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Aquatic Life Ambient Water Quality Criteria – Nonylphenol

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Ambient Aquatic Life Water Quality Criteria

Nonylphenol

(CAS Registry Number 84852-15-3)
(CAS Registry Number 25154-52-3)

FINAL

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U.S. Environmental Protection Agency
Office of Water
Office of Science and Technology
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NOTICE

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FOREWORD

Section 304(a)(l) of the Clean Water Act of 1977 (P.L. 95-217) requires the Administrator of the Environmental Protection Agency to publish water quality criteria that accurately reflect the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare that might be expected from the presence of pollutants in any body of water, including ground water. This document is a revision of proposed criteria based upon consideration of comments received from independent peer reviewers and the public. Criteria contained in this document replace any previously published EPA aquatic life criteria for the same pollutant(s).

The term "water quality criteria" is used in two sections of the Clean Water Act, section 304(a)(l) and section 303(c)(2). The term has a different program impact in each section. In section 304, the term represents a non-regulatory, scientific assessment of health or ecological effects. Criteria presented in this document are such scientific assessments. If water quality criteria associated with specific waterbody uses are adopted by a state or tribe as water quality standards under section 303, they become enforceable maximum acceptable pollutant concentrations in ambient waters within that state or tribe. Water quality criteria adopted in state or tribal water quality standards could have the same numerical values as criteria developed under section 304. However, in many situations states or tribes might want to adjust water quality criteria developed under section 304 to reflect local environmental conditions and human exposure patterns. Alternatively, states or tribes may use different data and assumptions than EPA in deriving numeric criteria that are scientifically defensible and protective of designated uses. It is not until their adoption as part of state or tribal water quality standards that criteria become regulatory. Guidelines to assist the states and tribes in modifying the criteria presented in this document are contained in the Water Quality Standards Handbook (U.S. EPA 1994). The handbook and additional guidance on the development of water quality standards and other water-related programs of this agency have been developed by the Office of Water.

This final document is guidance only. It does not establish or affect legal rights or obligations. It does not establish a binding norm and cannot be finally determinative of the issues addressed. Agency decisions in any particular situation will be made by applying the Clean Water Act and EPA regulations on the basis of specific facts presented and scientific information then available.

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1. INTRODUCTION

1.1. Physical-Chemical Properties

Nonylphenol ($C_{15}H_{24}O$) is produced from cyclic intermediates in the refinement of petroleum and coal-tar crudes. It is manufactured by alkylating phenol with mixed isomeric nonenes in the presence of an acid catalyst. The resulting product is a mixture of various isomers of nonylphenol, predominantly *para*-substituted nonylphenol, (phenol, 4-nonyl-branched, CAS No. 84852-15-3; 4-nonylphenol, CAS No. 104-40-5; and phenol, nonyl-, CAS No. 25154-52-3) with small amounts of *ortho*-substituted phenol (2-nonylphenol, CAS No. 136-83-4), and trace amounts of 2,4-dinonylphenol (phenol, dinonyl, branched, CAS No. 84962-08-3). Additional isomers, which represent the numerous branched structures that occur within the nonyl (nine carbon) group, add to the complexity of the compound. Commercial nonylphenol is most accurately described by CAS number 84852-15-3 (phenol, 4-nonyl-branched), but CAS numbers 104-40-5 (phenol, 4-nonyl-) and 25154-52-3 (phenol, nonyl) have also been used to describe these compounds commercially. The criteria derived in this document address the CAS numbers 84852-15-3 and 25154-52-3.

There is little direct use for nonylphenol except as a mixture with diisobutyl phthalate to color fuel oil for taxation purposes and with acylation to produce oxime as an agent to extract copper. Most nonylphenol is used as an intermediate in the production of other chemicals. Notably, nonionic surfactants of the nonylphenol ethoxylate type are produced through etherification of nonylphenol by condensation with ethylene oxide in the presence of a basic catalyst. The nonionic surfactants are used as oil soluble detergents and emulsifiers that can be sulfonated or phosphorylated to produce anionic detergents, lubricants, antistatic agents, high performance textile scouring agents, emulsifiers for agrichemicals, antioxidants for rubber manufacture, and lubricant oil additives (Reed 1978).

Nonylphenol is produced in large quantities in the United States. Production was 147.2 million pounds (66.8 million kg) in 1980 (USITC 1981), 201.2 million pounds (91.3 million kg) in 1988 (USITC 1989), 230 million pounds (104 million kg) in 1998 (Harvilicz 1999), and demand is increasing about 2 percent annually. Nonylphenol is a pale yellow highly viscous liquid with a slight phenolic odor, an approximate molecular weight of 215.0 to 220.4 g/mole, a

specific gravity of 0.953 g/mL at 20°C (Budavari 1989), and a vapor pressure of 4.55×10^{-3} ($\pm 3.54 \times 10^{-3}$) Pa (Roy F. Weston Inc. 1990). It has a dissociation constant (pK_a) of 10.7 ± 1.0 and a log octanol/water partition coefficient (log K_{ow}) of 3.80 to 4.77 (Roy F. Weston Inc. 1990). The water solubility of nonylphenol is pH-dependent; 4,600 µg/L at pH 5.0, 6,237 µg/L at pH 7.0, 11,897 µg/L at pH 9.0. Nonylphenol is soluble in seawater at 3,630 µg/L and is soluble in many organic solvents (Roy F. Weston Inc. 1990). Ahel and Giger (1993) measured the solubility of nonylphenol at different temperatures in distilled water and demonstrated a nearly linear increase in solubility between 2°C (4,600 µg/L) and 25°C (6,350 µg/L).

1.2. Nonylphenol in the Environment

Nonylphenol and nonylphenol ethoxylates have been found in the environment and a review of studies describing their distribution has been published (Bennie 1999). Shackelford et al. (1983) reported 4-nonylphenol at average concentrations ranging from 2 to 1,617 µg/L in eleven water samples associated with various industrial sources. Bennie et al. (1997) measured nonylphenol in water in 25 percent of sites sampled in the Great Lakes at concentrations from 0.01 to 0.92 µg/L. They found nonylphenol in all sediment samples with concentrations ranging from 0.17 to 72 µg/g (dry weight). Nonylphenol and its ethoxylates have been found in treatment plant wastewaters (Ellis et al. 1982, Giger et al. 1981) and in sewage sludges (Giger et al. 1984). In a study of airport runoff, nonylphenol was measured at 0.98 and 7.67 µg/L in the runoff as a result of aircraft deicer and antiicer fluid use (Corsi et al. 2003). A study was conducted of thirty river reaches in the continental U.S. in 1989 and 1990 to determine the frequency and concentrations of nonylphenol and its ethoxylates in water and sediments. Nonylphenol was found in approximately 30 percent of the water samples with concentrations ranging from about 0.20 to 0.64 µg/L. Approximately 71 percent of the sampling sites had measurable concentrations of nonylphenol in the sediments at concentrations ranging from about 10 to 2,960 µg/kg. Ethoxylates of nonylphenol were found in 59 to 76 percent of the water samples, with amounts varying by extent of ethoxylation (Naylor 1992, Naylor et al. 1992, Radian Corp. 1990).

Most nonylphenol enters the environment as 4-alkylphenol polyethoxylate surfactants which are degraded to 4-alkylphenol mono- and diethoxylates in active sewage sludges (Giger et al.

1984). It was theorized by Giger et al. (1984) that further transformation of 4-alkylphenol mono- and diethoxylates to 4-nonylphenol is favored by anaerobic environments. They conducted experiments with stabilized (anaerobic) and raw (aerobic) sewage sludge and found that concentrations of 4-nonylphenol increased four to eight times in the stabilized versus two times in the raw sludge, a finding which supported their theory.

A reconnaissance of 95 organic wastewater contaminants in 139 U.S. streams conducted in 1999-2000 revealed that nonylphenol was one of the most commonly occurring contaminants and was measured at higher concentrations than most of the other contaminants (Kolpin et al. 2002). Selection of streams sampled was biased toward streams susceptible to contamination. A number of studies on the persistence of nonylphenol in sewage treatment plant effluents and the environment have been conducted and are reviewed by Maguire (1999). Gaffney (1976) determined that 1 mg/L nonylphenol did not degrade during 135-hr incubation with domestic wastewater. In industrial wastewater, nonylphenol concentration was unchanged after 24 hr incubation, but decreased by 45 percent after 135 hr. Staples et al. (2001) determined that nonylphenol at 13 mg/L and 22°C was mineralized to CO_2 within 35 days in aerobic systems inoculated with sludge from a waste treatment plant. No intermediate compounds were formed and the calculated half-life for nonylphenol was 8.2 days.

Sundaram and Szeto (1981) studied nonylphenol fate incubated in open and closed containers of stream and pond waters. They found no degradation of nonylphenol incubated in open containers of the pond or stream waters. The observed half-life of 2.5 days, was attributed to volatilization. Incubation of nonylphenol in pond or stream waters in closed containers resulted in formation of a breakdown product. The observed half-life of nonylphenol in pond and stream water were estimated at 16.5 and 16.3 days, respectively. The same authors incubated nonylphenol in pond water with sediment present and found about 50 percent of the nonylphenol in the sediment after 10 days. About 80 percent of the nonylphenol in the sediment was degraded in 70 days. No degradation of nonylphenol occurred when autoclaved (sterilized) water and sediment samples were used. Staples et al. (1999) measured a half-life of 20 days for nonylphenol in water (31 mg/L) at 22°C. They suggested that the temperature of water and the initial concentration of the nonylphenol both affect the degradation rate of the chemical.

Ahel et al. (1994 a,b) studied the fate and transport of alkylphenol polyethoxylates (AP*n*EO)

and their degradates in the Glatt River system in Northern Switzerland from the Greifensee to the Rhine River. Water samples were collected at eight sites along the river hourly over several seasons. They found nonylphenol concentrations to be lower than other degradates and nonylphenol concentrations were most commonly detected in the 1 to 3 μg/L range. The concentration of AP*n*EO degradates varied with time of day reflecting fluctuations in wastewater treatment plant discharge. Concentrations of AP*n*EO degradates also varied seasonally, being found at higher concentrations in the winter due to lower water temperature. Nonylphenol had less seasonal variability than other AP*n*EO degradates. Nonylphenol was the predominant nonylphenolic compound found in sediments in this study. Sediment concentrations were 364 to 5,100 times those found in the river water.

Ahel and co-workers also reported that the abundance of particular AP*n*EO degradates is dependent on the conditions in the treatment plants studied along the Glatt River system (Ahel et al. 1994a; Ahel et al. 2000). Under aerobic conditions, the AP*n*EOs degrade through either the loss of ethylene oxide units to form low-molecular weight ethoxylates or through the formation of carboxylated ethoxylates ultimately terminating in CO_2 and water. Nonylphenol is formed during anaerobic breakdown of the AP*n*EOs and is therefore a minor component of wastewater treatment effluents because of aerobic conditions present during treatment. Another study by Ahel et al. (1996) demonstrated that nonylphenol can be reduced in ground water. The authors propose that biological processes are responsible provided that the ground water temperature does not become too cold for biological degradation. It has also been demonstrated (Ahel et al. 1994c) that nonylphenol can be degraded by photochemical processes. In bright summer sun, nonylphenol near the water surface has a half-life of 10-15 hr.

Heinis et al. (1999) studied the distribution and persistence of nonylphenol in natural pond systems in the temperate climate zone. They reported that nonylphenol partitioned to the pond enclosure wall material, macrophytes, and sediments within two days. After 440 days, the primary sink for nonylphenol was the sediment. Dissipation from the sediment was estimated to be 50 and 95 percent at 66 and 401 days, respectively. Hale et al. (2000) measured nonylphenol concentrations in sediments below wastewater outfalls and found one site that had a sediment concentration of 54,400 μg/kg more than twenty years after the treatment plant ceased operation. Bennett and Metcalfe (1998; 2000) found that nonylphenol was widely distributed in lower

Great Lakes sediments and reached 37,000 μg/kg in sediments near sewage treatment plants.

It appears that degradation of nonylphenol in sea water and saltwater sediments may be slower than in fresh water and freshwater sediments. Ekelund et al. (1993) found that nonylphenol degradation rate was initially slow in sea water, but increased after microorganism adaptation occurred. Approximately 50 percent of the nonylphenol was degraded after 58 days. In marine sediments, the initial rate of degradation faster than in sea water, but after 58 days about the same percentage of nonylphenol was degraded. Ethoxylated nonylphenol has a half-life of 60 days in marine sediments, similar to that of nonylphenol (Shang et al. 1999). Ferguson and Brownawell (2003) conducted degradation studies with AP*n*EOs in marine sediments and found that degradation occurred in oxic and anoxic conditions. They reported no clear evidence for net formation of nonylphenol from AP*n*EOs under anaerobic conditions during the 120 day study, but they speculated that the time scale of their study may not have been long enough to make the observation.

1.3. Metabolism and Bioconcentration

Nonylphenol is metabolized by hepatic cytochrome P450 enzymes in the rainbow trout (*Oncorhynchus mykiss*) and bile from the fish contained the glucuronic acid conjugates of nonylphenol (Meldahl et al. 1996; Thibaut et al. 1999). Arukwe et al. (2000) found that bile was the major route of nonylphenol excretion with a half-life of 24 to 48 hrs following either waterborne or dietary exposures.

The log K_{ow} of nonylphenol ranges from 3.80 to 4.77, indicating that moderate bioaccumulation in aquatic organisms may be expected. However, reported laboratory bioconcentration factors (BCFs) and field-derived bioaccumulation factors (BAFs) do not support the expected accumulations in tissues, indicating that some nonylphenol is metabolized. Bioconcentration was measured in two saltwater organisms, the blue mussel (*Mytilus edulis*) and Atlantic salmon (*Salmo salar*) by McLeese et al. (1980a). The estimated BCF for the blue mussel ranged from 1.4 to 7.9 and the estimated BCF for Atlantic salmon was 75 (McLeese et al. 1981). Hecht et al. (2004) reported nonylphenol BCFs for the three marine amphipod species, *Eohaustorius estuarium, Grandidierella japonica* and *Corophidum salmonis,* of 154, 185, and 46 to 133, respectively. Ahel et al. (1993) measured the bioconcentration of nonylphenol for

several species in rivers in Switzerland. They determined a BCF for algae of 487 (converted to a wet weight basis assuming 95 percent water in algae). Nonylphenol did not biomagnify in the food chain in the system studied; rather BCFs in fish and ducks were lower than in the algae. Keith et al. (2001) measured nonylphenol in fish tissues of seven species from the Kalamazoo River and in water at the river's confluence with Lake Michigan. They found 41 percent of the tissue samples had measurable concentrations of nonylphenol with a range of 3.3 to 29.1 μg/kg and a mean value of 12.0 μg/kg. A followup study was conducted in the same river (Kannan et al. 2003) to further examine the occurrence of nonylphenol and nonylphenol ethoxylates in fish, water and sediments and their association with two wastewater treatment plants. Ten fish from near each treatment plant were analyzed for nonylphenol and ethoxylated nonylphenol. Only one fish contained a measurable concentration of nonylphenol (3.4 μg/kg). Neither nonylphenol or its ethoxylates were detected in the sediments collected upstream of the treatment plants. However, five of twenty-four (21 %) sediment samples collected from below the treatment plants contained nonylphenol (no ethoxylates were found) at concentrations that ranged from 2 - 15.3 μg/kg dry weight. Downstream of one treatment plant, neither nonylphenol nor nonylphenol ethoxylates were measured above the method detection limit. Nonylphenol concentrations extracted from sediments in the Venice, Italy lagoon were higher in areas with large masses of decomposing macroalgae (primarily *Ulva rigida*) than in areas not associated with the decomposition (Marcomini et al. 1990). This may suggest that nonylphenol bioaccumulated by the macroalgae was transferred to the sediment as the algae died and decomposed.

1.4. Estrogenicity of Nonylphenol

There are several review articles that describe the estrogenicity of nonylphenol (Servos 1999; Sonnenschein and Soto 1998; Sumpter 1998). The majority of studies using aquatic species models report results for molecular or biochemical endpoints such as induction of the egg protein, vitellogenin, or are in vitro studies such as receptor binding assays. These types of studies and endpoints do not meet the data acceptability requirements outlined in EPA's *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (Stephan et al. 1985) and hence were not used deriving

ambient water quality criteria. However, studies identified in the literature search describing effects of nonylphenol on molecular and biochemical endpoints and activity in in vitro bioassays are discussed in Section 6 of this document.

Whole organism endpoints such as reproductive and growth effects are used to derive aquatic life ambient water quality criteria for nonylphenol. To the extent that such endpoints reflect the integration of molecular, biochemical and tissue-level effects at the whole organism level, the nonylphenol criteria address the estrogenicity of nonylphenol. For example, while vitellogenin is a commonly used biomarker indicative of exposure to estrogenic compounds, measurement of this molecular/biochemical endpoint alone does not necessarily indicate adverse effect on population relevant endpoints such as survival, growth and reproduction. However, several studies have demonstrated that vitellogenin induction can be accompanied by decreased fecundity (egg production) of breeding pairs of fathead minnows exposed chronically to estrogenic compounds (Ankley et al.). The chronic toxicity studies used in deriving the nonylphenol criteria (Table 6) included assessment of effects on growth and reproduction endpoints in aquatic organisms. Hence, to the extent that these endpoints are the result of effects on the endocrine system (although this was not definitively demonstrated in any of the tests by use of a concommittant measure of a estrogen-receptor specific endpoint), the estrogenic effects of nonylphenol have been considered in deriving the aquatic life ambient water quality criteria for nonylphenol.

EPA has activities underway to develop scientific methods for considering endocrine effects, such as the estrogenicity of nonylphenol, in Agency risk assessments. Under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act (FQPA), EPA is required to develop a screening program to determine whether certain substances (including all pesticide active and other ingredients) "may have an effect in humans that is similar to an effect produced by a naturally-occurring estrogen, or other such endocrine effects as the Administrator may designate". Following the recommendations of its Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC), EPA determined that there was scientific basis for including, as part of the program, the androgen and thyroid hormone systems, in addition to the estrogen hormone system. EPA also adopted EDSTAC's recommendation that the Program include evaluations of potential effects in aquatic life and wildlife. When the

appropriate screening and or testing protocols being considered under the Agency's Endocrine Disruptor Screening Program have been developed, nonylphenol may be subjected to additional screening and or testing to better characterize effects related to endocrine systems.

1.5. Derivation of Aquatic Life Ambient Water Quality Criteria

A comprehension of the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" (Stephan et al. 1985), hereafter referred to as the Guidelines, is necessary to fully understand the text, tables, and calculations presented in this criteria document. Results of intermediate calculations are presented to four significant figures to prevent round-off error in subsequent calculations, not to reflect the precision of the value. Final criteria values are presented to two significant figures.

Nonylphenol has been studied for its acute and chronic toxicity to aquatic organisms and results of many studies are summarized in a review article by Staples et al. (1998). This review article also addresses the ability of nonylphenol to bioaccumulate in aquatic organisms. Much of the data reported in the review article has been used in this document, as well as some newer data, to derive the aquatic life ambient water quality criteria. The latest comprehensive literature search for information used in developing this document was conducted in November 1999. Subsequently, forty-three newer studies have subsequently been identified and included. Data and analysis included in the U.S. EPA's Office of Pollution Prevention and Toxics nonylphenol risk assessment have also been evaluated in deriving the aquatic life criteria for nonylphenol. Freshwater criteria were derived using nonylphenol of CAS numbers 25154-52-3 and 84852-15-3; saltwater criteria were derived using only nonlyphenol of CAS number 84852-15-3.

Whenever adequately justified, a national criterion may be replaced by a site-specific criterion (U.S. EPA 1983), which may include not only site-specific criterion concentrations (U.S. EPA 1994), but also site-specific averaging periods and frequencies of allowed excursions (U.S. EPA 1991).

2. ACUTE TOXICITY TO AQUATIC ANIMALS

2.1. Freshwater

The acute toxicity of nonylphenol to freshwater animals has been determined for 18 species and 2 subspecies representing 15 genera (Table 1). Species Mean Acute Values (SMAV) ranged from 55.72 µg/L for an amphipod (*Hyalella azteca*) to 774 µg/L for a snail (*Physella virgata*).

The most sensitive freshwater species tested was the amphipod, *Hyalella azteca* (Tables 1 and 3). Brooke (1993a) and England and Bussard (1995) tested this species under similar conditions, except for water hardness levels which were 51.5 and 148-154 mg/L as $CaCO_3$, respectively. An LC50 of 20.7 µg/L was calculated in the lower hardness water and 150 µg/L in the higher hardness water. Insufficient data exist to demonstrate an effect of water hardness on the toxicity of nonylphenol; therefore, the results are given equal weight for determining the SMAV. Data for one cladoceran species (*Daphnia magna*) are available. Brooke (1993a) reported an EC50 of 104 µg/L from a test that had the solutions renewed daily and Comber et al. (1993) reported an EC50 of 190 µg/L in a static test. The *Daphnia magna* SMAV is 140.6 µg/L.

The least sensitive freshwater species to nonylphenol toxicity were also invertebrates (Tables 1 and 3). The annelid worm (*Lumbriculus variegatus*) had an LC50 of 342 µg/L, nymphs of the dragonfly *Ophiogomphus* sp. had an LC50 of 596 µg/L and the least sensitive species tested was a snail, *Physella virgata*, which had an LC50 of 774 µ/L (Brooke 1993a). The lower sensitivity to nonylphenol occurs even though this species of snail does not have an operculum and would not be able to completely enclose its body and thus protect itself against nonylphenol exposure. The midge, *Chironomus tentans*, had an LC50 of 160 µg/L (England and Bussard 1995), indicating intermediate sensitivity among invertebrate species tested (Figure 1).

The only amphibian toxicity test available was for the boreal toad, *Bufo boreas*. The toad tadpoles had a 96-hr LC50 of 120 µg/L and were ranked second in sensitivity to nonylphenol (Dwyer et al. 1999a).

Freshwater fish species were in the mid-range of sensitivity to nonylphenol (Figure 1). SMAVs ranged from 110 µg/L for the fountain darter (*Etheostoma rubrum*) to 289.3 µg/L for the bonytail chub (*Gila elegans*). Three trout species of the genus *Oncorhynchus* (rainbow trout, apache trout, and cutthroat trout) and two subspecies of the species *Oncorhynchus clarki* were

tested and had similar LC50s ranging from 140 to 270 µg/L (Dwyer et al. 1995; Brooke 1993a). Dwyer et al. (1995, 1999a) exposed nine species of fish that were classified as threatened/endangered or were surrogates of threatened or endangered fish species. Acute toxicity test results were based on static tests with unmeasured nonylphenol concentrations and the LC50s ranged from 110 µg/L for the fountain darter, *Etheostoma rubrum*, to a geometric mean of 289.3 µg/L calculated from two tests with the bonytail chub. In addition to the test conducted by Dwyer et al. (1995), two additional tests were available for the fathead minnows (*Pimephales promelas*). LC50s for this species ranged from 128 µg/L (Brooke 1993a) to 360 µg/L (Dwyer et al. 1995). The tests conducted using flow-through exposure conditions (Holcombe et al. 1984; Brooke 1993a), which is preferable to static exposure conditions (Stephan et al. 1985) were used in calculating the SMAV (158.9 µg/L). One test was available for the bluegill (*Lepomis macrochirus*) and the LC50 was 209 µg/L (Brooke 1993a).

Freshwater Species Mean Acute Values (SMAV) and Genus Mean Acute Values (GMAV) were derived from available acute values (Tables 1 and 3, respectively). GMAVs were available for 15 genera; the most sensitive was the amphipod, *H. azteca*, which was 13.9 times more sensitive than the least sensitive species, a snail *P. virgata* (Figure 1). The four most sensitive species were within a factor of 2.5 of one another. Based on available data for freshwater organisms summarized in Table 1 and the GMAVs presented in Table 3, the freshwater Final Acute Value (FAV) for nonylphenol is 55.69 µg/L (calculated using the procedure described in the "Guidelines"). This FAV is essentially the same as the lowest freshwater SMAV of 55.72 µg/L for the amphipod *H. azteca*.

2.2. Saltwater

The acute toxicity of nonylphenol to saltwater animals has been determined for 8 invertebrate and 3 fish species (Table 1). SMAVs ranged from 17 µg/L for the winter flounder, *Pleuronectes americanus,* to 209.8 µg/L for the sheepshead minnow, *Cyprinodon variegatus* (Lussier et al. 2000; Ward and Boeri 1990b), a difference of 12.3-fold. Fish (winter flounder), bivalves (coot clam, *Mulinia lateralis*) and crustaceans (the mysid, *Americamysis bahia*) were the most sensitive species.

Data for nine of the thirteen saltwater test values reported in Table 1 were from a single

multi-species test (Lussier et al. 2000). Nonylphenol concentrations were measured in seven of the nine tests (Table 1), with measurements made at test initiation and at the end of the test (48 or 96 hr). Test organisms were fed brine shrimp, *Artemia* sp., during chemical exposure because the tests were designed to extend beyond the usual 48- or 96-hr acute test interval to 168 hr. The extended exposure time required feeding to ensure survival of animals not affected by nonylphenol. Normally, data gathered from tests in which organisms are fed are not acceptable for use in deriving Final Acute Values. However, the brine shrimp fed during the tests were "reference grade" and not likely to change the exposure to nonylphenol. Further, additional tests conducted in a different laboratory are available for two of the saltwater species such that toxicity results obtained when the testing is conducted with and without food added can be compared. In a 96-hr test with the mysid, the estimated LC50 was somewhat higher when the organisms were fed (60.6 µg/L; Lussier et al. 2000) compared to when they were not fed (43 µg/L; Ward and Boeri 1990a) during the study. In contrast, in a 96-hr test with the sheepshead minnow, the LC50 determined when the organisms were fed (142 µg/L; Lussier et al. 2000) was lower than when the organisms were not fed (310 µg/L; Ward and Boeri 1990a) during the study. These data indicate that feeding during the tests did not consistently increase or decrease the LC50 estimates, and therefore feeding is assumed not to have altered the results in these tests. Hence, the data from the Lussier et al. (2000) tests were used in deriving a saltwater Final Acute Value.

Acute toxicity test data were available for a number of other saltwater species. Invertebrates tested include: coot clam, *Mulinia lateralis* (LC50 = 37.9 µg/L; Lussier et al. 2000), the copepod, *Acartia tonsa* (LC50 = 190 µg/L; Kusk and Wollenberger 1999), American lobster, *Homarus americanus* (LC50 = 71 µg/L; Lussier et al. 2000), mud crab, *Dyspanopeus sayii* (LC50 >195 µg/L; Lussier et al. 2000) and the amphipods, *Leptocheirus plumulosus* (LC50 = 61.6; Lussier et al. 2000) and *Eohaustorius estuarius* (LC50 = 138 µg/L; Hecht and Boese 2002). The test with the amphipod *E. estuarius* (Hecht and Boese 2002) was conducted as a 96-hr test with a mean LC50 for toxicity measured at 227 µg/L as the average of three tests (299, 194, 189 µg/L). The ability of the surviving organisms to bury themselves in sediment at 96 hr when placed on sediment was combined with the number of survivors to calculate an EC50. The mean EC50 for the three tests was 138 µg/L. The sensitivity of the saltwater fish inland silversides (*Menidia*

beryllina), was intermediate (LC50 = 70 µg/L) among the three saltwater fish species tested.

Saltwater Species Mean Acute Values (SMAV) and Genus Mean Acute Values (GMAV) were derived from available acute values (Tables 1 and 3, respectively). GMAVs were available for 11 genera; the most sensitive was the winter flounder, *Pleuronectes americanus*, which was 12.3 times more sensitive than the least sensitive species, the sheepshead minnow, *Cyprinodon variegates* (Table 1 and 3). GMAVs for the four most sensitive saltwater species differ by a factor of only 3.5 (Table 3 and Figure 2). Based on available data for freshwater organisms summarized in Table 1 and the GMAVs presented in Table 3, the freshwater Final Acute Value (FAV) for nonylphenol is 13.93 µg/L (calculated using the procedure described in the "Guidelines"). This FAV is lower than the lowest SMAV of 17 µg/L for the the winter flounder, *Pleuronectes americanus*.

3. CHRONIC TOXICITY TO AQUATIC ANIMALS

3.1. Freshwater

The chronic toxicity of nonylphenol was determined for 5 freshwater species, two fish and 3 invertebrates (Table 2). Concentrations of nonylphenol were measured in all the tests. England (1995) exposed neonates of a cladoceran, *Ceriodaphnia dubia*, to nonylphenol for seven days in a renewal test. The results showed a significant reproductive impairment at 202 µg/L, but not at 88.7 µg/L, and survival was reduced at 377 µg/L, but not at 202 µg/L. Based upon reproductive impairment, the Chronic Value for *C. dubia* was 133.9 µ/L. At the end of 48 hr in the same test, effects were observed and an EC50 of 69 µg/L was calculated. However, the animals had received food and according to the Guidelines acute tests with this species must not receive food during an acute toxicity test if the test is to be used to compute an Acute-Chronic Ratio (ACR).

Fliedner (1993) exposed 4 to 24 hr-old *Daphnia magna* neonates to nonylphenol for 22 days in a 20°C life-cycle test. Test solutions were renewed three times each week during which a 52.2 to 65.5 % decrease in nonylphenol concentration was measured. Mean measured nonylphenol test concentrations were: 0, 0, 1.55, 1.34, 3.45, 10.70, and 47.81 µg/L. No effects were observed during the study on mortality, the number of offspring per female, or the mean day of the first brood at any of the test concentratoins. A significant effect was observed on the total number of young per concentration on day nine of the study. Based on the No Observed Effect Concentration (NOEC) of 10.7 µg/L and the Lowest Observed Effect Concentration (LOEC) of 47.8 µ/L reported, the chronic value (geometric mean of the NOEC and LOEC) for *D. magna* in this test is 22.62 µ/L. An acute test with this species conducted by the same authors was not available to calculate an ACR.

Brooke (1993a) conducted a 21-day chronic exposure for the cladoceran *Daphnia magna*. Test solutions were renewed three times per week and concentrations of nonylphenol declined, on average, 57.4 ± 5.8 % between solution renewals. The author concluded that *D. magna* growth and reproduction were significantly affected at 215 µg/L, but not at 116 µg/L. Survival was reduced to 60 percent at 215 µg/L; however, this survival rate was not a significant reduction from the control survival rate because only 80 percent of organisms survived in the control group. Based on reproductive impairment, the chronic value, calculated as the geometric

mean of the lower (116 µg/L) and upper (215 µg/L) chronic limits, for this test was 157.9 µg/L. Dividing the acute value (104 µg/L), determined from a companion test for this species (Brooke 1993a; Table 1) by the chronic value (157.9 µg/L; Table 2) results in an ACR 0.6586 for *D. magna* (Table 2).

A third life-cycle test (21-day exposure) with *D. magna* was conducted by Comber et al. (1993). They found no significant effects in survival, reproduction or growth at concentrations ≤24 µg/L. The number of live young produced was significantly reduced at concentrations ≥ 39 µg/L when compared to controls. Growth was reduced at concentrations ≥ 71 µg/L and survival of adults was reduced at concentrations ≥ 130 µ/L. Based on reproductive impairment, the chronic value, calculated as the geometric mean of the lower (24 µg/L) and upper (39 µg/L) chronic limits, for this test was 30.59 µg/L. Dividing the acute value (190 µg/L), determined in in a companion test for this species (Table 1) by the chronic value (30.59 µg/L; Table 2) results in an ACR of 6.211 for *D. magna*. Calculating the geometric mean of the two ACRs for *D. magna* (0.6586 and 6.211) results in a species mean acute-chronic ration (SMACR) of 2.023 for *D. magna*.

The midge, *Chironomus tentans*, was exposed in a continuous-flow diluter to nonylphenol from ≤24-hr old larva through emergence (53 days) as adults (Kahl et al. 1997). Nominal exposure concentrations ranged from 12.5 to 200 µg/L, but mean measured concentrations were lower. Neither growth nor reproductive endpoints (sex ratio, emergence pattern, and egg production and viability) were negatively affected at any of the exposure concentrations. There was a significant effect on survival of larvae during the first 20 days of exposure, but no effect after 20 days. Based on survival at 20 days, the NOEC and LOEC for this study were 42 and 91 µg/L, respectively. The chronic value, calculated as the geometric mean of the NOEC and the LOEC, is 61.82 µg/L for this test. A companion acute toxicity test was not conducted; therefore, an ACR can not be calculated for this species.

A 91-day early life-stage test was conducted with embryos and fry of the rainbow trout, *Oncorhynchus mykiss* (Brooke 1993a). Five nonylphenol exposure concentrations were tested, ranging from 6.0 to 114 µg/L in the flow-through test. Time to hatch and percent survival at hatch were not affected by the nonylphenol concentrations tested; however, nearly all of the larvae were abnormal at the two highest exposure concentrations (≥ 53.0 µg/L). At the end of

the test, survival was significantly reduced at concentrations ≥ 23.1 µg/L but not at 10.3 µg/L. Growth (both weight and length) was a more sensitive chronic endpoint than survival. At the end of the test, the fish were significantly shorter (14 %) and weighed less (30 %, dry weight) than control fish at nonylphenol concentrations ≥ 10.3 µg/L, but not at 6.0 µg/L. Based on growth, the NOEC and LOEC determined in this study were 6.0 and ≥ 10.3 µg/L, respectively. The chronic value, calculated as the geometric mean of the NOEC and the LOEC, is 7.861 µg/L for rainbow trout. Dividing the acute value (221 µg/L), determined in in a companion test for this species (Table 1) by the chronic value (7.861 µg/L; Table 2) results in an ACR of 28.11 for rainbow trout.

An early-life-stage toxicity test was available for the fathead minnow, *Pimephales promelas* (Ward and Boeri 1991c). Embryos and larvae were exposed under continuous-flow conditions for a total of 33 days to five concentrations of nonylphenol that ranged from 2.8 to 23 µg/L. Embryos in the control group and those in the three lowest nonylphenol exposure concentrations (2.8, 4.5, and 7.4 µg/L) began to hatch on the third day of exposure, while the two higher concentrations (14 and 23 µg/L) began hatching on the fourth day. Growth (length or weight) of nonylphenol exposed fish was not significantly different from the control organisms at any of the nonylphenol treatment concentrations. Survival of the fish at the end of the test was significantly reduced at nonylphenol concentrations ≥ 14 µg/L. Fish survival averaged 56.7 % at 23 µg/L nonylphenol, 66.7 % at 14 µg/L nonylphenol, and 76.7 % at 7.4 µg/L nonylphenol. At concentrations ≤ 7.4 µg/L survival of nonylphenol exposed fish did not differ from the control fish survival, which averaged 86.7 %. Based on survival, the NOEC and LOEC determined in this study were 7.4 µg/L and 14 µg/L, respectively. The chronic value, calculated as the geometric mean of the NOEC and the LOEC, is 10.18 µg/L for fathead minnow (Table 2). A companion acute toxicity test was not conducted; therefore, an ACR can not be calculated for this species.

3.2. Saltwater

Two chronic toxicity tests with phenol have been conducted with the same saltwater animal species. A 28-day chronic toxicity test was conducted with mysids, *Americamysis bahia* (Ward

and Boeri 1991b). There was no effect on survival or reproduction at 6.7 μg/L, but there was a 18 % reduction in survival and a 53% reduction in reproduction at 9.1 μg/L. Effects on survival at the highest concentration tested (21 μg/L) were observed before the end of the third week of the test. Test organisms of each sex were measured separately for length and weight. The data show no obvious difference between the length of male and female mysids for all of the concentrations tested, therefore growth analysis was based on combined length data for both sexes. Growth (length) was the most sensitive endpoint for mysids. There was a 7% (statistically significant relative to control animals) reduction in the length of mysids exposed to 6.7 μg/L nonylphenol, but no difference in growth for mysids exposed to 3.9 μg/L nonylphenol. Based on growth, the NOEC and LOEC determined in this study were 3.9 μg/L and 6.7 μg/L, respectively. The chronic value, calculated as the geometric mean of the NOEC and the LOEC, is 5.112 μg/L for mysids (Table 2). Dividing the acute value (43 μg/L), determined in in a companion test for this species (Table 1) by the chronic value (5.112 μg/L; Table 2) results in an ACR of 8.412 for the mysid, *Americamysis bahia*.

A second 28-day life cycle test with mysids, *Americamysis bahia* (Kuhn et al. 2001) was conducted using the ASTM standardized life-cycle test methods. Time to first brood release appeared dose dependent, but was not statistically significant. Growth of the female mysid was dose dependent and was significantly affected at concentrations $\geq$ 27.56 μg/L. The most sensitive endpoint for this test was a reproduction. The average number of young per available female reproductive days was significantly reduced at test concentration $\geq$ 15.28 μg/L, but was not affected at 9.46 μg/L. Based on reproduction, the NOEC and LOEC determined in this study were 9.46 μg/L and 15.28 μg/L, respectively. The chronic value, calculated as the geometric mean of the NOEC and the LOEC, is 12.02 μg/L. A companion acute toxicity test was not conducted; therefore, an ACR can not be calculated from this test.

3.3. Acute-Chronic Ratios

Three nonylphenol ACRs, determined from the fourth (*Daphnia magna*) and eighth (rainbow trout) most sensitive freshwater species tested and the third (mysid) most sensitive saltwater species tested, are available (Table 3). Two ACRs (0.6586 and 6.211; Table 2) were available

for the cladoceran *Daphnia magna*, which differed by a factor of approximately 9.4-fold. The species mean ACR, calculated as the geometric mean of the two values, is 2.023. Acute-chronic ratios for the acutely sensitive mysid, *A. bahia*, was 8.412 and the moderately acutely sensitive rainbow trout, *Oncorhynchus mykiss*, was 28.11. An ACR of 0.515 would be calculated from the tests of England (1995) with the cladoceran, *Ceriodaphnia dubia*. However, the organisms were fed during the acute test and data demonstrating that feeding did not significantly affect the acute value were not available. According to the Guidelines, acute tests with this species must be done without food present in the test solutions. Therefore, the *C. dubia* ACR was not used.

The three valid species mean ACRs (2.023, 8.412 and 28.11) differed by 13.9-fold (Table 3). The Guidelines stipulate that if the species mean acute-chronic ratio seems to increase or decrease as the SMAV increases, the Final Acute-Chronic Ratio (FACR) should be based on the acute-chronic ratios for species whose SMAVs are close to the Final Acute Value (FAV). Examination of the SMACRs (Table 3) relative to the SMAVs indicates that the more acutely sensitive species (*A. bahia* and *D. magna*) have 3 to 14-fold lower SMACRs than for the less acutely sensitive rainbow trout, indicating a general trend of increasing SMACR with increasing SMAV. Therefore, the FACR should be based on the SMACR for species whose SMAVs are close to the FAV. The mysid SMAV (51.05 μg/L) is closest to both the freshwater FAV (55.49 μg/L) and the saltwater FAV (13.93 μg/L). Therefore, the SMACR for the mysid is used as the FACR and is 8.412.

4. TOXICITY TO AQUATIC PLANTS

4.1. Freshwater

Acceptable data on the toxicity of nonylphenol to freshwater plants were available for one species of algae (nonvascular plant) and no acceptable toxicity data are available for vascular plants (Table 4). Ward and Boeri (1990a) exposed the green alga, *Selenastrum capricornutum*, to nonylphenol for four days. They calculated an EC50 of 410 μg/L based on cell counts. At the end of the toxicity test, algae from the highest exposure concentration (720 μg/L) were transferred to fresh media solution. During the next seven days, cell counts increased exponentially, indicating that nonylphenol treatment at this concentration for four days did not have a persistent algistatic effect.

4.2. Saltwater

Acceptable data on the toxicity of nonylphenol to saltwater plants were available for one species of marine algae and no acceptable toxicity data are available for vascular plants (Table 4). The EC50 value for vegetative growth of the planktonic diatom, *Skeletonema costatum*, was 27 μg/L (Ward and Boeri 1990d). Although this value was lower than nearly all of the acute values for animals, it is for vegetative growth, which can recover rapidly. *Skeletonema* transferred from the highest nominal concentration of nonylphenol with survivors (120 μg/L) into control medium grew to a 76-fold increase in cells/mL within 48 hr (Ward and Boeri 1990d), demonstrating that nonylphenol treatment at this concentration for four days did not have a persistent algistatic effect.

Based on the vegetative growth test using the saltwater planktonic diatom, *Skeletonema costatum*, the Final Plant Value for nonylphenol is 27 μg/L. This plant species is more sensitive to nonylphenol than any tested species of freshwater animal and more sensitive than all but one tested saltwater animal species.

5. BIOACCUMULATION

5.1. Freshwater

Acceptable data on the bioconcentration of nonylphenol in two species of freshwater animals were available (Table 5). Ward and Boeri (1991a) measured the whole body concentrations of nonylphenol in juvenile fathead minnows at two exposure concentrations after 27 days of exposure. The bioconcentration factors (BCFs) were 271 and 344 (not lipid normalized) following exposure to 4.9 and 22.7 µg/L nonylphenol, respectively. Brooke (1993b) exposed juvenile fathead minnow (*Pimephales promelas*) and juvenile bluegill (*Lepomis macrochirus*) to nonylphenol at five concentrations for four and twenty-eight days. Lipid concentrations were measured (Brooke 1994) for the test fish and the bioconcentration results were lipid normalized which reduced the bioconcentration factors from 4.7 to 4.9 times. Nonylphenol concentrations that proved lethal to the organisms were not used to calculate bioconcentration factors. The short-term (4-day) tests showed that tissue concentrations reached steady-state within two days in both the fathead minnow and the bluegill. Therefore, there was good agreement between the results obtained in the 4-day and 28-day tests. Lipid-normalized BCFs for the fathead minnow ranged from 128.3 to 209.4 (Table 5) and for the bluegill ranged from 38.98 to 56.94. Giesy et al. (2000) measured the nonylphenol concentrations in whole bodies of the fathead minnow following a 42-day exposure. Exposure to sublethal concentrations of nonylphenol ranging from 0.4 to 3.4 µg/L resulted in BCFs ranging from 203 to 268

5.2. Saltwater

Bioconcentration factors are available for three species of saltwater animals, *Mytilus edulis*, *Crangon crangon* and *Gasterosteus aculeatus* (Ekelund et al. 1990;Table 5). *Crangon crangon* is a non-resident species, but the data are included because so little bioaccumulation data are available. Organisms were exposed to ^{14}C-labeled nonylphenol (CAS number not provided) for 16 days followed by an elimination period of 32 days. Lipid-normalized BCFs based on wet weight ranged from 78.75 for *C. crangon* to 2,168 for *M. edulis*. The BCF for *M. edulis* was an estimate, because steady-state tissue concentration was not reached during 16 days of exposure.

6. OTHER DATA

6.1. Freshwater

Additional data on the lethal and sublethal effects of nonylphenol on freshwater species that do not meet the data requirements described in the Guidelines (Stephan et al. 1985) for use in deriving aquatic life ambient water quality criteria are summarized in Table 6.

Three plant species (*Chlamydomonas reinhardtii, Salvinia molesta* and *Lemna minor)* were exposed in studies using media solutions that were not described. The effect levels determined for *Salvinia molesta* (2,500 µg/L) and *Chlamydomonas reinhardtii* (6,250 µg/L), indicates that these plant species are less sensitive to nonylphenol than animals (Prasad 1986; Weinberger and Greenhalgh 1984). Effect concentrations reported for the duckweed, *Lemna minor*, were highly variable, ranging from 125 to 5,500 µg/L (Weinberger and Iyengar 1983; Prasad 1986). Protozoa were affected in the concentration range from 50 to 747 µg/L (Preston et al. 2000, Schultz 1997, Yoshioka 1985).

Additional data on acute and chronic toxicity of a variety of invertebrates are summarized in Table 6. McLeese et al. (1980b) reported an LC50 of 5,000 µg/L for a clam, *Anodonta cataractae*, following a 144-hr exposure. The test organisms were fed in this test and the toxicity value is higher than those reported in Table 1 for similar species. In an acute test (96-hr) in which the cladoceran, *Daphnia magna* was fed, effect levels were reported as 136 and 302 µg/L for young and adult animals, respectively (Gerritsen et al. 1998). In a 48-hr test with the same species, EC50s ranged from 234 to 337 (Zang et al. 2003). Three 21-day tests with *Daphnia magna* (Baer and Owens 1999, Baldwin et al. 1997, LeBlanc et al. 2000), an additional *D. magna* test of 35-day duration in a high-hardness medium (Zang et al. 2003), and a 30-day test with *D. galeata mendotae* (Shurin and Dodson 1997) are included in this section because nonylphenol concentrations in the test water were not measured in these chronic tests. Negative effects on survival or reproduction were observed in all three tests with typical water hardness (i.e., between 25 and 200 µg/L). The results from these tests with *D. magna* (Table 6) agree reasonably well with those from tests with *D. magna* in which nonylphenol concentrations were measured (Table 2). Another cladoceran, *Daphnia pulex*, was exposed for 48 hr in tests in which nonylphenol concentrations decreased more than 50 percent during the exposures (Ernst et al.

1980). The LC50s determined in the test ranged from 140 to 190 µg/L, which agreed with LC50s for other cladoceran species. The cladoceran, *Ceriodaphnia dubia,* gave similar LC50 results of 276 and 225 µg/L following exposure to nonylphenol for 48 hr and 7 days, respectively (England 1995). The LC50 values reported in this table for the species are slightly higher than the chronic value for the species of 134 µg/L (Table 2). England and Bussard (1993) reported an EC50 and an LC50 for larva of the midge, *Chironomus tentans*, of 95 and 119 µg/L, respectively. These values, determined when the organisms were fed, are less than the than values reported in another study by the same authors in which organisms were not fed during the test (Table 1).

In a pair of tests in which the test organisms were fed, Brooke (1993b) measured a 96-hr LC50 for the fathead minnow, *Pimephales promelas*, of 138 µg/L and a 96-hr LC50 for the bluegill, *Lepomis macrochirus*, of 135 µg/L. The LC50 values for these species from tests in which the fish were fed, agree well with data from tests in which the fish were not fed (Table 1). McLeese et al. (1980b) reported an LC50 of 900 µg/L for the Atlantic salmon, *Salmo salar*, in a 96-hr exposure and Lech et al (1996) reported an LC50 of 193.65 for rainbow trout, *Oncorhyncus mykiss*, in a 72-hr exposure. Holmes and Kingsbury (1980) reported a 96-hr LC50 of 145 µg/L for brook trout juveniles (*Salvelinus fontinalis*), a 96-hr LC50 of 230 µg/L for rainbow trout juveniles (*Oncorhynchus mykiss*) and a 32-day LC50 of > 40 µg/L for lake trout juveniles (*Salvelinus naymaycush*). Fish were fed during these studies, but the resulting toxicity values are similar to comparable studies reported for salmonids in Table 1. Ernst et al (1980) reported 96-hr LC50s ranging from 560-920 µg/L for rainbow trout exposed to practical grade nonylphenol. A number of older studies were identified that report time to lethality (LT100) values for a number of freshwater species exposed to very high concentrations of nonylphenol (Applegate et al. 1957; MacPhee and Ruelle 1969; Wood 1952)

A long-term study was conducted with rainbow trout, *Onchorynchus mykiss*, exposing female fish immediately after hatch to 1, 10, and 30 or 50 µg/L of nonylphenol (Ashfield et al. 1998). They found reduced growth in fish exposed to 1 µg/L for 22 days and grown for 86 days beyond treatment. Growth was not reduced in the 10 µg/L treatment but was in the 50 µg/L treatment. In a second study in which exposure was for 35 days and grow-out was for 431 days beyond the last treatment day, reduced growth was observed at the 10 and 30 µg/L treatments on

day 55 of the study, but not at the 1 μg/L. At day 466, the fish exposed to 10 μg/L recovered the growth reductions seen earlier and only the 30 μg/L exposed fish showed reduced (approximately 25%) growth.

Five fish species (rainbow trout, Lahontan cutthroat trout, Apache trout, Colorado squawfish and fathead minnow) were exposed to nonylphenol for 96 hr to determine if nonylphenol inhibited brain acetylcholoinesterase enzymes. AChE inhibition was measured indirectly as a decrease in the number of muscarinic cholinergic receptors which is a compensatory response to acetycholine buildup (Jones et al. 1998). Responses at exposure concentrations $\leq$ 220 μg/L were observed in the rainbow trout, Lahontan cutthroat trout and Apache trout. The lack of a clear connection between this sublethal biochemical endpoint and population relevant effects precludes the use of these results as core data. An effect of nonylphenol on another sub-organismal endpoint, histology of epidermal mucous cells, was observed following intermittent exposure to technical grade nonylphenol (Burkhardt-Holm et al. 2000). Other histochemical or biochemical changes have been reported following exposure to nonylphenol including hemorrhage and lymphocyte infiltration in liver tissue of rainbow trout (Ugaz et al. 2003) and blood cell composition in carp (Schwaiger et al. 2000).

Brooke (1993b) measured the bioconcentration of nonylphenol in the fathead minnow and bluegill at concentrations near lethality. The fathead minnow BCF was 100.4 and the bluegill BCF was 35.31. The values were slightly less than the BCFs measured in the fish from lower exposure concentrations (Table 5). Blackburn et al. (1999) reported BCFs for adult male rainbow trout of 116 and 88 following 3 weeks exposure to 63 and 81 μg/L nonylphenol (purity unknown), respectively. Lewis and Lech (1996) found that bioconcentration of nonylphenol after short-term exposure (2-24 hr) was higher in rainbow trout viscera (BCF = 98.2) than in the remainder of the carcass (BCF = 24.21). They also measured the half-life of nonylphenol in various tissues and found that fat and muscle similarly depurated nonylphenol to half concentrations in about 19 hr. The liver depurated to half concentrations in about 6 hr.

Mesocosm studies were conducted with nonylphenol in which zooplankton, benthic macroinvertebrates, and fish were observed for effects. Zooplankton populations (O'Halloran et al. 1999) and benthic macroinvertebrate populations (Schmude et al. 1999) exposured to four concentrations of nonylphenol for 20 days showed no negative effects at the 23 μg/L

nonylphenol but were negatively affected at 76 μg/L nonylphenol. Various species of zooplankton and macroinvertebrates exhibited differences in sensitivity to nonylphenol. The authors of the zooplankton study stated that a MATC for the protection of all zooplankton taxa is approximately 10 μg/L. The fish (bluegill) in the mesocosms (Liber et al. 1999) were unaffected at nonylphenol exposures ≤ 76 μg/L, but survival was reduced at 243 μg/L. In one exposure replicate with a mean nonlyphenol concentration of 93 μg/L, survival of the fish was reduced after 20 days of exposure indicating that concentrations near 100 μg/L may be the toxicity threshold for this species. Hense et al. (2003) and Severin et al. (2003) conducted microcosm studies in Germany using 6-week exposures to nonylphenol. They found changes in phytoplankton species composition, but no change in biomass with nonylphenol concentrations up to 120 μg/L. The zooplankton in the study were not affected at concentrations ranging from 19 to 44 μg/L (mean of 30 μg/L), but species richness was affected at concentrations >30 μg/L. Effects observed in these mesocosm studies were all above the freshwater Final Chronic Value of 5.920 μg/L.

6.2. Saltwater

Additional data on the lethal and sublethal effects of nonylphenol on saltwater species that do not meet the data requirements described in the Guidelines (Stephan et al. 1985) for use in deriving aquatic life ambient water quality criteria are summarized in Table 6.

Results from a sexual reproduction test with red alga species, *Champia parvula*, indicated that reproduction was not inhibited at the highest measured concentration tested, 167 μg/L (Tagliabue 1993). Cypris larva of the barnacle, *Balanus amphitrite*, were exposed to nonylphenol for 48 hr and the settlement of the larva was reduced at 1.0 μg/L (Billinghurst et al. 1998). The soft-shell clam, *Mya arenaria*, showed no adverse effects on survival from a 360-hr exposure at 700 μg/L (McLeese et al. 1980b). Granmo et al. (1989) report LC50s of 3,000 μg/L and 500 μg/L at 96-hr and 360-hr, respectively, for the blue mussel, *Mytilus edulis*. Nonylphenol also reduced growth and byssus thread strength in the blue mussel at concentrations of ≥ 56 μg/L (Granmo et al. 1989) and caused effects on attachment activity at 22 μg/L (Etoh et al. 1997). Lussier et al. (2000) tested a number of saltwater invertebrates including coot clam, mysid, amphipod, grass shrimp, and American lobster and determined LC50s for various timepoints

(Table 6). Results from other studies with mysid (Ward and Boeri, 1990a) and American lobster (McLeese et al 1980b) are similar to those reported by Lussier et al (2000). The LC50 value (300 µg/L) reported by McLeese et al. (1980b) for the shrimp, *Crangon septemspinosa*, is higher than the grass shrimp values reported by Lussier et al (2000). Kusk and Wollenberger (1999) determined a 48-hr LC50 (280-360 µg/L for the copepod, *Acartia tonsa*, exposed to nonylphenol in a synthetic media. Nice et al (2000) reported developmental effects at 100 µg/L nonylphenol on the Pacific oyster (*Crassostrea gigas*) exposed for 72-hr. Nonylphenols have also been reported to have antifouling activity, but the test results are qualitative (Takasawa et al. 1990; Kitajima et al. 1995).

A fifty-five-day flow-through test with the mysid, *Americamysis bahia*, was conducted by Kuhn et al. (2001) to evaluate the efficacy of an age-classified projection matrix model for predicting population behavior. Organisms were exposed for more than three generations to nonylphenol. The measured mean concentrations of nonylphenol used for the 55-day exposure were 5.79, 7.56, 10.88, 15.75, 21.44, 33.19, and 106.00 µg/L. Thirty individuals were used in each replicate exposure chamber and the age distribution consisted of 15 (24-h newly hatched), 8 (8-d-old juveniles), 4 (17-d-old adults), 2 (23-d-old adults), and 1 (31-d-old adult) test organisms. Several generations were possible in this test (control organisms produced first brood in 14 days). It appears that the control populations grew in number of individuals for the first 28 to 36 days, then stabilized. Population growth was reduced from day 8 and beyond in all of the nonylphenol treated groups. The population exposed to 5.79 µg/L nonylphenol grew at the same rate as the control animals for the first 21 days, but then the rate fell below the control rate. The populations exposed to higher nonylphenol concentrations all decreased from day 8 and beyond. There appears to be a trend (not significant) in the shift in the sex ratios for the various treatments. At the end of the test, the sex ratios were one-third female in the control groups and half female in the 33.19 µg/L exposure group. The authors calculated a zero population growth value (λ) of 19 µg/L for the 55-day multigenerational test. The chronic values from the 28-day exposure in this study was 12.02 µg/L (Table 2) and from a similar 28-day study by Ward and Boeri (1991b) was 5.112 µg/L (Table 2), which are lower than the predicted value for "population protection" from the 55-day multigenerational test.

McLeese et al. (1980b) reported 96-hr test results for the Atlantic salmon, *Salmo salar*, that

were in general agreement with freshwater trout test results. In four tests, LC50 values ranged from 130 to 900 µg/L. Ward and Boeri (1990c) found similar toxicity results for sheepshead minnow, *Cyprinodon variegatus*, exposed in brackish water as those reported for salt water (Table 1). In brackish water, LC50s ranged from > 420 µg/L for a 24-hr exposure to 320 µg/L for a 72-hr exposure. Threespine stickleback, *Gasterosteus aculeatus*, exposed to a commercial mixture of nonylphenol had a 96-hr LC50 of 370 µg/L (Granmo et al. 1991a). Killifish (Kelly and Di Giulio 2000) were exposed as embryos and larva to nonylphenol for 96 hrs. Even though the solvent concentration used in the exposures exceeded the 0.5 mL/L recommended limit, the data are included in Table 6 because the results reported for the solvent controls do not show decreased hatching success or increased abnormalities at 10 days post-hatch. Embryos exposed to 2,204 µg/L for 96 hr were all abnormally developed at 10 days post-fertilization. The LC50 for the same exposure period was 5,444 µg/L. Killifish larva were similar in sensitivity to nonylphenol exposures at post hatch ages of 1, 14, and 28 days with LC50s of 214, 209, and 260, respectively.

Additional data on the effect of nonylphenol on saltwater species do not indicate greater sensitivities than the data summarized in Tables 1 and 2. Some of the data presented in Table 6 (e.g., sheepshead minnow, Inland silversides) were from the same acute tests listed in Table 1 (Lussier et al. 2000; Ward and Boeri 1990a,b), but for exposure durations other than 96 hr.

6.3. Reproductive, Devleopmental and Estrogenic Effects of Nonylphenol

There are several review articles that describe the estrogenic activity of nonylphenol (Servos 1999; Sonnenschein and Soto 1998; Sumpter 1998). The majority of studies describing the estrogenic activity of nonylphenol using aquatic species models exposed in vivo measure molecular, biochemical, or histological endpoints such as induction of the egg protein, vitellogenin, or occurrence of egg cells within testes (a condition known as intersex or ovo-testis). In addition, estrogenicity is commonly characterized using in vitro studies such as estrogen receptor binding assays. Molecular, biochemical and reproductive endpoints measured following in vivo exposures to nonylphenol and that are thought to result from estrogenic activity of nonylphenol are summarized in this section. In vitro studies are listed in Section 7 of this document.

The majority of reports of estrogenic effects in aquatic organism have been for fish, although some effects in invertebrates have also been reported. Bechmann (1999) found no effects in the marine copepod *Tisbe battagliai* exposed to nonylphenol at 55 μg/L, but estrogenic effects were reported to have occurred in the amphipod *Corophium volutator* (Brown et al. 1999) at 10 μg/L and in the larva of *Chironomus riparius* (Hahn et al. 2002) at 2,000 μg/L. The mechanism(s) by which estrogenic effects can be produced in invertebrates that do not possess estrogen receptors is unclear.

Vitellogenin is a protein produced in the liver of female oviparous vertebrate species and deposited in the ovaries as the primary material for yolk in the ova. Male fish normally produce very little vitellogenin. Islinger et al. (1999) estimated the estrogenic potential of nonylphenol to stimulate vitellogenin production in male rainbow trout at 2,000 to 3,000 times less potent than the natural estrogen, 17β-estradiol. Ren et al. (1996a) demonstrated significant increases in vitellogenin production in rainbow trout exposed to nonylphenol at 100 μg/L for 72 hr. In another study, Ren et al. (1996b) demonstrated that nonylphenol could stimulate the production of vitellogenin mRNA (which precedes vitellogenin protein synthesis) within 4 hr at 10 μg/L. Similarly, Lech et al. (1996) observed a significant increase in vitellogenin mRNA at 72 hr in rainbow trout at 14.14 μg/L nonylphenol. Vitellogenin was induced in green swordfish, *Xiphophorus helleri*, by exposure to 4 μg/L of technical grade nonylphenol (Kwak et al. 2001).

