish (Table 4-8). This result means that the proposed acute standard for cadmium would likely cause a reduction in bull trout survival of more than 10% of the exposed population every 3 years during the 25-year term of the proposed action."
>
> "Hansen et al. (2002, p. 171) concluded that bull trout exposed to cadmium at concentrations equivalent to 0.21 µg/L (at 100 mg/L CaCO3) experienced a 12.4% reduction in growth (weight) from the control after 55 days of exposure, while bull trout exposed to a much higher concentration of cadmium [equivalent to 0.9 µg/L (at 100 mg/L CaCO3)] experienced a 12.9% reduction in growth from the control. These results are somewhat ambiguous, as testing done at a concentration between these amounts [at 0.46. µg/L (at 100 mg/L CaCO3)] showed only a 9% reduction in weight. We conclude that a reduction in bull trout growth of about 13% (a reasonable worst case) is likely to occur every 3 years during the 25-year term of the proposed action when bull trout are subject to chronic exposure to cadmium at the proposed standard."
>
> **"**The available evidence for indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (high intensity), reduced growth (moderately-high-intensity), impairment of essential behaviors related to successful rearing and migration (moderate intensity), physiological trauma (moderate intensity), and reproductive failure (moderate intensity)."

While the Oregon BiOps from USFWS and NOAA clearly suggest a potential for adverse effects of the EPA 2001 freshwater acute and chronic cadmium criteria, the chronic criterion (0.25 µg/L) was accepted by EPA and incorporated into Oregon's aquatic life toxics criteria. One

potential reason for this acceptance is the inconsistent dose response curve in Hanson et al. (2002) that served as the basis for the "likely to adversely affect" determination for the chronic criterion, suggesting a questionable data set.

The 2016 EPA recommended freshwater chronic cadmium criterion of 0.72 µg/L has not undergone ESA consultation in other Pacific Northwest states.

Swinomish Tribe Biological Evaluation

The Swinomish BE concluded no effects of their submission of a freshwater acute cadmium criterion of 1.3 µg/L (hardness of 100 mg/L) and chronic cadmium criterion of 0.55 µg/L (hardness of 100 mg/L; USEPA, 2022a). The Swinomish submittal for cadmium aligns with previously approved Idaho freshwater acute (1.34 µg/L) and chronic (0.60 µg/L) cadmium criteria.

Criteria Calculations

Freshwater Acute Cadmium Criterion

The proposed freshwater acute cadmium criterion uses the same derivation methods as EPA's recommendations (USEPA, 2016). The freshwater acute cadmium criterion is based upon the commercially important rainbow trout (*Oncorhynchus mykiss*). EPA found that the rainbow trout SMAV was less than the 5th percentile of the GMAV toxicity distribution for the freshwater acute data set, necessitating the use of rainbow trout SMAV to derive criteria. Rather than using the geometric mean of acute toxicity values for rainbow trout to derive the acute criterion, we used the 20th percentile of available acute toxicity data for rainbow trout to add increased protection for endangered species. We sought to align our proposed freshwater acute cadmium criterion with Idaho's and Swinomish approved criterion of 1.3 µg/L to ensure protection of endangered species. We did not find new freshwater acute toxicity studies since EPA last updated the cadmium criteria that would lower the GMAV.

Table 20 shows the calculated 20th percentile of 30 rainbow trout LC50 values from the acute toxicity dataset presented in EPA's 2016 cadmium recommendations (USEPA, 2016). The 20th percentile was used to align with Idaho and the Swinomish Tribe freshwater acute cadmium criteria that has been demonstrated to be protective of endangered species and approved through ESA consultation. Calculation results are as follows:

CMC = 1.376 µg/L (hardness of 100 mg/L)

$CMC = e^{(0.9789 \times \ln(hardness) - 4.189)} \times CF$

Where CF (conversion factor from total to dissolved fraction) = 1.136672 - [(ln hardness) x (0.041838)]

FAV = 2.7518

CMC = FAV /2 = 2.7518 / 2 = 1.376 ug/L

Acute criterion (total) = 1.4 µg/L (hardness of 100 mg/L; rounded to two significant digits)

Acute criterion (dissolved) = 1.3 µg/L (hardness of 100 mg/L; rounded to two significant digits)

Table 20. Rainbow trout acute toxicity values used for criteria derivation (from USEPA, 2016).

Acute Value (µg/L)	Normalized Acute Value (µg/L)*	Reference
1.75	5.506	Davies 1976
1.3	5.479	Chapman 1978
1.0	4.214	Chapman 1978
3.0	6.641	Phipps and Holcombe 1985
1.88	3.565	Stubblefield 1990
2.66	5.569	Davies et al. 1993
3.15	1.567	Davies et al. 1993
3.02	6.070	Davies et al. 1993
6.12	2.779	Davies et al. 1993
2.79	9.371	Davies and Brinkman 1994
8.54	3.376	Davies and Brinkman 1994
13.4	4.873	Davies and Brinkman 1994
2.09	7.265	Davies and Brinkman 1994
10.5	3.886	Davies and Brinkman 1994
10.0	3.637	Davies and Brinkman 1994
0.71	2.255	Stratus Consulting 1999
0.47	1.563	Stratus Consulting 1999
0.51	1.570	Stratus Consulting 1999
0.38	1.227	Stratus Consulting 1999
1.29	4.191	Stratus Consulting 1999
2.85	3.183	Stratus Consulting 1999
3.7	3.594	Besser et al. 2007
5.2	5.051	Besser et al. 2007
3.061	2.945	Calfee et al. 2014
5.115	4.786	Calfee et al. 2014
2.933	2.745	Calfee et al. 2014
3.929	3.780	Calfee et al. 2014
4.808	5.003	Calfee et al. 2014

Acute Value (µg/L)	Normalized Acute Value (µg/L)*	Reference
3.135	3.045	Calfee et al. 2014
5.401	5.400	Wang et al. 2014
20th percentile of Normalized Acute Values	**2.7518 (FAV)**	
Acute criterion	**1.376 (CMC)**	

* Normalized to hardness of 100 mg/L

Freshwater Chronic Cadmium Criterion

The proposed freshwater chronic cadmium criterion was calculated from the 2016 EPA toxicity dataset and used the 1st percentile of the toxicity data distribution (Table 9 from USEPA, 2016).

FCV = 0.4618 µg/L (hardness of 100 mg/L)

$CCC = e^{(0.7977 \times \ln(\text{hardness}) - 4.446)} \times CF$

Where CF (conversion factor from total to dissolved fraction) = 1.101672 - [(ln hardness) x (0.041838)]

Chronic criterion (total) = 0.4527 µg/L (hardness of 100 mg/L; rounded to two significant digits)

Chronic criterion (dissolved) = 0.41 µg/L (hardness of 100 mg/L; rounded to two significant digits)

Saltwater Acute and Chronic Cadmium Criteria

Washington's saltwater acute and chronic cadmium criteria are outdated and do not match EPA recommendations. We propose to match EPA recommendations for the saltwater acute and chronic cadmium criteria. There are no known ESA consultation issues in other Region 10 states.

Chromium III

Summary of Criteria Recommendations and Changes

There are no known concerns regarding protection of endangered species in Washington using EPA recommendations for chromium III. We propose to adopt criteria that align with EPA recommendations (Table 21).

Table 21. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic chromium III criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	550*^ (1-hour)	180*^ (4-day)	-	-

EPA	570*^ (1-hour)	74*^ (4-day)	-	-
Proposed	570*^ (1-hour)	74*^ (4-day)	-	-

* Hardness based criteria (numeric value shown based on 100 mg/L)
^ Presented as the dissolved fraction

Endangered Species Act Consultation

There were no jeopardy calls for the freshwater acute (574 µg/L) and chronic (74 µg/L) chromium III criteria in Oregon (USFWS, 2012; NMFS, 2012). Furthermore, the Swinomish BE indicated a not likely to adversely affect (NLAA) determination for freshwater acute and chronic chromium III EPA recommendations (USEPA, 2022a).

Chromium VI

Summary of Criteria Recommendations and Changes

The proposed freshwater chromium VI criteria accounts for endangered species protection levels for species in Washington by incorporating the new science available since EPA last updated the freshwater criteria in 1995 (Table 22; USEPA, 1996).

While there were no jeopardy calls for chromium VI in Idaho or Oregon, the information presented as well as the Swinomish BE suggests that endangered species and their populations in Washington may be at risk at EPA recommendations. We therefore, decided to use new science available and the 1st percentile of the toxicity data distribution to derive chromium VI criteria. No changes were necessary for saltwater criteria because Washington's saltwater criteria are identical to EPA recommendations, and there are no endangered species protection issues highlighted in previous ESA consultations in Oregon.

Table 22. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic chromium VI criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	15^ (1-hour)	19^ (4-day)	1100^ (1-hour)	50^ (4-day)
EPA	16^ (1-hour)	11^ (4-day)	1100^ (1-hour)	50^ (4-day)
Proposed	18^ (1-hour)	4.5^ (4-day)	No change	No change

^ Presented as the dissolved fraction

Endangered Species Act Consultation

Idaho

The Idaho USFWS BiOp reported a likely to adversely affect (LAA) determination for the freshwater chronic chromium VI criterion (11 µg/L) for bull trout and white sturgeon but did not result in a jeopardy call (USFWS, 2015; Table 4). The information presented in Idaho BiOps presented concerns for Washington's endangered species. The USFWS Idaho BiOp specifically states:

> "Given the information discussed above that long-term exposure to chromium (VI) at the proposed chronic criterion level may cause reduced growth of juvenile bull trout, and depending on the magnitude of the growth reduction, reduced overwinter survival, the Service concludes that individual juvenile bull trout may be adversely affected by the proposed chronic chromium criterion. However, these effects are not likely to occur at a population level given the other above studies involving the chronic exposure effects of chromium that resulted in reduced salmonid growth only at chromium concentrations well above the proposed chronic criterion for chromium (VI) of 11 µg/L."

> "Given the information discussed above that long-term exposure to chromium (VI) at the proposed chronic criterion levels may cause reduced growth of juvenile bull trout, and depending on the magnitude of the growth reduction, reduced overwinter survival, the Service concludes that individual juvenile Kootenai River white sturgeon may be adversely affected by the proposed chronic criterion for chromium (VI). However, these effects are not likely to occur at a population level given the other above studies involving the chronic exposure effects of chromium that resulted in reduced salmonid growth only at chromium concentrations well above the proposed chronic criterion for chromium (VI) of 11 µg/L."

Oregon

The Oregon USFWS BiOps reported likely to adversely affect determinations but did not result in jeopardy for ESA listed species in Oregon (NMFS, 2012). The determinations present concerns for Washington's endangered species. The NMFS BiOp states:

> "Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration may not suffer acute toxic effects, but will suffer chronic toxic effects."

> "The available evidence for chromium (III) and chromium (VI), respectively, indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity, for chromium III, and low intensity for chromium VI) and reduced growth (moderately-high-intensity, for chromium III and chromium VI)."

> "In summary, the available evidence for saltwater chromium (VI) indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer

acute or chronic toxic effects including mortality (moderate intensity) and sublethal effects (moderately-high-intensity)."

Swinomish Tribe Biological Evaluation

The Swinomish biological evaluation found that there would likely be indirect effects to prey species for ESA listed species in Washington from exposure to the freshwater chronic and saltwater acute and chronic chromium VI criteria (USEPA, 2022a). EPA also references previous Oregon and Idaho BiOps mentioned previously:

> "EPA acknowledges that in the Oregon toxic consultation, NMFS determined some adverse effects from the acute chromium VI criteria were possible, but EPA defers to the more recent assessments in the Idaho consultation. Further, Chinook, steelhead, and bull trout exposure to the chromium VI at the criterion level in fresh waters of action area is unlikely due to the lack of current and anticipated sources of chromium VI."

Criteria Calculations

Freshwater Acute Chromium VI Criterion

The data used to derive the freshwater acute chromium VI criterion is presented in Table 23. New studies that met data acceptability requirements are presented in Table 24. Studies used in previous EPA derivations but not used in this derivation are found in Table 25. The proposed freshwater acute criterion for chromium VI was derived using 43 GMAVs. Calculation results are as follows:

FAV = 36.01

CMC = 18.01

Acute criterion (total) = 18 µg/L (rounded to two significant digits)

Conversion factor (total to dissolved fraction) = 0.982

Acute criterion (dissolved) = 18.01 x 0.982 = 17.69 µg/L

Acute criterion (dissolved) = 18 µg/L (rounded to two significant digits)

Table 23. Freshwater acute toxicity data used for criteria derivation.

Rank	GMAV (µg/L)	Species	SMAV (µg/L)
1	28.94	Daphnia magna	23.07
		Daphnia pulex	36.3
2	29	Pseudosida ramosa	29
3	36.35	Simocephalus serrulatus	40.9
		Simocephalus vetulus	32.3
4	67.1	Gammarus pseudolimaeus	67.1
5	80.87	Ceriodaphnia reticulata	45.1
		Ceriodaphnia dubia	145
6	125	Thamnocephalus platyurus	125

Rank	GMAV (µg/L)	Species	SMAV (µg/L)
7	170	Notodiaptomus conifer	170
8	177	Lecane papuana	177
9	300	Chironomus plumosus	300
10	456	Lampsilis siliquoidea	456
11	583	Amphipod	583
12	630	Hyalella azteca	630
13	650	Plumatella emarginata	650
14	919	Margaritifera falcata	919
15	1000	Culicoides furens	1000
16	1440	Pectinatella magnifica	1440
17	1560	Lophodella carteri	1560
18	2841	Bryocamptus zschokkei	1850
		Bryocamptus pygmaeus	3480
		Bryocamptus minutus	3560
19	3516	Tubifex tubifex	3516
20	3820	Attheyella crassa	3820
21	4000	Salmo salar	4000
22	23010	Physa heterostropha	23010
23	30450	Morone saxatilis	30450
24	32000	Xyrauchen texanus	32000
25	36300	Perca flavescens	36300
26	38000	Culex quinquefasciatus	38000
27	46000	Etheostoma nigrum	46000
28	47180	Pimephales notatus	54225
		Pimephales promelas	41050
29	49600	Ericymba buccata	49600
30	51250	Campostoma anomalum	51250
31	57300	Tanytarsus dissimilis	57300
32	59000	Salvelinus fontinalis	59000
33	61000	Chironomus tentans	61000
34	66000	Ptychocheilus Lucius	66000
35	67610	Notropis atherinoides	48400
		Notropis chrysocephalus	85600
		Notropis stramineus	74600
36	69000	Oncorhynchus mykiss	69000
37	72600	Promoxis annularis	72600
38	81000	Gila elegans	81000
39	123500	Lepomis cyanellus	114700
		Lepomis macrochirus	132900
40	140000	Enallagma aspersum	140000

Rank	GMAV (µg/L)	Species	SMAV (µg/L)
41	151950	Gambusia affinis	151950
42	176000	Orconectes rusticus	176000
43	1870000	Neophasganophora capitata	1870000

Table 24. New freshwater acute studies that met data acceptability requirements since EPA last updated chromium VI criteria (S = static, R = static renewal, U = unmeasured test concentrations, M = measured test concentrations).

Species	Method	LC50 (µg/L)	Used in Derivation?	Reference
Ceriodaphnia dubia	S, M	145	Yes.	Baral et al. 2006
Ceriodaphnia dubia	S, U	81.11	No. Other studies using the same species measured test concentrations.	Hockett 1996
Pimephales promelas	S, M	22464	No. FT, M available.	Baral et al. 2006
Gambusia affinis	R, U	151950	Yes.	Begum et al. 2006
Tubifex tubifex	S, U	2910	Yes.	Fargasova 1999
Notodiaptomus conifer	S, U	170	Yes.	Gutierrez et al. 2010
Lecane hamata	S, U	4410	No. LC50 10x higher than other species within genus.	Perez-Legaspi & Rico-Martinez 2001
Lecane luna	S, U	3260	No. LC50 10x higher than other species within genus.	Perez-Legaspi & Rico-Martinez 2001
Lecane quadridentata	S, U	4500	No. LC50 10x higher than other species within genus.	Perez-Legaspi & Rico-Martinez 2001
Culex quinquefasciatus	S, U	38000	Yes.	Sorenson et al. 2006
Salmo salar	R, M	4000	Yes.	Grande 1983
Thamnocephalus platyurus	S, U	125	Yes.	Centeno et al. 1995
Chironomus plumosus	S, U	300	Yes.	Vedamanikan & Shazilli 2008
Culicoides furens	S, U	1000	Yes.	Vedamanikan & Shazilli 2008
Ptychocheilus lucius	S, U	66000	Yes.	Buhl 1997
Gila elegans	S, U	81000	Yes.	Buhl 1997
Xyrauchen texanus	S, U	32000	Yes.	Buhl 1997

Species	Method	LC50 (µg/L)	Used in Derivation?	Reference
Bryocamptus pygmaeus	R, U	3480	Yes.	Di Marzio et al. 2009
Bryocamptus minutus	R, U	3560	Yes.	Di Marzio et al. 2009
Bryocamptus zschokkei	R, U	1850	Yes.	Di Marzio et al. 2009
Attheyella crassa	R, U	3820	Yes.	Di Marzio et al. 2009
Tubifex tubifex	S, U	5490	Yes.	Maestre et al. 2009
Oncorhynchus mykiss	R, U	12300	No. Other studies used flow-through design using the same species.	Kazlauskiene 1994
Tubifex tubifex	S, U	2720	Yes.	Rathore et al. 2002
Pseudosida ramosa	S, U	29	Yes.	Freitas & Rocha 2013
Lecane papuana	S, M	177	Yes.	Garza-Leon et al. 2021
Lampsilis siliquoidea	R, M	456	Yes.	Wang et al. 2017
Margaritifera falcata	R, M	919	Yes.	Wang et al. 2017

Table 25. Freshwater acute studies not used from previous EPA derivations.

Species	LC50 (µg/L)	Reason	Reference
Poecilia reticulata	30000	Non-North American species	USEPA, 1996
Carassius auratus	19500	Non-North American species	USEPA, 1996

Freshwater Chronic Chromium VI Criterion

There was inadequate freshwater chronic chromium VI data to calculate criteria using the eight-family method. The ACR of 2.917 was previously used to calculate the freshwater chronic chromium VI criterion as presented in 1995 updates to aquatic life (USEPA, 1996). Additional chronic chromium VI ACRs were available since last EPA updates (Table 26). The newly calculated ACR used to derive the chronic chromium VI criteria is 7.691. Calculation results are as follows:

FAV = 36.01

FACR = 7.691

CCC = 4.682 µg/L

Chronic criterion (total) = 4.7 µg/L (rounded to two significant digits)

Conversion factor (total to dissolved fraction) = 0.962

Chronic criterion (dissolved) = 4.682 x 0.962 = 4.5 µg/L (rounded to two significant digits)

Table 26. Acute to chronic ratios (ACR) used in chronic criterion derivation.

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
Daphnia pulex			5.92	5.92	1996 EPA doc
Simocephalus vetulus			5.267	5.267	1996 EPA doc
Simocephalus serrulatus			2.055	2.055	1996 EPA doc
Ceriodaphnia reticulata			1.13	1.13	1996 EPA doc
Pimephales promelas			18.55[A]	18.55[A]	1996 EPA doc
Daphnia carinata	423	71	5.96	5.96	Hickey 1989
Daphnia magna	224	50	4.48		Hickey 1989
Daphnia magna	290	17.7	16.4	8.572	Diamantino et al. 2000

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
Ceriodaphnia dubia	53	5	10.6	10.6	Hickey 1989
Notodiaptomus conifer	170	5.30	32.06	32.06	Gutierrez et al. 2010
Hypsiboas pulchellus	29600	1732	17.09	17.09	Natale et al. 2006
Pseudosida ramose	29	1.73	16.74	16.74	Frietas and Rocha 2013
Lecane papuana	177	62.58	2.83	2.83	Garza-Leon et al. 2021
Lampsilis siliquoidea	456	26.15	17.44	17.44	Wang et al. 2017
Geometric mean				7.691	

* Geometric mean of ACRs were calculated for similar species preceding the final acute chronic ratio calculation

[A] Previously excluded in 1995 update because was 10x greater than other species but new studies suggest it is within an acceptable range for inclusion into FACR calculations.

Saltwater Acute and Chronic Chromium VI Criteria

No changes are proposed to the saltwater acute and chronic chromium VI criteria. Washington's current saltwater chromium VI criteria are identical to EPA recommendations, and there are no known ESA consultation issues in other Region 10 states.

Copper

Summary of Criteria Recommendations and Changes

Washington's current rules have freshwater copper criteria based on hardness (Table 27). EPA recommends freshwater copper criteria using a model-based approach called the biotic ligand model (BLM) which is dependent on 12 different water quality inputs to determine the bioavailable fraction of copper. Washington proposes to use a different model-based approach for freshwater copper criteria using a multiple linear regression (MLR) model. Conceptually, this approach is simply a refinement of the current hardness-based approach, but considers three water quality parameters (hardness, pH, and dissolved organic carbon) compared to one. The MLR model is presented as a regression equation that uses water body specific inputs to calculate criteria. The copper MLR model has been published in the scientific literature. Furthermore, EPA has indicated that they are moving towards MLR based models for metals criteria in their Cooperative Research and Development Agreement (CRADA) project. Given the lack of data for the 12 parameters needed to run the BLM model throughout Washington, we propose using the copper MLR model for which we have adequate water quality information to develop default values. We propose a default freshwater copper acute criterion of 2.0 µg/L for western Washington and 2.5 µg/L for eastern Washington (boundaries for eastern and western are defined in the methodology below and in WAC 222-16-010). The freshwater default chronic copper criterion is 1.6 µg/L for western Washington and 1.8 µg/L for eastern Washington.

These default criteria are based on the 5th percentile of the MLR criteria for the respective west and east boundaries (Table 27). Criteria calculated using concurrently sampled pH, hardness, and DOC for a specific water body supersede the default criteria, regardless of whether the default criteria are higher or lower.

Table 27. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic copper criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	Hardness-based (1-hour)	Hardness-based (4-day)	4.8 (1-hour)	3.1 (4-day)
EPA	Biotic Ligand Model (1-hour)	Biotic Ligand Model (4-day)	4.8 (1-hour)	3.1 (4-day)
Proposed	West: 2.0# East: 2.5# (Multiple Linear Regression Model; 1-hour)	West: 1.6# East: 1.8# (Multiple Linear Regression Model; 4-day)	No change	No change

Represent 5th percentile default criteria values. The boundary between east and west designations is defined in WAC 222-16-010.

Copper MLR vs Copper BLM Models

The copper MLR offers several advantages compared with the BLM. EPA highlights these points in the aluminum MLR technical document (USEPA, 2018):

> "The EPA decided to use an empirical MLR approach in this aluminum criteria update rather than a BLM model due to: 1) the relative simplicity and transparency of the model, 2) the relative similarity to the available BLM model outputs, and 3) the decreased number of input data on water chemistry needed to derive criteria at different sites."

The copper MLR model is relatively new in that it was published in 2017 (Brix et al. 2017). EPA's CRADA project aim is to develop a simplified modeling frameworks for predicting the bioavailability of metals. This translates to developing MLR models for other metals in the future (Metals CRADA Phase 1 Report | US EPA[5]). Comparisons between the performance of MLR and BLM copper models have been completed. In an updated version of the copper MLR model, Brix et al. (2021) found performance between the two models were generally comparable. Brix et al. (2021) noted differences in performance on a species-specific basis and differences in criteria depending on water chemistry.

In an analysis to evaluate community protection levels by the copper MLR model, Mebane et al. (2023) compared the MLR-based chronic criteria from Brix et al. (2021) to an independently

[5] https://www.epa.gov/wqc/metals-crada-phase-1-report

compiled chronic criteria dataset and concluded the Brix et al. (2021) copper MLR model generated criteria protective of the 95th percentile level as intended by EPA's 1985 guidelines for deriving aquatic life toxics criteria. Mebane et al. (2023) also compared the MLR-based chronic copper criterion with field and experimental ecosystem studies with copper and found the MLR-based criteria were largely protective and performed better than the hardness-based or BLM-based criteria. Mebane et al. (2023) concludes:

> "Considering the state of the science, model performance, water quality goals to protect freshwater environments, USEPA policy directions, transparency, and simplicity, the MLR is the best candidate model presently available for statewide criteria updates."

Criteria Calculations

Methodology for Default Criteria

The default criteria were calculated using concurrently sampled pH, hardness, and dissolved organic carbon data from Washington's EIM database and the federal WQ Portal. Data from EIM and the federal WQ Portal were downloaded in March 2023. We also examined concurrently sampled total organic carbon (TOC), hardness, and pH as well as conductivity, pH, and DOC. We calculated conversion factors to translate TOC to DOC and conductivity to hardness as detailed below.

The data qualifiers and management decisions are presented in Appendix B. Data were reviewed for quality with respects to the intended use of the aquatic life toxics rulemaking. We reviewed sampling locations, the study purpose, outlier values and units, reported QA levels, and field collection comments. Records not meeting the intended use of the aquatic life toxics rulemaking were removed. The final count of concurrent samples was 3,337 events across 646 unique locations (Figure 1). Each of the 3,337 concurrent samples were entered into the MLR-based copper equation.

We then compiled the 3,337 calculated criteria values for waterbodies throughout the state and calculated the 5th percentile of those 3,337 different criteria to be representative of the default criteria. The 5th percentile of the criteria distribution represents conservative criteria values that are intended to protect the majority of waters with regulated discharge of copper. We considered ecoregional default values (e.g., EPA level III ecoregions), but we had limited geospatial representation in some ecoregions and therefore developed default values for western and eastern Washington. Eastern and western Washington is defined by definitions in WAC 222-16-010 (Figure 2). More specifically, **"Eastern Washington"** means the geographic area in Washington east of the crest of the Cascade Mountains from the international border to the top of Mt. Adams, then east of the ridge line dividing the White Salmon River drainage from the Lewis River drainage and east of the ridge line dividing the Little White Salmon River drainage from the Wind River drainage to the Washington-Oregon state line. **"Western Washington"** means the geographic area of Washington west of the Cascade crest and the drainages defined in Eastern Washington. We had 367 unique sample locations with 2,210 samples in western Washington and 279 unique locations with 1,127 samples in eastern Washington.

A 5th percentile default criteria was used to provide protection of all aquatic species. Washington developed west and east default values due to limited dispersion of concurrent sampling sites throughout the state that precluded the ability to develop ecoregional or watershed specific default criteria values. A 5th percentile default criteria is appropriate because individual ecoregions and watershed water chemistry are not accounted for using a default value but rather becomes integrated into the dataset. The 5th percentile default value is more protective of waters with higher bioavailability of copper.

The default acute copper criteria of 2.0 (west) and 2.5 (east) ug/L are similar to the calculated CMC (i.e., acute criterion) of 2.3 ug/L presented in the copper BLM technical support document and the most sensitive SMAV of 2.37 ug/L for *Daphnia pulicaria* (USEPA, 2007) under normalized BLM conditions: temperature = 20°C, pH = 7.5, DOC = 0.5 mg/L, Ca = 14.0 mg/L, Mg = 12.1 mg/L, Na = 26.3 mg/L, K = 2.1 mg/L, SO4 =81.4 mg/L, Cl = 1.90 mg/L, Alkalinity = 65.0 mg/L and S = 0.0003 mg/L. The calculated CCC (i.e., chronic criterion) in the copper BLM technical support document was 1.45 ug/L (under normalized BLM conditions; USEPA, 2007), which was similar to the 5th percentile default value proposed of 1.6 (west) and 1.8 (east) ug/L. Ultimately, protective levels of copper are dictated by water quality conditions and are subject to site-specific conditions, making direct comparisons difficult between BLM and MLR calculated criteria.

Permittees will have the opportunity to collect their own site-specific chemistry data to calculate site-specific criteria that may afford higher criteria values than the 5th percentile default criteria. If site-specific criteria are less than the 5th percentile default criteria, permittees will need to use the site-specific information to determine effluent limits.

Conversion Factors

Total Organic Carbon to Dissolved Organic Carbon

We also examined instances where we had concurrently sampled total organic carbon (TOC), hardness, and pH since 2000 to add additional sampling events and increase representation of water bodies throughout the state. We developed a conversion factor to translate TOC to DOC. We downloaded all concurrently sampled TOC and DOC data as of May 2023 and calculated the ratio of DOC to TOC or the proportion of TOC that is DOC. For the TOC conversion factor, we used the 10th percentile of all ratios for statewide data. We used a conservative value (i.e., 10th percentile) because it results in more protective criteria (i.e., the lower the DOC concentration the lower the criteria value) and the goal of default criteria are to be protective of the majority of state waters. When we converted TOC to DOC, 105 sampling events were added to our MLR dataset (105 sample events out of the total 3,337 total sampling events). The statewide conversion factor based on the 10th percentile of the ratio of DOC to TOC is 0.81 (see example below). The TOC to DOC conversion factor is similar to Oregon's conversion factor of 0.83 (ODEQ, 2021), the EPA national value of 0.86 (USEPA, 2007), and Massachusetts value of 0.86 (MassDEP, 2021).

Example:
TOC = 10 mg/L
DOC = 10 mg/L x 0.81 = 8.1 mg/L

Conductivity to Hardness

We also examined instances where we had concurrently sampled conductivity, hardness, and pH since 2000 to add additional sampling events and increase representation of water bodies throughout the state. We developed a conversion factor to translate conductivity to hardness (Figure 4). We downloaded all the concurrently sampled conductivity and hardness measurements data in August 2023. For the specific conductance versus hardness dataset, we first took the natural log of the values before developing a linear regression model between the two variables to improve model fit. The natural-log transformed data were used to establish the conversion equation used to estimate total hardness from conductivity. When we converted conductivity to hardness 910 sampling events were added to our MLR dataset (910 sample events out of the total 3,337 total sampling events). The linear regression equation that was used to convert conductivity to hardness is as follows:

LN(Hardness) = 1.0108*LN(conductivity) - 0.9233

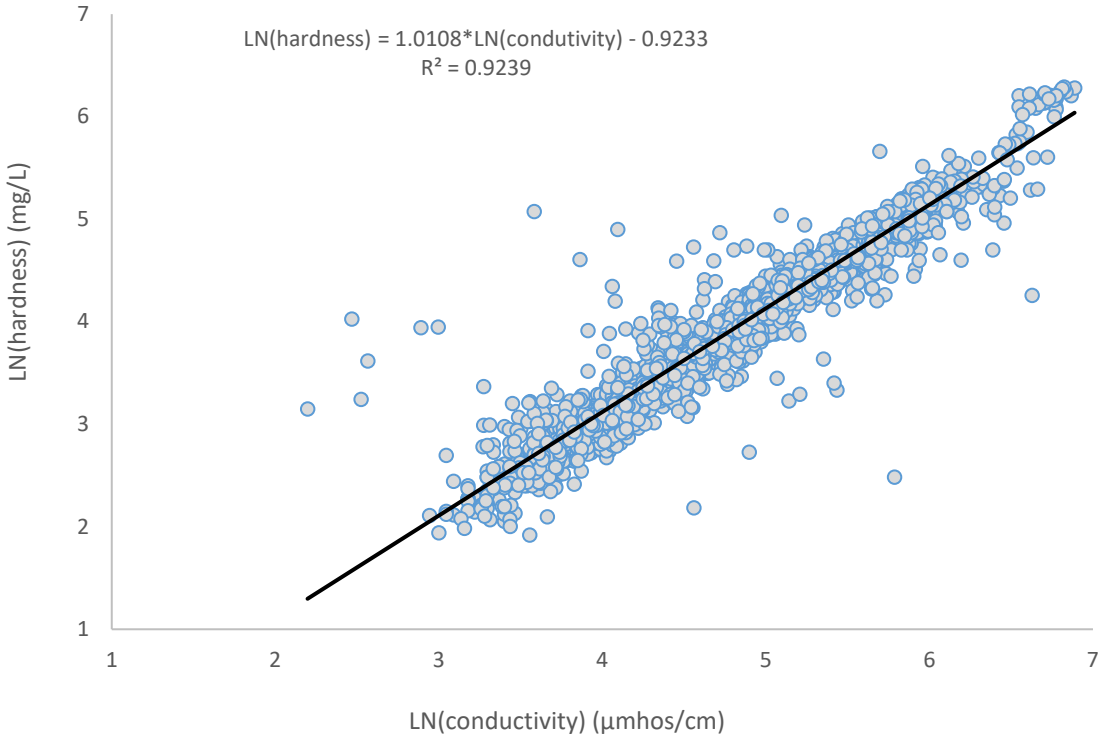

Figure 4. Relationship between hardness and conductivity (in micromhos per centimeter (µmhos/cm) for concurrent sampling throughout Washington.

Default Criteria

The default freshwater acute copper criterion is 2.0 µg/L for western Washington and 2.5 µg/L for eastern Washington. The default chronic copper criterion is 1.6 µg/L for western Washington and 1.8 µg/L for eastern Washington. The default criteria are based on data concurrently sampled in Ecology's EIM database and the federal WQ Portal. If site-specific water quality information exists for a water body, that information must be used to develop

site-specific copper criteria. In the absence of site-specific water chemistry data, the default copper criteria apply.

Freshwater Acute Copper Criteria

The freshwater acute copper criterion is represented by the higher value calculated from the two equations:

Equation 1) Acute criteria (empirical) = $e^{(0.700*\ln(DOC) + 0.579*\ln(hardness) + 0.778*pH - 6.738)}$, and

Equation 2) Acute criteria (reverse ACR) = $e^{(0.855*\ln(DOC) + 0.221*\ln(hardness) + 0.216*pH - 1.183)}$.

Equation 1 represents the acute copper MLR model presented in Brix et al. 2021. Equation 2 represents a reverse ACR based equation in which the ACR of 2.49 is applied to the chronic copper MLR model presented in Brix et al. 2021. The reverse ACR based equation is calculated by application of the ACR to the chronic criterion followed by division by two to be consistent with EPA methods for CMC calculations.

This approach was necessary because at low hardness and low DOC, low pH and low DOC, and high DOC and low hardness, the acute empirical model generates criteria lower than the chronic empirical model (examples presented in Figure 5). This is due to differences in the DOC, hardness, and pH slopes in the empirical acute model versus the empirical chronic model. To resolve these slope related issues, we developed rule language that uses the empirical acute model to the intersection of the acute empirical model and the applications of the reverse ACR based model at which point the reverse ACR based model becomes applicable (red dotted line in Figure 6). In other words, the applicable model for the acute criterion is whichever acute model is higher (the empirical based model or the reverse ACR based model). This method ensures the acute criterion is always greater than the chronic criteria. This concept is discussed in an upcoming publication (Brix et al. in prep).

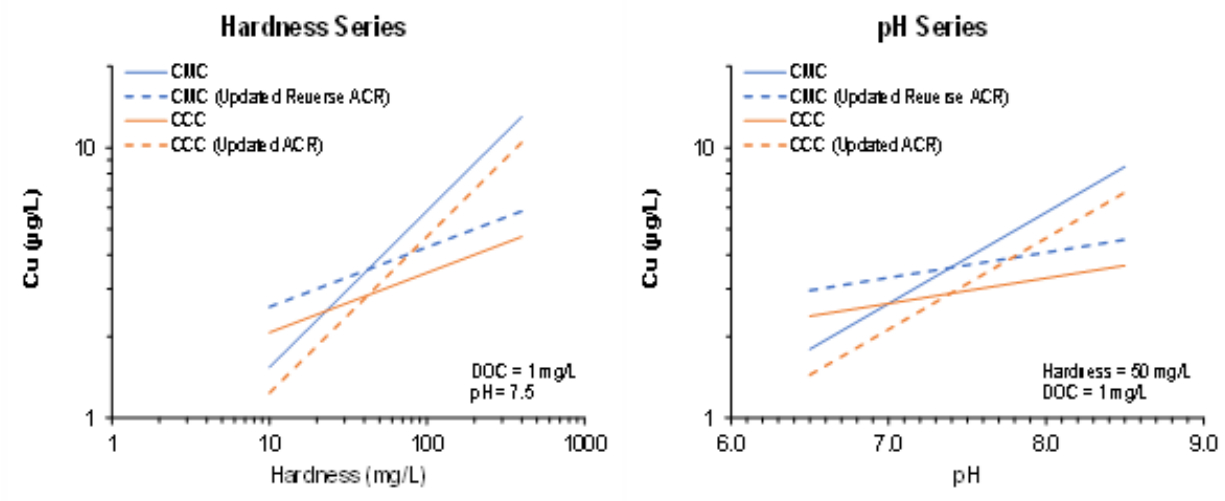

Figure 5. Demonstration of how the empirical based models (CMC and CCC), updated ACR, and the reverse ACR models function at different pH, hardness, and dissolved organic carbon levels.

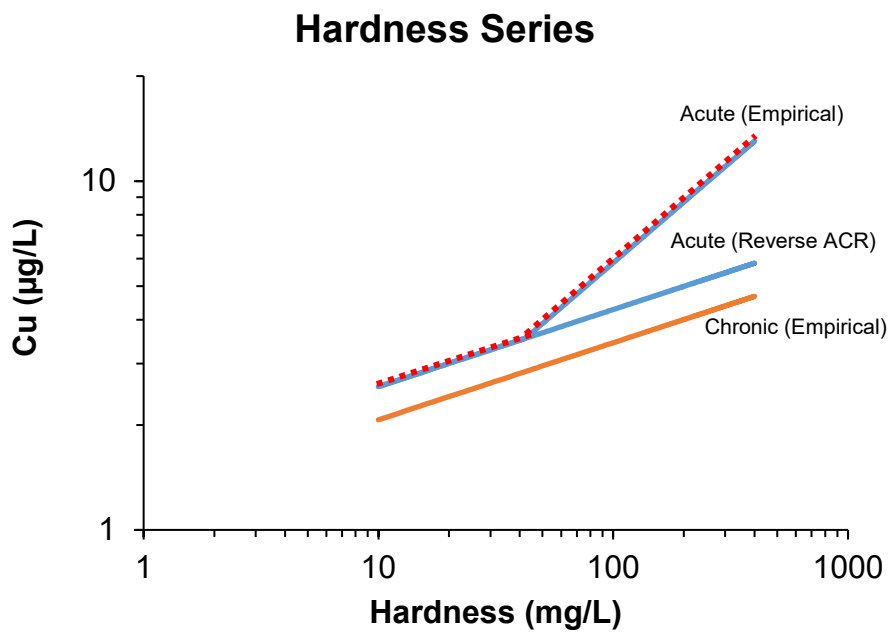

Figure 6. Depiction of how the acute MLR models functions in relation to the chronic MLR model. The proposed copper acute copper criterion states two separate equations, whichever is greater. Equation 1 represents the empirical acute based MLR model, while equation 2 represents the reverse ACR based model. The red dotted line depicts how the acute MLR model functions on the basis of these two models.

Table 28. Acute to chronic ratios used in the development of the copper multiple linear regression equation that are representative of data presented in Brix et al. 2021.

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
Ceriodaphnia dubia	28.42	7.90	3.60		Belanger et al. 1989
	63.33	19.36	3.27		Belanger et al. 1989
	17.97	9.17	1.96		Carlson et al. 1986
	25	12	2.1		Wang et al. 2011
	157	40	3.9		Wang et al. 2011
	267	41	6.5	3.27	Wang et al. 2011
Cottus bairdii	83	29.35	2.8	2.80	Besser et al. 2007
Daphnia magna	26	12.58	2.07		Chapman et al. Manuscript
	33.76	19.89	1.7		Chapman et al. Manuscript

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
	69	6.06	11.39		Chapman et al. Manuscript
	10.1	8.6	1.2		Villavicencio et al. 2011
	9.9	7.9	1.3		Villavicencio et al. 2011
	22.7	11.5	2.0		Villavicencio et al. 2011
	13.2	8.6	1.5		Villavicencio et al. 2011
	10.7	9.7	1.1		Villavicencio et al. 2011
	5.9	5.4	1.1		Villavicencio et al. 2011
	13.3	12.1	1.1		Villavicencio et al. 2011
	22.4	10.5	2.1		Villavicencio et al. 2011
	16.9	10.5	1.6		Villavicencio et al. 2011
	12	6.3	1.9		Villavicencio et al. 2011
	28	9.3	3.0		Villavicencio et al. 2011
	30.2	16	1.9		Villavicencio et al. 2011
	15.9	14.3	1.1		Villavicencio et al. 2011
	27.4	13.4	2.0		Villavicencio et al. 2011
	64.4	29.8	2.2		Villavicencio et al. 2011
	36.8	3.2	11.5		Villavicencio et al. 2011
	40.9	8.8	4.6	2.08	Villavicencio et al. 2011
Daphnia pulex	25.74	2.83	9.1		Winner 1985
	27.6	7.07	3.9		Winner 1985
	28.8	9.16	3.14	4.80	Winner 1985
Oncorhynchus mykiss	80	27.77	2.88		Seim et al. 1984
	58	40	1.5		Besser et al. 2007
	8.9	5.2	1.7		Cremazy et al. 2017
	12.7	6.6	1.9		Cremazy et al. 2017
	19.7	5	3.9		Cremazy et al. 2017
	5.9	5.5	1.1		Cremazy et al. 2017

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
	8.5	8.2	1.0		Cremazy et al. 2017
	41.3	33	1.3		Cremazy et al. 2017
	139.2	99	1.4		Cremazy et al. 2017
Oncorhynchus tshawytscha	33.1	5.92	5.59	5.59	Chapman 1975, 1982
Salvelinus fontinalis	198.7	141.0	1.4	1.40	Hansen et al. 2000, 2002
Villosa iris	15	10	1.5		Wang et al. 2011
	32	8.8	3.6		Wang et al. 2011
	72	38	1.9	2.20	Wang et al. 2011
Cyprinodon variegatus	368	249	1.48	1.48	Hughes et al. 1989
Geometric mean				2.49	

Table 29. Acute to chronic ratios not used for copper.

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Reason not used	Reference
Ceriodaphnia dubia	18	12	15	ACR is approximately 5 times greater than other ACRs for this species	Besser et al. 2007
Daphnia magna	8.8	9.2	0.96	ACR is <1	Villavicencio et al. 2011
Daphnia magna	3.6	8.5	0.42	ACR is <1	Villavicencio et al. 2011
Daphnia magna	3.1	10.2	0.30	ACR is <1	Villavicencio et al. 2011

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Reason not used	Reference
Lymnaea stagnalis	20	1.8	11.1	ACR species mean is approximately 10x greater than lowest species mean ACR	Brix et al. 2011
Oncorhynchus mykiss	29	31	0.94	ACR is <1	Cremazy et al. 2017
Oncorhynchus mykiss	46	49	0.94	ACR is <1	Cremazy et al. 2017
Oncorhynchus mykiss	12.7	16	0.79	ACR is <1	Cremazy et al. 2017
Oncorhynchus mykiss	6.7	18	0.37	ACR is <1	Cremazy et al. 2017
Pimephales promelas	88.3	5.1	17.3	ACR approximately 10x greater than lowest species mean ACR	Spehar and Fiandt, 1986

Freshwater Chronic Copper Criteria

The copper MLR based equation was used to calculate the default copper criteria and can be used to determine site-specific chronic criteria (Brix et al. 2021). The equation is as follows:

Chronic criteria = $e^{(0.855*\ln(DOC) + 0.221*\ln(hardness) + 0.216*pH - 1.402)}$

Saltwater Acute and Chronic Copper Criteria

No changes are proposed to the saltwater acute and chronic copper criteria. Washington's current saltwater copper criteria are identical to EPA recommendations, and there are no known ESA consultation issues in other Region 10 states.

Iron

Summary of Criteria Recommendations and Changes

We propose to not adopt EPA recommended freshwater chronic iron criterion (Table 30). The EPA iron criterion does not meet the minimum data requirements for the eight-family method or alternative methods. The EPA iron criterion of 1000 µg/L is based on few field studies outlined in an EPA document from 1976 (USEPA, 1976) and does not follow EPA 1985 guidelines (Stephan et al. 1985). Furthermore, it is difficult to develop statewide iron criteria because of

the variable natural iron concentrations in water bodies throughout Washington. Washington will continue to use their narrative criteria to protect against toxic and aesthetic effects of iron.

Table 30. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic iron criteria with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	-	1000	-	-
Proposed	-	-	-	-

Lead

Summary of Criteria Recommendations and Changes

Washington's freshwater and saltwater lead criteria are identical to EPA's recommendations (Table 31). There were LAA determinations for freshwater acute and chronic lead criteria in Oregon for bull trout but there were no jeopardy calls. The new science and 1st percentile resulted in higher freshwater criteria values than EPA recommendations. Therefore, we propose no changes to Washington's freshwater and saltwater lead criteria.

Table 31. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic lead criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	65*^ (1-hour)	2.5*^ (4-day)	210^ (1-hour)	8.1^ (4-day)
EPA	65*^ (1-hour)	2.5*^ (4-day)	210^ (1-hour)	8.1^ (4-day)
Proposed	No change	No change	No change	No change

* Hardness based criteria (numeric value shown based on 100 mg/L)
^ Presented as the dissolved fraction

Mercury

Summary of Criteria Recommendations and Changes

The only action for mercury criteria proposed is the adoption of the mercury freshwater acute criterion recommended by EPA (Table 32). EPA recommendations for mercury freshwater and saltwater chronic criteria are significantly higher than Washington's criteria. Idaho's mercury freshwater chronic criterion received a jeopardy call and was identical to Washington's current criteria. EPA has indicated that they are working on updating their aquatic life toxics national

recommendations for mercury. We have decided to wait until EPA's new recommendations to revise chronic criteria for mercury.

Table 32. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic mercury criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	2.1* (1-hour)	0.012^ (4-day)	1.8* (1-hour)	0.025^ (4-day)
EPA	1.4* (1-hour)	0.77* (4-day)	1.8* (1-hour)	0.94* (4-day)
Proposed	1.4* (1-hour)	No change	No change	No change

* Presented as dissolved fraction

^ Presented as total recoverable fraction

Endangered Species Act Consultation

Idaho

There was a jeopardy call in the Idaho USFWS BiOp for the freshwater mercury chronic criterion of 0.012 µg/L (USFWS, 2015). The Idaho USFWS BiOp specifically states:

> "The common occurrence of mercury tissue concentrations in the tissue of fish exceeding a threshold concentration for reproductive or neurologic harm considered applicable to bull trout (0.3 mg/kg ww) while water concentrations of mercury were considerably less than the proposed 12 ng/L chronic aquatic life criterion indicates that the proposed chronic criterion would not be sufficient to protect all fish species. As no species-specific information were available for bull trout, we consider this general "fish: endpoint to apply to bull trout as well."
>
> "Based on the above information, implementation of the proposed chronic criterion for mercury is likely to adversely affect growth, reproduction, and behavior in the bull trout throughout its distribution in Idaho. Considering that the state of Idaho harbors 44 percent of all streams and 34 percent of all lakes and reservoirs occupied by the bull trout rangewide, these effects are considered to be significant. These effects are likely to impede (1) maintaining/increasing the current distribution of the bull trout, (2) maintaining/increasing the current abundance of the bull trout, and (3) achieving stable/increasing trends in bull trout populations."

Nickel

Summary of Criteria Recommendations and Changes

The proposed freshwater nickel criteria accounts for endangered species protection levels by incorporating new science available since EPA last updated the freshwater criteria in 1995

(Table 33; USEPA, 1996). The proposed freshwater nickel criteria are more stringent than EPA recommendations. Although jeopardy calls were specific to the freshwater chronic criterion for species relevant to Washington, new science was used to update the freshwater acute criterion. The freshwater chronic criterion is dependent on the acute criterion because it uses an ACR to derive the criterion value. Furthermore, we decided it was necessary to incorporate the new science for nickel because of the abundance of new data that demonstrates there are more sensitive species than previously used in the 1995 derivation (USEPA, 1996). No changes were necessary for saltwater criteria because Washington's saltwater nickel criteria are identical to EPA recommendations and there are no endangered species protection issues highlighted in previous ESA consultations in other Region 10 states.

Table 33. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic nickel criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	1415*^ (1-hour)	157*^ (4-day)	74^ (1-hour)	8.2^ (4-day)
EPA	470* (1-hour)	52* (4-day)	74^ (1-hour)	8.2^ (4-day)
Proposed	34*^ (1-hour)	5.6*^ (4-day)	No change	No change

* Hardness based criteria (numeric value shown based on 100 mg/L)
^ Presented as the dissolved fraction

Endangered Species Act Consultation

Idaho

There were likely to adversely affect (LAA) determinations for ESA listed species for nickel in Idaho (NMFS, 2014; USFWS, 2015). However, no jeopardy calls were made for similarly listed species in Washington. The Idaho BiOps (NMFS, 2014; USFWS, 2015) specifically state:

> "Based on the research results referenced above, the Service concludes that the proposed approval of the chronic aquatic life criteria for nickel is likely to adversely affect the bull trout via effects to one component (amphipods) of its prey base. Given the variety of prey species in the diet of the bull trout, this adverse effect is not likely to cause a significant adverse effect to the bull trout."
>
> "Based on the above analysis, the Service concludes that the proposed approval of the chronic aquatic life criterion for nickel is likely to adversely affect PCE 3 of bull trout critical habitat via effects to one component (amphipods) of its prey base. However, given the variety of prey species in the diet of the bull trout, this adverse effect is not likely to cause a significant adverse effect to the capability of bull trout critical habitat in Idaho to provide for an abundant prey base for the bull trout."

Oregon

There were likely to adversely affect (LAA) determinations for the nickel freshwater acute and chronic criteria to bull trout in Oregon. However, no jeopardy calls were made for similarly listed species in Washington. The Oregon BiOps (USFWS, 2012; NMFS, 2012) specifically state:

> "Based on model results relying upon rainbow trout response data for exposure to nickel at the proposed chronic criterion concentration, we conclude that chronic exposure of bull trout to nickel at the proposed chronic standard is likely to kill up to 151 adult bull trout, and injure/reduce the fitness (via reduced growth) of up to 1,370 individual adult bull trout per 3- year period over the 25-year term of the proposed action in surface waters along 820.6 miles of bull trout habitat within the action area."
>
> "We are unable to quantify the exact number of bull trout eggs that may be affected as it is not possible to accurately inventory this life stage within the action area at this time. However, we assume that some small portion of eggs will be adversely affected every 3 years during the 25- year term of the proposed action along 260.8 miles of bull trout spawning and rearing habitat exposed to nickel concentrations at the proposed chronic criterion because modeling indicates a probable 7.9% of fecundity in bull trout when exposed at the proposed criterion."
>
> "In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for nickel, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for nickel, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute toxic effects, but may not suffer chronic toxic effects."
>
> "Several studies have determined that mortality of salmonid embryos occurs over longer-term exposures to concentrations that are below the chronic criterion. For example, Birge *et al.* (1978) determined a 30-day LC50 for rainbow trout embryos of 50 µg/L at a water hardness between 93 mg/L and 105 mg/L. The corresponding lethal threshold (LC1) was estimated to be approximately 0.6 µg/L. Birge and Black (1980; as cited in Eisler 1998, hardness not reported) determined an LC10 of 11 µg/L for rainbow trout embryos exposed from fertilization through hatching. In Eisler's (1998b) review, LC50s were reported of 60 µg/L and 90 µg/L at water hardness of 125 and 174 mg/L, respectively, for rainbow trout embryos that were exposed from fertilization through hatching. These results and the review by Birge *et al.* (1981) suggest that adverse effects are likely to occur to embryos exposed to nickel concentrations that are lower than the proposed chronic criterion."

Swinomish Tribe Biological Evaluation

EPA's BE concluded that its proposed approval of the Swinomish Tribe's adoption of EPA's 1995 CWA recommendations for nickel is likely to adversely affect (LAA) for ESA listed species in Washington through indirect effects (USEPA, 2022a).

Criteria Calculations

Freshwater Acute Nickel Criterion

The data used to derive the freshwater acute nickel criterion is presented in Table 34. New studies that met data acceptability requirements are presented in Table 35. Studies used in previous EPA derivations but not used in this derivation are found in Table 36. The proposed freshwater acute criterion for nickel was derived using 28 GMAVs. Calculation results are as follows:

FAV = 38.17 (hardness of 50 mg/L)

CMC = 19.09 µg/L (hardness of 50 mg/L)

CMC = $e^{(0.846 \times \ln(\text{hardness}) - 0.3604)} \times CF$

Where CF (conversion factor from total to dissolved fraction) = 0.998

Acute criterion (total) = 34.31 µg/L (hardness of 100 mg/L)

Acute criterion (dissolved) = 34 µg/L (hardness of 100 mg/L; rounded to two significant digits)

Table 34. Freshwater acute toxicity data used for criteria derivation reported as total recoverable nickel.

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
1	29.05	Leptoxis ampla	29.05
2	58.32	Ceriodaphnia dubia	58.32
3	81.94	Neocloeon triangulifer	81.94
4	264.9	Somatogyrus sp.	264.9
5	275.5	Hamiota perovalis	275.5
6	335.6	Tubifex tubifex	335.6
7	385.4	Hyalella azteca	385.4
8	416	Physa gyrina	416
9	448.9	Villosa nebulosa	448.9
10	1089	Daphnia publicaria Daphnia magna Daphnia pulex	2042 1033 612
11	4312	Ambloplites rupestris	4312
12	4636	Ephemerella subvaria	4636
13	6163	Danio rerio	6163
14	6707	Pimephales promelas	6707
15	8697	Morone americana	12790

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
		Morone saxatilis	5914
16	12180	Anguilla rostrata	12180
17	12548	Oncorhynchus mykiss	12548
18	12756	Lepomis gibbosus	12756
19	12770	Amnicola sp.	12770
20	13000	Gammarus sp.	13000
21	14100	Nais sp.	14100
22	21200	Damselfly (unidentified sp.)	21200
23	30200	Caddisfly (unidentified sp.)	30200
24	40460	Acroneuria lycorias	40460
25	43231	Chironomus riparius Chironomus dilutes	32800 56979
26	43250	Fundulus diaphanus	43250
27	53915	Gambusia affinis	53915
28	66100	Crangonyx pseudogracilis	66100

* Normalized to hardness of 50 mg/L

Table 35. New freshwater acute studies that met data acceptability requirements since EPA last updated nickel criteria (S = static, R = static renewal, U = unmeasured test concentrations, M = measured test concentrations).

Species	Method	LC50 (µg/L total nickel)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Pimephales promelas	R, M	3928	106	2080	No. Other studies with the same species used flow-through design.	Lynch et al. 2016
Neocloeon triangulifer	S, M	147	100	81.94	Yes.	Soucek et al. 2020
Hyalella azteca	S, M	917.8	100	510.6	Yes.	Wang et al. 2020
Hyalella azteca	S, M	75.15	18	208.7	Yes.	Borgman et al. 2005
Hyalella azteca	S, M	133.3	124	53.73	Yes.	Borgman et al. 2005
Oncorhynchus mykiss	FT, M	20842	91	9724	Yes.	Brix et al. 2004
Tubifex tubifex	S, U	537	80	335.6	Yes.	Fargasova 1999
Ceriodaphnia dubia	S, M	81.16	50	81.16	Yes.	Keithly et al. 2004
Ceriodaphnia dubia	S, M	148.3	113	65.62	Yes.	Keithly et al. 2004
Ceriodaphnia dubia	S, M	261.5	161	81.22	Yes.	Keithly et al. 2004
Ceriodaphnia dubia	S, M	400.8	253	79.21	Yes.	Keithly et al. 2004
Hyalella azteca	S, M	3051	98	1557	Yes.	Keithly et al. 2004
Ceriodaphnia dubia	S, U	29.3	80	19.69	Yes.	Hockett et al. 1996
Hyalella azteca	S, M	2000	120	953.6	Yes.	Liber et al. 2011
Chironomus dilutus	S, M	119500	120	56978	Yes.	Liber et al. 2011

Species	Method	LC50 (µg/L total nickel)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Chironomus riparius	S, M	79500	367.8	14696	Yes.	Powlesland 1986
Gambusia affinis	S, U	68000	60	53915	Yes.	Kallangoudar & Patil 1997
Oncorhynchus mykiss	R, U	1280	250	256	No. Other studies with same species used flow through design and measured concentrations.	Kazlausk et al. 1994
Danio rerio	R, M	13146	141	5469	Yes.	Alsop et al. 2011
Danio rerio	R, M	16694	141	6945	Yes.	Alsop et al. 2013
Danio rerio	R, M	>10000	142	>4132	No. Greater than value when other more definitive data exists.	Griffitt 2008
Daphnia pulex	R, M	1480	142	612	Yes.	Griffitt 2008
Ceriodaphnia dubia	R, M	19640	142		No. LC50 10x higher than other studies using the same species.	Griffitt 2008
Daphnia magna	S, M	1503	92.5	893.2	Yes.	Lari et al. 2017
Hamiota perovalis	R, U	313	43	275.5	Yes.	Gibson et al. 2018
Villosa nebulosa	R, U	510	43	448.9	Yes.	Gibson et al. 2018
Leptoxis ampla	R, U	33	43	29.05	Yes.	Gibson et al. 2018
Somatogyrus sp.	R, U	301	43	264.9	Yes.	Gibson et al. 2018

* Normalized to a hardness of 50 mg/L

Table 36. Freshwater acute studies not used from previous EPA criteria derivations.

Species	SMAV *(µg/L)	Reason	Reference
Poecilia reticulata	9661	Non-North American species	USEPA, 1996
Carassius auratus	21320	Non-North American species	USEPA, 1996
Cyprinus carpio	9839	Non-North American species	USEPA, 1996

Freshwater Chronic Nickel Criterion

There was inadequate freshwater chronic nickel data to calculate criteria using the eight-family method. The FACR of 17.99 was previously used to calculate the freshwater chronic nickel criterion as presented in 1995 updates to aquatic life (USEPA, 1996). Additional chronic nickel ACRs were available since EPA's last update. The newly calculated FACR used to derive the chronic nickel criterion is 12.29 (Table 37). Calculation results are as follows:

FAV = 38.17 (hardness of 50 mg/L)

FACR = 12.29

CCC = 3.106 (hardness of 50 mg/L)

$CCC = e^{(0.846 \times \ln(hardness) - 2.176)} \times CF$

Where CF (conversion factor from total to dissolved fraction) = 0.997

Chronic criterion (total) = 5.584 ug/L (hardness of 100 mg/L)

Chronic criterion (dissolved) = 5.6 ug/L (hardness of 100 mg/L; rounded to two significant digits)

Table 37. Acute to chronic ratios (ACR) used in chronic criterion derivation.

Species	Acute Value (ug/L)	Chronic Value (ug/L)	ACR*	Species Mean ACR	Reference
Daphnia magna	1800	14.77	122.4*		EPA 1986 doc
Daphnia magna	1920	123.1	15.60		EPA 1986 doc
Daphnia magna	4970	356.6	13.94	14.75	EPA 1986 doc
Pimephales promelas	27930	526.1	53.03		EPA 1986 doc
Pimephales promelas	5186	217.3	23.87	35.60	EPA 1986 doc
Mysidopsis bahia	508	92.74	5.478	5.478	EPA 1986 doc
Ceriodaphnia dubia	7.2	148	20.56		Keithly et al. 2004

Species	Acute Value (ug/L)	Chronic Value (ug/L)	ACR*	Species Mean ACR	Reference
Ceriodaphnia dubia	4.2	261	62.14		Keithly et al. 2004
Ceriodaphnia dubia	7.5	400	53.33	40.84	Keithly et al. 2004
Neocloeon triangulifer	8	147	18.38	18.38	Soucek et al. 2020
Bufo terrestris	0.77	2.984	3.875	3.875	Fort et al. 2006
Gastrophyne carolinesis	0.1131	1.149	10.16	10.16	Fort et al. 2006
Geometric mean				12.29	

* Not used because varies significantly from other ACRs of the same species

Saltwater Acute and Chronic Nickel Criteria

No changes are proposed to the saltwater acute and chronic nickel criteria. Washington's current saltwater nickel criteria are identical to EPA recommendations, and there are no known ESA consultation issues in other Region 10 states.

Selenium

Summary of Criteria Recommendations and Changes

EPA updated their freshwater selenium criteria in 2016 that includes both fish tissue and water column elements (Table 38; USEPA, 2016b). Washington's current selenium criteria are based on water column only exposures. EPA's updated criteria are based on chronic exposure to selenium and are intended to protect the entire aquatic community. The new freshwater selenium criteria are based on levels of hierarchy by which particular types of fish tissue has precedent over other types of fish tissue, and fish tissue supersedes water column concentrations under steady state conditions. Further discussion on assumptions related to steady-state conditions are in the rulemaking Draft Implementation Plan[6].

We propose to adopt EPA recommendations for freshwater selenium criteria and make no changes to Washington's saltwater acute and chronic selenium criteria (Table 38). We made slight modifications to the EPA recommended footnotes for the selenium freshwater criteria but they are conceptually similar. We are not aware of endangered species concerns for Washington's ESA-listed species related to EPA recommended criteria for selenium.

[6] https://fortress.wa.gov/ecy/publications/summarypages/2410008.html

Table 38. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic selenium criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute	FW Chronic	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	20 µg/L	5 µg/L	290	71
EPA	15.1 mg/kg dry weight (egg-ovary)[1,2] 8.5 mg/kg dry weight (whole-body)[1,3] 11.3 mg/kg dry weight (muscle)[1,3] 1.5 µg/L (lentic)[4] 3.1 µg/L (lotic)[4] $WQC_{int} = WQC_{30\text{-}day} - C_{bkgrnd} (1 - f_{int}) / f_{int}$ [4,5]		290	71
Proposed	15.1 mg/kg dry weight (egg-ovary)[1] 8.5 mg/kg dry weight (whole-body)[2] 11.3 mg/kg dry weight (muscle)[2] 1.5 µg/L (lentic)[3] 3.1 µg/L (lotic)[3] $WQC_{int} = WQC - C_{bkgrnd} (1 - f_{int}) / f_{int}$ [3,4]		No change	No change

[1] Egg-ovary supersedes any whole-body, muscle, or water column element when fish egg-ovary concentrations are measured, except as noted in footnote 3. Tissue criterion is not to be exceeded.

[2] Fish whole-body or muscle tissue supersedes the water column element when both fish tissue and water concentrations are measured, except as noted in footnote 3. Tissue criterion is not to be exceeded.

[3] Water column values are based on dissolved total selenium in water and are derived from fish tissue values via bioaccumulation modeling. When selenium inputs are increasing, water column values are the applicable criterion element in the absence of steady-state condition fish tissue data. Water column criteria are based on a 30-day average concentrations, except for WQC_{int} (see footnote 4). Water column criteria are not to be exceeded more than once every three years on average.

[4] Where WQC_{int} is the intermittent exposure concentration in µg/L; WQC is the applicable water column element, for either lentic or lotic waters; C_{bkgrnd} is the average daily background concentration occurring during the remaining time, integrated over 30 days; f_{int} is the fraction of any 30-day period during which elevated selenium concentrations occur, with f_{int} assigned a value ≥ 0.033 (corresponding to one day). Intermittent exposure criteria averaging period is the number of days per month with an elevated concentration.

Silver

Summary of Criteria Recommendations and Changes

The proposed freshwater silver criteria accounts for endangered species protection levels by incorporating new science available since EPA last updated the criteria in 1980 (Table 39; USEPA, 1980). The proposed freshwater acute silver criterion is more stringent than EPA recommendations. EPA does not have a recommendation for a freshwater chronic silver criterion, but during our review of new science, we found adequate data available to calculate a

chronic criterion. We updated the saltwater acute silver criterion in order to calculate a saltwater chronic criterion using the newly established FACR.

Table 39. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic silver criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	3.4*^ (1-hour)	-	1.9^ (instantaneous)	-
EPA	3.2*^ (instantaneous)	-	1.9^ (instantaneous)	-
Proposed	0.52*^ (1-hour)	0.21*^ (4-day)	2.2 (1-hour)	0.87 (4-day)

* Hardness based criteria (numeric value shown based on 100 mg/L)
^ Presented as the dissolved fraction

Endangered Species Act Consultation

Oregon

There were likely to adversely affect (LAA) determinations for the silver freshwater acute (3.2 µg/L at 100 mg/L hardness) and chronic (0.10 µg/L at 100 mg/L hardness) criteria in Oregon (USFWS, 2012) for bull trout, a species that is also on Washington's endangered species list. There was no jeopardy call. The Oregon BiOps specifically state:

> "The available evidence for silver indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (moderate intensity), and sublethal effects (moderate intensity)."
>
> "Since the proposed acute standard is 72% less than the LC10 acute concentration for silver, we conclude that while some adverse effects may occur to the bull trout, these effects are likely to be sub-lethal and not cause a significant disruption of breeding, feeding, migrating, or sheltering behavior during each 3-year period during the 25-year term of the proposed action."
>
> "We conclude that bull trout exposure to the proposed chronic criterion concentration of silver is likely to cause mortality of 263 adult bull trout during each 3-year period over the 25-year term of the proposed action, and injure another 1,371 individual adult bull due to reduced growth and fitness each 3-year period over the 25-year term of the proposed action in surface waters along 820.6 miles of bull trout habitat within the action area."

Criteria Calculations

Freshwater Acute Silver Criterion

The data used to derive the freshwater acute silver criterion is presented in Table 40. New studies that met data acceptability requirements are presented in Table 41. Studies used in

previous EPA derivations but not used in this derivation are found in Table 42. The proposed freshwater acute criterion for silver was derived using 20 GMAVs. Calculation results are as follows:

FAV = 0.3686 (hardness of 50 mg/L)

CMC = 0.1843 µg/L (hardness of 50 mg/L)

CMC = $e^{(1.72 \times \ln(\text{hardness}) - 8.420)}$ x CF

Where CF (conversion factor from total to dissolved fraction) = 0.85

Acute criterion (total) = 0.6071 µg/L (hardness of 100 mg/L)

Acute criterion (dissolved) = 0.52 µg/L (hardness of 100 mg/L; rounded to two significant digits)

Table 40. Freshwater acute toxicity data used for criteria derivation reported as total recoverable silver.

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
1	0.3620	Ceriodaphnia dubia	0.3620
2	0.7840	Daphnia magna	0.7840
3	2.565	Danio rerio	2.565
4	2.930	Hyalella azteca	2.930
5	3.222	Rhinichthys oscuius	3.222
6	3.351	Cottus bairdi	3.351
7	5.390	Gammarus pseudolimnaeus	5.390
8	7.421	Pimephales promelas	7.421
9	8.772	Oncorhynchus mykiss	8.772
10	10.66	Jordanella floridae	10.66
11	10.84	Leptophlebia sp.	10.84
12	18.32	Ictalurus punctatus	18.32
13	29	Hydra sp.	29
14	63.29	Nephelopsis obscura	63.29
15	93.94	Lepomis macrochirus	93.94
16	122.6	Lepomis macrochirus	122.6
17	241	Aplexa hypnorum	241
18	379.0	Chironomus tentans	379.0
19	3788	Tanytarsus dissimiliis	3788
20	4612	Philodina acuticornis	4612

* Normalized to 50 mg/L hardness

Table 41. New freshwater acute studies that met data acceptability requirements since EPA last updated silver criteria (S = static, R = static renewal, FT = flow-through, U = unmeasured test concentrations, M = measured test concentrations).

Species	Method	LC50 (µg/L total silver)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Daphnia magna	R, M	0.26	150	0.039	Yes.	Bianchini et al. 2002
Daphnia magna	R, M	0.18	115	0.043	Yes.	Bianchini et al. 2002
Ceriodaphnia dubia	R, M	0.5	90	0.18	Yes.	Bielmyer et al. 2002
Pimephales promelas	FT, M	30	103	8.66	Yes.	Birge et al. 1984
Pimephales promelas	R, M	7.8	48	8.37	No. Other studies with the same species used flow through design.	Erickson et al. 1998
Daphnia magna	S, M	0.58	49	0.60	Yes.	Erickson et al. 1998
Hydra sp.	S, M	29	50	29	Yes.	Brooke et al. 1986
Nephelopsis obscura	S, M	59	48	63.29	Yes.	Brooke et al. 1986
Leptophlebia sp.	S, M	8.7	44	10.84	Yes.	Brooke et al. 1986
Pimephales promelas	R, M	5.412	80	2.411	No. Other studies with the same species used flow through design.	Diamond et al. 1997
Pimephales promelas	R, M	8.471	80	3.774	No. Other studies with the same species used flow through design.	Diamond et al. 1997
Pimephales promelas	R, M	7.882	80	3.512	No. Other studies with the same species used flow through design.	Diamond et al. 1997

Species	Method	LC50 (µg/L total silver)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Pimephales promelas	R, M	5.294	80	2.359	No. Other studies with the same species used flow through design.	Diamond et al. 1997
Ceriodaphnia dubia	R, M	1.294	80	4.263	No. LC50 is 10x other studies with the same species.	Diamond et al. 1997
Ceriodaphnia dubia	R, M	1.294	80	4.263	No. LC50 is 10x other studies with the same species.	Diamond et al. 1997
Ceriodaphnia dubia	R, M	1.059	80	3.488	No. LC50 is 10x other studies with the same species.	Diamond et al. 1997
Ceriodaphnia dubia	R, M	0.8235	80	2.713	No. LC50 is 10x other studies with the same species.	Diamond et al. 1997
Pimephales promelas	S, M	2.43	50	2.43	No. Other studies with the same species used flow through design.	Karen et al. 1999
Pimephales promelas	S, M	2.24	100	0.68	No. Other studies with the same species used flow through design.	Karen et al. 1999
Pimephales promelas	S, M	2.79	200	0.26	No. Other studies with the same species used flow through design.	Karen et al. 1999
Daphnia magna	R, M	0.844	100	0.26	Yes.	Karen et al. 1999
Daphnia magna	R, M	1.009	200	0.31	Yes.	Karen et al. 1999

Species	Method	LC50 (µg/L total silver)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Pimephales promelas	FT, M	16	38	25.65	Yes.	LeBlanc et al. 1984
Oncorhynchus mykiss	R, M	14.7	140	2.501	No. Other studies with same species used flow through design.	Mann et al. 2004
Oncorhynchus mykiss	FT, M	13.3	130	2.571	Yes.	Morgan and Wood, 2004
Ceriodaphnia dubia	S, M	0.92	70	0.52	Yes.	Rodgers et al. 1997
Daphnia magna	S, M	1.06	70	0.59	Yes.	Rodgers et al. 1997
Hyalella azteca	S, M	6.8	70	3.81	Yes.	Rodgers et al. 1997
Chironomus tentans	S, M	676	70	388.0	Yes.	Rodgers et al. 1997
Pimephales promelas	S, M	11.6	70	6.5	No. Other studies with same species used flow through design.	Rodgers et al. 1997
Danio rerio	R, M	15.18	141	2.565	Yes.	Alsop et al. 2011
Lepomis macrochirus		60	33	122.6	Yes.	Buccafusco 1987
Pimephales promelas	FT, M	6.7	44	8.35	Yes.	Holcombe et al. 1983
Ictalurus punctatus	FT, M	17.3	44	21.55	Yes.	Holcombe et al. 1983
Aplexa hypnorum	S, M	241	50	241	Yes.	Holcombe et al. 1983

Species	Method	LC50 (µg/L total silver)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Daphnia magna	S, U	1.5	72	0.8	No. Other studies with same species measured chemical concentrations.	Leblanc 1980
Pimephales promelas	FT, M	10.7	45	12.83	Yes.	Lima 1982
Jordanella floridae	FT, M	9.2	45	11.03	Yes.	Lima 1982
Gammarus pseudolimnaeus	FT, M	4.5	45	5.39	Yes.	Lima 1982
Tanytarsus dissimiliis	FT, M	3160	45	3788	Yes.	Lima 1982
Pimephales promelas	FT, M	9	45	10.78	Yes.	Holcombe et al. 1987
Oncorhynchus mykiss	FT, M	6	45	7.19	Yes.	Holcombe et al. 1987
Ictalurus punctatus	FT, M	13	45	15.58	Yes.	Holcombe et al. 1987
Pimephales promelas	S, M	2.43	50	2.43	No. Other studies with same species used flow through design.	Forsythe 1996
Pimephales promelas	S, M	2.24	100	0.6799	No. Other studies with same species used flow through design.	Forsythe 1996
Pimephales promelas	S, M	2.79	200	0.2571	No. Other studies with same species used flow through design.	Forsythe 1996

Species	Method	LC50 (µg/L total silver)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Daphnia magna	S, U	10	240	0.673	No. Other studies with same species measured chemical concentrations.	Khangarot 1987
Pimephales promelas	FT, M	5.1	53	4.614	Yes.	Brooke et al. 1993
Hyalella azteca	FT, M	2.1	48	2.253	Yes.	Brooke et al. 1993
Ceriodaphnia dubia	S, M	0.4	88	0.1513	Yes.	Brooke et al. 1993
Oncorhynchus mykiss	FT, M	7.6	120	1.686	Yes.	Galvez et al. 2002

* Normalized to 50 mg/L hardness

Table 42. Freshwater acute studies not used from previous EPA criteria derivations.

Species	SMAV *(µg/L)	Reason	Reference
Gammarus pseudolimnaeus	4827	Repeat of Lima 1982 publication used in current derivation.	USEPA, 1980
Tanytarsus dissimiliis	3433	Repeat of Lima 1982 publication used in current derivation.	USEPA, 1980
Daphnia magna	1.733	LC50 10x higher than other studies using the same species.	USEPA, 1980

Freshwater Chronic Silver Criterion

EPA has not developed a freshwater chronic silver criterion, and the silver criterion has not been updated since 1980. We applied 1985 EPA derivation methods to calculate a silver criterion. There was not adequate toxicity data to calculate a chronic silver criterion using the eight-family method, and therefore, we applied a FACR to the FAV to calculate a criterion. The calculated FACR for silver is 5.028 (Table 43). Table 44 shows the ACR studies that met test acceptability requirements but were not used. Calculation results are as follows:

FAV = 0.3686 (hardness of 50 mg/L)

FACR = 5.028

CCC = 0.0733 (hardness of 50 mg/L)

$CCC = e^{(1.72 \times \ln(hardness) - 9.342)} \times CF$

Where CF (conversion factor from total to dissolved fraction) = 0.85

Chronic criterion (total) = 0.2414 µg/L (hardness of 100 mg/L)

Chronic criterion (dissolved) = 0.21 µg/L (hardness of 100 mg/L; rounded to two significant digits)

Table 43. Acute to chronic ratios (ACR) used in chronic criterion derivation.

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
Daphnia magna	43	22	2		USEPA, 1980
Daphnia magna	0.81	0.45	1.8		Kolkmeier and Brooks, 2013
Daphnia magna	2.12	6.88	3.25	2.27	Bianchini, 2008
Mysidopsis bahia	250	18	14	14	USEPA, 1980
Oncorhynchus mykiss	6.5	1.624	4.00	4	Davies, 1978
Geometric mean				5.028	

* Geometric mean of ACRs were calculated for similar species preceding the final acute chronic ratio calculation

Table 44. Studies with acute to chronic ratios (ACR) that met test acceptability requirements but were not used in the chronic criterion derivation.

Species	ACR	Reason	Reference
Oncorhynchus mykiss	54	ACR was 10X greater than other study using the same species.	USEPA, 1980
Ceriodaphnia dubia	158	ACR was 10x greater than the lowest ACR for a given species.	Bielmyer et al. 2002

Saltwater Acute Silver Criterion

EPA recommends a saltwater acute silver criterion of 1.9 µg/L with an instantaneous duration (Table 39). EPA recommendations for the saltwater acute silver criterion is based on pre-1985 EPA methods for deriving aquatic life toxics criteria. We used the data from EPA's 1980 document and any new science to recalculate the acute silver criterion using EPA's 1985 guidance. An evaluation of the saltwater acute silver criteria was done to align freshwater and saltwater averaging periods as well as use the latest science to derive a saltwater chronic silver criteria using the newly established FACR. Using EPA's 1985 methodology, we calculated a saltwater acute silver criterion of 2.2 µg/L using 17 GMAVs (Table 45). New studies since EPA last updated the saltwater acute silver criterion are found in Table 46. Calculation results are as follows:

FAV = 5.171

CMC = 2.586

Where CF (conversion factor from total to dissolved fraction) = 0.85

Acute criterion (total) = 2.586 µg/L

Acute criterion (dissolved) = 2.2 µg/L (rounded to 2 significant digits)

Table 45. Saltwater acute toxicity data used for criteria derivation reported as total recoverable silver.

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
1	4.7	Paralichthys dentatus	4.7
2	18.97	Strongylocentrotus purpuratus Stronglyocentrotus droebachiensis	15 24
3	20	Crassostrea virginica	20
4	21	Mercenaria mercenaria	21
5	24	Tigriopus japonicus	24
5	33	Argopecten irradians	33
6	33	Dendraster excentricus	33
7	33	Cancer magister	33

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
8	36	Acartia tonsa	36
9	77.41	Brachionus plicatilis	77.41
10	210	Menidia menidia	210
11	210.8	Mysidopsis bahia	210.8
12	331	Oligocottus maculosus	331
13		Oncorhynchus mykiss	404.5
14	500	Pseudopieuronectes americanus	500
15	550	Apeltes quadracus	550
16	1400	Cyprinodon variegatus	1400
17	2250	Opsanus beta	2250

Table 46. New saltwater acute studies that met data acceptability requirements since EPA last updated silver criteria (S = static, R = static renewal, FT = flow-through, U = unmeasured test concentrations, M = measured test concentrations).

Species	Method	LC50 (µg/L total silver)	Used in Derivation?	Reference
Oncorhynchus mykiss	S, U	404.5	Yes.	Ferguson and Hogstrand, 1998
Stronglyocentrotus droebachiensis	S, M	24	Yes.	Dinnel et al. 1989
Strongylocentrotus purpuratus	S, M	15	Yes.	Dinnel et al. 1989
Dendraster excentricus	S, M	33	Yes.	Dinnel et al. 1989
Cancer magister	S, M	33	Yes.	Dinnel et al. 1989
Brachionus plicatilis	S, M	77.41	Yes.	Saunders, 2012
Oligocottus maculosus	R, M	331	Yes.	Shaw et al. 1998
Mysidopsis bahia	FT, M	305.9	Yes.	Ward and Kramer, 2002
Opsanus beta	R, M	2250	Yes.	Wood et al. 2004
Mysidopsis bahia	FT, M	141	Yes.	McKenney, 1982
Mysidopsis bahia	FT, M	300	Yes.	McKenney, 1982
Mysidopsis bahia	FT, M	300	Yes.	McKenney, 1982
Mysidopsis bahia	FT, M	64	Yes.	McKenney, 1982
Mysidopsis bahia	FT, M	298	Yes.	McKenney, 1982
Tigriopus japonicus	R, U	24	Yes.	Lee et al. 2008

Saltwater Chronic Silver Criterion

EPA has not developed a saltwater chronic silver criterion. We applied 1985 EPA derivation methods to calculate a silver criterion. There was not adequate toxicity data to calculate a chronic silver criterion using the eight-family method, and therefore, we applied a FACR to the FAV to calculate a criterion. The calculated FACR for silver is 5.028 (Table 43). Calculation results are as follows:

FAV = 5.171

FACR = 5.028

CCC = FAV / FACR = 1.028

Where CF (conversion factor from total to dissolved fraction) = 0.85

Chronic criterion (total) = 1.028 µg/L

Chronic criterion (dissolved) = 0.87 µg/L (rounded to 2 significant digits)

Zinc

Summary of Criteria Recommendations and Changes

The proposed freshwater zinc criteria accounts for endangered species protection levels by incorporating new science available since EPA last updated the freshwater criteria in 1995. The proposed freshwater zinc criteria are more stringent than EPA recommendations (Table 47; USEPA, 1996). No changes were necessary for saltwater criteria because Washington's saltwater zinc criteria are identical to EPA recommendations and there are no endangered species protection issues highlighted in previous ESA consultations in other Region 10 states.

Table 47. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic zinc criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	114*^ (1-hour)	105*^ (4-day)	90^ (1-hour)	81^ (4-day)
EPA	120*^ (1-hour)	120*^ (4-day)	90^	81^
Proposed	57*^ (1-hour)	39^ (4-day)	No change	No change

* Hardness based criteria (numeric value shown based on 100 mg/L)

^ Presented as the dissolved fraction

Endangered Species Act Consultation

Oregon

There were likely to adversely affect designations for the zinc freshwater acute (120 µg/L at 100 mg/L hardness) and chronic (120 µg/L at 100 mg/L hardness) criteria in Oregon for bull trout, a species that is also on Washington's endangered species list. There were no jeopardy calls. The Oregon BiOps specifically state (NMFS, 2012; USFWS, 2012):

> "Bull trout exposure to zinc at the proposed acute criterion is likely to result in the mortality of up to 507 adult bull trout in surface waters along 820.6 miles of habitat within the action area over each 3-year period during the 25-year term of the proposed action."
>
> "Bull trout exposure to zinc at the proposed chronic criterion is likely to kill up to 266 adult bull trout, and injure (via reduced fitness) up to another 1,370 individual adult bull trout during each- 3 year period over the 25 year term of the proposed action in 820.6 miles of bull trout habitat within the action area."
>
> "The available evidence for zinc indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (moderately-high-intensity), cellular trauma (moderate intensity), physiological trauma (moderate intensity), and reproductive failure (moderately-high-intensity)."

Idaho

There were jeopardy calls for the zinc freshwater acute (120 µg/L at 100 mg/L hardness) and chronic (120 µg/L at 100 mg/L hardness) criteria in Idaho (NMFS, 2014; USFWS, 2015) for species (i.e., bull trout and white sturgeon) relevant to Washington. The Idaho BiOp specifically states:

> "For that reason, zinc concentrations at the proposed acute and chronic criteria level are likely to impair the capability of bull trout habitat to provide for the normal reproduction, growth, and survival of bull trout. Given that the state of Idaho represents 44 percent of streams and 34 percent of lakes and reservoirs occupied by the bull trout within its range, the above effects are considered to be significant and are likely to impede (1) maintaining/ increasing the current distribution of the bull trout, (2) maintaining/increasing the current abundance of the bull trout, and (3) achieving stable/increasing trends in bull trout populations within a significant portion of its range."
>
> "The proposed zinc criteria are likely to impair water quality (PCE 8) by allowing aquatic zinc concentrations to rise to levels that have been shown to be lethal to juvenile bull trout throughout the range of bull trout critical habitat in Idaho. For that reason, zinc concentrations at the proposed acute and chronic criteria level would impair the capability of the critical habitat to provide for the normal reproduction, growth, and survival of bull trout."
>
> "Given that existing data show adverse effects to multiple freshwater fish species, including potential prey species of the Kootenai River white sturgeon, at zinc concentrations below the proposed criteria, and given the likelihood that zinc concentrations will be even higher

in sediments, thus increasing adverse impacts to white sturgeon eggs and juveniles, we conclude the proposed criteria for zinc are likely to have significant adverse effects (in the form of reduced growth and survival) to the Kootenai River white sturgeon throughout its range in Idaho, which represents 39 percent of its range. Such impacts are likely to impede natural reproduction of the Kootenai River white sturgeon and the maintenance or increase of the wild population."

"Because the proposed water quality criteria would be implemented statewide, all of the designated white sturgeon critical habitat would be subjected to aquatic zinc concentrations up to 117 µg/L (acute) and 118 µg/L (chronic) at a water hardness value of 100 mg/L, in addition to unknown and unregulated concentrations in sediment. Thus, the proposed acute and chronic zinc criteria are likely to adversely affect sediment and water quality in 100 percent of the critical habitat within the distinct population segment and is reasonably certain to impair the ability of critical habitat to provide for the normal behavior, reproduction, and survival of white sturgeon."

Swinomish Tribe Biological Evaluation

EPA's biological evaluation concluded likely to adversely affect (LAA) determinations for the freshwater zinc acute and chronic criteria to ESA listed species in Washington (USEPA, 2022a).

Criteria Calculations

Freshwater Acute Zinc Criterion

The data used to derive the freshwater acute zinc criterion is presented in Table 48. New studies that met data acceptability requirements are presented in Table 49. Studies used in previous EPA derivations but not used in this derivation are found in Table 50. The proposed freshwater acute criterion for zinc was derived using 64 GMAVs. Calculation results are as follows:

FAV = 64.34 (hardness of 50 mg/L)

CMC = 32.17 µg/L (hardness of 50 mg/L)

CMC = $e^{(0.8473 \times \ln(\text{hardness}) + 0.1564)} \times CF$

Where CF (conversion factor from total to dissolved fraction) = 0.978

Acute criterion (total) = 58 µg/L (hardness of 100 mg/L)

Acute criterion (dissolved) = 57 µg/L (hardness of 100 mg/L; rounded to two significant digits)

Table 48. Freshwater acute toxicity data used for criteria derivation reported as total recoverable zinc.

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
1	40.24	Neocloeon triangulifer	40.24
2	51.96	Hyalella azteca	51.96
3	61.38	Euchlanis dilatata	61.38
4	72.10	Ceriodaphnia dubia	102.5

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
		Ceriodaphnia reticulata	50.7
5	76.13	Leptoxis ampla	76.13
6	102.3	Limnodrilus hoffmeisteri	102.3
7	102.5	Acipenser transmontanus	102.5
8	119.4	Morone saxatilis	119.4
9	175.7	Cottus bairdi	175.7
10	176.5	Lampsilis rafinesqueana	171.6
		Lampsilis siliquoidea	181.7
11	227.8	Agosia chrysogaster	227.8
12	255.4	Pomacea paludosa	255.4
13	303.1	Daphnia magna	232.5
		Daphnia pulex	252.9
		Daphnia carinata	188.9
		Daphnia prolata	759.7
14	344.6	Bryocamptus zschokkei	344.6
15	373.8	Somatogyrus sp.	373.8
16	474.3	Prosopium williamsoni	474.3
17	750.1	Oncorhynchus mykiss	623.7
		Oncorhynchus kisutch	1628
		Oncorhynchus nerka	1502
		Oncorhynchus tshawytscha	446.4
		Oncorhynchus clarkii	348.8
18	772.3	Anaxyrus boreas boreas	772.3
19	856.0	Villosa umbrans Villosa nebulosa	1479 495.4
20	863.0	Pimephales promelas	863.0
21	1224	Salvelinus fontinalis	1224
22	>1257	Limnodrilus hoffmeisteri	>1257
23	1307	Pectinatella magnifica	1307
24	1353	Physa gyrina	1683
25	1088	Physa heterostropha	1088
26	1370	Salmo salar	2176
		Salmo trutta	862.9
27	1578	Helisoma campanulatum	1578
28	1607	Plumatella emarginata	1607
29	1672	Jordanella floridae	1672
30	1707	Lophopodella carteri	1707
31	1746	Catostomus latipinnis Catostomus commersoni	583.4 5228
32	1769	Drunella grandis	1769

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
33	1913	Atyaephyra desmarestii	1913
34	1946	Rhinichthys cataractae	1946
35	2136	Xyrauchen texanus	2136
36	2545	Platygobio gracilis	2545
37	2791	Gila elegans	2791
38	2836	Ptychocheilus lucius	1222
		Ptychocheilus oregonesis	6580
39	2933	Hydra viridissima Hydra vulgaris Hydra oligactis	1719 3537 4150
40	3265	Lirceus alabamae	3265
41	3506	Chironomus riparius	3506
42	4341	Xiphophorus maculatus	4341
43	4900	Corbicula fluminea	4900
44	5135	Hyla chrysocelis	5135
45	5588	Tubifex tubifex	5588
46	6000	Notemigonus crysoleucas	6000
47	6315	Nais elinguis	2167
		Nais sp.	18400
48	8100	Gammarus sp.	8100
49	8157	Asellus bicrenate	5731
		Asellus communis	11610
50	8483	Lepomis gibbosus	18790
		Lepomis macrochirus	3830
51	9712	Lumbriculus variegatus	9712
52	11305	Rana pipiens	11305
53	11899	Baetis tricaudatus	11899
54	13630	Anguilla rostrata	13630
55	16820	Amnicola sp.	16820
56	17940	Fundulus diaphanous	17940
57	19800	Crangonyx pseudogracilis	19800
58	21608	Branchiura sowerbyi	21608
59	21890	Chironomus plumosus	21890
60	>48500	Lepidostoma sp.	>48500
61	>67543	Chloroperlidae	>67543
62	>67543	Ephemerella sp.	>67543
63	69062	Cinygmula sp.	69062
64	88960	Argia sp.	88960

* Normalized to 50 mg/L hardness

Table 49. New freshwater acute studies that met data acceptability requirements since EPA last updated zinc criteria (S = static, R = static renewal, FT = flow-through, U = unmeasured test concentrations, M = measured test concentrations).

Species	Method	LC50 (µg/L total zinc)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Tubifex tubifex	S, U	11150	113	5588	Yes.	Chatterjee et al. 2019
Branchiura sowerbyi	S, U	51097	120	24335	Yes. 24-hour LC50.	Dhara et al. 2020
Pimephales promelas	S, M	839	110	429.4	No. Other studies using the same species had a flow through design.	Lynch et al. 2016
Daphnia magna	S, M	696	90	423	Yes.	Meyer et al. 2015
Daphnia magna	S, M	330	90	200.6	Yes.	Santos-Medrano & Rico-Martinez 2015
Daphnia prolata	S, M	1250	90	759.7	Yes.	Santos-Medrano & Rico-Martinez 2015
Lampsilis rafinesqueana	S, U	163	44	181.7	Yes.	Wang et al. 2010
Lampsilis siliquoidea	S, U	145	41	171.6	Yes.	Wang et al. 2010
Hyalella azteca	S, U	101.2	107	53.13	Yes.	Wang et al. 2020
Oncorhynchus mykiss	FT, M	162	20	352.1	Yes	Alsop et al. 1999
Oncorhynchus mykiss	FT, M	869	120	413.9	Yes	Alsop et al. 1999
Oncorhynchus mykiss	FT, M	103	10	402.8	Yes	Alsop and Wood, 1999
Oncorhynchus mykiss	FT, M	2615	120	1245	Yes.	Alsop and Wood, 2000
Daphnia magna	S, M	121	46	129.9	Yes.	Barata et al. 1998
Daphnia magna	R, M	1425	150	561.9	No. Concentrations were not measured.	Bianchini et al. 2002
Pimephales promelas	R, M	483.8	44.8	531.0	No. Other studies using the same species had a flow through design.	Bringolf et al. 2006

Species	Method	LC50 (µg/L total zinc)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Pimephales promelas	R, M	745.3	49.3	754.2	No. Other studies using the same species had a flow through design.	Bringolf et al. 2006
Pimephales promelas	R, M	876.1	61.4	736.2	No. Other studies using the same species had a flow through design.	Bringolf et al. 2006
Cottus bairdi	FT, U	439	154	169.2	Yes.	Brinkman & Woodling 2005
Rhithrogena hageni	FT, M	51636	45	56458	No. LC50 10x higher than other species within genus.	Brinkman & Johnston 2012
Oncorhynchus clarkii	FT, M	189.2	47.4	197.9	Yes. Combined with other Brinkman & Johnston 2012 values	Brinkman & Johnston 2012
Oncorhynchus clarkii	FT, M	1452	144	592.0	Yes. Combined with other Brinkman & Johnston 2012 values	Brinkman & Johnston 2012
Oncorhynchus clarkii stomias	FT, M	321.1	47.4	335.9	Yes. Combined with other Brinkman & Johnston 2012 values	Brinkman & Johnston 2012
Oncorhynchus clarkii stomias	FT, M	1534	144	624.7	Yes. Combined with other Brinkman & Johnston 2012 values	Brinkman & Johnston 2012
Oncorhynchus clarkii virginalis	FT, M	145.2	41.7	169.3	Yes. Combined with other Brinkman & Johnston 2012 values	Brinkman & Johnston 2012
Oncorhynchus clarkii virginalis	FT, M	1063	144	432.5	Yes. Combined with other Brinkman & Johnston 2012 values	Brinkman & Johnston 2012
Prosopium williamsoni	FT, M	365.0	43.2	413.2	Yes. Combined with other Brinkman & Johnston 2012 values	Brinkman & Johnston 2012

Species	Method	LC50 (µg/L total zinc)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Prosopium williamsoni	FT, M	437.6	41.1	516.4	Yes. Combined with other Brinkman & Johnston 2012 values	Brinkman & Johnston 2012
Prosopium williamsoni	FT, M	481.6	47.8	500	Yes. Combined with other Brinkman & Johnston 2012 values	Brinkman & Johnston 2012
Cottus bairdi	FT, M	338.4	99.5	188.9	Yes.	Brinkman & Johnston 2012
Rhinichthys cataractae	FT, M	1943	49.9	1946	Yes.	Brinkman & Johnston 2012
Platygobio gracilis	FT, M	2648	52.4	2545	Yes.	Brinkman & Johnston 2012
Anaxyrus boreas boreas	FT, M	863.0	57	772.3	Yes.	Brinkman & Johnston 2012
Baetis tricaudatus	FT, M	10327	42.3	11899	Yes.	Brinkman & Johnston 2012
Cinygmula sp.	FT, M	70348	51.1	69062	Yes.	Brinkman & Johnston 2012
Drunella doddsi	FT, M	>64000	49.8	>63783	No. LC50 10x higher than other species within genus and definitive values exist for this species.	Brinkman & Johnston 2012
Chloroperlidae	FT, M	>68800	51.1	>67543	Yes.	Brinkman & Johnston 2012
Ephemerella sp.	FT, M	>68800	51.1	>67543	Yes.	Brinkman & Johnston 2012
Lepidostoma sp.	S, M	>48500	50	>48500	Yes.	Brinkman & Johnston 2012
Bryocamptus zschokkei	R, M	620	100	344.6	Yes.	Brown et al. 2005
Ptychocheilus lucius	S, U	3340	199	1036	Yes.	Buhl 1996
Gila elegans	S, U	5350	199	1660	Yes.	Buhl 1996
Xyrauchen texanus	S, U	2920	199	906	Yes.	Buhl 1996
Acipenser transmontanus	FT, M	150	100	83.37	Yes.	Calfee et al. 2014

Species	Method	LC50 (µg/L total zinc)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Oncorhynchus mykiss	FT, M	233.0	100	129.5	Yes.	Calfee et al. 2014
Salmo trutta	FT, M	890.6	51.9	862.9	Yes.	Davies et al. 2000
Salvelinus fontinalis	FT, M	1109	84.2	713.4	Yes.	Davies et al. 2001
Ceriodaphnia dubia	R, M	119.3	80	80.13	Yes.	Diamond et al. 1997
Ceriodaphnia dubia	R, M	203.5	80	136.6	Yes.	Diamond et al. 1997
Ceriodaphnia dubia	R, M	186.7	80	125.4	Yes.	Diamond et al. 1997
Ceriodaphnia dubia	R, M	307.4	80	206.4	Yes.	Diamond et al. 1997
Pimephales promelas	R, M	387.0	80	259.9	No. Other studies using the same species had a flow through design.	Diamond et al. 1997
Pimephales promelas	R, M	296.8	80	199.3	No. Other studies using the same species had a flow through design.	Diamond et al. 1997
Pimephales promelas	R, M	100	80	67.15	No. Other studies using the same species had a flow through design.	Diamond et al. 1997
Pimephales promelas	R, M	380	80	255.1	No. Other studies using the same species had a flow through design.	Diamond et al. 1997
Acipenser transmontanus	R, M	153.4	76	107.6	Yes.	Vardy et al. 2014
Oncorhynchus mykiss	S, U	12800	250	3273	No. Other studies using the same species had a flow through design.	Gundogdu 2008
Pomacea paludosa	R, M	136.0	28	222.3	Yes.	Hoang & Tong 2015
Pomacea paludosa	R, M	371.2	97	211.7	Yes.	Hoang & Tong 2015

Species	Method	LC50 (µg/L total zinc)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Pomacea paludosa	R, M	462.2	103	250.5	Yes.	Hoang & Tong 2015
Pomacea paludosa	R, M	587.9	108	306.2	Yes.	Hoang & Tong 2015
Pomacea paludosa	R, M	1098	230	301.4	Yes.	Hoang & Tong 2015
Hydra vulgaris	S, M	7400	204	2248	Yes.	Karntanut & Pascoe 2000
Hydra vularis (Zurich)	S, M	14000	210	4150	Yes.	Karntanut & Pascoe 2002
Hydra vulgaris	S, M	13000	210	3854	Yes.	Karntanut & Pascoe 2002
Hydra oligactis	S, M	14000	210	4150	Yes.	Karntanut & Pascoe 2002
Hydra viridissima	S, M	11000	210	3261	Yes.	Karntanut & Pascoe 2002
Hydra viridissima	S, M	2500	207	750.2	Yes.	Karntanut & Pascoe 2002
Daphnia magna	R, U	157.5	105	83.98	Yes.	Lazorchak et al. 2009
Pimephales promelas	S, M	839.5	110	430.4	No. Other studies using the same species had a flow through design.	Lynch et al. 2016
Oncorhynchus mykiss	R, M	130	24	242.1	No. Other studies using the same species had a flow through design.	Mebane et al. 2008
Ceriodaphnia dubia	R, M	119	181	40.01	Yes.	Naddy et al. 2015
Oncorhynchus mykiss	S, M	304	181	102.2	No. Other studies using the same species had a flow through design.	Naddy et al. 2015
Atyaephyra desmarestii	S, M	7810	263	1913	Yes.	Pestana et al. 2007
Nais elinguis	R, M	912	18	2167	Yes.	Shuhaimi et al. 2012
Lepomis macrochirus	FT, M	4500	214	1313	Yes.	Van der Schalie et al. 2004
Cottus bairdi	FT, M	159.5	48.6	163.4	Yes.	Woodling et al. 2002

Species	Method	LC50 (µg/L total zinc)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Drunella grandis	FT, M	1352	36.4	1769	Yes.	Brinkman & Vieira 2008
Daphnia magna	S, M	173.5	100	96.44	Yes.	Cooper et al. 2009
Daphnia carinata	S, M	339.8	100	188.9	Yes.	Cooper et al. 2009
Chironomus plumosus	S, U	32600	80	21890	Yes.	Fargasova 2001
Daphnia magna	S, U	550	90	334.3	Yes.	Jellyman et al. 2011
Ptychocheilus lucius	S, U	1700	197	532.0	Yes.	Hamilton 1995
Xyrauchen texanus	S, U	8900	197	2785	Yes.	Hamilton 1995
Gila elegans	S, U	15000	197	4694	Yes.	Hamilton 1995
Ptychocheilus lucius	S, U	8400	150	3311	Yes.	Hamilton & Buhl 1997
Xyrauchen texanus	S, U	9800	150	3863	Yes.	Hamilton & Buhl 1997
Catostomus latipinnis	S, U	1480	150	583.4	Yes.	Hamilton & Buhl 1997
Hydra vulgaris	S, U	2300	19.5	5108	Yes.	Holdway et al. 2001
Hydra viridissima	S, U	935	19.5	2076	Yes.	Holdway et al. 2001
Daphnia magna	S, M	1319	150	520.0	Yes	Yim et al. 2006
Daphnia magna	S, M	306.7	44	341.8	Yes	Yim et al. 2006
Hyla chrysocelis	S, M	4696	45	5135	Yes.	Gottschalk 1995
Rana pipiens	S, M	10339	45	11305	Yes.	Gottschalk 1995
Daphnia magna	R, M	233	250	59.58	Yes.	Li et al. 2019
Limnodrilus hoffmeisteri	R, M	400	250	102.3	Yes.	Li et al. 2019
Chironomus riparius	R, M	13710	250	3506	Yes.	Li et al. 2019
Neocloeon triangulifer	S, M	70.55	97	40.24	Yes.	Besser et al. 2021

Species	Method	LC50 (µg/L total zinc)	Hardness (mg/L)	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Villosa umbrans	R, U	1302	43	1479	Yes.	Gibson et al. 2018
Villosa nebulosa	R, U	436	43	495.4	Yes.	Gibson et al. 2018
Leptoxis ampla	R, U	67	43	76.13	Yes.	Gibson et al. 2018
Somatogyrus sp.	R, U	329	43	373.8	Yes.	Gibson et al. 2018
Euchlanis dilatata	S, M	101	90	61.38	Yes.	Hernandez-Flores et al. 2020

* Normalized to hardness of 50 mg/L

Table 50. Freshwater acute studies not used from previous EPA criteria derivations.