Jobling et al. (1996) demonstrated significant increases in vitellogenin in male rainbow trout,

Oncorhynchus mykiss, at three weeks of exposure to 20.3 and 54.3 μg/L of nonylphenol. Similar results were reported in another study with rainbow trout, plasma vitellogenin was increased after 21 days exposure to 50 μg/L of nonylphenol (Tremblay and Van Der Kraak 1998). Harris et al. (2001) also observed increased plasma vitellogenin levels in female rainbow trout exposed to 8.3 and 85.6 μg/L of nonylphenol. In the same study, nonylphenol also caused changes in several pituitary and plasma hormone levels. In contrast, vitellogenin induction was not observed in rainbow trout exposed for 9 days to 109 μg/L of nonylphenol (Pedersen et al. 1999) or in Atlantic salmon, *Salmo trutta*, exposed for 30 days to 20 μg/L (Moore et al. 2003). The influence of exposure route on nonylphenol-induced vitellogenin *m*RNA and plasma vitellogenin production in the male fathead minnow was studied by Pickford et al. (2003). Their results showed that exposure via water produced 10-fold higher vitellogenin induction than exposure via the dietary route.

In a study with the fathead minnow, Giesy et al. (2000) found that nonylphenol exposures to 0.5 to 3.4 μg/L nonylphenol were not acutely toxic to the adult fish and fecundity was variously affected over the reproductive season. When the cumulative reproduction was combined for the two experiments during different portions of the reproductive season, concentrations of > 0.3 to 0.4 μg/L did appear to reduce fecundity. However, fish exposed to 0.09 and 0.1 μg/L produced more eggs than control fish. These data appear to produce a U-shaped dose-response and indicate a possible hormetic response of fecundity to nonylphenol. Nonylphenol concentrations of 0.05 to 3.4 μg/L did not significantly change vitellogenin concentrations in the blood of males, and raised the 17β-estradiol titers in the blood of male and female fish at most treatment concentrations > 0.05 μg/L. An increase in the number of Sertoli cells may have occurred in the male fathead minnow exposed to nonylphenol at 1.6 μg/L for 42 days (Miles-Richardson et al. 1999). The evidence was not complete, but indicated the possibility of increased phagocytic action and Sertoli cell tissue in testes.

A non-resident fish species, Japanese medaka (*Oryzias latipes*), was exposed to nonylphenol for 28 days following hatch and survivors monitored for the following 55 days (Nimrod and Benson 1998). At the highest exposure concentration of 1.93 μg/L, survival, growth, egg production, egg viability, and gonadosomatic index (GSI) were not altered. In another study with the same species of fish, development of ovo-testis, an intersex condition, occurred after a

three month exposure to 50 μg/L of nonylphenol (Gray and Metcalf 1997). The sex ratio shifted in favor of females at the highest exposure concentration. Seki et al. (2003) found that in the same species of fish exposed to nonylphenol from fertilized egg to 60 days post-hatch, the lowest-observed-effect concentration for vitellogenin induction was 11.6 μg/L.

Yokota et al. (2001) conducted a two-generation flow-through study with the non-resident fish species medaka (*Oryzias latipes*). Concentrations of nonylphenol were measured during exposures that began with eggs and proceeded to 60-days post-hatch of the second (F_1) generation. Five exposure concentrations of nonylphenol in quadruplicate (4.2, 8.2, 17.7, 51.5, and 183 μg/L) and water-only and solvent controls were used. In the F_0 generation, egg hatchability was reduced (46.7%) by 183 μg/L nonylphenol exposure, survival was significantly decreased at 60 days post-hatch by nonylphenol exposures $\geq$ 17.7 μg/L, and no differences in growth (length or weight) were observed at 60 days post-hatch. Induction of ovo-testis was observed in the 17.7 μg/L treatment, with 20% of fish displaying male characteristics externally having ovo-testis tissues. In fish from the 51.5 μg/L treatment, 40% had ovo-testis and all of these fish exhibited female characteristics externally. Spermatogenesis was observed in the fish with ovo-testis exposed to 17.7 μg/L nonylphenol, but was not observed in the fish with ovo-testis exposed to 51.5 μg/L nonylphenol. Fecundity of paired fish during the reproductive phase (days 71 to 103 post-hatch) was not affected by nonylphenol treatments. GSI of male fish was reduced at 17.7 μg/L, but not significantly, and GSI of female fish was increased significantly by exposure to nonylphenol concentrations > 8.2 μg/L.

The effects of nonylphenol on F_1 fish from Yokota et al. (2001) were also reported. No embryological abnormalities or hatching failures of fertilized eggs were observed in any treatments. Growth was not affected at 60-days post-hatch by any of the nonylphenol exposure concentrationsl. The sex ratio, characterized by secondary sex characteristics, changed in treatments $\geq$ 17.7 μg/L to favor females 1:2. Induction of ovo-testis was observed at lower concentrations of nonylphenol in the F_1 generation than in the F_0 generation. Ovo-testis were observed in the 8.2 μg/L exposure group (10%) and in the 17.7 μg/L exposure group (25%). However, all fish with ovo-testis displayed external male characteristics and the degree of development of oocytes in each ovo-testis was not as severe as that in the F_0 generation in the 17.7 μg/L treatment. The overall results indicate a LOEC of 17.7 μg/L and a NOEC of 8.2 μg/L

with a chronic value, calculated as the geometric mean of the NOEC and LOEC, of 12.05 μg/L. The chronic value for this study is in good agreement with the Table 2 data for resident freshwater species, rainbow trout and fathead minnow.

A multi-generational exposure to nonylphenol has been conducted with rainbow trout by Schwaiger et al. (2002). Adult rainbow trout of both sexes were exposed intermittently to nonylphenol at 1 and 10 μg/L over a 4 month period. Mortality rate in the progeny was significantly reduced by parental exposure to both 1 and 10 μg/L nonylphenol and hatching in the progency was reduced by parental exposure to 10 μg/L nonylphenol. Vitellogenin was induced (approximately 10-fold) in adult male fish exposed to both 1 and 10 μg/L nonylphenol. In the male progeny of parental fish exposed to 10 μg/L nonylphenol, no effects were observed on plasma vitellogenin or testosterone concentrations, but plasma estradiol concentrations were elevated. In the female progeny of the same parental fish, plasma vitellogenin and plasma testosterone concentrations were elevated, but plasma estradiol concentrations were not different from control levels. Testicular tissue of nonylphenol exposed adult male fish was not affected by nonylphenol. Sex ratios of the offspring of exposed fish were also unaffected by parental exposure to nonylphenol.

As summarized in this section, the ability of nonylphenol to induce estrogenic effects has seldom been reported at concentrations below the freshwater Final Chronic Value of 6.5965 μg/L.

7. UNUSED DATA

Data from some studies were not used in this document, as they did not meet the criteria for inclusion as specified in the Guidelines (Stephan et al. 1985). The reader is referred to the Guidelines for further information regarding these criteria.

Results were not used when the test organism is not resident to North America (Gross-Sorokin et al. 2003; Yoshimura 1986). Tsuda et al. (2000) measured tissue concentrations from feral fish, but water concentrations greatly varied.

Test Organism or Test Material were Not Adequately Described

Folmar et al. (1998)
Hansen et al. (1998)
Kopf (1997)
Magliulo et al. (1998)
Muller (1980)
Palmer et al. (1998)
Weinberger and Rea (1981)

Nonylphenol was a Component of a Mixture or Sediment

Ahel et al. (1993)
Amato and Wayment (1998)
Bettinetti and Provini (2002)
Fay et al. (2000)
Dwyer et al. (1999a,b)
Escher et al. (1999)
Hansen et al. (1999)
Larsson et al. (1999)
Moore et al. (1987)
Purdom et al. (1994)
Sundaram et al. (1980)
Turner et al. (1985)
Ward and Boeri (1992)

Studies were Conducted with Ethoxylated Nonylphenols

Baldwin et al. (1998)
Braaten et al. (1972)
Dorn et al. (1993)
Maki et al. (1998)
Manzano et al. (1998, 1999)
Patoczka and Pulliam 1999

Organisms were Dosed by Injection, Gavage or in Artifical Medium

Arukwe et al. (1997a,b;1998)
Christiansen et al. (1998a,b,c; 1999)
Coldham et al. (1997, 1998)
Haya et al. (1997)
Madsen et al. (1997)
Nimrod and Benson (1996; 1997)
Rice et al. (1998)
Spieser et al. (1998)
Thibaut et al. (1998)
Weinberger et al. (1987)
Yadetie et al. (1999)

Experimental Model was Plasma, Enzymes, Receptors, Tissues or Cell Cultures

Andersen et al. (1999)
Celius et al. (1999)
Flouriot et al. (1995)
Hewitt et al. (1998)
Jobling et al. (1996)
Jobling and Sumpter (1993)
Knudsen and Pottinger (1999)
Lamche and Burkhardt-Holm (2000)
Levine and Cheney (2000)
Loomis and Thomas (1999)
Lutz and Kloas (1999)
Milligan et al. (1998)
Petit et al. (1997, 1999)
Routledge and Sumpter (1996, 1997)
Soto et al. (1991, 1992)
White et al. (1994)

Data were Compiled from Other Source

Bearden and Schultz (1997, 1998)
Lewis (1991)
Liber et al. (1999)
Varma and Patel (1988)
Veith and Mekenyan (1993

8. SUMMARY

8.1. Freshwater Data

Acute toxicity of nonylphenol was tested in 18 freshwater species and 2 subspecies from 15 genera (Figure 1 and Table 3). Species Mean Acute Values (SMAV) ranged from 55.72 µg/L for the amphipod *Hyalella azteca* to 774 µg/L for the snail *Physella virgata*. Eleven species of fish were tested and were in the mid-range of sensitivity (SMAVs = 110 to 289.3 µg/L) of tested species. The four most sensitive tested freshwater species were comprised of two invertebrate species and two vertebrate species (Figure 1). No relationships have been demonstrated between nonylphenol toxicity and water quality characteristics such as hardness and pH. The freshwater Final Acute Value is 55.49 µg/L which is equal to the LC50 for the most sensitive tested species, *Hyalella azteca*.

Chronic toxicity of nonylphenol was tested in 5 freshwater species from 5 genera (Figure 3 and Table 3). Two freshwater fish were tested; the rainbow trout, *Oncorhynchus mykiss*, had a chronic value of 7.861 µg/L based on growth, and the fathead minnow, *Pimephales promelas*, had a chronic value of 10.18 µg/L based on survival. Two species of freshwater cladocerans were tested and chronic values ranged from 22.62 to 157.9 µg/L based on reproduction. One species of freshwater midge was tested and its chronic value was 61.82 µg/L based on survival.

Data were available to calculate a Final Acute-Chronic Ratio (FACR) for a freshwater cladoceran, *Daphnia magna*, saltwater mysid, *Americamysis bahia*, and rainbow trout, *Oncorhynchus mykiss*. The Final Acute-Chronic Ratio for nonylphenol was the ACR for *A. bahia* because SMARs increased with increasing SMAV and the SMAV for *A. bahia* is closest to the freshwater and saltwater FAV. The FACR for nonylphenol is 8.412.

8.2. Saltwater Data

Acute toxicity of nonylphenol was tested in 11 saltwater species from 11 genera (Figure 2 and Table 3). Species Mean Acute Values (SMAV) ranged from 17 µg/L for the winter flounder, *Pleuronectes americanus*, to 209.8 µg/L for the sheepshead minnow, *Cyprinodon variegatus*. These two fish species were the only fish were tested. Nine different species of

invertebrates were tested. The four most sensitive tested saltwater species were comprised of three invertebrate species and one fish species (Figure 1). No relationships have been demonstrated between nonylphenol toxicity and water quality characteristics such as hardness and pH. The saltwater Final Acute Value is 13.93 μg/L.

Chronic toxicity of nonylphenol was tested on one saltwater species (Figure 3 and Table 3). The saltwater species tested was the mysid, *Americamysis bahia*, which was also the most sensitive of all species tested, both freshwater and saltwater. Two tests were available for *A. bahia*, with chronic values of 5.112 μg/L based on reduced growth and 12.02 μg/L based on a reproductive endpoint.

Data were available to calculate a Final Acute-Chronic Ratio (FACR) for a freshwater cladoceran, *Daphnia magna*, saltwater mysid, *Americamysis bahia*, and rainbow trout, *Oncorhynchus mykiss*. The Final Acute-Chronic Ratio for nonylphenol was the ACR for *A. bahia* because SMARs increased with increasing SMAV and the SMAV for *A. bahia* is closest to both the freshwater and saltwater FAV. The FACR for nonylphenol is 8.412.

8.3. Plant Data

Nonylphenol toxicity data for 2 species of aquatic plants, one freshwater alga and one saltwater diatom, were available. Algae were as sensitive as animals, showing effect concentrations that ranged from 27 μg/L for the freshwater alga to 410 μg/L for the saltwater diatom. Based on the vegetative growth endpoint in saltwater planktonic diatom *Skeletonema costatum*, the Final Plant Value for nonylphenol is 27 μg/L.

8.4. Bioaccumulation Data

Nonylphenol bioaccumulation in aquatic organisms is less than would be predicted from the log K_{ow} of nonylphenol. Nonylphenol is metabolized in animals which may account for the lower than expected BCFs. In freshwater fish, lipid-normalized BCFs ranged from 39 to 209. Bioaccumulation in saltwater organisms is apparently greater, with lipid-normalized BCFs 79 to 2,168.

9. NATIONAL CRITERIA

9.1. Freshwater

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" (Stephan et al. 1985) indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the one-hour average concentration of nonylphenol does not exceed 28 μg/L more than once every three years on the average and if the four-day average concentration of nonylphenol does not exceed 6.6 μg/L more than once every three years on the average.

9.2. Saltwater

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" (Stephan et al. 1985) indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the one-hour average concentration of nonylphenol does not exceed 7.0 μg/L more than once every three years on the average and if the four-day average concentration of nonylphenol does not exceed 1.7 μg/L more than once every three years on the average.

10. IMPLEMENTATION

As discussed in the Water Quality Standards Regulation (U.S. EPA 1983) and the Foreword to this document, a water quality criterion for aquatic life has regulatory impact only after it has been adopted in a state or tribal water quality standard. Such a standard specifies a criterion for a pollutant that is consistent with a particular designated use. With the concurrence of the U.S. EPA, states and tribes designate one or more uses for each body of water or segment thereof and adopt criteria that are consistent with the use(s) (U.S. EPA 1994, 1987). In each standard a state or tribe may adopt the national criterion, if one exists, or, if adequately justified, a site-specific criterion (if the site is an entire state, the site-specific criterion is also a state-specific criterion).

Site-specific criteria may include not only site-specific criterion concentrations (U.S. EPA 1994), but also site-specific, and possibly pollutant-specific, durations of averaging periods and frequencies of allowed excursions (U.S. EPA 1991). The averaging periods of "one hour" and "four days" were selected by the U.S. EPA on the basis of data concerning how rapidly some aquatic species react to increases in the concentrations of some pollutants, and "three years" is the Agency's best scientific judgment of the average amount of time aquatic ecosystems should be provided between excursions (Stephan et al. 1985; U.S. EPA 1991). However, various species and ecosystems react and recover at greatly different rates. Therefore, if adequate justification is provided, site-specific and/or pollutant-specific concentrations, durations, and frequencies may be higher or lower than those given in national water quality criteria for aquatic life.

Use of criteria, which have been adopted into state or tribal water quality standards, for developing water quality-based permit limits requires selection of an appropriate wasteload allocation model. Although dynamic models are preferred for the application of these criteria (U.S. EPA 1991), limited data or other considerations might require the use of a steady-state model (U.S. EPA 1986). Guidance on mixing zones and the design of monitoring programs is also available (U.S. EPA 1987, 1991).

Figure 1. Summary of Ranked Nonylphenol GMAVs

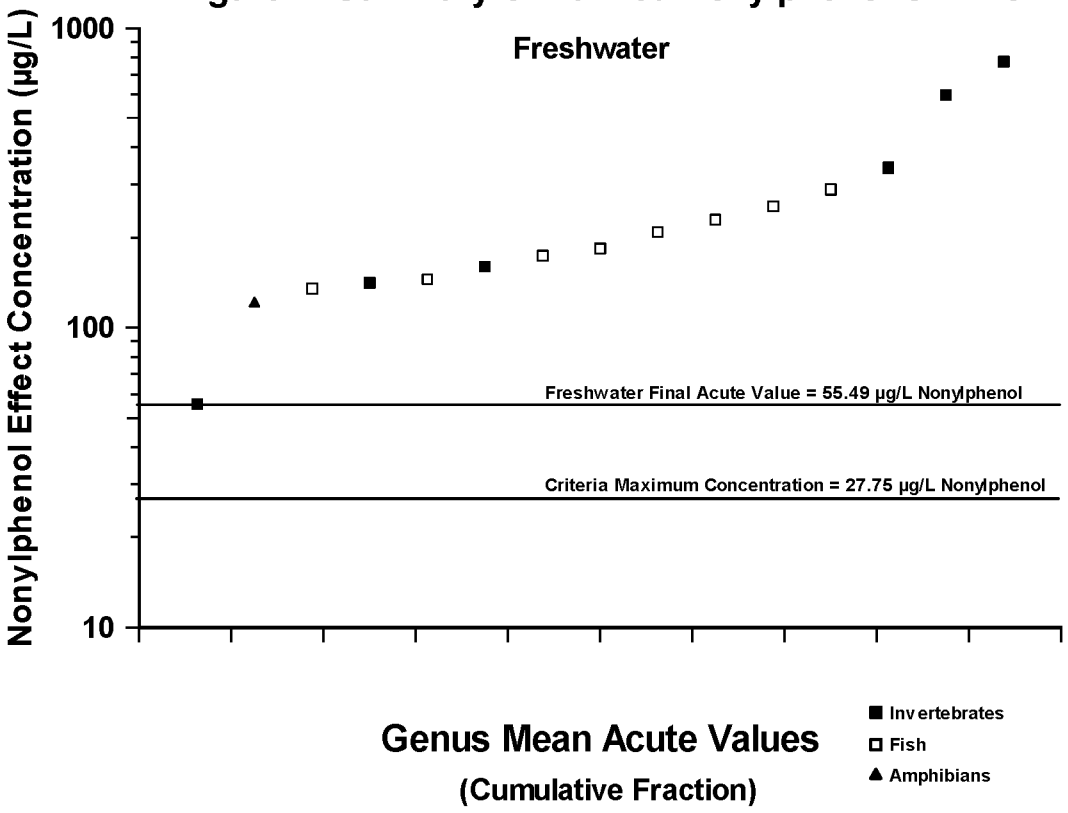

Figure 2. Summary of Ranked Nonylphenol GMAVs

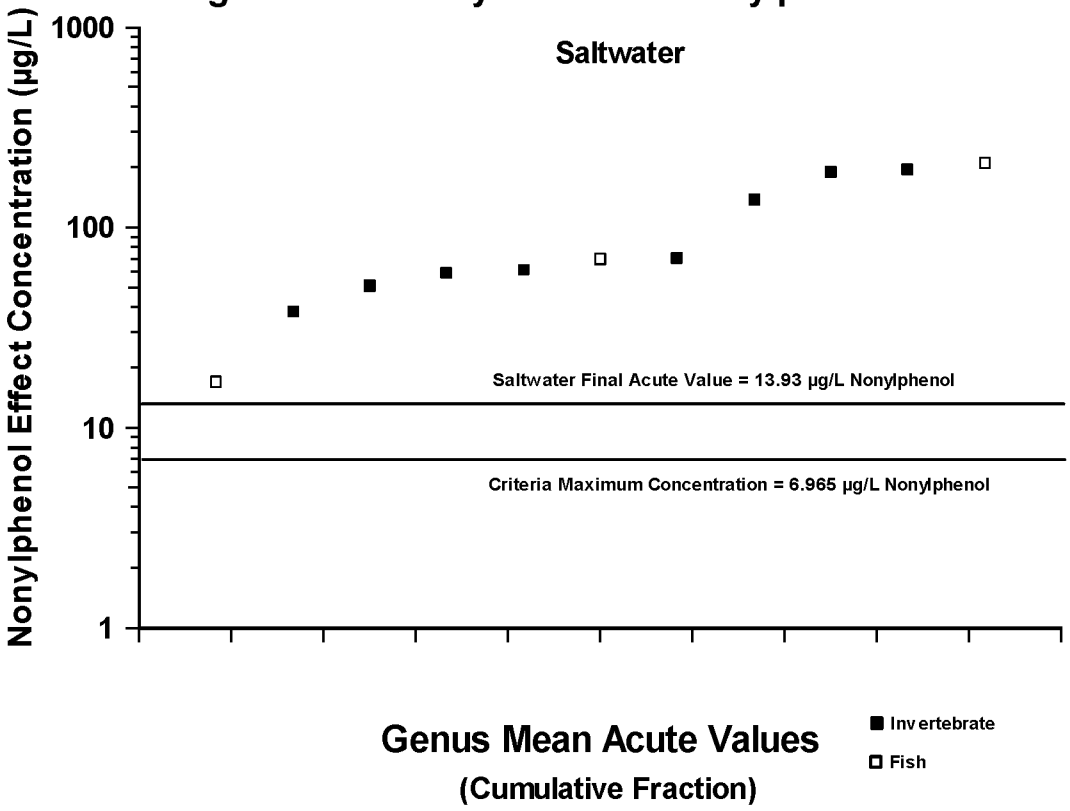

Figure 3. Chronic Toxicity of Nonylphenol to Aquatic Animals

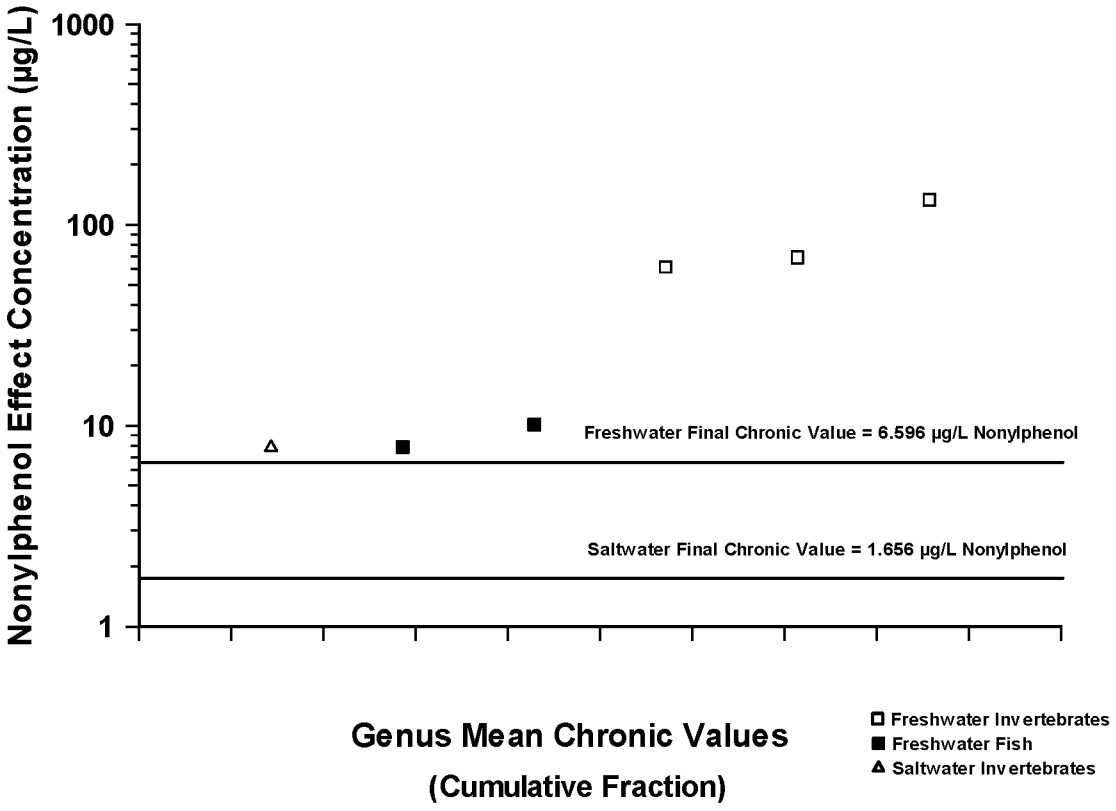

Table 1. Acute Toxicity of Nonylphenol to Aquatic Animals

Species	Method[a]	Chemical	pH	LC_{50} or EC_{50} (µg/L)	Species Mean Acute Value[b] (µg/L)	Reference
			FRESHWATER SPECIES			
Annelid (adult), *Lumbriculus variegatus*	F,M	>90%	6.75	**342**	342	Brooke 1993a
Snail (adult), *Physella virgata*	F,M	>90%	7.89	**774**	774	Brooke 1993a
Cladoceran (<24-hr old), *Daphnia magna*	R,M	>90%	7.87	**104**	-	Brooke 1993a
Cladoceran (<24-hr old), *Daphnia magna*	S,M	91.8%	8.25	**190**	140.6	Comber et al. 1993
Midge (2nd instar), *Chironomus tentans*	F,M	>95%	8.0-8.4	**160**	160	England and Bussard 1995
Dragonfly (nymph), *Ophiogomphus* sp.	F,M	>90%	8.06	**596**	596	Brooke 1993a
Amphipod, (juvenile, 2mm TL), *Hyalella azteca*	F,M	>90%	7.80	**20.7**	-	Brooke 1993a
Amphipod (juvenile, 2-3mm TL), *Hyalella azteca*	F,M	>95%	7.9-8.7	**150**	55.72	England and Bussard 1995
Rainbow trout (0.67 ± 0.35 g), *Oncorhynchus mykiss*	S,U	85%	7.8-7.9	190	-	Dwyer et al. 1995
Rainbow trout (1.25 ± 0.57 g), *Oncorhynchus mykiss*	S,U	85%	7.5-7.7	260	-	Dwyer et al. 1995
Rainbow trout (0.27 ± 0.07 g), *Oncorhynchus mykiss*	S,U	85%	7.9	140	-	Dwyer et al. 1995
Rainbow trout (1.09 ± 0.38 g), *Oncorhynchus mykiss*	S,U	85%	7.7-7.9	270	-	Dwyer et al. 1995

Table 1. Acute Toxicity of Nonylphenol to Aquatic Animals

Species	Method[a]	Chemical	pH	LC_{50} or EC_{50} (µg/L)	Species Mean Acute Value[b] (µg/L)	Reference
Rainbow trout (0.48 ± 0.08 g), *Oncorhynchus mykiss*	S,U	85%	7.5-7.9	160	-	Dwyer et al. 1995
Rainbow trout (0.50 ± 0.21 g), *Oncorhynchus mykiss*	S,U	85%	6.5-7.9	180	-	Dwyer et al. 1995
Rainbow trout (45 d), *Oncorhynchus mykiss*	F,M	>90%	6.72	**221**	221	Brooke 1993a
Apache trout (0.85 ± 0.49 g), *Oncorhynchus apache*	S,U	85%	7.8-7.9	**180**	-	Dwyer et al. 1995
Apache trout (0.38 ± 0.18 g), *Oncorhynchus apache*	S,U	85%	7.3-7.7	**160**	169.7	Dwyer et al. 1995
Greenback cutthroat trout (0.31 ± 0.17 g), *Oncorhynchus clarki stomais*	S,U	85%	7.5-7.6	**150**	-	Dwyer et al. 1995
Lahontan cutthroat trout (0.34 ± 0.08 g), *Oncorhynchus clarki henshawi*	S,U	85%	7.9	**140**	-	Dwyer et al. 1995
Lahontan cutthroat trout (0.57 ± 0.23 g), *Oncorhynchus clarki henshawi*	S,U	85%	7.6-7.7	**220**	166.6	Dwyer et al. 1995
Fathead minnow (0.32 ± 0.16 g), *Pimephales promelas*	S,U	85%	7.7-8.1	210	-	Dwyer et al. 1995
Fathead minnow (0.56 ± 0.19 g), *Pimephales promelas*	S,U	85%	7.8-8.1	360	-	Dwyer et al. 1995
Fathead minnow (0.45 ± 0.35 g), *Pimephales promelas*	S,U	85%	7.6-7.8	310	-	Dwyer et al. 1995

Table 1. Acute Toxicity of Nonylphenol to Aquatic Animals

Species	Method[a]	Chemical	pH	LC_{50} or EC_{50} (µg/L)	Species Mean Acute Value[b] (µg/L)	Reference
Fathead minnow (0.40 ± 0.21 g), *Pimephales promelas*	S,U	85%	7.5-7.9	330	-	Dwyer et al. 1995
Fathead minnow (0.34 ± 0.24 g), *Pimephales promelas*	S,U	85%	7.5-7.6	170	-	Dwyer et al. 1995
Fathead minnow (0.39 ± 0.14 g), *Pimephales promelas*	S,U	85%	7.8-8.2	290	-	Dwyer et al. 1995
Fathead minnow (32 d), *Pimephales promelas*	F,M	99%	7.29	**140**	-	Holcombe et al. 1984; Univ. Wisc.-Superior 1985
Fathead minnow (25-35 d), *Pimephales promelas*	F,M	>90%	7.23	**128**	133.9	Brooke 1993a
Bonytail chub (0.29 ± 0.08 g), *Gila elegans*	S,U	85%	7.7-7.9	**270**	-	Dwyer et al. 1995
Bonytail chub (0.52 ± 0.09 g), *Gila elegans*	S,U	85%	7.4-7.6	**310**	289.3	Dwyer et al. 1995
Colorado squawfish (0.32 ± 0.05 g), *Ptychocheilus lucius*	S,U	85%	8.1-8.2	**240**	-	Dwyer et al. 1995
Colorado squawfish (0.34 ± 0.05 g), *Ptychocheilus lucius*	S,U	85%	7.8-8.0	**270**	254.6	Dwyer et al. 1995
Razorback sucker (0.31 ± 0.04 g), *Xyrauchen texanus*	S,U	85%	7.8-8.1	**160**	-	Dwyer et al. 1995
Razorback sucker (0.32 ± 0.07 g), *Xyrauchen texanus*	S,U	85%	7.9-8.0	**190**	174.4	Dwyer et al. 1995
Gila topminnow (0.219 g, 27.2 mm), *Poeciliopsis occidentalis*	S,U	85%	8.0	**230**	230	Dwyer et al. 1999a

Table 1. Acute Toxicity of Nonylphenol to Aquatic Animals

Species	Method[a]	Chemical	pH	LC_{50} or EC_{50} (µg/L)	Species Mean Acute Value[b] (µg/L)	Reference
Fountain darter (0.062 g, 20.2 mm), *Etheostoma rubrum*	S,U	85%	8.0-8.1	**110**	110	Dwyer et al. 1999a
Greenthroat darter (0.133 g, 22.6 mm), *Etheostoma lepidum*	S,U	85%	8.0-8.2	**190**	190	Dwyer et al. 1999a
Bluegill (juvenile), *Lepomis macrochirus*	F,M	>90%	7.61	**209**	209	Brooke 1993a
Boreal toad (0.012 g, 9.6 mm), *Bufo boreas*	S,U	85%	7.9-8.0	**120**	120	Dwyer et al. 1999a
			SALTWATER SPECIES			
Coot clam (embryo/larva), *Mulinia lateralis*	S,U	90%	7.8-8.2	**37.9**	37.9	Lussier et al. 2000
Copepod (10-12 d), *Acartia tonsa*	S,U	-	-	**190**	190	Kusk and Wollenberger 1999
Mysid (<24-hr old), *Americamysis bahia*	F,M	>95%	7.3-8.2	**43**	-	Ward and Boeri 1990a
Mysid (<24-hr old), *Americamysis bahia*	F,M	90%	7.8-8.2	**60.6**	51.05	Lussier et al. 2000
Amphipod (adult), *Leptocheirus plumulosus*	F,M	90%	7.8-8.2	**61.6**	61.6	Lussier et al. 2000
Amphipod (adult), *Eohaustorius estuarius*	S,U	-	-	**138**	138	Hecht and Boese 2002
Grass shrimp (48-hr old), *Palaemonetes vulgaris*	F,M	90%	7.8-8.2	**59.4**	59.4	Lussier et al. 2000
American lobster (1st stage), *Homarus americanus*	R,U	90%	7.8-8.2	**71**	71	Lussier et al. 2000
Mud crab (4th and 5th stages), *Dyspanopeus sayii*	F,M	90%	7.8-8.2	**>195**	>195	Lussier et al. 2000

Table 1. Acute Toxicity of Nonylphenol to Aquatic Animals

Species	Method[a]	Chemical	pH	LC_{50} or EC_{50} (µg/L)	Species Mean Acute Value[b] (µg/L)	Reference
Winter flounder (48-hr-old), *Pleuronectes americanus*	S,M	90%	7.8-8.2	**17**	17	Lussier et al. 2000
Sheepshead minnow (juvenile), *Cyprinodon variegatus*	F,M	>95%	7.4-8.1	**310**	-	Ward and Boeri 1990b
Sheepshead minnow (juvenile), *Cyprinodon variegatus*	F,M	90%	7.8-8.2	**142**	209.8	Lussier et al. 2000
Inland silversides (juvenile), *Menidia beryllina*	F,M	90%	7.8-8.2	**70**	70	Lussier et al. 2000

[a] S = static; R = renewal; F = flow-through; M = measured; U = unmeasured.
[b] Each Species Mean Acute Value was calculated from the underlined number(s) in the preceding column.

Table 2. Chronic Toxicity of Nonylphenol to Aquatic Animals

Species	Test[a]	Chemical	pH	Chronic Limits (µg/L)[b]	Chronic Value (µg/L)	Reference
Cladoceran, *Ceriodaphnia dubia*	LC	>95%	8.3-8.6	88.7-202	133.9	England 1995
Cladoceran, *Daphnia magna*	LC	93.1	8.04	10.7-47.8	22.62	Fliedner 1993
Cladoceran, *Daphnia magna*	LC	>90%	8.46	116-215	157.9	Brooke 1993a
Cladoceran, *Daphnia magna*	LC	91.8%	8.25	24-39	30.59	Comber et al. 1993
Midge, *Chironomus tentans*	LC	95%	7.73	42-91	61.82	Kahl et al. 1997
Rainbow trout, *Oncorhynchus mykiss*	ELS	>90%	6.97	6.0-10.3	7.861	Brooke 1993a
Fathead minnow, *Pimephales promelas*	ELS	>95%	7.1-8.2	7.4-14	10.18	Ward and Boeri 1991c
		SALTWATER SPECIES				
Mysid, *Americamysis bahia*	LC	>95%	7.4-8.3	3.9-6.7	5.112	Ward and Boeri 1991b
Mysid, *Americamysis bahia*	LC	-	-	9.46-15.28	12.02	Kuhn et al. 2001

[a] LC = life-cycle or partial life-cycle; ELS = early life-stage.
[b] Based upon measured concentrations of nonylphenol.

Table 2. Acute-Chronic Ratios

Acute-Chronic Ratios

Species	pH	Acute Value (µg/L)	Chronic Value (µg/L)	Ratio	Reference
Cladoceran, *Daphnia magna*	7.87-8.46	104	157.9	0.6586	Brooke 1993a
Cladoceran, *Daphnia magna*	8.25	190	30.59	6.211	Comber et al. 1993
Mysid, *Americamysis bahia*	7.3-8.3	43	5.112	8.412	Ward and Boeri 1990a, 1991b
Rainbow trout, *Oncorhynchus mykiss*	6.72-6.97	221	7.861	28.11	Brooke 1993a

Table 3. Ranked Genus Mean Acute Values with Species Mean Acute-Chronic Ratios

Rank[a]	Genus Mean Acute Value (µg/L)	Species	Species Mean Acute Value (µg/L)[b]	Species Mean Acute-Chronic Ratio[c]
		FRESHWATER SPECIES		
15	774	Snail, *Physella virgata*	774	-
14	596	Dragonfly, *Ophiogomphus sp.*	596	-
13	342	Annelid, *Lumbriculus variegatus*	342	-
12	289.3	Bonytail chub, *Gila elegans*	289.3	-
11	254.6	Colorado squawfish, *Ptychocheilus lucius*	254.6	-
10	230	Gila topminnow, *Poeciliopsis occidentalis*	230	-
9	209	Bluegill, *Lepomis macrochirus*	209	-
8	184.2	Rainbow trout, *Oncorhynchus mykiss*	221	28.11
		Apache trout, *Oncorhynchus apache*	169.7	-
		Lahontan cutthroat trout, *Oncorhynchus clarki henshawi*, and	166.6	-
		Greenback cutthroat trout, *Oncorhynchus clarki stomais*	-	-
7	174.4	Razorback sucker, *Xyrauchen texanus*	174.4	-
6	160	Midge, *Chironomus tentans*	160	-
5	144.6	Greenthroat darter, *Etheostoma lepidum*	190	-
		Fountain darter, *Etheostoma rubrum*	110	-
4	140.6	Cladoceran, *Daphnia magna*	140.6	2.023

Table 3. Ranked Genus Mean Acute Values with Species Mean Acute-Chronic Ratios

Rank[a]	Genus Mean Acute Value (µg/L)	Species	Species Mean Acute Value (µg/L)[b]	Species Mean Acute-Chronic Ratio[c]
3	133.9	Fathead minnow, *Pimephales promelas*	133.9	-
2	120	Boreal toad, *Bufo boreas*	120	-
1	55.72	Amphipod, *Hyalella azteca*	55.72	-
		SALTWATER SPECIES		
11	209.8	Sheepshead minnow, *Cyprinodon variegatus*	209.8	-
10	>195	Mud crab, *Dyspanopeus sayii*	>195	-
9	190	Copepod, *Acartia tonsa*	190	
8	138	Amphipod, *Eohaustorius estuarius*	138	-
7	71	American lobster, *Homarus americanus*	71	-
6	70	Inland silversides, *Menidia beryllina*	70	-
5	61.6	Amphipod, *Leptocheirus plumulosus*	61.6	-
4	59.4	Grass shrimp, *Palaemonetes vulgaris*	59.4	-
3	51.05	Mysid, *Americamysis bahia*	51.05	8.412
2	37.9	Coot clam, *Mulinia lateralis*	37.9	-
1	17	Winter flounder, *Pleuronectes americanus*	17	-

[a] Ranked from the most resistant to the most sensitive based on Genus Mean Acute Value.
[b] From Table 1.
[c] From Table 2.

Table 3. Ranked Genus Mean Acute Values with Species Mean Acute-Chronic Ratios

Freshwater

Final Acute Value = 55.49 µg/L

Criterion Maximum Concentration = 55.49 ÷ 2 = 27.75 µg/L

Final Acute-Chronic Ratio = 8.412 (see text)

Final Chronic Value = 55.49 µg/L ÷ 8.412 = 6.5965 µg/L

Saltwater

Final Acute Value = 13.93 µg/L

Criterion Maximum Concentration = 13.93 ÷ 2 = 6.965 µg/L

Final Acute-Chronic Ratio = 8.412 (see text)

Final Chronic Value = 13.93 µg/L ÷ 8.412 = 1.6560 µg/L

Table 4. Toxicity of Nonylphenol to Aquatic Plants

Species	Chemical	pH	Duration (days)	Effect	Concentration (µg/L)	Reference
			FRESHWATER SPECIES			
Green algae, *Selenastrum capricornutum*	>95%	7.8	4	EC50, number of cells	410	Ward and Boeri 1990a
			SALTWATER SPECIES			
Diatom, *Skeletonema costatum*	>95%	30[a]	4	EC50, number of cells	27	Ward and Boeri 1990d

[a]Salinity (g/kg).

Table 5. Bioaccumulation of Nonylphenol by Aquatic Organisms

Species	Chemical	Water Conc. (µg/L)[a]	pH	Duration (days)	Tissue	Percent Lipids	BCF or BAF[b]	Normalized BCF or BAF[c]	Reference
				FRESHWATER SPECIES					
Fathead minnow (0.5-1 g), *Pimephales promelas*	>95%	4.9	7.0-7.6	27	Whole body	-	271	-	Ward and Boeri 1991a
Fathead minnow (0.5-1 g), *Pimephales promelas*	>95%	22.7	7.0-7.6	27	Whole body	-	344	-	Ward and Boeri 1991a
Fathead minnow (4-wk old), *Pimephales promelas*	99%	18.4	7.62	4	Whole body	4.7±1.7	751	159.8	Brooke 1993b
Fathead minnow (4-wk old), *Pimephales promelas*	99%	41.9	7.62	4	Whole body	4.7±1.7	677	144.0	Brooke 1993b
Fathead minnow (4-wk old), *Pimephales promelas*	99%	82.1	7.62	4	Whole body	4.7±1.7	945	201.1	Brooke 1993b
Fathead minnow (4-wk old), *Pimephales promelas*	99%	9.3	7.60	28	Whole body	4.7±1.7	769	163.6	Brooke 1993b
Fathead minnow (4-wk old), *Pimephales promelas*	99%	19.2	7.60	28	Whole body	4.7±1.7	984	209.4	Brooke 1993b

Table 5. Bioaccumulation of Nonylphenol by Aquatic Organisms

Species	Chemical	Water Conc. (µg/L)[a]	pH	Duration (days)	Tissue	Percent Lipids	BCF or BAF[b]	Normalized BCF or BAF[c]	Reference
Fathead minnow (4-wk old), *Pimephales promelas*	99%	38.1	7.60	28	Whole body	4.7±1.7	876	186.4	Brooke 1993b
Fathead minnow (4-wk old), *Pimephales promelas*	99%	77.5	7.60	28	Whole body	4.7±1.7	603	128.3	Brooke 1993b
Fathead minnow (adult), *Pimephales promelas*	>98%	0.4 1.6 3.4	-	42	Whole body	-	203 252 268	- - -	Giesy et al. 2000
Bluegill (4-wk old), *Lepomis macrochirus*	99%	21.6	7.79	4	Whole body	4.9±1.5	279	56.94	Brooke 1993b
Bluegill (4-wk old), *Lepomis macrochirus*	99%	43.9	7.79	4	Whole body	4.9±1.5	257	52.45	Brooke 1993b
Bluegill (4-wk old), *Lepomis macrochirus*	99%	86.5	7.79	4	Whole body	4.9±1.5	223	45.51	Brooke 1993b
Bluegill (4-wk old), *Lepomis macrochirus*	99%	5.6	7.55	28	Whole body	4.9±1.5	231	47.14	Brooke 1993b
Bluegill (4-wk old), *Lepomis macrochirus*	99%	12.4	7.55	28	Whole body	4.9±1.5	253	51.63	Brooke 1993b
Bluegill (4-wk old), *Lepomis macrochirus*	99%	27.6	7.55	28	Whole body	4.9±1.5	250	51.02	Brooke 1993b

Table 5. Bioaccumulation of Nonylphenol by Aquatic Organisms

Species	Chemical	Water Conc. (µg/L)[a]	pH	Duration (days)	Tissue	Percent Lipids	BCF or BAF[b]	Normalized BCF or BAF[c]	Reference
Bluegill (4-wk old), *Lepomis macrochirus*	99%	59.5	7.55	28	Whole body	4.9±1.5	191	38.98	Brooke 1993b
Bluegill (juvenile), *Lepomis macrochirus*	96.4%	1.0 3.0 30.0	7.7	20	Whole body	0.72± 0.46	76 60 37	105.6 83.33 51.39	Liber et al. 1999
				SALTWATER SPECIES					
Blue mussel, *Mytilus edulis*	^{14}C-labeled	5.9	-	16	Whole body	1.6	2,740	1,712	Ekelund et al. 1990
Blue mussel, *Mytilus edulis*	^{14}C-labeled	6.2	-	16	Whole body	1.9	4,120	2,168	Ekelund et al. 1990
Common shrimp, *Crangon crangon*[d]	^{14}C-labeled	6.4	-	16	Whole body	1.4	110	78.75	Ekelund et al. 1990
Common shrimp, *Crangon crangon*[d]	^{14}C-labeled	7.4	-	16	Whole body	1.7	900	529.4	Ekelund et al. 1990
Three-spined stickleback, *Gasterosteus aculeatus*	^{14}C-labeled	4.8	-	16	Whole body	6.7	1,200	179.1	Ekelund et al. 1990
Three-spined stickleback, *Gasterosteus aculeatus*	^{14}C-labeled	4.9	-	16	Whole body	7.8	1,300	166.7	Ekelund et al. 1990

Table 5. Bioaccumulation of Nonylphenol by Aquatic Organisms

Species	Chemical	Water Conc. (µg/L)[a]	pH	Duration (days)	Tissue	Percent Lipids	BCF or BAF[b]	Normalized BCF or BAF[c]	Reference

[a]Measured concentration of nonylphenol.
[b]Bioconcentration factors (BCFs) and bioaccumulation factors (BAFs) are based on measured concentrations of nonylphenol in water and in tissue.
[c]When possible, the factors were normalized to 1% lipid by dividing the BCFs and BAFs by the percent lipid measured in the test organism.
[d]Non-resident species.

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
FRESHWATER SPECIES						
Phytoplankton and Periphyton	>98%	8.8 - 10.6	6 wk	Dominant species changed	29 - 120	Hense et al. 2003
Green alga, *Chlamydomonas reinhardtii*	-	-	24 days	100% algistatic	6,250	Weinberger and Greenhalgh 1984
Floating moss, *Salvinia molesta*	-	-	9 days	Reduced frond production	2,500	Prasad 1986
Duckweed, *Lemna minor*	-	5.6	96 hr	IC50	5,500	Weinberger and Iyengar 1983
Duckweed, *Lemna minor*	-	-	4 days	Reduced frond production	125	Prasad 1986
Ciliate protozoan, *Tetrahymena pyriformis*	-	-	24 hr	EC50	460	Yoshioka 1985
Ciliate protozoan, *Tetrahymena pyriformis*	-	7.40	40 hr	Reduced population growth 50%	747	Schultz 1997
Rotifer (4 to 6 hr-old female) *Brachionus calyciflorus*	Technical	7.5	96 hr	Sexual reproduction reduced	50	Preston et al. 2000
Clam (15 g), *Anodonta cataractae*	-	-	144 hr (fed)	LC50	5,000	McLeese et al. 1980b
Zooplankton	96.4%	7.5 - 8.2	20 days	NOEC LOEC	23 76	O'Halloran et al. 1999
Zooplankton	>98%	8.8 - 10.4	6 wk	NOEC	19 - 44	Severin et al. 2003
Benthic macro-invertebrates	96.4%	7.5 - 8.2	20 days	NOEC LOEC	23 76	Schmude et al. 1999
Cladoceran (<24-hr old), *Daphnia magna*	-	8.0	21 days	NOEC LOEC (reduced fecundity)	50 100	Baldwin et al. 1997

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Cladoceran (<24-hr old and adults), *Daphnia magna*	~85%	7.8 - 8.4	96 hr (fed)	MATC (young) MATC (adults)	302 136	Gerritsen et al. 1998
Cladoceran (<24-hr old), *Daphnia magna*	~85%	7.7±0.02	21 days	No sex ratio change (high food rate) Increased ratio of males (low food rate)	25 25	Baer and Owens 1999
Cladoceran (<24-hr old), *Daphnia magna*	Technical	-	21 days	50% adult mortality NOEC (deformed offspring)	200.5 44	LeBlanc et al. 2000
Cladoceran (<24-hr old), *Daphnia magna*	~85%	-	48 hr	EC50	234 272 337	Zang et al. 2003
Cladoceran (<24-hr old), *Daphnia magna*	~85%	-	35 day	LOEC	>50	Zang et al. 2003
Cladoceran (<36-hr old), *Daphnia galeata mendotae*	-	-	30 day	NOEC LOEC (deformed offspring)	10 50	Shurin and Dodson 1997
Cladoceran (>48-hr old), *Daphnia pulex*	Practical grade	-	48 hr	LC50	140	Ernst et al. 1980
Cladoceran (>48-hr old), *Daphnia pulex*	Practical grade	-	48 hr	LC50	176	Ernst et al. 1980
Cladoceran (>48-hr old), *Daphnia pulex*	Practical grade	-	48 hr	LC50	190	Ernst et al. 1980
Cladoceran (<24-hr old), *Ceriodaphnia dubia*	>95%	8.3-8.6	48 hr	LC50 (fed)	276	England 1995

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Cladoceran (<24-hr old), *Ceriodaphnia dubia*	>95%	8.3-8.6	7 days	LC50 (fed)	225	England 1995
Midge (2nd instar), *Chironomus tentans*	>95%	8.2	14 days	LC50 EC50	119 95	England and Bussard 1993
Sea lamprey (larva), *Petromyzon marinus*	-	7.5-8.2	14 hr	LT100	5,000	Applegate et al. 1957
Brook trout (juvenile), *Salvelinus fontinalis*	-	-	96 hr	LC50	145	Holmes and Kingsbury 1980
Lake trout (juvenile), *Salvelinus naymaycush*	-	-	35 days	LC50 (fed)	>40	Holmes and Kingsbury 1980
Brown trout (fingerling), *Salmo trutta*	-	7.0	2 hr	LT100	5,000	Wood 1953
Atlantic salmon (4 g), *Salmo salar*	-	-	96 hr (fed)	LC50	900	McLeese et al. 1980b
Atlantic salmon (48.3 $\pm$ 2.6 mm TL), *Salmo salar*	-	-	30 days	No change in plasma vitellogenin or gill NaK ATPase activity or plasma Cl^- and Na^+	20	Moore et al. 2003
Chinook salmon (juvenile), *Oncorhynchus tshawytscha*	-	7.2	3 hr	LT100	10,000	MacPhee and Ruelle 1969
Rainbow trout (juvenile), *Oncorhynchus mykiss*	-	7.5-8.2	4 hr	LT100	5,000	Applegate et al. 1957

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Rainbow trout (juvenile), *Oncorhynchus mykiss*	Practical grade	-	96 hr	LC50	920	Ernst et al. 1980
Rainbow trout (juvenile), *Oncorhynchus mykiss*	Practical grade	-	96 hr	LC50	560	Ernst et al. 1980
Rainbow trout (juvenile), *Oncorhynchus mykiss*	-	-	96 hr	LC50	230	Holmes and Kingsbury 1980
Rainbow trout (adult males), *Oncorhynchus mykiss*	-	-	3 wk	Increased vitellogenin production	20.3	Jobling et al. 1996
Rainbow trout (adult males), *Oncorhynchus mykiss*	-	-	3 wk	Increased vitellogenin production	54.3	Jobling et al. 1996
Rainbow trout (50 - 200 g), *Oncorhynchus mykiss*	-	-	72 hr	LC50	193.65	Lech et al. 1996
Rainbow trout (50 - 200 g), *Oncorhynchus mykiss*	-	-	72 hr	Increased vitellogenin mRNA	14.14	Lech et al. 1996
Rainbow trout, (40 - 60 g), *Oncorhynchus mykiss*	>99%		8 hr	Tissue half-life fat 19.8 hr muscle 18.6 hr liver 5.9 hr	18	Lewis and Lech 1996
Rainbow trout, (40 - 60 g), *Oncorhynchus mykiss*	>99%		2 – 5 hr	Eviscerated carcass BAF = 24.21	18	Lewis and Lech 1996
Rainbow trout, (40 - 60 g), *Oncorhynchus mykiss*	>99%		2 – 5 hr	Viscera BAF = 98.2	18	Lewis and Lech 1996

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Rainbow trout (juvenile), *Oncorhynchus mykiss*	-	-	4 hr	Vitellogenin mRNA production	10	Ren et al. 1996a
Rainbow trout (juvenile), *Oncorhynchus mykiss*	-	-	72 hr	Vitellogenin mRNA production	100	Ren et al. 1996a
Rainbow trout (juvenile), *Oncorhynchus mykiss*	-	6.5	22 days	Reduced growth at 108 days	50	Ashfield et al. 1998
			35 days	Reduced growth at 466 days	30	
Rainbow trout (juvenile), *Oncorhynchus mykiss*	-	-	96 hr	Decreased number of muscarinic cholinergic receptors in brain	220	Jones et al. 1998
Rainbow trout (35-50 g, immature), *Oncorhynchus mykiss*	-	8.0 – 8.4	21 days	Increased vitellogenin in blood plasma	50	Tremblay and Van Der Kraak 1998
Rainbow trout (adult males), *Oncorhynchus mykiss*	-	-	3 wk	BCF = 116 BCF = 88	63 81	Blackburn et al. 1999
Rainbow trout (103-168 g, juvenile) *Oncorhynchus mykiss*	99%	-	9 days	No vitellogenin induction	109	Pedersen et al. 1999
Rainbow trout (adult males), *Oncorhynchus mykiss*	Technical	-	10 days per month for 4 months	Epidermal mucous cell granulation	1	Burkhardt-Holm et al. 2000

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Rainbow trout (598 g; juvenile females), *Oncorhynchus mykiss*	99%	-	18 wk	Reduced GSI; Reduced HSI; Induced vitellogenin; Lowered plasma estradiol; Lowered plasma FSH	85.6 85.6 8.3 85.6 8.3	Harris et al. 2001
Rainbow trout (1667±201.6 g; F_0 3 yr-old adults), *Oncorhynchus mykiss*	98%	7.6	4 months (exposed 10 days/month)	Reduced embryo survival; Reduced hatch; F_o Males increased vitellogenin; F_1 Females increased vitellogenin and testosterone; F_1 Males increased estradiol	1 10 1 10 10	Schwaiger et al. 2002
Rainbow trout (6-mo-old), *Oncorhynchus mykiss*	-	7.2	4 wk	Liver tissue showed hemorrhage and lymphocyte infiltration	220	Uguz et al. 2003
Lahontan cutthroat trout (juvenile), *Oncorhynchus clarki henshawi*	-	-	96 hr	Decreased number of muscarinic cholinergic receptors in brain	220	Jones et al. 1998
Apache trout (juvenile), *Oncorhynchus mykiss*	-	-	96 hr	Decreased number of muscarinic cholinergic receptors in brain	>130	Jones et al. 1998
Northern squawfish (juvenile), *Ptychocheilus oregonensis*	-	7.2	3 hr	LT100	10,000	MacPhee and Ruelle 1969

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Colorado squawfish (juvenile), *Ptychocheilus lucius*	-	-	96 hr	Decreased number of muscarinic cholinergic receptors in brain	>220	Jones et al. 1998
Goldfish (juvenile), *Carassius auratus*	-	7.0	5 hr	LT100	5,000	Wood 1953
Common carp (15.2 ± 3.8 g juvenile), *Cyprinus carpio*	Technical (90% 4-NP)	7.6	70 days	Decreased erythrocytes; Increased reticulocytes	10 10	Schwaiger et al. 2000
Common carp (50-150 g mature males), *Cyprinus carpio*	95%	7.57 ±0.03	28-31 days 11 °C	BCF = 546.5 No change in 17-estradiol, testosterone, or vitellogenin	5.36	Villenueve et al. 2002
Fathead minnow (4-wk old), *Pimephales promelas*	99%	7.62	4 days	LC50 (fed)	138	Brooke 1993b
Fathead minnow (4-wk old), *Pimephales promelas*	99%	7.60	28 days	BCF = 100.4	193	Brooke 1993b
Fathead minnow, *Pimephales promelas*	-	-	96 hr	Decreased number of muscarinic cholinergic receptors in brain	>220	Jones et al. 1998
Fathead minnow (mature), *Pimephales promelas*	>98%	-	42 days	Possible increased number of Sertoli cells in males	1.6	Miles-Richardson et al. 1999
Fathead minnow (mature), *Pimephales promelas*	>98%	-	42 days	Decreased fecundity	>3.4	Giesy et al. 2000

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Fathead minnow (mature), *Pimephales promelas*	>98%	-	42 days	Increased ♂ vitellogenin	>3.4	Giesy et al. 2000
Fathead minnow (mature), *Pimephales promelas*	>98%	-	42 days	Increased ♂ & ♀ 17β-estradiol	>0.05 (not all test concentrations)	Giesy et al. 2000
Bluegill (juvenile), *Lepomis macrochirus*	-	7.0	2 hr	LT100	5,000	Wood 1953
Bluegill (juvenile), *Lepomis macrochiru*	-	7.5-8.2	14 hr	LT100	5,000	Applegate et al. 1957
Bluegill (4-wk old), *Lepomis macrochirus*	99%	7.79	4 days	LC50 (fed)	135	Brooke 1993b
Bluegill (4-wk old), *Lepomis macrochirus*	99%	7.55	28 days	BCF=35.31	126	Brooke 1993b
Bluegill (juvenile), *Lepomis macrochirus*	96.4%	7.7 - 7.9	20 days	NOEC LOEC (survival)	76 243	Liber et al. 1999
Southern platyfish (adult, 0.62 to 1.15 g), *Xiphophorus maculatus*	Technical 85%	-	28 days	Reduced GSI	960	Kinnberg et al. 2000
Green Swordtail (adult males), *Xiphophorus helleri*	Technical	-	96 hr 72 hr	LC50 Vitellogenin induced	206 4	Kwak et al. 2001

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Green Swordtail (juvenile 30-d-old males), *Xiphophorus helleri*	Technical	-	60 days	Reduced sword length	0.2	Kwak et al. 2001
African clawed frog (larva), *Xenopus laevis*	ACS Grade	7.8 - 8.0	21 days	NOEC LOEC (increased rate of tail resorption)	25 50	Fort and Stover 1997
African clawed frog (larva), *Xenopus laevis*	-	-	12 wk	Increased female phenotypes	22	Kloas et al. 1999
			SALTWATER SPECIES			
Red alga, *Champia parvula*	>95%	-	2 days	No effect on sexual reproduction	167	Tagliabue 1993
Barnacle (cypris larva), *Balanus amphitrite*	-	-	48 h	Reduced cyprid settlement	1.0	Billinghurst et al. 1998
Soft-shell clam, *Mya arenaria*	-	-	360 hr	No mortality	700	McLeese et al. 1980b
Coot clam, *Mulinia lateralis*	90%	30-31[a]	24 hr	LC50	50	Lussier et al. 2000
Coot clam, *Mulinia lateralis*	90%	30-31[a]	48 hr	LC50	50	Lussier et al. 2000
Coot clam, *Mulinia lateralis*	90%	30-31[a]	72 hr	LC50	40	Lussier et al. 2000
Blue mussel, *Mytilus edulis*	90%	30-31[a]	96 hr	LC50	3,000	Granmo et al. 1989
Blue mussel, *Mytilus edulis*	-	32[a]	360 hr	LC50	500	Granmo et al. 1989
Blue mussel, *Mytilus edulis*	-	32[a]	13 days	Reduced byssus strength	56	Granmo et al. 1989
Blue mussel, *Mytilus edulis*	-	32[a]	30 days	Reduced byssus strength	56	Granmo et al. 1989

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Blue mussel, *Mytilus edulis*	-	32[a]	30 days	No byssus threads formed	100	Granmo et al. 1989
Blue mussel, *Mytilus edulis*	-	32[a]	32 days	Reduction in growth	56	Granmo et al. 1989
Blue mussel, *Mytilus edulis*	-	32[a]	24 hr	No effect on fertilization	200	Granmo et al. 1989
Blue mussel, *Mytilus edulis*	-		72 hr	No effect on development	200	Granmo et al. 1989
Blue mussel (40-50 mm length), *Mytilus edulis*	-	-	50 days	BCF = 350	40	Granmo et al. 1991a,b
Blue mussel, *Mytilus edulis galloprovincialis*	-	-	2 days	Repelled attachment	22	Etoh et al. 1997
Mysid, *Americamysis bahia*	90%	30-31[a]	24 hr	LC50	~114	Lussier et al. 2000
Mysid, *Americamysis bahia*	90%	30-31[a]	48 hr	LC50	~82	Lussier et al. 2000
Mysid, *Americamysis bahia*	90%	30-31[a]	72 hr	LC50	~66	Lussier et al. 2000
Mysid, *Americamysis bahia*	90%	30-31[a]	120 hr	LC50	~60	Lussier et al. 2000
Mysid, *Americamysis bahia*	90%	30-31[a]	144 hr	LC50	~60	Lussier et al. 2000
Mysid, *Americamysis bahia*	90%	30-31[a]	168 hr	LC50	~60	Lussier et al. 2000
Mysid, *Americamysis bahia*	>95%	20[a]	24 hr	LC50	>47	Ward and Boeri 1990a

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Mysid, *Americamysis bahia*	>95%	20[a]	48 hr	LC50	>47	Ward and Boeri 1990a
Mysid, *Americamysis bahia*	>95%	20[a]	72 hr	LC50	44	Ward and Boeri 1990a
Pacific Oyster (embryo-larva), *Crassostrea gigas*	-	35[a]	72 hr	Delayed development to D-shape stage	100	Nice et al. 2000
Copepod (10-12 d), *Acartia tonsa*	-	18[a]	48 hr	LC50 synthetic media	360 280	Kusk and Wollenberger 1999
Amphipod, *Leptocheirus plumulosus*	90%	30-31[a]	48 hr	LC50	~160	Lussier et al. 2000
Amphipod, *Leptocheirus plumulosus*	90%	30-31[a]	72 hr	LC50	~80	Lussier et al. 2000
Amphipod, *Leptocheirus plumulosus*	90%	30-31[a]	120 hr	LC50	~50	Lussier et al. 2000
Amphipod, *Leptocheirus plumulosus*	90%	30-31[a]	144 hr	LC50	~40	Lussier et al. 2000
Amphipod, *Leptocheirus plumulosus*	90%	30-31[a]	168 hr	LC50	~30	Lussier et al. 2000
Grass shrimp, *Palaemonetes vulgaris*	90%	30-31[a]	24 hr	LC50	~125	Lussier et al. 2000
Grass shrimp, *Palaemonetes vulgaris*	90%	30-31[a]	48 hr	LC50	~60	Lussier et al. 2000
Grass shrimp, *Palaemonetes vulgaris*	90%	30-31[a]	72 hr	LC50	~60	Lussier et al. 2000
Grass shrimp, *Palaemonetes vulgaris*	90%	30-31[a]	120 hr	LC50	~60	Lussier et al. 2000

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Shrimp, *Crangon septemspinosa*	>95%	-	96 hr	LC50	300	McLeese et al. 1980b
Shrimp, *Crangon septemspinosa*	>95%	-	96 hr	LC50	300	McLeese et al. 1980b
Shrimp, *Crangon septemspinosa*	>95%	-	96 hr	LC50	300	McLeese et al. 1980b
American lobster, *Homarus americanus*	90%	30-31[a]	24 hr	LC50	~140	Lussier et al. 2000
American lobster, *Homarus americanus*	90%	30-31[a]	48 hr	LC50	~140	Lussier et al. 2000
American lobster, *Homarus americanus*	90%	30-31[a]	72 hr	LC50	~140	Lussier et al. 2000
American lobster, *Homarus americanus*	>95%	-	96 hr	LC50	170	McLeese et al. 1980b
Atlantic salmon, *Salmo salar*	-	-	96 hr	LC50	190	McLeese et al. 1980b
Atlantic salmon, *Salmo salar*	-	-	96 hr	LC50	160	McLeese et al. 1980b
Atlantic salmon, *Salmo salar*	-	-	96 hr	LC50	130	McLeese et al. 1980b
Atlantic salmon, *Salmo salar*	-	-	96 hr	LC50	900	McLeese et al. 1980b
Sheepshead minnow, *Cyprinodon variegatu*	90%	30-31[a]	72 hr	LC50	~150	Lussier et al. 2000
Sheepshead minnow, *Cyprinodon variegatu*	90%	30-31[a]	120 hr	LC50	~125	Lussier et al. 2000

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Sheepshead minnow, *Cyprinodon variegatu*	90%	30-31[a]	144 hr	LC50	~120	Lussier et al. 2000
Sheepshead minnow, *Cyprinodon variegatu*	90%	30-31[a]	168 hr	LC50	~120	Lussier et al. 2000
Sheepshead minnow, *Cyprinodon variegatu*	>95%	15-17[a]	24 hr	LC50	>420	Ward and Boeri 1990c
Sheepshead minnow, *Cyprinodon variegatu*	>95%	15-17[a]	48 hr	LC50	340	Ward and Boeri 1990c
Sheepshead minnow, *Cyprinodon variegatu*	>95%	15-17[a]	72 hr	LC50	320	Ward and Boeri 1990c
Killifish (embryo), *Fundulus heteroclitus*	85 - 90% (technical)	20[a]	10 days	100% abnormal development	2,204	Kelly and Di Giulio 2000
Killifish (embryo), *Fundulus heteroclitus*	85 - 90% (technical)	20[a]	96 hr	LC50	5,444	Kelly and Di Giulio 2000
Killifish (1-day old larva), *Fundulus heteroclitus*	85 - 90% (technical)	20[a]	96 hr	LC50 (fed)	214	Kelly and Di Giulio 2000
Killifish (14-day old larva), *Fundulus heteroclitus*	85 - 90% (technical)	20[a]	96 hr	LC50 (fed)	209	Kelly and Di Giulio 2000
Killifish (28-day old larva), *Fundulus heteroclitus*	85 - 90% (technical)	20[a]	96 hr	LC50 (fed)	260	Kelly and Di Giulio 2000

Table 6. Other Data on Effects of Nonylphenol on Aquatic Organisms

Species	Chemical	pH	Duration	Effect	Concentration (µg/L)	Reference
Threespine stickleback *Gasterosteus aculeatus*	Commercial (para-substituted with branched nonyl chain)	32[a]	96 hr	LC50	370	Granmo et al. 1991a
Inland silversides, *Menidia beryllina*	90%	30-31[a]	24 hr	LC50	~120	Lussier et al. 2000
Inland silversides, *Menidia beryllina*	90%	30-31[a]	48 hr	LC50	~100	Lussier et al. 2000
Inland silversides, *Menidia beryllina*	90%	30-31[a]	72 hr	LC50	~80	Lussier et al. 2000
Inland silversides, *Menidia beryllina*	90%	30-31[a]	120 hr	LC50	~60	Lussier et al. 2000
Inland silversides, *Menidia beryllina*	90%	30-31[a]	144 hr	LC50	~60	Lussier et al. 2000
Inland silversides, *Menidia beryllina*	90%	30-31[a]	168 hr	LC50	~60	Lussier et al. 2000

[a]Salinity (g/kg).