Species	SMAV *(µg/L)	Reason	Reference
Mozambique tiliapia	790	Non-North American species	USEPA, 1996
Poecilia reticulata	6053	Non-North American species	USEPA, 1996
Cyprinus carpio	7233	Non-North American species	USEPA, 1996
Carrasius auratus	10250	Non-North American species	USEPA, 1996
Xenopus laevis	19176	Non-North American species	USEPA, 1996

Freshwater Chronic Zinc Criterion

There was inadequate freshwater chronic zinc data to calculate criteria using the eight-family method. The FACR of 2.00 was previously used to calculate the freshwater chronic zinc criterion as presented in 1995 updates to aquatic life (USEPA, 1996). Additional chronic zinc ACRs were available since EPA's last update. The newly calculated FACR used to derive the chronic zinc criterion is 2.950 (Table 51). Table 52 shows studies with ACR values that were not used in final calculations. Calculation results are as follows:

FAV = 64.34 (hardness of 50 mg/L)

FACR = 3.062

CCC = 21.81 µg/L (hardness of 50 mg/L)

$CCC = e^{(0.8473 \times \ln(hardness) - 0.2323)} \times CF$

Where CF (conversion factor from total to dissolved fraction) = 0.986

Chronic criterion (total) = 39.24 µg/L (hardness of 100 mg/L)

Chronic criterion (dissolved) = 39 µg/L (hardness of 100 mg/L; rounded to two significant digits)

Table 51. Acute to chronic ratios (ACR) used in chronic criterion derivation.

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
Daphnia magna	334	135.8	2.459		USEPA, 1987b
Daphnia magna	525	47.29	11.10		USEPA, 1987b
Daphnia magna	655	46.73	14.02	7.260	USEPA, 1987b
Oncorhynchus mykiss	430	276.7	1.554		USEPA, 1987b
Oncorhynchus mykiss	267	169	1.58		Wang et al. 2014

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
Oncorhynchus mykiss	2140	196	10.92		De Schamphelaere et al. 2005
Oncorhynchus mykiss	1470	1500	0.9800		De Schamphelaere et al. 2005
Oncorhynchus mykiss	194	114.4	1.695		De Schamphelaere et al. 2005
Oncorhynchus mykiss	904	247.7	3.650		De Schamphelaere et al. 2005
Oncorhynchus mykiss	2280	1146	1.990		De Schamphelaere et al. 2005
Oncorhynchus mykiss	130	88.18	1.474		De Schamphelaere et al. 2005
Oncorhynchus mykiss	153	88.62	1.726		De Schamphelaere et al. 2005
Oncorhynchus mykiss	214	225.9	0.9473		De Schamphelaere et al. 2005
Oncorhynchus mykiss	283	236.2	1.198		De Schamphelaere et al. 2005
Oncorhynchus mykiss	483	242.4	1.993		De Schamphelaere et al. 2005
Oncorhynchus mykiss	1510	566.4	2.666		De Schamphelaere et al. 2005
Oncorhynchus mykiss	548	412.6	1.328		De Schamphelaere et al. 2005
Oncorhynchus mykiss	610	200.5	3.042	1.936	De Schamphelaere et al. 2005
Salvelinus fontinalis	1996	854.7	2.335		USEPA, 1987b
Salvelinus fontinalis	1085	417	2.602	2.465	Davies et al. 2001
Pimephales promelas	600	106.3	5.644	5.644	USEPA, 1987b
Mysidopsis bahia	499	166.5	2.997	2.997	USEPA, 1987b

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
Cottus bairdi	439	255.3	1.719	1.719	Brinkman & Woodling 2005
Cottus bairdi	156	37.83	4.124	4.124	Davies et al. 2001
Prosopium williamsoni	471	269	1.590	1.590	Brinkman & Johnston 2012
Bryocamptus zschokkei	620	379.5	1.634	1.634	Brown et al. 2005
Salmo trutta	871	303	2.875	2.875	Davies et al. 2000
Ceriodaphnia dubia	173.5	18.06	9.605	9.605	Cooper et al. 2009
Hyalella azteca	99	33.94	2.917	2.917	Wang et al. 2020
Lampsilis siliquoidea	163	68.23	2.389	2.389	Wang et al. 2010
Geometric mean				3.062	

* Geometric mean of ACRs were calculated for similar species preceding the final acute chronic ratio calculation

Table 52. Studies not used in chronic zinc acute to chronic ratio calculations.

Species	ACR (µg/L)	Reason	Reference
Oncorhynchus nerka	<6.074	"Less than value" is not a definitive value.	USEPA, 1987b
Oncorhynchus tshawytscha	0.2614-1.889	Acute values presented as a range and juvenile fish were used.	USEPA, 1987b
Jordanella floridae	41.20	ACR is 10X greater than other values for zinc	USEPA, 1987b
Daphnia magna	1.742	IC25 was representative of the acute value	Lazorchak et al. 2009
Oncorhynchus mykiss	1.051	NOEC was a "less than value" making the acute value inaccurate	Mebane et al. 2008

Saltwater Zinc Criteria

No changes are proposed to the saltwater acute and chronic zinc criteria. Washington's current saltwater zinc criteria are identical to EPA recommendations, and to our knowledge there are no endangered species protection concerns.

Other Chemicals

The criteria in this section are for other chemicals besides metals listed in alphanumeric order. Toxics with an acute criteria duration of 1-hour are not to be exceeded more than once every three years on average. Toxics with an acute criteria duration of instantaneous are not to be exceeded at any time. Toxics with a chronic criteria duration of 4-day average concentration are not to be exceeded more than once every three years on average. Toxics with a chronic criteria duration of 24-hours are not to be exceeded at any time. Exceptions to these rules are otherwise noted in table footnotes (i.e., PFOS and PFOA).

4,4'-DDT and metabolites

Summary of Criteria Recommendations and Changes

Washington's freshwater and saltwater 4,4'-DDT and metabolites criteria are identical to EPA recommendations (Table 53). We are not aware of endangered species protection issues with EPA recommended 4,4'-DDT and metabolites criteria in Region 10 states. We propose no changes to Washington's current 4,4'-DDT and metabolites criteria.

Table 53. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic 4,4'-DDT and metabolites criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	1.1 (instantaneous)	0.001 (24-hour)	0.13 (instantaneous)	0.001 (24-hour)
EPA	1.1 (instantaneous)	0.001 (24-hour)	0.13 (instantaneous)	0.001 (24-hour)
Proposed	No change	No change	No change	No change

6-PPD-quinone (N-(1,3-Dimethylbutyl)-N'-phenyl-p-phenylenediamine-quinone)

Summary of Criteria Recommendations and Changes

The proposed criterion for 6PPD-q **(**N-(1,3-Dimethylbutyl)-N'-phenyl-p-phenylenediamine-quinone) is presented in Table 54. The common EPA methodology for developing criteria primarily relies on toxicity data from eight taxonomic families. We currently have freshwater acute toxicity data for five out of eight families for 6PPD-q and very limited chronic data. The eight-family minimum data requirement is intended to ensure evaluation of the most sensitive organisms with different life histories. As an alternate to the common EPA derivation method, if a commercially, recreationally, or culturally important organism is particularly sensitive, EPA recommends criteria be based on a single organism if it results in a criterion lower than the eight-family derivation method (Stephan et al. 1985).

Another, newer, alternative derivation method EPA has recommended is to set benchmarks for chemicals that do not meet the eight-family requirements by estimating toxicity data from missing families using the Web-based Interspecies Correlation Estimation (WEB-ICE) model. EPA states that these benchmarks are available for states to adopt as water quality criterion. We decided to apply EPA methods for developing benchmarks by using the WEB-ICE model to estimate toxicity for the three missing families of toxicity data for 6PPD-q (Appendix C). We required the WEB-ICE model to have a R^2 greater than 0.8 and that the surrogate be within the range of the model. As a result of that exercise, we found that the additional toxicity information estimated from the model combined with the five families of toxicity data from scientific literature resulted in a criterion value of 46 ng/L.

When we calculated a 6PPD-q criterion using EPA's single species alternative method (34 ng/L), it resulted in a more protective criterion than the eight-family method with the use of WEB-ICE (i.e., 46 ng/L). Coho salmon are significantly more sensitive to 6PPD-q than all other aquatic life and have cultural, recreational, and commercial significance. There are three median lethal concentration (LC50) values available for coho salmon that have a relatively small standard deviation (Table 55). Using the geometric mean of the three LC50 values for coho salmon and a safety factor of two results in a single species derived criterion of 34 ng/L. The single species method is more protective than the eight-family derivation method with the use of WEB-ICE. However, there are several concerns regarding protection of coho salmon at 34 ng/L.

The lowest LC50 reported for coho salmon is 41 ng/L, indicating that a criterion of 34 ng/L will likely result in significant toxicity to coho salmon. Furthermore, the toxicity tests available for coho salmon are 24 hours in duration. The standard toxicity test for vertebrates is 96 hours and is what is typically used for criteria derivations. A longer duration toxicity test is anticipated to result in additional toxicity, suggesting that 24-hour LC50s are a conservative estimate of coho salmon toxicity in terms of data used for criteria derivations. Brinkman et al. (2022) compared toxicity of the rainbow trout after 24 hours to 96 hours and reported an almost 2-fold increase in toxicity between 24 and 96 hours. These uncertainties suggest that 34 ng/L will not be adequately protective of coho salmon. Therefore, we explored additional methods to derive a protective freshwater acute 6PPD-q criterion.

The eight-family derivation method combines toxicity information from individual species within a genus. This method averages out individual species toxicity information. For example, the genus *Oncorhynchus* would require combining toxicity data for rainbow trout, chinook salmon, and coho salmon when using standard EPA derivation methods. The high sensitivity of coho salmon is therefore discounted using this method. To account for individual species toxicity, we developed a species sensitivity distribution rather than a genus sensitivity distribution. We used EPA's species sensitivity distribution calculator[7] to derive a 5th percentile of the toxicity data distribution for individual species. This method accounts for each individual species and derives a more protective criterion. The only available data with definitive toxicity values included five fish species. While toxicity studies have been conducted for invertebrates and other fish, LC50s were not determined and reported as greater than the highest test

[7] https://www.epa.gov/chemical-research/species-sensitivity-distribution-ssd-toolbox

concentration or greater than 6PPD-q solubility, indicating that aquatic invertebrates are not sensitive to 6PPD-q.

The 5th percentile of the species sensitivity distribution resulted in a value of 0.008 µg/L or 8 ng/L. We support that 8 ng/L will be protective of coho salmon and other aquatic life for the following reasons:

- 8 ng/L is approximately 5-fold lower than the lowest 24-hour LC50 for coho salmon of 41 ng/L (Lo et al. 2023)
- Greer et al. (2023) reported a coho salmon LC5 of 20.7 ng/L and a LC10 of 29.2 ng/L
- Lo et al. (2023) reported a coho salmon LC5 of 16.6 ng/L and a LC10 of 20.8 ng/L
- The most sensitive individuals in the three coho salmon toxicity tests experienced mortality between 10-20 ng/L
- The species sensitivity distribution method is more protective than other options explored, including EPA single species method, genus species sensitivity distribution, and extrapolating 24-hour coho salmon LC50s to 96 hours and applying the single species method

The information presented above indicates that coho salmon (the most sensitive aquatic species known to 6PPD-q) will be adequately protected using a FW acute criterion of 8 ng/L.

Table 54. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic 6PPD-quinone criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	-	-	-	-
Proposed	0.008 (1-hour)	-	-	-

Endangered Species Act Consultation

Since no state has adopted a water quality criterion for 6PPD-q, no ESA consultations have been completed on 6PPD-q water quality criteria.

Criteria Calculations

Freshwater Acute 6PPD-q Criterion

The data used to calculate the species sensitivity distribution is presented in Table 55. The species sensitivity distribution is presented in Figure 7.

Table 55. Acute toxicity data considered for criteria development for 6PPD-q.

Species	LC50 (µg/L)	Used for Derivation?	Reference
Oncorhynchus kisutch	0.0950	Yes.	Tian et al. 2022
Oncorhynchus kisutch	0.0410	Yes.	Lo et al. 2023
Oncorhynchus kisutch	0.0804	Yes.	Greer et al. 2023
Oncorhynchus mykiss	1.00	Yes.	Brinkman et al. 2022
Oncorhynchus mykiss	1.66	Yes.	Di et al. 2022
Salvelinus fontinalis	0.590	Yes.	Brinkman et al. 2022
Salvelinus leucomaenis Pluvius	0.510	Yes.	Hiki et al. 2022
Danio rerio	139	Yes.	Varshney et al. 2022
Salvelinus alpinus	>14.2	No. LC50 is not definitive.	Brinkman et al. 2022
Acipenser transmontanus	>14.2	No. LC50 is not definitive.	Brinkman et al. 2022
Oryzias latipes	34	No. Non-north American test species.	Hiki et al. 2021
Hyalella azteca	>43	No. LC50 is not definitive.	Hiki et al. 2021
Daphnia magna	>46	No. LC50 is not definitive	Hiki et al. 2021
Danio rerio	>54	No. LC50 is not definitive.	Hiki et al. 2021
Gobiocypris rarus	>500	No. LC50 is not definitive.	Di et al. 2022
Oncorhynchus tshawytscha	>67.3	No. LC50 is not definitive.	Lo et al. 2023
Oncorhynchus tshawytscha	>80	No. LC50 is not definitive.	Greer et al. 2023
Oncorhynchus nerka	>50	No. LC50 is not definitive.	Greer et al. 2023

Species	LC50 (µg/L)	Used for Derivation?	Reference
Salmo salar	>12.2	No. LC50 is not definitive.	Foldvik et al. 2022
Salmo trutta	>12.2	No. LC50 is not definitive.	Foldvik et al. 2022
Pimephales promelas	>39.27	No. LC50 is not definitive.	Anderson-Bain et al. 2023

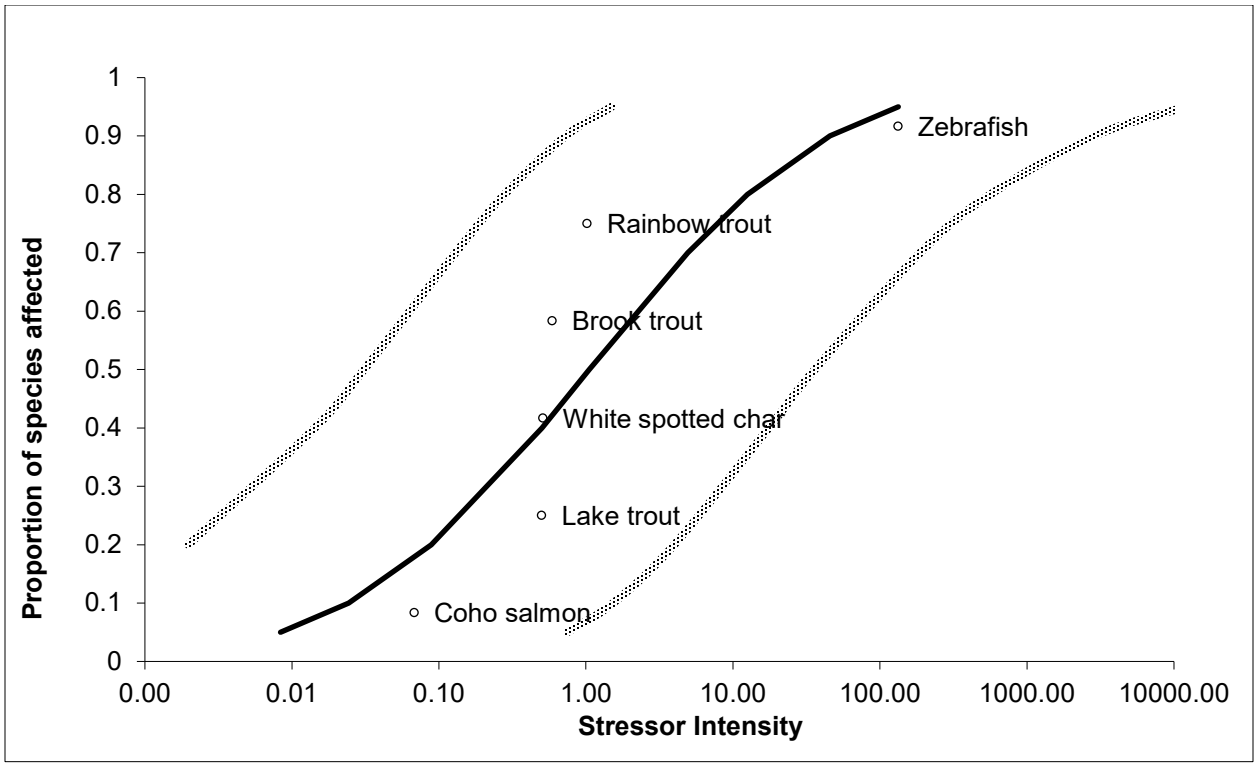

Figure 7. Species sensitivity distribution for fish species LC50 values for 6PPD-q.

Acrolein

Summary of Criteria Recommendations and Changes

Washington does not currently have acrolein criteria in the water quality standards. EPA recommended freshwater acute and chronic acrolein criteria in 2009 using 1985 EPA derivation methods. We propose that Washington adopt EPA recommendations for freshwater and acute acrolein criteria (Table 56). EPA does not have saltwater recommendations for acrolein. We are not aware of endangered species protection issues for Washington endangered species in regards to EPA's recommended acrolein criteria.

Table 56. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic acrolein criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	3 (1-hour)	3 (4-day)	-	-
Proposed	3 (1-hour)	3 (4-day)	-	-

Aldrin

Summary of Criteria Recommendations and Changes

Washington's freshwater and saltwater acute aldrin criteria are less than EPA recommendations (Table 57). We propose to adopt EPA recommendations for freshwater and saltwater acute aldrin criteria. We propose to retain Washington's current freshwater and saltwater aldrin chronic criteria to ensure existing protections are not removed for aquatic life. We are not aware of endangered species protection issues with EPA recommended aldrin criteria in Region 10 states.

Table 57. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic aldrin criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	2.5 (instantaneous)	0.0019 (24-hour)	0.71 (instantaneous)	0.0019 (24-hour)
EPA	3 (instantaneous)	-	1.3 (instantaneous)	-
Proposed	3 (instantaneous)	No change	1.3 (instantaneous)	No change

Carbaryl

Summary of Criteria Recommendations and Changes

Washington does not currently have carbaryl criteria in the water quality standards. EPA recommended freshwater acute, freshwater chronic, and saltwater acute carbaryl criteria in 2012 using 1985 EPA derivation methods. We propose that Washington adopt EPA recommendations for carbaryl in freshwater and saltwater (Table 58). We are not aware of endangered species protection issues with EPA recommended carbaryl criteria in Region 10 states. There are no saltwater chronic recommendations for carbaryl.

Table 58. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic carbaryl criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	2.1 (1-hour)	2.1 (4-day)	1.6 (1-hour)	-
Proposed	2.1 (1-hour)	2.1 (4-day)	1.6 (1-hour)	-

Chlordane

Summary of Criteria Recommendations and Changes

Washington's freshwater and saltwater chlordane criteria are identical to EPA recommendations (Table 59). We are not aware of endangered species protection issues with EPA recommended carbaryl criteria in Region 10 states. We propose no changes to Washington's current chlordane criteria.

Table 59. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic chlordane criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	2.4 (instantaneous)	0.0043 (24-hour)	0.09 (instantaneous)	0.004 (24-hour)
EPA	2.4 (instantaneous)	0.0043 (24-hour)	0.09 (instantaneous)	0.004 (24-hour)
Proposed	No change	No change	No change	No change

Chloride

Summary of Criteria Recommendations and Changes

Washington's freshwater chloride criteria are identical to EPA recommendations (Table 60). EPA does not have saltwater recommendations for chloride. We are not aware of endangered species protection issues with EPA recommended chloride criteria in Region 10 states. We propose no changes to Washington's current chloride criteria.

Table 60. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic chloride criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	860000 (1-hour)	230000 (4-day)	-	-
EPA	860000 (1-hour)	230000 (4-day)	-	-
Proposed	No change	No change	-	-

Chlorine

Summary of Criteria Recommendations and Changes

Washington's freshwater and saltwater acute and chronic chlorine criteria are identical to EPA recommendations (Table 61). We are not aware of endangered species protection issues with EPA recommended chlorine criteria in Region 10 states. The Swinomish Tribe BE suggested that the SW acute value may cause adverse effects to ESA species (USEPA, 2022a). However, the effects assessment concentration EPA developed of 12.56 µg/L rounded to two significant digits is 13 µg/L and equal to the saltwater acute chlorine criterion. We found the potential effects on ESA species negligible after considering rounding. Furthermore, the Swinomish Tribe BE has not been evaluated by NOAA/USFWS and do not represent official ESA consultation. We propose no changes to Washington's current chlorine criteria.

Table 61. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic chlorine criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	19 (1-hour)	11 (4-day)	13 (1-hour)	7.5 (4-day)
EPA	19 (1-hour)	11 (4-day)	13 (1-hour)	7.5 (4-day)
Proposed	No change	No change	No change	No change

Chlorpyrifos

Summary of Criteria Recommendations and Changes

Washington's freshwater and saltwater acute and chronic chlorpyrifos criteria are identical to EPA recommendations (Table 62). We are not aware of endangered species protection issues with EPA recommended chlorpyrifos criteria in Region 10 states. We propose no changes to Washington's current chlorpyrifos criteria.

Table 62. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic chlorpyrifos criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	0.083 (1-hour)	0.041 (4-day)	0.011 (1-hour)	0.0056 (4-day)
EPA	0.083 (1-hour)	0.041 (4-day)	0.011 (1-hour)	0.0056 (4-day)
Proposed	No change	No change	No change	No change

Cyanide

Summary of Criteria Recommendations and Changes

The proposed freshwater acute and chronic cyanide criteria are more stringent than EPA recommendations (Table 63). The freshwater criteria are based on any new science since EPA last updated the cyanide criteria in 1995 (USEPA, 1996) and used the 1st percentile of the toxicity data distribution to ensure protection of Washington's endangered species. The proposed cyanide saltwater criteria are identical to EPA recommendations.

Table 63. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic cyanide criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	22 (1-hour)	5.2 (4-day)	1 (1-hour)	1 (4-day)
EPA	22 (1-hour)	5.2 (4-day)	1 (1-hour)	1 (4-day)
Proposed	12 (1-hour)	2.7 (4-day)	No change	No change

Endangered Species Act Consultation

Idaho

There were jeopardy calls for freshwater acute (22 µg/L) and chronic (5.2 µg/L) cyanide criteria in Idaho (NMFS, 2014; USFWS, 2015). The jeopardy calls were for bull trout, a species relevant to Washington. The Idaho BiOps specifically state:

> "The proposed acute and chronic criteria can expose listed salmonids to harmful cyanide concentrations under specific situations. The acute criterion cannot be considered to be reliably protective when water temperatures drop to about 6°C or lower. Further, Leduc (1984) found that cyanide concentrations at the chronic criterion in water colder than 6°C may be associated with chronic toxicity effects. Temperatures in streams within the action area routinely drop below 6°C."
>
> "The proposed acute criterion for cyanide (22 µg/L) is likely to cause mortality of exposed bull trout; an only slightly higher concentration of cyanide at 27 µg/L killed 50 percent of exposed brook trout. In separate reviews, USFWS (2010) and NMFS (2010b) evaluated the same cyanide criteria from a national perspective. Both described scenarios in which impaired reproduction from diverse species was extrapolated to effects on listed anadromous salmonids, through the use of interspecies correlation estimates of acute toxicity. Under these scenarios, adverse effects were considered by USFWS and NMFS as likely to jeopardize the continued existence of a variety of species, including Snake River salmon and steelhead."

"Data on the long-term exposure effects of cyanide on the brook trout and the rainbow trout show reduced egg production for the brook trout, and reduced growth and swimming performance for rainbow trout at cyanide concentrations at or below the proposed chronic criterion."

"The proposed criteria for cyanide are likely to create habitat conditions that impair or preclude the capability of the critical habitat to provide for the normal reproduction, growth, movement, and survival of the bull trout within approximately 44 percent of the streams and 35 percent of the lakes and reservoirs designated range-wide as critical habitat. On that basis, implementation of the proposed criteria for cyanide are likely to appreciably impair or preclude the recovery support function (persistent core area populations of the bull trout) of critical habitat within a major portion of the designated area."

"Implementation of the proposed criteria for cyanide is likely to cause mortality, reduced swimming performance, reduced growth, and reduced egg production of exposed individuals within 39 percent of the sturgeon's range. Similar effects are expected to exposed individuals of fish species that sturgeon prey on. These impacts are likely to reduce reproduction and numbers of the Kootenai River white sturgeon within 39 percent of its range. Given the scale and magnitude of anticipated effects, implementation of the proposed criteria for cyanide are likely to impede natural reproduction and achievement of a stable or increasing sturgeon population within a major portion of its range."

"Implementation of the proposed criteria for cyanide is likely to create habitat conditions within the entire area of designated critical habitat for the Kootenai River white sturgeon that cause mortality, reduced swimming performance, reduced growth, and reduced egg production of exposed individuals of the sturgeon. Similar effects are expected to exposed individuals of fish species that sturgeon prey on. The impacts of these altered habitat conditions are likely to reduce the reproduction and numbers of the Kootenai River white sturgeon within the critical habitat."

Criteria Calculations

Freshwater Acute Cyanide Criterion

The data used to derive the freshwater acute cyanide criterion is presented in Table 64. New studies that met data acceptability requirements are presented in Table 65. Studies used in previous EPA derivations but not used in this derivation are found in Table 66. The proposed freshwater acute criterion for cyanide was derived using 14 GMAVs and the 1st percentile of the toxicity data distribution. Calculation results are as follows:

FAV = 23.07

CMC = 11.53 µg/L

Acute criterion = 12 µg/L (rounded to two significant digits)

Table 64. Freshwater acute toxicity data used for criteria derivation.

Rank	GMAV (µg/L)	Species	SMAV (µg/L)
1	42.61	Daphnia magna Daphnia pulex	19 95.55
2	44.73	Oncorhynchus mykiss	44.73
3	73	Salmo salar	73
4	85.8	Salvelinus fontinalis	85.8
5	92.64	Perca flavescens	92.64
6	100.3	Lepomis macrochirus	99.28
7	102	Pomoxis nigromaculatus	102
7	102	Micropterus salmoides	102
9	125.1	Pimephales promelas	125.1
10	167	Gammarus pseudolimnaeus	167
11	426	Pternoarcys dorsata	426
12	432	Physa heterostropha	432
13	500	Gambusia affinis	500
14	2326	Asellus communis	2326

Table 65. New freshwater acute studies that met data acceptability requirements since EPA last updated cyanide criteria (S = static, R = static renewal, FT = flow-through, U = unmeasured test concentrations, M = measured test concentrations).

Species	Method	LC50 (µg/L)	Used in Derivation?	Reference
Salmo salar	R, M	90	No. Other study used flow through design with measured concentrations.	Tryland & Grande 1983
Salmo salar	FT, M	73	Yes.	Alabaster 1983
Daphnia magna	FT, U	19	Yes.	Jaafarzadeh et al. 2013
Lepomis macrochirus	FT, M	110	Yes.	Van der Schalie et al. 2004

Table 66. Freshwater acute studies not used from previous EPA criteria derivations.

Species	SMAV (µg/L)	Reason	Reference
Poecilia reticulata	147	Non-North American species	USEPA, 1996
Carassius auratus	318	Non-North American species	USEPA, 1996

Freshwater Chronic Cyanide Criterion

There was not adequate toxicity data available to calculate a chronic cyanide criterion using the eight-family method, and therefore, an ACR was used. We did not find any new ACRs available since EPA last updated the freshwater cyanide criteria in 1995 aquatic life updates. We decided to use the FACR developed in EPA's 1995 cyanide derivation document of 8.57 (USEPA, 1996). We used the FAV derived from the proposed acute criterion using the 1st percentile to calculate the chronic criterion. Calculations results were as follows:

FACR = 8.57

FAV = 23.07

CCC = 2.6920

Chronic criterion = 2.7 µg/L (rounded to two significant digits)

Saltwater Acute and Chronic Cyanide Criteria

No changes are proposed to the saltwater acute and chronic cyanide criteria. Washington's current saltwater cyanide criteria are identical to EPA recommendations and to our knowledge there are no endangered species protection concerns in Washington.

Demeton

Summary of Criteria Recommendations and Changes

Washington does not currently have demeton criteria in the water quality standards. EPA has recommended freshwater chronic and saltwater chronic demeton criteria since 1985. We propose that Washington adopt EPA recommendations for freshwater and saltwater chronic demeton criteria (Table 67). We are not aware of endangered species protection issues with the EPA recommended demeton criteria in Region 10 states.

Table 67. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic demeton criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	-	0.1 (-)	-	0.1 (-)
Proposed	-	0.1 (4-day)	-	0.1 (4-day)

Diazinon

Summary of Criteria Recommendations and Changes

Washington does not currently have diazinon criteria in the water quality standards. EPA has recommendations for freshwater acute, freshwater chronic, saltwater acute, and saltwater chronic diazinon criteria. We propose that Washington adopt EPA recommendations for diazinon in freshwater and saltwater (Table 68). We are not aware of endangered species protection issues with EPA recommended diazinon criteria in Region 10 states.

Table 68. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic diazinon criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	0.17 (1-hour)	0.17 (4-day)	0.82 (1-hour)	0.82 (4-day)
Proposed	0.17 (1-hour)	0.17 (4-day)	0.82 (1-hour)	0.82 (4-day)

Dieldrin

Summary of Criteria Recommendations and Changes

The freshwater dieldrin criteria were updated by EPA in 1995 (USEPA, 1996). We propose to adopt EPA recommendations for freshwater dieldrin criteria (Table 69). The saltwater dieldrin criteria were not updated in 1995 and uses pre-1985 EPA methods. Washington's current saltwater dieldrin criteria matches EPA recommendations, and therefore, no changes were necessary.

Table 69. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic dieldrin criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	2.5 (instantaneous)	0.0019 (24-hour)	0.71 (instantaneous)	0.0019 (24-hour)
EPA	0.24 (1-hour)	0.056 (4-day)	0.71 (instantaneous)	0.0019 (24-hour)
Proposed	0.24 (1-hour)	0.056 (4-day)	No change	No change

Endosulfan (alpha)

Summary of Criteria Recommendations and Changes

Washington has freshwater and saltwater acute and chronic endosulfan criteria that are identical to EPA recommendations (Table 70). We are not aware of endangered species protection issues with EPA recommendations in Region 10 states. Washington's endosulfan criteria do not specify stereochemistry (i.e., alpha and beta isomers). We intend to clarify that Washington's criteria include both alpha and beta configurations, but we propose no changes to the freshwater and saltwater numeric criteria.

Table 70. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic endosulfan (alpha) criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	0.22 (instantaneous)	0.056 (24-hour)	0.034 (instantaneous)	0.0087 (24-hour)
EPA	0.22 (instantaneous)	0.056 (24-hour)	0.034 (instantaneous)	0.0087 (24-hour)
Proposed	No change	No change	No change	No change

Endosulfan (beta)

Summary of Criteria Recommendations and Changes

Washington has freshwater and saltwater acute and chronic endosulfan criteria that are identical to EPA recommendations (Table 71). We are not aware of endangered species protection issues with EPA recommendations in Region 10 states. Washington's endosulfan criteria do not specify stereochemistry (i.e., alpha and beta isomers). We intend to clarify that Washington's criteria include both alpha and beta configurations, but we propose no changes to the freshwater and saltwater numeric criteria.

Table 71. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic endosulfan (beta) criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	0.22 (instantaneous)	0.056 (24-hour)	0.034 (instantaneous)	0.0087 (24-hour)
EPA	0.22 (instantaneous)	0.056 (24-hour)	0.034 (instantaneous)	0.0087 (24-hour)
Proposed	No change	No change	No change	No change

Endrin

Summary of Criteria Recommendations and Changes

The freshwater endrin criteria were updated by EPA in 1995 (USEPA, 1996). We propose to adopt EPA recommendations for freshwater endrin criteria (Table 72). The saltwater endrin criteria were not updated in 1995 and uses pre-1985 EPA methods. Washington's current saltwater endrin criteria matches EPA recommendation, and therefore, no changes were necessary.

Table 72. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic endrin criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	0.18 (instantaneous)	0.0023 (24-hour)	0.037 (instantaneous)	0.0023 (24-hour)
EPA	0.086 (1-hour)	0.036 (4-day)	0.037 (instantaneous)	0.0023 (24-hour)
Proposed	0.086 (1-hour)	0.036 (4-day)	No change	No change

gamma-BHC (Lindane)

Summary of Criteria Recommendations and Changes

We propose to adopt EPA recommendations for freshwater acute gamma-BHC (lindane; Table 73). EPA removed the freshwater chronic gamma-BHC criterion because EPA disqualified some of the data used to derive the chronic criterion in their 1995 update (Table 73; USEPA, 1996). However, we have not changed the FW chronic lindane criteria because of existing protections the criteria provides for aquatic life. EPA did not update the saltwater gamma-BHC criterion in 1995, and their current recommendations use pre-1985 EPA methods. Washington's current saltwater gamma-BHC criteria matches EPA recommendations, and therefore, no changes were necessary.

Table 73. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic gamma-BHC criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	2 (instantaneous)	0.08 (24-hour)	0.16 (instantaneous)	-
EPA	0.95 (1-hour)	-	0.16 (instantaneous)	-
Proposed	0.95 (1-hour)	No change	No change	-

Guthion

Summary of Criteria Recommendations and Changes

Washington does not currently have guthion criteria in the water quality standards. EPA recommended freshwater and saltwater chronic guthion criteria. We propose that Washington adopt EPA recommendations for freshwater and saltwater chronic guthion criteria (Table 74). We are not aware of endangered species protection issues with EPA recommended guthion criteria in Region 10 states.

Table 74. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic guthion criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	-	0.01 (-)	-	0.01 (-)
Proposed	-	0.01 (4-day)	-	0.01 (4-day)

Heptachlor

Summary of Criteria Recommendations and Changes

Washington's freshwater and saltwater acute and chronic heptachlor criteria are identical to EPA recommendations. We are not aware of endangered species protection issues with EPA recommended heptachlor criteria in Region 10 states. We propose no changes to Washington's current heptachlor criteria (Table 75).

Table 75. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic heptachlor criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	0.52 (instantaneous)	0.0038 (24-hour)	0.053 (instantaneous)	0.0036 (24-hour)
EPA	0.52 (instantaneous)	0.0038 (24-hour)	0.053 (instantaneous)	0.0036 (24-hour)
Proposed	No change	No change	No change	No change

Heptachlor epoxide

Summary of Criteria Recommendations and Changes

Washington does not currently have heptachlor epoxide criteria in the water quality standards. EPA has recommended freshwater acute and chronic and saltwater acute and chronic heptachlor criteria. EPA recommendations for heptachlor epoxide are based on toxicity studies for heptachlor. Heptachlor is the parent component of the metabolite heptachlor epoxide. Metabolites or degrades of parent compounds do not have the same chemical structure and can result in toxicity greater or less than a parent compound. There is uncertainty regarding aquatic life species sensitivity to heptachlor epoxide. We propose not to adopt EPA recommendations and to apply Washington's narrative toxics criteria when needed (Table 76). EPA recommendations for heptachlor epoxide does not use EPA 1985 standard methods for deriving toxics and are based on limited toxicity studies.

Table 76. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic heptachlor epoxide criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	0.52 (instantaneous)	0.0038 (24-hour)	0.053 (instantaneous)	0.0036 (24-hour)
Proposed	-	-	-	-

Malathion

Summary of Criteria Recommendations and Changes

Washington does not currently have malathion criteria in the water quality standards. EPA has recommendations for freshwater and saltwater chronic malathion criteria. We propose that Washington adopt EPA recommendations for malathion in freshwater and saltwater (Table 77). We are not aware of endangered species protection issues with EPA recommended malathion criteria in Region 10 states.

Table 77. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic malathion criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	-	0.1 (-)	-	0.1 (-)
Proposed	-	0.1 (4-day)	-	0.1 (4-day)

Methoxychlor

Summary of Criteria Recommendations and Changes

Washington does not currently have methoxychlor criteria in the water quality standards. EPA has recommendations for freshwater and saltwater chronic methoxychlor criteria. We propose that Washington adopt EPA recommendations for methoxychlor in freshwater and saltwater (Table 78). We are not aware of endangered species protection issues with EPA recommended methoxychlor criteria in Region 10 states.

Table 78. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic methoxychlor criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	-	0.03 (-)	-	0.03 (-)
Proposed	-	0.03 (4-day)	-	0.03 (4-day)

Mirex

Summary of Criteria Recommendations and Changes

Washington does not currently have methoxychlor criteria in the water quality standards. EPA has recommendations for freshwater and saltwater chronic methoxychlor criteria. We propose that Washington adopt EPA recommendations for methoxychlor in freshwater and saltwater (Table 79). We are not aware of endangered species protection issues with EPA recommended methoxychlor criteria in Region 10 states.

Table 79. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic mirex criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	-	0.001 (-)	-	0.001 (-)
Proposed	-	0.001 (4-day)	-	0.001 (4-day)

Nonylphenol

Summary of Criteria Recommendations and Changes

Washington does not currently have nonylphenol criteria. EPA has recommendations for freshwater and saltwater nonylphenol criteria (USEPA, 2005; Table 80). The Swinomish Tribe BE suggests there could be a LAA but there are no completed BiOps in other Region 10 states. We examined the new science since EPA last updated nonylphenol criteria in 2005 and it resulted in a higher criterion value. We propose to match EPA recommendations for nonylphenol because there is not an existing BiOp with a LAA and EPA recommendations are intended to be protective of aquatic species (Table 80).

Table 80. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic nonylphenol criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	28 (1-hour)	6.6 (4-day)	7 (1-hour)	1.7 (4-day)
Proposed	28 (1-hour)	6.6 (4-day)	7 (1-hour)	1.7 (4-day)

Endangered Species Act Consultation

We are not aware of any completed nonylphenol ESA consultations in EPA Region 10 states that are relevant to this rulemaking. EPA's biological evaluation for the Swinomish Tribe suggested a likely to adversely affect determination but a BiOp has not been completed.

Swinomish Tribe Biological Evaluation

Below is an explanation of potential effects of the nonylphenol criteria in the Swinomish Tribe BE (USEPA, 2022a):

"The acute toxicity of nonylphenol in freshwaters was evaluated in fish only. The PCLTV for fish was 13.5 µg/L for mortality to *Lepomis macrochirus* of two tested fish species. As the lowest PCLTV of 13.5 µg/L was lower than the criterion of 28.0 µg/L, the criterion may not be protective of prey species relevant to listed species. Therefore, EPA calculated the percent of species with toxicity values less than the criterion and found that because 2 of 2 (100%; >20% threshold) species toxicity values were greater than the criterion, exposure at the level of the acute freshwater criterion is **likely to result in reductions** in the community of prey species."

"The nonylphenol marine acute criterion LAA call was not based on effects to any of the ESA listed fish species within the action area. Instead it was based on the 5th percentile of a SSD of eight 96 hour LC50 values for marine fish, five of which were found in a review of the literature published since the EPA (USEPA, 2005a) nonylphenol criteria document was issued. The 5th percentile of the fitted SSD (12.18 µg/L) divided by 2.27 resulted in a calculated acute toxicity threshold value of 5.37 µg/L, lower than the marine acute nonylphenol criterion of 7 µg/L. The same considerations apply to the chronic criterion, which was derived from the acute criterion. The nonylphenol chronic effects assessment concentration (0.6614 µg/L) is lower than the marine chronic nonylphenol criterion (1.0 µg/L). Our conclusion is that exposure at the level of the marine chronic nonylphenol criterion is likely to adversely affect rainbow trout (steelhead), Chinook salmon, chum salmon, bull trout, bocaccio and yelloweye rockfish."

Parathion

Summary of Criteria Recommendations and Changes

Washington's freshwater acute and chronic parathion criteria are identical to EPA recommendations. EPA does not have parathion saltwater criteria recommendations. We are not aware of endangered species protection issues with EPA recommended parathion criteria in Region 10 states. We propose no changes to Washington's current parathion criteria (Table 81).

Table 81. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic parathion criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	0.065 (1-hour)	0.013 (4-day)	-	-
EPA	0.065 (1-hour)	0.013 (4-day)	-	-
Proposed	No change	No change	-	-

Pentachlorophenol

Summary of Criteria Recommendations and Changes

The proposed freshwater pentachlorophenol criteria accounts for endangered species protection levels by incorporating the new science available since EPA last updated the criteria in 1995 (USEPA, 1996). The proposed freshwater pentachlorophenol criteria are more stringent than EPA recommendations (Table 82). The saltwater pentachlorophenol criteria are more stringent than EPA recommendations to account for endangered species protection levels. The pentachlorophenol saltwater criteria were calculated using new science available since EPA last updated the criteria in 1986.

Table 82. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic pentachlorophenol criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	20* (1-hour)	13* (4-day)	13 (1-hour)	7.9 (4-day)
EPA	19* (1-hour)	15* (4-day)	13 (1-hour)	7.9 (4-day)
Proposed	9.4* (1-hour)	4.7* (4-day)	No change	6.7 (4-day)

* pH dependent criteria (numeric values based on pH of 7.8)

Endangered Species Act Consultation

Oregon

The Oregon NMFS BiOp reported likely to adversely affect determinations for salmonids for EPA's freshwater acute (19 µg/L) and chronic (15 µg/L) criteria and saltwater chronic (7.9 µg/L) criterion (NMFS, 2012). The Oregon BiOp stated:

> "The available evidence for pentachlorophenol indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderately-high-intensity) and reduced growth (moderate intensity)."
>
> "In summary, the available evidence for saltwater PCP indicates that listed species exposed to waters equal to the chronic criterion concentrations will suffer chronic toxic effects including sublethal effects (moderately-high-intensity)."
>
> "Based on the direct mortality population modeling results, juvenile salmon and steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc is predicted to result in mortality at the population level—relative to the baseline population model."

Swinomish Tribe Biological Evaluation

The 2022 Swinomish BE indicated "likely to adversely affect" determinations for the saltwater acute pentachlorophenol criterion (USEPA, 2022a). More specifically it states:

> "Dividing the Pacific herring 25.3 µg/L SMAV by 2.27 to convert this LC50 to the lowest LCLOW or minimum acute effect concentration for any marine fish species yields a threshold acute effect concentration of 11.1 µg/L. This concentration is lower than the pentachlorophenol marine acute criterion of 13 µg/L. Assuming that this threshold acute effect concentration is the same as that for all ESA listed fish species in the marine portions of the action area, exposure at the level of the marine pentachlorophenol acute criterion of 13 µg/L is likely to adversely affect rainbow trout (steelhead), Chinook salmon, chum salmon, bull trout, bocaccio and yelloweye rockfish."

Criteria Calculations

Freshwater Acute Pentachlorophenol Criterion

The data used to derive the freshwater acute pentachlorophenol criterion are presented in Table 83. New studies that met data acceptability requirements are presented in Table 84. Studies used in previous EPA derivations but not used in this derivation are found in Table 85. The proposed freshwater acute criterion for pentachlorophenol was derived using 66 GMAVs. Calculation results are as follows:

FAV = 5.107 (pH of 6.5)

CMC = 2.554 ug/L (pH of 6.5)

CMC = e^[1.005(pH) – 5.595]

Acute criterion = 9.4 µg/L (at pH = 7.8; rounded to two significant digits)

Table 83. Freshwater acute toxicity data (normalized to pH of 6.5) used for criteria derivation.