11. REFERENCES

Ahel, M. and W. Giger. 1993. Aqueous solubility of alkylphenols and alkylphenol polyethoxylates. Chemosphere 26:1461-1470.

Ahel, M., J. McEvoy and W. Giger. 1993. Bioaccumulation of the lipophilic metabolites of nonionic surfactants in freshwater organisms. Environ. Pollut. 79:243-248.

Ahel, M., W. Giger and M. Koch. 1994a. Behaviour of alkylphenol polyethoxylate surfactants in the aquatic environment–I. Occurrence and transformation in sewage treatment. Wat. Res. 28:1131-1142.

Ahel, M., W. Giger and C. Schaffner. 1994b. Behavior of alkylphenol polyethoxylate surfactants in the aquatic environment–II. Occurrence and transformation in rivers. Wat. Res. 28:1143-1152.

Ahel, M., F.E. Scully, Jr., J. Hoigne and W. Giger. 1994c. Photochemical degradation of nonylphenol and nonylphenol polyethoxylates in natural waters. Chemosphere 28:1361-1368.

Ahel, M., C. Schaffner and W. Giger. 1996. Behaviour of alkylphenol polyethoxylates surfactants in the aquatic environment–III. Occurrence and elimination of their persistent metabolites during infiltration of river water to groundwater. Wat. Res. 30:37-46.

Ahel, M., E. Molnar, S. Ibric and W. Giger. 2000. Estrogenic metabolites of alkylphenol polyethoxylates in secondary sewage effluents and rivers. Wat. Sci. Tech. 42:15-22.

Allen, Y., A.P. Scott, P. Matthiessen, S. Haworth, J.E. Thain and S. Feist. 1999. Survey of estrogenic activity in United Kingdom estuarine and coastal waters and its effects on gonadal development of the flounder *Platichthys flesus*. Environ. Toxicol. Chem. 18:1791-1800.

Amato, J.R. and D.D. Wayment. 1998. Surfactant toxicity identification with a municipal wastewater. In: Environmental Toxicology and Risk Assessment. Little, E.S., A.J. DeLoney and B.M. Greenberg, Eds. ASTM STP 1333, Vol. 7, American Society for Testing and Materials, Philadelphia, PA. pp. 272-283.

Andersen, H.R., A. Andersson, S.F. Arnold, H. Autrup, M. Barfoed, N.A. Beresford, P. Bjerregaard, L.B. Christiansen, B. Gissel, R. Hummel, E.B. Jorgensen, B. Korsgaard, R. Le Guevel, H. Leffers, J. McLachlan, A. Moller, J.B. Nielsen, N. Olea, A. Oles-Karasko, F. Pakdel, K.L. Pedersen, P. Perez, N.E. Skakkeboek, C. Sonnenschein, A.M. Soto, J.P. Sumpter, S.M. Thorpe and P. Grandjean. 1999. Comparison of short-term

estrogenicity tests for identification of hormone-disrupting chemicals. Environ. Health Perspect. 107(Suppl. 1):89-108.

Applegate, V.C., J.H. Howell, A.E. Hall, Jr. and M.A. Smith. 1957. Toxicity of 4,346 chemicals to larval lampreys and fishes. Special Scientific Report-Fisheries, No. 207, U.S. Dept. Interior, Fish and Wildlife Service, Washington, DC. 157 pp.

Arukwe, A., L. Forlin and A. Goksoyr. 1997a. Xenobiotic and steroid biotransformation enzymes in Atlantic salmon (*Salmo salar*) liver treated with an estrogenic compound, 4-nonylphenol. Environ. Toxicol. Chem. 16:2576-2583.

Arukwe, A., F.R. Knudsen and A. Goksoyr. 1997b. Fish zona radiata (eggshell) protein: A sensitive biomarker for environmental estrogens. Environ. Health Perspect. 105:418-422.

Arukwe, A., T. Celius, B.T. Walther and A. Goksoyr. 1998. Plasma levels of vitellogenin and eggshell *zona radiata* proteins in 4-nonylphenol and *o,p*' nonylphenol treated juvenile Atlantic salmon (*Salmo salar*). Mar. Environ. Res. 46:133-136.

Arukwe, A., R. Thibaut, K. Ingebrigtsen, T. Celius, A. Goksoyr and J-P. Cravedi. 2000. *In vivo* and *in vitro* metabolism and organ distribution of nonylphenol in Atlantic salmon (*Salmo salar*). Aquat. Toxicol. 49:289-304.

Ashfield, L.A., T.G. Pottinger and J.P. Sumpter. 1998. Exposure of female juvenile rainbow trout to alkylphenolic compounds results in modifications to growth and ovosomatic index. Environ. Toxicol. Chem. 17:679-686.

Baer, K.N. and K.D. Owens. 1999. Evaluation of selected endocrine disrupting compounds on sex determination in *Daphnia magna* using reduced photoperiod and different feeding rates. Bull. Environ. Contam. Toxicol. 62:214-221.

Baldwin, W.S., S.E. Graham, D. Shea and G.A. LeBlanc. 1997. Metabolic androgenization of female *Daphnia magna* by the xenoestrogen 4-nonylphenol. Environ. Toxicol. Chem. 16:1905-1911.

Baldwin, W.S., S.E. Graham, D. Shea and G.A. Leblanc. 1998. Altered metabolic elimination of testosterone and associated toxicity following exposure to *Daphnia magna* to nonylphenol polyethoxylate. Ecotoxicol. Environ. Saf. 39:104-111.

Bearden, A.P. and T.W. Schultz. 1997. Structure-activity relationships for *Pimephales* and *Tetrahymena*: a mechanism of action approach. Environ. Toxicol. Chem. 16:1311-1317.

Bearden, A.P. and T.W. Schultz. 1998. Comparison of *Tetrahymena* and *Pimephales* toxicity based on mechanism of action. SAR QSAR Environ. Res. 9:127-153.

Bechmann, R.K. 1999. Effect of the endocrine disrupter nonylphenol on the marine copepod *Tisbe battagliai.* Sci. Total Environ. 233:33-46.

Bennett, E.R. and C.D. Metcalfe. 1998. Distribution of alkylphenol compounds in Great Lakes sediments, United States and Canada. Environ. Toxicol. Chem. 17:1230-1235.

Bennet, E.R. and C.D. Metcalfe. 2000. Distribution of degradation products of alkylphenol ethoxylates near sewage treatment plants in the Lower Great Lakes, North America.. Environ. Toxicol. Chem. 19: 784-792.

Bennie, D.T. 1999. Review of the environmental occurrence of alkylphenols and alkylphenol ethoxylates. Wat. Qual. Res. J. Can. 34:79-122.

Bennie, D.T., C.A. Sullivan, H. Lee, T.E. Peart and R.J. Maguire. 1997. Occurrence of alkylphenols and alkylphenol mono- and di-ethoxylates in natural waters of the Laurentian Great Lakes basin and the upper St. Lawrence River. Sci. Total Environ. 193:263-275.

Bettinetti, R. and A. Provini. 2002. Toxicity of 4-nonylphenol to *Tubifex tubifex* and *Chironomus riparius* in 28-day whole-sediment tests. Ecotoxicol. Environ. Safety 53:113-121.

Billinghurst, Z., A.S. Clare, T. Fileman, M. McEvoy, J. Readman and M.H. Depledge. 1998. Inhibition of barnacle settlement by the environmental oestrogen 4-nonylphenol and the natural oestrogen 17β oestradiol. Mar. Pollut. Bull. 36:833-839.

Blackburn, M.A., S.J. Kirby and M.J. Waldock. 1999. Concentrations of alkyphenol polyethoxylates entering UK estuaries. Mar. Pollut. Bull. 38:109-118.

Braaten, B., A. Granmo and R. Lange. 1972. Tissue-swelling in *Mytilus edulis* L. induced by exposure to a nonionic surface active agent. Norw. J. Zool. 20:137-140.

Brooke, L.T. 1993a. Acute and chronic toxicity of nonylphenol to ten species of aquatic organisms. Report to the U.S. EPA for Work Assignment No. 02 of Contract No. 68-C1-0034. Lake Superior Research Institute, University of Wisconsin-Superior, Superior, WI. March 24. 30 pp. Amended 18 October 2005. 34 pp.

Brooke, L.T. 1993b. Nonylphenol Toxicity. Accumulation and lethality for two freshwater fishes (fathead minnow and bluegill) to nonylphenol. Report to the U.S. EPA for Work Assignment No. 1-12 of Contract No. 68-C1-0034. Lake Superior Research Institute, University of Wisconsin-Superior, Superior, WI. September 30. 50 pp.

Brooke, L.T. 1994. Nonylphenol Analysis. Accumulation and lethality for two freshwater fishes (fathead minnow and bluegill) to nonylphenol. Report to the U.S. EPA for Work Assignment No. 2-15 of Contract No. 68-C1-0034. Lake Superior Research Institute, University of Wisconsin-Superior, Superior, WI. May 31. 49 pp.

Brown, R.J., M. Conradi and M.H. Depledge. 1999. Long-term exposure to 4-nonylphenol affects sexual differentiation and growth of the amphipod *Corophium volutator* (Pallas, 1766). Sci. Total Environ. 233:77-88.

Budavari, S. (Ed.). 1989. The Merck Index: An encyclopedia of chemicals, drugs, and biologicals. 11th ed. Merck and Co., Inc. Rahway, NJ.

Burkhardt-Holm, P., T. Wahli and W. Meier. 2000. Nonylphenol affects the granulation of pattern of epidermal mucous cells in rainbow trout, *Oncorhynchus mykiss*. Ecotoxicol. Environ. Safe. 46:34-40.

Celius, T., T.B. Haugen, T. Grotmol and B.T. Walther. 1999. A sensitive zonagenetic assay for rapid *in vitro* assessment of estrogenic potency of xenobiotics and mycotoxins. Environ. Health Perspect. 107:63-68.

Christiansen, T., B. Korsgaard and A. Jespersen. 1998a. Effects of nonylphenol and 17β-estradiol on vitellogenin synthesis, testicular structure and cytology in male eelpout *Zoarces viviparus*. J. Exp. Biol. 201:179-192.

Christiansen, T., B. Korsgaard and A. Jespersen. 1998b. Induction of vitellogenin synthesis by nonylphenol and 17β-estradiol and effects on the testicular structure in the eelpout *Zoarces viviparus*. Mar. Environ. Res. 46:141-144.

Christiansen, T., K.L. Pedersen, B. Korsgaard and P. Bjerregaard. 1998c. Estrogenicity of xenobiotics in rainbow trout (*Oncorhynchus mykiss*) using *in vivo* synthesis of vitellogenin as a biomarker. Mar. Environ. Res. 46(1-5): 137-140.

Christensen, L.J., B. Korsgaard and P. Bjerregaard. 1999. The effect of 4-nonylphenol on the synthesis of vitellogenin in the flounder *Platichthys flesus*. Aquat. Toxicol. 46:211-219.

Coldham, N.G., M.J. Sauer, S. Sivapathasundaram, L. Ashfield, T. Pottinger and C. Goodall. 1997. Tissue distribution, metabolism and excretion of 4-nonylphenol in rainbow trout. J. Vet. Pharmacol. Therap. 20(Suppl. 1): 256-257.

Coldham, N.G., S. Sivapathasundaram, M. Dave, L.A. Ashfield, T.G. Pottinger, C. Goodall and M.J. Sauer. 1998. Biotransformation, tissue distribution, and persistence of 4-nonylphenol residues in juvenile rainbow trout (*Oncorhynchus mykiss*). Drug Metabol. Dispos. 26:347-354.

Comber, M.H.I., T.D. Williams and K.M. Stewart. 1993. The effects of nonylphenol on *Daphnia magna*. Wat. Res. 27:273-276.

Corsi, S.R., D.H. Zitomer, J.A. Field and D.A. Cancilla. 2003. Nonylphenol ethoxylates and other additives in aircraft deicers, antiicers, and waters receiving airport runoff. Environ. Sci. Technol. 37:4031-4037.

Dorn, P.B., J.P. Salanitro, S.H. Evans and L. Kravetz. 1993. Assessing the aquatic hazard of some branched and linear nonionic surfactants by biodegradation and toxicity. Environ. Toxicol. Chem. 12:1751-1762.

Dwyer, F.J., L.C. Sappington, D.R. Buckler and S.B. Jones. 1995. Use of surrogate species in assessing contaminant risk to endangered and threatened species. EPA/600/R-96/029. National Technical Information Service, Springfield, VA. 71 pp.

Dwyer, F.J., D.K. Hardesty, C.E. Henke, C.G. Ingersoll, D.W. Whites, D.R. Mount and C.M. Bridges. 1999a. Assessing contaminant sensitivity of endandered and threatened species: Toxicant classes. EPA/600/R-99/098. National Technical Information Service, Springfield, VA. 15 pp.

Dwyer, F.J., D.K. Hardesty, C.E. Henke, C.G. Ingersoll, D.W. Whites, D.R. Mount and C.M. Bridges. 1999b. Assessing contaminant sensitivity of endangered and threatened species: Effluent toxicity tests. EPA/600/R-99/099. National Technical Information Service, Springfield, VA. 9 pp.

Ekelund, R., A. Bergman, A. Granmo, and M. Berggren. 1990. Bioaccumulation of 4-nonylphenol in marine animals--A re-evaluation. Environ. Poll. 64: 107-120.

Ekelund, R., A. Granmo, K. Magnusson and M. Berggren. 1993. Biodegradation of 4-nonylphenol in seawater and sediment. Environ. Pollut. 79:59-61.

Ellis, D.D., C.M. Jone, R.A. Larson and D.J. Schaeffer. 1982. Organic constituents of mutagenic secondary effluents from wastewater treatment plants. Arch. Environ. Contam. Toxicol. 11:373-382.

England, D.E. 1995. Chronic toxicity of nonylphenol to *Ceriodaphnia dubia*. Report No. 41756. ABC Laboratories, Inc. Columbia, MO. 409 pp.

England, D.E. and J.B. Bussard. 1993. Toxicity of nonylphenol to the midge *Chironomus tentans*. Report No. 40597. ABC Laboratories, Inc., Columbia, MO. 2528 pp.

England, D.E. and J.B. Bussard. 1995. Toxicity of nonylphenol to the amphipod *Hyalella azteca*. Report No. 41569. ABC Laboratories, Inc., Columbia, MO. 178 pp.

Ernst, B., G. Julien, K. Doe and R. Parker. 1980. Environmental investigations of the 1980 spruce budworm spray program in New Brunswick. Surveillance Report EPS-5-AR-81-3, November 1980. Environment Canada, Atlantic Region, Halifax, NS.

Escher, M., T. Wahli, S. Buttner, W. Meier and P. Burkhardt-Holm. 1999. The effect of sewage plant effluent on brown trout (*Salmo trutta fario*): a cage experiment. Aquat. Sci. 61:93-110.

Etoh, H., S. Hageshita and K. Ina. 1997. An improved assay for attachment-promoting substances of the blue mussel, *Mytilus edulis galloprovincialis*. J. Mar. Biotechnol. 5:24-26.

Fay, A.A., F.J. Brownawell, A.A. Elskus and A.E. McElroy. 2000. Critical body residues in the marine amphipod *Ampelisca abdita*: Sediment exposures with nonionic organic contaminants. Environ. Toxicol. Chem. 19:1028-1035.

Ferguson, P.L. and B.J. Brownawell. 2003. Degradation of nonylphenol ethoxylates in estuarine sediment under aerobic and anaerobic conditions. Environ. Toxicol. Chem. 22:1189-1199.

Fliedner, A. 1993. *Daphnia magna*, Reproduction test (OECD No. 202). Fraunhofer–Institute fur Umweltchemie und Okotoxikologie, Postfach 1260, W–5948 Schmallenberg – Grafschaft, Germany. Report No. UBA–002/4–22 February.

Flouriot, G., F. Pakdel, B. Ducouret and Y. Valotaire. 1995. Influence of xenobiotics on rainbow trout liver estrogen receptor and vitellogenin gene expression. J. Molecular Endocrin. 15:143-151.

Folmar, L., M. Hemmer, N. Denslow, K. Kroll, A. Cheek, H. Meredith, H. Richman and G. Grau. 1998. A comparison of an *in vivo* fish VTG assay with YES and E-screen to rank 'relative estrogenicity' of natural pharmaceutical and xenoestrogens. Am. Zool. 38:112A.

Fort, D.J. and E.L. Stover. 1997. Development of short-term, whole-embryo assays to evaluate detrimental effects on amphibian limb development and metamorphosis using *Xenopus laevis*. In: Environmental Toxicology and Risk Assessment: Modeling and Risk Assessment. F.J. Dwyer, T.R. Doane and M.L. Hinman (Eds.). ASTM, STP 1317, Vol. 6, American Society for Testing and Materials, Philadelphia, PA., pp. 376-390.

Gaffney, P.E. 1976. Carpet and rug industry case study II: Biological effects. J. Water Pollut. Control Fed. 48:2731-2737.

Gerritsen, A., N. van der Hoeven and A. Pielaat. 1998. The acute toxicity of selected alkylphenols to young and adult *Daphnia magna*. Ecotoxicol. Environ. Safety. 39:227-232.

Giesy, J.P., S.L. Pierens, E.M. Snyder, S. Miles-Richardson, V.J. Kramer, S.A. Snyder, K.M. Nichols and D.A. Villeneuve. 2000. Effects of 4-nonylphenol on fecundity of biomarkers of estrogenicity in fathead minnows (*Pimephales promelas*). Environ. Toxicol. Chem. 19:1368-1377.

Giger, W., E. Stephanou and C. Schaffner. 1981. Persistent organic chemicals in sewage effluents: I. Identifications of nonylphenols and nonylphenol ethoxylates by glass capillary gas chromatography/ mass spectrometry. Chemosphere 10:1253-1263.

Giger, W., P.H. Brunner and C. Schaffner. 1984. 4-Nonylphenol in sewage sludge; Accumulation of toxic metabolites from nonionic surfactants. Science 225:623-625.

Granmo, A., R. Ekelund, K. Magnusson and M. Berggren. 1989. Lethal and sublethal toxicity of 4-nonylphenol to the common mussel (*Mytilus edulis* L.). Environ. Poll.59: 115-127.

Granmo, A., R. Ekelund, M. Berggren and K. Magnusson. 1991a. Toxicity of 4-nonylphenol to aquatic organisms and potential for bioaccumulation. Proceedings, Swedish Environmental Protection Agency Seminar on Nonylphenol Ethoxylates/nonylphenol, Saltsjobaden, Sweden, February 6-8, pp. 53-75.

Granmo, A., S. Kollberg, M. Berggren, R. Ekelund, K. Magnusson, L. Renberg and C. Wahlberg. 1991b. Bioaccumulation of nonylphenol in caged mussels in an industrial coastal area on the Swedish west coast. In: Organic Micropollutants in the Aquatic Environment. Angeletti, G. (Ed.). Proceedings of the 6th European Symposium. Pp. 71-79.

Gray, M.A. and C.D. Metcalfe. 1997. Induction of testis-ova in Japanese medaka (*Oryzias latipes*) exposed to *p*-nonylphenol. Environ. Toxicol. Chem. 16:1082-1086.

Gross-Sorokin, M.Y., E.P.M. Grist, M. Cooke and M. Crane. 2003. Uptake and depuration of 4-nonylphenol by the benthic invertebrate *Gammarus pulex*: How important is feeding rate? Environ. Sci. Technol. 37:2236-2241.

Hahn, T., K. Schenk and R. Schulz. 2002. Environmental chemicals with known endocrine potential affect yolk protein content in the aquatic insect *Chironomus riparius*. Environ. Pollut. 120:525-528.

Hale, R.C., C.L. Smith, P.O. de Fur, E. Harvey, E.O. Bush, M.J. La Guardia and G.G. Vadas. 2000. Nonylphenols in sediments and effluents associated with diverse wastewater outfalls. Environ. Toxicol. Chem. 19:946-952.

Hansen, F.T., V.E. Forbes and T.L. Forbes. 1999. Effects of 4-n-nonylphenol on life-history traits and population dynamics of a polychaete. Ecol. Appl. 9:482-495.

Hansen, P., H. Dizer, B. Hock, A. Marx, J. Sherry, M. McMaster and C. Blaise. 1998. Vitellogenin - a biomarker for endocrine disruptors. Trends Anal. Chem. 17:448-451.

Harries, J.E., D.A. Sheahan, S. Jobling, P. Matthiessen, P. Neall, J.P. Sumpter, T. Tylor and N. Zaman. 1997. Estrogenic activity in five United Kingdom rivers detected by measurement of vitellogenesis in caged male trout. Environ. Toxicol. Chem. 16:534-542.

Harris, C.A., E.M. Santos, A Janbakhsh, T.G. Pottinger, C.R. Tyler and J.P. Sumpter. 2001. Nonylphenol affects gonadotropin levels in the pituitary gland and plasma of female rainbow trout. Environ. Sci. Technol. 35:2909-2916.

Harvilicz, H. 1999. NPE demand remains strong despite environmental concerns in Europe. Chemical Market Reporter. October 18, 1999. p. 15.

Haya, K., L.E. Burridge and T.J. Benfey. 1997. The effect of cortisol and nonylphenol on growth and ornithine decarboxylase activity of juvenile Atlantic salmon, *Salmo salar*. Can. Tech. Rep. Fish. Aquat. Sci. 2192: 85-86.

Hecht, S. and B.L. Boese. 2002. Sensitivity of an infaunal amphipod, *Eohaustorius estuarius*, to acute waterborne exposures of 4-nonylphenol: Evidence of a toxic hangover. Environ. Toxicol. Chem. 21:816-819.

Hecht, S.A., J.S. Gunnarsson, B.L. Boese, J.O. Lamberson, C. Schaffner, W. Giger and P.C. Jepson. 2004. Influences of sedimentary organic matter quality on the bioaccumulation of 4-nonylphenol by estuarine amphipods. Environ. Toxicol. Chem. 23:865-873.

Heinis, L.J., M.L. Knuth, K. Liber, B.R. Sheedy, R.L. Tunell and G.T. Ankley. 1999. Persistence and distribution of 4-nonylphenol following repeated application to littoral enclosures. Environ. Toxicol. Chem. 18:363-375.

Hemmer, M.J., B.L. Hemmer, C.J. Bowman, K.J. Kroll, L.C. Folmer, D. Marcovich, M.D. Hoglund and N.D. Denstow. 2001. Effects of p-nonylphenol, methoxychlor and endosulfan on vitellogenin induction and expression in sheepshead minnow (*Cyprindon variegatus*). Environ. Toxicol. Chem. 20:336-343.

Hense, B.A., I. Juttner, G. Welzl, G.F. Severin, G. Pfister, A. Behechti and K-W. Schramm. 2003. Effects of 4-nonylphenol on phytoplankton and periphyton in aquatic microcosms. Environ. Toxicol. Chem. 22:2727-2732.

Hewitt, L.M., L. Tremblay, G.J. Van Der Kraak, K.R. Solomon and M.R. Servos. 1998. Identification of the lampricide 3-trifluoromethyl-4-nitrophenol as an agonist for the rainbow trout estrogen receptor. Environ. Toxicol. Chem. 17:425-432.

Holcombe, G.W., G.L. Phipps, M.L. Knuth and T. Felhaber. 1984. The acute toxicity of selected substituted phenols, benzenes and benzoic acid esters to fathead minnows (*Pimephales promelas*). Environ. Pollut. (Series A) 35:367-381.

Holmes, S. B. and P. D. Kingsbury. 1980. The environmental impact of nonylphenol and the Matacil® formulation. Part 1: Aquatic ecosystems. Report FPMX-35. Forest Pest Management Institute, Canadian Forestry Service, Sault Ste. Marie, Ontario.

Islinger, M., S. Pawlowski, H. Hollert, A. Volkl and T. Braumbeck. 1999. Measurement of vitellogenin-mRNA expression in primary cultures of rainbow trout hepatocytes in a non-radioactive dot blot/RNAse protection-assay. Sci. Total Environ. 233:109-122.

Jobling, S. and J.P. Sumpter. 1993. Detergent components in sewage effluent are weakly oestrogenic to fish: An in vitro study using rainbow trout (*Oncorhynchus mykiss*) hepatocytes. Aquat. Toxicol. 27:361-372.

Jobling, S., D. Sheahan, J.A. Osborne, P. Matthiessen and J.P. Sumpter. 1996. Inhibition of testicular growth in rainbow trout (*Oncorhynchus mykiss*) exposed to estrogenic alkylphenolic chemicals. Environ. Toxicol. Chem. 15:194-202.

Jones, S.B., L.B. King, L.C. Sappington, F.J. Dwyer, M. Ellersieck and D.R. Buckler. 1998. Effects of carbaryl, permethrin, 4-nonylphenol, and copper on muscarinic cholinergic receptors in brain of surrogate and listed fish species. Comp. Biochem. Physiol. 120:405-414.

Kahl, M.D., E.A. Makynen, P.A. Kosian and G.T. Ankley. 1997. Toxicity of 4-nonylphenol in a life-cycle test with the midge *Chironomus tentans*. Ecotoxicol. Environ. Saf. 38:155-160.

Kannan, K., T.L. Keith, C.G. Naylor, C.A. Staples, S.A. Snyder and J.P. Giesy. 2003. Nonylphenol and nonylphenol ethoxylates in fish, sediment, and water from the Kalamazoo River, Michigan. Arch. Environ. Contam. Toxicol. 44:77-82.

Keith, T.L., S.A. Snyder, C.G. Naylor, C.A. Staples, C. Summer, K. Kannan and J.P. Giesy. 2001. Identification and quantitation of nonylphenol ethoxylates and nonylphenol in fish tissues from Michigan. Environ. Sci. Technol. 35:10-13.

Kelly, S.A. and R.T. Di Giulio. 2000. Developmental toxicity of estrogenic alkylphenols in killifish (*Fundulus heteroclitus*). Environ. Toxicol. Chem. 19:2564-2570.

Kinnberg, K., B. Korsgaard, P. Bjerregaard and A. Jespersen. 2000. Effects of nonylphenol and 17β-estradiol on vitellogenin synthesis and testis morphology in male platyfish *Xiphorphorus maculatus*. J. Exper. Biol. 203:171-181.

Kitajima, F., C.G. Satuito, H. Hirota, I. Katsuyama and N. Fusetani. 1995. A new screening method for antifouling substances against the young mussels *Mytilus edulis galloprovincialis*. Fish. Sci. 61:578-583.

Kloas, W., I. Lutz and R. Einspanier. 1999. Amphibians as a model to study endocrine disruptors: II. Estrogenic activity of environmental chemicals in vitro and in vivo. Sci. Total Environ. 225:59-68.

Knudsen, F.R. and T.G. Pottinger. 1999. Interaction of endocrine disrupting chemicals, singly and in combination, with estrogen-, androgen-, and corticosteroid-binding sites in rainbow trout (*Oncorhynchus mykiss*). Aquat. Toxicol. 44:159-170.

Kolpin, D.W., E.T. Furlong, M.T. Meyer, E.M. Thurman, S.D. Zaugg, L.B. Barber and H.T. Buxton. 2002. Pharmaceuticals, hormones, and other organic wastewater contaminants in U.S. streams, 1999 - 2000: A national reconnaissance. Environ. Sci. Technol. 36:1202-1211.

Kopf, W. 1997. Wirkung endodriner stoffe in biotests mit wasserorganismen. *In:* Stoffe mit endokriner wirkung in wasser. Bayerisches landesamt fur wasserwirt schaft, Institut fur Wasserforschung, Munchen. 12pp.

Kuhn, A., W.R. Munns, Jr., D. Champlin, R. McKinney, M. Tagliabue, J. Serbst and T. Gleason. 2001. Evaluation of the efficacy of extrapolation population modeling to predict the dynamics of *Americamysis bahia* populations in the laboratory. Environ. Toxicol. Chem. 20:213-221.

Kusk, K.O. and L. Wollenberger. 1999. Fully defined saltwater medium for cultivation of and toxicity testing with marine copepod *Acartia tonsa*. Environ. Toxicol. Chem. 18:1564-1567.

Kwak, H., M. Bae, M. Lee, Y. Lee, B. Lee, K. Kang, C. Chae, H. Sung, J. Shin, J. Kim, W. Mar, Y. Sheen and M. Cho. 2001. Effects of nonylphenol, bisphenol A, and their mixture on the viviparous swordtail fish (*Xiphophorus helleri*). Environ. Toxicol. Chem. 20:787-795.

Lamche, G. and P. Burkhardt-Holm. 2000. Nonylphenol provokes a vesiculation of the golgi apparatus in three fish epidermis cultures. Ecotoxicol. Environ. Saf. 47:137-148.

Larsson, D.G.J., M. Adolfsson-Erici, J. Parkkonen, M. Pettersson, A.H. Berg, P. Olsson and L. Forlin. 1999. Ethinyloestradiol - an undesired fish contraceptive? Aquat. Toxicol. 45:91-97.

Lavado, R., R. Thibaut, D. Raldua, R. Martin and C. Porte. 2004. First evidence of endocrine disruption in feral carp from the Ebro River. Toxicol. Appl. Pharm. 196:247-257.

LeBlanc, G.A., S. Mu and C.V. Rider. 2000. Embryotoxicity of the alkylphenol degradation product 4-nonylphenol to the crustacean *Daphnia magna*. Environ. Health Perspect. 108:1133-1138.

Lech, J. J., S. K. Lewis and L. Ren. 1996. *In vivo* estrogenic activity of nonylphenol in rainbow trout. Fund. Appl. Toxicol. 30:229-232.

Levine, C. and M.A. Cheney. 2000. Metabolic responses to acute toxicity of alkylphenols and alkylphenol polyethoxylates in *Elliptio complanata* measured by calorespirometry. Environ. Toxicol. Chem. 19:1906-1910.

Lewis, M.A. 1991. Chronic and sublethal toxicities of surfactants to aquatic animals: A review and risk assessment. Water Res. 25:101-113.

Lewis, S. K. and J. J. Lech. 1996. Uptake, disposition, and persistence of nonylphenol from water in rainbow trout (*Oncorhynchus mykiss*). Xenobiotica 26:813-819.

Liber, K., J. A. Gangl, T. D. Corry, L. J. Heinis and F. S. Stay. 1999. Lethality and bioaccumulation of 4-nonylphenol in bluegill sunfish in littoral enclosures. Environ. Toxicol. Chem. 18:394-400.

Loomis, A.K. and P. Thomas. 1999. Binding characteristics of estrogen receptor (ER) in Atlantic croaker (*Micropogonias undulatus*) testis: Different affinity for estrogens and xenobiotics from that of hepatic ER. Biol. Reprod. 61:51-60.

Lussier, S.M., D. Champlin, J. LiVolsi, S. Poucher and R.J. Pruell. 2000. Acute toxicity of *para*-nonylphenol to saltwater animals. Environ. Toxicol. Chem. 19:617-621.

Lutz, I. and W. Kloas. 1999. Amphibians as a model to study endocrine disruptors: I. Environmental pollution and estrogen receptor binding. Sci. Total Environ. 25:49-57.

Lye, C.M., C.L.J. Frid, M.E. Gill, D.W. Cooper and D.M. Jones. 1999. Estrogenic alkylphenols in fish tissues, sediments, and waters from the U.K. Tyne and Tees estuaries. Environ. Sci. Technol. 33:1009-1014.

MacPhee, C. and R. Ruelle. 1969. Lethal effects of 1888 chemicals upon four species of fish from western North America. Bulletin No. 3, Forest, Wildlife and Range Experiment Station, University of Idaho, Moscow, ID. 112 p.

Madsen, S.S., A.B. Mathiesen and B. Korsgaard. 1997. Effects of 17β-estradiol and 4-nonylphenol on smoltification and vitellogenesis in Atlantic salmon (*Salmo salar*). Fish Physiol. Biochem. 17:303-312.

Magliulo, L., M.P. Schreibman and J.L. Cepriano. 1998. Disruption of the brain-pituitary-gonad axis of platyfish due to hormone mimicking environmental pollutants. Am. Zool. 38:112A.

Maguire, R.J. 1999. Review of the persistence of nonylphenol and nonylphenol ethoxylates in aquatic environments. Wat. Qual. Res. J. Can. 34:37-78.

Maki, H., H. Okamura, I. Aoyama and M. Fujita. 1998. Halogenation and toxicity of the biodegradation products of a nonionic surfactant, nonylphenol ethoxylate. Environ. Toxicol. Chem. 17:650-654.

Manzano, M.A., J.A. Perales, D. Sales and J.M. Quiroga. 1998. Effect of concentration on the biodegradation of a nonylphenol polyethoxylate in river water. Bull. Environ. Contam. Toxicol. 61:489-496.

Manzano, M.A., J.A. Perales, D. Sales and J.M. Quiroga. 1999. The effect of temperature on the biodegradation of a nonylphenol polyethoxylate in river water. Water Res. 33:2593-2600.

Marcomini, A., B. Pavoni, A. Sfriso and A.A. Orio. 1990. Persistent metabolites of alkylphenol polyethoxylates in the marine environment. Mar. Chem. 29:307-324.

McLeese, D.W., D.B. Sargeant, C.D. Metcalfe, V. Zitko and L.E. Burridge. 1980a. Uptake and excretion of aminocarb, nonylphenol, and pesticides diluent 585 by mussels (*Mytilus edulis*). Bull. Environ. Contam. Toxicol. 24:575-581.

McLeese, D.W., V. Zitko, C.D. Metcalf and D.B. Sergeant. 1980b. Lethality of aminocarb and the components of the aminocarb formulation to juvenile Atlantic salmon, marine invertebrates and a freshwater clam. Chemosphere. 9:79-82.

McLeese, D.W., V. Zitko, D.B. Sargeant, L. Burridge and C.D. Metcalfe. 1981. Lethality and accumulation of alkylphenols in aquatic fauna. Chemosphere 10:723-730.

Meldahl, A. C., K. Nithipatikom and J. J. Lech. 1996. Metabolism of several ^{14}C-nonylphenol isomers by rainbow trout (*Oncorhynchus mykiss*): *in vivo* and *in vitro* microsomal metabolites. Xenobiotica 26:1167-1180.

Miles-Richardson, S.R., S.L. Pierens, K.M. Nichols, V.J. Kramer, E.M. Snyder, S.A. Snyder, J.A. Render, S.D. Fitzgerald and J.P. Giesy. 1999. Effects of waterborne exposure to 4-nonylphenol and nonylphenol ethoxylate on secondary sex characteristics and gonads of fathead minnows (*Pimephales promelas*). Environ. Res. (Series A) 80:S122-S137.

Milligan, S.R., O. Khan and M. Nash. 1998. Competitive binding of xenobiotic estrogens to rat alpha-fetoprotein and to sex steroid binding proteins in human and rainbow trout (*Oncorhynchus mykiss*) plasma. Gen. Comp. Endocrinol. 112:89-95.

Moore, A., A.P. Scott, N. Lower, I. Katsiadaki and L. Greenwood. 2003. The effects of 4-nonylphenol and atrazine on Atlantic salmon (*Salmo salar L.*) smolts. Aquaculture 222:253-263.

Moore, S.B., R.A. Diehl, J.M. Barnhardt and G.B. Avery. 1987. Aquatic toxicities of textile surfactants. Text. Chem. Color. 19:29-32.

Muller, R. 1980. Fish toxicity and surface tension of non-ionic surfactants: investigations of antifoam agents. J. Fish. Biol. 16:585-589.

Naylor, C.G. 1992. Environmental fate of alkylphenol ethoxylates. Soap Cosmetics Chemical Specialities. August.

Naylor, C.G., J.P. Mieure, W.J. Adams, J.A. Weeks, F.J. Castaldi, L.D. Ogle and R.R. Romano. 1992. Alkylphenol ethoxylates in the environment. J. Am. Oil Chem. Soc. 69:695-708.

Nice, H.E., M.C. Thorndyke, D. Morrittt, S. Steele and M. Crane. 2000. Development of the *Crassostrea gigas* larvae is affected by 4-nonylphenol. Mar. Pollut. Bull. 40:491-496.

Nimrod, A.C. and W.H. Benson. 1996. Estrogenic responses to xenobiotics in channel catfish (*Ictalurus punctatus*). Mar. Environ. Res. 42:155-160.

Nimrod, A.C. and W.H. Benson. 1997. Xenobiotic interaction with and alteration of channel catfish estrogen receptor. Toxicol. Appl. Pharmacol. 147:381-390.

Nimrod, A.C. and W.H. Benson. 1998. Reproduction and development of Japanese medaka following an early life stage exposure to xenoestrogens. Aquat. Toxicol. 44: 141-156.

O'Halloran, S. L., K. Liber, J. A. Gangl and M. L. Knuth. 1999. Effects of repeated exposure to 4-nonylphenol on the zooplankton community in littoral enclosures. Environ. Toxicol. Chem. 18:376-385.

Palmer, B.D., S.K. Palmer, C. Burch, J. Danyo and K.W. Selcer. 1998. Effects of endocrine disrupting chemicals on amphibians. Am. Zool. 38(5): Abstract No. 177A.

Patoczka, J. and G.W. Pulliam. 1990. Biodegradation and secondary effluent toxicity of ethoxylated surfactants. Water Res. 24:965-972.

Pedersen, S.N., L.B. Christiansen, K.L. Pedersen, B. Korsgaard and P. Bjerregaard. 1999. In vivo estrogenic activity of branched and linear alkylphenols in rainbow trout (*Oncorhynchus mykiss*). Sci. Tot. Environ. 233:89-96.

Petit, F., P. Le Goff, J.P. Cravedi, Y. Valotaire and F. Pakdel. 1997. Two complementary bioassays for screening the estrogenic potency of xenobiotics: recombinant yeast for trout estrogen receptor and trout hepatocyte cultures. J. Mol. Endocrinol. 19:321-335.

Petit, F., P. Le Goff, J.P. Cravedi, O. Kah, Y. Valotaire and F. Pakdel. 1999. Trout estrogen receptor sensitivity to xenobiotics as tested by different bioassays. Aquaculture. 177:353-365.

Pickford, K.A., R.E. Thomas-Jones, B. Wheals, C.R. Tyler and J.P. Sumpter. 2003. Route of exposure affects the oestrogenic response of fish to 4-*tert*-nonylphenol. Aq. Toxicol. 65:267-279.

Prasad, R. 1986. Effects of nonylphenol adjuvant on macrophytes. In: Adjuvants and Agrochemicals. Chow, P.N.P. (Ed.). Vol. 1, Mode of action and physiological activity. First International Symposium. Brandon, Manitoba, Canada. August 5-7. XIV+207 pp.

Preston, B.L., T.W. Snell, T.L. Robertson and B.J. Dingmann. 2000. Use of freshwater rotifer *Brachionus calyciflorus* in screening assay for potential endocrine disruptors. Environ. Toxicol. Chem. 19:2923-2928.

Purdom, C.E., P.A. Hardiman, V.J. Bye, N.C. Eno, C.R. Tyler and J.P. Sumpter. 1994. Estrogenic effects of effluents from sewage treatment works. Chem. and Ecol. 8:275-285.

Radian Corp. 1990. Nonylphenol and nonylphenol ethoxylates in river water and bottom sediments-January 1989-August 1990. Final Report to Alkylphenol and Ethoxylates Panel, Chemical Manufacturers Association.

Reed, H.W.B. 1978. Alkylphenols. In: Kirk-Othmer: Encyclopedia of chemical technology. Grayson M. and EcKroth D. (Eds.). 3rd ed., Vol. 2, John Wiley and Sons, New York. pp. 72-96.

Ren, L., D. Lattier and J. J. Lech. 1996a. Estrogenic activity in rainbow trout determined with a new cDNA probe for vitellogenesis, pSG5Vg1.1. Bull. Environ. Contam. Toxicol. 56:287-294.

Ren, L., S.K. Lewis and J.J. Lech. 1996b. Effects of estrogen and nonylphenol on the post-transcriptional regulation of vitellogenin gene expression. Chemico-Biol. Interact. 100:67-76.

Rice, C.D., L.E. Roszell, M.M. Banes and R.E. Arnold. 1998. Effects of dietary PCBs and nonyl-phenol on immune function and CYP1A activity in channel catfish, *Ictalurus punctatus*. Mar. Environ. Res. 46:351-354.

Routledge, E.J. and J.P. Sumpter. 1996. Estrogenic activity of surfactants and some of their degradation products assessed using a recombinant yeast screen. Environ. Toxicol. Chem. 15:241-248.

Routledge, E.J. and J.P. Sumpter. 1997. Structural features of alkylphenolic chemicals associated with estrogenic activity. J. Biol. Chem. 272:3280-3288.

Roy F. Weston Inc. 1990. Determination of the vapor pressure of 4-nonylphenol. Final Report Study No. 90-047. Roy F. Weston Inc., Environmental Fate and Effects Laboratory, 254 Welsh Pool Road, Lionville, PA. 15 August 1990.

Schmude, K. L., K. Liber, T. D. Corry and F. S. Stay. 1999. Effects of 4-nonylphenol on benthic macroinvertebrates and insect emergence in littoral enclosures. Environ. Toxicol. Chem. 18:386-393.

Schultz, T.W. 1997. Tetratox: *Tetrahymena pyriformis* population growth impairment endpoint - A surrogate for fish lethality. Toxicol. Methods. 7:289-309.

Schwaiger, J., O.H. Spieser, C. Bauer, H. Ferling, U. Mallow, W. Kalbfus and R.D. Negele. 2000. Chronic toxicity of nonylphenol and ethinylestradiol: haematological and histopathological effects in juvenile common carp (*Cyprinus carpio*). Aquat. Toxicol. 51:69-78.

Schwaiger, J., U. Mallow, H. Ferling, S. Knoerr, T. Braunbeck, W. Kalbfus and R.D. Negele. 2002. How estrogenic is nonylphenol: A transgenerational study using rainbow trout (*Oncorhynchus mykiss*) as a test organism. Aquat. Toxicol. 59:177-189.

Seki, M., H. Yokota, M. Maeda, H. Tadokoro and K. Kobayashi. 2003. Effects of 4-nonylphenol and 4-*tert*-octylphenol on sex differentiation and vitellogenin induction in medaka (*Oryzias latipes*). Environ. Toxicol. Chem. 22:1507-1516.

Servos, M.R. 1999. Review of the aquatic toxicity, estrogenic responses and bioaccumulation of alkylphenols and alkylphenol polyethoxylates. Wat. Qual. Res. J. Can. 34:123-177.

Severin, G.F., G. Welzl, I. Juttner, G. Pfister and K-W. Schramm. 2003. Effects of nonylphenol on zooplankton in aquatic microcosms. Environ. Toxicol. Chem. 22:2733-2738.

Shackelford, W.M., D.M. Cline, L. Faas and G. Kurth. 1983. Evaluation of automated spectrum matching for survey identification of wastewater components by gas chromatography-mass spectrometry. PB83-182931. National Technical Information Service, Springfield, VA.

Shang, D.Y., R.W. MacDonald and M.G. Ikonomou. 1999. Persistence of nonylphenol ethoxylate surfactants and their primary degradation products in sediments from near a municipal outfall in the Strait of Georgia, British Columbia, Canada. Environ. Sci. Technol. 33:1366-1372.

Shurin, J.B. and S.I. Dodson. 1997. Sublethal toxic effects of cyanobacteria and nonylphenol on environmental sex determination and development in *Daphnia*. Environ. Toxicol. Chem. 16:1269-1276.

Sonnenschein, C. and A.M. Soto. 1998. An updated review of environmental estrogen and androgen mimics and antagonists. J. Steroid Biochem. Molec. Biol. 65:143-150.

Soto, A.M., H. Justicia, J.W. Wray and C. Sonnenschein. 1991. *p*-Nonylphenol: An estrogenic xenobiotic released from modified polystyrene. Environ. Health Perspect. 92:167-173.

Soto, A.M., T.M. Lin, H. Justicia, R.M. Silvia and C. Sonnenschein. 1992. An "in culture" bioassay to assess the estrogenicity of xenobiotics (E-SCREEN). In: Advances in Modern Environmental Toxicology. Volume XXI. Chemically-induced alterations in sexual and functional development: The wildlife/human connection. T. Colburn and C. Clement (Eds.). Princeton Scientific Publishing Co., Inc., NJ. 403 pp.

Spieser, O.H., J. Schwaiger, H. Ferling, W. Kalbfus and R. Negele. 1998. Effects of nonylphenol and ethinyl-estradiol on swimming behavior of juvenile carp. Abstract for Annual Meeting. International Association for Great Lakes Research. Hamilton, Ontario, Canada. pp. 37-38.

Staples, C.A., J. Weeks, J.F. Hall and C.G. Naylor. 1998. Evaluation of aquatic toxicity and bioaccumulation of C8- C9-alkylphenol ethoxylates. Environ. Toxicol. Chem. 17:2470-2480.

Staples, C.A., J.B. Williams, R.L. Blessing and P.T. Varineau. 1999. Measuring the biodegradability of nonylphenol, ether carboxylates, octylphenol, ether carboxylates, and nonylphenol. Chemosphere 38:2029-2039.

Staples, C.A., C.G. Naylor, J.B. Williams and W.E. Gledhill. 2001. Ultimate biodegradation of alkylphenol ethoxylate surfactants and their biodegradation intermediates. Environ. Toxicol. Chem. 20:2450-2455.

Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman and W.A. Brungs. 1985. Guidelines for

deriving numerical national water quality criteria for the protection of aquatic organisms and their uses. http://www.epa.gov/waterscience/criteria/85guidelines.pdf

Sumpter, J.P. 1998. Xenoendocrine disrupters -- environmental impacts. Toxicol. Lett. 102-103:337-342.

Sundaram, K.M.S. and S. Szeto. 1981. The dissipation of nonylphenol in stream and pond water under simulated field conditions. J. Environ. Sci. Health (Part B) B16(6):767-776.

Sundaram, K.M.S., S. Szeto, R. Hindle and D. MacTavish. 1980. Residues of nonylphenol in spruce foliage, forest soil, stream water and sediment after its aerial application. J. Environ. Sci. Health (Part B) B15(4):403-419.

Tabira, Y., M. Nakai, D. Asai, Y. Yakabe, Y. Tahara, T. Shinmyozu, M. Noguchi, M. Takatsuki and Y. Shimohigashi. 1999. Structural requirements of *para*-alkylphenols to bind to estrogen receptor. Eur. J. Biochem. 262:240-245.

Tagliabue, M.D. 1993. Nonylphenol *Champia* results. Memo dated February 23 to S. Poucher, Science Applications International Corporation, Narragansett, RI. 6 pp.

Takasawa, R., H. Etoh, A. Yagi, K. Sakata and K. Ina. 1990. Nonylphenols as promising antifouling agents found by a simple bioassay method using the blue mussel, *Mytilus edulis*. Agric. Biol. Chem. 54:1607-1610.

Tanghe, T., G. Devriese and W. Verstraete. 1999. Nonylphenol and estrogenic activity in aquatic environmental samples. J. Environ. Qual. 28:702-709.

Thibaut, R., L. Debrauwer, D. Rao and J. Cravedi. 1998. Characterization of biliary metabolites of 4-n-nonylphenol in rainbow trout (*Oncorhynchus mykiss*). Xenobiotica. 28:745-757.

Thibaut, R., L. Debrauwer, D. Rao and J.P. Cravedi. 1999. Urinary metabolites of 4-n-nonylphenol in rainbow trout (Oncorhynchus mykiss). Sci. Total Environ. 233:193-200.

Tsuda, T., A. Takino, M. Kojima, H. Harada, K. Muraki and M. Tsuji. 2000. 4-Nonylphenols and 4-tert-octylphenol in water and fish from rivers flowing into Lake Biwa. Chemosphere 41:757-762.

Tremblay, L. and G. Van Der Kraak. 1998. Use of a series of homologous in vitro and in vivo assays to evaluate the endocrine modulating actions of β-sitosterol in rainbow trout. Aquat. Toxicol. 43:149-162.

Turner, A.H., F.S. Abram, V.M. Brown and H.A. Painter. 1985. The biodegradability of two primary alcohol ethoxylate nonionic surfactants under practical conditions, and the toxicity of biodegradation products to rainbow trout. Water Res. 19:45-51.

Uguz, C., M. Iscan, A. Erguven, B. Isgor and I. Togan. 2003. The bioaccumulation of nonyphenol and its adverse effect on the liver of rainbow trout (*Onchorynchus mykiss*). Environ. Res. 92:262-270.

University of Wisconsin-Superior. 1985. Acute toxicities of organic chemicals to fathead minnows (*Pimephales promelas*). Volume II. D.L. Geiger, C.E. Northcott, D.J. Call and L.T. Brooke (Eds.). Center for Lake Superior Environmental Studies, University of Wisconsin-Superior, Superior, WI.
326 pp.

U.S. EPA. 1983. Water quality standards regulation. *Federal Register* 48:51400-51413. November 8.

U.S. EPA 1986. Chapter I - Stream design flow for steady-state modeling. In: Book VI - Design conditions. In: Technical guidance manual for performing waste load allocation. Office of Water, Washington, DC.

U.S. EPA. 1987. Permit writers guide to water quality-based permitting for toxic pollutants. Office of Water, Washington, DC. EPA-440/4-87-005.

U.S. EPA. 1991. Technical support document for water quality-based toxics control. Office of Water, Washington, DC. EPA-505/2-90-001. Available for download at: http://www.epa.gov/npdes/pubs/owm0264.pdf

U.S. EPA. 1994. Water quality standards handbook. Second Edition. Office of Water, Washington, DC. EPA-823-B-94-005. Available for download at: http://www.epa.gov/waterscience/standards/handbook/

USITC. 1981. Synthetic organic chemicals: United States production and sales, 1980. United States International Trade Commission Publication No. 1183. U.S. Government Printing Office, Washington, DC 20402. 327 p.

USITC. 1989. Synthetic organic chemicals: United States production and sales, 1988. United States International Trade Commission Publication No. 2219. U.S. Government Printing Office, Washington, DC 20402.

Varma, M.M. and D. Patel. 1988. Nonionic surfactants in perspective. J. Environ. Systems 18:87-96.

Veith, G.D. and O.G. Mekenyan. 1993. A QSAR approach for estimating the aquatic toxicity of soft electrophiles (QSAR for soft electrophiles). Quant. Struct. Act. Relat. 12:349-356.

Villenueve, D.L., S.A. Villalobos, T.L. Keith, E.M. Snyder, S.D. Fitzgerald and J.P. Giesy. 2002. Effects of waterborne exposure to 4-nonylphenol on plasma sex steroid and vitellogenin concentrations in sexually mature male carp (*Cyprinus carpio*). Chemosphere 47:15-28.

Ward, T.J. and R.L. Boeri. 1990a. Acute static toxicity of nonylphenol to the freshwater alga *Selenastrum capricornutum*. EnviroSystems Study No. 8969-CMA. Resource Analysts, Inc., Hampton, NH. 41 pp.

Ward, T.J. and R.L. Boeri. 1990b. Acute flow through toxicity of nonylphenol to the mysid, *Mysidopsis bahia*. Study Number 8974-CMA. EnviroSystems, Hampton, NH. 35 pp.

Ward, T.J. and R.L. Boeri. 1990c. Acute flow through toxicity of nonylphenol to the sheepshead minnow, *Cyprinodon variegatus*. Study Number 8972-CMA. EnviroSystems, Hampton, NH. 34 pp.

Ward, T.J. and R.L. Boeri. 1990d. Acute static toxicity of nonylphenol to the marine alga *Skeletonema costatum*. Study Number 8970-CMA. EnviroSystems, Hampton, NH. 42 pp.

Ward, T.J. and R.L. Boeri. 1991a. Bioconcentration test with nonylphenol and the fathead minnow, *Pimephales promelas*. Study Number 8975-CMA. Envirosystems, Hampton, NH. 72 pp.