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
1	1.208	Plationus platulus	1.208
2	2.745	Keratella cochlearis	2.745
3	3.660	Lecane quadridentata	3.660
4	7.321	Triphysaria pusilla	7.321
5	7.840	Acipenser brevirostrum	10.371
		Acipenser oxyrinchus	<5.926
6	8.803	Hyalella azteca	8.803
7	12.55	Entosphenus tridentatus	12.55
8	21.96	Elliptio dilatate	21.96
9	22.93	Lithobates sphenocephalus	22.93
10	26.54	Ictalurus punctatus	26.54
11	28.69	Oncorhynchus mykiss	33.63
		Oncorhynchus kisutch	31.82

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
		Oncorhynchus nerka	32.85
		Oncorhynchus tshawytscha	25.85
		Oncorhynchus apache	19.93
		Oncorhynchus clarkii	30.79
12	33.91	Rana catesbeiana	33.91
13	34.13	Salvelinus fontinalis	34.13
14	42.40	Lepomis macrochirus	42.40
15	51.56	Simocephalus vetulus	51.56
16	58.18	Chaetocorophium lucasi	58.18
17	58.47	Varichaeta pacifica	58.47
18	60.43	Aplexa hypnorum	60.43
19	60.5	Gambusia affinis	60.5
20	60.61	Anaxyrus boreas boreas	60.61
21	65.80	Pimephales promelas	65.80
22	76.74	Ceriodaphnia dubia	87.73
		Ceriodaphnia reticulata	67.13
23	91.48	Gammarus pseudolimnaeus	91.48
24	95.17	Asplanchna girodi	95.17
25	105.0	Micropterus salmoides	105.0
26	105.1	Leptodea fragilis	105.1
27	109.8	Philodina acuticornis	109.8
28	120.0	Brachionus calyciflorus	120.0
29	122.1	Daphnia pulex	90.83
		Daphnia magna	78.51
		Daphnia carinata	255.1
30	128.4	Deleatidium sp.	128.4
31	132.1	Physa gyrina	132.1
32	146.7	Utterbackia imbecillis	146.7
33	151.3	Corbicula fluminea	151.3
34	155.8	Ligumia subrostrate	155.8
35	155.9	Branchiura sowerbyi	155.9
36	161.2	Megalonaias nervosa	161.2
37	172.1	Crangonyx pseudogracilis	172.1
38	182.5	Limnodrilus hoffmeisteri	182.5
39	212.3	Heteropneustes fossilis	212.3
40	224.2	Tubifex tubifex	224.2
41	234.3	Clarias batrachus	234.3
42	246.3	Lampsilis cardium	240.9
		Lampsilis siliquoidea	251.8
43	281.9	Channa punctatus	281.9

Rank	GMAV* (µg/L)	Species	SMAV* (µg/L)
44	306.7	Jordanella floridae	306.7
45	308.8	Lumbriculus variegatus	308.8
46	317.5	Quistradrilus multisetosus	317.5
47	361.6	Spirosperma ferox	239.5
		Spirosperma nikoiskyl	545.8
48	403.2	Gillia altilis	403.2
49	408.2	Stylodrilus heringianus	408.2
50	417.7	Rhyacodirilus montana	417.7
51	484.3	Prionchulus punctatus	484.3
52	492.3	Sphaerium novaezelandiae	492.3
53	805.6	Tanais standfordi	805.6
54	1145	Tobrilus gracilis	1145
55	1585	Dorylaimus stagnalis	1585
56	1672	Aporcelaimellus obtusicaudatus	1672
57	2818	Tylenchus elegans	2818
58	3881	Chironomus riparius	3881
59	8408	Plectus acuminatus	8408
60	10610	Sepedon fuscipennis	10610
61	11621	Diplogasteritus species	11621
62	11260	Tanytarsus dissimilis	11260
63	11914	Caenorhabditis elegans	11914
64	>14968	Rhabditis species	>14968
65	>14968	Cephalobus persegnis	>14968
66	35872	Culex pipiens fatigans	35872

* Normalized to pH of 6.5

Table 84. New freshwater acute studies that met data acceptability requirements since EPA last updated pentachlorophenol criteria (S = static, R = static renewal, FT = flow-through, U = unmeasured test concentrations, M = measured test concentrations).

Species	Method	LC50 (µg/L)	pH	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Entosphenus tridentatus	FT, M	31	7.4	12.55	Yes.	Anderson et al. 2010
Corbicula fluminea	R, M	250	7	151.3	Yes.	Basack et al. 1997
Lithobates sphenocephalus	S, M	140	8.3	22.93	Yes.	Bridges et al. 2002
Anaxyrus boreas boreas	S, M	370	8.3	60.61	Yes.	Bridges et al. 2002
Lepomis macrochirus	S, M	192	8.3	31.45	Yes.	Bridges et al. 2002
Oncorhynchus mykiss	S, M	160	8.2	28.98	Yes.	Dwyer et al. 2000
Pimephales promelas	S, M	250	8.3	40.95	Yes.	Dwyer et al. 2000
Oncorhynchus apache	S, M	110	8.2	19.93	Yes.	Dwyer et al. 2000
Oncorhynchus clarkii	S, M	>10	8.2	>1.811	No. LC50 10x more sensitive than other studies using the same species and LC50 is a "greater than value."	Dwyer et al. 2000
Oncorhynchus clarkii	S, M	170	8.2	30.79	Yes.	Dwyer et al. 2000
Gila elegans	S, M	230	8.3	37.68	Yes.	Dwyer et al. 2000
Ptychocheilus lucius	S, M	240	8.3	39.32	Yes.	Dwyer et al. 2000
Xyrauchen texanus	S, M	280	8.3	45.87	Yes.	Dwyer et al. 2000
Acipenser brevirostrum	S, M	70	8.4	10.37	Yes.	Dwyer et al. 2000
Acipenser oxyrinchus	S, M	<40	8.4	<5.926	Yes.	Dwyer et al. 2000

Species	Method	LC50 (µg/L)	pH	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Hyalella azteca	R, U	4	8.0	0.8859	Yes.	McNulty et al. 1999
Leptodea fragilis	S, M	580	8.2	105.1	Yes.	Milam et al. 2005
Lampsilis cardium	S, M	1330	8.2	240.9	Yes.	Milam et al. 2005
Lampsilis siliquoidea	S, M	1390	8.2	251.8	Yes.	Milam et al. 2005
Megalonaias nervosa	S, M	890	8.2	161.2	Yes.	Milam et al. 2005
Ligumia subrostrate	S, M	860	8.2	155.8	Yes.	Milam et al. 2005
Utterbackia imbecillis	S, M	810	8.2	146.7	Yes.	Milam et al. 2005
Ceriodaphnia dubia	S, M	470	8.2	85.13	Yes.	Milam et al. 2005
Daphnia magna	S, M	680	8.2	123.2	Yes.	Milam et al. 2005
Chironomus riparius	R, U	1421	6.8	1051	Yes.	Morales et al. 2014
Daphnia magna	S, U	150	7.3	67.13	Yes.	Oda et al. 2006
Brachionus calyciflorus	S, U	262	7.5	95.90	Yes.	Preston et al. 2001
Brachionus calyciflorus	S, U	1310	7.5	479.5	Yes.	Radix et al. 2000
Daphnia carinata	S, U	570	7.3	255.1	Yes.	Willis 1999
Ceriodaphnia dubia	S, U	202	7.3	90.40	Yes.	Willis 1999
Ceriodaphnia pulchella	S, U	1790	7.3	801.1	Yes.	Willis 1999
Simocephalus vetulus	S, U	140	7.3	62.65	Yes.	Willis 1999
Daphnia magna	S, U	187	7.3	83.69	Yes.	Willis 1999
Deleatidium sp.	S, U	287	7.3	128.4	Yes.	Willis 1999
Chaetocorophium lucasi	S, U	130	7.3	58.18	Yes.	Willis 1999
Sphaerium novaezelandiae	S, U	1100	7.3	492.3	Yes.	Willis 1999

Species	Method	LC50 (µg/L)	pH	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Lumbriculus variegatus	S, U	690	7.3	308.8	Yes.	Willis 1999
Tanais standfordi	S, U	1800	7.3	805.6	Yes.	Willis 1999
Simocephalus vetulus	S, M	140	7.8	37.91	Yes.	Willis et al. 1995
Pimephales promelas	FT, M	564	7.8	152.7	Yes. Combined with other LC50 values from Broderius et al. 1995.	Broderius et al. 1995
Pimephales promelas	FT, M	449	7.8	121.6	Yes. Combined with other LC50 values from Broderius et al. 1995.	Broderius et al. 1995
Pimephales promelas	FT, M	350	7.8	94.77	Yes. Combined with other LC50 values from Broderius et al. 1995.	Broderius et al. 1995
Heteropneustes fossilis	FT, M	580	7.5	212.3	Yes. Calculated mean pH value of range provided.	Farah et al. 2004
Clarias batrachus	FT, M	640	7.5	234.3	Yes. Calculated mean pH value of range provided.	Farah et al. 2004
Channa punctatus	FT, M	770	7.5	281.9	Yes. Calculated mean pH value of range provided.	Farah et al. 2004
Culex pipiens	FT, M	98000	7.5	35872	Yes. Calculated mean pH value of range provided.	Farah et al. 2004
Prionchulus punctatus	S, M	293	6.0	484.3	Yes.	Kammenga et al. 1994
Dorylaimus stagnalis	S, M	958.8	6.0	1585	Yes.	Kammenga et al. 1994
Aporcelaimellus obtusicaudatus	S, M	1012	6.0	1672	Yes.	Kammenga et al. 1994
Tobrilus gracilis	S, M	692.5	6.0	1145	Yes.	Kammenga et al. 1994
Plectus acuminatus	S, M	5087	6.0	8408	Yes.	Kammenga et al. 1994
Cephalobus persegnis	S, M	9056	6.0	>14968	Yes.	Kammenga et al. 1994

Species	Method	LC50 (µg/L)	pH	Normalized LC50* (µg/L)	Used in Derivation?	Reference
Rhabditis sp.	S, M	9056	6.0	>14968	Yes.	Kammenga et al. 1994
Diplogasteritus sp.	S, M	7031	6.0	11621	Yes.	Kammenga et al. 1994
Tylenchus elegans	S, M	1705	6.0	2818	Yes.	Kammenga et al. 1994
Philodina acuticornis	S, U	300	7.5	109.8	Yes.	McDaniel & Snell 1999
Asplanchna girodi	S, U	260	7.5	95.17	Yes.	McDaniel & Snell 1999
Asplanchna girodi	S, U	160	7.5	58.57	Yes.	McDaniel & Snell 1999
Elliptio dilatate	S, U	60	7.5	21.96	Yes.	McDaniel & Snell 1999
Triphysaria pusilla	S, U	20	7.5	7.321	Yes.	McDaniel & Snell 1999
Lecane quadrientata	S, U	10	7.5	3.660	Yes.	McDaniel & Snell 1999
Keratella cochelaris	S, U	7.5	7.5	2.745	Yes.	McDaniel & Snell 1999
Plationus patulus	S, U	3.3	7.5	1.208	Yes.	McDaniel & Snell 1999
Brachionus calyciflorus	S, U	210	7.5	76.87	Yes.	Preston et al. 1999
Caenorhabditis elegans	S, M	44000	7.8	11914	Yes.	Cressman & Williams 1997

* Normalized to pH of 6.5

Table 85. Freshwater acute studies not used from previous EPA derivations.

Species	SMAV (µg/L)	Reason	Reference
Cyprinus carpio	4.355	Non-North American species	USEPA, 1996
Carassius auratus	65.53	Non-North American species	USEPA, 1996
Poecilia reticulata	195.4	Non-North American species	USEPA, 1996
Orconectes immunis	>43920	Non-North American species	USEPA, 1996

Freshwater Chronic Pentachlorophenol Criterion

There was inadequate freshwater chronic pentachlorophenol data to calculate a chronic criterion using the eight-family method. The FACR of 2.608 was previously used to calculate the freshwater chronic pentachlorophenol criterion as presented in 1995 updates to aquatic life (USEPA, 1996). Additional chronic pentachlorophenol ACRs were available since EPA's last update. The newly calculated FACR used to derive the chronic pentachlorophenol criterion is 4.044 (Table 86). Calculation results are as follows:

FAV = 5.107 (pH of 6.5)

FACR = 4.044

CCC = FAV / FACR

CCC = 1.263 ug/L (pH of 6.5)

$CCC = e^{\wedge[1.005(pH) - 6.299]}$

Chronic criterion = 4.7 µg/L (at pH = 7.8; rounded to two significant digits)

Table 86. Acute to chronic ratios (ACR) used in chronic criterion derivation.

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
Daphnia magna	600	240	2.5	2.5	USEPA, 1986b
Simocephalus vetulus	160	177.2	0.9029		USEPA, 1986b
Simocephalus vetulus	196	221.2	0.8861	0.8944	USEPA, 1986b

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR*	Species Mean ACR	Reference
Oncorhynchus mykiss	66	14.46	4.564	4.564	USEPA, 1986b
Pimephales promelas	224.9	57.25	3.928		USEPA, 1986b
Pimephales promelas	95	23.89	3.977		USEPA, 1986b
Pimephales promelas	218	40.08	5.439		USEPA, 1986b
Pimephales promelas	261	48.99	5.328		USEPA, 1986b
Pimephales promelas	378	89.23	4.236	4.701	USEPA, 1986b
Cyprinodon variegatus	442	64.31	6.873	6.873	USEPA, 1986b
Lymnaea stagnalis	170	27.91	6.091	6.091	Besser et al. 2009
Pyrgulopsis idahoensis	143	16.25	8.801	8.801	Besser et al. 2009
Geometric mean				4.044	

* Geometric mean of ACRs were calculated for similar species preceding the final acute chronic ratio calculation

Saltwater Acute Pentachlorophenol Criterion

The data used to derive the saltwater acute nonylphenol criterion are presented in Table 87. New studies that met data acceptability requirements are presented in Table 88. The proposed saltwater acute criterion for pentachlorophenol was derived using 20 GMAVs. Calculation results are as follows:

FAV = 26.87

CMC = 13.43

Acute criterion = 13 µg/L (rounded to two significant digits)

Table 87. Saltwater acute toxicity data used for criteria derivation.

Rank	GMAV (µg/L)	Species	SMAV (µg/L)
1	25.29	Clupea pallasi	25.29
2	40.83	Crassostrea gigas	40.83
3	53.2	Lagodon rhomboides	53.2
4	62.81	Pseudodiaptomus coronatus	62.81
5	96	Eurytemora affinis	96
6	112.1	Mugil cephalus	112.1
7	170	Temora longicornis	170
8	188.0	Cyprinodon variegatus	442
		Cyprinodon bovinus	80
9	>306	Fundulus similis	>306
10	328.8	Mytilus edulis	328.8
11	397.2	Limnodriloides verrucosus	397.2
12	423.4	Tubificoides gabriellae	423.4
13	435	Nereis arenaceodentata	435
14	450	Solea solea	450
15	491.3	Palaemonetes pugio	491.3
16	598.2	Monopylephorus cuticulatus	598.2
17	862.6	Ophryotrocha diadema	862.6
18	>1045	Penaeus aztectus	>195
		Penaeus duorarum	5600
19	980	Acartia bifilosa	980
20	1200	Crepidula fornicate	1200

Table 88. New saltwater acute studies that met data acceptability requirements since EPA last updated pentachlorophenol criteria (S = static, R = static renewal, U = unmeasured test concentrations, M = measured test concentrations).

Species	Method	LC50 (µg/L)	Used in Derivation?	Reference
Cyprinodon variegatus	S, U	50	No. Other studies with the same species used a flow through design and measured test concentrations.	Sappington et al. 2001
Cyprinodon bovinus	S, U	80	Yes.	Sappington et al. 2001
Eurytemora affinis	S, M	96	Yes.	Lindley 1999
Acartia bifilosa	S, M	980	Yes.	Lindley 1999

Saltwater Chronic Pentachlorophenol Criterion

There was inadequate saltwater chronic pentachlorophenol data to calculate criteria using the eight-family method. The FACR of 2.608 was previously used to calculate the saltwater chronic pentachlorophenol criterion as presented in 1995 updates to aquatic life (USEPA, 1996). Additional chronic pentachlorophenol ACRs were available since EPA's last update. The newly calculated FACR used to derive the chronic pentachlorophenol criterion is 4.044 (Table 89). Calculation results are as follows:

FAV = 26.87

FACR = 4.044

CCC = FAV / FACR = 6.652

Chronic criterion = 6.7 µg/L (rounded to two significant digits)

Table 89. Acute to chronic ratios (ACR) used in chronic criterion derivation.

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR[1]	Species Mean ACR	Reference
Daphnia magna	600	240	2.5	2.5	USEPA, 1986b
Simocephalus vetulus	160	177.2	0.9029		USEPA, 1986b
Simocephalus vetulus	196	221.2	0.8861	0.8944	USEPA, 1986b
Oncorhynchus mykiss	66	14.46	4.564	4.564	USEPA, 1986b
Pimephales promelas	224.9	57.25	3.928		USEPA, 1986b
Pimephales promelas	95	23.89	3.977		USEPA, 1986b
Pimephales promelas	218	40.08	5.439		USEPA, 1986b
Pimephales promelas	261	48.99	5.328		USEPA, 1986b
Pimephales promelas	378	89.23	4.236	4.701	USEPA, 1986b
Cyprinodon variegatus	442	64.31	6.873	6.873	USEPA, 1986b

Species	Acute Value (µg/L)	Chronic Value (µg/L)	ACR[1]	Species Mean ACR	Reference
Lymnaea stagnalis	170	27.91	6.091	6.091	Besser et al. 2009
Pyrgulopsis idahoensis	143	16.25	8.801	8.801	Besser et al. 2009
Geometric mean				4.044	

Perfluorooctane sulfonic acid (PFOS)

Summary of Criteria Recommendations and Changes

Washington does not currently have PFOS criteria in the water quality standards. EPA has draft recommendations for freshwater acute and chronic PFOS criteria and a saltwater acute benchmark (USEPA, 2022c). In EPA's development of saltwater acute criteria, they found that there was inadequate toxicity data to meet the minimum data requirements for criteria development as outlined in EPA 1985 derivation guidelines. Thus, EPA filled data gaps with a WEB-ICE model and are recommending a benchmark value that is available for states to adopt rather than a 304(a) criteria recommendation. Washington proposes to adopt EPA draft recommendations for PFOS in freshwater and saltwater (Table 90). We intend to adopt EPA final recommendations if they are released during this rulemaking. If EPA's recommendations are not finalized during the proposal phase, we do not intend to adopt the draft recommendations. We are not aware of endangered species protection issues with EPA recommended PFOS criteria in Region 10 states.

Table 90. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic PFOS criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	3000 (1-hour)	Water: 8.4 µg/L[1,2] Tissue: 6.75 mg/kg fish whole body[1,3,4] Tissue: 2.91 mg/kg fish muscle[1,3,4] Tissue: 0.937 mg/kg invertebrate whole body[1,3,4]	550 (1-hour)	-
Proposed	3000 (1-hour)	Water: 8.4 µg/L[1,2] Tissue: 6.75 mg/kg fish whole body[1,3,4] Tissue: 2.91 mg/kg fish muscle[1,3,4]	550 (1-hour)	-

		Tissue: 0.937 mg/kg invertebrate whole body[1,3,4]		

[1] All water column and tissue criteria are intended to be independently applicable and no one criterion takes primacy.
[2] Water column criteria are based on a 4-day average concentration not to be exceeded more than once every three years on average.
[3] Tissue criteria derived from the chronic water column concentration with the use of bioaccumulation factors and are expressed as wet weight (ww) concentrations.
[4] Tissue data is an instantaneous point measurement that reflect integrative accumulation of PFOS over time and space. Criteria are not to be exceeded more than once every 10 years on average.

Perfluorooctanoic acid (PFOA)

Summary of Criteria Recommendations and Changes

Washington does not currently have PFOA criteria in the water quality standards. EPA has draft recommendations for freshwater acute and chronic PFOA criteria and a saltwater acute benchmark (USEPA, 2022d). In EPA's development of saltwater acute criteria, they found that there was inadequate toxicity data to meet the minimum data requirements for criteria development as outlined in EPA 1985 derivation guidelines. Thus, EPA filled data gaps with a WEB-ICE model and are recommending a benchmark value that is available for states to adopt rather than a 304(a) criteria recommendation. We intend to adopt EPA final recommendations if they are released during this rulemaking. If EPA's recommendations are not finalized during the proposal phase, we do not intend to adopt the draft recommendations. We are not aware of endangered species protection issues with EPA recommended PFOA criteria in Region 10 states.

Table 91. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic PFOA criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	49000 (1-hour)	Water: 94 µg/L Tissue: 6.10 mg/kg fish whole body Tissue: 0.125 mg/kg fish muscle Tissue: 1.11 mg/kg invertebrate whole body	7000 (1-hour)	-
Proposed	49000 (1-hour)	Water: 94 µg/L[1,2] Tissue: 6.10 mg/kg fish whole body[1,3,4] Tissue: 0.125 mg/kg fish muscle[1,3,4] Tissue: 1.11 mg/kg invertebrate whole body[1,3,4]	7000 (1-hour)	-

[1] All water column and tissue criteria are intended to be independently applicable and no one criterion takes primacy.

[2] Water column criteria are based on a 4-day average concentration not to be exceeded more than once every three years on average.
[3] Tissue criteria derived from the chronic water column concentration with the use of bioaccumulation factors and are expressed as wet weight (ww) concentrations.
[4] Tissue data is an instantaneous point measurement that reflect integrative accumulation of PFOS over time and space. Criteria are not to be exceeded more than once every 10 years on average.

Polychlorinated biphenyls (PCBs)

Summary of Criteria Recommendations and Changes

We are recommending no changes to Washington's freshwater and saltwater PCB criteria (Table 92). EPA has recommendations for freshwater and saltwater chronic criteria but do not have recommendations for freshwater or saltwater acute criteria. Washington currently has freshwater and saltwater acute criteria based on protective values described in EPA's 1986 Gold Book. We do not intend to modify our freshwater and saltwater acute PCB criteria because of existing protections the criteria provides for aquatic life. We are not aware of endangered species protection issues with EPA's PCB recommendations in Region 10 states.

Table 92. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic PCBs criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	2 (24-hour)	0.014 (24-hour)	10 (24-hour)	0.03 (24-hour)
EPA	-	0.014 (24-hour)	-	0.03 (24-hour)
Proposed	No change	No change	No change	No change

Sulfide-Hydrogen Sulfide

Summary of Criteria Recommendations and Changes

We propose to not adopt EPA recommendations for sulfide-hydrogen sulfide (Table 93). EPA recommendations are based on very limited toxicity data. We evaluated the new science and found that only three out of eight families have toxicity data and there is less information on chronic toxicity. We recommend using Washington's toxics narrative criteria to address any issues related to sulfide-hydrogen sulfide.

Table 93. Comparison of Washington current freshwater (FW) and saltwater (SW) acute and chronic hydrogen sulfide criteria, EPA recommendations, and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	-	2 (24-hour)	-	2 (24-hour)
Proposed	-	-	-	-

Toxaphene

Summary of Criteria Recommendations and Changes

Washington's freshwater and saltwater toxaphene criteria are identical to EPA recommendations. We are not aware of endangered species protection issues with EPA recommended toxaphene criteria in Region 10 states. We propose no changes to Washington's current toxaphene criteria (Table 94).

Table 94. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic toxaphene criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	0.73 (1-hour)	0.0002 (4-day)	0.21 (1-hour)	0.0002 (4-day)
EPA	0.73 (1-hour)	0.0002 (4-day)	0.21 (1-hour)	0.0002 (4-day)
Proposed	No change	No change	No change	No change

Tributyltin

Summary of Criteria Recommendations and Changes

Washington does not currently have tributyltin criteria in the water quality standards. EPA has recommendations for freshwater and saltwater acute and chronic tributyltin criteria. We propose that Washington adopt EPA recommendations for tributyltin in freshwater and saltwater (Table 95). We are not aware of endangered species protection issues with EPA recommended tributyltin criteria in Region 10 states.

Table 95. Comparison of Washington's current freshwater (FW) and saltwater (SW) acute and chronic tributyltin criteria (duration in parentheses) with EPA recommendations and the newly proposed criteria.

	FW Acute (µg/L)	FW Chronic (µg/L)	SW Acute (µg/L)	SW Chronic (µg/L)
Washington	-	-	-	-
EPA	0.46 (1-hour)	0.072 (4-day)	0.42 (1-hour)	0.0074 (4-day)
Proposed	0.46 (1-hour)	0.072 (4-day)	0.42 (1-hour)	0.0074 (4-day)

Conclusions

The work presented in this document represent the updates needed to aquatic life toxics criteria to be consistent with Clean Water Act recommendations as well as protection levels needed for aquatic life toxics in Washington.

Additional analyses not covered in the body of this document regarding methods used to describe permit impacts and analysis (Appendix D) and water quality assessment considerations (Appendix E) are provided in the Appendix.

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U.S. Environmental Protection Agency (USEPA).1980. *Ambient Water Quality Criteria for Silver* (No. 50). US Environmental Protection Agency, Office of Water Regulations and Standards, Criteria and Standards Division. EPA 440/5-80-071.

U.S. Environmental Protection Agency (USEPA). 1980b. *Ambient water quality criteria for aldrin/dieldrin*. Office of Water Regulations and Standards, Washington D.C. EPA 440/5-80-019.

U.S. Environmental Protection Agency (USEPA). 1980c. Ambient water quality criteria for endrin. Office of Water Regulations and Standards, Washington D.C. EPA 440/5-90-047.

U.S. Environmental Protection Agency (USEPA). 1980d. Ambient water quality criteria for hexachlorocyclohexane. Office of Water Regulations and Standards, Washington D.C. EPA 440/5-80-054.

US Environmental Protection Agency (USEPA). 1985. Ambient Water Quality Criteria for Arsenic-1984. Office of Water Regulations and Standards, Washington D.C. EPA 440/5-84-033.

U.S. Environmental Protection Agency (USEPA). 1986. Quality criteria for water 1986. EPA U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington DC.

U.S. Environmental Protection Agency (USEPA). 1986b. Ambient Water Quality Criteria for Pentachlorophenol – 1986. Office of Water: Washington D.C. EPA 440/5-86-009.

U.S. Environmental Protection Agency (USEPA). 1987. Ambient Aquatic Life Water Quality Criteria for Nickel. EPA 440/5-86-004. National Technical Information Service, Springfield, VA.
U.S. Environmental Protection Agency. 1987b. Ambient aquatic life water quality criteria for zinc. EPA 440/5-87-003. Washington, DC.
U.S. Environmental Protection Agency (USEPA). 1996. 1995 Updates: Water quality criteria documents for the protection of aquatic life in ambient water. Office of Water: Washington D.C. EPA 820-B-96-001.
U.S. Environmental Protection Agency (USEPA). 2005. *Aquatic life ambient water quality criteria—nonylphenol* (Vol. 5). EPA-822-R-05. Office of Water: Washington D.C.
U.S. Environmental Protection Agency (USEPA). 2007. Aquatic life ambient freshwater quality criteria—Copper. EPA/ 822-R-07-001. Technical Report. Office of Water, Washington, DC
U.S. Environmental Protection Agency (USEPA). 2016. Aquatic Life Ambient Water Quality Criteria Cadmium. Office of Water: Washington D.C. EPA 820-R-16-002.
U.S. Environmental Protection Agency (USEPA). 2016b. Aquatic life ambient criteria quality criterion for selenium – Freshwater. EPA 822-R-16-006. US Environmental Protection Agency, Office of Water.
U.S. Environmental Protection Agency (USEPA). 2018. Final aquatic life ambient water quality criteria for aluminum 2018. Washington (DC). EPA-822-R-18-001.
U.S. Environmental Protection Agency (USEPA). 2019. Analysis of the Protectiveness of Default Ecoregional Aluminum Criteria Values. EPA-HQ-OW-2016-0694-0114.
U.S. Environmental Protection Agency (USEPA). 2022a. Biological Evaluation of EPA's Proposed Approval Action on the Swinomish Tribe's Water Quality Standards. EPA Region 10: Seattle, WA.
U.S. Environmental Protection Agency (USEPA). 2022b. Biological Evaluation of Freshwater Aluminum Water Quality Criteria for Oregon. EPA Region 10. Main_010220_clean.pdf (epa.gov)[8].
U.S. Environmental Protection Agency (USEPA). 2022c. Draft Aquatic Life Ambient Water Quality Criteria for Perfluorooctane sulfonate (PFOS). Office of Water: Washington D.C. EPA-842-D-22-002.
U.S. Environmental Protection Agency (USEPA). 2022d. Draft Aquatic Life Ambient Water Quality Criteria for Perfluorooctanoic Acid (PFOA). Office of Water: Washington D.C. EPA-842-D-22-001.
U.S. Environmental Protection Agency (USEPA). 2023. Technical Support Document. EPA's Clean Water Act action on certain surface water quality standards of the Swinomish Tribe. Water Division Region 10: Seattle, WA.
U.S. Fish and Wildlife Service (USFWS). 2012. Formal section 7 consultation on USEPA's proposed approval of Oregon water quality criteria for toxics. Oregon Fish and Wildlife Office, Portland, Oregon. TAILS no.13420-2009-F-0011. 419 pp. + appendices.

[8] https://gaftp.epa.gov/region10/ORAI/Revised_BE/Main_010220_clean.pdf

U.S. Fish and Wildlife Service (USFWS). 2015. Formal section 7 consultation on USEPA's proposed approval of Idaho water quality criteria for toxics. Idaho Fish and Wildlife Office, Boise, Idaho. TAILS no. 01EIFW00-2014-F-0233. 352 pp.

Varshney, S., Gora, A.H., Siriyappagouder, P., Kiron, V. and Olsvik, P.A., 2022. Toxicological effects of 6PPD and 6PPD quinone in zebrafish larvae. *Journal of Hazardous Materials*, *424*, p.127623.

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Wang, N., Ingersoll, C.G., Ivey, C.D., Hardesty, D.K., May, T.W., Augspurger, T., Roberts, A.D., Van Genderen, E. and Barnhart, M.C., 2010. Sensitivity of early life stages of freshwater mussels (Unionidae) to acute and chronic toxicity of lead, cadmium, and zinc in water. *Environmental Toxicology and Chemistry*, *29*(9), pp.2053-2063.

Wang, N., Mebane, C.A., Kunz, J.L., Ingersoll, C.G., Brumbaugh, W.G., Santore, R.C., Gorsuch, J.W. and Arnold, W.R., 2011. Influence of dissolved organic carbon on toxicity of copper to a unionid mussel (Villosa iris) and a cladoceran (Ceriodaphnia dubia) in acute and chronic water exposures. *Environmental Toxicology and Chemistry*, *30*(9), pp.2115-2125.

Wang, N., Ingersoll, C.G., Dorman, R.A., Brumbaugh, W.G., Mebane, C.A., Kunz, J.L. and Hardesty, D.K., 2014. Chronic sensitivity of white sturgeon (Acipenser transmontanus) and rainbow trout (Oncorhynchus mykiss) to cadmium, copper, lead, or zinc in laboratory water-only exposures. *Environmental toxicology and chemistry*, *33*(10), pp.2246-2258.

Wang, N., Kunz, J.L., Ivey, C.D., Ingersoll, C.G., Barnhart, M.C. and Glidewell, E.A., 2017. Toxicity of chromium (vi) to two mussels and an amphipod in water-only exposures with or without a co-stressor of elevated temperature, zinc, or nitrate. *Archives of environmental contamination and toxicology*, *72*, pp.449-460.

Wang, N., Kunz, J.L., Cleveland, D.M., Steevens, J.A., Hammer, E.J., Van Genderen, E., Ryan, A.C. and Schlekat, C.E., 2020. Evaluation of acute and chronic toxicity of nickel and zinc to 2 sensitive freshwater benthic invertebrates using refined testing methods. *Environmental Toxicology and Chemistry*, *39*(11), pp.2256-2268.

Winner, R.W., 1985. Bioaccumulation and toxicity of copper as affected by interactions between humic acid and water hardness. *Water Research*, *19*(4), pp.449-455.

Appendix A. ECOTOX Database Results and References

The Environmental Protection Agency (EPA) Ecotoxicology Knowledgebase (ECOTOX) database was a primary source of new science to update aquatic life toxics criteria. Below are the results for each toxic that was updated using the ECOTOX database, including each citation that was evaluated for data acceptability (Tables A1-A28). A notes column was added to each table that provides an explanation on why the article was not used for criteria derivation. If the notes box is left blank for a corresponding citation, then that article was used in updating and deriving new toxic criteria. At the end of each section we added open literature studies that were evaluated but did not meet acceptability requirements.

Arsenic

Freshwater Acute

Table A1. List of citations from EPA ECOTOX database reviewed for arsenic freshwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Broderius,S.J., M.D. Kahl, and M.D. Hoglund. Use of Joint Toxic Response to Define the Primary Mode of Toxic Action for Diverse Industrial Organic Chemicals. Environ. Toxicol. Chem.14(9): 1591-1605, 1995. ECOREF #15031	Did not find relevant arsenic data
Brodeur,J.C., C.M. Asorey, A. Sztrum, and J. Herkovits. Acute and Subchronic Toxicity of Arsenite and Zinc to Tadpoles of Rhinella arenarum both Alone and in Combination. J. Toxicol. Environ. Health Part A72(14): 884-890, 2009. ECOREF #117667	Non-north american test species
Buhl,K.J.. The Relative Toxicity of Waterborne Inorganic Contaminants to the Rio Grande Silvery Minnow (Hybognathus amarus) and Fathead Minnow (Pimephales promelas) in a Water Quality Simulating that in the Rio Grande, New Mexico. Final Rep.to U.S.Fish and Wildl.Serv., Study No.2F33-9620003, U.S.Geol.Surv., Columbia Environ.Res.Ctr., Yankton Field Res.Stn., Yankton, SD:75 p., 2002. ECOREF #77828	Arsenate based study; EPA arsenic derivation based on arsenite
Buhl,K.J., and S.J. Hamilton. Comparative Toxicity of Inorganic Contaminants Released by Placer Mining to Early Life Stages of Salmonids. Ecotoxicol. Environ. Saf.20(3): 325-342, 1990. ECOREF #334	Arsenate based study; EPA arsenic derivation based on arsenite
Buhl,K.J., and S.J. Hamilton. Relative Sensitivity of Early Life Stages of Arctic Grayling, Coho Salmon, and Rainbow Trout to Nine Inorganics. Ecotoxicol. Environ. Saf.22:184-197, 1991. ECOREF #3956	

Citation	Notes
Burton,G.A.,Jr., J.M. Lazorchak, W.T. Waller, and G.R. Lanza. Arsenic Toxicity Changes in the Presence of Sediment. Bull. Environ. Contam. Toxicol.38(3): 491-499, 1987. ECOREF #12154	Study included sediment
Dyer,S.D., G.L. Brooks, K.L. Dickson, B.M. Sanders, and E.G. Zimmerman. Synthesis and Accumulation of Stress Proteins in Tissues of Arsenite-Exposed Fathead Minnows (Pimephales promelas). Environ. Toxicol. Chem.12:913-924, 1993. ECOREF #7266	Very little information on methods; 3-5 fish per replicate
Dyer,S.D., K.L. Dickson, and E.G. Zimmerman. A Laboratory Evaluation of the Use of Stress Proteins in Fish to Detect Changes in Water Quality. ASTM Spec. Tech. Publ.:247-261, 1993. ECOREF #45073	Repeated information
Fargasova,A.. Ecotoxicology of Metals Related to Freshwater Benthos. Gen. Physiol. Biophys.18(Focus Issue): 48-53, 1999. ECOREF #61824	Arsenate based study; EPA arsenic derivation based on arsenite
Ghosh,A.R., and P. Chakrabarti. Toxicity of Arsenic and Cadmium to a Freshwater Fish Notopterus notopterus. Environ. Ecol.8(2): 576-579, 1990. ECOREF #3440	Non-north american test species
Gupta,A.K., and P. Chakrabarti. Toxicity of Arsenic to Freshwater Fishes Mystus vittatus (Bloch) and Puntius javanicus (Blkr.). Environ. Ecol.11(4): 808-811, 1993. ECOREF #4456	Non-north american test species
Hamilton,S.J., and K.J. Buhl. Safety Assessment of Selected Inorganic Elements to Fry of Chinook Salmon (Oncorhynchus tshawytscha). Ecotoxicol. Environ. Saf.20(3): 307-324, 1990. ECOREF #3526	
Hamilton,S.J., and K.J. Buhl. Hazard Evaluation of Inorganics, Singly and in Mixtures, to Flannelmouth Sucker Catostomus latipinnis in the San Juan River, New Mexico. Ecotoxicol. Environ. Saf.38(3): 296-308, 1997. ECOREF #18979	Arsenate based study; EPA arsenic derivation based on arsenite
Hamilton,S.J., and K.J. Buhl. Hazard Assessment of Inorganics, Individually and in Mixtures, to Two Endangered Fish in the San Juan River, New Mexico. Environ. Toxicol. Water Qual.12:195-209, 1997. ECOREF #20368	Arsenate based study; EPA arsenic derivation based on arsenite
Hartwell,S.I., J.H. Jin, D.S. Cherry, and J.,Jr. Cairns. Toxicity Versus Avoidance Response of Golden Shiner, Notemigonus crysoleucas, to Five Metals. J. Fish Biol.35(3): 447-456, 1989. ECOREF #3286	Pulsed exposure to toxicant; did not follow standard methods
Hockett,J.R., and D.R. Mount. Use of Metal Chelating Agents to Differentiate Among Sources of Acute Aquatic Toxicity. Environ. Toxicol. Chem.15(10): 1687-1693, 1996. ECOREF #45021	
Hu,J., D. Wang, B.E. Forthaus, and J. Wang. Quantifying the Effect of Nanoparticles on As(V) Ecotoxicity Exemplified by Nano-Fe2O3 (Magnetic) and Nano-Al2O3. Environ. Toxicol. Chem.31(12): 2870-2876, 2012. ECOREF #165681	Nanoparticle study

Citation	Notes
Jeyasingham,K., and N. Ling. Acute Toxicity of Arsenic to Three Species of New Zealand Chironomids: Chironomus zealandicus, Chironomus sp. a and Polypedilum pavidus (Diptera, Chironomidae). Bull. Environ. Contam. Toxicol.64(5): 708-715, 2000. ECOREF #50648	Non-north american test species
Khangarot,B.S., A. Sehgal, and M.K. Bhasin. "Man and Biosphere" - Studies on the Sikkim Himalayas. Part 5: Acute Toxicity of Selected Heavy Metals on the Tadpoles of Rana hexadactyla. Acta Hydrochim. Hydrobiol.13(2): 259-263, 1985. ECOREF #11438	Non-north american test species
Klauda,R.J.. Acute and Chronic Effects of Waterborne Arsenic and Selenium on the Early Life Stages of Striped Bass (Morone saxatilis). Rep.No.JHU/APL PPRP-98, Rep.to Maryland Power Plant Siting Program, John Hopkins University, Laurel, MD:209 p., 1986. ECOREF #18109	Unable to retrieve article
Liber,K., L.E. Doig, and S.L. White-Sobey. Toxicity of Uranium, Molybdenum, Nickel, and Arsenic to Hyalella azteca and Chironomus dilutus in Water-Only and Spiked-Sediment Toxicity Tests. Ecotoxicol. Environ. Saf.74(5): 1171-1179, 2011. ECOREF #175087	
Lima,A.R., C. Curtis, D.E. Hammermeister, T.P. Markee, C.E. Northcott, and L.T. Brooke. Acute and Chronic Toxicities of Arsenic(III) to Fathead Minnows, Flagfish, Daphnids, and an Amphipod. Arch. Environ. Contam. Toxicol.13(5): 595-601, 1984. ECOREF #10695	Study used in previous EPA derivation
Liu,F., A. Gentles, and C.W. Theodorakis. Arsenate and Perchlorate Toxicity, Growth Effects, and Thyroid Histopathology in Hypothyroid Zebrafish Danio rerio. Chemosphere71(7): 1369-1376, 2008. ECOREF #111072	Arsenate based study; EPA arsenic derivation based on arsenite
Mayer,F.L.,Jr., and M.R. Ellersieck. Manual of Acute Toxicity: Interpretation and Data Base for 410 Chemicals and 66 Species of Freshwater Animals. USDI Fish and Wildlife Service, Publication No.160, Washington, DC:505 p., 1986. ECOREF #6797	This reference is a database; ecotox likely incorporated similar studies
Mount,D.I., and T.J. Norberg. A Seven-Day Life-Cycle Cladoceran Toxicity Test. Environ. Toxicol. Chem.3(3): 425-434, 1984. ECOREF #11181	Study used in previous EPA derivation
Palawski,D., J.B. Hunn, and F.J. Dwyer. Sensitivity of Young Striped Bass to Organic and Inorganic Contaminants in Fresh and Saline Waters. Trans. Am. Fish. Soc.114(5): 748-753, 1985. ECOREF #11334	Arsenate based study; EPA arsenic derivation based on arsenite
Rankin,M.G., and D.G. Dixon. Acute and Chronic Toxicity of Waterborne Arsenite to Rainbow Trout (Oncorhynchus mykiss). Can. J. Fish. Aquat. Sci.51(2): 372-380, 1994. ECOREF #14077	
Richie,J.P.,Jr., B.J. Mills, and C.A. Lang. The Verification of a Mammalian Toxicant Classification Using a Mosquito Screening Method. Fundam. Appl. Toxicol.4(6): 1029-1035, 1984. ECOREF #173907	Arsenate based study; EPA arsenic derivation based on arsenite
Shaw,J.R., K. Gabor, E. Hand, A. Lankowski, L. Durant, R. Thibodeau, C.R. Stanton, R. Barnaby, B. Coutermarsh, K.H. Kar. Role of Glucocorticoid Receptor in Acclimation of Killifish (Fundulus	Saltwater based study

Citation	Notes
heteroclitus) to Seawater and Effects of Arsenic. Am. J. Physiol., Regul. Integr. Comp. Physiol.292(2): R1052 - R1060, 2007. ECOREF #101073	
Shaw,J.R., S.P. Glaholt, N.S. Greenberg, R. Sierra-Alvarez, and C.L. Folt. Acute Toxicity of Arsenic to Daphnia pulex: Influence of Organic Functional Groups and Oxidation State. Environ. Toxicol. Chem.26(7): 1532-1537, 2007. ECOREF #100641	
Shukla,J.P., K.N. Shukla, and U.N. Dwivedi. Survivality and Impaired Growth in Arsenic Treated Fingerlings of Channa punctatus, a Fresh Water Murrel. Acta Hydrochim. Hydrobiol.15(3): 307-311, 1987. ECOREF #12594	Non-north american test species
Shukla,J.P., and K. Pandey. Toxicity and Long-Term Effect of Arsenic on the Gonadal Protein Metabolism in a Tropical Freshwater Fish, Colisa fasciatus (Bl. & Sch.). Acta Hydrochim. Hydrobiol.13(1): 127-131, 1985. ECOREF #11412	Non-north american test species
Spehar,R.L., and J.T. Fiandt. Acute and Chronic Effects of Water Quality Criteria-Based Metal Mixtures on Three Aquatic Species. Environ. Toxicol. Chem.5(10): 917-931, 1986. ECOREF #12093	Study used in EPA 1995 derivation
Tisler,T., and J. Zagorc-Koncan. Acute and Chronic Toxicity of Arsenic to Some Aquatic Organisms. Bull. Environ. Contam. Toxicol.69(3): 421-429, 2002. ECOREF #78709	Doesn't specify arsenic species
U.S. Environmental Protection Agency. Pesticide Ecotoxicity Database (Formerly: Environmental Effects Database (EEDB)). Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.:, 1992. ECOREF #344	This reference is to a database
Wang,D., J. Hu, B.E. Forthaus, and J. Wang. Synergistic Toxic Effect of Nano-Al2O3 and As(V) on Ceriodaphnia dubia. Environ. Pollut.159(10): 3003-3008, 2011. ECOREF #165959	Arsenate based study; EPA arsenic derivation based on arsenite

Open Literature

Table A2. List of open literature citations from EPA ECOTOX database reviewed for arsenic criteria derivation but did not meet acceptability requirements.

Citation	Notes
Gardner, S., Cline, G., Mwebi, N. and Rayburn, J., 2017. Developmental and interactive effects of arsenic and chromium to developing Ambystoma maculatum embryos: Toxicity, teratogenicity, and whole-body concentrations. Journal of Toxicology and Environmental Health, Part A, 80(2), pp.91-104.	12-day LC50

Freshwater Chronic

Table A3. List of citations from EPA ECOTOX database reviewed for arsenic freshwater chronic criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Chen,T.H., J.A. Gross, and W.H. Karasov. Chronic Exposure to Pentavalent Arsenic of Larval Leopard Frogs (Rana pipiens): Bioaccumulation and Reduced Swimming Performance. Ecotoxicology18(5): 587-593, 2009. ECOREF #119404	Study used arsenate; EPA used arsenite to derive arsenic criteria
Cockell,K.A., and W.J. Bettger. Investigations of the Gallbladder Pathology Associated with Dietary Exposure to Disodium Arsenate Heptahydrate in Juvenile Rainbow Trout (Oncorhynchus mykiss). Toxicology77(3): 233-248, 1993. ECOREF #7192	Study used arsenate; EPA used arsenite to derive arsenic criteria
Erickson,R.J., D.R. Mount, T.L. Highland, J.R. Hockett, E.N. Leonard, V.R. Mattson, T.D. Dawson, and K.G. Lott. Effects of Copper, Cadmium, Lead, and Arsenic in a Live Diet on Juvenile Fish Growth . Can. J. Fish. Aquat. Sci.67:1816-1826, 2010. ECOREF #156202	Study used arsenate; EPA used arsenite to derive arsenic criteria
Hoang,T.C., and S.J. Klaine. Influence of Organism Age on Metal Toxicity to Daphnia magna. Environ. Toxicol. Chem.26(6): 1198-1204, 2007. ECOREF #101846	Study limited to 1 test concentrations
Liber,K., L.E. Doig, and S.L. White-Sobey. Toxicity of Uranium, Molybdenum, Nickel, and Arsenic to Hyalella azteca and Chironomus dilutus in Water-Only and Spiked-Sediment Toxicity Tests. Ecotoxicol. Environ. Saf.74(5): 1171-1179, 2011. ECOREF #175087	Chronic toxicity value borrowed from another study
Tisler,T., and J. Zagorc-Koncan. Acute and Chronic Toxicity of Arsenic to Some Aquatic Organisms. Bull. Environ. Contam. Toxicol.69(3): 421-429, 2002. ECOREF #78709	Study used arsenate; EPA used arsenite to derive arsenic criteria
Vellinger,C., E. Gismondi, V. Felten, P. Rousselle, K. Mehennaoui, M. Parant, and P. Usseglio-Polatera. Single and Combined Effects of Cadmium and Arsenate in Gammarus pulex (Crustacea, Amphipoda): Understanding the Links Between Physiological and Behavioural Responses. Aquat. Toxicol.140/141:106-116, 2013. ECOREF #164550	Study used arsenate; EPA used arsenite to derive arsenic criteria
Okamoto, A., Masunaga, S. and Tatarazako, N., 2021. Chronic toxicity of 50 metals to Ceriodaphnia dubia. *Journal of Applied Toxicology*, *41*(3), pp.375-386.	10x threshold for ACR value and no MATC value reported; did not use flow through design
Irving, E.C., Lowell, R.B., Culp, J.M., Liber, K., Xie, Q. and Kerrich, R., 2008. Effects of arsenic speciation and low dissolved oxygen condition on the toxicity of arsenic to a lotic mayfly. *Environmental Toxicology and Chemistry: An International Journal*, *27*(3), pp.583-590.	12-day LC50 not relevant to ACR development
Gardner, S., Cline, G., Mwebi, N. and Rayburn, J., 2017. Developmental and interactive effects of arsenic and chromium to developing Ambystoma maculatum embryos: Toxicity, teratogenicity,	Study used arsenate; EPA used arsenite to derive arsenic criteria

Citation	Notes
and whole-body concentrations. *Journal of Toxicology and Environmental Health, Part A*, *80*(2), pp.91-104.	

Saltwater Acute

Table A4. List of citations from EPA ECOTOX database reviewed for arsenic saltwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Gaion,A., A. Scuderi, D. Pellegrini, and D. Sartori. The Influence of Solid Matrices on Arsenic Toxicity to Corophium orientale. Chem. Ecol.29(7): 653-659, 2013. ECOREF #166137	Non-north american test species; sediment study
Hwang,D.S., K.W. Lee, J. Han, H.G. Park, J. Lee, Y.M. Lee, and J.S. Lee. Molecular Characterization and Expression of Vitellogenin (Vg) Genes from the Cyclopoid Copepod, Paracyclopina nana Exposed to Heavy Metals. Comp. Biochem. Physiol. C Comp. Pharmacol. Toxicol.151(3): 360-368, 2010. ECOREF #153073	Not relevant endpoints
Lee,K.W., S. Raisuddin, D.S. Hwang, H.G. Park, H.U. Dahms, I.Y. Ahn, and J.S. Lee. Two-Generation Toxicity Study on the Copepod Model Species Tigriopus japonicus. Chemosphere72:1359-1365, 2008. ECOREF #104287	Non-north american test species
Lee,K.W., S. Raisuddin, J.S. Rhee, D.S. Hwang, I.T. Yu, Y.M. Lee, H.G. Park, and J.S. Lee. Expression of Glutathione S-Transferase (GST) Genes in the Marine Copepod Tigriopus japonicus Exposed to Trace Metals. Aquat. Toxicol.89(3): 158-166, 2008. ECOREF #107127	Non-north american test species
Liu,F., R.J. Kendall, and C.W. Theodorakis. Joint Toxicity of Sodium Arsenate and Sodium Perchlorate to Zebrafish Danio rerio Larvae. Environ. Toxicol. Chem.24(6): 1505-1507, 2005. ECOREF #110484	Arsenate used; EPA used arsenite to derive criteria
Shaw,J.R., K. Gabor, E. Hand, A. Lankowski, L. Durant, R. Thibodeau, C.R. Stanton, R. Barnaby, B. Coutermarsh, K.H. Kar. Role of Glucocorticoid Receptor in Acclimation of Killifish (Fundulus heteroclitus) to Seawater and Effects of Arsenic. Am. J. Physiol., Regul. Integr. Comp. Physiol.292(2): R1052 - R1060, 2007. ECOREF #101073	

Saltwater Chronic

Table A5. List of citations from EPA ECOTOX database reviewed for arsenic saltwater chronic criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Lee,K.W., S. Raisuddin, D.S. Hwang, H.G. Park, H.U. Dahms, I.Y. Ahn, and J.S. Lee. Two-Generation Toxicity Study on the Copepod Model Species Tigriopus japonicus. Chemosphere72:1359-1365, 2008. ECOREF #104287	Non-north american test species
Liu,F.J., J.S. Wang, and C.W. Theodorakis. Thyrotoxicity of Sodium Arsenate, Sodium Perchlorate, and Their Mixture in Zebrafish Danio rerio. Environ. Sci. Technol.40(10): 3429-3436, 2006. ECOREF #151957	Arsenate study; EPA derived arsenic criteria based on arsenite

Chromium VI

Freshwater Acute

Table A6. List of citations from EPA ECOTOX database reviewed for chromium vi freshwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Al-Akel,A.S.. Chromium Toxicity and Its Impact on Behavioural Responses in Freshwater Carp, Cyprinus carpio from Saudi Arabia. Pak. J. Zool.28(4): 361-363, 1996. ECOREF #46875	Non-north american species used
Al-Akel,A.S., and M.J.K. Shamsi. Hexavalent Chromium: Toxicity and Impact on Carbohydrate Metabolism and Haematological Parameters of Carp (Cyprinus carpio L.) from Saudi Arabia. Aquat. Sci.58(1): 24-30, 1996. ECOREF #19485	Non-north american species used
Anusuya,D., and I. Christy. Effects of Chromium Toxicity on Hatching and Development of Tadpoles of Bufo melanostictus. J. Environ. Biol.20(4): 321-323, 1999. ECOREF #47043	Non-north american species used
Arkhipchuk,V.V., C. Blaise, and M.V. Malinovskaya. Use of Hydra for Chronic Toxicity Assessment of Waters Intended for Human Consumption. Environ. Pollut.142(2): 200-211, 2006. ECOREF #90306	Ambient water subchronic study
Baral,A., R. Engelken, W. Stephens, J. Farris, and R. Hannigan. Evaluation of Aquatic Toxicities of Chromium and Chromium-Containing Effluents in Reference to Chromium Electroplating Industries. Arch. Environ. Contam. Toxicol.50(4): 496-502, 2006. ECOREF #119599	

Citation	Notes
Begum,G., J.V. Rao, and K. Srikanth. Oxidative Stress and Changes in Locomotor Behavior and Gill Morphology of Gambusia affinis Exposed to Chromium. Toxicol. Environ. Chem.88(2): 355-365, 2006. ECOREF #119520	
Bichara,D., N.B. Calcaterra, S. Arranz, P. Armas, and S.H. Simonetta. Set-up of an Infrared Fast Behavioral Assay Using Zebrafish (Danio rerio) Larvae, and Its Application in Compound Biotoxicity Screening. J. Appl. Toxicol.34:214-219, 2014. ECOREF #169111	Examined swimming behavior as endpoint
Buhl,K.J.. Relative Sensitivity of Three Endangered Fishes, Colorado Squawfish, Bonytail, and Razorback Sucker, to Selected Metal Pollutants. Ecotoxicol. Environ. Saf.37:186-192, 1997. ECOREF #18325	
Bulus Rossini,G.D., and A.E. Ronco. Sensitivity of Cichlasoma facetum (Cichlidae, Pisces) to Metals. Bull. Environ. Contam. Toxicol.72(4): 763-768, 2004. ECOREF #74230	Non-north american species used
Centeno,M.D.F., G. Persoone, and M.P. Goyvaerts. Cyst-Based Toxicity Tests. IX. The Potential of Thamnocephalus platyurus as Test Species in Comparison with Streptocephalus proboscideus (Crustacea: Branchiopoda: Anostraca). Environ. Toxicol. Water Qual.10(4): 275-282, 1995. ECOREF #14017	
Chu,K.W., and K.L. Chow. Synergistic Toxicity of Multiple Heavy Metals is Revealed by a Biological Assay Using a Nematode and Its Transgenic Derivative. Aquat. Toxicol.61(1/2): 53-64, 2002. ECOREF #65728	Transgenic nematode used in testing
Da Silva Kraus,L.A., A.C.T. Bonecker, N. De Almeida, and A. Vital. Acute Toxicity of Potassium Dichromate, Sodium Dodecyl Sulfate, Copper and Zinc to Poecilia vivipara (Osteichthyes, Cyprinodontiformes). Fresenius Environ. Bull.7(11/12): 654-658, 1998. ECOREF #60132	Non-north american species used
De Souza,J.P., L.S. Medeiros, E.U. Winkaler, and J.G. Machado-Neto. Acute Toxicity and Environmental Risk of Diflubenzuron to Daphnia magna, Poecilia reticulata and Lemna minor in the Absence and Presence of Sediment. Pesticidas21:1-12, 2011. ECOREF #174961	Non-north american species used
Di Marzio,W.D., D. Castaldo, C. Pantani, A. Di Cioccio, T. Di Lorenzo, M.E. Saenz, and D.M.P. Galassi. Relative Sensitivity of Hyporheic Copepods to Chemicals. Bull. Environ. Contam. Toxicol.82(4): 488-491, 2009. ECOREF #114244	
Diao,J., P. Xu, P. Wang, D. Lu, Y. Lu, and Z. Zhou. Enantioselective Degradation in Sediment and Aquatic Toxicity to Daphnia magna of the Herbicide Lactofen Enantiomers. J. Agric. Food Chem.58(4): 2439-2445, 2010. ECOREF #152904	Herbicide used in testing; sediment study
Elumalai,M., C. Antunes, and L. Guilhermino. Effects of Single Metals and Their Mixtures on Selected Enzymes of Carcinus maenas. Water Air Soil Pollut.141(1-4): 273-280, 2002. ECOREF #72944	Ambient estuary water used in testing

Citation	Notes
Fargasova,A.. Ecotoxicology of Metals Related to Freshwater Benthos. Gen. Physiol. Biophys.18(Focus Issue): 48-53, 1999. ECOREF #61824	
Gutierrez,M.F., A.M. Gagneten, and J.C. Paggi. Copper and Chromium Alter Life Cycle Variables and the Equiproportional Development of the Freshwater Copepod Notodiaptomus conifer (Sars.). Water Air Soil Pollut.213:275-286, 2010. ECOREF #169526	
Hockett,J.R., and D.R. Mount. Use of Metal Chelating Agents to Differentiate Among Sources of Acute Aquatic Toxicity. Environ. Toxicol. Chem.15(10): 1687-1693, 1996. ECOREF #45021	Unclear if resulting LC50 mixed with chelating agents
Joshi,S.N., and H.S. Patil. Differential Toxicity of Four Chromium Salts to Male Skipper Frog Rana cyanophlyctis. Environ. Ecol.12(1): 36-38, 1994. ECOREF #17526	Non-north american test species used
Kazlauskiene,N., A. Burba, and G. Svecevicius. Acute Toxicity of Five Galvanic Heavy Metals to Hydrobionts. Ekologiia1:33-36, 1994. ECOREF #17573	
Kungolos,A., S. Hadjispyrou, P. Samaras, M. Petala, V. Tsiridis, K. Aravossis, and G.P. Sakellaropoulos. Assessment of Toxicity and Bioaccumulation of Organotin Compounds. In: Proceedings of the 7th International Conference on Environmental Science and Technology, Syros, Greece:499-505, 2001. ECOREF #68179	Hexavalent chromium not used in study
Li,Y., F. Dong, X. Liu, J. Xu, Y. Han, and Y. Zheng. Chiral Fungicide Triadimefon and Triadimenol: Stereoselective Transformation in Greenhouse Crops and Soil, and Toxicity to Daphnia magna. J. Hazard. Mater.265:115-123, 2014. ECOREF #170571	Fungicide based study
Li,Y., F. Dong, X. Liu, J. Xu, Y. Han, and Y. Zheng. Enantioselectivity in Tebuconazole and Myclobutanil Non-Target Toxicity and Degradation in Soils. Chemosphere122:145-153, 2015. ECOREF #178194	Fungicide based study
Lin,K., S. Zhou, C. Xu, and W. Liu. Enantiomeric Resolution and Biotoxicity of Methamidophos. J. Agric. Food Chem.54(21): 8134-8138, 2006. ECOREF #99572	Pesticide based study
Madoni,P., D. Davoli, G. Gorbi, and L. Vescovi. Toxic Effect of Heavy Metals on the Activated Sludge Protozoan Community. Water Res.30(1): 135-141, 1996. ECOREF #16363	Test organisms from sludge
Madoni,P., D. Davoli, and G. Gorbi. Acute Toxicity of Lead, Chromium, and Other Heavy Metals to Ciliates from Activated Sludge Plants. Bull. Environ. Contam. Toxicol.53(3): 420-425, 1994. ECOREF #13671	Test organisms from sludge
Madoni,P., and M.G. Romeo. Acute Toxicity of Heavy Metals Towards Freshwater Ciliated Protists. Environ. Pollut.141(1): 1-7, 2006. ECOREF #95678	Single celled organism; inappropriate test organism
Maestre,Z., M. Martinez-Madrid, and P. Rodriguez. Monitoring the Sensitivity of the Oligochaete Tubifex tubifex in Laboratory Cultures Using Three Toxicants. Ecotoxicol. Environ. Saf.72:2083-2089, 2009. ECOREF #118134	

Citation	Notes
Mohammed,A.. Comparative Sensitivities of the Tropical Cladoceran, Ceriodaphnia rigaudii and the Temperate Species Daphnia magna to Seven Toxicants. Toxicol. Environ. Chem.89(2): 347-352, 2007. ECOREF #102662	Tests conducted in 24 well plates and the test chamber volume to organism ratio was too low. Possible organism density related effects.
Mohammed,A., and J.B.R. Agard. Comparative Sensitivity of Three Tropical Cladoceran Species (Diaphanosoma brachyurum, Ceriodaphnia rigaudii and Moinodaphnia macleayi) to Six Chemicals. J. Environ. Sci. Health. Part A, Environ. Sci. Eng. Toxic Hazard. Substance Control41(12): 2713-2720, 2006. ECOREF #101029	Tests conducted in 24 well plates and the test chamber volume to organism ratio was too low. Possible organism density related effects.
Nalecz-Jawecki,G., and J. Sawicki. Toxicity of Inorganic Compounds in the Spirotox Test: A Miniaturized Version of the Spirostomum ambiguum Test. Arch. Environ. Contam. Toxicol.34(1): 1-5, 1998. ECOREF #18997	Not relevant
Natale,G.S., L.L. Ammassari, N.G. Basso, and A.E. Ronco. Acute and Chronic Effects of Cr(VI) on Hypsiboas pulchellus Embryos and Tadpoles. Dis. Aquat. Org.72(3): 261-267, 2006. ECOREF #101072	Non-north american test species used
Oliveira-Filho,E.C., and F.J.R. Paumgartten. Comparative Study on the Acute Toxicities of alpha, beta, gamma, and delta Isomers of Hexachlorocyclohexane to Freshwater Fishes. Bull. Environ. Contam. Toxicol.59(6): 984-988, 1997. ECOREF #18622	Study does not involve chromium
Perez-Legaspi,I.A., and R. Rico-Martinez. Acute Toxicity Tests on Three Species of the Genus Lecane (Rotifera: Monogononta). Hydrobiologia446-447:375-381, 2001. ECOREF #65813	
Rathore,R.S., and B.S. Khangarot. Effects of Temperature on the Sensitivity of Sludge Worm Tubifex tubifex Muller to Selected Heavy Metals. Ecotoxicol. Environ. Saf.53(1): 27-36, 2002. ECOREF #69566	
Safadi,R.S.. The Use of Freshwater Planarians in Acute Toxicity Tests with Heavy Metals. Verh. Int. Ver. Theor. Angew. Limnol.26(5): 2391-2392, 1998. ECOREF #83191	Lacks detailed methods such as controls, methods, purity, etc.
Sivakami,R., G. Premkishore, and M.R. Chandran. Effect of Chromium on the Metabolism and Biochemical Composition of Selected Tissues in the Freshwater Catfish Mystus vittatus. Environ. Ecol.12(2): 259-266, 1994. ECOREF #12676	Non-north american test species used
Sivakumar,S., R. Karuppasamy, and S. Subathra. Acute Toxicity and Behavioural Changes in Freshwater Fish Mystus vittatus (Bloch) Exposed to Chromium (VI) Oxide. Nat. Environ. Pollut. Technol.5(3): 381-388, 2006. ECOREF #119339	Non-north american test species used
Sorensen,M.A., P.D. Jensen, W.E. Walton, and J.T. Trumble. Acute and Chronic Activity of Perchlorate and Hexavalent Chromium Contamination on the Survival and Development of Culex quinquefasciatus Say (Diptera: Culicidae). Environ. Pollut.144(3): 759-764, 2006. ECOREF #96296	

Citation	Notes
Sornaraj,R., P. Baskaran, and S. Thanalakshmi. Effects of Heavy Metals on Some Physiological Responses of Air-Breathing Fish Channa punctatus (Bloch). Environ. Ecol.13(1): 202-207, 1995. ECOREF #17380	Non-north american test species used
Sotero-Santos,R.B., O. Rocha, and J. Povinelli. Toxicity of Ferric Chloride Sludge to Aquatic Organisms. Chemosphere68(4): 628-636, 2007. ECOREF #118678	Sludge used in testing
Tsui,M.T.K., W.X. Wang, and L.M. Chu. Influence of Glyphosate and Its Formulation (Roundup) on the Toxicity and Bioavailability of Metals to Ceriodaphnia dubia. Environ. Pollut.138(1): 59-68, 2005. ECOREF #87704	Pesticide mixture study; LC50 not provided
Twagilimana,L., J. Bohatier, CA Groliere, F. Bonnemoy, and D. Sargos. A New Low-Cost Microbiotest with the Protozoan Spirostomum teres: Culture Conditions and Assessment of Sensitivity of the Ciliate to 14 Pure Chemicals. Ecotoxicol. Environ. Saf.41(3): 231-244, 1998. ECOREF #20057	Microbiotest not relevant
Vedamanikam,V.J., and N.A.M. Shazilli. The Effect of Multi-Generational Exposure to Metals and Resultant Change in Median Lethal Toxicity Tests Values over Subsequent Generations. Bull. Environ. Contam. Toxicol.80(1): 63-67, 2008. ECOREF #111291	
Wong,C.K., and A.P. Pak. Acute and Subchronic Toxicity of the Heavy Metals Copper, Chromium, Nickel, and Zinc, Individually and in Mixture, to the Freshwater Copepod Mesocyclops pehpeiensis. Bull. Environ. Contam. Toxicol.73(1): 190-196, 2004. ECOREF #80006	Non-north american test species
Yang,H.B., Z. Ya-Zhou, Y. Tang, G. Hui-Qin, F. Guo, S. Wei-Hua, L. Shu-Shen, H. Tan, and F. Chen. Antioxidant Defence System is Responsible for the Toxicological Interactions of Mixtures: A Case Study on PFOS and PFOA in Daphnia magna. Sci. Total Environ.667:435-443, 2019. ECOREF #182580	Test did not use chromium
Zhang,Q., and C. Wang. Toxicity of Binary Mixtures of Enantiomers in Chiral Organophosphorus Insecticides: The Significance of Joint Effects Between Enantiomers. Chirality25(11): 787-792, 2013. ECOREF #165491	Pesticide study; did not use chromium

Open Literature

Table A7. List of open literature citations from EPA ECOTOX database reviewed for chromium vi criteria derivation but did not meet acceptability requirements.

Citation	Notes
Gardner, S., Cline, G., Mwebi, N. and Rayburn, J., 2017. Developmental and interactive effects of arsenic and chromium to developing Ambystoma maculatum embryos: Toxicity, teratogenicity, and whole-body concentrations. Journal of Toxicology and Environmental Health, Part A, 80(2), pp.91-104.	12-day LC50
Hernández-Ruiz, E., Alvarado-Flores, J., Rubio-Franchini, I., Ventura-Juárez, J. and Rico-Martínez, R., 2016. Adverse effects and bioconcentration of chromium in two freshwater rotifer species. Chemosphere, 158, pp.107-115.	Low organism to volume ratio
Hose, G.C., Symington, K., Lott, M.J. and Lategan, M.J., 2016. The toxicity of arsenic (III), chromium (VI) and zinc to groundwater copepods. Environmental Science and Pollution Research, 23, pp.18704-18713.	Groundwater test organisms; non-north American test species; field collected organisms with no exposure information
Okamoto, A., Masunaga, S. and Tatarazako, N., 2021. Chronic toxicity of 50 metals to Ceriodaphnia dubia. Journal of Applied Toxicology, 41(3), pp.375-386.	Inhibition concentrations reported; very little details on test methods, ACR based on two different organisms; did not use flow through design

Freshwater Chronic

Table A8. List of citations from EPA ECOTOX database reviewed for chromium vi freshwater chronic criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Baral,A., R. Engelken, W. Stephens, J. Farris, and R. Hannigan. Evaluation of Aquatic Toxicities of Chromium and Chromium-Containing Effluents in Reference to Chromium Electroplating Industries. Arch. Environ. Contam. Toxicol.50(4): 496-502, 2006. ECOREF #119599	
Carriquiriborde,P., and A.E. Ronco. Distinctive Accumulation Patterns of Cd(II), Cu(II), and Cr(VI) in Tissue of the South American Teleost, Pejerrey (Odontesthes bonariensis). Aquat. Toxicol.86(2): 313-322, 2008. ECOREF #117068	Acceptable but ACR cannot be calculated
Diamantino,T.C., L. Guilhermino, E. Almeida, and A.M.V.M. Soares. Toxicity of Sodium Molybdate and Sodium Dichromate to Daphnia magna Straus Evaluated in Acute, Chronic, and	

Citation	Notes
Acetylcholinesterase Inhibition Tests. Ecotoxicol. Environ. Saf.45(3): 253-259, 2000. ECOREF #48695	
Gutierrez,M.F., A.M. Gagneten, and J.C. Paggi. Copper and Chromium Alter Life Cycle Variables and the Equiproportional Development of the Freshwater Copepod Notodiaptomus conifer (Sars.). Water Air Soil Pollut.213:275-286, 2010. ECOREF #169526	
Mishra,A.K., and B. Mohanty. Chronic Exposure to Sublethal Hexavalent Chromium Affects Organ Histopathology and Serum Cortisol Profile of a Teleost, Channa punctatus (Bloch). Sci. Total Environ.407(18): 5031-5038, 2009. ECOREF #119189	Non-north american test species used
Natale,G.S., L.L. Ammassari, N.G. Basso, and A.E. Ronco. Acute and Chronic Effects of Cr(VI) on Hypsiboas pulchellus Embryos and Tadpoles. Dis. Aquat. Org.72(3): 261-267, 2006. ECOREF #101072	
Nguyen,L.T.H., and C.R. Janssen. Comparative Sensitivity of Embryo-Larval Toxicity Assays with African Catfish (Clarias gariepinus) and Zebra Fish (Danio rerio). Environ. Toxicol.16(6): 566-571, 2001. ECOREF #68928	Acceptable but ACR cannot be calculated
Oner,M., G. Atli, and M. Canli. Effects of Metal (Ag, Cd, Cr, Cu, Zn) Exposures on Some Enzymatic and Non-Enzymatic Indicators in the Liver of Oreochromis niloticus. Bull. Environ. Contam. Toxicol.82(3): 317-321, 2009. ECOREF #112714	Non-north american test species used
Pickering,Q.H., and J.M. Lazorchak. Evaluation of the Robustness of the Fathead Minnow, Pimephales promelas, Larval Survival and Growth Test, U.S. EPA Method 1000.0. Environ. Toxicol. Chem.14(4): 653-659, 1995. ECOREF #45200	Acceptable but ACR cannot be calculated
Sofyan,A.. Toxicity of Metals to Green Algae and Ceriodaphnia dubia: The Importance of Water Column and Dietary Exposures. Ph.D.Thesis, University of Kentucky, Lexington, KY:161 p., 2004. ECOREF #78692	Chromium III study

Saltwater Acute

Table A9. List of citations from EPA ECOTOX database reviewed for chromium vi saltwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Andersen,H.R., L. Wollenberger, B. Halling-Sorensen, and K.O. Kusk. Development of Copepod Nauplii to Copepodites - a Parameter for Chronic Toxicity Including Endocrine Disruption. Environ. Toxicol. Chem.20(12): 2821-2829, 2001. ECOREF #66691	

Citation	Notes
Bookhout,C.G., R.J. Monroe, R.B.,Jr. Forward, and J.D.,Jr. Costlow. Effects of Hexavalent Chromium on Development of Crabs, Rhithropanopeus harrisii and Callinectes sapidus. Water Air Soil Pollut.21(1-4): 199-216, 1984. ECOREF #7013	Used in previous 1984 derivation
Bryant,V., D.S. McLusky, K. Roddie, and D.M. Newbery. Effect of Temperature and Salinity on the Toxicity of Chromium to Three Estuarine Invertebrates (Corophium volutator, Macoma balthica, Nereis diversicolor). Mar. Ecol. Prog. Ser.20(1-2): 137-149, 1984. ECOREF #11873	Threshold reported as LT50 (time based)
Cardin,J.A.. Results of Acute Toxicity Tests Conducted with Chromium at ERL, Narragansett. U.S.EPA, Narragansett, RI:2 p., 1985. ECOREF #3754	Unable to locate
D'Asaro,C.N.. Effects Assessment of Selected Chemicals on Estuarine and Marine Organisms. EPA-600/X-85/056, Environmental Research Laboratory, U.S. Environmental Protection Agency, Gulf Breeze,FL:77 p., 1985. ECOREF #82668	
Dave,G., E. Nilsson, and A.S. Wernersson. Sediment and Water Phase Toxicity and UV-Activation of Six Chemicals Used in Military Explosives. Aquat. Ecosyst. Health Manag.3(3): 291-299, 2000. ECOREF #157913	Study not relevant to Cr6 thresholds
Dorn,P.B., J.H.,Jr. Rodgers, K.M. Jop, J.C. Raia, and K.L. Dickson. Hexavalent Chromium as a Reference Toxicant in Effluent Toxicity Tests. Environ. Toxicol. Chem.6(6): 435-444, 1987. ECOREF #12660	
Ek,H., E. Nilsson, G. Birgersson, and G. Dave. TNT Leakage Through Sediment to Water and Toxicity to Nitocra spinipes. Ecotoxicol. Environ. Saf.67(3): 341-348, 2007. ECOREF #97664	Study not relevant to Cr6 thresholds
Espiritu,E.Q., C.R. Janssen, and G. Persoone. Cyst-Based Toxicity Tests. VII. Evaluation of the 1-h Enzymatic Inhibition Test (Fluotox) with Artemia nauplii. Environ. Toxicol. Water Qual.10:25-34, 1995. ECOREF #16031	1985 EPA guidance suggests not using Artemia data
Gao,S., and D. Zou. Acute Toxicity of Copper, Mercury and Chromium to Larvae of Penaeus penicillatus Alcock. Mar. Sci. Bull. (Haiyang-Tongbao Shuangyuekan)13(2): 28-32, 1994. ECOREF #16613	Non-north american tests species
Garcia,K., J.B.R. Agard, and A. Mohammed. Comparative Sensitivity of a Tropical Mysid Metamysidopsis insularis and the Temperate Species Americamysis bahia to Six Toxicants. Toxicol. Environ. Chem.90(4): 779-785, 2008. ECOREF #117932	
Hori,H., M. Tateishi, K. Takayanagi, and H. Yamada. Applicability of Artificial Seawater as a Rearing Seawater for Toxicity Tests of Hazardous Chemicals by Marine Fish Species. Nippon Suisan Gakkaishi(4): 614-622, 1996. ECOREF #16999	Wrong language

Citation	Notes
Hutchinson,T.H., T.D. Williams, and G.J. Eales. Toxicity of Cadmium, Hexavalent Chromium and Copper to Marine Fish Larvae (Cypinodon variegatus) and Copepods (Tisbe battagliai). Mar. Environ. Res.38(4): 275-290, 1994. ECOREF #14137	
Jop,K.M.. Acute and Rapid-Chronic Toxicity of Hexavalent Chromium to Five Marine Species. ASTM Spec. Tech. Publ.12:251-260, 1989. ECOREF #198	
Jop,K.M., J.H.,Jr. Rodgers, P.B. Dorn, and K.L. Dickson. Use of Hexavalent Chromium as a Reference Toxicant in Aquatic Toxicity Tests. ASTM Spec. Tech. Publ.9:390-403, 1986. ECOREF #7772	
Kidwai,S., and M. Ahmed. Heavy Metal Bioassays on Selected Fauna from the Karachi Coast (Northwest Arabian Sea). Pak. J. Zool.30(2): 147-157, 1999. ECOREF #62226	Non-north american test species
Kissa,E., M. Moraitou-Apostolopoulou, and V. Kiortsis. Effects of Four Heavy Metals on Survival and Hatching Rate of Artemia salina (L.). Arch. Hydrobiol.102(2): 255-264, 1984. ECOREF #11259	1985 EPA guidance suggests not using Artemia data
Krishnani,K.K., I.S. Azad, M. Kailasam, A.R. Thirunavukkarasu, B.P. Gupta, K.O. Joseph, M. Muralidhar, and M. Abraham. Acute Toxicity of Some Heavy Metals to Lates calcarifer Fry with a Note on Its Histopathological Manifestations. J. Environ. Sci. Health. Part A, Environ. Sci. Eng. Toxic Hazard. Substance Control38(4): 645-655, 2003. ECOREF #78035	Non-north american test species
Lussier,S.M., J.H. Gentile, and J. Walker. Acute and Chronic Effects of Heavy Metals and Cyanide on Mysidopsis bahia (Crustacea: Mysidacea). Aquat. Toxicol.7(1/2): 25-35, 1985. ECOREF #11331	
Marino-Balsa,J.C., E. Poza, E. Vazquez, and R. Beiras. Comparative Toxicity of Dissolved Metals to Early Larval Stages of Palaemon serratus, Maja squinado, and Homarus gammarus (Crustacea: Decapoda). Arch. Environ. Contam. Toxicol.39(3): 345-351, 2000. ECOREF #56995	Doesn't specify what type of chromium
McLusky,D.S., and L. Hagerman. The Toxicity of Chromium, Nickel and Zinc: Effects of Salinity and Temperature, and the Osmoregulatory Consequences in the Mysid Praunus flexuosus. Aquat. Toxicol.10:225-238, 1987. ECOREF #6039	Non-north american test species
Miliou,H., G. Verriopoulos, D. Maroulis, D. Bouloukos, and M. Moraitou-Apostolopoulou. Influence of Life-History Adaptations on the Fidelity of Laboratory Bioassays for the Impact of Heavy Metals (Co2+ and Cr6+) on Tolerance and Population Dynamics of Tisbe holothuriae. Mar. Pollut. Bull.40(4): 352-359, 2000. ECOREF #52250	Non-north american test species
Mortimer,M.R., and G.J. Miller. Susceptibility of Larval and Juvenile Instars of the Sand Crab, Portunus pelagicus (L.), to Sea Water Contaminated by Chromium, Nickel or Copper. Aust. J. Mar. Freshw. Res.45(7): 1107-1121, 1994. ECOREF #16331	Non-north american test species

Citation	Notes
Parametrix Inc.. Acute Toxicity of Sodium Cyanide to Marine Copepods (Acartia tonsa). Report 3555, Parametrix Environmental Research Laboratory, Albany, OR:158 p., 2006. ECOREF #167149	Not relevant; study used cyanide
Parker,J.G.. The Effects of Selected Chemicals and Water Quality on the Marine Polychaete Ophryotrocha diadema. Water Res.18(7): 865-868, 1984. ECOREF #10890	Not relevant; multi-generational study with tolerant species
Ramirez,P., G. Barrera, and C. Rosas. Effects of Chromium and Cadmium upon Respiration and Survival of Callinectes similis. Bull. Environ. Contam. Toxicol.43(6): 850-857, 1989. ECOREF #2549	Field collect organisms; only 4 test concentrations
Rao,K.R., and D.G. Doughtie. Histopathological Changes in Grass Shrimp Exposed to Chromium, Pentachlorophenol and Dithiocarbamates. Mar. Environ. Res.14:371-395, 1984. ECOREF #13291	Not relevant
Rao,K.R., and P.J. Conklin. Molt-Related Susceptibility and Regenerative Limb Growth as Sensitive Indicators of Aquatic Pollutant Toxicity to Crustaceans. In: M.F.Thompson, R.Sarojini, and R.Nagabhushanam (Eds.), Biology of Benthic Marine Organisms: Techniques and Methods as Applied to the Indian Ocean, A.A.Balkema, Rotterdam, Netherlands:523-534, 1986. ECOREF #14267	Endpoints were not relevant to criteria development
Reish,D.J., and J.A. Lemay. Toxicity and Bioconcentration of Metals and Organic Compounds by Polychaeta. Ophelia5(suppl.): 653-660, 1991. ECOREF #3785	
Savant,K.B., and G.V. Nilkanth. On Comparative Studies of Acute Toxicity of Hexavalent Chromium and Selenium to Scylla serrata (Forskal). Pollut. Res.10(4): 239-243, 1991. ECOREF #81814	Non-north american test species
Taylor,D., B.G. Maddock, and G. Mance. The Acute Toxicity of Nine "Grey List" Metals (Arsenic, Boron, Chromium, Copper, Lead, Nickel, Tin, Vanadium and Zinc) to Two Marine Fish Species. Aquat. Toxicol.7(3): 135-144, 1985. ECOREF #11451	
U.S. Environmental Protection Agency. Pesticide Ecotoxicity Database (Formerly: Environmental Effects Database (EEDB)). Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.:, 1992. ECOREF #344	Database reference
Van der Meer,C., C. Teunissen, and T.F.M. Boog. Toxicity of Sodium Chromate and 3,4-Dichloroaniline to Crustaceans. Bull. Environ. Contam. Toxicol.40(2): 204-211, 1988. ECOREF #2419	Lacking informaton on methods such as test concentrations and replicates
Verriopoulos,G., A.V. Catsiki, A. Pantelidou, and M. Moraitou-Apostolopoulo. Studies on the Impact of Chromium to the Marine Gastropod Monodonta turbinata (Toxicity, Bioaccumulation, Acclimation). Rev. Int. Oceanogr. Med.93:103-118, 1990. ECOREF #18853	

Citation	Notes
Verriopoulos,G., M. Moraitou-Apostolopoulou, and E. Milliou. Combined Toxicity of Four Toxicants (Cu, Cr, Oil, Oil Dispersant) to Artemia salina. Bull. Environ. Contam. Toxicol.38(3): 483-490, 1987. ECOREF #9336	
Vranken,G., R. Vanderhaeghen, and C. Heip. Effects of Pollutants on Life-History Parameters of the Marine Nematode Monhystera disjuncta. ICES J. Mar. Sci.48:325-334, 1991. ECOREF #7215	Non-north american test species
Wong,C.K., K.H. Chu, K.W. Tang, T.W. Tam, and L.J. Wong. Effects of Chromium, Copper and Nickel on Survival and Feeding Behaviour of Metapenaeus ensis Larvae and Postlarvae (Decapoda: Penaeidae). Mar. Environ. Res.36(2): 63-78, 1993. ECOREF #4127	Non-north american test species

Saltwater Chronic

Table A10. List of citations from EPA ECOTOX database reviewed for chromium vi saltwater chronic criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Ahsanullah,M., and A.R. Williams. Sublethal Effects and Bioaccumulation of Cadmium, Chromium, Copper, and Zinc in the Marine Amphipod Allorchestes compressa. Mar. Biol.108:59-65, 1991. ECOREF #331	LC50 reported as greater than value
Andersen,H.R., L. Wollenberger, B. Halling-Sorensen, and K.O. Kusk. Development of Copepod Nauplii to Copepodites - a Parameter for Chronic Toxicity Including Endocrine Disruption. Environ. Toxicol. Chem.20(12): 2821-2829, 2001. ECOREF #66691	
Goodfellow,W.L.,Jr., and W.J. Rue. Evaluation of a Chronic Estimation Toxicity Test Using Mysidopsis bahia. ASTM Spec. Tech. Publ.12:333-344, 1989. ECOREF #2048	
Hutchinson,T.H., T.D. Williams, and G.J. Eales. Toxicity of Cadmium, Hexavalent Chromium and Copper to Marine Fish Larvae (Cypinodon variegatus) and Copepods (Tisbe battagliai). Mar. Environ. Res.38(4): 275-290, 1994. ECOREF #14137	
Jop,K.M.. Acute and Rapid-Chronic Toxicity of Hexavalent Chromium to Five Marine Species. ASTM Spec. Tech. Publ.12:251-260, 1989. ECOREF #198	
Lussier,S.M., J.H. Gentile, and J. Walker. Acute and Chronic Effects of Heavy Metals and Cyanide on Mysidopsis bahia (Crustacea: Mysidacea). Aquat. Toxicol.7(1/2): 25-35, 1985. ECOREF #11331	
McCulloch,W.L., and W.J. Rue. Evaluation of Seven-Day Chronic Toxicity Estimation Test Using Cyprinodon variegatus. ASTM Spec. Tech. Publ.12:355-364, 1989. ECOREF #13864	

Citation	Notes
Mortimer,M.R., and G.J. Miller. Susceptibility of Larval and Juvenile Instars of the Sand Crab, Portunus pelagicus (L.), to Sea Water Contaminated by Chromium, Nickel or Copper. Aust. J. Mar. Freshw. Res.45(7): 1107-1121, 1994. ECOREF #16331	
Van der Meer,C., C. Teunissen, and T.F.M. Boog. Toxicity of Sodium Chromate and 3,4-Dichloroaniline to Crustaceans. Bull. Environ. Contam. Toxicol.40(2): 204-211, 1988. ECOREF #2419	Methods lacking information such as test concentrations and replicates

Cyanide

Freshwater Acute

Table A11. List of citations from EPA ECOTOX database reviewed for cyanide freshwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Alabaster,J.S., D.G. Shurben, and M.J. Mallett. The Acute Lethal Toxicity of Mixtures of Cyanide and Ammonia to Smolts of Salmon, Salmo salar L. at Low Concentrations of Dissolved Oxygen. J. Fish Biol.22(2): 215-222, 1983. ECOREF #10252	
Bailey,H.C., D.H.W. Liu, and H.A. Javitz. Time/Toxicity Relationships in Short-Term Static, Dynamic, and Plug-Flow Bioassays. ASTM Spec. Tech. Publ.:193-212, 1985. ECOREF #7398	Study explains a testing method and not test results
Beleau,M.H., and J.A. Bartosz. Colorado River Fisheries Project Acute Toxicity of Selected Chemicals: Data Base. In: Rep.No.6,Dep.of Fish.Resour.,Univ.of Idaho,Moscow,ID:243-254, 1982. ECOREF #86404	Database reference
Broderius,S., and M. Kahl. Acute Toxicity of Organic Chemical Mixtures to the Fathead Minnow. Aquat. Toxicol.6:307-322, 1985. ECOREF #14128	Sand and gravel as media in testing
Brooke,L.T., D.J. Call, D.L. Geiger, and C.E. Northcott. Acute Toxicities of Organic Chemicals to Fathead Minnows (Pimephales promelas), Vol. 1. Center for Lake Superior Environmental Studies, University of Wisconsin-Superior, Superior, WI:414 p., 1984. ECOREF #12448	Repeat of data
Buccafusco,R.J., S.J. Ells, and G.A. LeBlanc. Acute Toxicity of Priority Pollutants to Bluegill (Lepomis macrochirus). Bull. Environ. Contam. Toxicol.26(4): 446-452, 1981. ECOREF #5590	No cyanide info available
Call,D.J., L.T. Brooke, D.H. Hammermeister, C.E. Northcott, and A.D. Hoffman. Variation of Acute Toxicity with Water Source. Center for Lake Superior Environmental Studies, Report No. LSRI0273:58 p., 1983. ECOREF #152135	EPA used in 1984 cyanide derivation

Citation	Notes
Call,D.J., L.T. Brooke, N. Ahmad, and J.E. Richter. Toxicity and Metabolism Studies with EPA (Environmental Protection Agency) Priority Pollutants and Related Chemicals in Freshwater Organisms. EPA 600/3-83-095, U.S.EPA, Duluth, MN:120 p., 1983. ECOREF #10579	EPA used in 1984 cyanide derivation
Call,D.J., and L.T. Brooke. Report on Stonefly Toxicity Tests with Priority Pollutants. Ctr.for Lake Superior Environ.Stud., Univ.of Wisconsin-Superior, Superior, WI (Memo to R.E.Siefert, U.S.EPA, Duluth, MN):2 p., 1982. ECOREF #9498	EPA used in 1984 cyanide derivation
Calleja,M.C., G. Persoone, and P. Geladi. Comparative Acute Toxicity of the First 50 Multicentre Evaluation of In Vitro Cytotoxicity Chemicals to Aquatic Non-vertebrates. Arch. Environ. Contam. Toxicol.26(1): 69-78, 1994. ECOREF #13669	Not relevant; cytotoxicity study
Collins,S.. Toxicity of Deicing Salt Components to Early Amphibian Life Stages. M.S. Thesis, Saint Mary's University, Canada:109 p., 2010. ECOREF #157604	Not relevant; test compound is ferrocyanide
David,M., H. Ramesh, S.P. Deshpande, S.G. Chebbi, and G. Krishnamurthy. Respiratory Distress and Behavioral Changes Induced by Sodium Cyanide in the Fresh Water Teleost, Cyprinus carpio (Linnaeus). J. Basic Clin. Physiol. Pharmacol.18(2): 55-65, 2007. ECOREF #118154	Non-north american test species
Dube,P.N., and B.B. Hosetti. Modulation in the Protein Metabolism by Subacute Sodium Cyanide Intoxication in the Freshwater Fish, Labeo rohita (Hamilton). Drug Chem. Toxicol.35(1): 25-31, 2012. ECOREF #160876	Non-north american test species
Dube,P.N., and B.B. Hosetti. Inhibition of ATPase Activity in the Freshwater Fish Labeo rohita (Hamilton) Exposed to Sodium Cyanide. Toxicol. Mech. Methods21(8): 591-595, 2011. ECOREF #164481	Non-north american test species
ENSR Corporation. Acute Toxicity of Cyanide to the Frog, Rana pipiens, in Horsetooth Reservoir Water Under Flow-Through Test Conditions. Report 8503-124-020-075, ENSR Corporation, Fort Collins, CO:45 p., 2005. ECOREF #166858	Unable to locate article
ENSR Corporation. Acute Toxicity of Cyanide to the Frog, Rana berlandieri, in Horsetooth Reservoir Water Under Flow-Through Test Conditions. Report 8503-124-020-076, ENSR Corporation, Fort Collins, CO:38 p., 2005. ECOREF #166859	Unable to locate article
ENSR Corporation. Acute Toxicity of Cyanide to the Frog, Xenopus laevis, in Horsetooth Reservoir Water Under Flow-Through Test Conditions. Report 8503-124-020-074, ENSR Corporation, Fort Collins, CO:50 p., 2005. ECOREF #166860	Unable to locate article
Elaziz,M.A., M. Moustafa, and A.E. Eissa. Assessment of Acute and Chronic Toxicity of Sodium Cyanide on Some Egyptian Freshwater Fishes. Abbassa Int. J. Aquac.:113-127, 2009. ECOREF #165769	Non-north american test species

Citation	Notes
Ewell,W.S., J.W. Gorsuch, R.O. Kringle, K.A. Robillard, and R.C. Spiegel. Simultaneous Evaluation of the Acute Effects of Chemicals on Seven Aquatic Species. Environ. Toxicol. Chem.5(9): 831-840, 1986. ECOREF #11951	
Jin,H., X. Yang, H. Yu, and D. Yin. Identification of Ammonia and Volatile Phenols as Primary Toxicants in a Coal Gasification Effluent. Bull. Environ. Contam. Toxicol.63(3): 399-406, 1999. ECOREF #117105	Study objectives and methods don't align with criteria development
Kitamura,H.. Relation Between the Toxicity of Some Toxicants to the Aquatic Animals (Tanichthys albonubes and Neocaridina denticulata) and the Hardness of the Test Solution. Bull. Fac. Fish. Nagasaki Univ. (Chodai Sui Kempo)67:13-19, 1990. ECOREF #5459	Non-north american test species
Kovacs,T.G., and G. Leduc. Acute Toxicity of Cyanide to Rainbow Trout (Salmo gairdneri) Acclimated at Different Temperatures. Can. J. Fish. Aquat. Sci.39(10): 1426-1429, 1982. ECOREF #15601	EPA used in 1984 cyanide derivation
LeBlanc,G.A.. Acute Toxicity of Priority Pollutants to Water Flea (Daphnia magna). Bull. Environ. Contam. Toxicol.24(5): 684-691, 1980. ECOREF #5184	Did not find any cyanide toxicity data
LeBlanc,G.A., and D.C. Surprenant. The Chronic Toxicity of 8 of the 65 Priority Pollutants to the Water Flea (Daphnia magna). Draft Manuscript, EG&G Bionomics, Aquatic Toxicology Laboratory, Wareham, MA:36 p., 1980. ECOREF #121018	Chronic based study
Marking,L.L., T.D. Bills, and J.R. Crowther. Effects of Five Diets on Sensitivity of Rainbow Trout to Eleven Chemicals. Prog. Fish-Cult.46(1): 1-5, 1984. ECOREF #10656	Diet based study; not relevant to water exposure
McGeachy,S.M.. Acute and Sublethal Toxicity of Cyanide to Exercised and Non-Exercised Rainbow Trout (Salmo gairdneri) at Different Times of the Year. Ph.D.Thesis, Concordia Univ., Montreal, Quebec, Canada:71 p., 1984. ECOREF #118391	Repeat of other McGeachy study
McGeachy,S.M., and G. Leduc. The Influence of Season and Exercise on the Lethal Toxicity of Cyanide to Rainbow Trout (Salmo gairdneri). Arch. Environ. Contam. Toxicol.17(3): 313-318, 1988. ECOREF #2344	
Meyn,E.L., R.K. Zajdel, and R.V. Thurston. Acute Toxicity of Ferrocyanide and Ferricyanide to Rainbow Trout (Salmo gairdneri). Tech.Rep.No.84-1, Fish.Bioassay Lab., Montana State Univ., Bozeman, MT:19 p., 1984. ECOREF #12029	Ferrocyanide used (mixture of iron and cyanide)
Moore,S.B., R.A. Diehl, J.M. Barnhardt, and G.B. Avery. Aquatic Toxicities of Textile Surfactants. Text. Chem. Color.19(5): 29-32, 1987. ECOREF #12754	Did not find cyanide data
Mowbray,D.L.. Assessment of the Biological Impact of OK Tedi Mine Tailings, Cyanide and Heavy Metals. In: J.C.Pernetta (Ed.), Reg.Seas Rep.Stud.No.99, Potential Impacts of Mining on the Fly River, UNEP, Athens, Greece:45-74, 1988. ECOREF #17356	Not relevant; site-specific assessment

Citation	Notes
Nalecz-Jawecki,G., and J. Sawicki. Toxicity of Inorganic Compounds in the Spirotox Test: A Miniaturized Version of the Spirostomum ambiguum Test. Arch. Environ. Contam. Toxicol.34(1): 1-5, 1998. ECOREF #18997	Bacteria based test; can't use single celled orgs
Parametrix Inc.. 96-h Acute Toxicity of Cyanide to Gasterosteus aculeatus Under Flow-Through Conditions. Report 3539-15, Parametrix Environmental Research Laboratory, Albany, OR:10 p., 2005. ECOREF #167153	Unable to locate article
Prashanth,M.S.. Acute Toxicity, Behavioral and Nitrogen Metabolism Changes of Sodium Cyanide Affected on Tissues of Tilapia mossambica (Perters). Drug Chem. Toxicol.35(2): 178-183, 2012. ECOREF #160874	Non-north american test species
Prashanth,M.S., H.A. Sayeswara, and H.S.R. Patil. Impact of Copper Cyanide on Behavioral Changes and Oxygen Consumption in Indian Major Carp Catla catla (Hamilton). J. Environ. Agric. Food Chem.9(9): 1433-1442, 2010. ECOREF #158813	Non-north american test species
Qureshi,A.A., K.W. Flood, S.R. Thompson, S.M. Janhurst, C.S. Inniss, and D.A. Rokosh. Comparison of a Luminescent Bacterial Test with Other Bioassays for Determining Toxicity of Pure Compounds and Complex Effluents. ASTM Spec. Tech. Publ.:179-195, 1982. ECOREF #15923	Bacteria based test; can't use single celled orgs
Richie,J.P.,Jr., B.J. Mills, and C.A. Lang. The Verification of a Mammalian Toxicant Classification Using a Mosquito Screening Method. Fundam. Appl. Toxicol.4(6): 1029-1035, 1984. ECOREF #173907	Not relevant; details a testing method
Sabourin,T.D.. Methods for Aquatic Toxicity Tests Conducted with Acrolein and DEHP as well as the Methods and Results for Acrylonitrile Tests. September 18 Memo to D.Call, University of Wisconsin, Superior, WI:16 p., 1987. ECOREF #17132	Not relevant; no cyanide data available
Sangli,A.B., and V.V. Kanabur. Lethal Toxicity of Cyanide and Formalin to a Freshwater Fish Gambusia affinis. Environ. Ecol.18(2): 362-364, 2000. ECOREF #74408	
Sanoli,A.B., and V.V. Kanabur. Acute Toxicity of Cyanide and Formalin to a Freshwater Fish Lepidocepalichithys guntea (Catfish). Indian J. Fish.48(1): 99-101, 2001. ECOREF #118101	Non-north american test species
Sarkar,S.K.. Toxicity Evaluation of Sodium Cyanide to Fish and Aquatic Organisms: Effects of Temperature. Sci. Cult.56(4): 165-168, 1990. ECOREF #8886	
Schimmel,S.C.. Results of Toxicity Tests Conducted with Cyanide at ERL, Narragansett. U.S.EPA, Narragansett, RI, (Memo to John H.Gentile, U.S.EPA, Narragansett, RI):2 p., 1981. ECOREF #103809	Unable to locate article