Ward, T.J. and R.L. Boeri. 1991b. Chronic toxicity of nonylphenol to the mysid, *Mysidopsis bahia*. Study Number 8977-CMA. EnviroSystems, Hampton, NH. 61 pp.

Ward, T.J. and R.L. Boeri. 1991c. Early life stage toxicity of nonylphenol to the fathead minnow, *Pimephales promelas*. Study Number 8979 CMA. EnviroSystems, Hampton, NH. 59 pp.

Ward, T.J. and R.L. Boeri. 1992. Toxicity of nonylphenol to the tadpole *Rana catesbiana*. Study Number 8981-MA. EnviroSystems, Hampton, NH. 78 pp.

Weinberger, P. and R. Greenhalgh. 1984. Some adjuvant effects on the fate of fenitrothion and aminocarb. Environm. Toxicol. Chem. 3:325-334.

Weinberger, P. and S. Iyengar. 1983. Effects of aminocarb, fuel oil 585 and nonylphenol on the growth and development of *Lemna minor* L. Dev. Ecol. Environ. Qual. Shuval, H. I. (Ed.). Proc. Int. Meet. Isr. Ecol. Soc. pp. 595-607.

Weinberger, P. and M. Rea. 1981. Nonylphenol: A perturbant additive. Canadian Tech. Rep. Fish Aquat. Sci. 990:370-381.

Weinberger, P., C. DeChacin and M. Czuba. 1987. Effects of nonyl phenol, a pesticide surfactant, on some metabolic processes of *Chlamydomonas segnis*. Can. J. Bot. 65:696-702.

White, R., S. Jobling, S.A. Hoare, J.P. Sumpter and M.G. Parker. 1994. Environmentally persistent alkylphenolic compounds are estrogenic. Endocrinology 135:175-182.

Wood, E. M. 1953. The toxicity of 3400 chemicals to fish. Report of Fish and Wildlife Service, Kearneysville, West Virginia. EPA 560/6-87-002. Office of Toxic Substances, Washington, DC, August.

Yadetie, F., A. Arukwe, A. Goksoyr and R. Male. 1999. Induction of hepatic estrogen receptor in juvenile Atlantic salmon in vivo by the environmental estrogen, 4-nonylphenol. Sci. Total Environ. 233:201-210.

Yokota, H., M. Seki, M. Maeda, Y. Oshima, H. Tadokoro, T. Honjo and K. Kobayashi. 2001. Life-cycle toxicity of 4-nonylphenol to medaka (*Oryzias latipes*). Environ. Toxicol. Chem. 20:2552-2560.

Yoshimura, K. 1986. Biodegradation and fish toxicity of nonionic surfactants. J. Amer. Oil Chem. Soc. 63:1590-1596.

Yoshioka, Y. 1985. Testing for the toxicity of chemicals with *Tetrahymena pyriformis*. Sci. Total Environ. 43:149-157.

Zang, L., R. Gibble and K.N. Baer. 2003. The effects of 4-nonylphenol and ethanol on acute toxicity, embryo development, and reproduction in *Daphnia magna*. Ecotoxicol. Environ. Safety 55:330-337.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION IX
75 Hawthorne Street
San Francisco, CA 94105-3901

JUL 14 2017

Felicia Marcus, Chair
California State Water Resources Control Board
1001 I Street
Sacramento, CA 95814

Re: Water Quality Control Plan for Inland Surface Waters, Enclosed Bays, and Estuaries of California - Tribal and Subsistence Fishing Beneficial Uses and Mercury Provisions

Dear Chair Marcus:

I am pleased to approve the new statewide water quality standards in the subject Provisions[1] consistent with the requirements of section 303(c) of the Clean Water Act and 40 C.F.R. Part 131. Supported by robust science and stakeholder outreach, these standards encompass specific beneficial uses to account for tribal cultural use and subsistence fish consumption, mercury water quality objectives to safeguard human health and aquatic wildlife, and flexibility to achieve permit compliance.

Summarized below are the approved standards, which take effect immediately for Clean Water Act purposes. Incorporated as part of this letter are Enclosure A, Table of Approved Standards, and Enclosure B, EPA's detailed analysis of the standards and rationale for approval.

1. New Human Health Beneficial Uses[2]

EPA approves the additions in Chapter II of three important, broadly defined human health beneficial uses applicable to all pollutants:

- Tribal Tradition and Cultural Use (CUL), which protects California Tribes' cultural, spiritual, ceremonial, and traditional uses of water;
- Tribal Subsistence Fishing Use (T-SUB), which protects non-commercial fishing by Tribal communities to meet sustenance needs; and

[1] The State's approval package, which includes State Water Resources Control Board Resolution 2017-0027, was certified by the California Office of Administrative Law on June 28, 2017 (OAL Matter 2017-0516-03) and submitted to EPA on the same date. The public process leading to the Resolution, which included notice of opportunity for public comment, a public hearing, public meetings and written response to comments, is consistent with the procedural requirements of CWA §303(c) and its implementing regulations, including 40 C.F.R. §131.20.

[2] Under CWA §303 and 40 C.F.R. Part 131, water quality standards include designated uses of the waters and water quality criteria to protect those uses. California refers to "designated use" as "beneficial use" and "water quality criteria" as "water quality objectives."

- Subsistence Fishing Use (SUB), which protects non-commercial fishing by non-Tribal communities to meet sustenance needs.

2. New Mercury Water Quality Objectives[3]

Today's action covers five new provisions for mercury water quality objectives in Chapter III. One provision (the Sport Fish Objective) is applicable both to human health use and to aquatic life and aquatic-dependent wildlife uses. There are three new human health objectives (two numeric and one narrative) and three new numeric wildlife objectives, as discussed below. The numeric objectives are measured in the amount of methylmercury, an organic and toxic form of mercury, in fish tissue. This is appropriate as bioaccumulation of methylmercury through diet is the primary route of exposure to toxic levels of mercury.

a. Human Health Mercury Objectives

The approved human health mercury objectives are:
- Tribal Subsistence Fishing Objective of 0.04 mg/kg for waters with the T-SUB use;
- Narrative Subsistence Fishing Objective, which prohibits levels of mercury in fish that cause adverse effects in people for waters with the SUB use or, in the North Coast Region, waters with the Subsistence Fishing (FISH) use; and
- Sport Fish Objective of 0.2 mg/kg for waters with the Commercial and Sport Fishing use or the CUL use.

The numeric objectives reflect consideration of detailed, site-specific fish consumption rates and default values published by EPA, and the protective narrative prohibition further provides the Regional Water Quality Control Boards flexibility to consider diverse consumption patterns and the relevant EPA default rate to translate to a numeric fish tissue value.

b. Wildlife Mercury Objectives[4]

The approved mercury objectives for the protection of aquatic life and aquatic-dependent wildlife are:
- Sport Fish Objective of 0.2 mg/kg, as applied to wildlife;
- Prey Fish Objective of 0.05 mg/kg; and
- California Least Tern Prey Fish Objective of 0.03 mg/kg.

All three objectives are for waters with one or more wildlife beneficial uses, including: Wildlife Habitat, Marine Habitat, Warm Freshwater Habitat, Cold Freshwater Habitat, Estuarine Habitat, Inland Saline Water Habitat, and/or Preservation of Rare and Endangered Species. For such waters, the Sport Fish Objective and one of the two Prey Fish Objectives will apply, depending

[3] This section provides the numeric objectives in summary form. See Enclosures A and B for their specific applications.

[4] EPA has initiated consultation on the approval of the mercury wildlife objectives under Section 7 of the Endangered Species Act and has the authority to take additional measures regarding these objectives if warranted by the consultation.

on whether the endangered California Least Tern or its habitat exists. If so, the more stringent Least Tern prey fish objective applies.

The State's detailed scientific studies, including analyses of species of concern selected from a thorough review of the federal and state listed species lists, offer clear support that these objectives are protective of California's sensitive aquatic wildlife. As certain threatened and endangered species may be particularly sensitive to mercury exposure, based on further evaluation of relevant mercury toxicity studies, California may wish to consider future adoption of site-specific criteria for waters inhabited by those species.

3. Applicability of New Mercury Objectives

Per Chapter III.D.3, the new mercury objectives apply to all inland surface waters and enclosed bays and estuaries but do not supersede the currently applicable, EPA-approved numeric objectives for the following waters:

- the San Francisco Bay;
- the Sacramento-San Joaquin Delta, including the Yolo Bypass;
- the fresh water portions of Walker Creek, Soulajule Reservoir, and tributaries (Arroyo Sausal, Salmon Creek, Chileno Creek, and Keyes Creek);
- Sulphur Creek (Schoolhouse Canyon to confluence with Bear Creek);
- Clear Lake;
- Cache Creek (including North Fork);
- Bear Creek;
- Harley Gulch; and
- the Guadalupe River Watershed (except Los Gatos Creek and its tributaries upstream of Vasona Dam, Lake Elsman, Lexington Reservoir, and Vasona Lake).

Except for lower Sulphur Creek, these waters' mercury objectives, developed with site-specific information and similar fish tissue methodology, are consistent with the new statewide objectives. Since lower Sulphur Creek has naturally occurring levels of mercury that do not support suitable fish habitat, its objectives reflect background conditions.[5]

The new mercury objectives, however, replace two less stringent objectives previously approved by EPA: the San Francisco Bay Basin Water Quality Control Plan's 25 microgram per liter water column objective and the Central Coastal Basin Water Quality Control Plan's 0.5 mg/kg fish tissue objective.

The general applicability of the new mercury objectives and the specified exceptions are reasonable and appropriate for the protection of human health and wildlife in California's waters.

4. Compliance Schedule Authorizing Provision

The mercury objectives associated with the new human health uses are significantly more stringent than those associated with current uses. California has in place an EPA-approved statewide "2008 Compliance Schedule Policy" applicable to the new mercury objectives.

[5] For further details on these site-specific, previously approved wildlife objectives, see Enclosure B, Attachment 1.

However, the State has indicated that its 2008 Policy does not apply to permits issued during the interim between the adoption of a new human health use for a waterbody with an existing mercury Total Maximum Daily Load (TMDL) and the adoption of a new mercury TMDL based on that more stringent new use, and that it adopted Chapter IV.D.2.c.2.ii. to address that gap and supplement the 2008 Policy.

EPA finds that Chapter IV.D.2.c.2.ii includes language constituting a compliance schedule authorizing provision, which EPA approves under 40 C.F.R. § 131.15, but only to the extent it authorizes granting mercury discharges not covered by the 2008 Policy a compliance schedule that is: (i) "as soon as possible" to meet final effluent limitations based on the more stringent new use, not to exceed 10 years from the time the permit first includes interim limitations consistent with the existing TMDL; and (ii) not based solely on time needed to develop a new TMDL.

I look forward to our continued partnership to protect California waters and advance human health and wildlife protection. Please call me if you would like to discuss further, or your staff may contact Diane Fleck of the Water Quality Assessment Section at (415) 972-3527 for specific questions concerning this approval.

Sincerely,

Tomás Torres July 14, 2017
Director, Water Division

Enclosures (2)
A. Table of Approved Standards
B. EPA Analysis of Approved Standards

cc: Michael Lauffer, Acting Executive Director, SWRCB
Karen Larsen, Deputy Director, SWRCB
Jacob Iversen, Environmental Scientist, SWRCB

Enclosure A

Table of Approved Standards

Beneficial Use	Discussion/Definition	
Chapter II. 2nd paragraph	Confirmation of Tribal use designation by a California Native American Tribe.	
Chapter II. 1): Tribal Tradition and Culture (CUL)	"Uses of water that support the cultural, spiritual, ceremonial, or traditional rights or LIFEWAYS of CALIFORNIA NATIVE AMERICAN TRIBES, including, but not limited to: navigation, ceremonies, or fishing, gathering, or consumption of natural aquatic resources, including fish, shellfish, vegetation, and materials."	
Chapter II. 2): Tribal Subsistence Fishing (T-SUB)	"Uses of water involving the non-commercial catching or gathering of natural aquatic resources, including fish and shellfish, for consumption by individuals, households, or communities of California Native American Tribes to meet needs for sustenance."	
Chapter II. 3): Subsistence Fishing (SUB)	"Uses of water involving the non-commercial catching or gathering of natural aquatic resources, including fish and shellfish, for consumption by individuals, households, or communities, to meet needs for sustenance."	
Water Quality Objective	**Applicable Beneficial Uses**	**Objective Value – Annual Average methylmercury in fish tissue, wet weight**
Chapter III.D.2.b: Tribal Subsistence Fishing Objective (Human Health)	Tribal Subsistence Fishing (T-SUB)	**0.04 milligrams per kilogram (mg/kg)** in 70% Trophic Level (TL) 3 fish and 30% TL 4 fish, skinless fillet.
Chapter III.D.2.c: Subsistence Fishing Objective (Human Health)	Subsistence Fishing (SUB); Subsistence Fishing (FISH – Regional Board 1)	Narrative: Waters…shall be maintained free of mercury at concentrations which accumulate in fish and cause adverse biological, reproductive, or neurological effects. (Fish consumption rate shall be site-specific; default: 142 grams/day)
Chapter III.D.2.a: Sport Fish Objective (Human Health and Wildlife)	**Human Health Uses**: Commercial & Sport Fishing (COMM); Tribal Tradition & Culture (CUL). **Wildlife Uses:** Wildlife Habitat (WILD); Marine Habitat (MAR); Warm Freshwater Habitat (WARM); Cold Freshwater Habitat (COLD); Estuarine Habitat (EST); Inland Saline Water Habitat (SAL); Preservation of Rare & Endangered Species (RARE).	**0.2 mg/kg** in highest TL fish, skinless fillet; If TL 3 fish, 150 – 500 millimeters (mm) total length; If TL 4 fish, 200 – 500 mm total length.
Chapter III.D.2.d: Prey Fish Objective (Wildlife)	WILD; MAR; WARM; COLD; EST; SAL; RARE; Where least tern objective does not apply.	**0.05 mg/kg** in whole fish 50-150 mm total length, between Feb 1 – July 31.
Chapter III.D.2.e: California Least Tern Prey Fish Objective (Wildlife)	WILD; MAR; WARM; COLD; EST; SAL; RARE; Where least tern or least tern habitat exists.	**0.03 mg/kg** in whole fish < 50 mm total length, between April 1 – August 31.
Chapter III.D.3.	Interaction of Mercury Water Quality Objectives with Basin Plans	(N/A)
Compliance Schedule Authorizing Provision	**When Applicable**	
Chapter IV. D.2.c.2.ii.	Where a mercury TMDL exists and the State adopts a more stringent human health use associated with CUL, T-SUB or SUB for the same waterbody, provisions in the existing mercury TMDL may continue to apply, if certain requirements are met.	

Enclosure B

EPA Review of State Water Resources Control Board Water Quality Standards for Mercury and New Beneficial Uses

I. Background

Around 2004, the State Water Resources Control Board (SWRCB) started working on a package to adopt statewide human health and wildlife mercury water quality objectives and implementation procedures. The SWRCB subsequently added human health beneficial uses for Native American Tribes and subsistence fisherpeople to the package and conducted significant public outreach during 2014, 2015, and 2016. On December 29, 2016, the SWRCB issued a public notice entitled, *Notice of Opportunity for Public Comment, Staff Workshop, Public Hearing, and Notice of Filing*, concerning the availability of documents, workshops, and hearings for its proposal. The proposed package was posted at the SWRCB's website on January 3, 2017, and workshops were held on January 9, 2017 and February 1, 2017. A hearing to take oral public comment was held in Sacramento on February 7, 2017, and written public comment was accepted through noon on February 17, 2017. The SWRCB prepared a Response to Comment document, and posted its final proposed package at its website[1] on April 21, 2017.

At a public meeting on May 2, 2017, the SWRCB adopted Resolution No. 2017-0027, *Part 2 of the Water Quality Control Plan for Inland Surface Waters, Enclosed Bays, and Estuaries of California—Tribal and Subsistence Fishing Beneficial Uses and Mercury Provisions* (Provisions). On June 28, 2017, the State's Office of Administrative Law (OAL) completed its review of the package and approved the Resolution (see OAL Matter Number: 2017-0516-03). On June 28, 2017, EPA received a complete package from the State requesting review and approval of the beneficial uses, the water quality objectives, and a compliance schedule authorizing provision contained in the Provisions.[2]

II. Summary of Water Quality Standards at Issue

Clean Water Act (CWA) section 303(c) directs states to adopt water quality standards (designated uses, criteria, and anti-degradation requirements) for their waters that are subject to the CWA and implementing regulations at 40 CFR Part 131. This regulation requires, among other things, that a state's water quality standards specify appropriate designated uses of the waters and water quality criteria that protect those uses. California uses the term "beneficial use" to mean the same as "designated use" under the CWA and the term "water quality objective" to mean the same as "water quality criteria" under the CWA.

California's new water quality standards included in the Provisions are consistent with CWA section 303(c) and 40 CFR Part 131. The Provisions: 1) add three new human health beneficial

[1] The SWRCB's website can be found here: http://www.waterboards.ca.gov/water_issues/programs/mercury/

[2] The public process leading to the Resolution adopting the Provisions includes notice of opportunity for public comment, a public hearing, public meetings, and written response to comments, and is consistent with the procedural requirements of CWA section 303(c) and its implementing regulations, including 40 CFR §131.20.

uses for subsistence fishing and for California Native American Tribal subsistence fishing and culture to protect Tribal members and other subsistence fisherpeople; 2) add five new mercury water quality objectives to protect human health, aquatic life, and aquatic-dependent wildlife from the toxic effects of mercury through diet; and 3) add a compliance schedule authorizing provision to facilitate the implementation of the new, more stringent mercury objectives for California.

III. New Beneficial Uses

The Provisions add three new human health beneficial uses (in italics below) in Chapter II. Beneficial Uses. Chapter II. Beneficial Uses states:

> * * *
>
> *For the State Water Resources Control Board (State Water Board) or the Regional Water Boards to designate the Tribal Tradition and Culture or Tribal Subsistence Fishing beneficial uses in a water quality control plan for a particular waterbody segment and time(s) of year, a CALIFORNIA NATIVE AMERICAN TRIBE[2] must confirm the designation is appropriate.*
>
> *** * ***
>
> *1) Tribal Tradition and Culture (CUL): Uses of water that support the cultural, spiritual, ceremonial, or traditional rights or LIFEWAYS of CALIFORNIA NATIVE AMERICAN TRIBES, including, but not limited to: navigation, ceremonies, or fishing, gathering, or consumption of natural aquatic resources, including fish, shellfish, vegetation, and materials.*
>
> *2) Tribal Subsistence Fishing (T-SUB): Uses of water involving the non-commercial catching or gathering of natural aquatic resources, including fish and shellfish, for consumption by individuals, households, or communities of California Native American Tribes to meet needs for sustenance.*
>
> *3) Subsistence Fishing (SUB): Uses of water involving the non-commercial catching or gathering of natural aquatic resources, including fish and shellfish, for consumption by individuals, households, or communities, to meet needs for sustenance.*
>
> ---
>
> *[2] Terms in "all cap" font (excepting the beneficial use abbreviations) are defined in Attachment A (Glossary).*

The beneficial uses are available for the SWRCB and the Regional Water Quality Control Boards (RWQCBs) to consider applying to specific waterbodies. The T-SUB use and CUL use will become effective for specific waterbodies when the SWRCB or RWQCB confirms with a California Native American Tribe that the designation is appropriate and the RWQCB and/or SWRCB adopts the use for a specific waterbody and EPA approves the State action. The SUB use will become effective for specific waterbodies when the SWRCB or RWQCB adopts the use for a specific waterbody and EPA approves the State action.

The new beneficial uses are not intended to protect or enhance fish populations or aquatic habitat, as explained in the Provisions at Chapter II. Beneficial Uses, third paragraph. Fish populations and aquatic habitats are protected and enhanced by aquatic life beneficial uses (e.g., fish spawning and warm freshwater habitat) which are designed to support habitats intended for fish reproduction and development. See Provisions, Chapter II. Beneficial Uses.

Analysis of New Beneficial Uses

The three new human health beneficial uses are reasonable and appropriate to protect California Native American Tribal traditions and culture, Tribal subsistence fishing, and other subsistence fishing. The uses are described in more detail in Chapter 6.4 of the *Final Staff Report, Including Substitute Environmental Documentation for Part 2 of the Water Quality Control Plan for Inland Surface Waters, Enclosed Bays, and Estuaries of California – Tribal and Subsistence Fishing Beneficial Uses and Mercury Provisions* (Final Staff Report). The CUL use provides protection for cultural, spiritual, ceremonial, and traditional uses of the water for California Tribal members. The protections apply to natural aquatic resources such as fish, shellfish, vegetation, and other aquatic materials. The T-SUB and SUB uses provide protection for subsistence fishing by Tribal and non-Tribal fisherpeople, respectively. The State refers to subsistence fishing as the consumption by individuals, households or communities for sustenance, and includes the non-commercial catching and gathering of natural aquatic resources such as fish and shellfish. The uses are broadly defined to cover a range of activities concerning tradition, culture and subsistence, and are intended to apply for all pollutants. Many subsistence fisherpeople use California waters for fishing and many California Native American Tribal members practice cultural traditions using California waters or resources from California waters. The uses are important for RWQCBs to consider to protect human health when adopting beneficial uses for specific waterbodies.

The State conducted extensive outreach over several years with many California Native American Tribes and environmental justice groups to develop the CUL, T-SUB, and SUB uses. See Final Staff Report, Chapter 2.6. EPA supports these collaborative efforts and the resulting beneficial uses to protect California Native American Tribal members and other subsistence fisherpeople. The beneficial uses are appropriate uses of California's waters subject to the CWA.

IV. New Mercury Water Quality Objectives

The Provisions add four new numeric mercury water quality objectives and one new narrative mercury water quality objective (in italics below) in Chapter III. Water Quality Objectives, D. Mercury, 2. Mercury Water Quality Objectives. Subsection 2. Mercury Water Quality Objectives has five subparts (a. through e.) which state:

> ***a. Sport Fish Water Quality Objective***
>
> *1) Application of the Sport Fish Water Quality Objective*
>
> *The Sport Fish Water Quality Objective for mercury applies to waters with the beneficial uses of COMM, CUL[5], WILD, or MAR.*
>
> *With respect to the WILD and MAR beneficial uses, the Sport Fish Water Quality Objective may be used to evaluate whether all species are supported only when applied to TROPHIC LEVEL 4 fish, except with respect to the California least tern (as discussed in Chapter III.D.2.e). If the objective is measured using TROPHIC LEVEL 3 fish, protection of all wildlife species within the WILD and MAR beneficial uses is not ensured. Therefore, if TROPHIC LEVEL 3 fish are used, then the Prey Fish Water Quality Objective (as described in Chapter III.D.2.d) shall be used, but if the water body is habitat for California least tern, then the California Least Tern Prey Fish Objective (as described in Chapter III.D.2.e) shall be used. However, if the Sport Fish Water Quality Objective is exceeded when applied to TROPHIC LEVEL 3 fish, that is sufficient evidence to indicate that the Prey Fish Water Quality Objective or, if applicable, the California*

Least Tern Prey Fish Objective is also exceeded without having to measure the two latter objectives (see flow chart in Attachment B).

2) Sport Fish Water Quality Objective

The Sport Fish Water Quality Objective is: The average methylmercury concentrations shall not exceed 0.2 milligrams per kilogram (mg/kg) fish tissue within a CALENDAR YEAR[6]. The water quality objective applies to the WET WEIGHT concentration in skinless fillet in TROPHIC LEVEL 3 or TROPHIC LEVEL 4 fish, whichever is the HIGHEST TROPHIC LEVEL FISH in the water body. Freshwater TROPHIC LEVEL 3 fish are between 150 to 500 millimeters (mm) in total length and TROPHIC LEVEL 4 fish are between 200 to 500 mm in total length, except for sizes specified in Attachment C, or as additionally limited in size in accordance with the LEGAL SIZE LIMIT for the species caught. Estuarine fish shall be within the LEGAL SIZE LIMIT and greater than 150 mm, or as otherwise specified in Attachment C.

b. Tribal Subsistence Fishing Water Quality Objective

1) Application of the Tribal Subsistence Fishing Water Quality Objective

The Tribal Subsistence Fishing Water Quality Objective applies to waters with the T-SUB beneficial use.

2) Tribal Subsistence Fishing Water Quality Objective

The Tribal Subsistence Fishing Water Quality Objective is: The average methylmercury concentrations shall not exceed 0.04 mg/kg fish tissue within a CALENDAR YEAR. The objective applies to the WET WEIGHT concentration in skinless fillet from a mixture of 70 percent TROPHIC LEVEL 3 fish and 30 percent TROPHIC LEVEL 4 fish as detailed in Attachment C.

c. Subsistence Fishing Water Quality Objective

1) Application of the Subsistence Fishing Water Quality Objective

The Subsistence Fishing Water Quality Objective applies to waters with the SUB beneficial use or to waters with the FISH beneficial use (see footnote 2).

2) Subsistence Fishing Water Quality Objective

The Subsistence Fishing Water Quality Objective is: Waters with the Subsistence Fishing (SUB) beneficial use shall be maintained free of mercury at concentrations which accumulate in fish and cause adverse biological, reproductive, or neurological effects in people.

The fish consumption rate used to evaluate this objective shall be derived from water body- and population-specific data and information on the subsistence fishers' rate and form (e.g. whole, fillet with skin, skinless fillet) of fish consumption.[7]

When a water quality control plan designates a water body or water body segment with the Subsistence Fishing (SUB) beneficial use, development of a region-wide or site-specific numeric fish tissue mercury water quality objective is recommended to account for the wide variation of consumption rate and fish species encompassed by the SUB beneficial use.

d. Prey Fish Water Quality Objective

1) Application of the Prey Fish Water Quality Objective

The Prey Fish Water Quality Objective applies to waters with the WILD or MAR beneficial uses. However, the objective does not apply to water body segments where the California Least Tern Prey Fish Water Quality Objective applies (see Chapter III.D.2.e). As discussed in Chapter III.D.2.a, it is not necessary to measure the Prey Fish Water Quality Objective if the Sport Fish Water Quality Objective applies to the same water body and is evaluated using TROPHIC LEVEL 4 fish. However, if the Sport Fish Water Quality Objective is exceeded when applied to TROPHIC LEVEL 3 fish, that is sufficient evidence to indicate that the Prey Fish Water Quality Objective is also exceeded without having to measure the latter objective (see flow chart in Attachment B).

2) Prey Fish Water Quality Objective

The Prey Fish Water Quality Objective is: The average methylmercury concentrations shall not exceed 0.05 mg/kg in WET WEIGHT whole fish tissue of any species between 50 to 150 mm in total length during

the breeding season. The breeding season is February 1 through July 31, unless site-specific information indicates another appropriate breeding period.

e. California Least Tern Prey Fish Water Quality Objective

1) Application of the California Least Tern Prey Fish Water Quality Objective

The California Least Tern Prey Fish Water Quality Objective applies to water with the WILD, MAR, or RARE beneficial uses at water bodies where the least tern or least tern habitat exists, including but not limited to the water bodies identified in Attachment D.

2) California Least Tern Prey Fish Water Quality Objective

The California Least Tern Prey Fish Water Quality Objective is: The average methylmercury concentrations shall not exceed 0.03 mg/kg fish tissue from April 1 through August 31. The objective applies to the WET WEIGHT concentration in whole fish less than 50 mm total length.

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[5] *If site-specific studies indicate a consumption pattern under the CUL beneficial use higher than the consumption rate used for the objective to support the COMM beneficial use, then the Regional Water Board should consider adopting a site-specific objective to protect consumption of fish under the CUL beneficial use.*

[6] *Any explicit reference in the MERCURY PROVISIONS to "CALENDAR YEAR" means a fixed period of twelve CALENDAR MONTHS (i.e., the period of months would not be moving or rolling).*

[7] *U.S. EPA recommended national subsistence fishing consumption rate of 142 grams per day (4 to 5 meals per week) shall be used to translate the narrative objective unless a site-specific numeric water quality objective is developed or an external peer-reviewed consumption study uses a different methodology to translate the narrative water quality objective.*

Attachment B, Mercury Prey Fish Decision Diagram, is a flowchart for determining when it is necessary to monitor mercury levels in prey fish. Attachment C, Fish Trophic Level Classifications, is a list of fish species and sizes associated with specific trophic levels, and Attachment D, Waters Protected by the Mercury California Least Tern Prey Fish Water Quality Objective, is a list of identified waters to which the California Least Tern Objective applies.

In footnote 3 of the Provisions, the SWRCB states that the SUB beneficial use also applies to the Subsistence Fishing (FISH) beneficial use contained in the North Coast Regional Water Quality Control Board's water quality control plan (see Water Quality Control Plan for the North Coast (May 2011), p. 2-3.00). In footnote 4 of the Provisions, the SWRCB states that any explicit reference in the Provisions to the WILD or MAR beneficial use includes the WARM, COLD, EST and SAL beneficial uses. Footnotes 3 and 4 are found in Chapter III.D.1., Applicability.

The State's mercury objectives are measured in the amount of methylmercury in fish tissue. Methylmercury is an organic and toxic form of mercury that readily bioaccumulates in living organisms. Bioaccumulation of methylmercury through diet is the primary route of exposure of toxic levels of mercury. Therefore, the amount of methylmercury in fish tissue is an appropriate surrogate for mercury, for water quality objectives. See also Final Staff Report, Chapters 4 and 6.1.

Analysis of New Mercury Water Quality Objectives

The five new mercury water quality objectives to protect human health and wildlife in California are reasonable, protective of the applicable beneficial uses, and based on sound scientific rationale. Three objectives (the Tribal Subsistence Fishing, Subsistence Fishing, and Sport Fish Water Quality Objectives) are for the protection of human health, and three objectives (the Sport Fish, Prey Fish, and California Least Tern Prey Fish Water Quality Objectives) are for the protection of aquatic-dependent wildlife.

1. Human Health Mercury Objectives

Tribal Subsistence Water Quality Objective: The Tribal Subsistence Water Quality Objective applies to waters assigned the T-SUB beneficial use, and the average methylmercury concentration in fish (skinless fillet) must not exceed 0.04 milligrams of methylmercury per kilogram of fish tissue wet weight within a calendar year. The fish must be a mixture of 70% trophic level 3 fish and 30% trophic level 4 fish, as detailed in Attachment C, Fish Trophic Level Classifications.

The SWRCB's fish tissue human health objectives in the Provisions are derived using EPA's equation for deriving fish tissue human heath criteria (USEPA, 2001):

$$FTC = BW * (RfD – RSC)/FI$$

Where:
FTC = Fish Tissue Criterion in milligrams (mg) methylmercury (mehg) per kilogram (kg) fish tissue (or mg/kg) in wet weight (ww)
BW = Body Weight of 70 kg for an average person
RfD = Reference Dose of 0.0001 mg mehg/kg body weight (EPA default value)[3]
RSC = Relative Source Contribution of 2.7×10^{-5} mg mehg/kg body weight (EPA default value); this value is subtracted from the Reference Dose to account for other sources of mehg e.g., marine fish
FI = Fish Intake, i.e., the fish consumption rate, in kilograms per day (kg/day).

The SWRCB used the same input values for BW, RfD, and RSC that EPA used in its 2001 human health CWA 304(a) recommendation for methylmercury (USEPA, 2001) in their calculations to derive the FTC, which is the Tribal Subsistence Water Quality Objective.

The most important variable in the equation is the Fish Intake, or the fish consumption rate. The consumption rate in EPA's 2001 CWA 304(a) recommendation for methylmercury for the general U.S. population (90^{th} percentile of people who do and do not eat fish) is 17.5 grams per

[3] EPA's Integrated Risk Information System (IRIS) Glossary defines Reference Dose as: an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. See: IRIS Glossary.

day (g/day), resulting in a national recommended FTC of 0.3 mg/kg[4] (USEPA, 2001). EPA's default consumption rate for subsistence fishers (99th percentile) is 142 g/day (USEPA, 2000).

The SWRCB used a subsistence consumption rate of 142 g/day, or 4 to 5 fish meals per week. This value is based on a detailed study (through a fish use/consumption survey) of California Native American Tribes conducted as part of the development of the Provisions, *California Tribes Fish-Use: Final Report. A Report for the State Water Resources Control Board and the U.S. Environmental Protection Agency, Agreement Number 11-146-250*, July 2014, by Fraser Shilling, A. Negrette, L. Biondini, and S. Cardenas (California Tribes Fish-Use Report). See Final Staff Report, Chapters 4.9 and 6.5.

The California Tribes Fish-Use Report found that the 95th percentile consumption rate of California Native American Tribes is 141.8 (142 rounded) g/day (Shilling et al., 2014, Table 6). The current California Tribal subsistence consumption rate (95th percentile) is the same value as the current EPA national default subsistence rate (99th percentile), although the California Tribal rate was independently derived using detailed information from California Native American Tribes.

Using a consumption rate of 142 g/day in the equation above results in a FTC value of 0.04 mg/kg, the Tribal Subsistence Water Quality Objective. The objective is applied to the skinless fillet (muscle) portion of fish since most people eat the fillet portion of fish. Fillets also contain the highest concentration of methylmercury compared to other edible parts of fish. The objective is an annual average value; samples collected over a calendar year will be averaged. Since the objective is a chronic objective, i.e. a long-term objective, it is appropriate to determine attainment over a longer averaging period. In addition, mercury is a bioaccumulative pollutant and accumulates in tissue from diet through the food chain. This bioaccumulation process takes time before the methylmercury is reflected in the muscle tissue. See also Final Staff Report, Appendix H. Calculation of Human Health Objective, Section H.4 Averaging Period for the Water Quality Objectives.

The SWRCB chose to determine attainment of the objective from a mixture of 70% trophic level (TL) 3 and 30% TL 4 fish to reflect the species of fish and amount consumed by Tribal members as discussed in the California Tribes Fish-Use Report. EPA's implementation guidance for the CWA section 304(a) methylmercury water quality criterion (USEPA, 2010) says that states should consider factoring the consumption of different TLs when computing the average methylmercury concentration in fish tissue. The California Tribes Fish-Use Report found that most fish currently consumed by Tribal members were TL 3 fish.

EPA national default and/or California-specific Trophic Level Ratios (TLRs) can be used to determine the methylmercury levels in TL 3 and TL 4 fish necessary to achieve attainment of the 0.04 mg/kg objective. TLRs are determined by measuring the amount of methylmercury in different TL fish (and within specific size ranges) in the same waterbody or same type of waterbody. California-specific TLRs were determined using statewide fish tissue data. See Final Staff Report, Appendix L. Derivation of Trophic Level Ratios. For example, using California TLRs, consumption of 70% TL 3 fish and 30% TL 4 fish would result in a TL 3 fish value of

[4] All fish tissue values in this document are in wet weight.

0.03 mg/kg, and a TL 4 fish value of 0.06 mg/kg to attain the objective of 0.04 mg/kg of methylmercury in fish tissue. See Final Staff Report, Appendix H. Calculation of the Human Health Objectives, Table H-4. Potential Subsistence Objectives Using Mixed Consumption Scenarios.

The calculation of the Tribal Subsistence Water Quality Objective is reasonable and appropriate, and based on sound scientific rationale: it uses local information based on a detailed study of California Tribes. The consumption rate and percentages of TL 3 and TL 4 fish consumed are specific to California Tribes. Other factors in the derivation of the objective value are based on EPA recommended values and are reasonable for California.

Subsistence Water Quality Objective: The Subsistence Water Quality Objective applies to waters assigned the SUB beneficial use, and for waters in the North Coast Region, the Subsistence Fishing (FISH) beneficial use. The objective is expressed as a narrative and states that waters shall be maintained free of mercury concentrations which accumulate in fish and cause adverse effects in people. The consumption rate shall be derived from site-specific information; in the absence of site-specific information, the EPA default subsistence consumption rate shall be used to translate the narrative objective or an external peer-reviewed consumption study using a different methodology may be used to translate the narrative objective to numeric fish tissue values.

The narrative objective prohibits levels of mercury in fish that cause adverse effects in people, and therefore, is protective of human health. Site-specific consumption rates may be used to translate the objective into a numeric fish tissue value giving the RWQCBs flexibility to consider broad and/or locally diverse consumption patterns, or EPA's default subsistence consumption rate of 142 g/day (99^{th} percentile) or 4 to 5 fish meals per week (USEPA, 2000) may be used. Appendix H. Calculation of the Human Health Objectives of the Final Staff Report provides different translations based on the percentages of TL 2, 3 and 4 fish consumed using the EPA default rate of 142 g/day. Since a default breakout is not included in the narrative objective, the breakout (if any – since the State may apply the consumption rate to 100% TL 3 or 100% TL 4 fish) would be based on site-specific information on the amounts and species of fish consumed from the waterbody by the targeted consumer group. The RWQCBs will determine whether to apply the tissue value to one or several TLs based on site-specific information on what TL fish (and in what amounts) the subsistence population is consuming from the waterbody. Appendix H, Table H-4. Potential Subsistence Objectives Using Mixed Consumption Scenarios, provides numeric fish tissue values using different percentages of TL 2, 3, and 4 fish. The calculations use TLRs to determine the allowable methylmercury levels in the different TL fish.

The narrative Subsistence Water Quality Objective is reasonable and appropriate, and based on sound scientific rationale.

Sport Fish Water Quality Objective: The Sport Fish Water Quality Objective, as applied to human health, applies to waters with the Commercial and Sport Fishing (COMM) and/or CUL beneficial use. The COMM use includes uses of water for commercial or recreational collection of fish, shellfish, or other organisms intended for human consumption. Recreational (or sport) fishing occurs on many waterbodies in California, but some waterbodies have not been

designated with the COMM use. Historically, the RWQCBs had associated sport fishing with the Water Contact Recreation (REC-1) use because its definition includes "fishing" (but not consumption). See Final Staff Report, Chapter 5.1. Where sport fishing and the consumption of fish occurs, but the COMM use has not been designated, the Sport Fish Water Quality Objective applies (R.Rasmussen, SWRCB, personal communication on March 6, 2017 with D. Fleck, J. Hashimoto, and P. Kozelka, EPA).

The objective states that the average methylmercury concentration in fish (skinless fillet) must not exceed 0.2 mg/kg within a calendar year. The concentration applies to TL 3 or TL 4 fish, whichever is the highest TL in the waterbody. Freshwater TL 3 fish must be between 150 – 500 millimeters (mm) total length, and TL 4 fish must be between 200 – 500 mm total length unless specified in Attachment C or limited by the legal size limit for the species caught. Estuarine fish must be within the legal size limit and greater than 150 mm (or as otherwise specified in Attachment C).

The objective is derived using EPA's equation for deriving fish tissue criteria for human health (USEPA, 2001) as discussed above:

$$FTC = BW * (RfD – RSC)/FI$$

Where:
FTC = Fish Tissue Criterion in mg mehg/kg fish tissue
BW = Body Weight of 70 kg for an average person
RfD = Reference Dose of 0.0001 mg mehg/kg body weight (EPA default value)
RSC = Relative Source Contribution of 2.7×10^{-5} mg mehg/kg body weight (EPA default value); this value is subtracted from the Reference Dose to account for other sources of mehg e.g., marine fish
FI = Fish Intake, i.e., the fish consumption rate, in kg/day.

The SWRCB used the same input values for BW, RfD, and RSC that EPA used in its 2001 human health CWA 304(a) recommendation for methylmercury (USEPA, 2001) in their calculations to derive the FTC, which is the Sport Fish Water Quality Objective.

The most important variable in the equation is the Fish Intake, or the fish consumption rate. The consumption rate in EPA's 2001 CWA 304(a) recommendation for methylmercury for the general U.S. population (90^{th} percentile of people who do and do not eat fish) is 17.5 grams per day (g/day), resulting in a national recommended FTC of 0.3 mg/kg (USEPA, 2001).

The SWRCB used a consumption rate of 32 g/day, or 1 fish meal (approximately 8 ounces) per week. This value was chosen after a thorough review of all sport fish consumption studies for waters in California. See Final Staff Report, Appendix G. Fish Consumption Studies. The rate was derived from a survey of anglers in San Francisco Bay completed in 2000, *The San Francisco Bay Seafood Consumption Study* by the San Francisco Estuary Institute (SFEI) (SFEI, 2000). The SWRCB stated that it was "probably one of the highest-quality studies done to date" in California (Final Staff Report, Appendix G. Fish Consumption Studies, page 1). The study (and the 32 g/day consumption rate) was used to derive mercury fish tissue objectives for the San Francisco Bay and the Sacramento-San Joaquin Delta (previously approved by EPA), as well as

the Fish Contaminant Goal for the Office of Environment Health Hazard Assessment. The value is the 95th percentile of consumption rates from anglers in the study.

Using a consumption rate of 32 g/day in the equation above results in a FTC value of 0.2 mg/kg, the Sport Fish Water Quality Objective. The objective is applied to the skinless fillet (muscle) portion of fish, since most people eat the fillet portion of fish. The objective is an annual average value; samples collected over a calendar year will be averaged. Since the objective is a chronic objective, i.e., a long-term objective, it is appropriate to determine attainment over a longer averaging period. In addition, mercury is a bioaccumulative pollutant and accumulates in tissue from diet through the food chain. This bioaccumulation process takes time before methylmercury levels are reflected in fish tissue. See also Final Staff Report, Appendix H. Calculation of Human Health Objective, Section H.4 Averaging Period for the Water Quality Objectives.

A footnote on the objective states that if site-specific studies indicate a consumption pattern under the CUL use that is higher than the consumption rate used to determine the objective (i.e., 32 g/day), the RWQCB should consider adopting a site-specific objective to protect for the consumption of fish under the CUL use. Use of a higher site-specific consumption rate is reasonable and necessary to assure that human health is protected in waterbodies with a CUL use where consumption is occurring.

The Sport Fish Objective applies to the highest TL fish in the waterbody, either TL 4 fish, or if no TL 4 fish are present in the waterbody, to TL 3 fish. The size ranges specified reflect large TL 3 and TL 4 fish within legal catch limits, because most fisherpeople desire to catch and consume large fish. Since mercury bioaccumulates up the food chain, applying the objective to larger fish results in a more stringent objective. Applying the 0.2 mg/kg objective to large TL3 and TL 4 fish will protect human health.

The Sport Fish Water Quality Objective is reasonable and appropriate, and based on sound scientific rationale. It uses a local recreational fisher consumption rate and applies to the highest TL fish in the waterbody and to the larger size ranges of each TL fish within legal size limits. Other factors in the derivation of the objective value are based on EPA recommended values.

In conclusion, the three new human health water quality objectives are consistent with CWA section 303(c) and implementing regulations at 40 CFR Part 131. The water quality objectives are appropriate for the protection of human health in California's waters subject to the CWA.

2. Wildlife Mercury Objectives

Sport Fish Water Quality Objective, Prey Fish Water Quality Objective, and the California Least Tern Prey Fish Water Quality Objective: The Sport Fish Water Quality Objective, as applied to aquatic life and aquatic-dependent wildlife, and the Prey Fish Water Quality Objective apply to waters with the following beneficial uses: Wildlife Habitat (WILD); Marine Habitat (MAR); Warm Freshwater Habitat (WARM); Cold Freshwater Habitat (COLD); Estuarine Habitat (EST); and Inland Saline Water Habitat (SAL). Waters assigned the Preservation of Rare and Endangered Species (RARE) use, the Fish Migration (MIGR) use, or the Spawning, Reproduction and/or Early Development (SPWN) use are designated with at least one of the

applicable beneficial uses, i.e., WILD, MAR, WARM, COLD, EST and/or SAL (R.Rasmussen, SWRCB, personal communication on March 6, 2017 with D. Fleck, J. Hashimoto, and P. Kozelka, EPA).[5] Therefore, the Sport Fish Water Quality Objective and the Prey Fish Water Quality Objective apply to waters designated with the following beneficial uses: WILD, MAR, WARM, COLD, EST, SAL, RARE, MIGR, and SPWN. These uses are the beneficial uses that protect aquatic life and aquatic-dependent wildlife in California.

The Sport Fish Water Quality Objective of 0.2 mg/kg was calculated to protect human health, using a 32 g/day fish consumption rate and applies to large TL 3 and 4 fish, whichever is the highest TL fish in the waterbody. However, the objective also serves to protect aquatic life and aquatic-dependent wildlife because limiting methylmercury levels in large fish for human consumption results in lower methylmercury levels in smaller fish (i.e., prey fish). The SWRCB completed a thorough review to determine whether the Sport Fish Objective would sufficiently protect aquatic life and aquatic-dependent wildlife in California and found that additional protections may be necessary for some aquatic-dependent (avian) species. See Final Staff Report, Chapters 6.7 and 6.8, Appendix J. Review of Effects on Wildlife, and Appendix K. Wildlife Targets.

The Prey Fish Objective of 0.05 mg/kg applies to whole fish between 50 and 150 mm total length during the breeding season (February 1 through July 31, unless site-specific information indicates another appropriate period). The objective is an average value. The Prey Fish Water Quality Objective does not apply where the California Least Tern Prey Fish Water Quality Objective applies.

The California least tern is a small, piscivorous bird that consumes large quantities (relative to its size) of very small fish less than 50 mm, and therefore, is more vulnerable to mercury bioaccumulation in the aquatic food web. The U.S. Fish and Wildlife Service (FWS) listed them as an endangered species under the Endangered Species Act (ESA). Therefore, an additional objective is included to protect them. The California Least Tern Prey Fish Water Quality Objective applies to waters with the WILD, MAR, WARM, COLD, EST, SAL, and RARE uses (and to waters with MIGR and SPWN uses through one of the other uses), where least tern or least tern habitat exist. The Least Tern Objective of 0.03 mg/kg applies to whole fish less than 50 mm total length from April 1 through August 31. The objective is an average value.

<u>For waterbodies with wildlife beneficial uses, the Sport Fish Objective and EITHER the Prey Fish Objective OR the California Least Tern Prey Fish Objective will apply to the waterbody</u>. Although the Sport Fish Objective protects most wildlife species, it was designed to protect human health and not wildlife; therefore, one of the prey fish objectives will also apply to all waters with a wildlife use to ensure that all aquatic life and wildlife species are protected.

[5] The Final Staff Report at Chapter 5.6 Inapplicable Uses, states that the MIGR use is not applicable because mercury does not impede migration, and the SPWN use is not applicable because the wildlife objectives are not meant to protect for fish reproduction, although waters designated with the SPWN use are also designated with WILD, COLD and/or WARM and protective mercury thresholds for fish reproduction are higher than the objectives (and thus fish reproduction is protected). EPA believes that waters with MIGR and SPWN uses should be included as applicable uses. However, since waters with the MIGR or SPWN use are covered through another wildlife use, no issue remains.

Because the SWRCB determined that the Sport Fish Objective would be protective of wildlife in most but not all situations, the Prey Fish Objective must be monitored for attainment only in certain situations. The Prey Fish Objective must be monitored for attainment in water bodies: 1) where the Least Tern Objective does not apply, and 2) when the Sport Fish Objective is NOT exceeded using TL 3 fish. When a waterbody meets the Sport Fish Objective using TL 4 fish (the most stringent application), the Prey Fish Objective will also be met, and additional monitoring to determine attainment is not necessary. When the waterbody meets the Sport Fish Objective using TL 3 fish, although the Sport Fish Objective is met for human health, the Prey Fish Objective must be measured to determine whether the Sport Fish Objective is met for wildlife, i.e., the waterbody is attaining wildlife uses. When the waterbody does not meet the Sport Fish Objective using TL 3 (or 4) fish, the Prey Fish Objective is not met for wildlife and there is no need to measure it. See Final Staff Report, Attachment B. Mercury Prey Fish Decision Diagram.

Prey Fish and Least Tern Prey Fish Objective Values

In Appendix K. Wildlife Targets, of the Final Staff Report, the SWRCB explains how it developed the prey fish objectives. The SWRCB followed the FWS's methodology used in several reports prepared in collaboration with EPA and the State to evaluate methylmercury fish tissue levels to protect wildlife in California. The reports include the FWS's October 2003 report *Evaluation of the Clean Water Act Section 304(a) Human Health Criterion for Methylmercury: Protectiveness for Threatened and Endangered Wildlife in California*, prepared by Daniel Russell, USFWS, Sacramento Fish and Wildlife Service (USFWS, 2003). The report found that when EPA's CWA section 304(a) human health guidance criterion of 0.3 mg/kg was applied to a diet consisting of 100% large TL 4 fish in California (the most stringent human dietary application of the criterion), the resulting fish tissue levels in smaller TL 2 and 3 fish species (e.g., prey fish for aquatic-dependent piscivorous wildlife) would protect most California wildlife species, but would likely not protect the most sensitive listed wildlife bird species in California, the California least tern. The report concluded that the least tern would be protected with a TL 3 (prey) fish tissue value of 0.03 mg/kg. FWS prepared similar reports using the same methodology for Cache Creek and the Sacramento-San Joaquin Delta Watersheds (USFWS, 2004), and the Guadalupe River Watershed (USFWS, 2005). The FWS and the National Marine Fisheries Service (NMFS) (collectively, the Services) assisted the State and EPA on the development of other site-specific methylmercury fish tissue objectives including objectives for the San Francisco Bay. The SWRCB coordinated closely with the Services and EPA on the development of the wildlife objectives in the Provisions.

The methodology to develop wildlife objectives uses the following equation (Final Staff Report, Appendix K. Wildlife Targets, equation 1):

$$WV = RfD \times BW/FIR$$

Where:
WV = Wildlife Value in mg mehg/kg tissue (or mg/kg) in the prey of a species diet
RfD = Reference Dose of the species of concern in mg mehg/kg body weight per day (mg/kg-bw/day)
BW = Body Weight of the species of concern in kg

FIR = Food Ingestion Rate of the species of concern in kg/day of food consumed.

The WV is the average safe concentration of methylmercury in the overall diet (food) of the wildlife species necessary to keep the species' daily ingested amount at or below the RfD.

Since methylmercury bioaccumulates and biomagnifies, the SWRCB (and FWS in their previous reports to the State) focused on birds and mammals that prey directly on fish because they are generally higher-order predators that would have a greater potential for dietary exposure and subsequent toxicity than lower order aquatic and aquatic-dependent species such as reptiles or amphibians (Final Staff Report, Appendix K. Wildlife Targets)[6]. The SWRCB focused on the species of concern that the FWS focused on in their previous reports to the State. After thoroughly reviewing the current lists of federal and state listed species, the SWRCB finalized the list of species of concern for their analysis for the Provisions. The list consisted of 18 species: 3 mammals and 15 birds. The species, and associated RfD, BW, FIR, and WV values are summarized in Table K-1 in Appendix K. Wildlife Targets of the Final Staff Report, which is included below.

The SWRCB used values for RfDs, BWs FIRs and WVs that FWS used in their previous reports to the State, except for the common loon. The RfD values (one for birds and one for mammals) are from the 2003 FWS study that evaluated EPA's CWA section 304(a) human health criterion (USFWS, 2003). The FWS study used an avian RfD of 0.021 mg/kg-bw/day based on a mallard duck study (Heinz, 1979) and uncertainty factors from the Mercury Study Report to Congress (MSRC) (EPA, 1997)[7]; and a mammalian RfD of 0.018 mg/kg–bw/day based on analyses from both the MSRC and the Great Lakes Initiative (EPA, 1995) using data from Wobeser et al., 1976a,b. For the common loon, the values were taken from the analysis for Clear Lake completed by the Central Valley Regional Water Quality Control Board (Central Valley Water Board) (Central Valley Water Board, 2002). The Services concurred on a Not Likely to Adversely Affect determination for the ESA consultation for Clear Lake.

[6] In Appendix J. Review of Effects on Wildlife, at section J.4 Exposure and Effects on Fish, the SWRCB reviewed the literature on methylmercury effects on fish. The SWRCB summarized their findings citing Crump and Trudeau, 2009 and Sandheinrich and Wiener, 2011; these authors found that effects on survival, growth, behavior and reproduction in freshwater fish occur at concentrations of 0.3 – 0.7 mg/kg or greater in whole body, and 0.5 – 1.2 mg/kg or greater in muscle (Appendix J, Final Staff Report). The SWRCB also found that Depew et al., 2012 found a dietary threshold of 0.05 mg/kg for reproductive and biochemical effects. The SWRCB concluded that top predator fish would be protected by its prey fish objective of 0.05 mg/kg in TL 3 fish since it met the lowest threshold found in the literature, i.e., Depew et al., 2012 (Appendix J, Final Staff Report).

[7] Three uncertainty factors (UFs) may be considered when developing a RfD: a UF(A) to account for interspecies uncertainty, a UF(S) to account for subchronic to chronic uncertainty, and a UF(L) to account for LOAEL to NOAEL uncertainty. (The LOAEL is the lowest observed adverse effect level and the NOAEL is the no observed adverse effect level.) A RfD = Test Dose / (UF(A) x UF(S) x (UF(L)). The MSRC (and the FWS and SWRCB) used a UF(A) and UF(S) of 1 and a UF(L) of 3 for birds.

Table K-1. Wildlife Values (mg/kg in diet) (From the Final Staff Report, Appendix K)

Species	RfD (mg/kg/day)	Body Weight (kg)	FIR (kg/day)	Wildlife Value[a] (mg/kg in diet)
Mink	0.018	0.60	0.140	0.077
River otter	0.018	6.70	1.124	0.107
Belted kingfisher	0.021	0.15	0.068	0.046
Common merganser	0.021	1.23	0.302	0.085 (0.099[b])
Western grebe	0.021	1.19	0.296	0.084
Double-crested cormorant	0.021	1.74	0.390	0.094
Osprey	0.021	1.75	0.350	0.105 (0.112[b])
Bald eagle	0.021	5.25	0.566	0.195 (0.184[c])
Peregrine falcon	0.021	0.89	0.134	0.139
Southern sea otter [FT]	0.018	19.8	6.5	0.055
California least tern [FE]	0.021	0.045	0.031	0.030
California Ridgeway's rail [FE]	0.021	0.346	0.172	0.042
Light-footed Ridgeway's rail [FE]	0.021	0.271	0.142	0.040
Yuma Ridgeway's rail [FE]	0.021	0.271	0.142	0.040
Western snowy plover [FT]	0.021	0.041	0.033	0.026
Great blue heron	0.021	2.20	0.378	0.122 [b]
Forster's tern	0.021	0.16	0.071	0.047 [b]
Common loon	0.021 [d]	4[d]	0.800 [d]	0.105

[a] from the USFWS Cache Creek Targets (USFWS 2004) and the USFWS Evaluation of the U.S. EPA Human Health Criterion (USFWS 2003), except as otherwise noted
[b] from Guadalupe River Watershed targets (USFWS 2005)
[c] the two references (USFWS 2004 and USFWS 2003) provided different values
[d] from Clear Lake analysis (Central Valley Water Board 2002)
[FT / FE] on federal list of threatened or endangered species

The Wildlife Value, or WV, is the safe prey fish concentration, if the species mostly eats one size of fish from the same TL. If the species eats different sizes of fish from multiple TLs, and/or other aquatic prey, the following equation is used to determine each safe prey fish concentration for each size category and TL (Final Staff Report, Appendix K. Wildlife Targets, equation 2):

$$WV = (\%TL2 \times [Hg]TL2) + (\%TL3 \times [Hg]TL3) + (\%TL4 \times [Hg]TL4)$$

Where:
%TL2 = Percent of trophic level 2 biota in diet
%TL3 = Percent of trophic level 3 biota in diet
%TL4 = Percent of trophic level 4 biota in diet
[Hg]TL2 = concentration in food from trophic level 2
[Hg]TL3 = concentration in food from trophic level 3
[Hg]TL4 = concentration in food from trophic level 4

Since most piscivorous wildlife eat a variety of sizes of fish, often from different TLs, the FWS in their 2003 report and the RWQCBs in their reports to derive site-specific methylmercury objectives compiled information on the diets of the species of concern from the scientific literature. The SWRCB consolidated the information from the previous FWS and RWQCB reports on diet into Table K-2 in Appendix K. Wildlife Targets.

Table K-2. Trophic Level (TL) Compositions (Expressed as Decimal Fractions) for Wildlife Species, Including Omnivorous Birds (OB), Piscivorous Birds (PB) and Other Foods (OF) (From the Final Staff Report, Appendix K)

Species	TL2	TL2/3 < 50 mm	TL3 < 150 mm	TL3 150 – 500 mm	TL4 150 – 500 mm	OB	PB	OF
Mink			1.00					
River otter			0.80		0.20			
Belted kingfisher			1.00					
Common Merganser				1.00				
Western grebe				1.00[a]				
Double-crested cormorant			1.00					
Osprey				0.90	0.10			
Bald eagle				0.58	0.13	0.13	0.05	0.11
Peregrine falcon						0.10	0.05	0.85
Southern sea otter	0.80		0.20					
California least tern		1.00						
California Ridgeway's rail	0.85		0.05					
Light-footed Ridgeway's rail	0.82		0.18					
Yuma Ridgeway's rail	0.23		0.72					0.05
Western snowy plover	0.25							.75
Great blue heron			1.00[b]					
Forster's tern		1.00[b]						
Common loon				0.80[c]				

Note: most data are from the USFWS evaluation of the U.S. EPA human health criterion (Table 4, USFWS 2003), the USFWS Cache Creek targets (Table 4, USFWS 2004) and the Sacramento-San Joaquin Delta targets (Table 4.1 and Table 4.3, Central Valley Water Board 2010), except as otherwise noted.

[a] The U.S. Geological Survey grebe study team caught fish 18 – 123 mm as representative grebe prey (Ackerman et al. 2015). Also, fish found in the stomachs of western grebes were 27 – 88 mm (1 – 3.5 in) long (CDFW 1990). In any case, the larger size (used in Table K-2) is more protective.

[b] from Guadalupe River Watershed targets (Table 4 and 5, USFWS 2005).

[c] from Clear Lake targets (Table C-3, Central Valley Water Board 2002), reclassified based on the 200 – 400 mm size and CDFW 1990. Clear Lake report has the loon diet as "TL2" but "200 – 400 mm". Because of the size the fish are shown here as TL3. The CDFW life history account for loon: "Diet varies; usually about 80% fish, with crustaceans the next largest item… Most fish eaten are not sought by humans…" Burgess and Meyer report "We sampled small fish (76 – 127 mm in length) typically consumed as prey by loons (Barr 1996)"

Using equation 2 from Appendix K, the WVs from Table K-1 and the dietary breakout of each species from Table K-2[8] (and using Food Chain Multipliers, FCMs[9], and TLRs developed for the human health objectives to calculate tissue concentrations for different TLs), the SWRCB performed extensive analyses to determine each tissue concentration for each size range and TL to protect each of the species of concern. These values are shown in Table K-3 of Appendix K of the Final Staff Report, included below.

The shaded values in Table K-3 represent the lowest necessary values for each category of TL and size range, i.e., final wildlife objectives must be at least as stringent as the shaded values to protect all the species of concern. Species of concern with shaded values are: belted kingfisher, western grebe, osprey, California least tern, light-footed Ridgeway's rail, and Yuma Ridgeway's rail. Since the shaded values are in various TLs and size ranges, the SWRCB converted each set of shaded values into the same TL and size range for comparison purposes, using FCMs and TLRs. The SWRCB converted each set of shaded gray values into TL 4 150 – 500 mm fish (values for osprey were not converted since osprey eat from that TL and size range). Once each was converted, the SWRCB could choose the lowest value as the objective, and all wildlife species would be protected.

After performing the calculations, the SWRCB found that belted kingfisher, western grebe, osprey, light-footed Ridgeway's rail, and Yuma Ridgeway's rail would be protected by an objective of 0.2 mg/kg in TL 4 fish 150 – 500 mm total length, the Sport Fish Water Quality Objective for human health. All other species in Table K-3, except the California least tern, would also be protected because each of the other species was not the most sensitive species in the TL and size range category from which it ate. See Final Staff Report, Appendix K. Wildlife Targets.

Since California least tern would not be protected by the Sport Fish Water Quality Objective when applied to TL 4 fish, the SWRCB recommended a separate, additional objective of 0.03 mg/kg in fish less than 50 mm to protect least tern (Final Staff Report, Appendix K. Wildlife Targets).

Since the Sport Fish Water Quality Objective of 0.2 mg/kg applies to the highest TL fish in the waterbody to protect human health, if TL 4 fish are not found in the waterbody, the objective applies to TL 3 fish. The objective applies to large TL 3 and 4 fish, 150 – 500 mm and 200 – 500 mm, respectively, because people prefer to catch and consume large fish. However, 0.2 mg/kg in TL 3 fish 150 – 500 mm is not protective of the species of concern in Table K-3, and thus would not provide protection to wildlife (Final Staff Report, Appendix K. Wildlife Targets).

To protect the species of concern in waters where TL 4 fish do not exist, and where the Sport Fish Objective applies to TL 3 fish, the SWRCB found that an additional objective of 0.05 mg/kg

[8] Prey food for the California Ridgeway's Rail included 10% vegetation, which was considered to have negligible methylmercury.

[9] Food Chain Multipliers (FCMs) are similar to Trophic Level Ratios (TLRs) in that they both express the relationship between TLs in a waterbody. FCMs reflect a direct predator-prey relationship between TLs, while TLRs reflect the relationship between similarly sized fish in different TLs.

for TL 3 fish 50 – 150 mm was necessary, based on the belted kingfisher in Table K-3. Based on previous calculations in the Appendix, the SWRCB found that the 0.05 mg/kg objective for TL 3 fish 50 – 150 mm was consistent with achieving 0.08 mg/kg in TL 3 fish 150 – 500 mm for the western grebe. These calculations are reasonable and based on sound scientific rationale.

In conclusion, the three new wildlife water quality objectives are consistent with CWA section 303(c) and implementing regulations at 40 CFR Part 131. The water quality objectives are appropriate for the protection of aquatic life and aquatic-dependent wildlife in California's waters subject to the CWA.

Table K-3. Protective Wildlife Targets (in mg/kg, wet weight) in Various Trophic Levels (TL), Omnivorous Birds (OB) or Piscivorous Birds (PB), and the Most Sensitive Species in Each TL Category (Shaded Gray) (From the Final Staff Report, Appendix K)

Species	TL 2	TL2/3 < 50 mm	TL3 < 150 mm	TL3 150 – 500 mm	TL4 150 – 500 m	OB	PB
Mink			0.077 a,b				
River Otter			0.04 a 0.059 b 0.067 g		0.30 b 0.36 a 0.27 g		
Belted Kingfisher			0.046 a,b,c				
Common Merganser				0.085 a,b 0.099 c (150–300 mm)			
Western Grebe				0.084 a,b (150 – 300 mm)			
Double-crested Cormorant			0.094 a,b				
Osprey				0.09 a,d,g 0.10 b,c,e	0.26 a 0.17 b 0.20 c,g 0.19 d 0.18 e		
Bald Eagle				0.11a,g 0.12 b,e 0.09 d 0.08 f	0.31 a 0.20 b 0.22 d 0.23 e 0.28 f 0.24 g	0.19 a 0.21 b 0.20 g	1.35 a 1.50 b 1.29 d 1.43 g
Peregrine Falcon				(0.17) a,b,e		0.30 a,b,e	2.17 a,b,e
Southern sea otter FT	0.028 f		0.16 f				
California least tern FE		0.03 b					
California Ridgeway's rail FE	0.037 f		0.21 f				
Light-footed Ridgeway's rail FE	0.022 f		0.12 f				
Yuma Ridgeway's rail FE	0.009 f		0.050 f				
Western snowy plover FT	0.104 f						
Great blue heron			0.12 c				
Forster's tern		0.047 c					
Common loon				0.11 d			

[a] from Sacramento-San Joaquin Delta targets (Table 4.3, Central Valley Water Board 2010)
[b] from the Cache Creek targets (USFWS 2004, Table 5 and Table 6)
[c] from Guadalupe River Watershed targets (Table 5, USFWS 2005)
[d] from Clear Lake analysis (Table C-3,C-4 Central Valley Water Board 2002).
[e] from Cache Creek targets (Central Valley Water Board 2005)
[f] calculated from information in the USFWS evaluation of the human heath criterion (USFWS 2003)
[g] calculated as part of this report for California, see text above.
FT / FE on federal list of threatened or endangered species

V. Applicability of New Human Health and Wildlife Water Quality Objectives

The Provisions add a new subsection, 3. Interaction of Mercury Water Quality Objectives with Basin Plans, in Chapter III (in italics below). Chapter III. Water Quality Objectives, D. Mercury. Subsection 3. Interaction of Mercury Water Quality Objectives with Basin Plans states:

> *The MERCURY WATER QUALITY OBJECTIVES do not supersede any site-specific numeric mercury water quality objectives established in a Basin Plan, except (i) the freshwater mercury water quality objective for chronic effects to aquatic life (0.025 µg/L) established in the San Francisco Bay Basin Water Quality Control Plan (Table 3-4, and corresponding note); and (ii) the total body burden of 0.5 µg/g wet weight established for the mercury water quality objective for aquatic organisms in the Water Quality Control Plan for the Central Coastal Basin (see note accompanying Table 3-5).*

The objectives in the Provisions apply to inland surface waters and enclosed bays and estuaries (they do not apply to ocean waters). The new mercury water quality objectives do not apply to the following waters where there are existing State-adopted and EPA-approved site-specific numeric water quality objectives for mercury in inland surface waters and enclosed bays and estuaries (with 2 exceptions as explained below):[10]

- all segments of the San Francisco Bay;
- all segments of the Sacramento-San Joaquin Delta including the Yolo Bypass;
- the freshwater portions of Walker Creek, Soulajule Reservoir, and all tributary waters (Arroyo Sausal, Salmon Creek, Chileno Creek, and Keyes Creek);
- Sulphur Creek, from Schoolhouse Canyon to its confluence with Bear Creek;
- Clear Lake;
- Cache Creek (including North Fork);
- Bear Creek;
- Harley Gulch; and
- Waters of the Guadalupe River Watershed except Los Gatos Creek and its tributaries upstream of Vasona Dam, Lake Elsman, Lexington Reservoir, and Vasona Lake.

Analysis of Applicability of New Human Health and Wildlife Water Quality Objectives

The application of the new mercury objectives to all inland surface waters and enclosed bays and estuaries except where the State has adopted, and EPA has approved, site-specific numeric water quality objectives with the two exceptions in the Provisions is reasonable. For each of the listed waters except Sulphur Creek, the State has adopted, and EPA has approved, fish tissue objectives designed to protect human health and wildlife in those areas. The objectives were developed using the same methodology as the statewide fish tissue objectives, and are similar (or identical) in value, and are protective of aquatic life and aquatic-dependent wildlife including federally listed threatened and endangered species. See Attachment A, Previously Approved Site-Specific Mercury Objectives. They are site-specific to each area, and were developed using site-specific information such as the wildlife that live in the area and consumption patterns of the population in the area.

[10] See Attachment 1: Previously Approved Site-Specific Water Quality Objectives.

For Sulphur Creek, from Schoolhouse Canyon to its confluence with Bear Creek, the site-specific mercury objectives reflect naturally occurring, pre-anthropogenic, background mercury levels. Fish do not exist in the reach because the naturally occurring conditions do not support suitable habitat. In the summer, the water in the waterbody originates from geothermal sources naturally high in mercury; in the winter, the water in the waterbody contains elevated levels of suspended solids naturally enriched with mercury from the surrounding geology.

The two exceptions listed in the Provisions are less stringent than the current statewide fish tissue objectives. Therefore, these two site-specific water quality objectives that were previously approved by EPA are superseded. The water column objective in the San Francisco Bay Basin Plan is 0.025 micrograms per liter (µg/L) or 25 nanograms per liter (ng/L), while the new fish tissue objectives, translated in Chapter IV. Implementation of Water Quality Objectives, will be either 12 ng/L or 4 ng/L in the water column depending on whether the waterbody is fast or slow moving (or if a lake or reservoir, the permitting authority will calculate the value using EPA's recommended national bioaccumulation factors and chemical translators, but even so, will be in the 4 to 12 ng/L range). The fish tissue objective of 0.5 microgram per gram (µg/g) (or mg/kg) ww in the Water Quality Control Plan for the Central Coastal Basin is clearly less stringent than the least stringent fish tissue objective, the Sport Fish Objective, of 0.2 mg/kg ww. The new statewide objectives are necessary to adequately protect human health and wildlife where these two site-specific objectives currently apply.

It is reasonable and appropriate for the SWRCB to retain the currently effective State-adopted and EPA-approved site-specific fish tissue objectives, and the site-specific objectives for Sulphur Creek, except for the two listed exceptions in the Provisions (the Provisions replace the water column objective in the San Francisco Bay Basin Plan and the fish tissue objective in the Central Coastal Basin Plan).

VI. Addition of New Compliance Schedule Authorizing Provision

Two sections of the Provisions contain language relating to compliance schedules (CSs): Chapter IV.D.2.c.2. and Chapter IV.D.2.d. As explained below, EPA's 303(c) action concerns the language included in Chapter IV.D.2.c.2.ii. that constitutes a compliance schedule authorizing provision (CSAP); other portions of Chapter IV.D.2.c.2. and Chapter IV.D.2.d. are discussed for background purposes.

Chapter IV.D.2.c.2.ii. focuses on the calculation of interim and final effluent limits issued to a discharger subject to an existing mercury TMDL who demonstrates it cannot immediately achieve compliance with more stringent limits based on new mercury water quality objectives. Chapter IV.D.2.c.2.ii. states:

> *ii. Existing mercury TMDL*
>
> *If the discharger is assigned a waste load allocation by the EXISTING MERCURY TMDL, the interim effluent limitation and final effluent limitation may be established as follows:*
>
> ***Interim effluent limitations**. If the discharger demonstrates that the discharger is not immediately able to achieve compliance with the effluent limitation calculated by applying Chapter IV.D.2.c.2.i, above, the interim effluent limitation may be based on the requirements of the applicable waste load allocation in the*

EXISTING MERCURY TMDL applicable to the discharger, so long as: (a) the discharger is subject to a time schedule to complete FEASIBLE tasks to control mercury, if any, in addition to those currently underway, including the development of a proposed schedule for future source control tasks, and (b) the discharger makes a commitment to support, participate in, and expedite the development of a TMDL to implement any of the MERCURY WATER QUALITY OBJECTIVES and associated beneficial uses (CUL, T-SUB, SUB) (i.e., referred to herein as the new mercury TMDL). The time schedule to complete the additional tasks shall be specified in the permit and shall reflect a realistic assessment of the shortest practicable time required to perform each task.

The interim effluent limitation may apply until the new mercury TMDL is in effect, provided the new mercury TMDL is in effect within ten years from the effective date of the first permit that included the interim effluent limitation.

***Final effluent limitations**. If no new mercury TMDL is in effect within ten years from the effective date of the first permit that included the interim effluent limitation, the final effluent limitation shall be calculated in accordance with Chapter IV.D.2.c.2.i and shall take effect ten years from the effective date of the first permit that included the interim effluent limitation. If a new mercury TMDL is in effect within ten years from the effective date of the first permit that included the interim effluent limitation, the final effluent limitation shall be based on the applicable waste load allocation assigned to the discharger by the new mercury TMDL for the water quality standard under evaluation.*

Chapter IV.D.2.d. clarifies that the EPA-approved SWRCB 2008 Compliance Schedule Policy applies to the new mercury objectives, and provides that such a compliance schedule may include requirements consistent with Chapter IV.D.2.c.2.ii, if applicable. Chapter IV.D.2.d. states:

4) Compliance Schedule. The PERMITTING AUTHORITY may include a compliance schedule in NPDES permits to achieve the mercury effluent limitation in accordance with the Policy for Compliance Schedules in National Pollutant Discharge Elimination System Permits (State Water Board Resolution No. 2008-0025). (Compliance Schedule Policy).

The duration of the compliance schedule in a permit may not exceed ten years from the date of the adoption, revision, or new interpretation of the applicable water quality objective, except where a compliance schedule in a permit is established in a "single permitting action" or implements or is consistent with the waste load allocations specified in a TMDL, as provided by the Compliance Schedule Policy. If a compliance schedule is authorized in a permit, interim requirements and final effluent limitation shall be included, as provided by the Compliance Schedule Policy. The compliance schedule may also include requirements consistent with Chapter IV.D.2.c.2.ii, if applicable

The Region understands it is the State's position that its 2008 Policy does not apply to permit limits issued during the interim between the adoption of a new human health use for a waterbody with an existing mercury Total Maximum Daily Load (TMDL) and the adoption of a new mercury TMDL based on that more stringent new use, and that it adopted Chapter IV.D.2.c.2.ii. to address that gap and supplement the 2008 Policy. The State has asked the Region to review and approve Chapter IV.D.2.c.2.ii. as a CSAP.

Analysis of Addition of New Compliance Schedule Authorizing Provision

In 2015, EPA promulgated revised water quality standards (WQS) regulations, which clarified issues surrounding CSAP in the WQS context. 80 Fed Reg 51019, 51041 (August 21, 2015), states:

> In 1990, EPA concluded that before a permitting authority can include a compliance schedule for a [water quality-based effluent limit] WQBEL in an NPDES permit, the state or authorized tribe must affirmatively authorize its use in its WQS or implementing regulations. In the Matter of Star-Kist Caribe, Inc. 3 EAD 172 (April 16, 1990). EPA approval of the state's or authorized tribe's permit compliance schedule authorizing provision as a WQS ensures that any NPDES permit WQBEL with a compliance schedule derives from and complies with applicable WQS as required by § 122.44(d)(1)(vii)(A). Because the state's or authorized tribe's approved WQS authorize extended compliance, any delay in compliance with a WQBEL pursuant to an appropriately issued permit compliance schedule is consistent with the statutory implementation timetable in CWA section 301(b)(1)(C).

The preamble also explained that "the authorizing provision must be consistent with the CWA and is subject to EPA review and approval as a WQS. This rule adds § 131.5(a)(5) to explicitly specify that EPA has the authority to determine whether any provision authorizing the use of schedules of compliance for WQBELs in NPDES permits adopted by a state or authorized tribe is consistent with the requirements at § 131.15." Id. The preamble to the final rule clarified that it does not change any permit compliance schedule requirements at § 122.47. 80 Fed. Reg. 51020, 51041 (Aug. 21, 2015).

Under the Provisions, where a discharger is subject to an existing mercury TMDL (i.e., one that has been "approved by the U.S. EPA for a COMM, WILD or RARE beneficial use" per the Provisions' Glossary) and the SWRCB or a RWQCB adopts one (or more) of the three new human health beneficial uses for the same waterbody (i.e., CUL, SUB or T-SUB), the discharger may seek a CS under the 2008 Compliance Schedule Policy to meet the new, more stringent mercury objectives. However, the State will need time to develop and adopt the new TMDL. The State Chapter IV.D.2.c.2.ii. addresses the question of what interim effluent limits the discharger may be required to meet until the new TMDL is developed.

Chapter IV.D.2.c.2.ii. contains both language addressing permit implementation outside EPA's 303(c) approval authority and language consisting of a CSAP under 33 CFR § 131.5. It provides that, where a mercury TMDL already exists, at the time the State adopts a more stringent human health use for the same waterbody, the permitting authority may authorize a CS and assign interim effluent limitations based on the requirements of the wasteload allocations in the existing TMDL, as described in IV.D.2.c.2.ii., where certain conditions are met. It further provides that the permitting authority may assign limitations consistent with the existing TMDL if the discharger: (i) demonstrates that it cannot immediately achieve compliance with an effluent limit based on a water quality objective for one of the new human health uses; (ii) is subject to a "time schedule" to implement "feasible" mercury control measures, including the development of a proposed schedule for future source control tasks, and (iii) "makes a commitment" to support, participate in, and expedite the development of a new TMDL to implement requirements necessary for the waterbody to attain the newly assigned human health beneficial use, among other things.

The additional time that an interim effluent limitation may be authorized under the Provisions is limited to 10 years from the effective date of the first permit that included the interim limitation. If the new mercury TMDL (that has wasteload allocations based on the new human heath uses and objectives) is not in effect within 10 years from the effective date of the first permit that included the interim limitation, the final limitation will be calculated as described in Chapter IV.D.2.c.2.i. Thus the Provisions effectively limit a CS under IV.D.2.c.2.ii. to 10 years, unless a new TMDL to attain the new beneficial use is completed earlier (i.e., within the 10 years).

It is reasonable that dischargers be accorded additional time (subject to appropriate interim and final requirements and time limitations "as soon as possible") to meet limits based on the significantly more stringent new mercury water quality objectives necessary to meet the new human health beneficial uses. For example, the T-SUB (Tribal Subsistence Fishing) fish tissue objective is 0.04 mg/kg for a mixture of TL 3 and TL 4 fish, while current site-specific fish tissue objectives in the Delta for the protection of human health (recreational fishing) are 0.08 and 0.24 mg/kg in TL 3 and TL 4 fish, respectively. The new fish tissue objective for subsistence (0.04 mg/kg) is significantly more stringent than the current fish tissue objective for recreational fishing (0.24 mg/kg in TL 4 fish and 0.08 mg/kg in TL 3 fish).

The mercury objectives associated with the new human health uses are significantly more stringent than those associated with current uses. California has in place an EPA-approved statewide "2008 Compliance Schedule Policy" applicable to the new mercury objectives. However, the State says that its 2008 Policy does not apply to permits issued during the interim between the adoption of a new human health use for a waterbody with an existing mercury Total Maximum Daily Load (TMDL) and the adoption of a new mercury TMDL based on that more stringent new use, and that it adopted Chapter IV.D.2.c.2.ii.to address that gap and supplement the 2008 Policy.

EPA finds that Chapter IV.D.2.c.2.ii includes language constituting a compliance schedule authorizing provision (CSAP), which EPA approves, under 40 C.F.R. § 131.15, but only to the extent it authorizes granting mercury discharges not covered by the 2008 Policy a compliance schedule that is: (i) "as soon as possible" to meet final effluent limitations based on the more stringent new use, not to exceed 10 years from the time the permit first includes interim limitations consistent with the existing TMDL; and (ii) not based solely on time needed to develop a new TMDL.

VII. Conclusion

The Provisions provide a sound regulatory approach to water quality standards for human health, aquatic life, and aquatic-dependent wildlife. The Provisions include new beneficial uses to protect waters for California Native American Tribal traditions and subsistence fishing, as well as for subsistence fishing by other groups. The Provisions include four new numeric methylmercury fish tissue water quality objectives for the protection of human health and wildlife and one new narrative methylmercury objective for subsistence fishing. Lastly, the Provisions include a compliance schedule authorizing provision to allow municipal and

industrial dischargers additional time, when necessary, to implement more stringent water quality mercury standards associated with the new human health beneficial uses. The Provisions will significantly enhance California waters when they are implemented.

VIII. References

Ackerman et al., 2015: Ackerman J.T., Hartman C.A., Eagles-Smith C.A., Herzog M.P., Davis J., Ichikawa G., Bonnema A. 2015. Estimating Mercury Exposure to Piscivorous Birds and Sport Fish in California Lakes Using Prey Fish Monitoring: A Tool for Managers: U.S. Geological Survey Open-File Report 2015-1106.

Barr, 1996: Barr J.F. 1996. Aspects of common loon (*Gavia immer*) feeding biology on its breeding ground. Hydrobiologia (321) 119–144.

Burgess and Meyer, 2008: Burgess N.M., Meyer M.W. 2008. Methylmercury exposure associated with reduced productivity in common loons. Ecotoxicology 17 (2) 83-91.

CDFW, 1990: California Department of Fish and Wildlife. 1990. California Wildlife Habitat Relationships System. Originally published in: Zeiner, D.C., W.F.Laudenslayer, Jr., K.E. Mayer, and M. White, eds. 1988-1990. California's Wildlife. Vol. I-III. Sacramento, California. Updated. California Department of Fish and Wildlife, Sacramento, CA

Central Valley Water Board, 2002: Central Valley Regional Water Quality Control Board. 2002. Amendments to the Water Quality Control Plan for the Sacramento River and San Joaquin River Basins for the Control of Mercury in Clear lake (Lake County) Clear Lake TMDL for Mercury. Staff Report and Functional Equivalent Document. Final Report, December 2002. Rancho Cordova, CA.

Central Valley Water Board, 2005: Central Valley Regional Water Quality Control Board. 2005. Amendments to the Water Quality Control Plan for the Sacramento River and San Joaquin River Basins For The Control of Mercury in Cache Creek, Bear Creek, Sulphur Creek, and Harley Gulch. Staff Report, October 2005. Rancho Cordova, CA.

Central Valley Water Board, 2010: Central Valley Regional Water Quality Control Board. 2010. Sacramento - San Joaquin Delta Estuary TMDL for Methylmercury. Staff Report, April 2010. Rancho Cordova, CA.

Crump and Trudeau, 2009: Crump K.L., Trudeau V.L. 2009. Mercury-induced reproductive impairment in fish. Environmental Toxicology and Chemistry 28 (5) 895-907.

Depew et al., 2012: Depew D.C., Basu N., Burgess N.M., Campbell L.M., Evers D.C., Grasman K.A., Scheuhammer A.M. 2012. Derivation of screening benchmarks for dietary methylmercury exposure for the common loon (*Gavia immer*): Rational for use in ecological risk assessment. Environmental Toxicology and Chemistry 31 (10) 2399–2407.

Heinz, 1979: Heinz G.H. 1979. Methylmercury: reproductive and behavioral effects on three generations of mallard ducks. Journal of Wildlife Management 43 (2) 394-401.

Sandheinrich and Weiner, 2011: Sandheinrich M.B., Wiener J.G. 2011. Methylmercury in freshwater fish: recent advances in assessing toxicity of environmentally relevant exposures. In: Beyer W.N., Meador J.P. (Eds.), Environmental Contaminants in Biota: Interpreting Tissue Concentrations, 2011. CRC Press, Boca Raton, FL, USA, pp. 169-190.

SFEI, 2000: San Francisco Estuary Institute. 2000. San Francisco Bay Seafood Consumption Study. Richmond, CA.

Shilling et al., 2014: Shilling F., Negrette A., Biondini L., Cardenas S. 2014. California Tribes Fish-Use: Final Report. A Report for the State Water Resources Control Board and the U.S. Environmental Protection Agency. Agreement # 11-146-250. July 2014.

USEPA, 1995: U.S. Environmental Protection Agency. 1995. Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife. EPA-820-B-95-008. Office of Water. Washington, DC.

USEPA, 1997: U.S. Environmental Protection Agency. 1997. Mercury Study Report to Congress Volume VII: Characterization of Human Health and Wildlife Risks from Mercury Exposure in the United States. EPA-452/R-97-009. Office of Research and Development. Washington, DC.

USEPA, 2000: U.S. Environmental Protection Agency. 2000. Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000). Office of Science and Technology, Office of Water. Washington, D.C. EPA-822-B-00-004. October.

USEPA, 2001: U.S. Environmental Protection Agency. 2001. Water Quality Criteria for the Protection of Human Health: Methylmercury. EPA-823-R-01-001. January 2002. U.S. EPA, Office of Water, Washington, DC.

USEPA, 2010: U.S. EPA (U.S. Environmental Protection Agency). 2010. Guidance for Implementing the January 2001 Methylmercury Water Quality Criterion. EPA 823-R-10-001. U.S. Environmental Protection Agency, Office of Water, Washington, DC.

USFWS, 2003: U.S. Fish and Wildlife Service. 2003. Evaluation of the Clean Water Act Section 304(a) Human Health Criterion for Methylmercury: Protectiveness for Threatened and Endangered Wildlife in California. October. Sacramento Fish and Wildlife Office, Environmental Contaminants Division, Sacramento, CA.

USFWS, 2004: U.S. Fish and Wildlife Service. 2004. Evaluation of Numeric Wildlife Targets for Methylmercury in the Development of Total Maximum Daily Loads for the Cache Creek and Sacramento-San Joaquin Delta Watersheds. March. Sacramento Fish and Wildlife Office, Environmental Contaminants Division, Sacramento, CA.

USFWS, 2005: U.S. Fish and Wildlife Service. 2005. Derivation of Numeric Wildlife Targets for Methylmercury in the Development of a Total Maximum Daily Load for the Guadalupe River Watershed. April. Sacramento Fish and Wildlife Office, Environmental Contaminants Division.

Wobeser, et al., 1976a: Wobeser, G., N.O. Nielsen, and B. Schiefer. 1976a. Mercury and mink I. The use of mercury contaminated fish as food for ranch mink. Can. J. Comp. Med. 40:30-33.

Wobeser, et al., 1976b: Wobeser, G., N.O. Nielsen, and B. Schiefer. 1976b. Mercury and mink II. Experimental methylmercury intoxication. Can. J. Comp. Med. 40:34-45.

Attachment 1. Previously Approved Site-Specific Wildlife Objectives

The State previously adopted, and EPA previously approved, seven groups of site-specific fish tissue water quality objectives for mercury for the protection of human health and/or wildlife, and one set of site-specific objectives reflecting natural background conditions for an area naturally-enriched with mercury, a small segment of lower Sulphur Creek.

The State-adopted, EPA-approved site-specific mercury objectives are summarized in Table 1.

Table 1. State-Adopted and EPA-Approved Mercury Water Quality Objectives

Applicable Water Bodies	State Adoption /EPA Approval	Type	Definition (all values in average wet weight)
San Francisco Bay (all segments including the Delta within the San Francisco Bay Regional Water Quality Control Board)*	State Adoption 2007; EPA Approval 02/12/2008	Human Health (HH)	**0.2 mg/kg** mercury in edible portions of trophic level 3 and trophic level 4 fish 250 – 1350 mm total length (Specific species and sizes in Plan)
		Aquatic Life (AL) Wildlife (WL)	**0.03 mg/kg** mercury in whole fish 30 – 50 mm total length
Sacramento-San Joaquin Delta, including Yolo Bypass (all segments)	State Adoption 2011; EPA Approval 10/20/2011	HH, AL, and WL	**0.24 mg/kg** methylmercury in muscle of trophic level 4 fish 150 – 500 mm total length **0.08 mg/kg** methylmercury in muscle of trophic level 3 fish 150 – 500 mm total length (Specific species and sizes in Plan)
		AL and WL	**0.03 mg/kg** methylmercury in whole fish less than 50 mm in length (Specific species and sizes in Plan)
Freshwater portions of Walker Creek, Soulajule Reservoir, and all tributary waters (Arroyo Sausal, Salmon Creek, Chileno Creek, and Keyes Creek)	State Adoption 2008; EPA Approval 09/29/2008	AL and WL	**0.1 mg/kg** methylmercury in whole fish 150 – 350 mm total length **0.05 mg/kg** methylmercury in whole fish 50 – 150 mm total length
Sulphur Creek, from Schoolhouse Canyon to its confluence with Bear Creek	State Adoption 2008; EPA Approval 09/04/2009	(Naturally occurring, mercury-enriched background conditions including geothermal waters; waters do not support fish)	During low flow conditions (less than 3 cfs), instantaneous maximum total mercury concentration of 1800 ng/L; During high-flow conditions (greater than 3 cfs), instantaneous maximum ratio of total mercury to total suspended solids of 35 mg/kg.
Clear Lake	State Adoption 2003; EPA Approval 09/26/2003	HH, AL, and WL	**0.19 mg/kg** methylmercury in the muscle of trophic level 4 fish 200-400 mm in total length **0.09 mg/kg** methylmercury in the muscle of trophic level 3 fish < 300 mm total length (Specific species and sizes in Plan)
Cache Creek (including North Fork); Bear Creek; Harley Gulch	State Adoption 2006; EPA Approval 02/06/2007	HH, AL, and WL	Cache Creek and Bear Creek: **0.23 mg/kg** methylmercury in muscle of trophic level 4 fish 250-350 mm total length **0.12 mg/kg** methylmercury in muscle of trophic level 3 fish 250-350 mm total length (Specific species and sizes in Plan)
		AL and WL	Harley Gulch: **0.05 mg/kg** methylmercury in whole fish trophic level 2-3 fish 75-100 mm total length (Specific species and sizes in Plan)
Waters of Guadalupe River Watershed except Los Gatos Creek and its tributaries upstream of Vasona Dam, Lake Elsman, Lexington Reservoir, and Vasona Lake	State Adoption 2009; EPA Approval 06/01/2010	AL and WL	**0.01 mg/kg** methylmercury in whole fish trophic level 3 fish > 150-350 mm total length **0.05 mg/kg** methylmercury in whole fish trophic level 3 fish 50-150 mm total length

* The State vacated the existing water column objective of 0.025 µg/L total mercury as a 4-day average, in waters of San Francisco Bay north of the Dumbarton Bridge.

United States
Environmental Protection
Agency

Region 10

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Columbia River Basin:
State of the River Report for Toxics
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www.epa.gov/region10/columbia

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Acronyms

BMP	best management practice
BPA	Bonneville Power Administration
CRITFC	Columbia River Inter-Tribal Fish Commission
DDD	dichlorophenyldichloroethane
DDE	dichlorophenyldichloroethylene
DDT	dichlorodiphenyltrichloroethane
DOE	U.S. Department of Energy
EPA	U.S. Environmental Protection Agency
IDEQ	Idaho Department of Environmental Quality
LCREP	Lower Columbia River Estuary Partnership
NOAA	National Oceanic Atmospheric Administration
NPCC	Northwest Power and Conservation Council
NPDES	National Pollutant Discharge Elimination System
ODEQ	Oregon Department of Environmental Quality
OSU	Oregon State University
PAH	polycyclic aromatic hydrocarbon
PBDEs	polybrominated diphenyl ethers
PBT	persistent, bioaccumulative, and toxic contaminant
PCBs	polychlorinated biphenyls
PNNL	Pacific Northwest National Laboratory
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PSP	Pesticide Stewardship Partnership
TMDL	total maximum daily load
TRI	Toxics Release Inventory
UC	University of California
U.S.	United States
USACE	U.S. Army Corps of Engineers
USDOE	see DOE
USEPA	see EPA
USFWS	U.S. Fish and Wildlife Service
USGS	U.S. Geological Survey
WADOE	Washington Department of Ecology
WADOH	Washington Department of Health
WDFW	Washington Department of Fish and Wildlife

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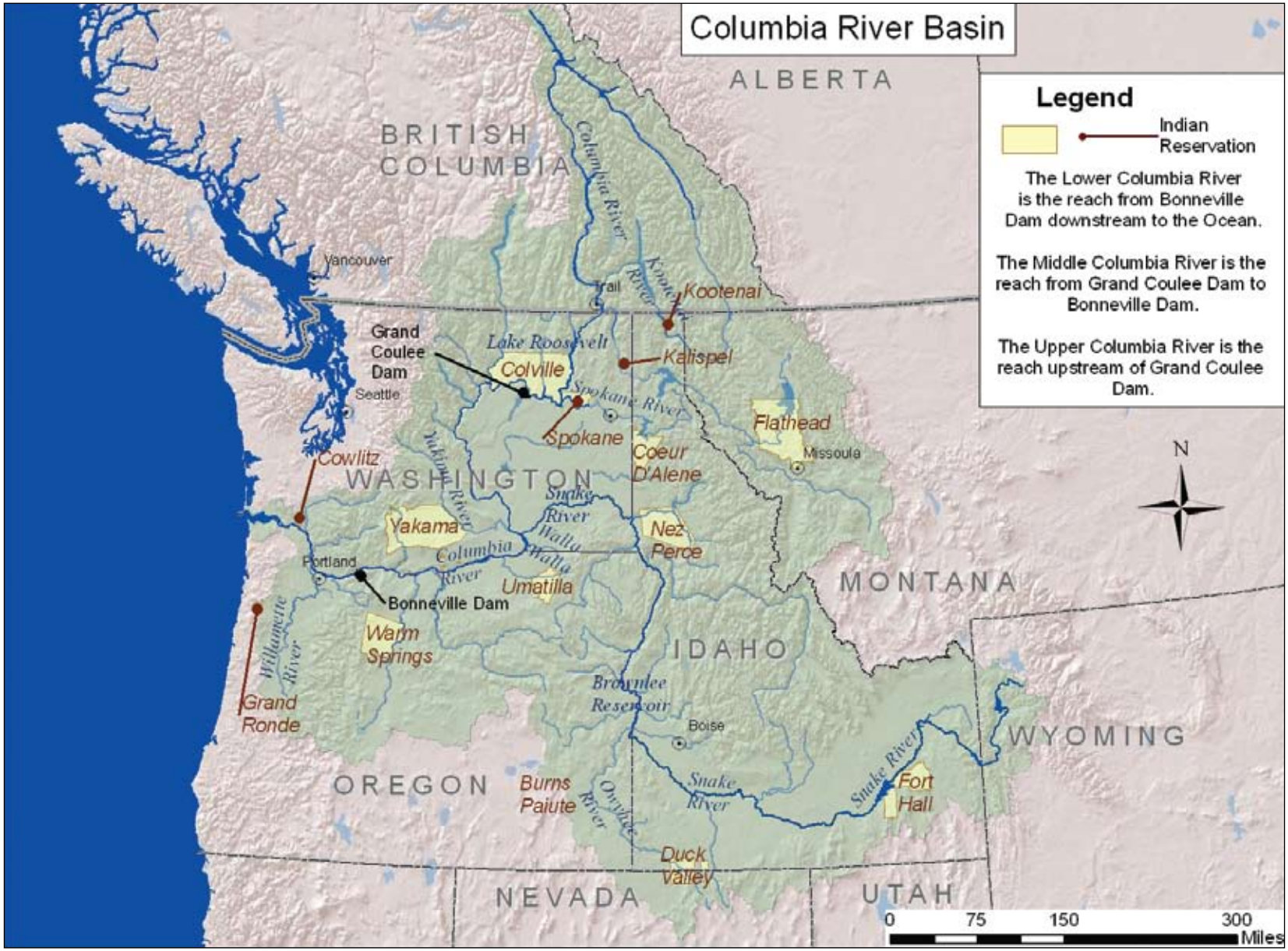


The Columbia River Basin

1.0 Executive Summary

The Columbia River Basin, one of the world's great river basins, is contaminated with many toxic contaminants, some of which are moving through the food web. These toxics in the air, water, and soil threaten the health of people, fish, and wildlife inhabiting the Basin.

In this report, the U.S. Environmental Protection Agency (EPA), Region 10, summarizes what we currently know about four main contaminants in the Basin and the risks they pose to people, fish, and wildlife. We also identify major gaps in current information that we must fill to understand and reduce these contaminants. Current information in the Basin indicates that toxics are a health concern for people, fish, and wildlife, but this information is sparse. In many locations, toxics have not been monitored at all. We do not have enough information in the majority of the Basin to know whether contaminant levels are increasing or decreasing over time. We need to fill these information gaps to understand the impacts on the ecosystem and to plan and prioritize toxics reduction actions.

This report focuses primarily on the following four contaminants: mercury, dichlorodiphenyltrichloroethane (DDT) and its breakdown products, polychlorinated biphenyls (PCBs), and polybrominated diphenyl ether (PBDE) flame retardants. We focus on these contaminants because they are found throughout the Basin at levels that could adversely impact people, fish, and wildlife. Many other contaminants are found in the Basin, including arsenic, dioxins, radionuclides, lead, pesticides, industrial chemicals, and "emerging contaminants" such as pharmaceuticals found in wastewater. This report does not focus on those contaminants, in part because there is a lack of widespread information on their presence in the Basin.

Mercury contaminates the Basin from industrial and energy-related activities occurring within and outside of the Basin. Mercury poses a special challenge because much of the Basin's mercury pollution comes from sources outside of the Basin via atmospheric deposition. At a watershed scale, however, local and regional sources can be significant contributors of mercury to the Basin. Fish consumption advisories for mercury continue to be issued in every state throughout the Basin.

The pesticide DDT and industrial chemicals known as PCBs have been banned since the 1970s, and reduction efforts have lowered their levels in the environment. Unfortunately, these chemicals persist in the environment and continue to pollute the Basin's waterbodies from various sources, including stormwater and agricultural land runoff and hazardous waste releases. In many areas, DDT and PCB concentrations still exceed levels of concern, and fish consumption advisories for these contaminants continue to be issued in every state throughout the Basin.

PBDE flame retardants and other emerging contaminants of concern—such as pharmaceuticals and personal care products—are a growing concern because their levels are increasing in fish and wildlife throughout the Basin. We are just beginning to conduct the research needed to better understand the impacts to the ecosystem from emerging contaminants.

This report provides preliminary information on the presence of mercury, DDT, PCBs, and PBDEs in the following species: juvenile salmon; resident fish (sucker, bass, and mountain whitefish); sturgeon; predatory birds (osprey and bald eagles); aquatic mammals (mink and otter); and sediment-dwelling shellfish (Asian clams). These species can help us understand trends in the levels of toxics in the Basin and judge the effectiveness of toxics reduction efforts.

Some initial steps to address the problem of toxics have already been taken. In 2005, EPA joined other federal, state, tribal, local, and nonprofit partners to form the Columbia River Toxics Reduction Working Group to better coordinate toxics reduction work and share information. The goal of the Working Group is to reduce toxics in the Columbia River Basin and prevent further contamination. This *State of the River Report for Toxics* was identified as a priority by this multi-stakeholder group and was prepared under the leadership of EPA Region 10 with the support and guidance of the Working Group.

Meanwhile, there are many ongoing efforts to reduce toxics in the Basin. Some examples include erosion control efforts in the Yakima Basin; Pesticide Stewardship Partnerships in the Hood River and Walla Walla Basins; PCB cleanup at Bonneville Dam; legacy pesticide collection throughout the Basin;

and investigation and cleanup of the Portland Harbor, Hanford, and Upper Columbia/Lake Roosevelt contamination sites. These and other combined efforts have reduced toxics over the years, but we still need to further reduce toxics to make the Basin a healthier place for people, fish, and wildlife.

To ensure a more coordinated strategy, EPA and our Working Group partners developed a set of six broad Toxics Reduction Initiatives needed to reduce toxics in the Basin. Over the next year, the Working Group will develop a detailed work plan to provide a roadmap for future reduction efforts with input from Basin citizens; local watershed councils; Basin communities and other entities; and tribal, federal, and state governments.

Reducing toxics in the Basin will require a comprehensive, coordinated effort by all levels of government, nongovernmental organizations, and the public. The problems are too large, widespread, and complex to be solved by only one organization. Our hope is that this report and the subsequent toxics reduction work plan will help us make this ecosystem healthier for all who live, work, and play in the Basin.

2.0 Introduction

The Columbia River Basin is one of the world's great river basins in terms of its land area and river volume, as well as its environmental and cultural significance. However, public and scientific concern about the health of the Basin ecosystem is increasing, especially with regard to adverse impacts on the Basin associated with the presence of toxic contaminants. A full understanding of the toxics problem is essential because the health of the Basin's ecosystem is critical to the approximately 8 million people who inhabit the Basin and depend on its resources for their health and livelihood. [1] The health of the ecosystem is also critical to the survival of the hundreds of fish and wildlife species that inhabit the Basin. In this *State of the River Report for Toxics*, we make our first attempt to describe the risks to the Basin's human and animal communities from toxics and to set forth current and future efforts needed to reduce toxics.

The Basin drains about 259,000 square miles across seven U.S. states and British Columbia, Canada. Of that total, about 219,400 square miles, or 85 percent of the Pacific Northwest region, are in the United States; the remaining 39,500 square miles are in Canada. [2] The Basin's rivers and streams carry the fourth largest volume of runoff in North America. The Columbia River begins at Columbia Lake in the Canadian Rockies and travels 1,243 miles over 14 dams to reach the Pacific Ocean a hundred miles downstream from Portland, Oregon. The River's final 300 miles, including the dramatic Columbia River Gorge Scenic Area, form the border between Washington and Oregon. In this report, the Lower Columbia River is considered to be the reach from Bonneville Dam downstream to the Pacific Ocean, the Middle Columbia River is considered to be the reach from Bonneville Dam upstream to Grand Coulee Dam, and the Upper Columbia River is considered to be the reach above Grand Coulee Dam.

Major tributaries to the Columbia River include the Snake, Willamette, Spokane, Deschutes, Yakima, Wenatchee, John Day, Umatilla, Walla Walla, Pend Oreille/Clark Fork, Okanogan, Kettle, Methow, Kootenai, Flathead, Grande Ronde, Lewis, Cowlitz, Salmon, Clearwater, Owyhee, and Klickitat Rivers. The Snake River is the largest tributary to the Columbia River, with a drainage area of 108,500 square miles, or 49 percent of the U.S. portion of the watershed. Another major tributary is the Willamette River, which drains 11,200 square miles and is located entirely within the State of Oregon. [2]

The Basin's salmon and steelhead runs were once the largest runs in the world, with an estimated peak of between 10 million and 16 million fish returning to the Basin annually to about 1 million upriver adult salmon passing Bonneville Dam in recent years. [3] For thousands of years, the tribal people of the Basin have depended on these salmon runs and other native fish for physical, spiritual, and cultural sustenance. Bald eagles, osprey, bears, and many other animals also rely on fish from the Columbia River and its tributaries to survive and feed their young. Historically, the large annual returns of adult salmon and steelhead have contributed important marine nutrients to the ecosystems of the interior Columbia River Basin. The Basin is also economically vital to many Pacific Northwest industries such as sport and commercial fishing, agriculture, transportation, recreation, and tourism. Throughout history, and up to the present day, the Basin has supported settlement and development, agriculture, transportation, and recreation.

There are more than 370 major dams on tributaries of the Columbia River Basin. [4] With its many major federal and nonfederal hydropower dams, the River is one of the most intensive hydroelectric developments in the world. About 65 percent (approximately 33,000 megawatts) of the Pacific Northwest's generating capacity comes from hydroelectric dams. Under normal precipitation, the dams produce about three-quarters (16,200 average megawatts) of the region's electricity. Some of the other major uses of the multi-purpose dams on the Columbia and Snake Rivers include flood control, commercial navigation, irrigation, and recreation. [3]

A National Priority

In 2006, EPA designated the Columbia River Basin as a Critical Large Aquatic Ecosystem in our *2006-2011 Strategic Plan*. [5] The Plan's Goal 4, Healthy Communities and Ecosystems, is "to protect, sustain, or restore the health of people, communities, and ecosystems using integrated and comprehensive approaches and partnerships."

The Columbia River Basin goal states:

> "By 2011, prevent water pollution and improve and protect water quality and ecosystems in the Columbia River Basin to reduce risks to human health and the environment."

The focus of the *2006-2011 Strategic Plan* was achieving more measurable environmental results. Working with state, tribal, and local partners, we selected the following strategic targets for the Columbia River Basin:

- By 2011, protect, enhance, or restore 13,000 acres of wetland habitat and 3,000 acres of upland habitat in the Lower Columbia River watershed.
- By 2011, clean up 150 acres of known highly contaminated sediments in the Lower Columbia River Basin, including Portland Harbor.
- By 2011, demonstrate a 10 percent reduction in mean concentration of contaminants of concern found in water and fish tissue. Contaminants of concern include chlorpyrifos and azinphos methyl in the Little Walla Walla River, DDT in the Walla Walla and Yakima Rivers, and DDT and PCBs in the mainstem.

We selected these targets because historical data were available and each represented measurable outcomes for reduction of toxics in the Basin. Meeting these targets and the overarching goal depends on the states, tribes, local governments, federal government, and nongovernmental agencies working together to improve the health of the Columbia River Basin.

The Story of Contamination in the Columbia River Basin

Fish, wildlife, and people are exposed to many contaminants polluting the water and sediment of the Columbia River Basin. These contaminants come from current and past industrial discharges (point sources) to the air, land, and water and from more widespread sources such as runoff from farms and roads (nonpoint sources) and atmospheric deposition. Some contaminants, such as mercury, also come from natural sources. Even when released in small amounts, some of these contaminants can build up over time to toxic levels in plants and animals.

In 1992, an EPA national survey of contaminants in fish in the United States alerted EPA and others to a potential health threat to tribal and other people who eat fish from the Columbia River Basin. [6] The Columbia River Inter-Tribal Fish Commission (CRITFC) and its four member tribes—the Confederated Tribes of the Warm Springs Reservation of Oregon, the Confederated Tribes and Bands of the Yakama Nation, the Confederated Tribes of the Umatilla Indian Reservation, and Nez Perce Tribe—were concerned for their tribal members who consume fish.

To evaluate the likelihood that tribal people may be exposed to high levels of contaminants in fish, EPA funded the CRITFC tribes to conduct a Columbia River Basin tribal fish consumption survey, which was then followed by an EPA and tribal study of contaminant levels in fish caught at traditional tribal fishing sites. [7,8] The consumption survey showed that the tribal members were

Human activities have contributed many toxic contaminants to the Columbia River Basin over the last 150 years:

- Dioxins, PCBs, metals, and other toxic chemicals were spilled and dumped in Portland Harbor. The sources: boat-building, steel-milling, and sewer discharges.
- "Legacy pollutants"—chemicals banned in the 1970s such as PCBs and chlorinated pesticides such as DDT—still contaminate the river. The sources: farmland, roads, construction sites, and stormwater runoff.
- Newer chemicals, including modern pesticides, flame retardants such as PBDEs, pharmaceuticals, and personal care products, contaminate the river. The sources: runoff and sewers.
- Metals wash into Lake Roosevelt. The sources: metal smelters in Washington and British Columbia.
- Metals wash into the Spokane River. The source: mines in northern Idaho.

eating six to eleven times more fish than EPA's estimated national average at that time of 6.5 grams per day. The fish contaminant study showed the presence of 92 contaminants in fish consumed by CRITFC tribal members and other people in the Columbia River Basin. Some of these contaminant levels were above the levels of concerns for aquatic life or human health. [8] Contaminants measured in Columbia River fish included PCBs, dioxins, furans, arsenic, mercury, and DDE, a toxic breakdown product of the pesticide DDT.

The Origin and Purpose of the Columbia River Toxics Reduction Working Group

Over the past two decades, much information was collected on the levels of contaminants in water, sediment, and fish in the Columbia River Basin. The result was an accumulation of scattered data that needed to be compiled into a Basin-wide report of the potential impacts from contaminants to people, fish, and wildlife. In 2005, EPA joined other federal, state, tribal, local, and non-profit partners to form the Columbia River Toxics Reduction Working Group to better coordinate this work and share information. Our goal is to reduce toxics in the Basin and prevent further contamination. This goal includes reducing toxics in the plants and animals that people eat and ensuring the survival, reproduction, and growth of fish and wildlife in the Basin.

One of the first actions this multi-stakeholder group identified was the development of a report for the Columbia River Basin describing the state of the River. The Working Group recognized toxics as one of several important factors affecting the health of the Basin's people, plants, and animals. We also recognized that toxics had received less attention than other factors and that a report on the influence of toxics was a good first step in understanding the health of the Basin's ecosystem.

This *State of the River Report for Toxics* was prepared under the leadership of EPA Region 10 with the support and guidance of the Working Group. This report sets in motion the process by which we will address the following questions:

- Which toxics are we most concerned about in the Columbia River Basin, and why? Which toxics are the highest priority for cleanup?
- Where are the toxics coming from? How can they be controlled and cleaned up? How can we prevent contamination in the future?
- What can indicator species tell us about the health of the Columbia River Basin? What indicator species should we use to evaluate the health of the ecosystem? Is the health of the ecosystem improving or declining? What additional information do we need to collect so that we can determine changes over time to better understand and deal with the toxics problem?
- What toxics reduction actions are currently under way? Have they been successful? What actions are planned to further reduce toxics?
- What are the next steps to improve the health of the Columbia River Basin ecosystem? What are the short- and long-term monitoring and research needs?

This report will be used to inform people, communities, and decision-makers in the Basin about the toxics problem and to begin a dialogue to identify potential solutions for improving the Basin's health.

VISIT THE WEB In addition to this report, EPA's Columbia River Basin website (http://www.epa.gov/region10/columbia) will provide more detailed and up-to-date information on the health of the Columbia River Basin as work continues.

3.0
Toxic Contaminants

What are Toxic Contaminants?

Toxic contaminants (or toxics) are chemicals introduced to the environment in amounts that can be harmful to fish, wildlife, or people. Some are naturally occurring, but many of these contaminants were manufactured for use in industry, agriculture, or for personal uses such as hygiene and medical care. These synthetic and naturally occurring chemicals can be concentrated to toxic levels and transported to streams through a combination of human activities such as mining or wastewater treatment and through natural processes such as erosion (Figure 3.1).

The fate of a contaminant is determined by its properties—for example, whether the contaminant mixes readily with water or sediment particles, or whether it changes form when exposed to sunlight, bacteria, or heat. A contaminant's location and level of concentration in a river help determine whether fish, wildlife, and people are exposed to it and, if so, whether they experience harmful health effects.

Why are Persistent Toxics a Concern?

Chemicals with well-known effects are generally those chemicals that remain in the environment for a long time (persistent contaminants), contaminate food sources, and increase in concentration in fish and birds. Animals can take in these contaminants directly while foraging for food or drinking water, or they can eat other animals and plants that have absorbed the contaminants. Many contaminants break down slowly, so they accumulate and concentrate in plants, wildlife, and people. The concentration of persistent contaminants through water, sediment, and food sources and within a plant or animal is called *bioaccumulation*. An example of a persistent chemical in the Columbia River is DDT and its breakdown product DDE, both of which are still present in the River nearly 40 years after DDT was banned.

Contaminants in water and sediment are absorbed by microscopic plants and animals, called phytoplankton and zooplankton, as they take in food and water. Many of these chemicals are not easily metabolized, so they persist in living organisms and concentrations build up in their tissues. Plankton, which are

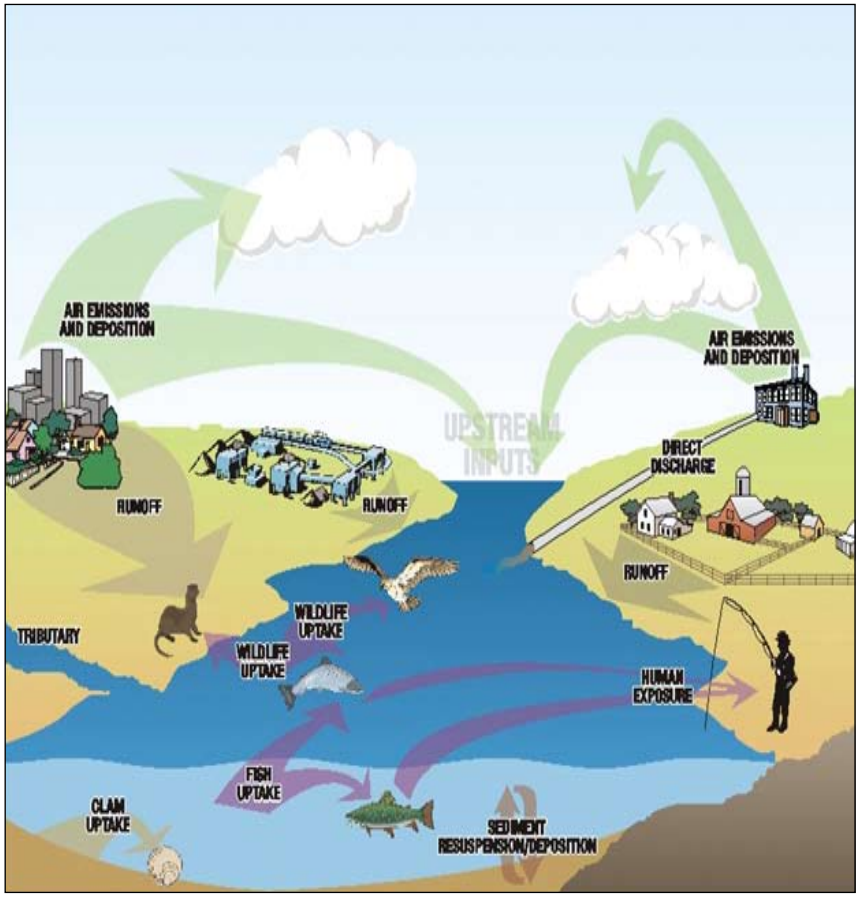


Figure 3.1: Toxic Contaminant Pathways in the Environment

at the bottom of the food web, carry the toxic burden all their lives. As larger animals eat the plankton, the accumulated chemicals are absorbed into each animal's body. Fish and other animals eat the plants, microorganisms, and small fish; the chemical moves into their bodies, and ultimately into larger fish-

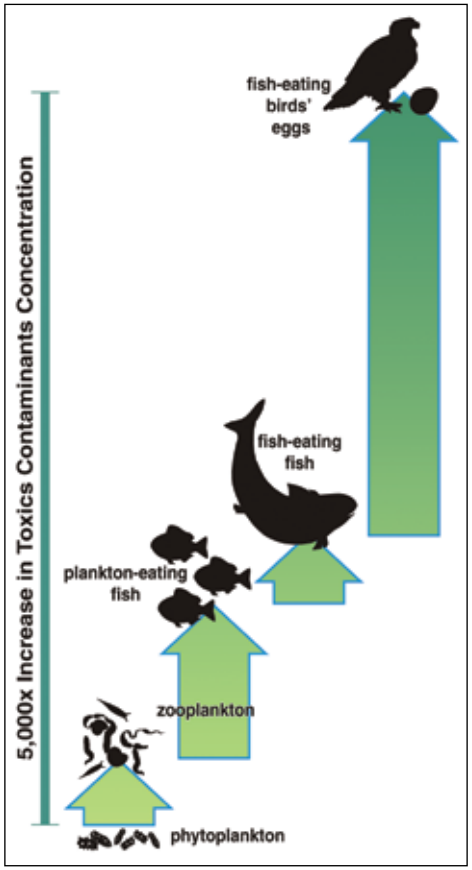


Figure 3.2: Persistent contaminants biomagnify, increasing in concentration up the food web. The highest biomagnification levels can be found in the eggs of fish-eating birds.

eating birds and mammals higher in the food web. This is how contaminant concentrations exponentially increase in fish and fish-eating animals at levels much higher than the concentrations found in the waters the fish live in. Through this *biomagnification* process, top predators, including birds of prey and humans, can accumulate contaminants in higher concentrations than those found in the plants and animals they consume (Figure 3.2). This toxic load builds up in their bodies throughout their lives.

What are the Contaminants of Concern in the Columbia River Basin?

While many contaminants have the potential to be of concern, this report focuses primarily on four contaminants: mercury (including methylmercury); DDT and its breakdown products; PCBs; and PBDEs.

These contaminants are of primary concern because (1) they are widely distributed throughout the Basin; (2) they may have adverse effects on wildlife, fish, and people; (3) they are found at levels of concern in many locations throughout the Basin; and (4) there is an opportunity to build on current efforts to reduce these contaminants within the Basin. [1]

In addition to these four contaminants, many other contaminants of concern were also identified in the Basin. These included metals such as arsenic and lead; radionuclides; several types of pesticides, including current-use pesticides; industrial chemicals; combustion byproducts such as dioxin; and "emerging contaminants" such as pharmaceuticals and personal care products. These contaminants are not the focus of this report, either because there is a lack of widespread information on their presence in the Basin or because they are best suited to more geographically targeted studies within the Basin.

VISIT THE WEB For more information on biomagnification, go to: http://toxics.usgs.gov/definitions/biomagnification.html.

Which Contaminants are Found in People?

Two studies recently investigated the amount and type of toxic contaminants found in people. In 2005, ten Washington residents volunteered to have their hair, blood, and urine tested for the presence of toxics as part of the "Pollution in People" investigative study by the Toxic-Free Legacy Coalition. [2] Each person tested positive for at least 26, and as many as 39, of the 66 toxics tested for, including common pesticides; plasticizers and fragrances found in vinyl, toys, and personal care products; flame retardants found in electronics, mattresses, and furniture; lead, mercury, and arsenic; and both DDT and PCBs.

In 2007, ten Oregon residents representing a diverse group of people from rural and urban areas throughout the state volunteered to have their bodies tested in a study of chemicals in people conducted by the Oregon Environmental Council and the Oregon Collaborative for Health and the Environment. [3] Each person had at least 9, and as many as 16, of the 29 toxics tested for in their bodies. Similar to the Washington study, these toxics included pesticides, mercury, plasticizers, and PCBs. Every participant had mercury, PCBs, and plasticizers in their blood.

While some of these toxics found in people may come from consuming fish or wildlife in the Columbia River Basin, the majority of the toxics found in people come from everyday activities and products such as food, cosmetics, home electronics, plastic products, and furniture. A greater effort to reduce toxics in the products we produce and consume will be needed to limit human exposure and intake of toxics and to reduce the amount of toxics that we put into the ecosystem.

What about Hanford and radionuclides?

For more than 40 years, the U.S. government produced plutonium for nuclear weapons at the Hanford Site along the Columbia River. Production began in 1944 as part of the Manhattan Project, the World War II effort to build an atomic bomb. Plutonium production ended and cleanup began at Hanford in 1989. Over 600 waste sites have been identified in the immediate vicinity of the nuclear reactors. These waste sites have contaminated the groundwater with radionuclides (nuclear waste) and toxic chemicals, above drinking water standards. In certain areas, the contaminated groundwater has reached the Columbia River.

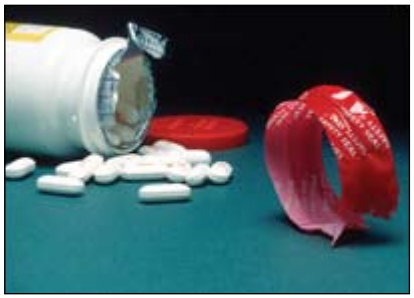
The waste sites and facilities near the River are undergoing an intensive investigation and cleanup effort. One part of that investigation will evaluate the risk to humans and other organisms in the Columbia River ecosystem from Hanford contaminants, including radionuclides, heavy metals, and some organic chemicals. The risk assessment results will be available in 2011. [5] Because of the ongoing investigation and cleanup efforts, this *State of the River Report for Toxics* does not focus on effects on the river from Hanford.