Citation	Notes
Skibba,W.D.. The Trout Test with Salmo gairdneri Rich. for Determining the Acute Toxicity of Aggressive Substances as Well as Measurement Results for. Acta Hydrochim. Hydrobiol.9(1): 3-15, 1981. ECOREF #5639	EPA used in 1984 derivation
Slabbert,J.L., and E.A. Venter. Biological Assays for Aquatic Toxicity Testing. Water Sci. Technol.39(10/11): 367-373, 1999. ECOREF #61447	Non-north american test species
Solbe,J.F.D., V.A. Cooper, C.A. Willis, and M.J. Mallett. Effects of Pollutants in Fresh Waters on European Non-Salmonid Fish I: Non-Metals. J. Fish Biol.27(suppl.A): 197-207, 1985. ECOREF #11655	Non-north american test species
Thurston,R.V., and T.A. Heming. Acute Toxicity of Iron Cyanides and Thiocyanate to Trout. In: EPA-600/9-86/024, R.C.Ryans (Ed.), Proc.of USA-USSR Symp., Jul.30-Aug.1, 1984, Borok, Jaroslavl Oblast, U.S.EPA, Athens, GA:55-71, 1984. ECOREF #67837	Unable to locate article
Tong,Z., Z. Huailan, and J. Hongjun. Chronic Toxicity of Acrylonitrile and Acetonitrile to Daphnia magna in 14-d and 21-d Toxicity Tests. Bull. Environ. Contam. Toxicol.57(4): 655-659, 1996. ECOREF #13070	Chronic study
Tonogai,Y., S. Ogawa, Y. Ito, and M. Iwaida. Actual Survey on TLM (Median Tolerance Limit) Values of Environmental Pollutants, Especially on Amines, Nitriles, Aromatic Nitrogen Compounds. J. Toxicol. Sci.7(3): 193-203, 1982. ECOREF #10132	Study not relevant; cyanide data not available
Tryland,O., and M. Grande. Removal of Cyanide from Scrubber Effluents and Its Effect on Toxicity to Fish. Vatten39:168-174, 1983. ECOREF #20723	Study not relevant ; examined wastewater
Tscheu-Schluter,M.. On the Toxicity of Simple and Complex Cyanides to Aquatic Organisms (Zur Toxizitat Einfacher und Komplexer Cyanide Gegenuber Wasserorganismen). Acta Hydrochim. Hydrobiol.11(2): 169-179, 1983. ECOREF #12314	Non-north american test species
U.S. Environmental Protection Agency. Pesticide Ecotoxicity Database (Formerly: Environmental Effects Database (EEDB)). Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.:, 1992. ECOREF #344	Reference to a database
Van der Schalie,W.H., T.R. Shedd, M.W. Widder, and L.M. Brennan. Response Characteristics of an Aquatic Biomonitor Used for Rapid Toxicity Detection. J. Appl. Toxicol.24(5): 387-394, 2004. ECOREF #77525	
Wellens,H.. Comparison of the Sensitivity of Brachydanio rerio and Leuciscus idus by Testing the Fish Toxicity of Chemicals and Wastewaters. Z. Wasser-Abwasser-Forsch.51(2): 49-52, 1982. ECOREF #11037	Wrong language
Zhang,T., H. Jin, and H. Zhu. Quality Criteria of Acrylonitrile for the Protection of Aquatic Life in China. Chemosphere32(10): 2083-2093, 1996. ECOREF #16884	Non-north american test species

Freshwater Chronic

Table A12. List of citations from EPA ECOTOX database reviewed for cyanide freshwater chronic criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Authman,M.M.N., W.T. Abbas, I.M.K. Abumourad, and A.M. Kenawy. Effects of Illegal Cyanide Fishing on Vitellogenin in the Freshwater African Catfish, Clarias gariepinus (Burchell, 1822). Ecotoxicol. Environ. Saf.91(0): 61-70, 2013. ECOREF #164180	Non-north american test species
LeBlanc,G.A., and D.C. Surprenant. The Chronic Toxicity of 8 of the 65 Priority Pollutants to the Water Flea (Daphnia magna). Draft Manuscript, EG&G Bionomics, Aquatic Toxicology Laboratory, Wareham, MA:36 p., 1980. ECOREF #121018	No cyanide data
Moore,S.B., R.A. Diehl, J.M. Barnhardt, and G.B. Avery. Aquatic Toxicities of Textile Surfactants. Text. Chem. Color.19(5): 29-32, 1987. ECOREF #12754	No cyanide data
Rippon,G.D., C.A. Le Gras, R.V. Hyne, and P.J. Cusbert. Toxic Effects of Cyanide on Aquatic Animals of the Alligator Rivers Region. Tech.Memorandum No.39, Commonwealth of Australia, Supervising Scientist for the Alligator Rivers Region, N.S.W.2022, Australia:10 p., 1992. ECOREF #6598	Non-north american test species
Szabo,A., S.M. Ruby, F. Rogan, and Z. Amit. Changes in Brain Dopamine Levels, Oocyte Growth and Spermatogenesis in Rainbow Trout, Oncorhynchus mykiss, Following Sublethal Cyanide Exposure. Arch. Environ. Contam. Toxicol.21(1): 152-157, 1991. ECOREF #117809	Endpoints not relevant
Tong,Z., Z. Huailan, and J. Hongjun. Chronic Toxicity of Acrylonitrile and Acetonitrile to Daphnia magna in 14-d and 21-d Toxicity Tests. Bull. Environ. Contam. Toxicol.57(4): 655-659, 1996. ECOREF #13070	No cyanide data
Zhang,T., H. Jin, and H. Zhu. Quality Criteria of Acrylonitrile for the Protection of Aquatic Life in China. Chemosphere32(10): 2083-2093, 1996. ECOREF #16884	Non-north american test species

Nickel

Freshwater Acute

Table A13. List of citations from EPA ECOTOX database reviewed for nickel freshwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Alam,M.K., and O.E. Maughan. Acute Toxicity of Heavy Metals to Common Carp (Cyprinus carpio). J. Environ. Sci. Health. Part A, Environ. Sci. Eng. Toxic Hazard. Substance Control30(8): 1807-1816, 1995. ECOREF #45566	Non-north american test species used; no hardness
Alkahem,H.F.. The Toxicity of Nickel and the Effects of Sublethal Levels on Haematological Parameters and Behaviour of the Fish, Oreochromis niloticus. J. Univ. Kuwait Sci.21(2): 243-251, 1994. ECOREF #16861	Non-north american test species used
Alkahem,H.F.. Effects of Nickel on Carbohydrate Metabolism of Oreochromis niloticus. Dirasat Ser. B Pure Appl. Sci.22(1): 83-88, 1995. ECOREF #20533	Non-north american test species used
Alsop,D., and C.M. Wood. Metal Uptake and Acute Toxicity in Zebrafish: Common Mechanisms Across Multiple Metals. Aquat. Toxicol.105(3/4): 385-393, 2011. ECOREF #158223	
Alsop,D., and C.M. Wood. Metal and Pharmaceutical Mixtures: Is Ion Loss the Mechanism Underlying Acute Toxicity and Widespread Additive Toxicity in Zebrafish?. Aquat. Toxicol.140/141:257-267, 2013. ECOREF #166490	
Bechard,K.M., P.L. Gillis, and C.M. Wood. Acute Toxicity of Waterborne Cd, Cu, Pb, Ni, and Zn to First-Instar Chironomus riparius Larvae. Arch. Environ. Contam. Toxicol.54(3): 454-459, 2008. ECOREF #108924	24-hr LC50; control mortality after 24 hr
Borgmann,U., Y. Couillard, P. Doyle, and D.G. Dixon. Toxicity of Sixty-Three Metals and Metalloids to Hyalella azteca at Two Levels of Water Hardness. Environ. Toxicol. Chem.24(3): 641-652, 2005. ECOREF #80935	
Brix,K.V., J. Keithly, D.K. DeForest, and J. Laughlin. Acute and Chronic Toxicity of Nickel to Rainbow Trout (Oncorhynchus mykiss). Environ. Toxicol. Chem.23(9): 2221-2228, 2004. ECOREF #80785	
Chu,K.W., and K.L. Chow. Synergistic Toxicity of Multiple Heavy Metals is Revealed by a Biological Assay Using a Nematode and Its Transgenic Derivative. Aquat. Toxicol.61(1/2): 53-64, 2002. ECOREF #65728	No hardness data
Fargasova,A.. Ecotoxicology of Metals Related to Freshwater Benthos. Gen. Physiol. Biophys.18(Focus Issue): 48-53, 1999. ECOREF #61824	

Citation	Notes
Griffitt,R.J., J. Luo, J. Gao, J.C. Bonzongo, and D.S. Barber. Effects of Particle Composition and Species on Toxicity of Metallic Nanomaterials in Aquatic Organisms. Environ. Toxicol. Chem.27(9): 1972-1978, 2008. ECOREF #104806	
Herkovits,J., C.S. Perez-Coll, and F.D. Herkovits. Evaluation of Nickel-Zinc Interactions by Means of Bioassays with Amphibian Embryos. Ecotoxicol. Environ. Saf.45(3): 266-273, 2000. ECOREF #50151	No hardness data
Herkovits,J., L. Corro, C. Perez-Coll, and O. Dominguez. Fluid Motion Effect on Metal Toxicity in Bufo arenarum Embryos. Bull. Environ. Contam. Toxicol.68(4): 549-554, 2002. ECOREF #65778	No hardness data
Hockett,J.R., and D.R. Mount. Use of Metal Chelating Agents to Differentiate Among Sources of Acute Aquatic Toxicity. Environ. Toxicol. Chem.15(10): 1687-1693, 1996. ECOREF #45021	
Kallanagoudar,Y.P., and H.S. Patil. Influence of Water Hardness on Copper, Zinc and Nickel Toxicity to Gambusia affinis (B&G). J. Environ. Biol.18(4): 409-413, 1997. ECOREF #19028	
Kazlauskiene,N., A. Burba, and G. Svecevicius. Acute Toxicity of Five Galvanic Heavy Metals to Hydrobionts. Ekologiia1:33-36, 1994. ECOREF #17573	
Keithly,J., J.A. Brooker, D.K. DeForest, B.K. Wu, and K.V. Brix. Acute and Chronic Toxicity of Nickel to a Cladoceran (Ceriodaphnia dubia) and an Amphipod (Hyalella azteca). Environ. Toxicol. Chem.23(3): 691-696, 2004. ECOREF #106584	
Keller,A.E.. Personal Communication to U.S. EPA: Water Quality and Toxicity Data for Unpublished Unionid Mussel Tests. Memo to R.Pepin and C.Roberts,U.S.EPA Region 5,Chicago, IL:14 p., 2000. ECOREF #76251	Unpublished work; no access
Khan,S., and D. Nugegoda. Sensitivity of Juvenile Freshwater Crayfish Cherax destructor (Decapoda: Parastacidae) to Trace Metals. Ecotoxicol. Environ. Saf.68(3): 463-469, 2007. ECOREF #106705	Non-north american test species
Khunyakari,R.P., V. Tare, and R.N. Sharma. Effects of Some Trace Heavy Metals on Poecilia reticulata (Peters). J. Environ. Biol.22(2): 141-144, 2001. ECOREF #62227	Non-north american test species
Liber,K., L.E. Doig, and S.L. White-Sobey. Toxicity of Uranium, Molybdenum, Nickel, and Arsenic to Hyalella azteca and Chironomus dilutus in Water-Only and Spiked-Sediment Toxicity Tests. Ecotoxicol. Environ. Saf.74(5): 1171-1179, 2011. ECOREF #175087	
Madoni,P.. The Acute Toxicity of Nickel to Freshwater Ciliates. Environ. Pollut.109(1): 53-59, 2000. ECOREF #51792	Single celled test organism; not appropriate
Madoni,P., and M.G. Romeo. Acute Toxicity of Heavy Metals Towards Freshwater Ciliated Protists. Environ. Pollut.141(1): 1-7, 2006. ECOREF #95678	Single celled test organism; not appropriate

Citation	Notes
Nalecz-Jawecki,G., and J. Sawicki. Toxicity of Inorganic Compounds in the Spirotox Test: A Miniaturized Version of the Spirostomum ambiguum Test. Arch. Environ. Contam. Toxicol.34(1): 1-5, 1998. ECOREF #18997	Bacteria test; not appropriate
Nanda,P., B.N. Panda, and M.K. Behera. Nickel Induced Alterations in Protein Level of Some Tissues of Heteropneustes fossilis. J. Environ. Biol.21(2): 117-119, 2000. ECOREF #52565	Non-north american test species used
Phipps,G.L., V.R. Mattson, and G.T. Ankley. Relative Sensitivity of Three Freshwater Benthic Macroinvertebrates to Ten Contaminants. Arch. Environ. Contam. Toxicol.28(3): 281-286, 1995. ECOREF #14907	10-day LC50; not appropriate
Pourkhabbaz,A., T. Khazaei, S. Behravesh, M. Ebrahimpour, and H. Pourkhabbaz. Effect of Water Hardness on the Toxicity of Cobalt and Nickel to a Freshwater Fish, Capoeta fusca. Biomed. Environ. Sci.24(6): 656-660, 2011. ECOREF #166472	Non-north american test species used
Puttaswamy,N., and K. Liber. Influence of Inorganic Anions on Metals Release from Oil Sands Coke and on Toxicity of Nickel and Vanadium to Ceriodaphnia dubia. Chemosphere86(5): 521-529, 2012. ECOREF #165122	Mixture study; inappropriate water quality test conditions
Sanchez-Moreno,S., J.A. Camargo, and A. Navas. Ecotoxicological Assessment of the Impact of Residual Heavy Metals on Soil Nematodes in the Guadiamar River Basin (Southern Spain). Environ. Monit. Assess.116(1-3): 245-262, 2006. ECOREF #101819	Soil nematodes used as test organism
Sharma,S., S. Sharma, P.K. Singh, R.C. Swami, and K.P. Sharma. Exploring Fish Bioassay of Textile Dye Wastewaters and Their Selected Constituents in Terms of Mortality and Erythrocyte Disorders. Bull. Environ. Contam. Toxicol.83(1): 29-34, 2009. ECOREF #158330	Test material isn't relevant
Shuhaimi-Othman,M., N. Yakub, N.A. Ramle, and A. Abas. Toxicity of Metals to a Freshwater Ostracod: Stenocypris major. J. Toxicol.2011:8 p., 2011. ECOREF #165793	Non-north american test species used
Shuhaimi-Othman,M., N. Yakub, N.S. Umirah, and A. Abas. Toxicity of Eight Metals to Malaysian Freshwater Midge Larvae Chironomus javanus (Diptera, Chironomidae). Toxicol. Ind. Health27(10): 879-886, 2011. ECOREF #163320	Non-north american test species used
Shuhaimi-Othman,M., R. Nur-Amalina, and Y. Nadzifah. Toxicity of Metals to a Freshwater Snail, Melanoides tuberculata. Sci. World J.:10 p., 2012. ECOREF #166664	Non-north american test species used
Shuhaimi-Othman,M., Y. Nadzifah, N.S. Umirah, and A.K. Ahmad. Toxicity of Metals to Tadpoles of the Common Sunda Toad, Duttaphrynus melanostictus. Toxicol. Environ. Chem.94(2): 364-376, 2012. ECOREF #159422	Non-north american test species used
Shuhaimi-Othman,M., Y. Nadzifah, N.S. Umirah, and A.K. Ahmad. Toxicity of Metals to an Aquatic Worm, Nais elinguis (Oligochaeta, Naididae). Res. J. Environ. Toxicol.6(4): 122-132, 2012. ECOREF #163848	Non-north american test species used

Citation	Notes
Sornaraj,R., P. Baskaran, and S. Thanalakshmi. Effects of Heavy Metals on Some Physiological Responses of Air-Breathing Fish Channa punctatus (Bloch). Environ. Ecol.13(1): 202-207, 1995. ECOREF #17380	Non-north american test species used
Sztrum,A.A., J.L. D'Eramo, and J. Herkovits. Nickel Toxicity in Embryos and Larvae of the South American Toad: Effects on Cell Differentiation, Morphogenesis, and Oxygen Consumption. Environ. Toxicol. Chem.30(5): 1146-1152, 2011. ECOREF #153688	Non-north american test species used
Tatara,C.P., M.C. Newman, J.T. McCloskey, and P.L. Williams. Predicting Relative Metal Toxicity with Ion Characteristics: Caenorhabditis elegans LC50. Aquat. Toxicol.39(3-4): 279-290, 1997. ECOREF #18605	No hardness data
Tsui,M.T.K., W.X. Wang, and L.M. Chu. Influence of Glyphosate and Its Formulation (Roundup) on the Toxicity and Bioavailability of Metals to Ceriodaphnia dubia. Environ. Pollut.138(1): 59-68, 2005. ECOREF #87704	Pesticide mixture study
Vedamanikam,V.J., and N.A.M. Shazili. The Chironomid Larval Tube, a Mechanism to Protect the Organism from Environmental Disturbances?. Toxicol. Environ. Chem.91(1): 171-176, 2009. ECOREF #115860	No hardness data
Vedamanikam,V.J., and N.A.M. Shazilli. Comparative Toxicity of Nine Metals to Two Malaysian Aquatic Dipterian Larvae with Reference to Temperature Variation. Bull. Environ. Contam. Toxicol.80(6): 516-520, 2008. ECOREF #107050	No hardness data
Vedamanikam,V.J., and N.A.M. Shazilli. The Effect of Multi-Generational Exposure to Metals and Resultant Change in Median Lethal Toxicity Tests Values over Subsequent Generations. Bull. Environ. Contam. Toxicol.80(1): 63-67, 2008. ECOREF #111291	No hardness data
Virk,S., and R.C. Sharma. Effect of Nickel and Chromium on Various Life Stages of Cyprinus carpio Linn. Indian J. Ecol.22(2): 77-81, 1995. ECOREF #18750	Non-north american test species used
Wong,C.K., and A.P. Pak. Acute and Subchronic Toxicity of the Heavy Metals Copper, Chromium, Nickel, and Zinc, Individually and in Mixture, to the Freshwater Copepod Mesocyclops pehpeiensis. Bull. Environ. Contam. Toxicol.73(1): 190-196, 2004. ECOREF #80006	Non-north american test species used

Open Literature

Table A14. List of open literature citations from EPA ECOTOX database reviewed for nickel criteria derivation but did not meet acceptability requirements.

Citation	Notes
Zidour, M., Boubechiche, Z., Pan, Y.J., Bialais, C., Cudennec, B., Grard, T., Drider, D., Flahaut, C., Ouddane, B. and Souissi, S., 2019. Population response of the estuarine copepod Eurytemora affinis to its bioaccumulation of trace metals. Chemosphere, 220, pp.505-513.	LC50s are sex specific (male and female); tests were 96 hr and not the standard 48-hr for invertebrates
Panneerselvam, K., Marigoudar, S.R. and Dhandapani, M., 2018. Toxicity of nickel on the selected species of marine diatoms and copepods. Bulletin of environmental contamination and toxicology, 100, pp.331-337.	Marine study
Okamoto, A., Masunaga, S. and Tatarazako, N., 2021. Chronic toxicity of 50 metals to Ceriodaphnia dubia. Journal of Applied Toxicology, 41(3), pp.375-386.	Very little study details; Effect level reported as inhibitory concentrations; did not use flow through design
Ghosh, A., Kaviraj, A. and Saha, S., 2018. Deposition, acute toxicity, and bioaccumulation of nickel in some freshwater organisms with best-fit functions modeling. Environmental Science and Pollution Research, 25, pp.3588-3595.	Non-north American test species;
Ansari, S., Ansari, B.A. and Ansari, B.A., 2015. Effects of heavy metals on the embryo and larvae of Zebrafish, Danio rerio (Cyprinidae). Scholars Academic Journal of Biosciences, 3(1b), pp.52-56.	No hardness data
Leung, J., Witt, J.D., Norwood, W. and Dixon, D.G., 2016. Implications of Cu and Ni toxicity in two members of the Hyalella azteca cryptic species complex: Mortality, growth, and bioaccumulation parameters. Environmental toxicology and chemistry, 35(11), pp.2817-2826.	No 48-hour LC50s calculated
McKinley, K., McLellan, I., Gagné, F. and Quinn, B., 2019. The toxicity of potentially toxic elements (Cu, Fe, Mn, Zn and Ni) to the cnidarian Hydra attenuata at environmentally relevant concentrations. Science of the Total Environment, 665, pp.848-854.	Multi-well plates test chambers; 48-hour LC50 not reported; fed during study

Freshwater Chronic

Table A15. List of citations from EPA ECOTOX database reviewed for nickel freshwater chronic criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Brix,K.V., J. Keithly, D.K. DeForest, and J. Laughlin. Acute and Chronic Toxicity of Nickel to Rainbow Trout (Oncorhynchus mykiss). Environ. Toxicol. Chem.23(9): 2221-2228, 2004. ECOREF #80785	
Jaworska,M., A. Gorczyca, J. Sepiol, and P. Tomasik. Effect of Metal Ions on the Entomopathogenic Nematode Heterorhabditis bacteriophora Poinar (Nematode: Heterohabditidae) Under Laboratory Conditions. Water Air Soil Pollut.93:157-166, 1997. ECOREF #40155	Bacteria study
Keithly,J., J.A. Brooker, D.K. DeForest, B.K. Wu, and K.V. Brix. Acute and Chronic Toxicity of Nickel to a Cladoceran (Ceriodaphnia dubia) and an Amphipod (Hyalella azteca). Environ. Toxicol. Chem.23(3): 691-696, 2004. ECOREF #106584	
Kienle,C., H.R. Kohler, and A. Gerhardt. Behavioural and Developmental Toxicity of Chlorpyrifos and Nickel Chloride to Zebrafish (Danio rerio) Embryos and Larvae. Ecotoxicol. Environ. Saf.72(6): 1740-1747, 2009. ECOREF #119259	No hardness data
Ku,T.T., W. Yan, W.Y. Jia, Y. Yun, N. Zhu, G.K. Li, and N. Sang. Characterization of Synergistic Embryotoxicity of Nickel and Buprofezin in Zebrafish. Environ. Sci. Technol.49(7): 4600-4608, 2015. ECOREF #173640	Toxicity test endpoints aren't relevant
Lahnsteiner,F., N. Mansour, and B. Berger. The Effect of Inorganic and Organic Pollutants on Sperm Motility of Some Freshwater Teleosts. J. Fish Biol.65(5): 1283-1297, 2004. ECOREF #112446	Toxicity test endpoints aren't relevant
Langer-Jaesrich,M., H.R. Kohler, and A. Gerhardt. Can Mouth Part Deformities of Chironomus riparius Serve as Indicators for Water and Sediment Pollution? A Laboratory Approach. J. Soils Sediments10(3): 414-422, 2010. ECOREF #121124	Toxicity test endpoints aren't relevant
Liber,K., L.E. Doig, and S.L. White-Sobey. Toxicity of Uranium, Molybdenum, Nickel, and Arsenic to Hyalella azteca and Chironomus dilutus in Water-Only and Spiked-Sediment Toxicity Tests. Ecotoxicol. Environ. Saf.74(5): 1171-1179, 2011. ECOREF #175087	Water only test duration too short for chronic study
Mwangi,J.N., N. Wang, C.G. Ingersoll, D.K. Hardesty, E.L. Brunson, H. Li, and B. Deng. Toxicity of Carbon Nanotubes to Freshwater Aquatic Invertebrates. Environ. Toxicol. Chem.31(8): 1823-1830, 2012. ECOREF #158582	Nanotube study

Citation	Notes
Ouellette,J.D., M.G. Dube, and S. Niyogi. A Single Metal, Metal Mixture, and Whole-Effluent Approach to Investigate Causes of Metal Mine Effluent Effects on Fathead Minnows (Pimephales promelas). Water Air Soil Pollut.224(1462): 44 p., 2013. ECOREF #166026	Study mimicked effluent and didn't aim find threshold value
Pane,E.F., A. Haque, and C.M. Wood. Mechanistic Analysis of Acute, Ni-Induced Respiratory Toxicity in the Rainbow Trout (Oncorhynchus mykiss): An Exclusively Branchial Phenomenon. Aquat. Toxicol.69(1): 11-24, 2004. ECOREF #89704	Test endpoints are not relevant to criteria development
Pavlaki,M.D., R. Pereira, S. Loureiro, and A.M.V.M. Soares. Effects of Binary Mixtures on the Life Traits of Daphnia magna. Ecotoxicol. Environ. Saf.74(1): 99-110, 2011. ECOREF #166654	
Puttaswamy,N., and K. Liber. Influence of Inorganic Anions on Metals Release from Oil Sands Coke and on Toxicity of Nickel and Vanadium to Ceriodaphnia dubia. Chemosphere86(5): 521-529, 2012. ECOREF #165122	Mixture study; inappropriate water quality test conditions
Zuiderveen,J.A., and W.J. Birge. The Relationship Between Chronic Values in Toxicity Tests with Ceriodaphnia dubia. ASTM Spec. Tech. Publ.6:551-556, 1997. ECOREF #76252	Did not include analytical chemistry
Besser,J.M., C.D. Ivey, J.A. Steevens, D. Cleveland, D. Soucek, A. Dickinson, E.J. Van Genderen, A.C. Ryan, C.E. Schlek. Modeling the Bioavailability of Nickel and Zinc to Ceriodaphnia dubia and Neocloeon triangulifer in Toxicity Tests with Natural Waters. Environ. Toxicol. Chem.40(11): 3049-3062, 2021. ECOREF #188814	
Cremazy,A., K.V. Brix, and C.M. Wood. Chronic Toxicity of Binary Mixtures of Six Metals (Ag, Cd, Cu, Ni, Pb and Zn) to the Great Pond Snail Lymnaea stagnalis. Environ. Sci. Technol.52(10): 5979-5988, 2018. ECOREF #188091	EC20 useful; study duration too long for acute toxicity value
De Schamphelaere,K., L.V. Laer, N. Deleebeeck, B.T. Muyssen, F. Degryse, E. Smolders, and C. Janssen. Nickel Speciation and Ecotoxicity in European Natural Surface Waters: Development, Refinement and Validation of Bioavailability Models. Ghent University Laboratory for Environmental Toxicology and Aquatic Ecology:125 p., 2006. ECOREF #187751	Wrong language
Deleebeeck,N.M.E., K.A.C. De Schamphelaere, and C.R. Janssen. A Novel Method for Predicting Chronic Nickel Bioavailability and Toxicity to Daphnia magna in Artificial and Natural Waters. Environ. Toxicol. Chem.27(10): 2097-2107, 2008. ECOREF #187752	EC20 useful; study duration too long for acute toxicity value
Keithly,J., J.A. Brooker, D.K. DeForest, B.K. Wu, and K.V. Brix. Acute and Chronic Toxicity of Nickel to a Cladoceran (Ceriodaphnia dubia) and an Amphipod (Hyalella azteca). Environ. Toxicol. Chem.23(3): 691-696, 2004. ECOREF #106584	Repeat

Open Literature

Table A16. List of open literature citations from EPA ECOTOX database reviewed for nickel criteria derivation but did not meet acceptability requirements.

Citation	Notes
Nys, C., Janssen, C.R., Van Sprang, P. and De Schamphelaere, K.A., 2016. The effect of pH on chronic aquatic nickel toxicity is dependent on the pH itself: Extending the chronic nickel bioavailability models. Environmental toxicology and chemistry, 35(5), pp.1097-1106.	Static-renewal test design; according to EPA 1985 guidance chronic studies should be flow-through
Nys, C., Van Regenmortel, T., Janssen, C.R., Blust, R., Smolders, E. and De Schamphelaere, K.A., 2017. Comparison of chronic mixture toxicity of nickel-zinc-copper and nickel-zinc-copper-cadmium mixtures between Ceriodaphnia dubia and Pseudokirchneriella subcapitata. Environmental Toxicology and Chemistry, 36(4), pp.1056-1066.	Static-renewal test design; according to EPA 1985 guidance chronic studies should be flow-through
Niyogi, S., Brix, K.V. and Grosell, M., 2014. Effects of chronic waterborne nickel exposure on growth, ion homeostasis, acid-base balance, and nickel uptake in the freshwater pulmonate snail, Lymnaea stagnalis. Aquatic toxicology, 150, pp.36-44.	Static-renewal test design; according to EPA 1985 guidance chronic studies should be flow-through
Klemish, J.L., Bogart, S.J., Luek, A., Lannoo, M.J. and Pyle, G.G., 2018. Nickel toxicity in wood frog tadpoles: Bioaccumulation and sublethal effects on body condition, food consumption, activity, and chemosensory function. Environmental toxicology and chemistry, 37(9), pp.2458-2466.	Static-renewal test design; according to EPA 1985 guidance chronic studies should be flow-through
Gissi, F., Wang, Z., Batley, G.E., Leung, K.M., Schlekat, C.E., Garman, E.R. and Stauber, J.L., 2020. Deriving a chronic guideline value for nickel in tropical and temperate marine waters. Environmental Toxicology and Chemistry, 39(12), pp.2540-2551.	Marine study
Deleebeeck, N.M., De Schamphelaere, K.A. and Janssen, C.R., 2007. A bioavailability model predicting the toxicity of nickel to rainbow trout (Oncorhynchus mykiss) and fathead minnow (Pimephales promelas) in synthetic and natural waters. Ecotoxicology and Environmental Safety, 67(1), pp.1-13.	Good study but ACRs not reported; data usable for 8 family method

Pentachlorophenol

Freshwater Acute

Table A17. List of citations from EPA ECOTOX database reviewed for pentachlorophenol freshwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Andersen,H.B., R.S. Caldwell, J. Toll, T. Do, and L. Saban. Sensitivity of Lamprey Ammocoetes to Six Chemicals. Arch. Environ. Contam. Toxicol.59(4): 622-631, 2010. ECOREF #153571	
Ashauer,R., A.B.A. Boxall, and C.D. Brown. New Ecotoxicological Model to Simulate Survival of Aquatic Invertebrates After Exposure to Fluctuating and Sequential Pulses of Pesticides. Environ. Sci. Technol.41(4): 1480-1486, 2007. ECOREF #115493	Modeling study; methods lack detail
Basack,S.B., M.L. Oneto, N.R. Verrengia Guerrero, and E.M. Kesten. Accumulation and Elimination of Pentachlorophenol in the Freshwater Bivalve Corbicula fluminea. Bull. Environ. Contam. Toxicol.58(3): 497-503, 1997. ECOREF #18004	
Bitton,G., K. Rhodes, B. Koopman, and M. Cornejo. Short-Term Toxicity Assay Based on Daphnid Feeding Behavior. Water Environ. Res.67(3): 290-293, 1995. ECOREF #19602	6-hour study; not standardized test
Bridges,C.M., F.J. Dwyer, D.K. Hardesty, and D.W. Whites. Comparative Contaminant Toxicity: Are Amphibian Larvae More Sensitive than Fish?. Bull. Environ. Contam. Toxicol.69(4): 562-569, 2002. ECOREF #72411	
Broderius,S.J., M.D. Kahl, and M.D. Hoglund. Use of Joint Toxic Response to Define the Primary Mode of Toxic Action for Diverse Industrial Organic Chemicals. Environ. Toxicol. Chem.14(9): 1591-1605, 1995. ECOREF #15031	
Centeno,M.D.F., G. Persoone, and M.P. Goyvaerts. Cyst-Based Toxicity Tests. IX. The Potential of Thamnocephalus platyurus as Test Species in Comparison with Streptocephalus proboscideus (Crustacea: Branchiopoda: Anostraca). Environ. Toxicol. Water Qual.10(4): 275-282, 1995. ECOREF #14017	Not relevant testing method; cyst based study
Cheng,Y., M. Ekker, and H.M. Chan. Relative Developmental Toxicities of Pentachloroanisole and Pentachlorophenol in a Zebrafish Model (Danio rerio). Ecotoxicol. Environ. Saf.112:7-14, 2015. ECOREF #170681	No details on bioassay. Does not use standard methods.
Cressman III,C.P., and P.L. Williams. Reference Toxicants for Toxicity Testing Using Caenorhabditis elegans in Aquatic Media. ASTM Spec. Tech. Publ.6:518-532, 1997. ECOREF #19999	

Citation	Notes
Donkin,S.G., and P.L. Williams. Influence of Developmental Stage, Salts and Food Presence on Various End Points Using Caenorhabditis elegans for Aquatic Toxicity Testing. Environ. Toxicol. Chem.14(12): 2139-2147, 1995. ECOREF #16377	LC50 reported as range of values
Dwyer,F.J., D.K. Hardesty, C.E. Henke, C.G. Ingersoll, D.W. Whites, D.R. Mount, and C.M. Bridges. Assessing Contaminant Sensitivity of Endangered and Threatened Species: Toxicant Classes. EPA 600/R-99/098, U.S.EPA, Washington, D.C.:15 p., 1999. ECOREF #56161	Not accessible
Dwyer,F.J., D.K. Hardesty, C.G. Ingersoll, J.L. Kunz, and D.W. Whites. Assessing Contaminant Sensitivity of American Shad, Atlantic Sturgeon and Shortnose Strugeon, Final Report - February 2000. Final Rep., U.S.Geol.Surv., Columbia Environ.Res.Ctr., Columbia, MO:30 p., 2000. ECOREF #77827	
Dwyer,F.J., F.L. Mayer, L.C. Sappington, D.R. Buckler, C.M. Bridges, I.E. Greer, D.K. Hardesty, C.E. Henke, C.G. Ingers. Assessing Contaminant Sensitivity of Endangered and Threatened Aquatic Species: Part I. Acute Toxicity of Five Chemicals. Arch. Environ. Contam. Toxicol.48(2): 143-154, 2005. ECOREF #81380	
Dwyer,F.J., L.C. Sappington, D.R. Buckler, and S.B. Jones. Use of Surrogate Species in Assessing Contaminant Risk to Endangered and Threatened Fishes. EPA/600/R-96/029, U.S.EPA, Washington, DC:78 p., 1995. ECOREF #73668	
Farah,M.A., B. Ateeq, M.N. Ali, R. Sabir, and W. Ahmad. Studies on Lethal Concentrations and Toxicity Stress of Some Xenobiotics on Aquatic Organisms. Chemosphere55(2): 257-265, 2004. ECOREF #73350	
Fisher,S.W., H. Hwang, M. Atanasoff, and P.F. Landrum. Lethal Body Residues for Pentachlorophenol in Zebra Mussels (Dreissena polymorpha) Under Varying Conditions of Temperature and pH. Ecotoxicol. Environ. Saf.43(3): 274-283, 1999. ECOREF #20453	Body residue study
Fort,D.J., E.L. Stover, and J.A. Bantle. Integrated Ecological Hazard Assessment of Waste Site Soil Extracts Using FETAX and Short-Term Fathead Minnow Teratogenesis Assay. ASTM Spec. Tech. Publ.4:93-109, 1996. ECOREF #45211	FETAX assay of waste site
Fort,D.J., and E.L. Stover. Effect of Low-Level Copper and Pentachlorophenol Exposure on Various Early Life Stages of Xenopus laevis. ASTM Spec. Tech. Publ.:188-203, 1996. ECOREF #61813	FETAX assay with non-north american test species used
Janssen,C.R., G. Persoone, and T.W. Snell. Cyst-Based Toxicity Tests. VIII. Short-Chronic Toxicity Tests with the Freshwater Rotifer Brachionus calyciflorus. Aquat. Toxicol.28(3/4): 243-258, 1994. ECOREF #16572	Cytotoxicity test

Citation	Notes
Jin,X., J. Zha, Y. Xu, J.P. Giesy, and Z. Wang. Toxicity of Pentachlorophenol to Native Aquatic Species in the Yangtze River. Environ. Sci. Pollut. Res.19(3): 609-618, 2012. ECOREF #160738	Non-north american test species used
Jordao,R., B. Campos, M.F.L. Lemos, A.M.V.M. Soares, R. Tauler, and C. Barata. Induction of Multixenobiotic Defense Mechanisms in Resistant Daphnia magna Clones as a General Cellular Response to Stress. Aquat. Toxicol.175:132-143, 2016. ECOREF #173580	Molecular study; endpoints not relevant
Kammenga,J.E., C.A.M. Van Gestel, and J. Bakker. Patterns of Sensitivity to Cadmium and Pentachlorophenol Among Nematode Species from Different Taxonomic and Ecological Groups. Arch. Environ. Contam. Toxicol.27(1): 88-94, 1994. ECOREF #13656	
Keller,A.E.. Personal Communication to U.S. EPA: Water Quality and Toxicity Data for Unpublished Unionid Mussel Tests. Memo to R.Pepin and C.Roberts,U.S.EPA Region 5,Chicago, IL:14 p., 2000. ECOREF #76251	Not accessible
Kim,K.T., Y.G. Lee, and S.D. Kim. Combined Toxicity of Copper and Phenol Derivatives to Daphnia magna: Effect of Complexation Reaction. Environ. Int.32(4): 487-492, 2006. ECOREF #87184	Mixture toxicity study
Kishino,T., and K. Kobayashi. Relation Between Toxicity and Accumulation of Chlorophenols at Various pH, and Their Absorption Mechanism in Fish. Water Res.29(2): 431-442, 1995. ECOREF #13717	Non-north american test species used
Kishino,T., and K. Kobayashi. Acute Toxicity and Structure-Activity Relationships of Chlorophenols in Fish. Water Res.30(2): 387-392, 1996. ECOREF #16366	Non-north american test species used
Kishino,T., and K. Kobayshi. Studies on the Mechanism of Toxicity of Chlorophenols Found in Fish Through Quantitative Structure-Activity Relationships. Water Res.30(2): 393-399, 1996. ECOREF #16365	Non-north american test species used
Kurume Laboratory. Final Report. Bioconcentration Test of 2-Perfluoroalkyl (C=4-16) Ethanol [This Test was Performed Using 2-(Perfluorooctyl) Ethanol (Test Substance Number K-1518)] in Carp. Test Substance K-1518, Kurame Laboratory, Chemicals Evaluation and Research Institute, Japan:94 p., 2001. ECOREF #181458	Bioconcentration study
Lee,S.I., E.J. Na, Y.O. Cho, B. Koopman, and G. Bitton. Short-Term Toxicity Test Based on the Algal Uptake by Ceriodaphnia dubia. Water Environ. Res.69:1207-1210, 1997. ECOREF #61914	Dietary exposure route; not relevant
Markle,P.J., J.R. Gully, R.B. Baird, K.M. Nakada, and J.P. Bottomley. Effects of Several Variables on Whole Effluent Toxicity Test Performance and Interpretation. Environ. Toxicol. Chem.19(1): 123-132, 2000. ECOREF #51911	LC50s not provided

Citation	Notes
Martinez-Jeronimo,F., and G. Munoz-Mejia. Evaluation of the Sensitivity of Three Cladoceran Species Widely Distributed in Mexico to Three Reference Toxicants. J. Environ. Sci. Health. Part A, Environ. Sci. Eng. Toxic Hazard. Substance Control42(10): 1417-1424, 2007. ECOREF #119176	No pH reported
Mayer,F.L., D.R. Buckler, F.J. Dwyer, M.R. Ellersieck, L.C. Sappington, J.M. Besser, and C.M. Bridges. Endangered Aquatic Vertebrates: Comparative and Probabilistic-Based Toxicology. EPA/600/R-08/045, U.S.EPA, Washington, DC:43 p., 2008. ECOREF #153255	Repeat of Dwyer/other EPA studies
McDaniel,M., and T.W. Snell. Probability Distributions of Toxicant Sensitivity for Freshwater Rotifer Species. Environ. Toxicol.14(3): 361-366, 1999. ECOREF #76116	
McNulty,E.W., F.J. Dwyer, M.R. Ellersieck, E.I. Greer, C.G. Ingersoll, and C.F. Rabeni. Evaluation of Ability of Reference Toxicity Tests to Identify Stress in Laboratory Populations of the Amphipod Hyalella azteca. Environ. Toxicol. Chem.18(3): 544-548, 1999. ECOREF #52121	
Milam,C.D., J.L. Farris, F.J. Dwyer, and D.K. Hardesty. Acute Toxicity of Six Freshwater Mussel Species (Glochidia) to Six Chemicals: Implications for Daphnids and Utterbackia imbecillis as Surrogates for Protection of Freshwater Mussels (Unionidae). Arch. Environ. Contam. Toxicol.48(2): 166-173, 2005. ECOREF #81810	
Morales,M., P. Martinez-Paz, R. Martin, R. Planello, J. Urien, J.L. Martinez-Guitarte, and G. Morcillo. Transcriptional Changes Induced by In Vivo Exposure to Pentachlorophenol (PCP) in Chironomus riparius (Diptera) Aquatic Larvae. Aquat. Toxicol.157:1-9, 2014. ECOREF #170699	
Nikkila,A., A. Halme, and J.V.K. Kukkonen. Toxicokinetics, Toxicity and Lethal Body Residues of Two Chlorophenols in the Oligochaete Worm, Lumbriculus variegatus, in Different Sediments. Chemosphere51(1): 35-46, 2003. ECOREF #71410	Sediment based toxicity study
Oda,S., N. Tatarazako, H. Watanabe, M. Morita, and T. Iguchi. Genetic Differences in the Production of Male Neonates in Daphnia magna Exposed to Juvenile Hormone Analogs. Chemosphere63(9): 1477-1484, 2006. ECOREF #97744	
Powell,R.L., E.M. Moser, R.A. Kimerle, D.E. McKenzie, and M. McKee. Use of a Miniaturized Test System for Determining Acute Toxicity of Toxicity Identification Evaluation Fractions. Ecotoxicol. Environ. Saf.35(1): 1-6, 1996. ECOREF #109574	Test design modified from standard methods
Preston,B.L., T.W. Snell, D.M. Fields, and M.J. Weissburg. The Effects of Fluid Motion on Toxicant Sensitivity of the Rotifer Brachionus calyciflorus. Aquat. Toxicol.52(2): 117-131, 2001. ECOREF #60075	
Preston,B.L., T.W. Snell, and R. Kneisel. UV-B Exposure Increases Acute Toxicity of Pentachlorophenol and Mercury to the Rotifer Brachionus calyciflorus. Environ. Pollut.106(1): 23-31, 1999. ECOREF #20344	

Citation	Notes
Ra,J.S., S.Y. Oh, B.C. Lee, and S.D. Kim. The Effect of Suspended Particles Coated by Humic Acid on the Toxicity of Pharmaceuticals, Estrogens, and Phenolic Compounds. Environ. Int.34(2): 184-192, 2008. ECOREF #155080	Sediment study
Radix,P., M. Leonard, C. Papantoniou, G. Roman, E. Saouter, S. Gallotti-Schmitt, H. Thiebaud, and P. Vasseur. Comparison of Four Chronic Toxicity Tests Using Algae, Bacteria, and Invertebrates Assessed with Sixteen Chemicals. Ecotoxicol. Environ. Saf.47(2): 186-194, 2000. ECOREF #60083	
Saka,M.. Examination of an Amphibian-Based Assay Using the Larvae of Xenopus laevis and Ambystoma mexicanum. Ecotoxicol. Environ. Saf.55(1): 38-45, 2003. ECOREF #69555	LC50s not reported in text; non-north american test species used
Sappington,L.C., F.L. Mayer, F.J. Dwyer, D.R. Buckler, J.R. Jones, and M.R. Ellersieck. Contaminant Sensitivity of Threatened and Endangered Fishes Compared to Standard Surrogate Species. Environ. Toxicol. Chem.20(12): 2869-2876, 2001. ECOREF #65396	Repeat of dwyer info
Sawle,A.D., E. Wit, G. Whale, and A.R. Cossins. An Information-Rich Alternative, Chemicals Testing Strategy Using a High Definition Toxicogenomics and Zebrafish (Danio rerio) Embryos. Toxicol. Sci.118(1): 128-139, 2010. ECOREF #158552	Genotoxic based study
Shedd,T.R., M.W. Widder, M.W. Toussaint, M.C. Sunkel, and E. Hull. Evaluation of the Annual Killifish Nothobranchius guentheri as a Tool for Rapid Acute Toxicity Screening. Environ. Toxicol. Chem.18(10): 2258-2261, 1999. ECOREF #20487	Non-north american test species used
Trapido,M., Y. Veressinina, and R. Munter. A Study of the Toxicity of the Ozonation Products of Phenols and Chlorophenols by Daphnia magna Test. Proc. Estonian Acad. Sci.46(3): 130-139, 1997. ECOREF #65394	Lacking study design details; pH is very high
Twagilimana,L., J. Bohatier, CA Groliere, F. Bonnemoy, and D. Sargos. A New Low-Cost Microbiotest with the Protozoan Spirostomum teres: Culture Conditions and Assessment of Sensitivity of the Ciliate to 14 Pure Chemicals. Ecotoxicol. Environ. Saf.41(3): 231-244, 1998. ECOREF #20057	Single cell test; not relevant
Van der Schalie,W.H., T.R. Shedd, M.W. Widder, and L.M. Brennan. Response Characteristics of an Aquatic Biomonitor Used for Rapid Toxicity Detection. J. Appl. Toxicol.24(5): 387-394, 2004. ECOREF #77525	Rapid toxicity test; deviates from standard methods
Willis,K.J.. Acute and Chronic Bioassays with New Zealand Freshwater Copepods Using Pentachlorophenol. Environ. Toxicol. Chem.18(11): 2580-2586, 1999. ECOREF #20641	
Willis,K.J., N. Ling, and M.A. Chapman. Effects of Temperature and Chemical Formulation on the Acute Toxicity of Pentachlorophenol to Simocephalus vetulus (Schoedler, 1858) (Crustacea: Cladocera). N. Z. J. Mar. Freshw. Res.29(2): 289-294, 1995. ECOREF #18919	

Citation	Notes
Xia,X., H. Chunxiu, S. Xue, B. Shi, G. Gui, D. Zhang, X. Wang, and L. Guo. Response of Selenium-Dependent Glutathione Peroxidase in the Freshwater Bivalve Anodonta woodiana Exposed to 2,4-Dichlorophenol, 2,4,6-Trichlorophenol and Pentachlorophenol. Fish Shellfish Immunol.55:499-509, 2016. ECOREF #188367	Molecular based study / endpoints
Yin,D., Y. Gu, Y. Li, X. Wang, and Q. Zhao. Pentachlorophenol Treatment In Vivo Elevates Point Mutation Rate in Zebrafish p53 Gene. Mutat. Res.609(1): 92-101, 2006. ECOREF #91629	Molecular based study / endpoints
Zhao,Y.. Application of Survival Analysis Methods to Pulsed Exposures: Exposure Duration, Latent Mortality, Recovery Time, and the Underlying Theory of Survival Distribution Models. Ph.D.Thesis, The College of William and Mary, Williamsburg, VA:127 p., 2006. ECOREF #169510	LC50 not reported in figure only
Zhao,Y., and M.C. Newman. Shortcomings of the Laboratory-Derived Median Lethal Concentration for Predicting Mortality in Field Populations: Exposure Duration and Latent Mortality. Environ. Toxicol. Chem.23(9): 2147-2153, 2004. ECOREF #77534	LC50 not reported in figure only

Freshwater Chronic

Table A18. List of citations from EPA ECOTOX database reviewed for pentachlorophenol freshwater chronic criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Arthur,A.D., and D.G. Dixon. Effects of Rearing Density on the Growth Response of Juvenile Fathead Minnow (Pimephales promelas) Under Toxicant-Induced Stress. Can. J. Fish. Aquat. Sci.51(2): 365-371, 1994. ECOREF #14078	
Besser,J.M., D.L. Hardesty, I.E. Greer, and C.G. Ingersoll. Sensitivity of Freshwater Snails to Aquatic Contaminants: Survival and Growth of Endangered Snail Species and Surrogates in 28-Day Exposures to Copper, Ammonia and Pentachlorophenol. Administrative Report CERC-8335-FY07-20-10, Columbia, MO:51 p., 2009. ECOREF #151380	
Besser,J.M., N. Wang, F.J. Dwyer, F.L.,Jr. Mayer, and C.G. Ingersoll. Assessing Contaminant Sensitivity of Endangered and Threatened Aquatic Species: Part II. Chronic Toxicity of Copper and Pentachlorophenol to Two Endangered Species and Two Surrogate Species. Arch. Environ. Contam. Toxicol.48(2): 155-165, 2005. ECOREF #91632	
Yu,L.Q., G.F. Zhao, M. Feng, W. Wen, K. Li, P.W. Zhang, X. Peng, W.J. Huo, and H.D. Zhou. Chronic Exposure to Pentachlorophenol Alters Thyroid Hormones and Thyroid Hormone Pathway mRNAs in Zebrafish. Environ. Toxicol. Chem.33(1): 170-176, 2014. ECOREF #170360	Endpoints are not relevant