VISIT THE WEB For more information on the "Pollution in People" studies, visit the Toxic-Free Legacy Coalition: http://www.toxicfreelegacy.org/index.html and the Oregon Environmental Council: http://www.oeconline.org/pollutioninpeople.

VISIT THE WEB For more information about the Hanford cleanup, go to: http://yosemite.epa.gov/R10/CLEANUP.NSF/sites/Hanford and www.hanford.gov.

What are Emerging Contaminants of Concern?

A growing number of substances that we use every day, including pharmaceuticals, cosmetics, and personal care products, are turning up in our lakes and rivers, including the Columbia River. [4] These "emerging chemical contaminants" often occur at very low levels. With improved detection technologies, we are becoming more aware of their widespread distribution in the environment, and concerns are increasing about their potential impacts on fish and shellfish, wildlife, and human health. Hormones, antibiotics, and other drugs, which are commonly found in animal and human waste sources, are examples of emerging contaminants. Current-use pesticides and perfluorinated compounds—chemicals used in consumer products to make them stain- and stick-resistant—are other examples of emerging contaminants.



Emerging chemical contaminants include pharmaceuticals and other products that are not properly disposed. These contaminants are increasingly accumulating in waterways, including the Columbia River.

Although several of these emerging contaminants have been detected in water and sediment in the Lower Columbia River, information from locations elsewhere in the Basin is extremely limited. In response to these newly recognized contaminants, the U.S. Geological Survey (USGS) is sponsoring a four-year study in the Lower Columbia River addressing the movement of emerging contaminants from water to sediment, and through the food web to fish-eating birds, to evaluate the threat to the environment and human health.

Dioxins: A success story in toxics reductions

A 1987 EPA study showed unsafe levels of dioxin in fish from the Columbia River [6] Dioxins are persistent bioaccumulative toxins that can cause developmental and reproductive problems and potentially increase the risk of cancer. Dioxins are a byproduct of combustion and manufacturing processes, including bleaching paper pulp with chlorine.

In response to the study, in 1991 EPA collaborated with Oregon and Washington to require reductions in the amount of dioxin discharged by 13 paper mills to the Columbia, Snake, and Willamette Rivers. These pulp and paper mills subsequently changed their bleaching process, which reduced releases of dioxins into the Columbia River Basin.

Since 1991, dioxin concentrations in resident fish in the Columbia have decreased dramatically (Figure 3.3). [7,8,9,10,11,12] The dioxin content of osprey eggs has also shown a significant reduction in the lower part of the river. [13] However, dioxin is extremely persistent, and fish consumption advisories are still in place for some locations in the Basin.

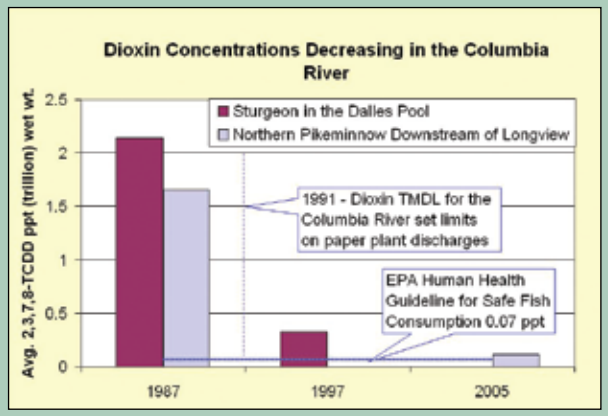


Figure 3.3: Dioxin levels in Columbia River fish have decreased significantly since pulp and paper mills changed their bleaching process, which reduced dioxin discharges in the early 1990s.

VISIT THE WEB For more information about dioxins in the Columbia River Basin, go to: www.deq.state.or.us/wq/TMDLs/columbia.htm and www.ecy.wa.gov/biblio/97342.html.

Fish Consumption Advisories for Toxics are Widespread across the Basin

When a river or lake becomes contaminated, it is not only an ecological loss but also a significant resource loss for people who depend on those fish for their diet. Fish consumption advisories are issued for lakes and rivers where various levels of fish consumption are no longer safe due to toxics in fish.

State health departments have issued public fish consumption advisories about the types and amounts of fish that are safe to eat from specific waters, including waters of the Columbia River Basin (Figure 3.4). In Washington, Oregon, Idaho, and Montana, people are advised to limit meals of fish such as bass, trout, walleye, and bottom fish from certain streams and lakes due to concerns about high levels of mercury, PCBs, and other contaminants. Because testing has shown high mercury concentrations in certain species, and because there is a lack of data from many water bodies, Washington has issued a statewide mercury advisory for consumption of bass and Idaho has issued a statewide mercury advisory for bass and walleye.

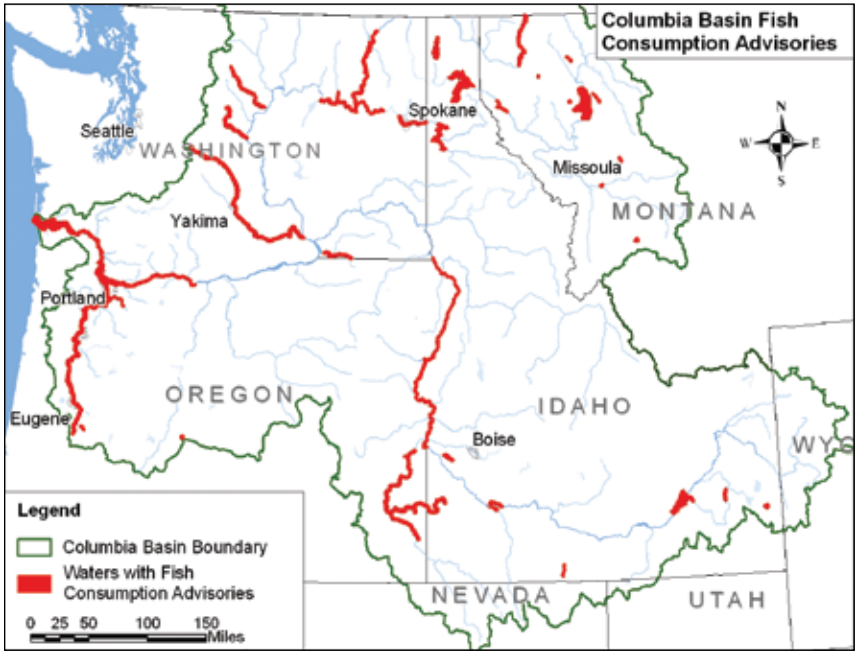


Figure 3.4: State-issued fish consumption advisories are in effect throughout the Columbia River Basin for certain contaminants and species. Not all waters have been tested, so the absence of an advisory does not necessarily mean it is safe to consume unlimited quantities of fish from untested waters.

VISIT THE WEB

Find information about fish consumption advisories for Washington: http://www.doh.wa.gov/ehp/oehas/fish/
Oregon: www.oregon.gov/DHS/ph/envtox/fishconsumption.html
Idaho: www.Idahohealth.org and Montana: www.dphhs.mt.gov/fish2005.pdf.

4.0 Indicators

What are Indicators?

Environmental indicators are tools used to help citizens and decision-makers better understand the health of the environment and whether we are reaching our environmental goals. Indicators may be specific organisms, specific media such as water or sediment, or a specific sampling location or contaminant. The indicators used in this report are animal species living in the Columbia River Basin or dependent on food from the River. Studying these species over time will help scientists track changes in the Basin's ecosystem.

Which Indicator Species are Used in this Report?

For this report, the following indicator species were selected to help assess the health of the Basin ecosystem: juvenile salmon; resident fish, both native and introduced (e.g., sucker, bass, and mountain whitefish); sturgeon; predatory birds (osprey and bald eagle); aquatic mammals (mink and otter), and sediment-dwelling shellfish (Asian clam).

Why were These Species Selected as Indicators for the Columbia River Basin?

The indicator species listed above were chosen for this report because they have some or most of the following characteristics:

- The species has a clear connection with important aspects of the Basin's ecosystem.
- Information is available to describe contaminant status and/or trend information for the species.
- The species can be used to track progress on toxics reduction activities.
- The species represents an important functional level (e.g. predator, prey) of the Basin's food web.
- The species may be compared with the same species living in other aquatic ecosystems.

Juvenile salmon

There are five species of salmon in the Basin: Chinook, coho, sockeye, chum, and pink salmon. Salmon are *anadromous*, meaning their eggs are laid and hatch in freshwater, and their young spend part of their early lives in freshwater before swimming to the ocean to grow and mature (Figure 4.1). Upon returning to their native stream, the adults spawn and then die. Cutthroat trout and steelhead are closely related to salmon. These two species can exhibit both anadromous and resident fish behaviors and are capable of spawning. In the 1990s, the federal fish and wildlife agencies listed several of the anadromous salmon species as threatened and/or endangered.

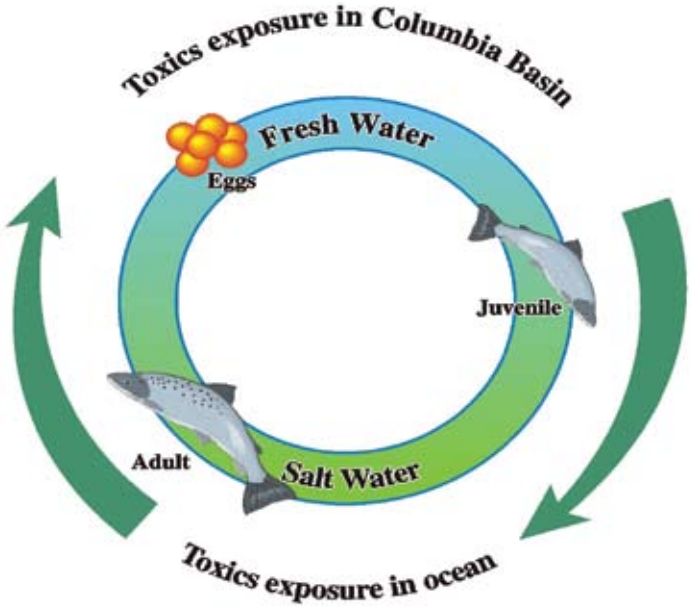


Figure 4.1: Salmon spend a significant part of their adult lives in the ocean. Therefore, it is primarily in their juvenile stages that they are exposed to contaminants in the Columbia River Basin.

Salmon as a Food Source

Because adult salmon spend the majority of their lives in the ocean, the percentage of contaminant accumulation in their tissue from sources in the Columbia River Basin cannot be determined. Regardless of the source, contaminants in adult salmon could pose a threat to people who consume large amounts of salmon, especially Columbia River Basin tribal people for whom the salmon is an important part of their culture and a major food source. In addition, some recreational anglers and their families may consume large amounts of salmon. Given this, it is important to ensure that both tribes and anglers have the most up-to-date information to make informed decisions on how much salmon can be safely consumed.

Pacific salmon die within days of digging their nests, or "redds," and mating. Their remains decompose, releasing nutrients for plants and other animals. Live and dead salmon are also important food for birds and mammals such as bald eagles, otters, and bears. In this way, salmon contribute to the health of freshwater ecosystems.

Juvenile salmon are an important indicator of ecosystem health in the Basin because: (1) they are relatively widespread throughout the Basin; (2) they both forage in the River system and serve as a major food source for larger fish, birds, and mammals; (3) they use many habitat types and therefore provide a means of assessing environmental conditions throughout the River system and estuary; (4) they go through physiological changes from juvenile to adult and therefore can be more susceptible to toxic contaminants; and (5) currently, 13 species of salmon and steelhead in the Basin are listed as either threatened or endangered under the Endangered Species Act.

The National Oceanic and Atmospheric Administration (NOAA) Fisheries and the University of California (UC) Davis are investigating how chemical contaminants affect juvenile salmon health and survival in the Lower Columbia River. In a recently published paper, they concluded that the adverse health effects of chemical contaminant exposure are similar to adverse health effects associated with passage through the hydropower system in the Columbia River. [1]

Resident fish

There are many native and nonnative resident fish species in the Basin, including rainbow trout, cutthroat trout, mountain whitefish, large scale sucker, bass, walleye, and northern pikeminnow. They are a common source of food for people and wildlife and are widely distributed throughout the Basin. Resident fish live their entire lives in the Basin and thus are exposed to contaminants present in the water and sediments through their food, by breathing in oxygenated water through their gills, and by continuous contact with the water and sediments. In many of the Basin's water bodies, these resident species have accumulated levels of some contaminants that are harmful to predators and to people.

Resident fish are useful indicators because: (1) they are widely distributed throughout the Basin; (2) most of the existing data on contaminants in the Basin are from resident fish species; (3) many species of resident fish spend their lives in relatively small areas, so their tissue concentrations are indicative of the contaminant loads in those areas; and (4) they occupy a central place in the food web, are exposed to contaminants through their diet, and in turn expose those who eat them, including people, to any accumulated contaminants.

VISIT THE WEB For more information about salmon in the Columbia River Basin, go to: http://www.nwr.noaa.gov/Salmon-Recovery-Planning/ESA-Recovery-Plans/Draft-Plans.cfm.

Sturgeon

White sturgeon are the largest freshwater fish in North America, occurring in Pacific Coast rivers from central California to Alaska's Aleutian Islands. Some white sturgeon spend their entire life cycle in freshwater, while others use estuarine or coastal saltwater resources for growth and food, only entering freshwater to reproduce.

White Sturgeon (photo courtesy of Gretchen Kruse, Free Run Aquatic Research)

White sturgeon inhabit the Columbia River and its larger tributaries, such as the Snake and Kootenai Rivers. Sturgeon can live 100 years and grow up to 1,500 pounds and 15 feet long. Sturgeon are primarily bottom-dwelling fish. Juvenile sturgeon feed primarily on plankton and aquatic insects, whereas adults feed mainly on live or decaying fish, aquatic insects, and shellfish (e.g., Asian clams).

Sturgeon are not reproducing successfully throughout the Columbia River system. In Canada's portion of the River, there has been no successful reproduction recorded in the wild over the last decade. For similar reasons, the Kootenai River population of white sturgeon has been listed on the federal endangered species list since 1994.

White sturgeon are a good Columbia River indicator species for several reasons: (1) they are widely distributed in large rivers of the Basin; (2) they are long-lived and thus have prolonged exposure to toxic contaminants; (3) sturgeon migration is curtailed by dams in some portions of the Basin, allowing for evaluation of local toxics effects; (4) they are near the top of the food web; and (5) effects of contaminants on sturgeon are likely similar for other benthic, bottom-dwelling species.

Predatory birds—osprey and bald eagle in the Lower Columbia River

Osprey and bald eagle are large birds of prey that live in much of the Basin, but they are concentrated in the Lower Columbia River. While the bald eagle is found exclusively in North America, the osprey has a nearly world-wide distribution. Bald eagles feed primarily on live or scavenged fish and aquatic birds, while the osprey has a diet almost exclusively of live fish captured near the nest.

Osprey and bald eagles are useful indicators for evaluating the health of an aquatic ecosystem for several reasons: (1) they are widely distributed; (2) they are long-lived (bald eagles, for instance, can live up to 28 years in the wild); (3) they primarily prey on fish and other aquatic predators, usually near their nests; and (4) they are at the top of the food web and are therefore exposed to high concentrations of contaminants through their diet.

Osprey

Bald Eagle

(photos courtesy of NOAA/Dept. of Commerce)

Aquatic mammals—mink and river otter

Mink and river otter are members of the weasel family. They are excellent swimmers and are active predators that feed on fish, frogs, crayfish, and sometimes small mammals and waterfowl. The average lifespan of mink in the

Mink (photo courtesy of U.S. Forest Service)

North American River Otter (photo courtesy of USGS)

wild is three to six years, whereas river otter average over eight years. Both are found throughout the Basin in appropriate habitat; however, mink populations have not recovered from a decline in the 1950s and 1960s, even though suitable habitat is available for them in the Lower Columbia River.

Mink and otter are useful indicators of ecosystem health in the Basin because they: (1) prey on other aquatic species; (2) are particularly sensitive to contaminants which accumulate and can impact their reproduction; (3) have smaller home ranges compared to osprey and bald eagles; and (4) occur throughout the Basin.

Sediment-dwelling shellfish—Asian clam

First found in North America at Vancouver Island, British Columbia, in 1924, the nonnative, freshwater Asian clam is a small, light-colored bivalve now abundant throughout North America. It is widely distributed throughout a large portion of the Basin and has an average life span of three to five years. Located primarily in flat-bottom sand or clay areas, Asian clams feed by filtering particles from the surrounding water. They also routinely bury in the sediment for extended periods and filter sediment pore water.

Asian clams are a good indicator species for several reasons: (1) they are filter feeders and, like other freshwater shellfish, can collect and concentrate contaminants in their bodies; (2) they are not very mobile, so data on clams can be more useful to pinpoint the location where they were exposed to the contaminants than similar or more mobile species; (3) because of their distribution and feeding habits, they are a useful indicator of sediment and water quality conditions in the Basin; and (4) they occupy a lower position in the food web than other indicator species.

Lamprey

Pacific lamprey are scaleless, jawless fish that are culturally important to the Columbia River tribes. Lamprey have declined drastically in the past 20 years and are no longer found in many streams in their traditional range. Pacific lamprey spawn in freshwater streams. Juvenile lamprey (ammocoetes) spend their first five to seven years in the sediment as filter feeders. Adult lamprey migrate to the ocean, where they feed parasitically on other fish for up to three years before returning to freshwater streams to spawn.

Because lamprey spend their developing years in the Basin's streams, there are concerns that toxics may be a contributing factor in their declining numbers. Studies in locations outside the Columbia River Basin have documented the sensitivity of juvenile lamprey to toxics in their environment. [2,3] The unique life cycle of the lamprey with its potential for exposure to Basin contaminants distinguishes it as a potential indicator of ecosystem health. However, very little data have been collected on toxics in lamprey in the Columbia Basin. Because of this lack of data, lamprey are not discussed as an environmental indicator in this report. Given the cultural importance of lamprey to the Columbia River tribes, however, we will evaluate whether lamprey should be added as an indicator species after additional data on toxics in lamprey are collected and evaluated.

5.0
Status and Trends for Mercury, DDT, PCBs, and PBDEs

The contaminants discussed in this report—mercury, DDT, PCBs, and PBDEs—come from a variety of sources and can potentially result in health concerns for wildlife or people. Table 5.1 summarizes the sources and health concerns of these four contaminants.

In order to evaluate whether the toxics reduction efforts currently under way in the Basin are having an impact or if other activities are needed, it is important to understand whether the levels of contaminants are increasing or decreasing over time. While considerable information has been collected over the past 20 years, the data are limited with regard to whether the contaminants are increasing or decreasing Basin-wide. There is some trend information for specific areas of the Basin such as the Lower Columbia. While not comprehensive, this report highlights trend data when such data are available.

Table 5.1: Contaminants of concern summary

Contaminant	Sources/Pathways	Concern
Mercury	Atmospheric deposition from sources inside and outside the region is thought to be a major pathway for mercury. Other possible sources/ pathways include releases from past and current mining and smelting activities; erosion of native soils; agricultural activities; discharge of wastewater and stormwater; and resuspension and recirculation of sediments.	Mercury can cause neurological, developmental, and reproductive problems in people and animals.
DDT	DDT was banned in the United States in 1972, but DDT and its breakdown products are still found in the environment in sediments and soil. The main pathway to the River is via runoff from agricultural land.	DDT thins bird eggshells and causes reproductive and development problems. It is linked to cancer, liver disease, and hormone disruption in laboratory-test animals.
PCBs	PCBs were banned in the United States in 1976, but they are still widely found in the environment in fish tissue and sediments. Industrial spills and improper disposal are known sources locally, while incineration and atmospheric deposition bring PCBs from distant sources. Stormwater runoff and erosion may also be important pathways.	PCBs can harm immune systems, reproduction, and development; increase the risk of cancer; and disrupt hormone systems in both people and aquatic life.
PBDEs	PBDE flame retardants are present in many consumer products, including electronics, textiles, and plastics. There is limited information on the transport pathways to the River, but some possible pathways include atmospheric deposition, municipal and industrial wastewater, stormwater discharge, and runoff.	PBDEs accumulate in the environment, harming mammals' reproduction, development, and neurological systems. They can increase the risk of cancer and disrupt hormone systems.

VISIT THE WEB Additional information and updates about mercury, DDT, PCBs, and PBDEs can be found by visiting EPA's Columbia River website: http://www.epa.gov/region10/columbia.

Mercury: Most Fish Consumption Advisories in the Basin are due to High Concentrations of Mercury

Mercury can affect the nervous system and brain, and even low doses can impair the physical and mental development of human fetuses and infants exposed via the mother's diet. Fish consumption advisories generally discourage the consumption of larger fish and predatory fish, as they typically contain higher concentrations of mercury. Figure 5.1 shows mercury concentrations found in fish from U.S. waters in the Columbia River Basin.

As a metallic element, mercury is never destroyed, but cycles between a number of chemical and physical forms. Mercury in the aquatic environment can be converted by bacteria to a more toxic form, called methylmercury. This process is important because methylmercury can biomagnify, so predators at the top of the food web will have much higher concentrations of mercury in their bodies than are found in the surrounding water or the algae and insects at the base of the food web.

Methylmercury is the dominant form of mercury found in fish, and the concentrations of methylmercury found in fish are directly related to the amount available in the aquatic environment. The rate at which methylation of mercury occurs varies according to water body characteristics such as the amount of organic matter, sulfate, and iron present and the acidity, temperature, and water velocity.

Several pathways introduce mercury into the Columbia River Basin

Mercury enters the Columbia River and its tributaries via several pathways, including atmospheric deposition, runoff, wastewater discharges, industrial discharges, and mines. Based on available data, atmospheric deposition appears to be the major pathway for mercury loading to the Columbia River Basin. [1] Mercury air deposition includes both emissions from industrial facilities within and near the Basin and fallout from the pool of global mercury that has been transported from sources as far away as Asia and Europe.

EPA estimates that the total mercury air deposition in the Columbia River Basin is 11,500 pounds per year. [2] Approximately 84 percent of that load comes from global sources. At a watershed scale, however, local and regional sources

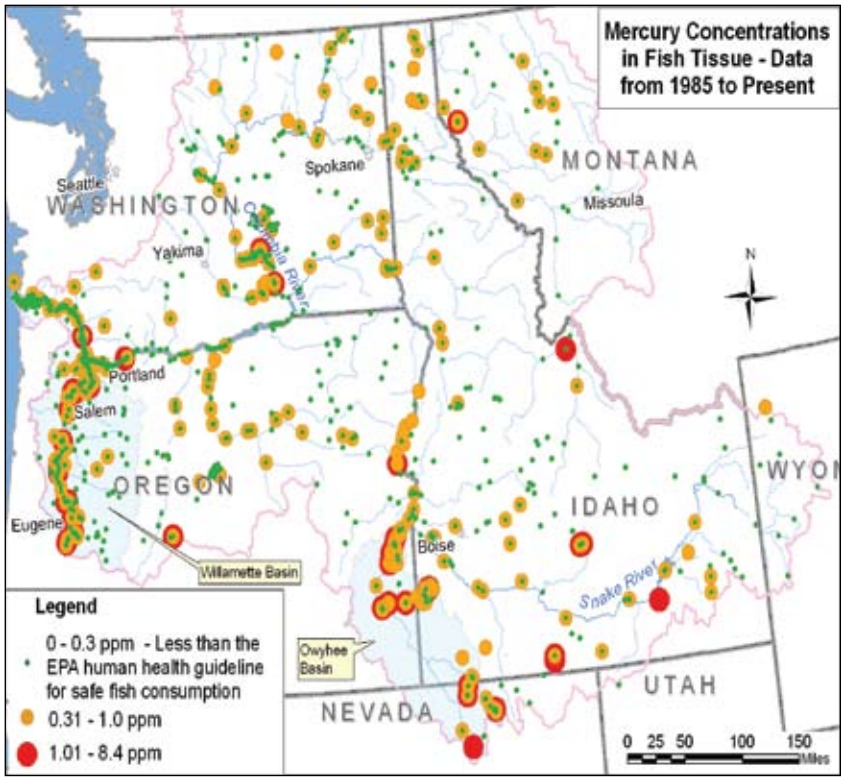


Figure 5.1: Seventy-five percent of fish consumption advisories in the Columbia River Basin are due to mercury contamination. In the fish tested, high levels of mercury have been consistently found downstream of historic mining areas in the Willamette and Owyhee River Basins. There is no information about mercury levels in fish from waters that are unmarked on the map.

can contribute the majority of mercury deposited on the local landscape. For example, a cement plant in Durkee, Oregon, emits more than 2,500 pounds of mercury per year. [3] Although just over 140 pounds of this amount are deposited in the sub-basin in which this plant is located, that deposition constitutes an estimated 62 percent of the air-deposited load in that area. [4]

As for regional sources, in northern Nevada near the Basin's southeast boundary, several gold mines emit mercury from their ore roasters. One of these mines discharges more than 1,700 pounds of mercury per year. [3] Although only part of this load ends up in the Columbia River Basin, almost 160 pounds are deposited in the nearby Upper Owyhee watershed in Idaho, accounting for 58 percent of the atmospheric mercury loading there. [4] In Idaho, the largest source of mercury emissions is an elemental phosphorus plant in Soda Springs. This plant emits more than 900 pounds per year [3] and contributes 36 percent of the mercury deposited in the adjacent watershed. [4]

Across the United States, coal-fired power plants are a major local source, but they are less significant sources in the Northwest because so few are located here. There is a single coal-fired power plant in the Columbia River Basin located near Boardman, in eastern Oregon. This plant emits about 168 pounds of mercury per year. [3] There are also three coal-fired power plants near the boundary of the Basin (one in Washington and two in Nevada) that could contribute some mercury load to the watershed, depending upon their emissions and prevailing wind patterns.

Not all of the mercury that falls onto land gets transported to water bodies. Forests and other undisturbed landscapes can retain mercury for years.

Other point sources directly discharge mercury to rivers and streams. Wastewater treatment plants, industrial discharges, and stormwater runoff from streets and other developed areas are more direct sources of mercury to streams than air deposition or erosion. These sources may be low in concentration, but high in volume. Nine of the 23 largest municipal and industrial wastewater point sources located in the U.S. portion of the Columbia River have reported discharging a total of 33 pounds of mercury per year. [5] This may be an underestimate, however, because mercury reporting is not always required and mercury detection limits are often too high to provide useful information. Although these sources contribute less mercury to the basin than the air pathway, they may be significant at a local scale because they discharge directly to water bodies. A smelter just north of the Canadian border directly discharged an average of 184 pounds of mercury per year to the Upper Columbia from 1994 through 1998. This load was reduced to an average of 38 pounds of mercury per year for the 1999-2007 time period. [6] Historic mercury and gold mining can also be important sources that load mercury directly to streams and have significant impacts at a watershed scale.

Mercury is also still found in several commonly used products such as fluorescent light tubes, compact fluorescent lamps, thermometers, thermostats, switches in vehicles, some batteries and pumps, and medical equipment such as blood pressure measuring devices. Although mercury has been or will be removed from some of these products, many of the older versions still contain mercury. If these older products are not handled and disposed of properly, they can add mercury to the environment.

Regional trends and spatial patterns of mercury levels in the Basin can be difficult to evaluate

Although data on mercury concentrations are available for resident fish species in the Basin from the 1960s to the present, there are few locations with consistent, comparable data from different time periods that can be used to evaluate changes in mercury concentrations over time. Two exceptions, noted in Figure 5.2, are mercury concentrations in northern pikeminnow from the Willamette River Basin and mercury concentrations in osprey eggs in the Lower Columbia River, both of which have been increasing in the last decade. [7,8,9] The osprey egg concentrations, however, were still below levels that are of concern in birds. Another study shows that mercury concentrations increased in pikeminnows (1.12 to 1.91 parts per million [ppm]) from the Upper Willamette River between 1993 and 2001. [10]

The Columbia River sturgeon population living in the pool behind Bonneville Dam has much higher concentrations of mercury in their livers than sturgeon in the estuary or other Columbia River reservoir pools. Sturgeon tissues from the Kootenai, Upper Columbia, and Snake Rivers contained mercury concentrations in the range of 0.02 to 0.6 ppm, but Bonneville pool sturgeon have mean concentrations of 4 ppm. [11,12,13,14] Also, high mercury levels in liver and other organs from Lower Columbia River white sturgeon are correlated with lower physical health indices and reproductive defects in the fish. [15,16,17,18,19]

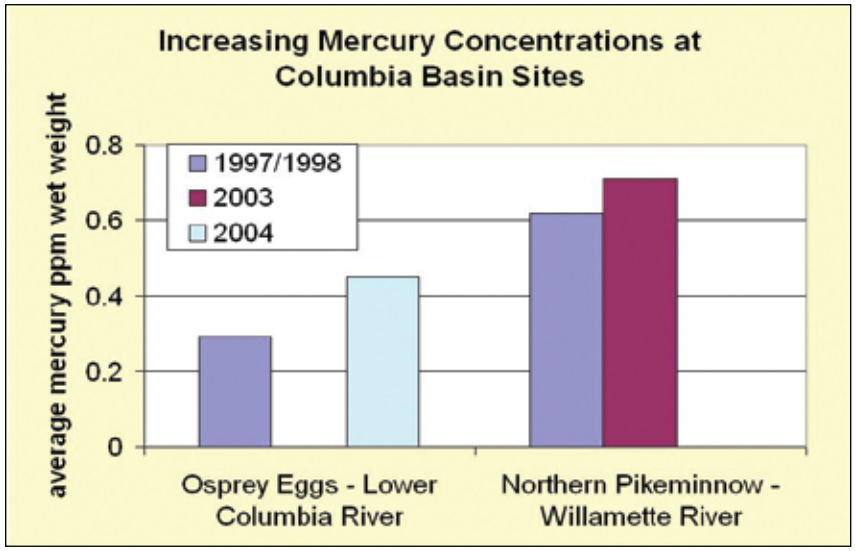


Figure 5.2: Mercury levels in Willamette River northern pikeminnow and Lower Columbia River osprey eggs have increased over the last decade. Mercury level trends have not been studied in other Columbia River Basin organisms over the

Mercury concentrations vary across the basin, but only in some cases are the sources known. For example, in reservoirs in the Owyhee River basin [20,21] and in the Snake River downstream of the Owyhee confluence, mercury levels are found above EPA's 0.3-ppm mercury human health guideline due to mercury used in gold mining there in the 1800s (Figure 5.3). [22,23,24,25,26,27,28,29]

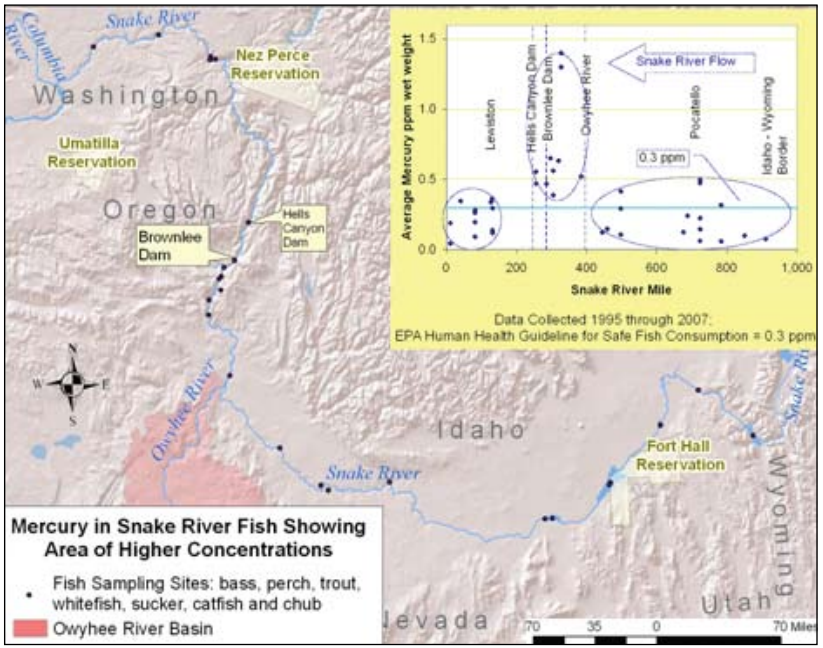


Figure 5.3: Mercury levels are highest in fish collected at Brownlee Dam reservoir, downstream from the Owyhee River inflow. The Owyhee River is contaminated by mercury from historic mining.

DDT: Banned in 1972, This Pesticide Still Poses a Threat to the Environment

DDT is the most well-known of a class of pesticides that were widely used from the 1940s until EPA banned them in the United States in 1972. However, DDT continues to be used in other parts of the world. DDT and its breakdown products—dichlorophenyldichloroethylene (DDE) and dichlorophenyldichloroethane (DDD)—have been linked to neurological and developmental disorders in birds and other animals. DDT has also been linked to eggshell thinning that caused declines in many bird species and inspired Rachel Carson's 1962 book *Silent Spring*, which documented detrimental effects of pesticides on bird species and ultimately led to the banning of DDT.

The chemical structure of DDT is very stable in the environment, which is why DDT and its breakdown products DDE and DDD continue to be an ecological and human health threat. Figure 5.4 shows DDE concentrations found in fish from U.S. waters in the Columbia River Basin.

Soil erosion from agricultural runoff is the main source of DDT into the Basin

The primary source of DDT to the Columbia River Basin is the considerable acreage of agricultural soils in which DDT accumulated over three decades of intensive use (1940s to early 1970s). DDT reaches the River when the soils are eroded by wind and water. Some irrigation practices increase soil erosion on agricultural lands. Other potential sources of DDT are areas where pesticides were handled or stored, such as barns or agricultural supply sheds, or areas where containers or unused product were disposed. The main pathway for these sources is erosion and runoff. Disturbance of contaminated sediments within the Columbia River and its tributaries may also release DDT to the water column, which can directly or indirectly be taken up by fish.

DDT levels are declining with better soil conservation practices, but DDT still exceeds human health levels of concern

The ban on DDT combined with significant improvements in soil conservation by farmers reduced DDT loading to the Columbia River Basin. [1] A number of state water quality improvement plans currently aim to reduce DDT compounds, and continued monitoring is critical to demonstrating the effectiveness of these actions.

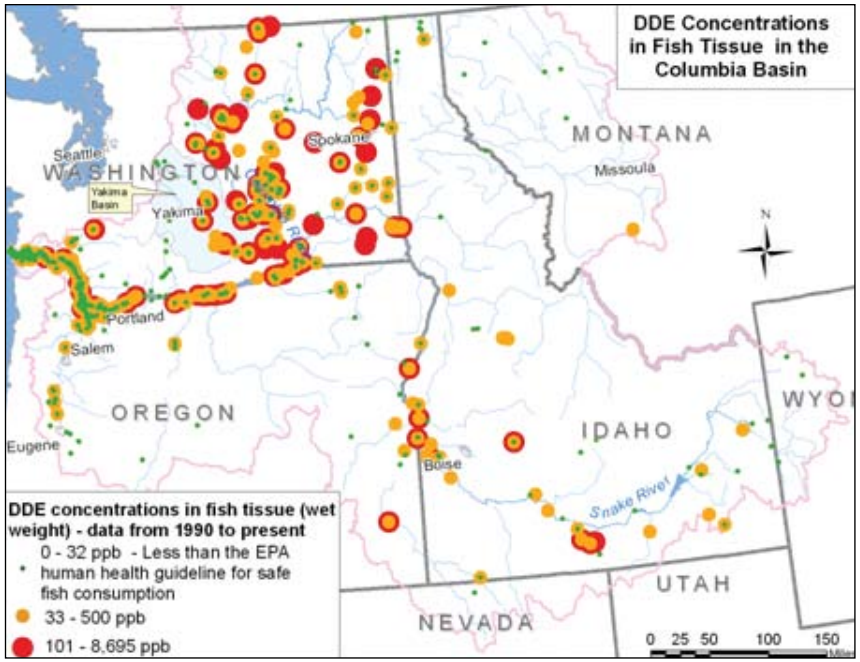


Figure 5.4: High levels of DDE in fish are found in areas where DDT pesticide use was historically high, such as in eastern Washington and the Snake River Plain. There is no information about DDE levels in fish from waters that are unmarked on the map.

Concentrations of DDT compounds in the Columbia River and its wildlife have decreased over the last 20 years. However, DDT is still regularly detected in the fish, plants, and sediments of the River and many of its tributaries, indicating that DDT continues to cycle through the food web. In addition, fish consumption advisories continue to be issued for DDT in Lake Chelan.

DDT levels have declined in several of the key species of resident fish in areas of the Columbia River Basin. DDT contamination has been most intensively studied in the Yakima River, which is a major tributary to the Columbia in Washington State and is in one of the most diverse agricultural areas of the country. [2] Data collected in the 1980s showed that fish in the Yakima River Basin had some of the highest concentrations of DDT in the nation. [3]

In the late 1990s, a partnership of farmers, irrigation districts, the Confederated Tribes and Bands of the Yakama Nation, and many governmental agencies initiated changes in farming and irrigation practices that have dramatically reduced erosion from farmland in the Yakima Basin (see Section 6.0 of this report). Sampling of resident fish conducted between 1996 and 2006 showed an overall decline in DDT levels in several species, including bass and sucker (Figure 5.5). [4,5]

By contrast, liver tissues from Columbia River white sturgeon residing in the pool upstream of Bonneville Dam contained much higher concentrations of DDT than other sub-populations of sturgeon residing in the Columbia River Basin (Figure 5.6). [6,7,8,9,10,11,12,13] The cause of these elevated concentrations is not known.

DDT is also a problem for fish-eating birds such as bald eagles and osprey. Severe declines in eagle populations in the Lower Columbia River occurred from the 1950s to1975. Studies conducted along the Lower Columbia River from 1980 to 1987 found elevated concentrations of DDE in bald eagles. [14] High concentrations of DDE are associated with eggshell thinning and low reproductive success.

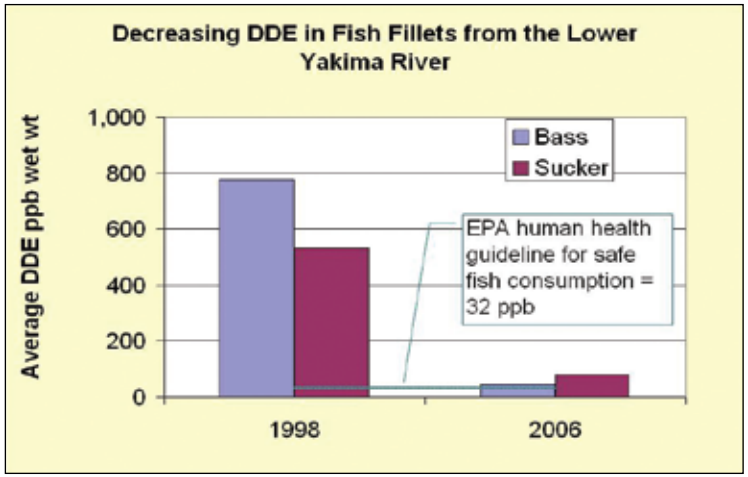


Figure 5.5: DDE levels in Yakima River fish have declined significantly since 1998.

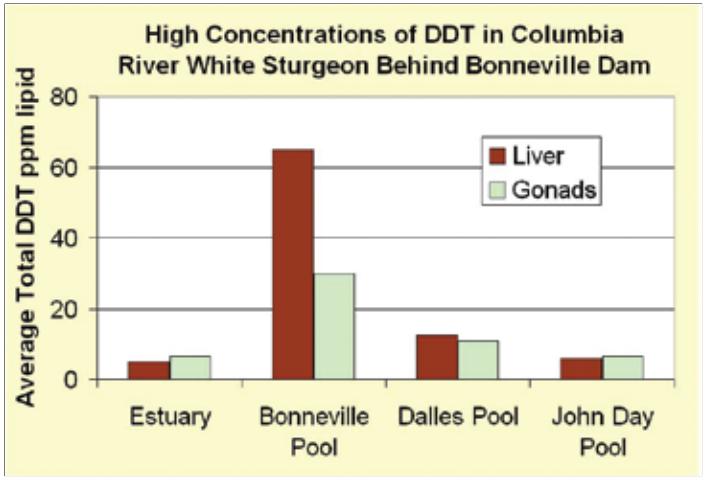


Figure 5.6: Sturgeon in the pool behind Bonneville Dam have much higher levels of DDT and other contaminants (such as mercury and PCBs) than do sturgeon downstream of the dam or sturgeon in pools behind upstream dams.

Successful reproduction of bald eagles along the Columbia River was also found to be considerably lower than the statewide average for Oregon. [15,16] DDE concentrations in Columbia River eagle eggs in the 1980s were the highest recorded for bald eagles in the western United States, surpassed only by levels found in eagle eggs from highly contaminated areas of the eastern United States. [14]

In a similar study in the mid-1990s, researchers found that total DDE concentrations in Columbia River eagle eggs declined significantly in comparison to concentrations found in the mid-1980s (Figure 5.7). [15,16]

Prior to the use of DDT, nesting osprey were common along the Lower Columbia and Willamette Rivers, [17] but populations declined dramatically from the 1950s to the 1970s. As with eagles, DDT was the primary cause of osprey population decline because of eggshell thinning. Figure 5.8 shows the

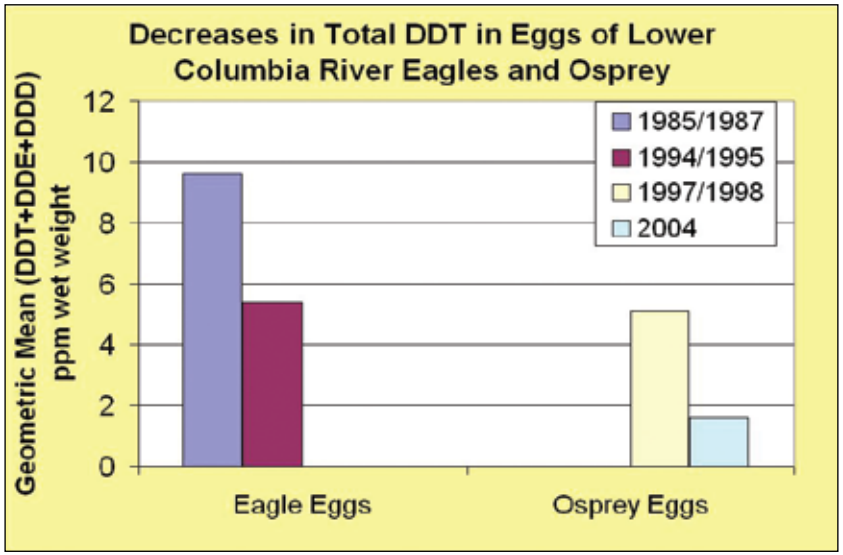
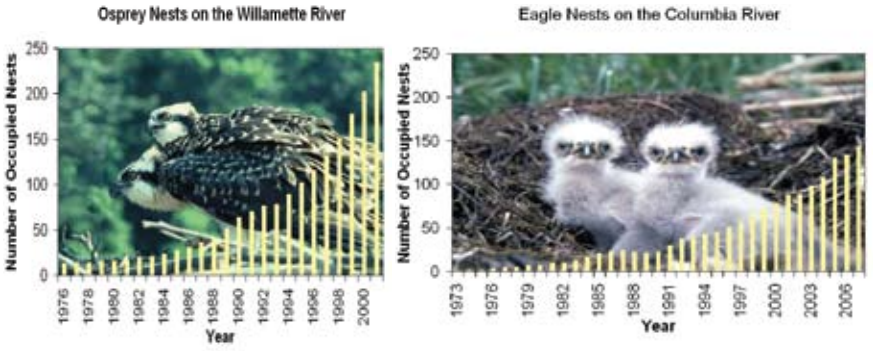


Figure 5.7: DDT levels have decreased significantly in eagle and osprey eggs from the Lower Columbia River over the past 20 years.



(photos courtesy of Peter McGowan, U.S. Fish and Wildlife Service)

Figures 5.8 and 5.9: Nesting pairs of osprey and bald eagle have increased significantly from near-regional extinction in the 1970s, due to reductions of DDT and other contaminants in the environment. [19,21]

increase in nesting osprey along the Willamette River, an important tributary of the Columbia River, from 1976 to 2001. Similar trends have been found in the Columbia River. A 1976 survey of the 300-mile-long Oregon side of the Columbia River found only one occupied osprey nest. [18,19] In 2004, there were 225 osprey nests in the same area. Scientists recorded a 69 percent decrease in DDT levels in osprey eggs from the Lower Columbia River between 1997 and 2004, coinciding with an increase from 94 to 225 osprey nests. [20]

Since the late 1970s, the number of bald eagle nesting pairs along the Lower Columbia River also has increased (Figure 5.9). In 2006, there were over 133 nesting pairs of bald eagles, up from 22 in 1980. However, researchers also found that long-established eagle pairs that had been breeding for many years along the Lower Columbia River produced about half the number of young as eagles that had more recently begun nesting there. The greater reproductive success of the newer nesting bald eagle population is attributed in large part to reduced exposure to DDT. [16]

PCBs: Stable PCB Compounds Continue to Persist in the Environment

PCBs are a class of man-made compounds known for their chemical and thermal stability. PCBs were manufactured to take advantage of these properties in such applications as electric transformers and capacitors, heat exchange and hydraulic fluids, lubricants, fluorescent light ballasts, fire retardants, plastics, epoxy paints, and other materials. Before PCBs were banned in the 1970s, approximately 700 million tons of PCBs were produced in the United States, and hundreds of tons remain in service today.

Environmental concentrations of PCBs decrease very slowly because they are stable and persistent. PCBs tend to concentrate in the fatty tissue of fish and other animals and can be passed from mother to young. PCBs have been linked to liver damage, disruption of neuro-development, reproductive problems, and some forms of cancer. PCB levels have triggered fish and shellfish advisories in the Lower Columbia River and several other water bodies in the Basin.

Figure 5.10 shows PCB concentrations found in fish from U.S. waters in the Columbia River Basin.

PCBs enter the ecosystem from multiple sources and through multiple pathways

PCBs in the Columbia River Basin tend to be associated with industrial locations, where spills or historic handling practices (such as disposing of PCB-contaminated materials in unlined landfills near the River or dumping such materials directly into the River) were more likely to occur. Several examples of known PCB disposal sites in the Lower Columbia River include Bradford Island at Bonneville Dam; Alcoa Smelter in Vancouver, Washington; and Portland Harbor on the Willamette. In addition, historically, many pieces of electrical equipment used to generate power at dams in the Columbia River Basin used cooling and insulating oil that contained PCBs. Past practices such as the use of PCB-laden paint in fish hatcheries and the use of oils tainted with PCBs to control dust on unpaved roads also led to PCB contamination.

Inefficient incineration of PCB-containing materials, followed by atmospheric deposition, is the primary means by which PCBs from other parts of the world reach the Columbia River Basin. Regionally, snowmelt, stormwater runoff and discharge, and soil erosion are pathways by which PCBs deposited on land are transported to water. PCBs entering rivers and streams from stormwater runoff and discharge are a growing concern. PCBs are not very water-soluble, but they do adhere to organic matter and sediment particles, so they have a high potential to be transported when sediment is transported (such as during storms and floods) and then accumulate in pools or reservoirs.

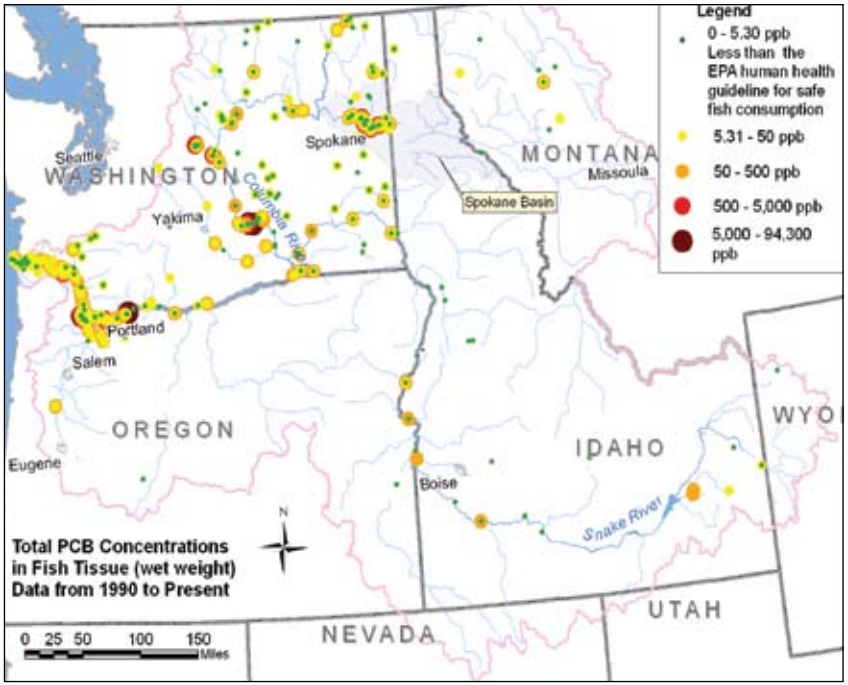


Figure 5.10: A legacy contaminant, PCB hot spots correspond to areas of historic industrial use or disposal sites. There is no information about PCB levels in fish from waters that are unmarked on the map.

PCBs in fish are declining but still exceed EPA human and ecological health concern levels in some areas

In the early 1990s, the Washington Department of Ecology (WADOE) found high concentrations of PCBs in rainbow trout, mountain whitefish, and large-scale sucker in the Spokane River. [1] The Department took steps to identify and clean up hazardous waste sites and reduce PCB inputs from municipal and industrial wastewater dischargers. As a result, concentrations of PCBs in rainbow trout, mountain whitefish, and sucker have decreased between 1992 and 2005 in almost every reach of the Spokane River (Figure 5.11). [1,2,3,4,5]

As with mercury and DDT, several studies have revealed that Columbia River sturgeon living in the pool behind Bonneville Dam contained much higher concentrations of PCBs in their livers than sturgeon in other areas of the Basin. [6]

Recent studies indicate that juvenile fall Chinook salmon from throughout the Basin are accumulating toxic contaminants, including PCBs, in their tissues. [7,8,9] As shown in Figure 5.12, PCB concentrations in juvenile salmon are higher in out-migrating juveniles sampled in the Lower Columbia River near the confluence of the Willamette River than in juveniles sampled at Warrendale just below the Bonneville Dam. Two studies of PCB

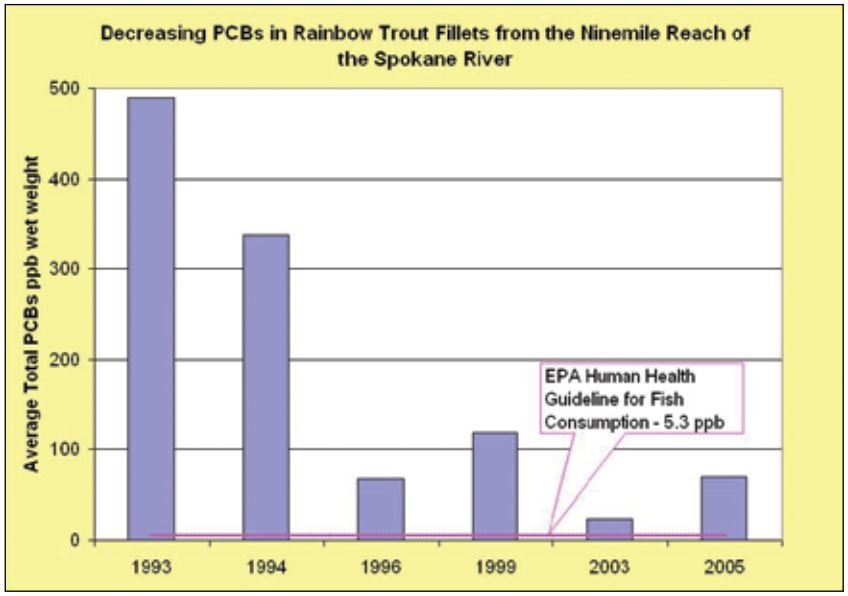


Figure 5.11: PCB levels in rainbow trout from throughout the Spokane River have declined due to hazardous waste cleanup efforts and a reduction in the amount of PCBs discharged in wastewater.

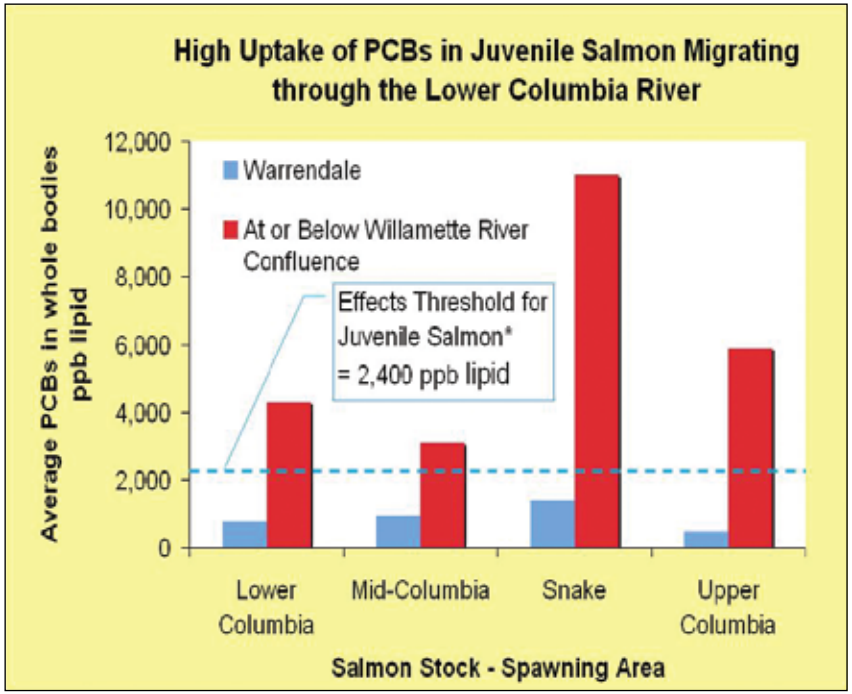


Figure 5.12: Migrating juvenile salmon, regardless of where they began their migration, consistently show higher levels of PCBs when captured in the Lower Columbia River below the Bonneville Dam.

concentrations in water also showed higher dissolved PCBs near the Portland/Vancouver area and downstream of the Willamette River than were found upstream near Bonneville Dam. [7,10] This suggests that there are significant sources of PCBs in the Lower Columbia River.

There are currently no data to indicate whether PCB levels in the mainstem of the Columbia River are increasing or decreasing. However, at some sites PCB concentrations in salmon were as high as or higher than those observed in juvenile salmon from industrial contamination sites in Puget Sound (Duwamish Waterway Superfund site in Seattle, Washington). At several sites in the Columbia River, salmon PCB concentrations were above levels at which juvenile salmon may be harmed (Figure 5.13).

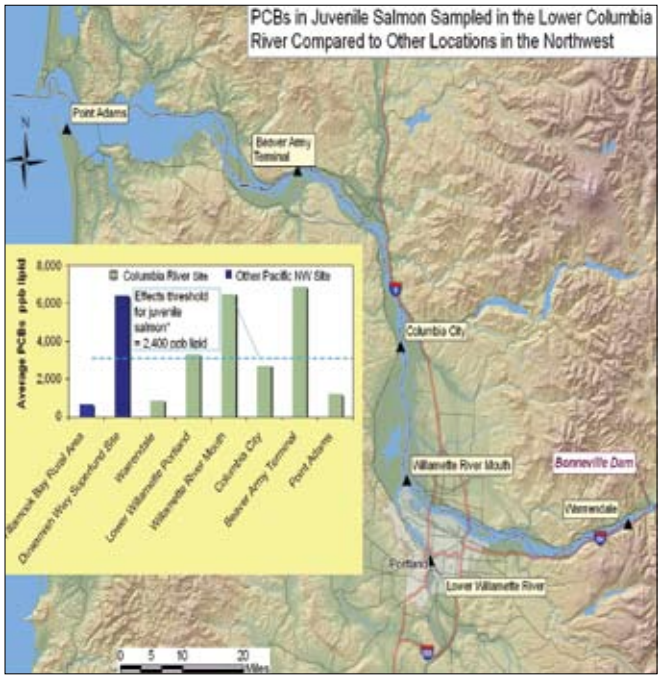


Figure 5.13: PCBs in juvenile salmon from several Lower Columbia River sites are similar to levels found in juvenile salmon at the Duwamish Waterway Superfund site in Seattle, Washington.

PCBs can also adversely affect the ability of mink and otter to reproduce. Mink are especially sensitive to the toxic effects of PCBs. Studies in the late 1970s showed that PCBs in mink from the Lower Columbia River were as high as those levels that are reported to cause total reproductive failure in female mink. [11]

Concentrations of PCBs in mink and otter have declined dramatically since the 1970s (Figure 5.14). [11,12,13] Despite these declines in contaminant concentrations and the presence of suitable habitat, mink remain scarce in the Lower Columbia. While there is a relatively dense otter population distributed throughout the Lower Columbia River, otters there have higher PCB concentrations compared to otters in other areas of Oregon and Washington. [14]

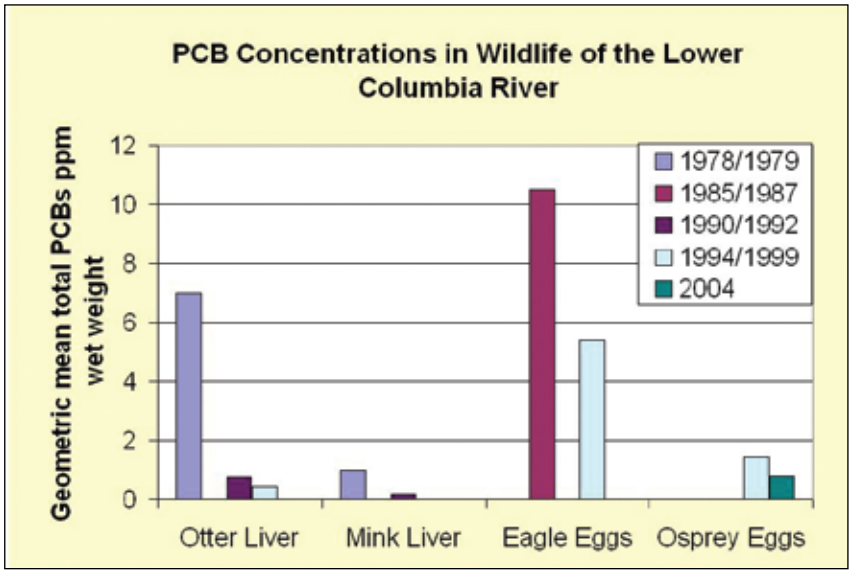


Figure 5.14: PCBs are decreasing in multiple fish-eating predators from the Lower Columbia River, due to decreased PCB use and contaminated site cleanup.

Like DDT, PCBs bioaccumulate in bald eagles and osprey. While PCB concentrations in eagle eggs from the Lower Columbia River were the highest recorded in the western United States in the 1980s, PCB levels are decreasing in both of these top predators (Figure 5.14). [15,16,17]

In 2005, U.S. Army Corps of Engineers (USACE) researchers used the Asian clam to describe distribution patterns of PCBs in the Lower Columbia River. [18] After analyzing samples from 36 stations, the researchers found distinctive spatial patterns related to the specific site from which the clams were collected. All clams collected had detectable levels of PCBs. Especially high levels of PCBs, ranging from 382 to 3,500 parts per billion (ppb), were found downstream of the Alcoa plant, a WADOE hazardous waste cleanup site (Figure 5.15) on the Washington side of the River.

Although "safe" levels for PCB consumption have not been formally established, the Clark County Health Office, State of Washington, recommends that seafood with PCB levels of up to 50 ppb should generally be eaten no more than two or three times per month.

VISIT THE WEB

For more information on PCBs and the Alcoa cleanup, go to: http://www.ecy.wa.gov/programs/swfa/industrial/alum_alcoavan.htm.

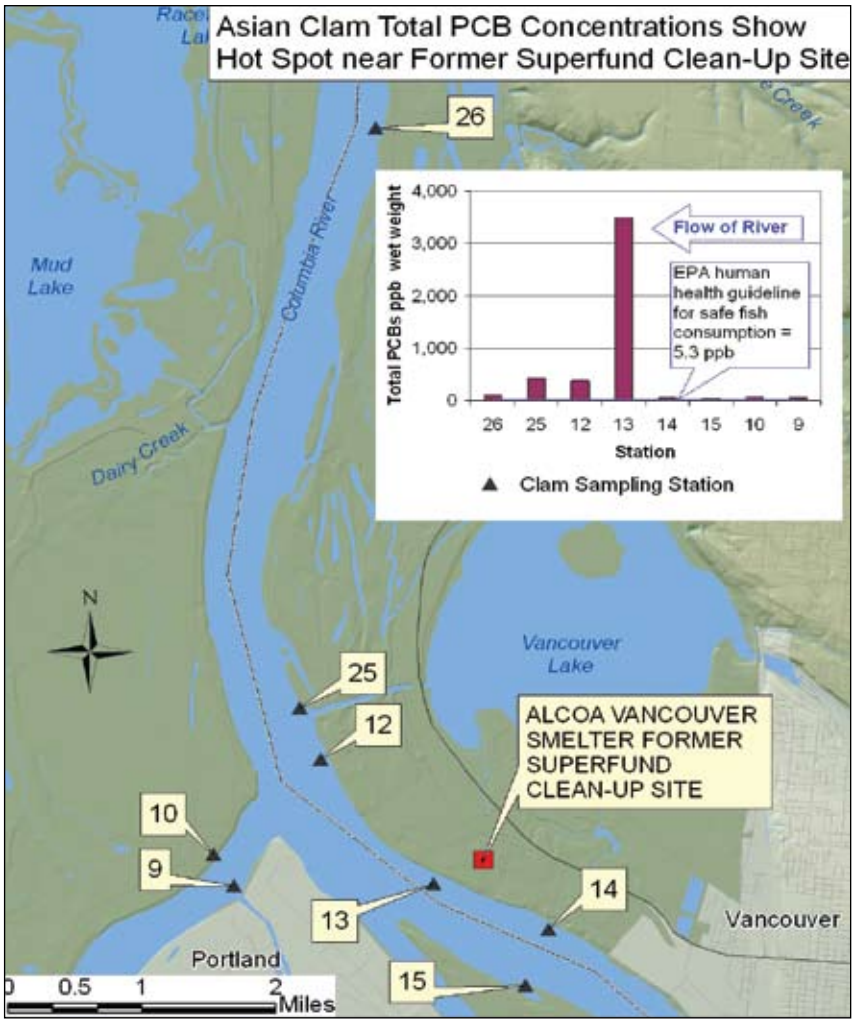


Figure 5.15: Clams collected in the Portland/Vancouver metropolitan area indicate PCB hot spots near the Alcoa plant, a WADOE hazardous waste cleanup site.

PBDEs: Concern over Flame Retardants is Growing

PBDEs are a commonly used flame retardant. Many industries and states, including Washington, are phasing out products containing PBDEs. PBDEs are of concern because their levels have increased rapidly in soil, air, wildlife, and human tissue and breast milk.

The health effects of PBDEs have not been studied in people. Laboratory animal studies show neurological, behavioral, reproductive, and developmental effects and even cancer at very high doses.

PBDEs are in many everyday products

Since the 1960s, PBDEs have been added to plastics and fabrics to reduce the likelihood that these materials will catch fire or burn easily when exposed to flame or high heat. PBDEs are used in electrical appliances; TV sets; building materials; home, auto, and business upholstery; and rug and drapery textiles. They are released slowly to the environment from production, use, and disposal of these products. PBDEs, like PCBs, remain in the environment for a long time. PBDEs accumulate in all animals, but the concentrations continue to increase as an animal ages. However, unlike PCBs, EPA does not currently regulate PBDEs and only recently published a standard method for measuring PBDEs in environmental samples.

Figure 5.16 shows PBDE concentrations found in fish from U.S. waters in the Columbia River Basin.

Information on how PBDEs enter the environment is limited

While there is limited understanding on how PBDEs enter the environment, several studies have indicated that municipal wastewater may be a significant pathway. [1,2,3,4,5] PBDEs in dust and air are a direct pathway of exposure to people, but the importance of air and atmospheric deposition of PBDEs as a source to the Columbia River Basin is unknown. Runoff from municipal sewage sludge placed on land is also being examined as a possible source of PBDEs to surface water. [4,5,6] A study of PBDE contamination in the Canadian portion of the Columbia River found a correlation between high PBDE levels and areas where septic systems were concentrated near the River. [7]

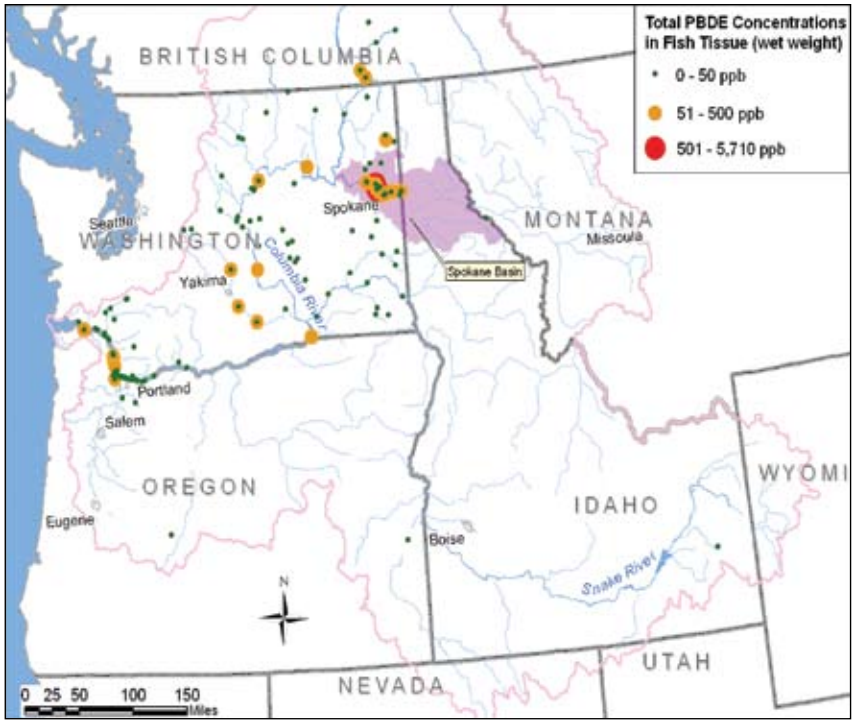


Figure 5.16: PBDEs are being detected and are increasing in fish in the Columbia River Basin. There is no information about PBDE levels in fish from waters that are unmarked on the map.

Levels of PBDEs in the Columbia River are increasing

In 1996, 1999, and 2005, the WADOE studied PBDE concentrations in sucker, mountain whitefish, and rainbow trout in the Spokane River (Figure 5.17). [8,9,10] PBDE levels in these species are increasing in most reaches of the Spokane River. The most dramatic increases were found in mountain whitefish downstream from the Spokane metropolitan area at Ninemile Reach.

Although relatively little PBDE data have been collected in the Columbia River Basin, the studies show that PBDEs are present and are increasing in the waters of the Columbia and several of its tributaries. [7] The studies further show that PBDEs are not only accumulating in larger fish [9] but are being taken up by juvenile salmon as well. [11]

In 2005, PBDEs were detected in all Asian clams collected from 36 stations throughout the Lower Columbia River. [12] The Lower Columbia appears to be an important source of PBDEs for salmon on their migration to the ocean based on the difference in PBDE concentrations in juvenile salmon above and below Bonneville Dam (Figure 5.18).

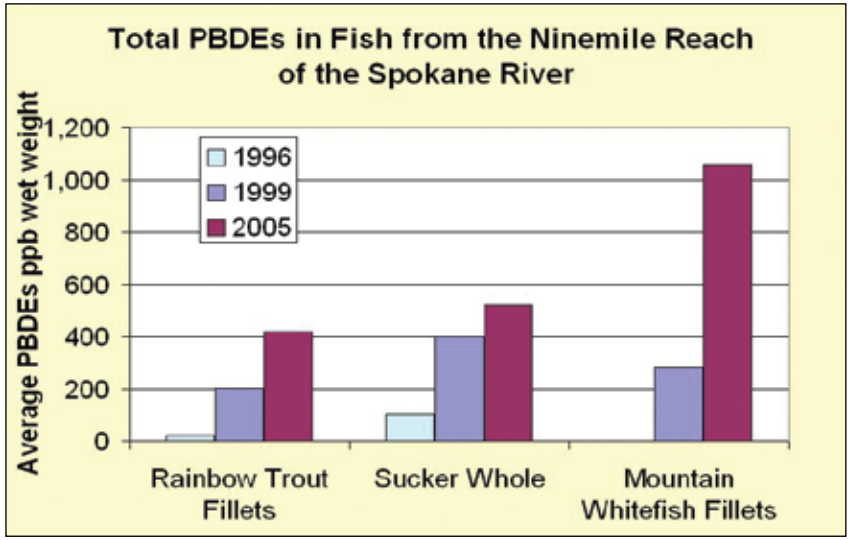


Figure 5.17: PBDE levels in Spokane River fish have increased since 1996.

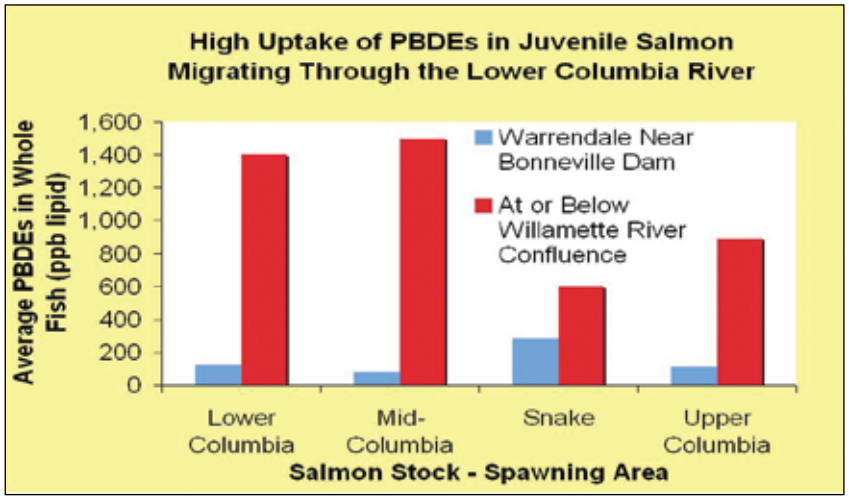


Figure 5.18: Migrating juvenile salmon, regardless of where they began their migration, consistently show higher levels of PBDEs when captured in the Lower Columbia River below the Bonneville Dam.

Summary of Status and Trends for Mercury, DDT, PCBs, and PBDEs

Table 5.2 summarizes the status of concentration levels for the four contaminants discussed in this report and their concentration trends where available.

Table 5.2. Summary of status and concentration trends for the selected indicator species

MERCURY		
Indicator Species	**Status**	**Concentration Trend over Time**
Resident fish - bass, whitefish, sucker, trout, walleye, northern pikeminnow	Increasing concentrations in fish tissue and bird eggs have been seen in the Snake and Willamette River Basins and other locations affected by regional sources compared to other areas within the Basin.	↑
Juvenile salmon		No Trend Data
Sturgeon		No Trend Data
Predatory birds – bald eagle and osprey		↑
Fish-eating mammals - mink and otter		No Trend Data
Sediment-dwelling shellfish - Asian clam		No Trend Data

Note: An upward-pointing red arrow indicates an increasing trend.

DDT AND BREAKDOWN PRODUCTS		
Indicator Species	**Status**	**Concentration Trend over Time**
Resident fish - bass, whitefish, sucker, trout, walleye, northern pikeminnow	The Columbia River Basin received some of the heaviest DDT loadings in the United States prior to the 1972 ban. Levels have decreased dramatically since the 1970s but are still above health effects limits for people, fish, and wildlife in many areas of the Basin.	↓
Juvenile salmon		No Trend Data
Sturgeon		No Trend Data
Predatory birds - bald eagle and osprey		↓
Fish-eating mammals - mink and otter		↓
Sediment-dwelling shellfish - Asian clam		No Trend Data

Note: A downward-pointing green arrow indicates a decreasing trend.

Table 5.2. Summary of status and concentration trends for the selected indicator species (cont)

PCBs		
Indicator Species	**Status**	**Concentration Trend over Time**
Resident fish - bass, whitefish, sucker, trout, walleye, northern pikeminnow	PCB levels have generally declined since they were banned in the 1970s. Because PCBs are very stable and bioaccumulate in long- lived species and top predators, they are still a concern. Every state in the basin still has areas with fish consumption advisories and levels that exceed species effects levels. Sources are still being discovered.	↓
Juvenile salmon		No Trend Data
Sturgeon		No Trend Data
Predatory birds - bald eagle and osprey		↓
Fish-eating mammals - mink and otter		↓
Sediment-dwelling shellfish - Asian clam		No Trend Data

Note: An upward-pointing red arrow indicates a decreasing trend.

PBDEs		
Indicator Species	**Status**	**Concentration Trend over Time**
Resident fish - bass, whitefish, sucker, trout, walleye, northern pikeminnow	In areas where data have been collected, levels of these chemicals are showing rapid increases. Though some studies have detected developmental and other impacts for humans and other species, there are currently no established effects levels for human or other species' health.	↑
Juvenile salmon		No Trend Data
Sturgeon		No Trend Data
Predatory birds – bald eagle and osprey		↑
Fish-eating mammals - mink and otter		No Trend Data
Sediment-dwelling shellfish - Asian clam		No Trend Data

Note: An upward-pointing red arrow indicates an increasing trend.

6.0
Toxics Reduction Efforts—Current and Planned

States, tribes, communities, non-profit groups, EPA, and other federal agencies have launched a long-term recovery effort to improve the water, land, and air quality of the Basin. These groups are working together to enhance and accomplish critical ecosystem restoration efforts. A number of toxics reduction efforts are under way throughout the Basin as a part of this recovery effort.

States are Improving Water Quality and Reducing Toxics

State agencies are developing water quality improvement plans

The Federal Clean Water Act requires states to list all water bodies under their control that do not meet water quality standards. The states are then required to develop water quality improvement plans for those impaired waters so they will meet water quality standards. These plans, also known as total maximum daily loads (TMDLs) (Table 6.1), are in place or are being developed throughout the Basin for toxics.

Through implementation of these TMDLs, water quality is improved using a combination of pollution controls on point sources; programs to reduce non-point sources such as urban stormwater and agricultural runoff; and cleanup of known sources of contaminants such as abandoned mines or hazardous waste sites.

Oregon is using human health criteria to limit toxics

In October 2008, the Oregon Environmental Quality Commission recommended that the Oregon Department of Environmental Quality (ODEQ) revise the human health criteria as a part of Oregon's water quality standards. The Commission has asked for a proposed rule with a fish consumption rate of 175 grams per day (instead of the current rate of 17.5 grams per day) and a broader toxics reduction implementation strategy. This recommendation was a result of a two-year collaborative process led by EPA, ODEQ, and the Confederated Tribes of the Umatilla Indian Reservation. The recommended fish consumption rate of 175 grams per day represents approximately the 90th to 95th percentile of Oregon's fish-consuming populations, as indicated by studies of tribes, Asians, and Pacific Islanders in Oregon and Washington. [1]

ODEQ's water quality standards play an important role in maintaining and restoring environmental quality. Human health criteria are used to limit the amount of toxic pollutants that enter Oregon's waterways and accumulate in the fish and shellfish consumed by Oregonians. The criteria also serve as the framework for wastewater permits, nonpoint source reduction activities, stormwater permits, and sediment cleanup efforts. The criteria help ensure that people may eat fish and shellfish from local waters without incurring unacceptable health risks. A final rule on the revised criteria is expected in October 2009.

EPA and States are Using Permits to Control Toxics

The Clean Water Act's National Pollutant Discharge Elimination System (NPDES) program controls the quality of water discharged into the Basin from point sources such as wastewater treatment plants, mines, and pulp and paper plants. Federal, state, and local NPDES permits limit the amount of pollutants from municipal, industrial, and stormwater discharges so that the quality of the water body receiving the discharge is not impacted or further impaired. Facilities that have an NPDES permit must conduct routine monitoring and are fined or required to install pollution controls if their NPDES permit conditions for water quality are not met. However, data on the amounts of many toxics (including DDT, PCBs, and PBDEs) entering the Columbia River from stormwater and from municipal and industrial dischargers are limited.

Stormwater and erosion controls are increasingly important in urban and developing areas to keep contaminants from reaching lakes, rivers, and streams. This is done through stormwater NPDES permitting and a combination of best management practices (BMPs) and public education. Many communities and industries

Combined sewer overflow (CSO) outfall (photo courtesy of WADOE)

Table 6.1: Toxics TMDLs that have been approved or are under development in the Washington, Oregon, and Idaho areas of the Columbia River Basin

State	River	Toxics
Washington	Yakima	Chlorinated Pesticides (e.g., DDT) and PCBs
	Spokane	Metals, PCBs
	Okanogan	DDT, PCBs
	Walla Walla	Chlorinated Pesticides and PCBs
	Palouse	Chlorinated Pesticides and PCBs
	Lake Chelan	DDT, PCBs
	Mission Creek (Wenatchee)	DDT
	Columbia	Dioxins
	Similkameen	Arsenic
Oregon	Columbia	Dioxins
	Columbia Slough	Lead, PCBs, Dioxins, DDT, Dieldrin
	Coast Fork Willamette	Mercury
	Cottage Grove Reservoir	Mercury
	Pudding	DDT, Dieldrin, Chlordane
	Johnson Creek	DDT, Dieldrin
	Willamette	Mercury
	Row River	Mercury
	Snake River	DDD, DDE, DDT, Dieldrin
Idaho	Salmon Falls Reservoir	Mercury
	Jordan Creek	Mercury
	East Fork Eagle Creek (North Fork Coeur D'Alene)	Metals
	Snake River	DDD, DDE, DDT, Dieldrin
	Columbia	Dioxins

are adopting innovative stormwater management techniques that improve the quality of the discharged water before it reaches lakes, rivers, and streams. These include porous pavement to reduce runoff; diversion of runoff from storm sewers into natural systems (e.g., vegetated buffers); retention and treatment wetlands; and filtration through vegetated swales. Such stormwater management practices also reduce flooding, erosion, and direct runoff of contaminants to waterways.

Federal Government and States are Working to Clean up Hazardous Waste in the Basin

Several contaminated sites in the Basin are being cleaned up and managed under EPA Superfund or state toxic cleanup programs. For example, since 1983, EPA has been working with the State of Idaho, the Coeur d'Alene Tribe, and mining companies to clean up the Bunker Hill Mining and Metallurgical Superfund site in the Coeur d'Alene Basin. The area's many mines were once a primary source of our nation's zinc, copper, lead, and precious metals. A comprehensive, integrated approach, using all available regulatory tools such as the Clean Water Act and the Comprehensive Environmental Response, Compensation and Liability Act, has been employed to help protect human health and the environment in this heavily contaminated watershed.

Cleanup of an Idaho mine near the Salmon River (photo courtesy of EPA)

Furthermore, in the Upper Columbia River above Grand Coulee Dam, several investigations and cleanups are ongoing in the areas that drain into Lake Roosevelt. In Montana, cleanup efforts in the upper Clark Fork and Flathead basins have reduced copper, lead, arsenic, and zinc contamination into the Columbia River tributaries. [2] In the Middle Columbia River, the U.S. Department of Energy (DOE) is working to prevent contaminated groundwater on the Hanford Nuclear Reservation from reaching the Columbia River. Work is also under way to clean up contaminated sediment from the Portland Harbor Superfund site, located on the lower Willamette River near its confluence with the Lower Columbia to reduce PCBs, DDT, and many other toxic contaminants.

In addition to the federally listed Superfund sites, each state manages its own list of contaminated site cleanup projects. States work with the federal agencies and with businesses and property owners to develop site assessment and cleanup plans and then conduct cleanup activities. Many contaminated sites in the Basin are in various stages of planning and cleanup for a variety of contaminants. Two examples of PCB-contaminated sites on the Columbia River are the Bradford Island site at the Bonneville Dam and the Alcoa plant in Vancouver, Washington. An accelerated cleanup is planned by the State of Washington at the Alcoa site, where sediment removal is scheduled for November 2008.

Upper Columbia River Investigation and Cleanup

EPA is studying hazardous waste contamination in the Upper Columbia River from the U.S./Canadian border down to Grand Coulee Dam and the surrounding upland areas. The investigation and cleanup site under EPA Superfund authority, located in northeastern Washington, consists of 150 miles of river and lake environment. From about 1930 to 1995, the Teck Cominco smelter in Trail, B.C., located 10 miles north of the U.S./Canadian border, discharged millions of tons of metals-laden slag and other wastes directly into the Columbia River. The waste discharged from the facility was carried downstream into the United States and has settled in the River's low-flow areas, beaches, and stream banks, potentially impacting the ecosystem in and around the Upper Columbia River.

In 2004, EPA began investigating the contamination problems in the Upper Columbia. In the first phase of the investigation, EPA collected over 400 sediment and 1,000 fish samples, along with samples from 15 beaches. Over the next several years, additional sediment, fish, and beach samples will be collected.

Bradford Island PCB Cleanup

In 1997 and 1998, USGS biologists found higher levels of PCBs in osprey eggs collected near Bonneville Dam than in eggs from other reaches of the Columbia River. [3] Also, in the late 1990s, very high levels of PCBs were found in crayfish collected near Bradford Island, which is part of the Bonneville Lock and Dam Complex. Based on this information, the Oregon Department of Human Services issued an advisory cautioning people against consuming crayfish, clams, or other bottom-dwelling organisms between Bonneville Dam and Ruckel Creek, about a mile upstream.

The PCB contamination came from disposal of electrical equipment on Bradford Island and the Columbia River during the 1950s. In response, the USACE removed PCB-containing equipment and some sediments in 2002. In 2007, the Corps completed the removal of PCB sediment "hot-spots" over a one-acre area that was estimated to contain over 90 percent of the PCB contamination on Bradford Island. The Corps continues to work with ODEQ to evaluate and remove the remaining PCB-contaminated sediments.

Portland Harbor Superfund Cleanup Site

The Portland Harbor Superfund site study area is focused on an 11-mile stretch of the lower Willamette River from downtown Portland, Oregon, to the Columbia River. Sediments at the site are contaminated with metals, pesticides (e.g., DDT), polycyclic aromatic hydrocarbons (PAHs), PCBs, and dioxin/furans from a variety of sources. EPA is overseeing a remedial investigation and feasibility study being conducted by a group of potentially responsible parties referred to as the Lower Willamette Group. EPA is the lead agency for investigating and cleaning up contaminated sediment in the Willamette. The ODEQ is the lead agency for investigating and cleaning up the upland sites that are potential sources of contamination to the Willamette. A draft feasibility study, which will evaluate cleanup strategies and methods, is targeted for late 2010. EPA will then issue a proposed cleanup plan for public comment before making a final decision on the harbor-wide cleanup. In addition to the harbor-wide investigation, several early actions are under way to clean up individual sites that need more immediate attention.

VISIT THE WEB

Additional information about the Upper Columbia, Bradford Island, and Portland Harbor investigations and cleanups can be found by visiting EPA's Columbia River Basin website: http://www.epa.gov/region10/columbia.

State and Local Partnerships are Working to Improve Farming Practices

Partnerships and volunteer efforts are reducing runoff from farms

The Columbia River Basin supports some of the most important agricultural regions in the United States. Clean water for food production is critical, but agricultural practices can degrade water quality by contributing eroded soil, nutrients, and pesticides to nearby waters. Agricultural BMPs are used to improve water quality, often with the added benefits of improving water and soil conservation and soil fertility.

BMPs are usually developed and implemented by partnerships between farmers, local conservation districts and university extension services, state and federal agriculture and water quality agencies, tribal governments, and local watershed groups. They have become a critical component of TMDLs in agricultural watersheds such as the Yakima River.

The agricultural community can be leaders in reducing toxics in the Columbia River Basin. Voluntary agricultural activities provide a great opportunity to reduce toxics in the Basin by reducing legacy toxics such as DDT and current-use pesticides, especially organophosphates. Toxic contaminants reach the Columbia River Basin from sediment transport and deposition and have contributed to the long-time degradation of water quality and fish and wildlife habitat. Sediments may transport trace metals (such as arsenic and copper) and organic compounds (such as polycyclic aromatic hydrocarbons [PAHs], PCBs, and pesticides such as DDT, chlordane, and atrazine). Most of these contaminants cling to particles suspended in the water and settle to the bottom; therefore, their concentrations in sediments are typically much higher than in water.

Yakima Valley irrigation ditch before implementation of BMPs (left) and Yakima Valley irrigation ditch with BMPs to control erosion and reduce runoff (right) (photos courtesy of the Confederated Tribes and Bands of the Yakama Nation Environmental Management Program)

Washington is working to control soil erosion and reduce pesticide runoff in the Yakima River Basin

The Yakima River Basin serves as a successful example of sediment cleanup and pesticide reduction efforts. [4] DDT was used extensively in the Yakima Valley from the 1940s until it was banned in 1972, and it persists in Yakima Basin soils. Erosion of these soils allows pesticides to reach the aquatic environment, where they accumulate in fish and in the people and wildlife that eat fish. Recognizing this, the WADOE, Yakima Valley growers, water purveyors, local conservation districts, and the Confederated Tribes and Bands of the Yakama Nation worked together to implement BMPs to reduce DDT and other pesticides by modifying irrigation practices to reduce the amount of soil carried to the Yakima River by irrigation returns.

DDT clings to organic particles in soil; therefore, reducing soil erosion from agricultural fields and the associated sediments should reduce runoff polluted with pesticides like DDT.

After the BMPs were initiated, suspended sediment loading to the Lower Yakima River during the irrigation season was reduced between 67 and 80 percent. Total DDT

concentrations in fish were reduced by 30 to 85 percent in the same area after implementation of the BMPs. The accompanying photos show soil eroded by surface irrigation into a return drain before BMPs were implemented; later, with BMPs, the soil is retained by a grass filter strip between crop and drain.

Oregon is working with farmers to reduce pesticide runoff

Another example of toxics reduction from agriculture in the Columbia River Basin is Oregon's Pesticide Stewardship Partnerships. These partnerships are voluntary collaborations to reduce pesticide use and improve water quality. Such collaborations typically include local watershed councils, ODEQ, agricultural growers, Oregon State University (OSU) Extension Service, and tribes. Pilot projects in the Columbia Gorge, Hood River, and Fifteen-Mile Creek near The Dalles, Oregon, showed substantial improvements in water quality due to changes in pesticide management and application practices. In addition, ODEQ has launched Pesticide Stewardship Partnerships in six watersheds in the Basin: the Walla Walla, Clackamas, Pudding, Yamhill, Willamette, and Hood River Basins.

For example, the Walla Walla partnership has reduced pesticide concentrations in Oregon's Walla Walla River Basin. [5] In 2006, high levels of five toxic pesticides were found in tributaries of the Little Walla Walla River. In response, the ODEQ, OSU Extension Service, fruit growers (Blue Mountain Horticultural Society), and Walla Walla Basin Watershed Council worked together to monitor and control current-use pesticides that reach surface water by spray drift and runoff from fruit orchards. To accomplish this, ODEQ and its partners installed vegetated buffers adjacent to surface waters, switched to using less toxic pesticides and mineral oil, provided individualized applicator training, and calibrated sprayers to avoid overspray.

The monitoring results in 2007-2008, after implementation of the practices described above, showed dramatic declines in several pesticides, including large reductions of one of the most toxic pesticides, chlorpyrifos (Figure 6.1).

In addition, ODEQ has worked with partners in the Walla Walla Basin to conduct two agricultural pesticide collection events to remove unwanted waste pesticides from the watershed. Over 17,000 pounds of pesticide waste were collected and properly disposed of from these events.

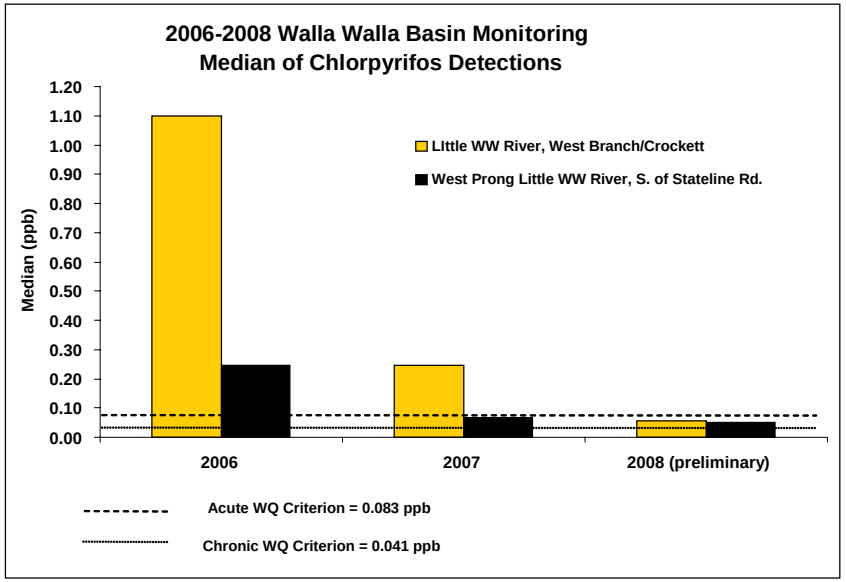


Figure 6.1: Concentrations of chlorpyrifos dropped after measures were implemented to keep pesticides from reaching nearby surface waters in Oregon.

State and Local Governments are Removing Toxics from Communities

The State of Washington passed one of the first state bans on PBDEs in the summer of 2007. This ban is part of the state's overall initiative to reduce the threat from persistent and bioaccumulative toxics (PBTs) by keeping toxics out of products and industrial processes. The ban is being phased in over a two-year period, with an emphasis on finding a safer and feasible alternative. Oregon is also working to reduce and control PBTs, particularly for large municipal wastewater dischargers. All of the Basin states have mercury reduction

programs to promote recycling of thermometers and fluorescent lamps containing mercury, and each state works with dentists, hospitals, and vehicle recyclers to capture and recycle mercury. For example, separating mercury from wastewater in dental offices prevents mercury from reaching wastewater treatment plants and the Columbia River. Oregon and Washington also sponsor collection of mercury recovered by small-scale mineral miners from streams and rivers.

State, county, and local toxics reduction programs help businesses and private citizens reduce the use of toxic chemicals and ensure the proper disposal of hazardous wastes such as pesticides, solvents, batteries, electronics, PBDE-containing materials, and pharmaceuticals. For example, Idaho's pesticide disposal program prevents thousands of pounds of unusable pesticides from reaching the environment each year. Under this program, the Idaho State Department of Agriculture assists growers, homeowners, dealers, and applicators with the disposal of pesticides that have become unusable because of expiration, cancellation, deterioration, or crop changes. Individuals can dispose of up to 1,000 pounds of pesticide at no charge. Permanent collection points are established throughout the state; materials are collected annually and taken to a licensed facility for incineration. From 2003 to 2007, 328,000 pounds of unusable pesticides have been collected, and over 870,000 pounds have been collected since the program's inception in 1993 (Figure 6.2). [6] The program also collects and recycles empty pesticide containers. Washington and Oregon are also sponsoring pesticide take-back programs, which have recovered thousands of pounds of banned pesticides such as DDT.

Another Idaho initiative is the Idaho Department of Environmental Quality's (IDEQ's) school laboratory and chemical cleanup project. This project assists schools in understanding and implementing best practices for managing and disposing of their large stockpiles of hazardous chemicals and wastes, including mercury.

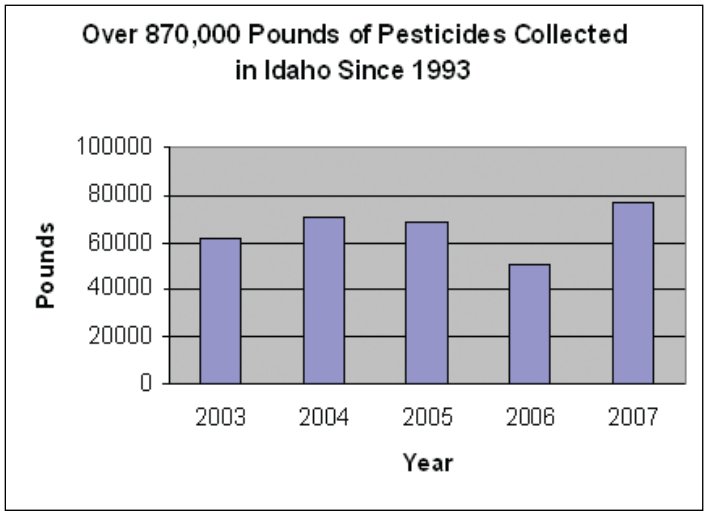


Figure 6.2: Amount of pesticides collected under Idaho's pesticide disposal program (2003–2007).

At the county level, Clark County, on the Lower Columbia River in Washington, recently implemented an unwanted medications take-back program that allows residents to drop off unwanted pharmaceuticals at participating pharmacies. The drugs are then incinerated at a licensed facility. Washington has implemented a pilot pharmaceutical take-back program in King County (through 2008) and plans to expand it to a statewide program. In Oregon, a proposal may be presented to the 2009 legislative session for a pharmaceutical take-back program. These partnerships between state and local governments, pharmacies, medical facilities, and the U.S. Drug Enforcement Administration reduce pharmaceutical pollution in wastewater and unlined solid waste landfills which can contaminate groundwater and surface waterways.

Oregon and Nevada are Reducing Industrial Mercury Emissions

A number of regulatory agencies in the Basin have recently introduced controls on industrial mercury discharges to the air. EPA expanded its Toxics Release Inventory (TRI) reporting requirements in 1999 to include mercury reporting for a variety of industries. The TRI data showed that some of the highest discharges of mercury in the country were in or bordering the Basin and that the single highest emitter of mercury was a cement plant in eastern Oregon. To reduce these emissions, ODEQ worked with the cement plant operators, who, through a 2008 mutual agreement and order, agreed to "…endeavor to meet a goal of 85% reduction in mercury emissions on a rolling 12-month average basis…". The agreement also stipulates that if the goal is not met within a specified timeframe, plant operators will develop an action plan and implement corrective actions in a further effort to achieve the 85 percent reduction. ODEQ will oversee these efforts to determine whether the cement plant "…exhaust[s] all reasonable alternatives…" to meet the goal. [7]

Approximately a dozen mines in the Battle Mountain Mining District in northern Nevada produce 11 percent of the world's gold and 74 percent of the nation's gold. [8] TRI reporting showed that these gold mining operations were releasing a total of over 12,000 pounds of mercury per year. Between 2002 and 2005, EPA and the Nevada Department of Environmental Protection worked with four mining companies to set up a program of voluntary reductions for mercury emissions that resulted in an 82 percent decrease of mercury discharges to air at these mines. In March 2008, the State of Nevada enacted the nation's first regulations limiting mercury air emissions from precious metal mining operations. These regulations set limits on mercury emissions from all the mines in the Battle Mountain District.

The only coal-fired power plant in the Columbia River Basin is located near the Columbia River at Boardman. This plant discharges an average of 168 pounds of mercury to the atmosphere per year. [9] In December 2006, Oregon adopted regulations applicable to coal-fired power plants that require the Boardman plant to control and reduce mercury emissions by 90 percent by 2012 and cap state-wide mercury emissions from coal-fired power plants by 2018. There are also three coal-fired power plants near the boundary of the Basin (one in Washington and two in Nevada) that could contribute some mercury load to the watershed, depending upon their emissions and prevailing wind patterns.

Idaho Agencies and Kootenai Tribe are Monitoring Toxics in Fish, Water, and Air

For several years, the State of Idaho has monitored rivers, lakes, and reservoirs for a number of toxics. In 2006, IDEQ sampled 15 large rivers for mercury in fish. In 2007, IDEQ sampled 50 lakes and reservoirs for arsenic, mercury, and selenium in fish tissue. In 2008, an additional 34 large rivers were sampled for arsenic, mercury, and selenium in both fish and water; the water samples were also tested for methylmercury.

IDEQ has also conducted or supported other local efforts, most notably in support of the Salmon Falls Creek mercury TMDL, submitted to EPA in December 2007 and approved in February 2008. The state's air quality program has also been conducting some mercury deposition monitoring.

Other noteworthy studies include the following:

- The Kootenai Tribe of Idaho has conducted studies of numerous contaminants in sturgeon, fish, water, sediment, and lower food web organisms from the Kootenai River between 1999 and 2007. The tribe has also studied biomarkers in sturgeon for the effects of contaminants.
- The Idaho Department of Fish and Game conducted studies of contaminants and biomarkers in Kootenai River adult and juvenile sturgeon in 1997 and 1998.
- Idaho Power Company has conducted several studies of contaminants in the Snake River area along the Oregon-Idaho state line.

PCBs and Hydroelectric Facilities

Historically, many pieces of electrical equipment used to generate power at dams in the Columbia River Basin used cooling and insulating oil that contained PCBs. In recent years, efforts have been made to reduce the presence of, and risk from, PCBs. These efforts include reducing or removing PCBs from electrical equipment; conducting operator self-assessments and EPA inspections; confirming that turbine oil does not contain PCBs; and reducing the potential for PCB spills. EPA will continue to work with the operators of hydroelectric facilities to better understand the remaining risk of PCBs at dams.

7.0 Conclusions

The Columbia River Basin is a unique and vibrant ecosystem that is at risk from toxic contaminants. Many challenges lie ahead to restore this ecosystem. This *State of the River Report for Toxics* is EPA Region 10's first attempt to understand and describe the current status and trends of toxics in this region of the United States. This report is intended to serve as a starting point for increasing public understanding about the impacts of toxics in the Basin and for finding ways to work in partnership with others to improve and expand current toxics reduction efforts. Specifically, its primary purposes are to inform citizens and decision-makers about the toxics problem and potential solutions; serve as a catalyst for increased citizen involvement and increased action; and inspire additional, more-efficient use of resources for increased toxics reduction and assessment actions.

While several monitoring studies are under way in the Basin to improve our understanding of the toxics problem, we must develop a more comprehensive and collaborative monitoring and research program. In addition, we must expand efforts to identify the sources of toxics in the Basin, characterize the types of contaminants, and quantify the contaminant load from these sources. We must also identify additional effective actions to reduce toxics and protect the health of the Columbia River Basin ecosystem, and we must continue to implement those actions.

This report focused on four contaminants: mercury, DDT and its breakdown products, PCBs, and PBDEs. However, we recognize that other toxics, including additional metals, dioxins, radionuclides, and pesticides as well as pharmaceuticals and personal care products, are also potential contaminants of concern. We know that these other contaminants need to be addressed in the future.

Meanwhile, many groups are conducting pollution prevention and cleanup efforts to reduce toxics overall and to reduce toxics in water, sediment, plants, and animals in the Columbia River Basin. Despite limited resources, these groups are making significant strides in reducing toxics in certain areas, but additional efforts need to be expanded throughout the Basin. The following Toxics Reduction Initiatives represent a first attempt at describing the next steps in the effort to reduce toxics. We look forward to a future public dialogue throughout the Basin as we refine and implement these initiatives.

8.0
Toxics Reduction Initiatives

The Columbia River Toxics Reduction Working Group has developed the following set of six Toxics Reduction Initiatives, which provide a broad overview of major actions needed to further reduce toxics in the Basin. A more in-depth and detailed work plan will be developed over the next year with stakeholder and public input.

Initiative #1: Expand toxics reduction activities

Federal, state, and local agencies have multiple regulatory mechanisms available to reduce toxics. Such mechanisms include TMDLs, NPDES permits, water quality standards, contaminated site cleanup, and programs to control pesticide usage. These programs need to be expanded. For example, additional toxics TMDLs and implementation plans are needed, and additional work is needed to identify other contaminated sites for cleanup.

It is also important to promote voluntary/nonregulatory initiatives. States and tribes have worked to reduce toxics using a variety of voluntary and nonregulatory activities. They have focused much of their work on the tributaries to the Columbia River. Excellent examples of voluntary programs are Oregon's Pesticide Stewardship Partnerships and the Pesticide Take Back Program. Support of local watershed groups in their efforts to complete toxics reduction projects should be continued. In addition, more partnerships should be developed with nongovernmental programs such as Salmon Safe and organizations such as Columbia Riverkeeper, other local nonprofit groups, and area industries.

Initiative #2: Identify, inventory, and characterize the sources of toxics in the Columbia River Basin

There have been past efforts to identify and characterize sources of toxics in the Columbia River and its tributaries,[1] some of which are ongoing (e.g., Upper Columbia River, Hanford, and Portland Harbor investigations; Working Group efforts; and TMDL development in the Basin). However, additional information is needed to better identify, inventory, and characterize the sources of these toxics. This information will be used to prioritize reduction efforts and develop long-term monitoring and research plans.

To fill in these critical information gaps, the Working Group has started to identify important "next steps." These steps include, but are not limited to, (1) identifying, inventorying, and mapping all potential sources of toxics, both within and outside the Basin; (2) determining the contaminants of concern from these sources; (3) collecting information on the concentrations of the contaminants of concern, where available; (4) determining the quantities of contaminants reaching the Columbia River and its tributaries, where possible; (5) evaluating the fate and transport of contaminants and their breakdown products from air and soil into the Columbia River and its tributaries; (6) determining the role of sediments as a source of contamination; and (7) prioritizing those sources where the greatest reduction efforts are needed and can be implemented.

Initiative #3: Develop a regional, multi-agency long-term monitoring program

There is no comprehensive, integrated monitoring plan for the Columbia River and its tributaries. This initiative will allow the Working Group to develop such a plan; ultimately, this plan would provide information on the locations and concentrations of toxics in the Basin, fill in data gaps in our scientific knowledge, evaluate the impact of toxics on the ecosystem, and characterize the information on the status and trends of toxics in the Basin. With this information, the Working Group will be able to target limited resources and tailor the monitoring program to obtain data from areas that have not been previously monitored (such as the mid-Columbia River and the Snake River).

Critical steps in the development of this monitoring plan include (1) completing a data gaps analysis of the Basin's contaminant data collected from 1994 to the present; (2) determining the geographic extent of the areas to be sampled and identifying which contaminants would be monitored; (3) determining the types of media to be sampled (e.g., water, sediments, and/or fish tissue); and (4) determining the frequency, specific locations, and techniques for sampling. Because of limited resources, any monitoring program needs to be coordinated among the different federal, state, tribal, local, and nongovernmental entities to leverage resources and avoid duplication.

Initiative #4: Develop a regional, multi-agency research program

While research is being conducted by different agencies on toxics in the Basin, no coordinated effort has been made to identify the highest priorities for research. A collaborative plan will help the Working Group further understand the Basin's contaminant problems and their relation to the food web, which will allow the Working Group to efficiently leverage resources among agencies. It will also enable us to develop an integrated approach that focuses on issues specific to the Columbia River Basin (for example, PBDE concentrations in osprey eggs) that can be addressed by scientists within the region (NOAA Fisheries, EPA Corvallis Laboratory, USGS Science Center, and others).

Initiative #5: Develop a data management system that will allow us to share information on toxics in the Basin

The ability to access information is critical to effectively evaluating toxics information. It is also necessary when prioritizing which reduction activities will provide the most benefits. Currently, no single database contains all of the data from monitoring efforts within the Basin. In addition, some of the data are not publicly accessible or are often available only in hard copy records. Some records are of unknown quality, and most are in differing formats.

While a single database would be useful, its development would be very expensive and would require dedicated resources to operate and maintain. As an alternative to a single database, the Working Group will explore the possibility of working with existing efforts such as the Northwest Data Exchange Network and the Pacific Northwest Aquatic Monitoring Partnership.

Initiative #6: Increase public education about the toxics problems and resource needs

Public support and concern related to toxics and their impact on human health and the environment are growing. Furthermore, there is a base of support in the Basin among citizens, watershed groups, and other stakeholders associated with local, state, tribal, and federal governments. Many of these groups are interested in working together to better understand and reduce toxics in the Columbia River Basin, with the goal of moving toward a Basin ecosystem that is healthier for all.

It will be important to educate the public further about the Columbia River Basin toxics problem, current efforts, and the need for increased action and resources to reduce toxics. The Working Group intends to work closely with the partners of the Columbia River Toxics Reduction Working Group and with Basin stakeholders to coordinate outreach to the public (including schools, business/industry groups, nonprofit organizations, farm associations, and watershed councils). Outreach efforts will include (1) holding public workshops and other public events throughout the Basin; (2) using multi-media tools, including websites, postcards, and posters, to educate and inform Basin residents about toxics; and (3) encouraging public participation in Columbia River toxics reduction activities.

9.0
A Path Forward

To a great extent, success will depend on a commitment to join forces to make the best use of available resources. This approach will require strong communication and collaboration among Basin agencies, organizations, and the public. We recognize that the citizens of the Northwest place a high value on a healthy Columbia River Basin ecosystem. Therefore, we plan to reach out to those who live, work, and play in the Basin; share information on risks to the Basin posed by toxics; and solicit help in restoring the Basin's magnificent ecosystem.

In 2009, the Columbia River Toxics Reduction Working Group will develop a draft work plan that will build on the successful and numerous toxics reduction efforts already accomplished or under way and will also identify new efforts to reduce toxics in the Basin. We will do this by hosting a number of watershed-based workshops in the Basin. The outcome of these workshops should be a toxics reduction work plan for the Columbia River Basin that will involve citizens; local watershed councils; Basin communities; other entities; and tribal, federal, and state governments in a collaborative partnership.

Columbia River Toxics Reduction Work Plan and Watershed Workshops

Late Winter – Early Spring 2009: The Columbia River Toxics Reduction Working Group develops draft toxics reduction work plan.

Late Spring – Summer 2009: Watershed workshops are held for Basin residents, local watershed councils and communities, tribal governments, and the general public to learn about, and contribute to, the draft work plan. Actions are initiated to evaluate the extent of toxic contamination in the Basin and reduce impacts.

Fall – Winter 2009: The Working Group finalizes a collaborative, watershed-based work plan that focuses efforts on implementation.

VISIT THE WEB More detailed information, including expanded data and reports, can be found by visiting EPA's Columbia River website: http://www.epa.gov/region10/columbia.

10.0 References

Section 2.0: Introduction

1. NPCC (2007), "Human Population Impacts on Columbia River Basin Fish and Wildlife," ISAB 2007-3, June.
2 Pacific Northwest River Basins Commission (1979), *Water—Today and Tomorrow*, Vancouver, Washington.
3. NPCC (2000), *Columbia River Basin Fish and Wildlife Program*, Council Document 2000-19.
4. BPA, USACE, USBR (2001), *The Columbia River System: Inside Story*, Federal Columbia River Power System, DOE/BP-3372, second edition, April.
5. EPA (2006), *2006-2011 EPA Strategic Plan: Charting Our Course*, EPA 190-R-06-001.
6. EPA (1992), *National Study of Chemical Residues in Fish*, EPA 823-R-92-008a.
7. CRITFC (1994), *A Fish Consumption Survey of the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin*, Technical report 94-3, Columbia River Inter-Tribal Fish Commission, Portland, Oregon.
8. EPA (2002), *Columbia River Basin Fish Contaminant Survey*, 1996-1998, EPA 910-R-02-006.

Section 3.0: Toxic Contaminants

1. Columbia River Toxics Reduction Working Group (2007), Contaminants Subgroup, "Contaminants of Concern."
2. Washington Toxics Coalition (2006), *Pollution in People: A Study of Toxic Chemicals in Washingtonians, A Toxic-Free Legacy Coalition Report*, May.
3. Oregon Environmental Council and the Oregon Collaborative for Health and the Environment (2007), *Pollution in People: A Study of Toxic Chemical in Oregonians*, November.
4. Nilsen, E.B., R.R. Rosenbauer, E.T. Furlong, M.R. Burkhardt, S.L. Werner, L. Greaser, and M. Noriega (2007), "Pharmaceuticals, personal care products and anthropogenic waste indicators detected in streambed sediments of the Lower Columbia River and selected tributaries," in *6th International Conference on Pharmaceuticals and Endocrine Disrupting Chemicals in Water*, National Ground Water Association, Costa Mesa, CA; Paper 4483, p 15.
5. Buelow, L. (2008), EPA, personal communication, June 2008 e-mail.
6. EPA (1987), *National Dioxin Study*, Report to Congress, EPA Office of Solid Waste and Emergency Response, EPA/530-SW-025, Washington, D.C., August.
7. EPA (1992), *National Study of Chemical Residues in Fish*, EPA 823-R-92-008a.
8. Seiders, K. (2007), *Washington State Toxics Monitoring Program: Contaminants in Fish Tissue from Freshwater Environments 2004 - 2005*, ECY publication #02-03-65 at http://www.ecy.wa.gov/biblio/0703024.html, Washington Department of Ecology.
9. EVS Consultants for EPA (1998), *Assessment of Dioxins Furans and PCBs in Fish Tissue from Lake Roosevelt Data from Washington Ecology EIM*, http://apps.ecy.wa.gov/eimreporting/Search.asp.
10. Henny, C., R.A. Grove, and J.L. Kaiser (2008), "Osprey distribution, abundance, reproductive success and contaminant burdens along the Lower Columbia River, 1997/1998 versus 2004", in *Archives of Environmental Contaminant Toxicology*, 54:525-534.

Section 4.0: Indicators

1. Loge, F.J., M.R. Arkoosh, T.R. Ginn, L.L. Johnson, and T.K. Collier (2005), "Impact of environmental stressors on dynamics of disease transmission," in *Environmental Science and Technology*, 39:7329–7336.
2. Mallatt, J., M.G. Barron, and C. McDonough (1986), "Acute toxicity of methyl mercury to the larval lamprey, *Petromyzon marinus*," in *Bulletin of Environmental Contamination and Toxicology*, 37:281-288.
3. Haas, J.E. and G. Ichikawa (2007), "Mercury bioaccumulation in Pacific Lamprey Ammocoetes: the roles life history plays," U. S. Fish and Wildlife Service, Sacramento Fish and Wildlife Office, Sacramento, CA. (poster).

Section 5.0: Status and Trends for Mercury, DDT, PCBs, and PBDEs

Mercury

1. Lorraine Edmond, EPA Region 10, compilation of data from GIS analysis of modeling results documented in ICF 2008, cited below.
2. Dwight Atkinson, EPA Office of Water, Washington, D.C., personal communication based on GIS analysis of modeling results documented in ICF 2008, cited below.
3. ICF International (2008), *Model-Based Analysis and Tracking of Airborne Mercury Emissions to Assist in Watershed Planning*, prepared for EPA Office of Water, Washington, D.C., final report August 5, 2008, 350 p.
4. Dwight Atkinson (2008), "Mercury Deposition Modeling for States & Tribes in Regions 8, 9, and 10," Power Point presentation, August 2008.
5. Columbia Riverkeeper (2008), "Columbia River Toxic Discharges Assessment and Mixing Zone Mapping," draft report, based on data reported to Washington Department of Ecology and Oregon Department of Environmental Quality.
6. Environment Canada National Pollutant Release Inventory database, data for Teck Cominco smelter at Trail, British Columbia, available online at http://www.ec.gc.ca/pdb/npri/npri_home_e.cfm.
7. ODEQ (2004), *Willamette Basin Mercury Study*. Oregon Department of Environmental Quality, Laboratory Division, Portland, Oregon.
8. Oregon Department of Human Services, 1997, *Elevated Levels of Mercury in Sport-Caught Bass and Squawfish from the Willamette River*, issued February 13, 1996.
9. Henny, C., R.A. Grove, and J.L. Kaiser (2008), "Osprey distribution, abundance, reproductive success and contaminant burdens along the Lower Columbia River, 1997/1998 versus 2004" in *Archives of Environmental Contaminant Toxicology*, 54:525-534.
10. Henny, C.J., J.L Kaiser, and R.A. Grove (2008), "PCDDs, PCDFs, PCBs, OC pesticides and mercury in fish and osprey eggs from Willamette River, Oregon (1993, 2001 and 2006) with calculated biomagnifications factors." in *Ecotoxicology*, http://www.springerlink.com/content/k63kx77k8117p1x3/fulltext.html.
11. Kruse, G.O., and D.L. Scarnecchia (2002), "Assessment of bioaccumulated metal and organochlorine compounds in relation to physiological biomarkers in Kootenai River white sturgeon," in *Journal of Applied Ichthyology*, 18:430-438.
12. Kruse, G.O., and D.L. Scarnecchia (2002), "Contaminant uptake and survival of white sturgeon embryos," presented at the American Fisheries Society Symposium, 28:151-160.
13. CRITFC (1994), *A Fish Consumption Survey of the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin*, technical report 94-3, Columbia River Inter-Tribal Fish Commission, Portland, Oregon.
14. Lepla, K.B., and J.A. Chandler (1994), revised edition March 1998, *A Survey of White Sturgeon in the Bliss Reach of the Middle Snake River, Idaho*, report prepared for Idaho Power Company, Technical Report FERC No. 1975.
15. Kruse, G.O., and M.H. Webb (2006), *Upper Columbia River White Sturgeon Contaminant and Deformity Evaluation and Summary*, report prepared for the Upper Columbia River White Sturgeon Recovery Team Contaminants Sub-Committee to the Upper Columbia River White Sturgeon Recovery Team, http://uppercolumbiasturgeon.org/Research/Research.html.
16. Foster, E.P., M.S. Fitzpatrick, G.W. Feist, C.B. Schreck, J. Yates, and J.M. Spitsbergen (2001), "Plasma androgen correlation, EROD induction, reduced condition factor, and the occurrence of organochlorine pollutants in reproductively immature white sturgeon (*Acipenser transmontanus*) from the Columbia River, U.S.A," in *Archives of Environmental Contamination and Toxicology*, 41:182-191.
17. Foster, E.P., M.S. Fitzpatrick, G.W. Feist, C.B. Schreck, and J. Yates (2001), "Gonad organochlorine concentrations and plasma steroid levels in white sturgeon *(Acipenser transmontanus)* from the Columbia River, U.S.A.," in *Bulletin of Environmental Contamination and Toxicology*, 67:239-245.
18. Feist, G.W., M.A.H. Webb, D.T. Gunderson, E.P. Foster, C.B. Schreck, A.G. Maule, and M.S. Fitzpatrick (2005), "Evidence of detrimental effects

of environmental contaminants on growth and reproductive physiology of white sturgeon in impounded areas of the Columbia River," in *Environmental Health Perspectives*, Vol. 113, No. 12.

19. Webb, M.A.H., G.W. Feist, M.S. Fitzpatrick, E.P. Foster, C.B. Schreck, M. Plumlee, C. Wong, and D.T. Gunderson (2005), "Mercury concentrations in gonad, liver and muscle tissue of white sturgeon (*Acipenser transmontanus*) in the Columbia River," in *Archives of Environmental Contamination and Toxicology*, 50:443-451.
20. Hill, S. (1975), "Study of mercury and heavy metals pollution in the Jordan Creek drainage," grant number Gy/10816, Student-Originated Studies, National Science Foundation, College of Mines, University of Idaho, Moscow, Idaho.
21. Rinella, F.A., W.H. Mullins, and C.A. Schuler (1994), *Reconnaissance Investigation of Water Quality, Bottom Sediment, and Biota Associated with Irrigation Drainage in the Owyhee and Vale Projects, Oregon and Idaho, 1990-91*, USGS Water-Resources Investigations Report 93 4156.
22. EPA (2003), "National Study of Chemical Residues in Lake Fish Tissue - 1999 to 2003," http://www.epa.gov/waterscience/fish/study/.
23. Seiders, K., C. Deligeannis, and P. Sandvik (2007), Washington Department of Ecology, *Washington State Toxics Monitoring Program: Contaminants in Fish Tissue from Freshwater Environments 2004 and 2005*, Publication No. 07-03-024; June.
24. Stone, H. (2006), "Brownlee Reservoir mercury TMDL fish tissue study: results and field summary," Idaho Department of Environmental Quality, June.
25. Clark, G.M., and T.R. Maret (1998), *Organochlorine Compounds and Trace Elements in Fish Tissue and Bed Sediments in the Lower Snake River Basin, Idaho and Oregon*, USGS Water-Resources Investigations Report 98-4103.
26. Data shared by T.R. Maret, USGS trmaret@usgs.gov, 2004-2007 data collection.
27. EPA (2002), *Columbia River Basin Fish Contaminant Survey, 1996-1998*. EPA Region 10, Seattle, WA. EPA 910/R-02-006, July.
28. Large Rivers Idaho Frame EMAP sampling (2006), (unpublished).
29. Idaho Fish Consumption Advisory Program Data, J. Vannoy, Idaho Department of Health, VannoyJ@dhw.idaho.gov.