Saltwater Acute

Table A19. List of citations from EPA ECOTOX database reviewed for pentachlorophenol saltwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Espiritu,E.Q., C.R. Janssen, and G. Persoone. Cyst-Based Toxicity Tests. VII. Evaluation of the 1-h Enzymatic Inhibition Test (Fluotox) with Artemia nauplii. Environ. Toxicol. Water Qual.10:25-34, 1995. ECOREF #16031	Cyst based study
Hori,H., M. Tateishi, K. Takayanagi, and H. Yamada. Applicability of Artificial Seawater as a Rearing Seawater for Toxicity Tests of Hazardous Chemicals by Marine Fish Species. Nippon Suisan Gakkaishi(4): 614-622, 1996. ECOREF #16999	Wrong language
Lawrence,A.J., and C. Poulter. Development of a Sub-lethal Pollution Bioassay Using the Estuarine Amphipod Gammarus duebeni. Water Res.32(3): 569-578, 1998. ECOREF #18971	Non-north american test species; no evidence of its presence on the coast North America.
Lindley,J.A., P. Donkin, S.V. Evans, C.L. George, and K.F. Uil. Effects of Two Organochlorine Compounds on Hatching and Viability of Calanoid Copepod Eggs. J. Exp. Mar. Biol. Ecol.242:59-74, 1999. ECOREF #59982	
Mayer,F.L., D.R. Buckler, F.J. Dwyer, M.R. Ellersieck, L.C. Sappington, J.M. Besser, and C.M. Bridges. Endangered Aquatic Vertebrates: Comparative and Probabilistic-Based Toxicology. EPA/600/R-08/045, U.S.EPA, Washington, DC:43 p., 2008. ECOREF #153255	No saltwater data
Palau-Casellas,A., and T.H. Hutchinson. Acute Toxicity of Chlorinated Organic Chemicals to the Embryos and Larvae of the Marine Worm Platynereis dumerilii (Polychaeta: Nereidae). Environ. Toxicol. Water Qual.13(2): 149-155, 1998. ECOREF #60056	Non-north american test species; no evidence of its presence on the coast North America.
Perez,S., D. Rial, and R. Beiras. Acute Toxicity of Selected Organic Pollutants to Saltwater (Mysid Siriella armata) and Freshwater (Cladoceran Daphnia magna) Ecotoxicological Models. Ecotoxicology24(6): 1229-1238, 2015. ECOREF #170705	Non-north american test species; no evidence of its presence on the coast North America.
Rinna,F., F. Del Prete, V. Vitiello, G. Sansone, and A.L. Langellotti. Toxicity Assessment of Copper, Pentachlorophenol and Phenanthrene by Lethal and Sublethal Endpoints on Nauplii of Tigriopus fulvus. Chem. Ecol.27(S2): 77-85, 2011. ECOREF #166814	Non-north american test species; no evidence of its presence on the coast North America.
Sappington,L.C., F.L. Mayer, F.J. Dwyer, D.R. Buckler, J.R. Jones, and M.R. Ellersieck. Contaminant Sensitivity of Threatened and Endangered Fishes Compared to Standard Surrogate Species. Environ. Toxicol. Chem.20(12): 2869-2876, 2001. ECOREF #65396	
Silva,J., L. Troncoso, E. Bay-Schmith, and A. Larrain. Utilization of Odontesthes regia (Atherinidae) from the South Eastern Pacific as a Test Organism for Bioassays: Study of Its	Non-north american test species; Lacks some method details

Citation	Notes
Sensitivity to Six Chemicals. Bull. Environ. Contam. Toxicol.66(5): 570-575, 2001. ECOREF #62074	
Smith,S., V.J. Furay, P.J. Layiwola, and J.A. Menezes-Filho. Evaluation of the Toxicity and Quantitative Structure-Activity Relationships (QSAR) of Chlorophenols to the Copepodid Stage of a Marine Copepod (Tisbe battagliai) and Two Species of Benthic Flatfish, the Flounder (Plati. Chemosphere28(4): 825-836, 1994. ECOREF #4071	Flatfish and flounder were collected in ambient waters that were not characterized

Silver

Freshwater Acute

Table A20. List of citations from EPA ECOTOX database reviewed for silver freshwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Alsop,D., and C.M. Wood. Metal Uptake and Acute Toxicity in Zebrafish: Common Mechanisms Across Multiple Metals. Aquat. Toxicol.105(3/4): 385-393, 2011. ECOREF #158223	
Bianchini,A., K.C. Bowles, C.J. Brauner, J.W. Gorsuch, J.R. Kramer, and C.M. Wood. Evaluation of the Effect of Reactive Sulfide on the Acute Toxicity of Silver (I) to Daphnia magna. Part 2: Toxicity Results. Environ. Toxicol. Chem.21(6): 1294-1300, 2002. ECOREF #66362	
Bianchini,A., M. Grosell, S.M. Gregory, and C.M. Wood. Acute Silver Toxicity in Aquatic Animals is a Function of Sodium Uptake Rate. Environ. Sci. Technol.36(8): 1763-1766, 2002. ECOREF #66367	Toxicity values not provided
Bianchini,A., and C.M. Wood. Does Sulfide or Water Hardness Protect Against Chronic Silver Toxicity in Daphnia magna? A Critial Assessment of the Acute-to-Chronic Toxicity Ratio for Silver. Ecotoxicol. Environ. Saf.71:32-40, 2008. ECOREF #104819	Organisms fed during study artifically raising LC50
Bielmyer,G.K., K.V. Brix, and M. Grosell. Is Cl- Protection Against Silver Toxicity Due to Chemical Speciation?. Aquat. Toxicol.87(2): 81-87, 2008. ECOREF #104888	Hardness too low - water quality not adequate
Bielmyer,G.K., R.A. Bell, and S.J. Klaine. Effects of Ligand-Bound Silver on Ceriodaphnia dubia. Environ. Toxicol. Chem.21(10): 2204-2208, 2002. ECOREF #68229	
Birge,W.J., J.A. Black, J.F. Hobson, A.G. Westerman, and T.M. Short. Toxicological Studies on Aquatic Contaminants Originating from Coal Production and Utilization: The Induction of Tolerance to Silver in Laboratory Populations of Fish and the Chronic Toxicity of Nickel to Fish	

Citation	Notes
Early Li. Proj.No.G-844-02, Water Resources Research Institute Research Rep.No.151, University of Kentucky, Lexington, KY:36 p., 1984. ECOREF #18858	
Brooke,L.T.. The Effects of Food and Test Solution Age on the Toxicity of Silver to Three Freshwater Organisms. Contract No.68-C1-0034, Work Assignment No.1-10, Environ.Health Lab, Univ.of Wisconsin-Superior, Superior, WI:19 p., 1993. ECOREF #77568	
Brooke,L.T., D.J. Call, C.A. Lindberg, T.P. Markee, S.H. Poirier, and D.J. McCauley. Acute Toxicity of Silver to Selected Freshwater Invertebrates. Report to: Battelle Memorial Research Institute, Collumbus, Ohio, Subcontract No.F-4114(8834)-411; Center for Lake Superior Environmental Studies, University of Wisconsin-Superior, Superior, WI:11 p.:, 1986. ECOREF #3658	
Buccafusco,R.J., S.J. Ells, and G.A. LeBlanc. Acute Toxicity of Priority Pollutants to Bluegill (Lepomis macrochirus). Bull. Environ. Contam. Toxicol.26(4): 446-452, 1981. ECOREF #5590	Test material had 80% purity; insufficient
Bury,N.R., F. Galvez, and C.M. Wood. Effects of Chloride, Calcium, and Dissolved Organic Carbon on Silver Toxicity: Comparison Between Rainbow Trout and Fathead Minnows. Environ. Toxicol. Chem.18(1): 56-62, 1999. ECOREF #19262	Mixture toxicity study
Bury,N.R., J. Shaw, C. Glover, and C. Hogstrand. Derivation of a Toxicity-Based Model to Predict how Water Chemistry Influences Silver Toxicity to Invertebrates. Comp. Biochem. Physiol. C Comp. Pharmacol. Toxicol.133(1-2): 259-270, 2002. ECOREF #65742	Mixture toxicity study
Chapman,G.A., S. Ota, and F. Recht. Effects of Water Hardness on the Toxicity of Metals to Daphnia magna. U.S.EPA, Corvallis, OR:17 p., 1980. ECOREF #3621	Already incorporated into 1980 EPA criteria
De Medeiros,A.M.Z., L.U. Khan, G.H. Da Silva, C.A. Ospina, O.L. Alves, V.L. De Castro, and D.S.T. Martinez. Graphene Oxide-Silver Nanoparticle Hybrid Material: An Integrated Nanosafety Study in Zebrafish Embryos. Ecotoxicol. Environ. Saf.209:14 p., 2021. ECOREF #186027	Nanoparticle study
Diamond,J.M., D.E. Koplish, J. McMahon III, and R. Rost. Evaluation of the Water-Effect Ratio Procedure for Metals in a Riverine System. Environ. Toxicol. Chem.16(3): 509-520, 1997. ECOREF #17591	
Diamond,J.M., D.G. Mackler, M. Collins, and D. Gruber. Derivation of a Freshwater Silver Criteria for the New River, Virginia, Using Representative Species. Environ. Toxicol. Chem.9(11): 1425-1434, 1990. ECOREF #3774	Test water contained silver; field collected orgs; hardness reported as range
Erickson,R.J., L.T. Brooke, M.D. Kahl, F.V. Venter, S.L. Harting, T.P. Markee, and R.L. Spehar. Effects of Laboratory Test Conditions on the Toxicity of Silver to Aquatic Organisms. Environ. Toxicol. Chem.17(4): 572-578, 1998. ECOREF #18938	
Forsythe II,B.L.. Silver in a Freshwater Ecosystem: Acute Toxicity and Trophic Transfer. Ph.D. Thesis, Clemson University, Clemson, SC:149 p., 1996. ECOREF #83754	

Citation	Notes
Galvez,F., and C.M. Wood. The Mechanisms and Costs of Physiological and Toxicological Acclimation to Waterborne Silver in Juvenile Rainbow Trout (Oncorhynchus mykiss). J. Comp. Physiol., B Biochem. Syst. Environ. Physiol.172(7): 587-597, 2002. ECOREF #76331	
Griffitt,R.J., J. Luo, J. Gao, J.C. Bonzongo, and D.S. Barber. Effects of Particle Composition and Species on Toxicity of Metallic Nanomaterials in Aquatic Organisms. Environ. Toxicol. Chem.27(9): 1972-1978, 2008. ECOREF #104806	Nanoparticle study
Grosell,M., C. Hogstrand, C.M. Wood, and H.J.M. Hansen. A Nose-to-Nose Comparison of the Physiological Effects of Exposure to Ionic Silver Versus Silver Chloride in the European Eel (Anguilla anguilla) and the Rainbow Trout (Oncorhynchus mykiss). Aquat. Toxicol.48(2-3): 327-342, 2000. ECOREF #49762	No hardness data
Hobson,J.F.. Acclimation-Induced Changes in Toxicity and Induction of Metallothionein-Like Proteins in the Fathead Minnow Following Sublethal Exposure to Cobalt, Silver, and Zinc. Ph.D.Thesis, University of Kentucky, Lexington, KY:145 p., 1986. ECOREF #150469	Zinc acclimization study
Hockett,J.R., and D.R. Mount. Use of Metal Chelating Agents to Differentiate Among Sources of Acute Aquatic Toxicity. Environ. Toxicol. Chem.15(10): 1687-1693, 1996. ECOREF #45021	Unclear if resulting LC50 mixed with chelating agents
Hogstrand,C., F. Galvez, and C.M. Wood. Toxicity, Silver Accumulation and Metallothionein Induction in Freshwater Rainbow Trout During Exposure to Different Silver Salts. Environ. Toxicol. Chem.15(7): 1102-1108, 1996. ECOREF #17253	No hardness data
Holcombe,G.W., G.L. Phipps, A.H. Sulaiman, and A.D. Hoffman. Simultaneous Multiple Species Testing: Acute Toxicity of 13 Chemicals to 12 Diverse Freshwater Amphibian, Fish, and Invertebrate Families. Arch. Environ. Contam. Toxicol.16:697-710, 1987. ECOREF #12665	
Holcombe,G.W., G.L. Phipps, and J.T. Fiandt. Toxicity of Selected Priority Pollutants to Various Aquatic Organisms. Ecotoxicol. Environ. Saf.7(4): 400-409, 1983. ECOREF #10417	
Hook,S.E., and N.S. Fisher. Sublethal Effects of Silver in Zooplankton: Importance of Exposure Pathways and Implications for Toxicity Testing. Environ. Toxicol. Chem.20(3): 568-574, 2001. ECOREF #59900	Could not relate LC50s to particular species
Karen,D.J., D.R. Ownby, B.L. Forsythe, T.P. Bills, T.W. LaPoint, G.B. Cobb, and S.J. Klaine. Influence of Water Quality on Silver Toxicity to Rainbow Trout (Oncorhynchus mykiss), Fathead Minnows (Pimephales promelas), and Water Fleas (Daphnia magna). Environ. Toxicol. Chem.18(1): 63-70, 1999. ECOREF #19218	
Keller,A.E.. Personal Communication to U.S. EPA: Water Quality and Toxicity Data for Unpublished Unionid Mussel Tests. Memo to R.Pepin and C.Roberts,U.S.EPA Region 5,Chicago, IL:14 p., 2000. ECOREF #76251	Could not find

Citation	Notes
Khangarot,B.S., A. Sehgal, and M.K. Bhasin. "Man and Biosphere" - Studies on the Sikkim Himalayas. Part 5: Acute Toxicity of Selected Heavy Metals on the Tadpoles of Rana hexadactyla. Acta Hydrochim. Hydrobiol.13(2): 259-263, 1985. ECOREF #11438	Non-north american test species used
Khangarot,B.S., P.K. Ray, and H. Chandra. Daphnia magna as a Model to Assess Heavy Metal Toxicity: Comparative Assessment with Mouse System. Acta Hydrochim. Hydrobiol.15(4): 427-432, 1987. ECOREF #12575	
Khangarot,B.S., and P.K. Ray. Sensitivity of Toad Tadpoles, Bufo melanostictus (Schneider), to Heavy Metals. Bull. Environ. Contam. Toxicol.38(3): 523-527, 1987. ECOREF #12339	Non-north american test species used
Khangarot,B.S., and P.K. Ray. Sensitivity of Freshwater Pulmonate Snails, Lymnaea luteola L., to Heavy Metals. Bull. Environ. Contam. Toxicol.41(2): 208-213, 1988. ECOREF #12943	Non-north american test species used
Khangarot,B.S., and P.K. Ray. The Acute Toxicity of Silver to Some Freshwater Fishes. Acta Hydrochim. Hydrobiol.16(5): 541-545, 1988. ECOREF #13149	Non-north american test species used
Kim,J., S. Kim, and S. Lee. Differentiation of the Toxicities of Silver Nanoparticles and Silver Ions to the Japanese Medaka (Oryzias latipes) and the Cladoceran Daphnia magna. Nanotoxicology5(2): 208-214, 2011. ECOREF #160065	Nanoparticle study
Klaine,S.J., T.W. La Point, G.P. Cobb, B.L. Forsythe II, T.P. Bills, M.D. Wenholz, and R.D. Jeffers. Influence of Water Quality Parameters on Silver Toxicity: Preliminary Result. In: A.W.Andren and T.W.Bober (Eds.), Silver in the Environment: Transport, Fate and Effects, Washington, DC:65-77, 1996. ECOREF #20261	Preliminary results
LeBlanc,G.A.. Acute Toxicity of Priority Pollutants to Water Flea (Daphnia magna). Bull. Environ. Contam. Toxicol.24(5): 684-691, 1980. ECOREF #5184	
LeBlanc,G.A., J.D. Mastone, A.P. Paradice, and B.F. Wilson. The Influence of Speciation on the Toxicity of Silver to Fathead Minnow (Pimephales promelas). Environ. Toxicol. Chem.3(1): 37-46, 1984. ECOREF #10538	
Lemke,A.E.. Interlaboratory Comparison Acute Testing Set. EPA-600/3-81-005, U.S.EPA, Duluth, MN:29 p., 1981. ECOREF #9479	Already used in the 1980 criteria derivation
Lima,A.R., C. Curtis, D.E. Hammermeister, D.J. Call, and T.A. Felhaber. Acute Toxicity of Silver to Selected Fish and Invertebrates. Bull. Environ. Contam. Toxicol.29(2): 184-189, 1982. ECOREF #15327	
Mann,R.M., M.J. Ernste, R.A. Bell, J.R. Kramer, and C.M. Wood. Evaluation of the Protective Effects of Reactive Sulfide on the Acute Toxicity of Silver to Rainbow Trout (Oncorhynchus mykiss). Environ. Toxicol. Chem.23(5): 1204-1210, 2004. ECOREF #75078	

Citation	Notes
Morgan,T.P., and C.M. Wood. A Relationship Between Gill Silver Accumulation and Acute Silver Toxicity in the Freshwater Rainbow Trout: Support for the Acute Silver Biotic Ligand Model. Environ. Toxicol. Chem.23(5): 1261-1267, 2004. ECOREF #75070	
Mouneyrac,C., O. Mastain, J.C. Amiard, C. Amiard-Triquet, P. Beaunier, A.Y. Jeantet, B.D. Smith, and P.S. Rainbow. Trace-Metal Detoxification and Tolerance of the Estuarine Worm Hediste diversicolor Chronically Exposed in Their Environment. Mar. Biol.143(4): 731-744, 2003. ECOREF #75379	Lacks method details- controls/replicates; LC50 not reported
Mount,D.I., and T.J. Norberg. A Seven-Day Life-Cycle Cladoceran Toxicity Test. Environ. Toxicol. Chem.3(3): 425-434, 1984. ECOREF #11181	Organisms were fed
Nalecz-Jawecki,G., K. Demkowicz-Dobrzanski, and J. Sawicki. Protozoan Spirostomum ambiguum as a Highly Sensitive Bioindicator for Rapid and Easy Determination of Water Quality. Sci. Total Environ.Suppl(Pt.2):1227-1234, 1993. ECOREF #83577	Bacterial test; single cell organism not appropriate
Nalecz-Jawecki,G., and J. Sawicki. Toxicity of Inorganic Compounds in the Spirotox Test: A Miniaturized Version of the Spirostomum ambiguum Test. Arch. Environ. Contam. Toxicol.34(1): 1-5, 1998. ECOREF #18997	Bacterial test; single cell organism not appropriate
Nebeker,A.V., C.K. McAuliffe, R. Mshar, and D.G. Stevens. Toxicity of Silver to Steelhead and Rainbow Trout, Fathead Minnows and Daphnia magna. Environ. Toxicol. Chem.2:95-104, 1983. ECOREF #10525	Already used in 1980 criteria derivation
Norberg-King,T.J.. An Evaluation of the Fathead Minnow Seven-Day Subchronic Test for Estimating Chronic Toxicity. Environ. Toxicol. Chem.8(11): 1075-1089, 1989. ECOREF #5313	7-day study
Patil,H.S., and M.B. Kaliwal. Relative Sensitivity of a Freshwater Prawn Macrobrachium hendersodyanum to Heavy Metals. Environ. Ecol.4(2): 286-288, 1986. ECOREF #12787	Silver sulfate exposure; 1980 criteria used only silver nitrate
Rodgers,J.H.J., E. Deaver, B.C. Suedel, and P.L. Rogers. Comparative Aqueous Toxicity of Silver Compounds: Laboratory Studies with Freshwater Species. Bull. Environ. Contam. Toxicol.58:851-858, 1997. ECOREF #17981	
Shivaraj,K.M., and H.S. Patil. Toxicity of Silver Chloride to a Fresh Water Fish Lepidocephalichthyes guntea. Environ. Ecol.6(3): 713-716, 1988. ECOREF #806	Non north american test species used
Tsuji,S., Y. Tonogai, Y. Ito, and S. Kanoh. The Influence of Rearing Temperatures on the Toxicity of Various Environmental Pollutants for Killifish (Oryzias latipes). Jpn. J. Toxicol. Environ. Health32(1): 46-53, 1986. ECOREF #12497	Non north american test species used
U.S. Environmental Protection Agency. Pesticide Ecotoxicity Database (Formerly: Environmental Effects Database (EEDB)). Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.:, 1992. ECOREF #344	Reference to a database

Citation	Notes
VanGenderen,E.J., A.C. Ryan, J.R. Tomasso, and S.J. Klaine. Influence of Dissolved Organic Matter Source on Silver Toxicity to Pimephales promelas. Environ. Toxicol. Chem.22(11): 2746-2751, 2003. ECOREF #71734	Test waters included DOC
Vedamanikam,V.J., and N.A.M. Shazili. The Chironomid Larval Tube, a Mechanism to Protect the Organism from Environmental Disturbances?. Toxicol. Environ. Chem.91(1): 171-176, 2009. ECOREF #115860	No hardness data
Vedamanikam,V.J., and N.A.M. Shazilli. Comparative Toxicity of Nine Metals to Two Malaysian Aquatic Dipterian Larvae with Reference to Temperature Variation. Bull. Environ. Contam. Toxicol.80(6): 516-520, 2008. ECOREF #107050	No hardness data
Vedamanikam,V.J., and N.A.M. Shazilli. The Effect of Multi-Generational Exposure to Metals and Resultant Change in Median Lethal Toxicity Tests Values over Subsequent Generations. Bull. Environ. Contam. Toxicol.80(1): 63-67, 2008. ECOREF #111291	No hardness data
Williams,P.L., and D.B. Dusenbery. Aquatic Toxicity Testing Using the Nematode, Caenorhabditis elegans. Environ. Toxicol. Chem.9(10): 1285-1290, 1990. ECOREF #3437	No hardness data
Wu,Y., Q. Zhou, H. Li, W. Liu, T. Wang, and G.Z. Jiang. Effects of Silver Nanoparticles on the Development and Histopathology Biomarkers of Japanese Medaka (Oryzias latipes) Using the Partial-Life Test. Aquat. Toxicol.100:160-167, 2010. ECOREF #151150	Non-north american test species used
Ziegenfuss,P.S., W.J. Renaudette, and W.J. Adams. Methodology for Assessing the Acute Toxicity of Chemicals Sorbed to Sediments: Testing the Equilibrium Partitioning Theory. ASTM Spec. Tech. Publ.9:479-493, 1986. ECOREF #7884	Sediment based study

Open Literature

Table A21. List of open literature citations from EPA ECOTOX database reviewed for silver criteria derivation but did not meet acceptability requirements.

Citation	Notes
Hoheisel, S.M., Diamond, S. and Mount, D., 2012. Comparison of nanosilver and ionic silver toxicity in Daphnia magna and Pimephales promelas. Environmental toxicology and chemistry, 31(11), pp.2557-2563.	No hardness data

Freshwater Chronic

Table A22. List of citations from EPA ECOTOX database reviewed for silver freshwater chronic criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Bielmyer,G.K., R.A. Bell, and S.J. Klaine. Effects of Ligand-Bound Silver on Ceriodaphnia dubia. Environ. Toxicol. Chem.21(10): 2204-2208, 2002. ECOREF #68229	
Call,D.J., C.N. Polkinghorne, T.P. Markee, L.T. Brooke, D.L. Geiger, J.W. Gorsuch, and K.A. Robillard. Silver Toxicity to Chironomus tentans in Two Freshwater Sediments. Environ. Toxicol. Chem.18(1): 30-39, 1999. ECOREF #19468	Sediment study
Davies,P.H., J.P.,Jr. Goettl, and J.R. Sinley. Toxicity of Silver to Rainbow Trout (Salmo gairdneri). Water Res.12(2): 113-117, 1978. ECOREF #2129	
Diamond,J.M., D.G. Mackler, M. Collins, and D. Gruber. Derivation of a Freshwater Silver Criteria for the New River, Virginia, Using Representative Species. Environ. Toxicol. Chem.9(11): 1425-1434, 1990. ECOREF #3774	Field collected orgs and test water
Diamond,J.M., E.L. Winchester, D.G. Mackler, and D. Gruber. Use of the Mayfly Stenonema modestum (Heptageniidae) in Subacute Toxicity Assessments. Environ. Toxicol. Chem.11(3): 415-425, 1992. ECOREF #16355	
Goettl,J.P.,Jr., J.R. Sinley, and P.H. Davies. Water Pollution Studies. Job Progress Report, Federal Aid Project F-33-R-8, DNR, Denver, CO:123 p., 1973. ECOREF #56144	Unable to locate
Goettl,J.P.,Jr., and P.H. Davies. Water Pollution Studies. Job Progress Report, Federal Aid Project F-33-R-11, DNR, Boulder, CO:58 p., 1976. ECOREF #10208	Unable to locate

Citation	Notes
Hobson,J.F.. Acclimation-Induced Changes in Toxicity and Induction of Metallothionein-Like Proteins in the Fathead Minnow Following Sublethal Exposure to Cobalt, Silver, and Zinc. Ph.D.Thesis, University of Kentucky, Lexington, KY:145 p., 1986. ECOREF #150469	Endpoints not relevant
Kolkmeier,M.A., and B.W. Brooks. Sublethal Silver and NaCl Toxicity in Daphnia magna: A Comparative Study of Standardized Chronic Endpoints and Progeny Phototaxis. Ecotoxicology22(4): 693-706, 2013. ECOREF #163942	
LeBlanc,G.A., J.D. Mastone, A.P. Paradice, and B.F. Wilson. The Influence of Speciation on the Toxicity of Silver to Fathead Minnow (Pimephales promelas). Environ. Toxicol. Chem.3(1): 37-46, 1984. ECOREF #10538	Lacking NOEC/LOEC data for silver nitrate
Morgan,T.P., C.M. Guadagnolo, M. Grosell, and C.M. Wood. Effects of Water Hardness on Toxicological Responses to Chronic Waterborne Silver Exposure in Early Life Stages of Rainbow Trout (Oncorhynchus mykiss). Environ. Toxicol. Chem.24(7): 1642-1647, 2005. ECOREF #83081	Only 2 test concentrations
Naddy,R.B., A.B. Rehner, G.R. McNerney, J.W. Gorsuch, J.R. Kramer, C.M. Wood, P.R. Paquin, and W.A. Stubblefield. Comparison of Short-Term Chronic and Chronic Silver Toxicity to Fathead Minnows in Unamended and Sodium Chloride-Amended Waters. Environ. Toxicol. Chem.26(9): 1922-1930, 2007. ECOREF #104889	
Naddy,R.B., J.W. Gorsuch, A.B. Rehner, G.R. McNerney, R.A. Bell, and J.R. Kramer. Chronic Toxicity of Silver Nitrate to Ceriodaphnia dubia and Daphnia magna, and Potential Mitigating Factors. Aquat. Toxicol.84(1): 1-10, 2007. ECOREF #105683	
Nebeker,A.V., C.K. McAuliffe, R. Mshar, and D.G. Stevens. Toxicity of Silver to Steelhead and Rainbow Trout, Fathead Minnows and Daphnia magna. Environ. Toxicol. Chem.2:95-104, 1983. ECOREF #10525	Data included in previous EPA derivation
Norberg-King,T.J.. An Evaluation of the Fathead Minnow Seven-Day Subchronic Test for Estimating Chronic Toxicity. Environ. Toxicol. Chem.8(11): 1075-1089, 1989. ECOREF #5313	
Norberg-King,T.J.. An Evaluation of the Fathead Minnow Seven-Day Subchronic Test for Estimating Chronic Toxicity. M.S.Thesis, University of Wyoming, Laramie, WY:80 p., 1987. ECOREF #17878	Repeat of data from published Norberg-King, 1989
Cremazy,A., K.V. Brix, and C.M. Wood. Chronic Toxicity of Binary Mixtures of Six Metals (Ag, Cd, Cu, Ni, Pb and Zn) to the Great Pond Snail Lymnaea stagnalis. Environ. Sci. Technol.52(10): 5979-5988, 2018. ECOREF #188091 Google Scholar	
Bianchini,A., and C.M. Wood. Does Sulfide or Water Hardness Protect Against Chronic Silver Toxicity in Daphnia magna? A Critial Assessment of the Acute-to-Chronic Toxicity Ratio for Silver. Ecotoxicol. Environ. Saf.71:32-40, 2008. ECOREF #104819	Repeat of other studies

Open Literature

Table A23. List of open literature citations from EPA ECOTOX database reviewed for silver criteria derivation but did not meet acceptability requirements.

Citation	Notes
Okamoto, A., Masunaga, S. and Tatarazako, N., 2021. Chronic toxicity of 50 metals to Ceriodaphnia dubia. Journal of Applied Toxicology, 41(3), pp.375-386.	Very little study details; Effect level reported as inhibitory concentrations; did not use flow through design

Saltwater Acute

Table A24. List of citations from EPA ECOTOX database reviewed for silver saltwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Cardin,J.A.. Unpublished Laboratory Data. U.S.EPA, Narragansett, RI:9 p., 1980. ECOREF #3751	Unpublished data; cannot find
Cardin,J.A.. Results of Acute Toxicity Tests Conducted with Silver at ERL, Narragansett. Memo to J.H.Gentile, U.S.EPA, Narragansett, RI:6 p., 1981. ECOREF #66501	Unpublished data; cannot find
Dinnel,P.A., J.M. Link, Q.J. Stober, M.W. Letourneau, and W.E. Roberts. Comparative Sensitivity of Sea Urchin Sperm Bioassays to Metals and Pesticides. Arch. Environ. Contam. Toxicol.18(5): 748-755, 1989. ECOREF #2264	
Dinnel,P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani. Methodology and Validation of a Sperm Cell Toxicity Test for Testing Toxic Substances in Marine Waters. Final Rep.FRI-UW-8306, Fish.Res.Inst., Schl.of Fish., Univ.of Washington, Seattle, WA:208 p., 1983. ECOREF #3752	Not relevant; gamete study design
Ferguson,E.A., and C. Hogstrand. Acute Silver Toxicity to Seawater-Acclimated Rainbow Trout: Influence of Salinity on Toxicity and Silver Speciation. Environ. Toxicol. Chem.17(4): 589-593, 1998. ECOREF #18940	
Heitmuller,P.T., T.A. Hollister, and P.R. Parrish. Acute Toxicity of 54 Industrial Chemicals to Sheepshead Minnows (Cyprinodon variegatus). Bull. Environ. Contam. Toxicol.27(5): 596-604, 1981. ECOREF #10366	Uncertain data reported
Hook,S.E., and N.S. Fisher. Sublethal Effects of Silver in Zooplankton: Importance of Exposure Pathways and Implications for Toxicity Testing. Environ. Toxicol. Chem.20(3): 568-574, 2001. ECOREF #59900	No specific information on copepod and cladocerans

Citation	Notes
Lee,K.W., S. Raisuddin, J.S. Rhee, D.S. Hwang, I.T. Yu, Y.M. Lee, H.G. Park, and J.S. Lee. Expression of Glutathione S-Transferase (GST) Genes in the Marine Copepod Tigriopus japonicus Exposed to Trace Metals. Aquat. Toxicol.89(3): 158-166, 2008. ECOREF #107127	
Lussier,S.M., J.H. Gentile, and J. Walker. Acute and Chronic Effects of Heavy Metals and Cyanide on Mysidopsis bahia (Crustacea: Mysidacea). Aquat. Toxicol.7(1/2): 25-35, 1985. ECOREF #11331	Already used in EPA 1980 derivation
Lussier,S.M., and J.A. Cardin. Results of Acute Toxicity Tests Conducted with Silver at ERL, Narragansett. U.S.EPA, Narragansett, RI:14 p., 1985. ECOREF #3825	Repeat
Mathew,R., and N.R. Menon. Effects of Heavy Metals on Byssogenesis in Perna viridis (Linn.). Indian J. Mar. Sci.12(2): 125-127, 1983. ECOREF #11120	Non-north American test species
McKenney,C.L.,Jr., and S.H. Hong. Interlaboratory Comparison of Chronic Toxicity Testing Using the Estuarine Mysid (Mysidopsis bahia): A Final Report. U.S.EPA, Gulf Breeze, FL:35 p., 1982. ECOREF #3736	Chronic study
Menasria,R., and J.F. Pavillon. Toxic Effects of Two Trace Metals, Copper and Silver, on a Crustacean Harpacticoid Copepod Tigriopus brevicornis (Muller). Lethal and Sublethal Effects at Different Development Stages (Effets Biologiques de Deux Metaux . J. Rech. Oceanogr.19(3-4): 157-165, 1994. ECOREF #18833	Non-north American test species
Nelson,D.A., J.E. Miller, and A. Calabrese. Effect of Heavy Metals on Bay Scallops, Surf Clams, and Blue Mussels in Acute and Long-Term Exposures. Arch. Environ. Contam. Toxicol.17(5): 595-600, 1988. ECOREF #15056	Adult life stage used for blue mussel
Pavillon,J.F., C. Douez, R. Menasria, J. Forget, J.C. Amiard, and R. Cosson. Impact of Dissolved and Particulate Organic Carbon on the Bioavailability of the Trace Metals Silver and Mercury for the Harpacticoid Copepod Tigriopus brevicornis. J. Rech. Oceanogr.27(1): 43-52, 2002. ECOREF #76315	Used particulate matter in test
Pesch,C.E., and G.L. Hoffman. Interlaboratory Comparison of a 28-Day Toxicity Test with the Polychaete Neanthes arenaceodentata. ASTM Spec. Tech. Publ.:482-493, 1983. ECOREF #10168	Chronic study
Saunders,C.E.. Effects of Dissolved Organic Matter and Salinity on the Toxicity of Individual and Metal Mixtures of Copper with Zinc and Silver to the Saltwater Rotifer, Brachionus plicatilis. M.S. Thesis, Stephen F. Austin State University, Nacogdoches, TX:189 p., 2012. ECOREF #167104	
Schimmel,S.C.. Results: Interlaboratory Comparison - Acute Toxicity Tests Using Estuarine Animals. Final Draft, EPA 600/4-81-003, U.S.EPA, Gulf Breeze, FL:13 p., 1981. ECOREF #3740	

Citation	Notes
Shaw,J.R., C. Hogstrand, M.D. Kercher, and W.J. Birge. The Acute and Chronic Toxicity of Silver to Marine Fish. In: A.W.Andren and T.W.Bober (Eds.), Silver in the Environment: Transport, Fate and Effects, Washington, DC:317-324, 1997. ECOREF #83117	Literature review
Shaw,J.R., C.M. Wood, W.J. Birge, and C. Hogstrand. Toxicity of Silver to the Marine Teleost (Oligocottus maculosus): Effects of Salinity and Ammonia. Environ. Toxicol. Chem.17(4): 594-600, 1998. ECOREF #18941	Repeat
Shaw,J.R., W.J. Birge, and C. Hostrand. Parameters that Influence Silver Toxicity: Ammonia and Salinity. In: 4th Int.Conf.Proc.: Transport, Fate and Effects of Silver in the Environment, Aug.25-28, 1996, Madison, WI:155-159, 1996. ECOREF #20142	Repeat
U.S. Environmental Protection Agency. Pesticide Ecotoxicity Database (Formerly: Environmental Effects Database (EEDB)). Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.:, 1992. ECOREF #344	Database reference
Vijayavel,K., S. Gopalakrishnan, and M.P. Balasubramanian. Sublethal Effect of Silver and Chromium in the Green Mussel Perna viridis with Reference to Alterations in Oxygen Uptake, Filtration Rate and Membrane Bound ATPase System as Biomarkers. Chemosphere69(6): 979-986, 2007. ECOREF #105682	Non-north American test species
Ward,T.J., and J.R. Kramer. Silver Speciation During Chronic Toxicity Tests with the Mysid, Americamysis bahia. Comp. Biochem. Physiol. C Comp. Pharmacol. Toxicol.133(1-2): 75-86, 2002. ECOREF #65743	
Wood,C.M., M.D. McDonald, P. Walker, M. Grosell, J.F. Barimo, R.C. Playle, and P.J. Walsh. Bioavailability of Silver and Its Relationship to Ionoregulation and Silver Speciation Across a Range of Salinities in the Gulf Toadfish (Opsanus beta). Aquat. Toxicol.70:137-157, 2004. ECOREF #75372	

Zinc

Freshwater Acute

Table A25. List of citations from EPA ECOTOX database reviewed for zinc freshwater acute criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Adebayo,O.A., D.P.N. Kio, and O.O. Emmanuel. Assessment of Potential Ecological Disruption Based on Heavy Metal Toxicity, Accumulation and Distribution in Media of the Lagos Lagoon. Afr. J. Ecol.45(4): 454-463, 2007. ECOREF #151240	Non-north american test species used
Agrawal,U.. Effect of Sublethal Concentration of Zinc on Some Hematological Parameters of Freshwater Indian Catfish, Heteropneustes fossilis. J. Adv. Zool.15(2): 86-89, 1994. ECOREF #82971	Non-north american test species used
Alam,M.K., and O.E. Maughan. Acute Toxicity of Heavy Metals to Common Carp (Cyprinus carpio). J. Environ. Sci. Health. Part A, Environ. Sci. Eng. Toxic Hazard. Substance Control30(8): 1807-1816, 1995. ECOREF #45566	Non-north american test species used
Ali,D., S. Alarifi, S. Kumar, M. Ahamed, and M.A. Siddiqui. Oxidative Stress and Genotoxic Effect of Zinc Oxide Nanoparticles in Freshwater Snail Lymnaea luteola L.. Aquat. Toxicol.124/125(0): 83-90, 2012. ECOREF #160562	Nanoparticle study
Alsop,D., and C.M. Wood. Metal Uptake and Acute Toxicity in Zebrafish: Common Mechanisms Across Multiple Metals. Aquat. Toxicol.105(3/4): 385-393, 2011. ECOREF #158223	Hardness <10 mg/L can't compute/unsuitable conditions
Alsop,D.H., J.C. McGeer, D.G. McDonald, and C.M. Wood. Costs of Chronic Waterborne Zinc Exposure and the Consequences of Zinc Acclimation on the Gill/Zinc Interactions of Rainbow Trout in Hard and Soft Water. Environ. Toxicol. Chem.18(5): 1014-1025, 1999. ECOREF #46946	
Alsop,D.H., and C.M. Wood. Influence of Waterborne Cations on Zinc Uptake and Toxicity in Rainbow Trout, Oncorhynchus mykiss. Can. J. Fish. Aquat. Sci.56(11): 2112-2119, 1999. ECOREF #46945	
Alsop,D.H., and C.M. Wood. Kinetic Analysis of Zinc Accumulation in the Gills of Juvenile Rainbow Trout: Effects of Zinc Acclimation and Implications for Biotic Ligand Modeling. Environ. Toxicol. Chem.19(7): 1911-1918, 2000. ECOREF #46947	Study design and endpoints not relevant
Aquatic Toxicology Group. Brenda Mines Sulphate and Molybdenum Toxicity Testing. Proj.Rep.No.2-11-825/826, Prepared for Noranda Mining and Exploration Inc., Brenda Mines Div., B.C.:222 p., 1998. ECOREF #116817	Not available

Citation	Notes
Arambasic,M.B., S. Bjelic, and G. Subakov. Acute Toxicity of Heavy Metals (Copper, Lead, Zinc), Phenol and Sodium on Allium cepa L.., Lepidium sativum L. and Daphnia magna St.: Comparative Investigations and the Practical Applications. Water Res.29(2): 497-503, 1995. ECOREF #13712	No hardness data
Barata,C., D.J. Baird, and S.J. Markich. Influence of Genetic and Environmental Factors on the Tolerance of Daphnia magna Straus to Essential and Non-Essential Metals. Aquat. Toxicol.42(2): 115-137, 1998. ECOREF #19146	
Bechard,K.M., P.L. Gillis, and C.M. Wood. Acute Toxicity of Waterborne Cd, Cu, Pb, Ni, and Zn to First-Instar Chironomus riparius Larvae. Arch. Environ. Contam. Toxicol.54(3): 454-459, 2008. ECOREF #108924	24-hr LC50; control mortality observed after 24 hours
Bianchini,A., K.C. Bowles, C.J. Brauner, J.W. Gorsuch, J.R. Kramer, and C.M. Wood. Evaluation of the Effect of Reactive Sulfide on the Acute Toxicity of Silver (I) to Daphnia magna. Part 2: Toxicity Results. Environ. Toxicol. Chem.21(6): 1294-1300, 2002. ECOREF #66362	
Bianchini,A., and P. Carvalho de Castilho. Effects of Zinc Exposure on Oxygen Consumption and Gill Na+, K+-ATPase of the Estuarine Crab Chasmagnathus granulata Dana, 1851 (Decapoda - Grapsidae). Bull. Environ. Contam. Toxicol.62(1): 63-69, 1999. ECOREF #47569	Non-north american test species used
Bringolf,R.B., B.A. Morris, C.J. Boese, R.C. Santore, H.E. Allen, and J.S. Meyer. Influence of Dissolved Organic Matter on Acute Toxicity of Zinc to Larval Fathead Minnows (Pimephales promelas). Arch. Environ. Contam. Toxicol.51(3): 438-444, 2006. ECOREF #96586	
Brinkman,S., and J. Woodling. Zinc Toxicity to the Mottled Sculpin (Cottus bairdi) in High-Hardness Water. Environ. Toxicol. Chem.24(6): 1515-1517, 2005. ECOREF #84053	
Brinkman,S., and N. Vieira. Water Pollution Studies. Federal Aid Project F-243-R15, Job Progress Report, Colorado Div.of Wildlife, Fort Collins, Co:38 p., 2008. ECOREF #117718	Could not find
Brinkman,S.F., and J.D. Woodling. Acclimation and Deacclimation of Brown Trout (Salmo trutta) to Zinc and Copper Singly and in Combination with Cadmium or Copper. Arch. Environ. Contam. Toxicol.67(2): 214-223, 2014. ECOREF #169219	Acclimization chronic study
Brinkman,S.F., and W.D. Johnston. Acute Toxicity of Aqueous Copper, Cadmium, and Zinc to the Mayfly Rhithrogena hageni. Arch. Environ. Contam. Toxicol.54(3): 466-472, 2008. ECOREF #101773	
Brinkman,S.F., and W.D. Johnston. Acute Toxicity of Zinc to Several Aquatic Species Native to the Rocky Mountains. Arch. Environ. Contam. Toxicol.62(2): 272-281, 2012. ECOREF #161667	

Citation	Notes
Brodeur,J.C., C.M. Asorey, A. Sztrum, and J. Herkovits. Acute and Subchronic Toxicity of Arsenite and Zinc to Tadpoles of Rhinella arenarum both Alone and in Combination. J. Toxicol. Environ. Health Part A72(14): 884-890, 2009. ECOREF #117667	Non-north american test species used
Brooks,A., R.M. White, and D.C. Paton. Effects of Heavy Metals on the Survival of Diacypris compacta (Herbst) (Ostracoda) from the Coorong, South Australia. Int. J. Salt Lake Res.4(2): 133-163, 1995. ECOREF #59762	Non-north american test species used
Brown,R.J., S.D. Rundle, T.H. Hutchinson, T.D. Williams, and M.B. Jones. A Microplate Freshwater Copepod Bioassay for Evaluating Acute and Chronic Effects of Chemicals. Environ. Toxicol. Chem.24(6): 1528-1531, 2005. ECOREF #84071	
Buhl,K.J., and S.J. Hamilton. Toxicity of Inorganic Contaminants, Individually and in Environmental Mixtures, to Three Endangered Fishes (Colorado Squawfish, Bonytail, and Razorback Sucker). Arch. Environ. Contam. Toxicol.30(1): 84-92, 1996. ECOREF #16423	
Bulus Rossini,G.D., and A.E. Ronco. Sensitivity of Cichlasoma facetum (Cichlidae, Pisces) to Metals. Bull. Environ. Contam. Toxicol.72(4): 763-768, 2004. ECOREF #74230	Non-north american test species used
Calfee,R.D., E.E. Little, H.J. Puglis, E. Scott, W.G. Brumbaugh, and C.A. Mebane. Acute Sensitivity of White Sturgeon (Acipenser transmontanus) and Rainbow Trout (Oncorhynchus mykiss) to Copper, Cadmium, or Zinc in Water-Only Laboratory Exposures. Environ. Toxicol. Chem.33(10): 2259-2272, 2014. ECOREF #188154	
Canli,M.. Dietary and Water-Borne Zn Exposures Affect Energy Reserves and Subsequent Zn Tolerance of Daphnia magna. Comp. Biochem. Physiol. C Comp. Pharmacol. Toxicol.141(1): 110-116, 2005. ECOREF #84070	Dietary exposure/preexposure
Centeno,M.D.F., G. Persoone, and M.P. Goyvaerts. Cyst-Based Toxicity Tests. IX. The Potential of Thamnocephalus platyurus as Test Species in Comparison with Streptocephalus proboscideus (Crustacea: Branchiopoda: Anostraca). Environ. Toxicol. Water Qual.10(4): 275-282, 1995. ECOREF #14017	Non-north american test species used
Chan,K.M., L.L. Ku, P.C.Y. Chan, and W.K. Cheuk. Metallothionein Gene Expression in Zebrafish Embryo-Larvae and ZFL Cell-Line Exposed to Heavy Metal Ions. Mar. Environ. Res.62(suppl.1): S83 - S87, 2006. ECOREF #94046	Molecular based endpoints; not relevant
Chen,H.C., and Y.K. Yuan. Acute Toxicity of Copper, Cadmium and Zinc to Freshwater Fish Acrosscheilus paradoxus. Dongwu Xuekan5(2): 45-60, 1994. ECOREF #18913	Non-north american test species used
Chu,K.W., and K.L. Chow. Synergistic Toxicity of Multiple Heavy Metals is Revealed by a Biological Assay Using a Nematode and Its Transgenic Derivative. Aquat. Toxicol.61(1/2): 53-64, 2002. ECOREF #65728	Transgenic test organism

Citation	Notes
Ciji,P.P., and S.B. Nandan. Toxicity of Copper and Zinc to Puntius parrah (Day, 1865). Mar. Environ. Res.93:38-46, 2014. ECOREF #166483	Non-north american test species used
Collyard,S.A., G.T. Ankley, R.A. Hoke, and T. Goldenstein. Influence of Age on the Relative Sensitivity of Hyalella azteca to Diazinon, Alkylphenol Ethoxylates, Copper, Cadmium, and Zinc. Arch. Environ. Contam. Toxicol.26(1): 110-113, 1994. ECOREF #13554	LC50 values not found
Cooper,N.L., J.R. Bidwell, and A. Kumar. Toxicity of Copper, Lead, and Zinc Mixtures to Ceriodaphnia dubia and Daphnia carinata. Ecotoxicol. Environ. Saf.72:1523-1528, 2009. ECOREF #115778	
Crane,M.. Effect of Zinc on Four Populations and Two Generations of Gammarus pulex (L.). Freshw. Biol.33(1): 119-126, 1995. ECOREF #14884	LC50 values not reported; field caught organisms preexposed to zinc
Da Silva Kraus,L.A., A.C.T. Bonecker, N. De Almeida, and A. Vital. Acute Toxicity of Potassium Dichromate, Sodium Dodecyl Sulfate, Copper and Zinc to Poecilia vivipara (Osteichthyes, Cyprinodontiformes). Fresenius Environ. Bull.7(11/12): 654-658, 1998. ECOREF #60132	Non-north american test species used
Dalal,R., and S. Bhattacharya. Effect of Cadmium, Mercury, and Zinc on the Hepatic Microsomal Enzymes of Channa punctatus. Bull. Environ. Contam. Toxicol.52(6): 893-897, 1994. ECOREF #13692	Non-north american test species used
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Van der Schalie,W.H., T.R. Shedd, M.W. Widder, and L.M. Brennan. Response Characteristics of an Aquatic Biomonitor Used for Rapid Toxicity Detection. J. Appl. Toxicol.24(5): 387-394, 2004. ECOREF #77525	
Vedamanikam,V.J., and N.A.M. Shazili. The Chironomid Larval Tube, a Mechanism to Protect the Organism from Environmental Disturbances?. Toxicol. Environ. Chem.91(1): 171-176, 2009. ECOREF #115860	No hardness data
Vedamanikam,V.J., and N.A.M. Shazilli. Comparative Toxicity of Nine Metals to Two Malaysian Aquatic Dipterian Larvae with Reference to Temperature Variation. Bull. Environ. Contam. Toxicol.80(6): 516-520, 2008. ECOREF #107050	No hardness data
Vedamanikam,V.J., and N.A.M. Shazilli. The Effect of Multi-Generational Exposure to Metals and Resultant Change in Median Lethal Toxicity Tests Values over Subsequent Generations. Bull. Environ. Contam. Toxicol.80(1): 63-67, 2008. ECOREF #111291	No hardness data
Viljoen,A., G.J. Steyn, J.H.J. Van Vuren, and P.W. Wade. Zinc Effects on the Embryos and Larvae of the Sharptooth Catfish, Clarias gariepinus (Burchell, 1822). Bull. Environ. Contam. Toxicol.70(5): 1022-1027, 2003. ECOREF #71916	Non-north american test species used
Vyskushenko,A.D.. Effects of Copper Sulfate and Zinc Chloride on Lymnaea stagnalis L.. Hydrobiol. J.42(1): 107-113, 2006. ECOREF #102012	Field collected organisms; lacks study details
Wang,H., R.L. Wick, and B. Xing. Toxicity of Nanoparticulate and Bulk ZnO, Al2O3 and TiO2 to the Nematode Caenorhabditis elegans. Environ. Pollut.157(4): 1171-1177, 2009. ECOREF #108200	Nanoparticle study
Wang,N., C.G. Ingersoll, R.A. Dorman, W.G. Brumbaugh, C.A. Mebane, J.L. Kunz, and D.K. Hardesty. Chronic Sensitivity of White Sturgeon (Acipenser transmontanus) and Rainbow Trout (Oncorhynchus mykiss) to Cadmium, Copper, Lead, or Zinc in Laboratory Water-Only Exposures. Environ. Toxicol. Chem.33(10): 2246-2258, 2014. ECOREF #188097	Chronic data

Citation	Notes
Widianarko,B., F.X.S. Kuntoro, C.A.M. Van Gestel, and N.M. Van Straalen. Toxicokinetics and Toxicity of Zinc Under Time-Varying Exposure in the Guppy (Poecilia reticulata). Environ. Toxicol. Chem.20(4): 763-768, 2001. ECOREF #60205	72 hr toxicity studies, euryhaline species, salinity not reported; possibly invasive
Williams,N.D., and D.A. Holdway. The Effects of Pulse-Exposed Cadmium and Zinc on Embryo Hatchability, Larval Development, and Survival of Australian Crimson Spotted Rainbow Fish (Melanotaenia fluviatilis). Environ. Toxicol.15(3): 165-173, 2000. ECOREF #76127	Non-north american test species used
Wong,C.K., and A.P. Pak. Acute and Subchronic Toxicity of the Heavy Metals Copper, Chromium, Nickel, and Zinc, Individually and in Mixture, to the Freshwater Copepod Mesocyclops pehpeiensis. Bull. Environ. Contam. Toxicol.73(1): 190-196, 2004. ECOREF #80006	Non-north american test species used
Woodling,J., S. Brinkman, and S. Albeke. Acute and Chronic Toxicity of Zinc to the Mottled Sculpin Cottus bairdi. Environ. Toxicol. Chem.21(9): 1922-1926, 2002. ECOREF #68304	
Yang,H.N., and H.C. Chen. The Influence of Temperature on the Acute Toxicity and Sublethal Effects of Copper, Cadmium and Zinc to Japanese Eel, Anguilla japonica. Dongwu Xuekan7(1): 29-38, 1996. ECOREF #18914	Non-north american test species used
Yim,J.H., K.W. Kim, and S.D. Kim. Effect of Hardness on Acute Toxicity of Metal Mixtures Using Daphnia magna: Prediction of Acid Mine Drainage Toxicity. J. Hazard. Mater.B138(1): 16-21, 2006. ECOREF #112477	
Yu,L.P., T. Fang, D.W. Xiong, W.T. Zhu, and X.F. Sima. Comparative Toxicity of Nano-ZnO and Bulk ZnO Suspensions to Zebrafish and the Effects of Sedimentation, OH Production and Particle Dissolution in Distilled Water. J. Environ. Monit.13(7): 1975-1982, 2011. ECOREF #158590	No hardness data
Zhu,X., L. Zhu, Y. Chen, and S. Tian. Acute Toxicities of Six Manufactured Nanomaterial Suspensions to Daphnia magna. J. Nanopart. Res.11:67-75, 2009. ECOREF #153603	Nanoparticle study
Zou,E., and S. Bu. Acute Toxicity of Copper, Cadmium, and Zinc to the Water Flea, Moina irrasa (Cladocera). Bull. Environ. Contam. Toxicol.52(5): 742-748, 1994. ECOREF #13762	Less than value for hardness; hardness too low

Open Literature

Table A26. List of open literature citations from EPA ECOTOX database reviewed for zinc criteria derivation but did not meet acceptability requirements.

Citation	Notes
Moyson, S., Vissenberg, K., Fransen, E., Blust, R. and Husson, S.J., 2018. Mixture effects of copper, cadmium, and zinc on mortality and behavior of Caenorhabditis elegans. Environmental toxicology and chemistry, 37(1), pp.145-159.	No hardness data
Loro, V.L., Nogueira, L., Nadella, S.R. and Wood, C.M., 2014. Zinc bioaccumulation and ionoregulatory impacts in Fundulus heteroclitus exposed to sublethal waterborne zinc at different salinities. Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology, 166, pp.96-104.	Saltwater study
Hose, G.C., Symington, K., Lott, M.J. and Lategan, M.J., 2016. The toxicity of arsenic (III), chromium (VI) and zinc to groundwater copepods. Environmental Science and Pollution Research, 23, pp.18704-18713.	Groundwater test organisms; non-north American test species; field collected organisms with no exposure information
Gawad, S.S.A., 2018. Acute toxicity of some heavy metals to the fresh water snail, Theodoxus niloticus (Reeve, 1856). The Egyptian Journal of Aquatic Research, 44(2), pp.83-87.	Non-north American test species

Freshwater Chronic

Table A27. List of citations from EPA ECOTOX database reviewed for zinc freshwater chronic criteria derivation. If the citation was reviewed but not used for criteria derivation, we provided an explanation in the notes column.

Citation	Notes
Alsop,D.H., S.B. Brown, and G.J. Van der Kraak. Dietary Retinoic Acid Induces Hindlimb and Eye Deformities in Xenopus laevis. Environ. Sci. Technol.38(23): 6290-6299, 2004. ECOREF #110332	Feeding/diet study; not water exposure
Araujo,G.S., C. Pinheiro, J.L.T. Pestana, A. Soares, D.M.S. Abessa, and S. Loureiro. Toxicity of Lead and Mancozeb Differs in Two Monophyletic Daphnia Species. Ecotoxicol. Environ. Saf.178:230-238, 2019. ECOREF #182062	No zinc exposure
Asparch,Y., G. Svartz, and C.S. Perez Coll. Toxicity Characterization and Environmental Risk Assessment of Mancozeb on the South American Common Toad Rhinella arenarum. Environ. Sci. Pollut. Res. Int.27(3): 3034-3042, 2020. ECOREF #182173	Non-north american test species used

Citation	Notes
Atli,G., and M. Canli. Responses of Metallothionein and Reduced Glutathione in a Freshwater Fish Oreochromis niloticus Following Metal Exposures. Environ. Toxicol. Pharmacol.25(1): 33-38, 2008. ECOREF #117067	Non-north american test species used
Balch,G.C., R.D. Evans, P. Welbourn, and R. Prairie. Weight Loss and Net Abnormalities of Hydropsyche betteni (Caddisfly) Larvae Exposed to Aqueous Zinc. Environ. Toxicol. Chem.19(12): 3036-3043, 2000. ECOREF #59272	No hardness data
Barry,M.J.. Effects of Copper, Zinc and Dragonfly Kairomone on Growth Rate and Induced Morphology of Bufo arabicus Tadpoles. Ecotoxicol. Environ. Saf.74(4): 918-923, 2011. ECOREF #161496	Non-north american test species used
Bianchini,A., and C.M. Wood. Does Sulfide or Water Hardness Protect Against Chronic Silver Toxicity in Daphnia magna? A Critical Assessment of the Acute-to-Chronic Toxicity Ratio for Silver. Ecotoxicol. Environ. Saf.71:32-40, 2008. ECOREF #104819	Doesn't include zinc exposure
Bieniarz,K., P. Epler, and M. Sokolowska-Mikolajczyk. Goldfish (Carassius auratus gibelio Bloch) Breeding in Different Concentrations of Zinc. Pol. Arch. Hydrobiol.43(3): 365-371, 1996. ECOREF #84088	Wrong language
Borgmann,U., and W.P. Norwood. Toxicity and Accumulation of Zinc and Copper in Hyalella azteca Exposed to Metal-Spiked Sediments. Can. J. Fish. Aquat. Sci.54:1046-1054, 1997. ECOREF #67044	Sediment study
Brinkman,S., and J. Woodling. Zinc Toxicity to the Mottled Sculpin (Cottus bairdi) in High-Hardness Water. Environ. Toxicol. Chem.24(6): 1515-1517, 2005. ECOREF #84053	
Brinkman,S., and N. Vieira. Water Pollution Studies. Federal Aid Project F-243-R15, Job Progress Report, Colorado Div.of Wildlife, Fort Collins, Co:38 p., 2008. ECOREF #117718	
Brinkman,S.F., and J.D. Woodling. Acclimation and Deacclimation of Brown Trout (Salmo trutta) to Zinc and Copper Singly and in Combination with Cadmium or Copper. Arch. Environ. Contam. Toxicol.67(2): 214-223, 2014. ECOREF #169219	Only 2 test concentrations
Brodeur,J.C., C.M. Asorey, A. Sztrum, and J. Herkovits. Acute and Subchronic Toxicity of Arsenite and Zinc to Tadpoles of Rhinella arenarum both Alone and in Combination. J. Toxicol. Environ. Health Part A72(14): 884-890, 2009. ECOREF #117667	Non-north american test species used
Brown,J.M.. Net Effects of Batrachochytrium dendrobatidis (Bd) and Fungicides on Anurans Across Life Stages. M.S.Thesis, University of South Florida, Tampa, FL:48 p., 2013. ECOREF #175870	Fungicide study

Citation	Notes
Brown,R.J., S.D. Rundle, T.H. Hutchinson, T.D. Williams, and M.B. Jones. A Microplate Freshwater Copepod Bioassay for Evaluating Acute and Chronic Effects of Chemicals. Environ. Toxicol. Chem.24(6): 1528-1531, 2005. ECOREF #84071	
Ciereszko,A., I. Babiak, and K. Dabrowski. Efficacy of Animal Anti-Fertility Compounds Against Sea Lamprey (Petromyzon marinus) Spermatozoa. Theriogenology61(6): 1039-1050, 2004. ECOREF #79860	Study endpoints not relevant
Cooper,N.L., J.R. Bidwell, and A. Kumar. Toxicity of Copper, Lead, and Zinc Mixtures to Ceriodaphnia dubia and Daphnia carinata. Ecotoxicol. Environ. Saf.72:1523-1528, 2009. ECOREF #115778	
Davies,P.H., S. Brinkman, and D. Hansen. Water Pollution Studies. Federal Aid Project F-243R-6, Colorado Division of Wildlife, Fort Collins, CO:47 p., 2000. ECOREF #161558	
Davies,P.H., and S. Brinkman. Water Pollution Studies. Fed.Aid Proj.#F-33, Colorado Div.of Wildl., Fish Res.Sect., Fort Collins, CO:138 p., 1994. ECOREF #90601	
De Schamphelaere,K.A.C., S. Lofts, and C.R. Janssen. Bioavailability Models for Predicting Acute and Chronic Toxicity of Zinc to Algae, Daphnids, and Fish in Natural Surface Waters. Environ. Toxicol. Chem.24(5): 1190-1197, 2005. ECOREF #84052	
De Schamphelaere,K.A.C., and C.R. Janssen. Bioavailability and Chronic Toxicity of Zinc to Juvenile Rainbow Trout (Oncorhynchus mykiss): Comparison with Other Fish Species and Development of a Biotic Ligand Model. Environ. Sci. Technol.38(23): 6201-6209, 2004. ECOREF #84051	
Dorgelo,J., H. Meester, and C. Van Velzen. Effects of Diet and Heavy Metals on Growth Rate and Fertility in the Deposit-Feeding Snail Potamopyrgus jenkinsi (Smith) (Gastropoda: Hydrobiidae). Hydrobiologia316(3): 199-210, 1995. ECOREF #16506	Non-north American test species used
Du,J., J. Tang, S. Xu, J. Ge, Y. Dong, H. Li, and M. Jin. Parental Transfer of Perfluorooctane Sulfonate and ZnO Nanoparticles Chronic Co-Exposure and Inhibition of Growth in F1 Offspring. Regul. Toxicol. Pharmacol.98:41-49, 2018. ECOREF #179529	Nanoparticle study
Du,J., S. Wang, H. You, and Z. Liu. Effects of ZnO Nanoparticles on Perfluorooctane Sulfonate Induced Thyroid-Disrupting on Zebrafish Larvae. J. Environ. Sci.47:153-164, 2016. ECOREF #177092	Nanoparticle study
Fort,D.J., E.L. Stover, and J.A. Bantle. Integrated Ecological Hazard Assessment of Waste Site Soil Extracts Using FETAX and Short-Term Fathead Minnow Teratogenesis Assay. ASTM Spec. Tech. Publ.4:93-109, 1996. ECOREF #45211	Soil study

Citation	Notes
Guo,F., R. Tu, and W.X. Wang. Different Responses of Abalone Haliotis discus hannai to Waterborne and Dietary-Borne Copper and Zinc Exposure. Ecotoxicol. Environ. Saf.91:10-17, 2013. ECOREF #166247	Study endpoints not relevant for criteria development
Heijerick,D.G., C.R. Janssen, and W.M. De Coen. The Combined Effects of Hardness, pH, and Dissolved Organic Carbon on the Chronic Toxicity of Zn to D. magna: Development of a Surface Response Model. Arch. Environ. Contam. Toxicol.44(2): 210-217, 2003. ECOREF #71981	Modeling study; high DOC in testing
Heijerick,D.G., K.A.C. De Schamphelaere, P.A. Van Sprang, and C.R. Janssen. Development of a Chronic Zinc Biotic Ligand Model for Daphnia magna. Ecotoxicol. Environ. Saf.62:1-10, 2005. ECOREF #188078	
Ingersoll,C.G., R.D. Calfee, E. Beahan, W.G. Brumbaugh, R.A. Dorman, D.K. Hardesty, J.L. Kunz, E.E. Little, C.A. Mebane. Acute and Chronic Sensitivity of White Sturgeon (Acipenser transmontanus) and Rainbow Trout (Oncorhynchus mykiss) to Cadmium, Copper, Lead, or Zinc in Laboratory Water-Only Exposures. Sci. Investig. Rep.:120 p., 2014. ECOREF #169495	Found in Wang et al. 2014
Lazorchak,J.M., M.E. Smith, and H.J. Haring. Development and Validation of a Daphnia magna Four-Day Survival and Growth Test Method. Environ. Toxicol. Chem.28(5): 1028-1034, 2009. ECOREF #118322	
Lazorchak,J.M., and M.E. Smith. Rainbow Trout (Oncorhynchus mykiss) and Brook Trout (Salvelinus fontinalis) 7-Day Survival and Growth Test Method. Arch. Environ. Contam. Toxicol.53(3): 397-405, 2007. ECOREF #100026	No hardness data
Magliette,R.J., F.G. Doherty, D. McKinney, and E.S. Venkataramani. Need for Environmental Quality Guidelines Based on Ambient Freshwater Quality Criteria in Natural Waters--Case Study "Zinc". Bull. Environ. Contam. Toxicol.54(4): 626-632, 1995. ECOREF #14962	Case study; not relevant
Martin-Diaz,M.L., S.R. Tuberty, C.L.,Jr. McKenney, D. Sales, and T.A. Del Valls. Effects of Cadmium and Zinc on Procambarus clarkii: Simulation of the Aznalcollar Mining Spill. Cienc. Mar.31(1B): 197-202, 2005. ECOREF #84097	Lacks study details; no hardness data
Mebane,C.A., D.P. Hennessy, and F.S. Dillon. Developing Acute-to-Chronic Toxicity Ratios for Lead, Cadmium, and Zinc Using Rainbow Trout, a Mayfly, and a Midge. Water Air Soil Pollut.:21 p., 2007. ECOREF #97672	
Mebane,C.A., D.P. Hennessy, and F.S. Dillon. Developing Acute-to-Chronic Toxicity Ratios for Lead, Cadmium, and Zinc Using Rainbow Trout, a Mayfly, and a Midge. Water Air Soil Pollut.188(1-4): 41-66, 2008. ECOREF #111766	Repeat
Muyssen,B.T.A., K.A.C. De Schamphelaere, and C.R. Janssen. Mechanisms of Chronic Waterborne Zn Toxicity in Daphnia magna. Aquat. Toxicol.77(4): 393-401, 2006. ECOREF #97407	NOEC/LC50 not provided for ACR development

Citation	Notes
Muyssen,B.T.A., and C.R. Janssen. Age and Exposure Duration as a Factor Influencing Cu and Zn Toxicity Toward Daphnia magna. Ecotoxicol. Environ. Saf.68(3): 436-442, 2007. ECOREF #101832	No hardness data
Nguyen,L.T.H., and C.R. Janssen. Comparative Sensitivity of Embryo-Larval Toxicity Assays with African Catfish (Clarias gariepinus) and Zebra Fish (Danio rerio). Environ. Toxicol.16(6): 566-571, 2001. ECOREF #68928	Non-north american test species used
Oner,M., G. Atli, and M. Canli. Effects of Metal (Ag, Cd, Cr, Cu, Zn) Exposures on Some Enzymatic and Non-Enzymatic Indicators in the Liver of Oreochromis niloticus. Bull. Environ. Contam. Toxicol.82(3): 317-321, 2009. ECOREF #112714	Non-north american test species used
Rohr,J.R., J. Brown, W.A. Battaglin, T.A. McMahon, and R.A. Relyea. A Pesticide Paradox: Fungicides Indirectly Increase Fungal Infections. Ecol. Appl.27(8): 2290-2302, 2017. ECOREF #175858	Fungicide study
Saxena,S., and H. Chaturvedi. Effect of Zinc on the Development of Toad, Bufo fergusonii. J. Ecotoxicol. Environ. Monit.10(4): 259-263, 2000. ECOREF #84089	Non-north american test species used
Shenoy,K., B.T. Cunningham, J.W. Renfroe, and P.H. Crowley. Growth and Survival of Northern Leopard Frog (Rana pipiens) Tadpoles Exposed to Two Common Pesticides. Environ. Toxicol. Chem.28(7): 1469-1474, 2009. ECOREF #118251	Pesticide based study
Vardy,D.W., A.R. Tompsett, J.L. Sigurdson, J.A. Doering, X. Zhang, J.P. Giesy, and M. Hecker. Effects of Subchronic Exposure of Early Life Stages of White Sturgeon (Acipenser transmontanus) to Copper, Cadmium, and Zinc. Environ. Toxicol. Chem.30(11): 2497-2505, 2011. ECOREF #156324	Endpoints not relevant for criteria derivation
Wang,N., C.G. Ingersoll, R.A. Dorman, W.G. Brumbaugh, C.A. Mebane, J.L. Kunz, and D.K. Hardesty. Chronic Sensitivity of White Sturgeon (Acipenser transmontanus) and Rainbow Trout (Oncorhynchus mykiss) to Cadmium, Copper, Lead, or Zinc in Laboratory Water-Only Exposures. Environ. Toxicol. Chem.33(10): 2246-2258, 2014. ECOREF #188097	
Waykar,B., and S.M. Shinde. Assessment of the Metal Bioaccumulation in Three Species of Freshwater Bivalves. Bull. Environ. Contam. Toxicol.87(3): 267-271, 2011. ECOREF #166615	Bioaccumulation study; no toxicity data

Open Literature

Table A28. List of open literature citations from EPA ECOTOX database reviewed for zinc criteria derivation but did not meet acceptability requirements.

Citation	Notes
Okamoto, A., Masunaga, S. and Tatarazako, N., 2021. Chronic toxicity of 50 metals to Ceriodaphnia dubia. Journal of Applied Toxicology, 41(3), pp.375-386.	Study did not use flow through design; very little method details
Calfee, R.D. and Little, E.E., 2017. Toxicity of cadmium, copper, and zinc to the threatened Chiricahua leopard frog (Lithobates [Rana] chiricahuensis). Bulletin of environmental contamination and toxicology, 99, pp.679-683.	Questionable data due to unusual dose-response results

Appendix B. Multiple Linear Regression Dataset and Decisions

Database Qualifiers and Management Decisions

- Locations: irrigation ditches, proximity to salt water bodies, proximity to mining/rock quarry, outside state border

- Studies removed: targeting any kind of discharge event – storm, WWTP, construction, pesticide, fertilizer, CSO. Remediation/taxonomic studies at sites with known pollution and significant human disturbance

- Reviewed "field collection" & "Result" comments for key words like storm sample, discharge event, pesticide application, fertilizer application, QC failed, rain

- Units and outlier parameters – DOC with unit as %, pH above 14, pH with ppm units, TOC parameters labeled as dissolved and vice versa

- Result Data Qualifiers – Qualifiers U, UJ, REJ, E, EQP were removed *While data with EST, J, FS, K, B, JK, JL, NJ, and T were included, the majority of final concurrent data used in the MLR and conversion factors had no qualifier. The J qualifier was the most frequent to remain.

  U = analyte was detected at or above the reported results.
  UJ = analyte was not detected at or above the reported estimate.
  REJ = data was unusable for all purposes.
  E = reported result is an estimate because it exceeds the calibration range.
  EQP = inconsistent equipment performance.
  EST = measurement value reported is estimated.
  J = analyte was positively identified.
  FS = stagnant water – no flow.
  K = reported results with unknown bias.
  B = analyte detected in sample and method blank.
  JK = analyte was positively identified. Reported result is an estimate with unknown bias.
  JL = analyte was positively identified. Value may be less than the reported estimate.
  NJ = there is evidence that the analyte is present in the sample. Reported result is an estimate.
  T = reported result below associated quantitation limit but above MDL.

- EIM QA level 1 was removed (data neither verified nor assessed for usability)

- Federal WQ Portal Result Status Identifier – Rejected

- Data only included fresh/surface waters – all groundwater, marine, springs, estuary, tidal waters, wetlands and canals/ditches were removed

- Data prior to 1/1/2000 was excluded

- Data from the federal WQ Portal that was found to be a duplicate from EIM was removed. The EIM version was retained in use of the MLR dataset

- Locations outside the boundaries of the state were removed. Locations on the Columbia River in the shared waters of Oregon and Washington remained in the dataset

- Duplicates were removed if the percent difference from one another was less than 10%

- Samples were averaged on a daily basis

Database Data Counts

Results from Ecology's Environmental Information Management (EIM) and the Federal Water Quality Portal (WQ Portal):

Data was downloaded on:

- EIM MLR (Including TOC) – March 2023
- Federal WQ Portal MLR (Including TOC) – March 2023
- EIM TOC-DOC Conversion – May 2023 (The practice download was in late Jan/early Feb)
- EIM SpCon-T.Hardness Conversion – August 2023
- Federal WQ Portal SpCon-T.Hardness MLR – August 2023

Count of total download:

- pH
    - EIM – 336,597
    - WQ Portal – 50,876
- DOC
    - EIM – 14,892
    - WQ Portal – 3,231
- Total Hardness
    - EIM – 8,904
    - WQ Portal – 2,314
- TOC (for MLR)
    - EIM – 17,985
    - WQ Portal – 5,361
- Specific Conductivity for MLR [WQ Portal] – 64,109
- DOC for *Conversion Factor* [EIM] – 15,802
- TOC for *Conversion Factor* [EIM] – 18,475
- Total Hardness for *Conversion Factor* [EIM] – 9,445
- Specific Conductivity for *Conversion Factor* [EIM] – 109,392

Total MLR Dataset – 3,337

- Unique locations - 646

Count of concurrent samples for tradition MLR

- EIM – 1,234
- WQ Portal - 1,088

Count of concurrent samples for TOC based MLR

- EIM - 71
- WQ Portal - 34

Count of concurrent samples for Conductivity based MLR - 910

Count of concurrent samples for TOC conversion factor – 6,317

Count of concurrent samples for Specific Conductivity conversion factor - 3,459

The final MLR dataset produced 3,337 concurrent sampling events across 646 unique locations.

Appendix C. 6PPD-quinone WEB-ICE Results

Table C1. 6PPD-quinone WEB-ICE Results

Surrogate	Common Name	Species Name	Predicted LC50 (ug/L)	R^2	Notes
Rainbow trout	Western toad		0.556	0.883	surrogate outside of model range
Rainbow trout	Midge	Chironomus tentans	0.297	0.819	surrogate outside of model range
Rainbow trout	Bullfrog	Lithobates catesbeianus	7.13	0.977	
Rainbow trout	Stonefly	Pteronarcys californica	1.33	0.64	
Rainbow trout	Daphnid	Daphnia magna	2.03	0.83	
Rainbow trout	Daphnid	Daphnia pulex	2.23	0.21	
Rainbow trout	Polychaete	Hydroides elegans	212.71	0.21	surrogate outside of model range
Rainbow trout	Southern leopard frog	Lithobates sphenocephala	2.45	0.95	surrogate outside of model range
Rainbow trout	Stonefly	Claassenia sabulosa	0.41	0.55	
Rainbow trout	Stonefly	Pteronarcella badia	0.715	0.49	
Rainbow trout	Snipefly	Atherix variegata	6.91	0.91	
Rainbow trout	Eastern oyster	Crassostrea virginica	11.1	0.5	
Rainbow trout	Amphipod	Gammarus fasciatus	1.22	0.41	
Rainbow trout	Amphipod	Gammarus lacustris	0.951	0.26	
Rainbow trout	Amphipod	Gammarus pseudolimnaeus	0.258	0.67	
Rainbow trout	Amphipod	Hyalella azteca	0.252	0.57	
Rainbow trout	Beaver tail fairy shrimp	Thamnocephalus platyurus	1.16	0.61	surrogate outside of model range
Rainbow trout	Daphnid	Ceriodaphnia dubia	0.0376	0.64	
Rainbow trout	Daphnid	Simocephalus vetulus	2.08	0.99	surrogate outside of model range
Rainbow trout	Isopod	Caecidotea brevicauda	3.17	0.65	
Rainbow trout	Midge	Chironomus plumosus	2.47	0.5	
Rainbow trout	Midge	Paratanytarsus dissimilis	35.22	0.84	
Rainbow trout	Midge	Paratanytarsus parthenogeneticus	35.12	0.78	surrogate outside of model range
Rainbow trout	Mysid	Americamysis bahia	0.396	0.6	
Rainbow trout	Paper pondshell	Utterbackia imbecillis	12.29	0.6	surrogate outside of model range

Surrogate	Common Name	Species Name	Predicted LC50 (ug/L)	R^2	Notes
Rainbow trout	Pink shirmp	Farfantepenaeus duorarum	0.0591	0.72	
Rainbow trout	Tadpole physa	Physa gyrina	0.671	0.75	surrogate outside of model range
Rainbow trout	Swamp lymnae	Lymnaea stagnalis	0.756	0.73	surrogate outside of model range
Rainbow trout	Versatile fairy shrimp	Branchinecta lindahli	3.94	0.99	surrogate outside of model range
Brook trout	Amphipod	Salvelinus fontinalis	1.03	0.58	
Brook trout	Eastern oyster	Crassostrea virginica	24.12	0.92	
Brook trout	Stonefly	Claassenia sabuolsa	0.21	0.67	
Brook trout	Stonefly	Pteronarcella badia	0.869	0.76	
Brook trout	Stonefly	Pteronarcys californica	1.91	0.41	
Brook torut	Amphipod	Gammarus lacustris	1.03	0.58	
Brook trout	Fowler's toad	Anaxyrus fowleri	106.35	0.94	
Coho salmon	Amphipod	Crangonyx pseudogracilis	4.49	0.8	surrogate outside of model range
Coho salmon	Amphipod	Gammarus fasciatus	0.203	0.41	surrogate outside of model range
Coho salmon	Amphipod	Thamnocephalus platyurus	0.012	0.83	surrogate outside of model range
Coho salmon	Amphipod	Lithobates catesbeianus	0.639	0.63	surrogate outside of model range
Coho salmon	Beaver tail fairy shrimp	Thamnocephalus platyurus	0.012	0.83	surrogate outside of model range
Coho salmon	Bullfrog	Lithobates catesbeianus	0.639	0.99	surrogate outside of model range
Coho salmon	Daphnid	Daphnia magna	0.324	0.35	surrogate outside of model range
Coho salmon	Isopod	Caecidotea brevicauda	0.274	0.63	surrogate outside of model range
Coho salmon	Rainbow mussel	Villosa iris	0.00204	0.99	surrogate outside of model range
Coho salmon	Snipefly	Atherix variegata	0.73	0.94	
Coho salmon	Southern leopard frog	Lithobates sphenocephala	0.00201	0.99	surrogate outside of model range
Coho salmon	Stonefly	Pteronarcella badia	0.433	0.86	surrogate outside of model range
zebrafish	Flagfish	Jordanella floridae	61.09	0.99	
zebrafish	Medaka	Oryzias latipes	595.94	0.78	

Appendix D. PARIS Query

Identifying Future Changes to Permits (PARIS Query)

As part of this rulemaking, we conducted a permitting and reporting information system (PARIS) query to evaluate how permits may be impacted as a result of this rulemaking. We used discharge monitoring report (DMR) data and priority pollutant scan information to determine the potential for permitted effluent discharges to cause an exceedance of revised toxics criteria. This analysis is not definitive, and methods used do not account for all facets of developing effluent limits. However, this analysis provides an approximation of which permits may need closer review since they do have these chemicals in their effluent. The costs to permitting is evaluated in the Preliminary Regulatory Analysis[9].

Methods

Ecology evaluates the need for water quality-based effluent limits in each individual permit based on effluent variability, sampling frequencies, dilution factors (if applicable), and the water quality criteria. Permittees report data on toxics in the effluent on their routine DMRs and priority pollutant scans, which is stored in PARIS. We selected the following parameters in PARIS for inclusion into the query spreadsheet: water quality name, permit number, permit type, permit status, feature name, city, county, monitoring point code, parameter, unit, fraction, statistical base, is report only, benchmark min, benchmark max, limit min, limit max, param impairment, parameter notes, feature latitude, and feature longitude. We searched for permits for toxic chemicals that are proposed to have lower criteria or are new to the water quality standards.

We searched PARIS for effluent data for the following toxic chemicals:

[9] https://apps.ecology.wa.gov/publications/summarypages/2410009.html

- aluminum
- arsenic
- cadmium
- chromium III
- chromium VI
- copper
- nickel
- mercury
- selenium
- silver
- zinc
- 6PPD-quinone
- acrolein
- carbaryl
- cyanide
- demeton
- diazinon
- endrin
- gamma-BHC (lindane)
- guthion
- malathion
- methoxychlor
- mirex
- nonylphenol
- pentachlorophenol
- PFOS
- PFOA
- tributyltin

For hardness-based metals, we used a default hardness of 70.2 mg/L to calculate the criteria, which represents the statewide mean value based on data in the EIM database collected by Ecology's Environmental Assessment Program since 2000. We set the matrix for water, filtered out data for only river/streams, used Quality Assurance (QA) level 2 or higher, and removed samples during storm events.

For pH-based pentachlorophenol, we used a default pH of 7.8, which represents the statewide mean value based on data in EIM. The pH data used to calculate a statewide mean value used all pH data in the EIM database under the study type of RoutineMonitor, HabitatMonitoring, or GenEnvironmentalStudy Field Collection, collected on or after October 1, 2013, with a sample matrix of water and a sample source of fresh/surface water. We filtered the pH data to include QA level 2 or higher and data for rivers/streams.

For aluminum and copper, we used statewide values for pH, hardness, and DOC to calculate criteria using the multiple linear regression (MLR) as the representative criteria for comparison to effluent data. The statewide mean for concurrently sampled data was a pH of 7.58, hardness of 59.69 mg/L, and 2.71 mg/L DOC. The copper criteria are 9.3 ug/L for freshwater acute and 7.3 mg/L for freshwater chronic using statewide mean values for pH, hardness, and DOC. The aluminum criteria are 2100 ug/L for freshwater acute and 780 ug/L for freshwater chronic using statewide mean values for pH, hardness, and DOC. We reviewed the last 10 years for individual permits because permit renewal can be delayed and priority pollutant scan information from the last renewal is relevant to this analysis. We reviewed only the last two years for general permits because of corrective actions that are employed when a discharger is not meeting effluent limits. The most recent monitoring data are relevant because if there was an exceedance demonstrated during monitoring, actions should currently be underway to make a correction. Effluent exceedances prior to 2021 should have already been corrected; thus, only the most recent effluent data are relevant to evaluating permittees compliance with current and proposed aquatic life criteria for general permits.

For analysis of individual permits, we applied the acute and chronic dilution factors from each individual permit fact sheet to the proposed acute and chronic aquatic life criteria. The application of dilution factors to the newly proposed aquatic life criteria was representative of the potential effluent limit for each pollutant. We then compared the maximum reported effluent concentration from each permit's dataset to the respective calculated limit (aquatic life criterion divided by the dilution factor). Some permits do not have a dilution factor, for example if they discharge to a 303(d) listed water body. If the calculated limit was less than the maximum concentration reported in the monitoring data, then that discharge was deemed to have a reasonable potential to cause an exceedance of the proposed criterion, which could result in a new or revised effluent limits. This method for estimating permit limits is a conservative approach because it does not account for effluent variability, sampling frequencies, flow, and statistical based approaches typically used to calculate effluent limits that would likely drive effluent limits lower than the approach used in this analysis. We tallied all the individual NPDES permits for industrial and municipal entities that could potentially need changes to the effluent limits based on their effluent exceeding calculated limits using the methods described above. Individual permits were removed from consideration in this analysis when they did not have a reported pollutant concentration above the calculated limit.

For determining whether general permits could be affected by this rule, we compared maximum concentrations reported in DMRs or priority pollutant scans in PARIS to the applicable acute aquatic life toxics criteria. The acute toxics criteria are the more pertinent criteria to the general permits based on the short-term duration of general permit discharges such as stormwater runoff and time-limited discharges. If the maximum toxic concentration in effluent for a given permit exceeded the proposed aquatic life toxics acute criteria, the permit was listed as potentially of concern under the new criteria. Comparing the acute toxics criteria to the effluent data represents a conservative estimate of the number of permits potentially affected in this rulemaking. For example, the industrial stormwater general permit uses benchmark values rather than direct comparisons to the acute toxics criteria. The benchmark values are usually equal to or higher than the acute toxics criteria. Furthermore, the industrial stormwater permit allows for corrective actions in their stormwater pollution prevention plan (SWPPP) to meet benchmarks. An exceedance of the benchmark does mean there is a violation of permit requirements. For other general permits without numeric limits, a qualitative analysis was completed based on the permit description to determine where this rulemaking could potentially impact the permit.

Results

The PARIS query found reported information for the following permits listed below based on the filtering methods described in the methodology section. Other permit types are not included here because they do not discharge into surface waters of the state, the permit may not require monitoring of toxics in the effluent, or their effluent data was below the revised criteria or calculated limits. The impacts of new toxics to the water quality standards are not captured here because they are not currently incorporated into existing permits. A reasonable potential analysis will need to be conducted on new toxics to determine if a given permit requires a permit condition or limit.

Individual Permits

We identified 28 industrial and 18 municipal individual NPDES permits, for a total of 46 individual permits, that may require new or revised effluent limits based on the proposed criteria. The maximum reported discharge levels in DMR data from 46 different individual permits are anticipated to exceed potential limits based on the proposed criteria in this rulemaking. The parameters that have potential to affect permitted effluent limits are listed in Table D1.

Table D1. The number of individual permits that have potential to require new or revised limits based on the proposed criteria.

Toxic chemical	Industrial NPDES	Municipal NPDES
Acrolein	2	1
Aluminum	2	-
Arsenic	2	3
Cadmium	3	3

Chromium VI	4	2
Copper	15	7
Cyanide	2	4
Mercury	-	3
Nickel	6	3
Pentachlorophenol	4	-
Selenium	3	1
Silver	3	6
Zinc	18	8

State Waste Discharge Permit: Individual Pretreatment Permit

There are 46 individual pretreatment permits that could be impacted by this rulemaking (based on direct comparison of the effluent pollutant levels to the calculated limits described above using dilution factors). However, pretreatment dischargers, industrial facilities discharging to publicly owned treatment works (POTWs), do not receive effluent limits calculated directly from water quality criteria. Instead, to protect operations and to ensure compliance with state and federal requirements, POTWs will design local limits based on site-specific criteria such as applicable water quality criteria.

Ecology delegates authority to municipalities for discharge permits for industries discharging to their POTW and also issues permits for industries discharging to non-delegated municipalities. This rulemaking may require delegated municipalities, POTWs, and Ecology to reevaluate local limits and/or modify discharge permits for industries if necessary for the POTW to comply with new limits in their NPDES permit and changing water quality criteria. We cannot definitively determine whether pretreatment permits will be impacted. Of the 50 individual pretreatment permits, potential impacts for specific parameters in permits include aluminum (6), arsenic (3), cadmium (23), copper (40), cyanide (18), lead (30), mercury (5), nickel (31), pentachlorophenol (1), selenium (11), silver (19), and zinc (39).

Industrial Stormwater General Permit

We identified 540 industrial stormwater general permits that could be impacted by this rulemaking. The maximum reported discharge in DMRs from 634 different permits are anticipated to exceed limits based on the proposed criteria in this rulemaking. Potential exceedances by parameter in the 540 permits were as follows: arsenic (1), copper (371), mercury (2), and zinc (499). Industrial stormwater general permits are based on benchmarks, and an exceedance does not necessarily equate to violation of permit conditions. Industrial stormwater general permits have a SWPPP that allows for corrective actions to take place to maintain compliance.

Boatyard General Permit

We identified eight boatyard permits that could be impacted by this rulemaking. The maximum reported discharge in DMRs from eight different boatyard permits are anticipated to exceed limits based on the proposed criteria in this rulemaking. Of the eight boatyard permits, copper was exceeded in all eight permits and zinc in five of the permits.

Construction Stormwater General Permit

We identified five construction stormwater general permits that could be impacted by this rulemaking. The maximum reported discharge in DMRs from six different construction stormwater general permits are anticipated to exceed limits based on the proposed criteria in this rulemaking. Of the six construction stormwater general permits, the following toxics were of concern: cadmium (1), copper (3), mercury (1), and zinc (2).

Municipal Stormwater General Permit

The municipal stormwater general permit does not require numeric effluent limits that permittees need to meet (except in some cases to meet TMDL-related requirements; e.g., total suspended solids). These permits are written to require stormwater management programs that establish narrative effluent limits, based on best management practices, to meet water quality standards. Thus, the proposed criteria in this rulemaking could result in an assessment of appropriate best management practices to ensure water quality standards will continue to be met.

Irrigation System Aquatic Weed Control General Permit

The irrigation system aquatic weed control general permit contains limits for copper and acrolein, two toxics that are part of this rulemaking. The freshwater copper criteria are currently hardness-based, which requires hardness data. The copper criteria proposed are based on the MLR model and will now require hardness, pH, and dissolved organic carbon levels to calculate criteria. The proposed copper criteria will also include default copper criteria based on a 5^{th} percentile of criteria calculated from concurrently monitored hardness, pH, and dissolved organic carbon collected throughout the state. If there is sufficient water quality data, a copper criterion will be calculated use site-specific data. If there is not water quality data available for a water body, Ecology may decide to use the 5^{th} percentile default criteria in the irrigation general permit or require permittees to sample hardness, pH, and dissolved organic carbon in receiving waters or compliance points for this permit. Copper criteria may increase or decrease compared with current irrigation permit requirements based on the unique water quality of a site-specific location or water body.

Washington does not currently have acrolein criteria in the surface water quality standards. In this rulemaking, we are proposing to adopt EPA recommendations for acrolein. Future acrolein permits may include a lower limit given that current limits are based on outdated EPA criteria.

Aquatic Invasive Species Management General Permit

The aquatic invasive species management (AISM) general permit includes the application of chelated copper to water bodies to control aquatic invasive species. This rulemaking is proposing a MLR-based copper criteria which may result in higher or lower copper criteria

based on the unique water quality characteristics of the water body. The AISM permit currently uses short-term modifications during the application of chelated copper that allows for a temporary zone of impact with recognition of the benefits of the application to the water body and full restoration following application. We anticipate that if the proposed copper aquatic life criteria are adopted, short-term modifications will continue to be used for chelated copper treatments in the AISM permit and that it will have minimal impact to this permit.

Aquatic Plant and Algae Management General Permit

This rulemaking is proposing the addition of an aluminum criteria to Washington's surface water quality standards. The aquatic plant and algae management (APAM) general permit includes ALUM treatments to control aquatic plants. ALUM treatment consists of the application of high levels of aluminum to water bodies. We anticipate that ALUM treatments could result in short-term exceedances of the proposed aluminum aquatic life criteria. Currently, the APAM permit uses short-term modifications to apply ALUM treatments that allows for a temporary zone of impact with recognition of the benefits of the application to the water body and full restoration following application. We anticipate that if aluminum aquatic life criteria are adopted, short-term modifications will continue to be used for ALUM treatments in the APAM permit and that it will have minimal impact to this permit. Future monitoring of aluminum during ALUM applications may need to be considered for this permit.

Appendix E. Water Quality Assessment Analysis

Analysis of Water Concentrations Relative to Criteria

This analysis is not representative of the water quality assessment process but rather provides a rough estimate on how statewide water quality samples compare to the criteria. This analysis provides speculation around where the proposed criteria may result in a need to update 303(d) listings. We extracted all the data from January 2013 to January 2023 for toxics that are new or becoming more stringent in the proposed rulemaking from Ecology's EIM database. We evaluated the amount of data that exceeds the current criteria versus the proposed criteria to get an estimate of the percent increase in exceedances of the data available for statewide water quality assessments. When the criteria were less than the reporting limit for the analytical method, the U and UJ qualifiers (which signify non-detects) were removed from consideration because the reporting limit was greater than the criteria and would count toward an exceedance.

We also removed quality assurance and planning levels of one and two from this analysis to ensure the data we used in our analysis were of high quality. In our analysis, a single sampling event was considered the average daily concentration for a given location. We compared the average concentration to the current criteria and the proposed criteria to determine if the sample exceeded the respective criteria. For hardness-based metals criteria, we used a default hardness of 70.2 mg/L, which represents the statewide mean value based on data in EIM since 2000. We used mean statewide inputs for concurrently sampled pH (7.58), hardness (59.69 mg/L), and DOC (2.71 mg/L) to calculate the MLR based aluminum and copper criteria being proposed.

The results from this analysis in Table 5 demonstrated that revising some criteria may result in additional 303(d) listings. Of the highest concerns in this analysis are the following criteria (>3% percent increase in exceedance of all state data): 6PPD-quinone freshwater (FW) acute, cyanide FW acute, cyanide FW chronic, endrin FW acute, nickel FW chronic, pentachlorophenol FW acute, pentachlorophenol FW chronic, selenium FW chronic, and zinc FW chronic. This analysis does not mean there will be any new 303(d) listings because this analysis did not follow all steps of Policy 1-11, and exceedance data may be from one or multiple locations (e.g., if there are 10 exceedances, all samples may be from one stream, or they could be from 10 different streams).

Table E1. Evaluation of statewide data in comparison to the current and proposed criteria for new toxics or toxics becoming more stringent.

Toxic Criteria	No. of Samples	Percent Exceedance Current Criteria	Percent Exceedance Proposed Criteria	Percent Increase in Exceedances	Notes
6PPD-quinone FW Acute	4	N/A	75.0%	75.0%	
Acrolein FW Acute	0	N/A	0.00%	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Acrolein FW Chronic	0	N/A	0.00%	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Aluminum FW Acute	452	N/A	0.00%	N/A	Used statewide mean input values for concurrently sampled pH, hardness, and DOC for the aluminum MLR model.
Aluminum FW Chronic	452	N/A	1.55%	N/A	Used statewide mean input values for concurrently sampled pH, hardness, and DOC for the aluminum MLR model.
Arsenic FW Acute	799	0.13%	0.13%	0.00%	
Arsenic FW Chronic	799	1.00%	2.75%	1.75%	
Arsenic SW Acute	17	0.00%	0.00%	0.00%	
Arsenic SW Chronic	17	0.00%	0.00%	0.00%	

Toxic Criteria	No. of Samples	Percent Exceedance Current Criteria	Percent Exceedance Proposed Criteria	Percent Increase in Exceedances	Notes
Cadmium FW Acute	335	3.28%	4.48%	1.20%	
Cadmium FW Chronic	335	4.48%	7.16%	2.68%	
Cadmium SW Acute	14	0.00%	0.00%	0.00%	
Cadmium SW Chronic	14	0.00%	0.00%	0.00%	
Carbaryl FW Acute	532	N/A	20.68%	N/A	
Carbaryl FW Chronic	532	N/A	20.68%	N/A	
Carbaryl SW Acute	1	N/A	0.00%	N/A	
Chromium III FW Chronic	0	N/A	N/A	N/A	No chromium II samples.
Chromium VI FW Chronic	0	N/A	N/A	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Copper FW Acute	868	0.57%	1.15%	0.58%	Used mean hardness of 70.2 mg/L for current copper hardness based criteria and statewide mean input values for concurrently sampled pH, hardness, and DOC for the copper MLR model.
Copper FW Chronic	868	1.38%	1.61%	0.23%	Used mean hardness of 70.2 mg/L for current copper hardness based criteria and statewide mean

Toxic Criteria	No. of Samples	Percent Exceedance Current Criteria	Percent Exceedance Proposed Criteria	Percent Increase in Exceedances	Notes
					input values for concurrently sampled pH, hardness, and DOC for the copper MLR model.
Cyanide FW Acute	21	4.76%	9.52%	4.76%	
Cyanide FW Chronic	21	66.67%	100%	33.33%	
Demeton FW Chronic	0	N/A	0.00%	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Demeton SW Chronic	0	N/A	0.00%	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Diazinon FW Acute	551	N/A	0.73%		
Diazinon FW Chronic	551	N/A	0.73%		
Diazinon SW Acute	4	N/A	0.00%	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Diazinon SW Chronic	4	N/A	0.00%	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Dieldrin FW Acute	255	0.00%	0.39%	0.39%	
Endrin FW Acute	225	0.00%	8.44%	8.44%	

Toxic Criteria	No. of Samples	Percent Exceedance Current Criteria	Percent Exceedance Proposed Criteria	Percent Increase in Exceedances	Notes
Gamma-BHC FW Acute	225	0.00%	0.00%	0.00%	
Guthion FW Chronic	0	N/A	N/A	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Guthion SW Chronic	0	N/A	N/A	N/A	No saltwater samples.
Malathion FW Chronic	535	N/A	1.12%	N/A	
Malathion SW Chronic	0	N/A	N/A	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Mercury FW Acute	392	0.00%	0.00%	0.00%	
Methoxychlor FW Chronic	0	N/A	N/A	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Methoxychlor SW Chronic	0	N/A	N/A	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Mirex FW Chronix	0	N/A	N/A	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Mirex SW Chronic	0	N/A	N/A	N/A	Criteria < Reporting Limit.

Toxic Criteria	No. of Samples	Percent Exceedance Current Criteria	Percent Exceedance Proposed Criteria	Percent Increase in Exceedances	Notes
					Removed non-detects. No samples to evaluate.
Nickel FW Acute	410	0.00%	0.24%	0.24%	
Nickel FW Chronic	410	0.24%	3.41%	3.17%	
Nonylphenol FW Acute	3	N/A	0.00%	0.00%	
Nonylphenol FW Chronic	3	N/A	0.00%	0.00%	
Nonylphenol SW Acute	15	N/A	0.00%	0.00%	
Nonylphenol SW Chronic	15	N/A	0.00%	0.00%	
Pentachlorophenol FW Acute	596	0.00%	5.20%	5.20%	
Pentachlorophenol FW Chronic	596	0.00%	5.20%	5.20%	
Pentachlorophenol SW Chronic	0	N/A	N/A	N/A	Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
PFOS FW Acute	0	N/A	N/A	N/A	
PFOS FW Chronic	0	N/A	N/A	N/A	
PFOS SW Acute	0	N/A	N/A	N/A	
PFOS SW Chronic	0	N/A	N/A	N/A	
PFOA FW Acute	0	N/A	N/A	N/A	
PFOA FW Chronic	0	N/A	N/A	N/A	
PFOA SW Acute	0	N/A	N/A	N/A	
PFOA SW Chronic	0	N/A	N/A	N/A	
Selenium FW Acute	126	0.79%	N/A	N/A	Proposed criteria does not include acute criteria.
Selenium FW Chronic	126	0.79%	3.97%	3.18%	
Silver FW Acute	516	0.19%	1.37%	1.18%	Some Reporting Limits less than

Toxic Criteria	No. of Samples	Percent Exceedance Current Criteria	Percent Exceedance Proposed Criteria	Percent Increase in Exceedances	Notes
					the criteria were removed.
Silver FW Chronic	409	N/A	3.91	N/A	Currently do not have chronic criteria. Criteria < Reporting Limit. Removed non-detects. No samples to evaluate.
Silver SW Chronic	8	0.00%	0.00%	0.00%	
Tributyltin FW Acute	0	N/A	N/A	N/A	
Tributyltin FW Chronic	0	N/A	N/A	N/A	
Tributyltin SW Acute	0	N/A	N/A	N/A	
Tributyltin SW Chronic	0	N/A	N/A	N/A	
Zinc FW Acute	6706	1.17%	2.94%	1.77%	
Zinc FW Chronic	6706	1.35%	4.88%	3.53%	