DDT

1. Fuhrer, G.J., J. Morace, H.M. Johnson, J. Rinella, J.C. Ebbert, S.S. Embrey, I.R. Waite, K.D. Carpenter, D.R. Wise, and C.A. Hughes (2004), "Water Quality in the Yakima River Basin, Washington 1999-2000," USGS Circular 1237.
2. USGS Circular 1090, USGS WRIR 98-4113.
3. EPA (1992), *National Study of Chemical Residues in Fish*, EPA 823-R-92-008a.
4. Johnson, A., B. Era-Miller, and R. Coots (2007), *Chlorinated Pesticides, PCBs, and Dioxins in Yakima River Fish in 2006: Data Summary and Comparison to Human Health Criteria*, Washington Department of Ecology, Publication No. 07-03-036, July.
5. EPA (2002), *Columbia River Basin Fish Contaminant Survey, 1996-1998*, EPA Region 10, Seattle, Washington, EPA 910/R-02-006, July.
6. Foster, E.P., M.S. Fitzpatrick, G.W. Feist, C.B. Schreck, J. Yates, and J.M. Spitsbergen (2001), "Plasma androgen correlation, EROD induction, reduced condition factor, and the occurrence of organochlorine pollutants in reproductively immature white sturgeon (*Acipenser Transmontanus*) from the Columbia River, U.S.A.," in *Archives of Environmental Contamination and Toxicology*, 41, 182-191.
7. Feist, G.W., M.A.H. Webb, D.T. Gunderson, E.P. Foster, C.B. Schreck, A.G. Maule, and M.S. Fitzpatrick (2005), "Evidence of detrimental effects of environmental contaminants on growth and reproductive physiology of white sturgeon in impounded areas of the Columbia River," in *Environmental Health Perspectives*, Vol.113, No. 12.
8. Foster, E.P., M.S. Fitzpatrick, G.W. Feist, C.B. Schreck, J. Yates, J.M. Spitsbergen, and J. Heidel (2001), "Altered reproductive physiology, EROD induction, reduced condition factor, and the occurrence of organochlorine pollutants in white sturgeon (*Acipenser transmontanus*) from the Columbia River," in *Archives of Environmental Contamination and Toxicology*, 41(2):182-191.

9. Foster, E.P., M.S. Fitzpatrick, G.W. Feist, C.B. Schreck, and J. Yates (2001), "Gonad organochlorine concentrations and plasma sex steroid levels in white sturgeon (*Acipenser transmontanus*) from the Columbia River, USA.," in *Bull. Environ. Contam. Toxicol.*, 67:239-245.
10. Foster, E.P., M.S. Fitzpatrick, G.W. Feist, C.B. Schreck, J. Yates, J.M. Spitsbergen, and J.R. Heidel (2001), "Plasma androgen correlation, EROD induction, reduced condition factor, and the occurrence of organochlorine pollutants in reproductively immature white sturgeon (*Acipenser transmontanus*) from the Columbia River, USA.," in *Archives of Environmental Contamination and Toxicology*, 41(2):182-91.
11. Lepla, K.B., and J.A. Chandler (1994), revised edition March 1998, *A Survey of White Sturgeon in the Bliss Reach of the Middle Snake River, Idaho*, report prepared for Idaho Power Company, Technical Report FERC No. 1975.
12. Kruse, G.O., and M.H. Webb (2006), *Upper Columbia River White Sturgeon Contaminant and Deformity Evaluation and Summary*, report prepared for the Upper Columbia River White Sturgeon Recovery Team Contaminants Sub-Committee to the Upper Columbia River White Sturgeon Recovery Team, http://uppercolumbiasturgeon.org/Research/Research.html.
13. Kruse, G.O., and D.L. Scarnecchia (2002), "Assessment of bioaccumulated metal and organochlorine compounds in relation to physiological biomarkers in Kootenai River white sturgeon," in *Journal of Applied Ichthyology*, 18:430-438.
14. Anthony, R.G., M.G. Garrett, and C.A. Schuler (1993), "Environmental contaminants in bald eagles in the Columbia River estuary," in *Journal of Wildlife Management*, 57(1): 10-19.
15. Buck, J.A., R.G. Anthony, and F.B. Isaacs (1999), *Changes in Productivity and Environmental Contaminants in Bald Eagles Nesting Along the Lower Columbia River*, USFWS and Oregon Cooperative Wildlife Research Unit, Study ID 13420-1130-1F16.
16. Buck, J.A., R.G. Anthony, C.A. Schuler, F.B. Isaacs, and D.E. Tillitt (2005), "Changes in productivity and contaminants in bald eagles nesting along the Lower Columbia River, USA.," in *Environmental Toxicology and Chemistry*, Vol. 24, No. 7, pp. 1779-1792.
17. Gabrielson, I.N., and S.G. Jewett (1940), *Birds of Oregon*, Oregon State College Press, Corvallis.
18. Henny, C.J., J.A. Collins, and W.J. Deibert (1978), "Osprey distribution, abundance, and status in western North America: II. the Oregon population," *The Murrelet*, 59:14-25.
19. Henny, C.J., J.L. Kaiser, and R.A. Grove (2005), "Ospreys in Oregon and the Pacific Northwest," USGS Fact Sheet 153-02 (revised), 4 pp., http://fresc.usgs.gov/products/fs/fs-153-02.pdf.
20. Henny, C.J., R.A. Grove, and J.L. Kaiser (2008), "Osprey distribution, abundance, reproductive success and contaminant burdens along the Lower Columbia River, 1997/1998 versus 2004," in *Archives of Environmental Contaminant Toxicology*, 54:525-534.
21. Isaacs, F. (2002), "25-Year Study Shows Oregon Bald Eagles Doing Well," unpublished data, OSU, http://oregonstate.edu/dept/ncs/newsarch/2002/Dec02/eagles.htm.

PCBs

1. Johnson, A., D. Serdar, and D. Davis (1994), "Results of 1993 Screening Survey on PCBs and Metals in the Spokane River, April 1994," in Washington Department of Ecology Publication No. 94-e24 : July 11, 1994 memo to Carl Nuechterlein from Dale Davis and Dave Serdar with corrections.
2. Serdar, D., and A. Johnson (2006), "PCBs, PBDEs and selected metals in Spokane River fish, 2005," Ecology Publication 06-03-025.
3. Johnson, A. (1996), "Results on PCBs in Upper Spokane River Fish, Washington Department of Ecology memo to Carl Neuchterlein and Dave Knight," in Washington DOE Publication 97-304, July 8, 1997.
4. Johnson, A., (2000), "Results from Analyzing PCBs in 1999 Spokane River Fish and Crayfish Samples, September," in Washington Department of Ecology Publication No. 00-03-040.

5. Seiders, K., C. Deligeannis, and K. Kinney (2006), "Washington State Toxics Monitoring Program: Toxic Contaminants in Fish Tissue and Surface Water from Freshwater Environments, 2003," in Washington State Department of Ecology Publication No. 06-03-019.
6. Feist, G.W., M.A.H. Webb, D.T. Gunderson, E.P. Foster, C.B. Schreck, A.G. Maule, and M.S. Fitzpatrick (2005), "Evidence of detrimental effects of environmental contaminants on growth and reproductive physiology of white sturgeon in impounded areas of the Columbia River," in *Environmental Health Perspectives*, Vol.113 No. 12.
7. LCREP (Lower Columbia River Estuary Partnership) (2007), *Lower Columbia River Estuary Ecosystem Monitoring: Water Quality and Salmon Sampling Report*, prepared by the Lower Columbia River Estuary Partnership, Portland, Oregon.
8. Johnson, L.L., G.M. Ylitalo, C.A. Sloan, B.F. Anulacion, A.N. Kagley, M.R. Arkoosh, R.A. Lundrigan, K. Larson, M. Siipola, and T.K. Collier (2007), "Persistent organic pollutants in outmigrant juvenile chinook salmon from the Lower Columbia Estuary, USA," in *Science of the Total Environment*, 374:342-366.
9. Meador, J.P., T.K. Collier, and J.E. Stein (2002), "Use of tissue and sediment-based threshold concentrations of polychlorinated biphenyls (PCBs) to protect juvenile salmonids listed under the US Endangered Species Act," in *Aquatic Conserv: Mar. Freshw. Ecosyst.* 12: 493–516 (2002).
10. Johnson, A., and D. Norton (2005), *Concentrations of 303(d) Listed Pesticides, PCBs and PAHs, Measured with Passive Samplers Deployed in the Lower Columbia River*, Washington Department of Ecology, Publication No. 05-03-006.
11. Henny, C.J., R.A. Grove, and O.P. Hedstrom (1996), *Field evaluation of mink and river otter on the Lower Columbia River and the influence of environmental contaminants*, final report submitted to the Lower Columbia River Bi-State Water Quality Program, http://www.lcrep.org/pdfs/44.%20 01538.pdf.
12. Henny, C.J., L.J. Blus, S.V. Gregory, and C.J. Stafford (1981), "PCBs and organochlorine pesticides in wild mink and river otters from Oregon," in *Proceedings of the Worldwide Furbearer Conference* 3:1763-780.
13. Elliott et al. (1998), "Chlorinated hydrocarbons in livers of American mink (*Mustela vison*) and river otter (*Lontra canadensis*) from the Columbia and Fraser River Basins, 1990-1992," in *Environmental Monitoring and Assessment*, 57: 229-252.
14. Grove, R.A., and C.J. Henny (2007), "Environmental contaminants in male river otters from Oregon and Washington, USA, 1994-1999," in *Environmental Monitoring and Assessment*, 145:49-73.
15. Buck, J.A., R.G. Anthony, and F.B. Isaacs (1999), "Changes in productivity and environmental contaminants in bald eagles nesting along the Lower Columbia River," USFWS and Oregon Cooperative Wildlife Research Unit. Study ID 13420-1130-1F16.
16. Buck, J.A., R.G. Anthony, C.A. Schuler, F.B. Isaacs, and D.E. Tillitt (2005), "Changes in productivity and contaminants in bald eagles nesting along the Lower Columbia River, USA.," in *Environmental Toxicology and Chemistry*, Vol. 24, No. 7, pp. 1779-1792.
17. Henny, C.J., R.A. Grove, and J.L. Kaiser (2008), "Osprey distribution, abundance, reproductive success and contaminant burdens along the Lower Columbia River, 1997/1998 versus 2004", in *Archives of Environmental Contaminant Toxicology*, 54:525-534.
18. Siipola, M., T. Sherman, R. Abney, D. Ebner, J. Stevens, J. Clarke, and G. Ray (2007), "*Corbicula fluminea*: a potential freshwater bioaccumulative test species," USACE Portland District and the Engineer Research Development Center (poster).

PBDEs

1. Samara, F., C. W. Tsai, D. S. Aga (2006) "Determination of potential sources of PCBs and PBDEs in sediments of the Niagara River", in Environmental Pollution 139:3 489-497.

2. Hale, R.C., M.J. La Guardia, E.P. Harvey, T.M. Mainor, W.H. Duff, and M.O. Gaylor, (2001), "Polybrominated diphenyl ethers flame retardants in Virginia Freshwater Fishes (USA)," in *Environmental Science and Technology*, 35, 23:4585 – 4591.
3. North, K.D., "Tracking Polybrominated Diphenyl Ether (PBDE) Releases in a Wastewater Treatment Plant Effluent, Palo Alto, California, USA" Karin.North@cityofpaloalto.org.
4. Song, M., C. Shaogang, J. Letcher, and R. Seth (2006), "Fate, partitioning and mass loading of polybrominated diphenyl ethers (PBDEs) during the treatment processing of municipal waste," in *Environmental Science and Technology*, 40:6241-6246.
5. Hale, R.C., M. Alaee, J.B. Manchester-Neesvig, H.M. Stapleton, and M.G. Ikonomou (2003), "Polybrominated diphenyl ethers flame retardants in the North American environment," in *Environmental International*, 29:771–779.
6. Watanabe, I., and S. Sakai (2003), "Environmental release and behavior of brominated flame retardants," in *Environmental International*, 29:665-682.
7. Rayne, S., M.G. Ikonomou, and B. Antcliffe (2003), "Rapidly increasing brominated diphenyl ethers concentrations in the Columbia River System from 1992 to 2000," in *Environmental Science and Technology*, 37(13): 2847-2854.
8. Serdar, D., and A. Johnson (2006), *PCBs, PBDEs and Selected Metals in Spokane River Fish*, 2005, Ecology Publication 06-03-025.
9. Johnson, A., K. Seiders, C. Deligeannis, K. Kinney, P. Sandvik, B. Era-Miller, and D. Alkire (2006), *PBDE Flame Retardants in Washington Rivers and Lakes: Concentrations in Fish and Water, 2005-06*, Washington Department of Ecology Publication No. 06-03-027, August, Olympia, Washington.
10. Johnson, A., and N. Olson, (2001), "Analysis and Occurrence of Polybrominated Diphenyl Ethers in Washington State Freshwater Fish," in *Archives of Environmental Contamination and Toxicology*, 41:339-344, April.
11. LCREP (2007), *Lower Columbia River and Estuary Ecosystem Monitoring: Water Quality and Salmon Sampling Report*, 70 p. Lower Columbia River Estuary Partnership, Portland, Oregon.
12. Siipola, M., T. Sherman, R. Abney, D. Ebner, J. Stevens, J. Clarke, and G. Ray (2007), "*Corbicula fluminea*: a potential freshwater bioaccumulative test species," USACE Portland District and the Engineer Research Development Center (poster).

Section 6.0: Current and Planned Toxics Reduction Efforts

1. ODEQ (2008), *Human Health Focus Group Report: Oregon Fish and Shellfish Consumption Rate Project*, June.
2. EPA Region 8 website, http://epa.gov\region8\superfund\mt\milltowncfr.
3. Henny, C.J., R.A. Grove, J.L. Kaiser, and V.R. Bentley (2004), "An evaluation of Osprey eggs to determine spatial residue patterns and effects of contaminants along the lower Columbia River, USA", pp. 369-388, in *Raptors Worldwide*, R.D. Chancellor and B.-U. Meyburg, eds., WWGBP and MME, Budapest, Hungary.
4. Fuhrer, G.J., J. Morace, H.M. Johnson, J. Rinella, J.C. Ebbert, S.S. Embrey, I.R. Waite, K.D. Carpenter, D.R. Wise, and C. A. Hughes (2004), "Water Quality in the Yakima River Basin, Washington 1999-2000," USGS Circular 1237.
5. Walla Walla Pesticide Stewardship Partnership (2008), *Summary Report: Pesticide Monitoring in the Walla Walla Basin 2005-2007 Results, Department of Environmental Quality*, Oregon Department of Environmental Quality, September 2007, or http://www.deq.state.or.us/wq/pubs/factsheets/community/pesticide.pdf.
6. Idaho State Department of Agriculture, Pesticide Disposal Program, http://www.agri.state.id.us/Categories/Pesticides/pdp/indexdisposalmain.php.
7. ODEQ (2008), Environmental Quality Commission, *Mutual Agreement and Order*, July 17, 2008, DEQ no. AQ/V-ER-08-022.
8. USGS (2007), http://minerals.usgs.gov/west/projects/nngd.htm.

9. Portland General Electric average of 2000-2006 emissions reported for the Boardman, Oregon, plant.

Section 8.0: Toxics Reduction Initiatives

1. ODEQ (1996), *Identification of Sources of Pollutants to the Lower Columbia River Basin*, prepared by the Lower Columbia River Bi-State Water Quality Program, T. Rosetta and D. Borys, June.

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Science Advisory Board Consultation Document
Proposed Revisions to Aquatic Life Guidelines

Tissue-Based Criteria for "Bioaccumulative" Chemicals

Prepared by the
Tissue-based Criteria Subcommittee

August 2005

NOTICE

THIS DOCUMENT IS A PRELIMINARY DRAFT

It has been prepared for consultation with U.S. EPA's Science Advisory Board. It has not been formally released by the U.S. Environmental Protection Agency and should not at this stage be construed to represent Agency guidance or policy.

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Table of Contents

1 Introduction

1.1 Purpose and Scope of this Document

This document summarizes the current thinking of the U.S. Environmental Protection Agency (EPA)/U.S. Department of the Interior (DOI) Tissue-based Criteria Subcommittee (hereafter called the "Subcommittee") regarding approaches for revising EPA's Aquatic Life Criteria Guidelines to address risks from so called "bioaccumulative" chemicals. The Subcommittee drafted this document specifically to facilitate the 2005 Science Advisory Board (SAB) consultation on EPA's revision of its Aquatic Life Criteria Guidelines. Although nearly all chemicals bioaccumulate to some degree in aquatic organisms, we use the term "bioaccumulative" in this document to delineate chemicals which bioaccumulate extensively in aquatic food webs such that exposure from the diet becomes toxicologically important to relevant ecological receptors. Such chemicals generally persist in the aquatic environment, exhibit high hydrophobicity (e.g., log K_{ow} generally > 5), and are poorly metabolized by aquatic biota. The ecological receptors of primary concern for bioaccumulative chemicals in aquatic systems include aquatic life and aquatic-dependent wildlife (i.e., terrestrial wildlife that feed extensively on aquatic organisms).

We have organized this document into five main sections. First is an introduction that presents a brief history of EPA aquatic life criteria and exposes the need for a revised methodology that specifically addresses bioaccumulative chemicals. The second section provides the rationale and an overview of the proposed tissue-based criteria approach for both aquatic life and aquatic-dependent wildlife. The third and fourth sections present the salient features of tissue-based criteria for aquatic life and wildlife, respectively. The last section presents technical issues that the Subcommittee is seeking input from the SAB.

We think it is important to point out that this document represents "a work in progress" and that many issues and ideas have yet to be fully discussed or even explored by the Subcommittee. For example, the Subcommittee focused to date on national-level criteria but we recognize the need to address regional or site-specific criteria that account for regional or site-specific concerns. Further, the Subcommittee has not discussed in detail the application of population models for deriving aquatic life criteria for bioaccumulative chemicals, choosing instead to defer to the work of the water-based criteria subcommittee since most of the methodological issues will be the same. Finally, the Subcommittee clearly recognizes that the concept of tissue-based criteria can be appropriate for other types of chemicals (i.e., not just those where dietary exposure is important) and in particular where mixtures of chemicals with similar modes of action are of concern. To date, however, the Subcommittee has chosen to focus its efforts first on bioaccumulative chemicals due to concerns with the ability of existing Guidelines to adequately address risks from this group of chemicals.

The criteria process outlined in this document strives to make the best use of the data currently available. As this process evolves and guidance is developed for use by implementers in State, Tribal and local agencies, we intend for that guidance to also indicate what types of additional data or studies could have the greatest impact on improving the quality of the

assessment. In this way, the quality of criteria could continue to improve as data become available.

1.2 History of Aquatic Life Criteria

Prior to 1980, EPA derived aquatic life criteria for toxic chemicals using an "ad hoc" approach (i.e., formal procedures for their derivation were not codified). Criteria were usually established by citing the data deemed most relevant by those selected to derive the criterion for a given pollutant. This approach allowed for substantial inconsistencies in how toxicity data were used and the resulting level of protection provided, particularly since no minimum data requirements were established.

In 1980, EPA established for the first time written guidelines for deriving aquatic life criteria. These guidelines were last updated in 1985 (Stephan et al., 1985). In order to place the proposed approach for deriving tissue-based criteria into perspective, pertinent features of the 1985 Guidelines are summarized below.

Selected Features of the 1985 Guidelines

(1) Criteria are represented by a two-number system (an acute criterion derived for short-term exposures and a chronic criterion derived for long-term exposures) and are expressed as water concentrations.

(2) Species sensitivity is characterized using a species sensitivity distribution (SSD) with interpolation or extrapolation to obtain a criterion concentration protective of 95% of tested taxa.

(3) Minimum database requirements must be met in order to derive criteria (8 genera for acute criteria and 3 genera for chronic criteria from diverse taxonomic groups).

(4) Toxicity test data are based on water only exposures with only negligible exposure to chemicals from food.

(5) Acute criteria are based on 48-hr – 96 hr acute toxicity tests involving severe endpoints (e.g., survival, immobilization). Chronic criteria are based on longer term toxicity tests of early life stages, a partial life cycle, or a full life cycle involving endpoints such as survival, growth, reproduction and development. Data not conforming to these exposure durations are generally not used.

(6) Due to the limited amount of chronic toxicity data, derivation of chronic criteria often involves the use of acute-chronic ratios (ACRs) for extrapolating from acute to chronic effect concentrations.

(7) The 1985 Guidelines contain a procedure to derive a "final residue value" that attempts to address exposure to bioaccumulative chemicals. However, the science concerning bioaccumulation and subsequent EPA guidance for addressing bioaccumulation have evolved substantially over the last two decades such that this procedure is considered obsolete.

(8) The 1985 Guidelines also recommend an "averaging period" (1 hour for acute, 4 days for chronic) that is designed to address fluctuating exposures. The Guidelines also recommend an "allowable frequency" for exceeding the criterion (once in three years on average) which is intended to address the time needed for aquatic ecosystem recovery between criteria violations.

1.3 Limitations of 1985 Guidelines for Bioaccumulative Chemicals

In 1990, EPA convened a workgroup of scientists with the charge of revising the 1985 Guidelines to reflect the latest available science. Among other findings, ***the workgroup concluded that a separate set of procedures were needed for deriving aquatic life criteria for bioaccumulative chemicals.*** This conclusion grew out of recognition that the 1985 Guidelines contain a number of fundamental limitations with respect to deriving criteria for bioaccumulative chemicals. Specifically, the 1985 Guidelines:

(1) Lack a prescriptive procedure for addressing risks to aquatic life that result from exposure to chemicals from the diet (food web).

(2) Rely heavily on toxicity test data that often do not account for the slow accumulation kinetics of many bioaccumulative chemicals and consequently, may underestimate effects associated with long-term (steady state) accumulation.

(3) Lack a scientifically rigorous procedure for addressing chemical risks to aquatic-dependent wildlife (e.g., piscivorous birds and mammals).

1.4 Revision Efforts of the 1990s

Much of the effort of the Guidelines revision workgroup in the 1990s focused on developing a new framework for deriving aquatic life criteria for so-called "non-bioaccumulative" chemicals (i.e., chemicals where exposure via the diet is not a primary concern). As discussed in the companion SAB Consultation Document titled: *"Water-based Criteria,"* competing priorities impeded EPA's progress on the revising the 1985 Guidelines in the 1990s.

One of these competing priorities was the Great Lakes Water Quality Initiative (GLWQI) rulemaking, whereby EPA developed new chemical criteria for aquatic life, wildlife, and human health for the Great Lakes system (USEPA 1995a). Of particular relevance here is the GLWQI criteria focused on bioaccumulative chemicals and contained new procedures for deriving wildlife and human health criteria that accounted for chemical bioaccumulation in aquatic food webs[1]. These new procedures consisted of the use of bioaccumulation factors, biota-sediment accumulation factors and food web bioaccumulation models to estimate chemical accumulation in the aquatic diet of wildlife and humans residing in the Great Lakes system (USEPA, 1995b). These bioaccumulation methods were subsequently modified and extended to a national level with EPA's publication of its *Methodology for Deriving Water Quality Criteria for the Protection of Human Health* (USEPA, 2000; 2003). With appropriate modifications, the Subcommittee believes the bioaccumulation methods published in EPA's human health criteria methodology are applicable to aquatic life and aquatic-dependent wildlife receptors. To date, however, EPA has no national criteria methodology that specifically addresses risks from bioaccumulative chemicals to aquatic life and aquatic-dependent wildlife.

[1] The GLWQI criteria for aquatic life did not address food web bioaccumulation.

2 General Overview of Tissue-based Approach

2.1 What Are "Tissue-based Criteria?"

The Subcommittee is proposing to use a tissue-based approach for deriving criteria that protect aquatic life and aquatic-dependent wildlife from harmful exposure to bioaccumulative chemicals. We use the term "tissue-based criteria" to represent criteria that are derived from toxicological data expressed as concentrations in target organisms (e.g., commonly referred to in the literature as critical body residues, lethal body burdens, tissue residue-response relationships) as opposed to concentrations in ambient media (water, sediment). For aquatic-dependent wildlife, we consider tissue-based criteria to also include criteria that are based on toxicological data expressed as concentrations in aquatic organisms that compose their diet (e.g., mg chemical/kg food). The use of diet-based toxicological data will likely be reserved for aquatic-dependent wildlife because: (1) such data are more plentiful than toxicity data expressed as concentrations in wildlife tissues, and (2) exposure of wildlife to bioaccumulative chemicals from water ingestion is generally considered negligible relative to the diet, unlike aquatic organisms where exposure to chemicals via both food and water can be important. Thus, we use the term "tissue-based criteria" broadly to represent criteria derived both from toxicity data expressed as concentrations in tissues of the target organisms or their diet.

> We use the term "tissue-based criteria" broadly to represent criteria derived both from toxicity data expressed as concentrations in tissues of the target organisms or their diet (for wildlife).

2.2 Why Use a Tissue-based Approach for Bioaccumulative Chemicals?

The primary motive behind our pursuit of a tissue-based approach for bioaccumulative chemicals is the desire to account for multiple routes of exposure (e.g., diet, sediment, water) in the derivation of criteria. Chemical accumulation in the aquatic food web and subsequent dietary exposure is a dominant concern for bioaccumulative chemicals. Toxicological data based on internal (tissue) concentrations are attractive because they incorporate chemical uptake from different routes of exposure. Another motivating factor is that appropriate expressions of toxicity on a tissue concentration basis inherently account for toxicokinetic differences that exist among species. Conceptually, this should act to reduce variability in toxicity measurements between species caused by differing rates of uptake, distribution, metabolism, and elimination that would otherwise be reflected in media-based expressions of toxicity. Tissue-based expressions of toxicity also account for factors affecting the bioavailability of chemical concentrations in exposure media which can be a major source of variability in water-based toxicity test data. Finally, tissue-based expressions of toxicity appear to be promising for addressing exposure to chemical mixtures, particularly for those with common mode(s) of action.

The Subcommittee notes that the concept of expressing toxicological data for aquatic organisms on the basis of tissue or whole body concentrations is not new (e.g., Könemann, 1981) Veith et al. 1983; McCarty, 1986; Cook et al., 1989; 1993; McCarty and Mackay, 1993) and a substantial body of literature has evolved around this approach. For organic chemicals exhibiting a narcotic mode of action, the lethal tissue residue or body burden concept has its foundations in the early developments of quantitative structure activity relationships (QSARs)

involving octanol-water partition coefficients (K_{ow}), bioconcentration and acute toxicity (Veith et al., 1979; Veith et al. 1983; McCarty 1986). More recently, the lethal body-burden concept has been advanced as a method for deriving criteria for narcotic chemicals, including PAH mixtures (Di Toro, et al., 2000; Di Toro and McGrath 2000), although the toxicological basis for these criteria is driven mostly by measurements of acute lethality. EPA's development of the Biotic Ligand Model for cationic metals and application to deriving criteria for copper is also based implicitly on a lethal tissue residue approach (e.g., accumulation on the gill for fish; Di Toro et al., 2001; Paquin et al., 2002).

The use of tissue concentrations for expressing toxicological effects has been evaluated for compounds with reactive and specific modes of action (Verhaar et al., 1999; Legierse et al., 1999) and has been the subject of several critical reviews (Barron et al., 2002; Escher and Hermens, 2002; Beyer et al., 1996). For specific modes of action that involve irreversible (or less than reversible) binding of the toxicant with target sites, a critical target occupation model has been proposed for describing the time dependent toxicity (Legierse et al., 1999). This model does not assume a constant internal effect concentration with time, as often assumed with baseline toxicity. Furthermore, databases containing tissue-based toxicity data have been developed (USACE, 2004; Jarvenin and Ankley, 1999). Recently, EPA published draft aquatic life criteria for selenium that use a tissue-based approach (USEPA, 2004).

2.3 Guiding Principles of Tissue-based Criteria

The following principles or attributes helped guide the Subcommittee's thinking on how construct a methodology for deriving tissue-based criteria for bioaccumulative chemicals.

1. **Scientific Defensibility.** The methodology produces criteria that use the best available science and are scientifically defensible.
2. **Flexibility**. The methodology is flexible enough to accommodate the heterogeneous nature of available data including "data poor" and "data rich" situations.
3. **Transparency.** The methodology is transparent in how criteria are derived and how they can be set to satisfy different risk management objectives.
4. **Consistency.** The methodology is sufficiently prescribed such that its repeated application to the same dataset by appropriate users should result in the same (or similar) criteria values.
5. **Uncertainty.** The methodology does not discourage the generation of new data or methods for reducing uncertainty in the criterion.
6. **Site-Specificity.** The methodology is readily adaptable to enable derivation of criteria that reflect site- or region-specific attributes.
7. **Level of Effort.** The data requirements of the methodology are not be so onerous such that essentially no tissue-based criteria could be derived in the near future (i.e., the next 5 years) without the generation of a substantial amount of new data.
8. **Implementation.** The methodology facilitates the translation of tissue criteria to corresponding concentrations in various environmental compartments (e.g., water, food web) to address implementation and monitoring needs.

2.4 What is the General Process for Deriving Tissue-based Criteria?

This framework focuses primarily on a national-level process for deriving tissue-based criteria. The derivation of a national-level criterion will provide an analysis of all available toxicity data and a description and background on the parameter estimates used for representative species. National-level criteria may be adopted by State, Tribal, or local agencies or may be modified at state or local scales if sufficient additional information is available to improve the characterization of risk while maintaining the intended level of protection for aquatic life and wildlife. The framework will be expanded in the future to provide guidance on when and how site-specific criteria could be derived.

The current view of the Subcommittee is that guidelines for deriving tissue-based criteria for bioaccumulative chemicals would consist of two primary components:

1) Procedures for deriving a national tissue criterion (or criteria)

2) Procedures for translating a national tissue criterion (criteria) into corresponding concentrations in media and the aquatic food web.

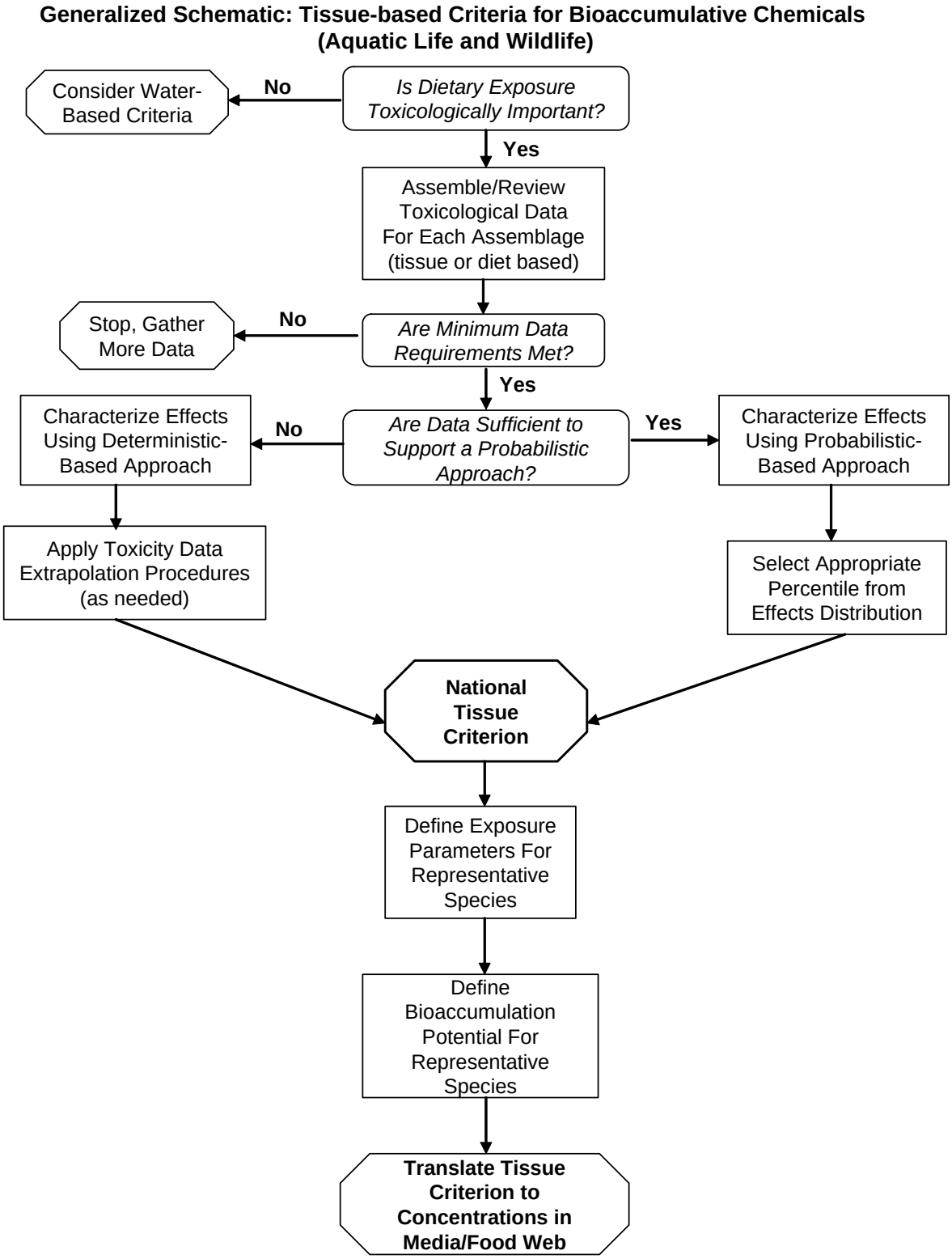
The second component (translating tissue criteria to media and food web concentrations) addresses both scientific and regulatory needs concerning the relationship between chemical loadings and accumulated chemical residues in tissue (i.e., bioaccumulation). Below, we provide an overview of these two components as they pertain to both aquatic life and aquatic-dependent wildlife with additional details following in Sections 3 and 4, respectively.

2.4.1 Derivation of a National Tissue Criterion

Figure 1 illustrates some of the primary decisions points and steps the Subcommittee is considering for deriving tissue-based criteria for aquatic life and aquatic-dependent wildlife with respect to bioaccumulative chemicals. For simplicity in presentation, we have chosen to represent only some of the decisions steps in the derivation process or have combined several steps into a single box.

As discussed in the Overview SAB Consultation Document, the derivation of a "water-based" or "tissue-based" criterion would begin with a "problem formulation" step, whereby critical assessment questions are formulated and addressed, a conceptual model developed, and an overall plan for analyzing the data is produced. Details of the problem formulation step are described in the Overview document and in Sections 3 and 4 of this document for aquatic life and aquatic-dependent wildlife, respectively. Therefore, it is assumed in Figure 1 that a complete problem formulation phase would be conducted, of which only certain steps are captured in this schematic. The primary steps of the schematic are as follows:

Figure 1.



1) **Determine Need for Tissue-based Criteria.** One of the first steps will be to determine the relative utility of a tissue-based approach for the chemical of concern as compared to a water-based approach. In the context of bioaccumulative chemicals, we expect the primary determinant to be the relative importance of chemical exposure via the diet to overall risks experienced by aquatic life and aquatic-dependent wildlife. Generally, the greater the importance of diet in governing chemical exposure and effects, the less likely that a water-based approach would be suitable and more likely a tissue-based approach would be used. Information on chemical properties (e.g, K_{ow}, persistence, etc.), trophic transfer, and toxicology of diet-borne chemical would be consulted as part of this decision step.

2) **Gather, Synthesize Toxicity Data.** Once an initial decision to pursue a tissue-based approach is made[2], all relevant data on the toxicity of the chemical would be assembled, with the primary focus on data that relate toxicological effects to chemical concentrations in tissue(s) and/or diet (in the case of wildlife). It is at this step in problem formulation where decisions are made about the form(s) of the chemical of concern, the most appropriate tissue(s) for expressing toxicological effects, the most appropriate toxicological endpoints to consider, relative sensitivity of taxa and life stages, in addition to proper screening of data for quality purposes.

3) **Determine Feasibility.** Once the appropriate toxicological data are reviewed, evaluated and synthesized, acceptable studies will be evaluated in the context of "minimum data requirements" which are established for each taxonomic assemblage to ensure that coverage of a diverse range of species is achieved. The assemblages being considered are aquatic plants, aquatic invertebrates, aquatic vertebrates (i.e., fish and some forms of amphibians), birds, mammals, and reptiles.

4) **Select General Derivation Approach.** If data meet the minimum data requirements, another key step within problem formulation will be to determine the approach to be used to characterize the effects on aquatic/wildlife species. The body of available toxicological data is evaluated to determine whether data are sufficient to support a probabilistic basis for setting the tissue criterion (e.g., species sensitivity distribution) or a deterministic basis (e.g., using data from a good quality study on an appropriately sensitive species). The selection of a probabilistic or deterministic approach is based on the quality and quantity of available toxicity data.

5) **Probabilistic Methods.** Probabilistic approaches have several advantages over deterministic approaches for deriving criteria, however they generally require that data be available from a relatively large number of species in order to reliably describe the overall distribution in species sensitivity. The decision on how much data are adequate to conduct a probabilistically-based approach has not been made by the Subcommittee but is one of many issues it intends to address in the future. In the case of species sensitivity distributions, one advantage is that the tissue criterion could be set using an appropriate

[2] Note: the tissue or water-based approaches are not mutually exclusive, and both may be initially applied for some chemicals where the relative value of one approach over the other is ambiguous.

percentile from the species sensitivity distribution. This is analogous to the current approach used in the 1985 Guidelines. An example of how a species sensitivity distribution can be applied to tissue-based toxicity data is illustrated by Steevens et al. (2005) for 2,3,7,8-TCDD.

6) **Deterministic Methods.** Deterministic approaches (e.g., characterizing effects based on data for an appropriately selected species or set of species) can be used with substantially less data but often are accompanied by the use of toxicity extrapolation procedures (e.g., uncertainty factors) which introduce uncertainty in the analysis.

7) **National Tissue Criterion.** Whichever approach is used to characterize toxicological effects, the goal of the methodology is to derive a tissue criterion (or criteria) that represents a concentration in tissue of aquatic life and/or aquatic-dependent wildlife that is deemed appropriately protective of the respective assemblages of species. In the case of aquatic-dependent wildlife, this tissue concentration may be expressed as concentrations in the aquatic diet.

2.4.2 Translation of Tissue Criterion to Concentrations in Media, Food Web

The Subcommittee anticipates the need to develop guidelines for translating tissue-based aquatic life and wildlife criteria into corresponding concentrations in environmental media (e.g., water) and/or other components of the aquatic food web for the following reasons:

- **Implementation.** Monitoring and enforcing pollutant discharge limits on the basis of measured chemical concentrations in tissues of organisms may not be practical or desirable in all situations (e.g., aquatic-dependent wildlife).

- **Intrinsic Toxicity vs. Risk.** The distribution of species sensitivity on the basis of tissue concentration-effect values (e.g., mg/kg-tissue) does not necessarily equate to the distribution of "risks" that would be experienced by those species from a given chemical concentration in water. While tissue-based toxicity data reflect the "intrinsic toxicity" of a chemical because bioavailability and toxicokinetic factors are addressed, such data do not reflect species-specific differences in exposure potential. For bioaccumulative chemicals, exposure potential can vary substantially among species due to differences in trophic position, habitat zone, and consumption rates. Therefore, the most sensitive species on a tissue concentration basis may not be the species "most at risk" on a water concentration (and chemical loading) basis.

The Subcommittee recognizes that translating tissue-based criteria for bioaccumulative chemicals into corresponding media concentrations involves a number of processes and parameters (e.g., bioaccumulation, food consumption patterns and rates, etc.) that can vary substantially across sites. Therefore, the current thinking is that procedures for translating tissue criteria into media concentrations would be developed in order to facilitate the use of appropriate site-specific data when available. In situations where such site-specific data are not available,

the Subcommittee anticipates that appropriate "nationally representative" parameter values could be used.

Continuing with Figure 1, the following general steps would be followed for translating tissue criteria into media concentrations.

1) **Define Exposure Potential of Representative Species.** It appears likely that the exact identity of species corresponding to the national tissue criterion (summarized above) will not be known. This situation is likely to occur because both deterministic and probabilistic approaches for characterizing effects will probably involve some type of extrapolation or interpolation of toxicity values among species (e.g., selecting a percentile from an SSD, applying uncertainty factors) in order to determine a tissue criterion that is protective of the overall assemblage. For example, the identity of a hypothetical species corresponding to the 5^{th} percentile from a SSD would likely be unknown, as would the components of its diet. Because the translation of tissue concentrations to media concentration requires knowledge of dietary composition, growth rates, feeding rates etc., we are proposing that a set of "representative species" be used to define exposure potential and the translation to media concentrations. Such species would be representative of the range of exposure potential likely to be encountered in the site(s) of concern, including "high end" exposure scenarios. Ideally, the representative species and associated exposure parameters (diet, body weight, food consumption rates, etc.) would be defined on a site or regional basis. In situations where this is not possible, the Subcommittee envisions that a "default" set of nationally representative species and parameter values would be developed. For aquatic life, these species would reflect a range of feeding guilds (e.g., carnivory, piscivory, omnivory, herbivory), habitat preferences (e.g., benthic, pelagic), and taxonomic groups within each assemblage. Similarly for wildlife, a set of representative species would reflect a range of feeding guilds, taxonomic groups, and habitat types across the United States.

2) **Define Bioaccumulation for Representative Species.** Once the representative species have been defined and exposure parameters characterized (either on a site or national basis), the next step is to define the bioaccumulation potential for the chemical in the context of each representative species. Since chemical bioaccumulation in aquatic organisms can vary on across sites, bioaccumulation would ideally be characterized using site-specific information. For nonionic organic chemicals, some key factors include disequilibrium between chemical concentrations in sediment and water, lipid content, dissolved and particulate organic carbon, food web structure, trophic position, metabolism, and hydrophobicity. The Subcommittee envisions using a combination of empirical (e.g., field-derived bioaccumulation factors, biota-sediment accumulation factors) and mechanistic models (e.g., food web bioaccumulation models) for assessing a chemical's bioaccumulation potential. For situations where a site-specific assessment of bioaccumulation potential is not possible, the Subcommittee is considering the need to derive a set of nationally representative bioaccumulation factors (BAFs) that could be used to characterize bioaccumulation potential. This appears to be most applicable to organic chemicals where factors such as lipid fraction and dissolved and particulate organic carbon that can be readily adjusted to reflect local or regional conditions. This

approach is consistent with EPA's bioaccumulation assessment guidance developed for deriving human health water quality criteria (USEPA, 2000, 2003).

3) **Translation to Media Concentrations.** For aquatic life, translation of the tissue criterion to corresponding water concentrations would be accomplished by dividing the tissue criterion by the appropriate BAF derived for each representative species. An analogous approach could be constructed for translating to sediment concentrations. For wildlife criteria derived from dietary toxicity data, BAFs would be applied and appropriately weighted for each component of the aquatic diet of the representative wildlife species.

2.5 Challenges to Deriving Tissue-based Criteria for Bioaccumulative Chemicals

Basic toxicological principles suggest that measurements of exposure closer the site(s) of toxic action (e.g., tissue or body residues) is preferred over measurements in external media (water). In practice, however, a number of factors can act to mitigate the conceptual advantages of tissue-based criteria over water-based criteria. Some of these include:

1. The scope and quantity of applicable toxicological measurements based on tissue concentrations appears far more limited compared to water-based measurements. Given that aquatic life criteria are intended to protect entire aquatic communities from harmful exposures, a reduction in the number of species from which to estimate such criteria generally translates into greater uncertainty associated with the criterion.

2. Related to #1 above, the applicability of existing tissue-based toxicological measurements for criteria derivation appears to vary substantially. A sizable portion of the tissue-based toxicity data compiled to date reflects measurements of chemical concentrations in multiple types of tissues (even within the same study) in combination with a given toxicological response. Notably, the mere measurement of a chemical concentration in tissue(s) in tandem with a toxicological effect does not solely constitute a valid toxicological linkage between a given tissue concentration and an associated effect. Of critical importance for making toxicological inferences is establishing a valid tissue concentration-response relationship for appropriate tissues in conjunction with an understanding of the mode(s) and site(s) of action.

3. Ambiguity in tissue concentration-response relationships can also result from incomplete knowledge of the bioavailable form(s) of chemicals in tissue (particularly problematic with metals; Rainbow 2002), the effect of exposure route on the potency of a given tissue concentration, and even duration of exposure (e.g., Landrum et al., 2004).

3 Proposed Process for Deriving Aquatic Life Criteria for "Bioaccumulative" Chemicals

3.1 Importance of Problem Formulation

As described in the SAB Consultation "Overview Document," problem formulation is the initial step in a risk assessment where information about the chemical stressor, its exposure potential, and its effect on the ecological receptors of concern is evaluated for defining the scope of the assessment and for ensuring that the risk management goals are met. We believe most, if not all, elements of problem formulation are relevant to the derivation of aquatic life criteria. It is in the problem formulation step where the decision to apply a tissue-based approach is made. Assessment questions are formulated and addressed, important data gaps are identified, and a conceptual model is developed. Importantly, a plan is devised for analyzing the data and formulating the criterion that makes best use of the available information. This analysis plan is particularly relevant to tissue-based aquatic life criteria since a flexible approach is being proposed for deriving criteria depending on the availability of data and assessment needs (Figure 1). The summary below presents the current thinking of the Subcommittee regarding several important issues related to deriving tissue-based aquatic life criteria for bioaccumulative chemicals.

3.1.1 Deciding Between a Tissue or Water-based Approach

The Tissue-based Criteria Subcommittee is focusing on developing criteria for chemicals for which water concentration is not a reasonable surrogate for target tissue toxicant concentration expected under natural exposure conditions. In other words, the Subcommittee is focused on chemicals for which water concentration does not adequately capture exposure and subsequent toxicological effects expected in the natural environment. Such chemicals generally bioaccumulate extensively in aquatic food webs such that trophic transfer and subsequent dietary exposure become toxicologically important. Organic chemicals in this category generally have high hydrophobicity (e.g., log $K_{ow} > 5$), long environmental persistence, and are poorly metabolized by biota. A few obvious examples include polychlorinated dioxins, furans, and biphenyls, DDT & metabolites, and dieldrin. Selected organometallics and metalloids also fall into this category (e.g., methylmercury, selenium).

In many cases the decision to pursue a tissue-based approach will be obvious from the onset. All relevant information on the toxicological importance of dietary exposure will be considered. For some chemicals, however, the Subcommittee expects this decision to be ambiguous (e.g., perhaps for some organic chemicals with log K_{ow} values in the 4-5 range). In such cases, both a water and tissue-based approach may be pursued with a final decision being based on the relative uncertainty among the two approaches. Furthermore, the Subcommittee notes that the relative importance of dietary exposure can vary widely across species for a given chemical. Some groups of organisms with high food intake rates and high chemical assimilation efficiencies (e.g., high volume filter feeders) may be especially prone to chemical exposure via the diet. If such organisms are among the most toxicologically sensitive to the chemical in question, then they may be particularly relevant in the decision to use a tissue-based approach. In addition to direct evidence of the relative toxicological importance of dietary exposure,

indirect evidence via bioaccumulation modeling involving multiple exposure pathways may be considered. The Subcommittee emphasizes that the toxicological importance of dietary exposure is key (not just the existence of dietary exposure), as some organisms may be highly exposed via the diet but have evolved storage and detoxification mechanisms that can render the toxicological importance of accumulated chemical concentrations in tissue as being minimal or ambiguous (e.g., selected marine invertebrates; Rainbow, 2002).

3.1.2 Addressing Key Assessment Questions

The problem formulation step is where assessment questions are formulated and addressed. Examples of assessment questions are provided in the SAB Consultation "Overview Document." Highlighted below are several assessment questions and issues the Subcommittee has discussed to date in the context of deriving tissue-based criteria for bioaccumulative chemicals.

Understanding Mode(s) of Action. Understanding the mode(s) of action is important for a number of reasons. First, information on mode of action can aid in distinguishing among taxonomic groups in terms of their expected sensitivity, particularly when combined with information on key physiological attributes (e.g., presences/activity of AhR receptors for exposure to dioxin-like compounds). Knowledge on mode of action can also be important for interpreting tissue concentration-based toxicity data. Specifically, the relative reversibility/irreversibility of the mode of action may aid in understanding the importance of exposure duration in affecting the potency of chemical concentrations in tissue. For example, there is evidence in the literature that some specific modes of action may involve irreversible (or less than reversible) binding to toxicological receptors such that the potency of a given tissue concentration increases with increasing exposure time (Lee et al., 2002a; 2002b; Landrum et al., 2004; 2005). This in turn may affect how one chooses to aggregate tissue-based toxicity data or conduct toxicity data extrapolations. Mode of action information is also important in the decision to derive criteria on the basis of chemical mixtures. The Subcommittee notes that there may be ambiguity in identifying the critical mode(s) of action or limitations to making inferences based on mode of action data because: (1) most mode of action data for aquatic organisms have been gathered from acute toxicity tests involving fish, (2) mode of action might vary across species, life stages and with the magnitude and duration of exposure, (3) multiple or unknown modes of action may be involved with the expression of toxicological effects.

Understanding Potency of Tissue Concentrations Derived from Different Exposure Routes. A critical issue for interpreting tissue concentration-based toxicity data is how one addresses the potency of chemical concentrations in tissues that are derived from different exposure routes (e.g., water vs. food). One of the most attractive features of a tissue-based approach is the notion that toxicity from different exposure routes can be integrated by a chemical concentration measured in an appropriate tissue. If this were not the case, then the utility of tissue concentration-based toxicity data would be significantly compromised due to the highly heterogeneous nature of toxicity test designs (e.g., exposures from water, sediment, food, injection). For organic chemicals that obviously fall into the

"bioaccumulative" category, the Subcommittee is not aware of evidence that tissue concentration-based toxicity values routinely vary by exposure route. For metals, there is evidence that the route of exposure can affect the potency of a given concentration in tissue. However, most metals would not be considered in the context of criteria for bioaccumulative chemicals. The Subcommittee invites SAB comment on the importance of exposure route in affecting toxicity expressed as concentrations in tissue, particularly with regard to organic chemicals.

Understanding the Importance of Temporal Variability in Exposure Concentrations. As described in the SAB consultation document on Water-based Criteria, modeling toxicity as a function of short-term (daily) fluctuations in water concentrations is a fundamental component of the proposed water-based criteria methodology. For bioaccumulative chemicals (e.g., persistent organic chemicals w/ log K_{ow} >5), the current thinking of the Subcommittee is that such short-term fluctuations will generally be much less important in affecting chemical uptake and tissue concentration-based toxicity. The basis for this thinking originates in the notion that for most aquatic species of concern (e.g., especially larger bodied animals at higher trophic levels such as piscivorous fish), accumulation kinetics of "bioaccumulative" chemicals is sufficiently slow such that risks from short-term (acute) exposures are generally not nearly as important relative to risks from long-term exposures. An illustration of this phenomenon is shown in Figure 2 using tissue concentrations predicted by the Gobas (1993) food web bioaccumulation model. It can be seen in Figure 2 that concentrations of highly hydrophobic chemicals (e.g., log K_{ow} > 5) in piscivorous fish are dampened temporally compared to concentrations in water. As a result, the Subcommittee expects that tissue-based aquatic life criteria for bioaccumulative chemicals will be concerned with chronic exposures and conditions approximating steady state. However, the Subcommittee recognizes that exceptions to this generalization might occur, possibly for small-bodied organisms lower in the food web where accumulation kinetics might be relatively rapid (e.g., zooplankton). If such organisms are among the most sensitive species to the chemical in question, then steps to address risks associated with short-term exposures will need to be taken. This might involve using dynamic bioaccumulation modeling for translating critical tissue residues back to media concentrations and/or the use of shorter-term averaging periods per the 1985 Guidelines methodology.

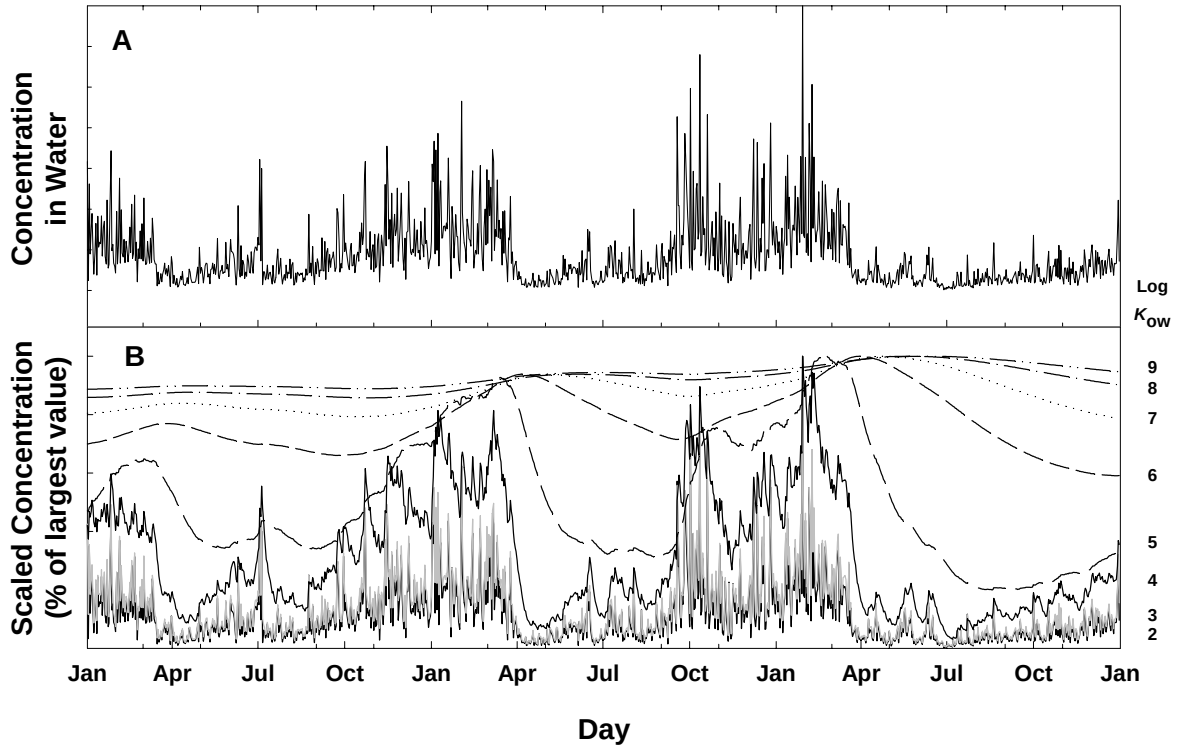


Figure 2 (A). Daily concentrations of a hypothetical nonionic organic chemical over time in the water column, predicted using a simple dilution model and daily flow data for the Mississippi River at St. Paul, Minnesota. **(B)** Daily chemical concentrations in piscivorous fish found using the kinetic food web models of Gobas (1993) with the daily chemical concentrations in the water column for nonionic organic chemicals with log *n*-octanol-water partition coefficients (log K_{ow}s) of 2, 3, ... and 9. The daily chemical concentrations in piscivorous fish have been scaled to the largest value for each log K_{ow}. As hydrophobicity increases, temporal variability in chemical concentrations in piscivorous fish decreases dramatically. From Burkhard (2003)

3.1.3 Screening and Evaluation of Toxicity Data

An important step in the derivation of tissue-based aquatic life criteria for bioaccumulative chemicals involves the screening and evaluation of toxicity data. All toxicity data will be first reviewed for acceptability based on quality prior to their use in deriving a criterion. Most of the evaluation criteria used to determine acceptable toxicity test quality will be the same or similar to those used in the derivation of water-based aquatic life criteria. A few examples include:

- Sufficient written documentation must be available from which to judge the quality of the methods, measurements, and statistical analyses conducted. Peer reviewed publications are preferred.
- Laboratory studies must contain a control treatment with an acceptable response rate for control organisms (often specific to the test design).
- Test organism handling, holding, acclimation, and loading rates should conform to standard practices appropriate to the test design (e.g., ASTM or other similar peer reviewed guidelines).

- Water quality parameters (e.g., pH, temperature, D.O., etc.) and their rates of change must be within accepted ranges for test design and/or the environmental tolerances of the test organisms.

In addition to data quality considerations, the Subcommittee recognizes that a number of other attributes of tissue concentration-based toxicity data will likely need to be evaluated for determining their acceptability and/or utility for deriving aquatic life criteria. Some of these attributes are listed below.

- **Study Duration.** The primary focus for characterizing effects of "bioaccumulative" chemicals will be those toxicity data that are indicative of effects resulting from long-term (chronic) exposure. This focus is based in part on the notion that risks will likely be driven from chronic exposure not from acute exposures. Furthermore, mode of action may vary as a function of magnitude and duration of exposure. We note, however, that certain tissue concentration-based toxicity data derived from short-term exposures are appropriate. For example, short-term exposure to critical life stages (e.g., egg, embryo) form the basis of estimating risks of 2,3,7,8-TCDD to aquatic life (Cook et al., 1993). The Subcommittee recognizes that toxicity data expressed on the basis of tissue concentrations may have the capacity to integrate differences in the magnitude and duration of chemical exposure, particularly for non-specific, reversible modes of action (e.g., narcosis). However, for some specific modes of action that involve irreversible or partially irreversible binding to receptors involved in expressing toxicity, both theoretical and empirical evidence suggest that exposure duration can contribute to variance in tissue-based toxicological effect levels. For example, exposure duration has been suggested as a source of variability observed in the potency of PAH concentrations in tissue (Lee et al., 2002a; 2002b). Duration of exposure may be particularly important for chemicals which are slow to reach steady state with respect to their distribution in organism tissues. Therefore, the effect of exposure duration on the potency of a particular tissue concentration is an attribute that would receive specific evaluation for characterizing effects using tissue-based toxicity data.

- **Toxicological Endpoints.** Similar to the 1985 Guidelines, studies will be screened for those that measure effects based on toxicological endpoints that are thought to have most relevance to potential impacts on populations (e.g., survival, growth, reproduction, development). Other endpoints would be used provided that a sufficient ecological and toxicological linkage can be made to impacts on populations.

- **Strength of Tissue Concentration-Response Relationship.** Analogous to the evaluation of water-based toxicity data, the strength of the tissue concentration-response relationship is an important criterion for evaluating the acceptability of tissue-based expressions of toxicity. This point cannot be overemphasized for evaluating tissue-based toxicity data, since often times chemical concentrations are analyzed and reported for multiple tissues along with a common set of adverse effects. Chemical concentrations in some tissues may have little or no correlation with toxicological effects. However, this lack of correlation may be reflect confounding factors such as the use of too few

treatments or treatment levels that are beyond the range of the concentration-response curve.

In order to be used directly in the derivation of a tissue-based criterion, a "valid" tissue concentration-response relationship must be available from the toxicity test in question. Validity of the tissue concentration-response relationship will likely be judged quantitatively based on its statistical and toxicological significance and qualitatively based on consistency of increasing response with increasing tissue concentrations. Tissue concentration-response relationships that can be expressed quantitatively (e.g., via regression equations) are generally preferred over those that can only be expressed qualitatively (i.e., increasing response with increasing tissue concentration). Tissue concentration-effects data for which a concentration-response relationship is not observed may have some utility for characterizing effects (e.g., in the case of unbounded NOAELs, indicating levels where effects have not occurred).

- **Tissue Type.** The choice of tissue(s) used to relate chemical concentrations to toxicological effects is an important attribute to consider when developing tissue-based criteria. Other factors aside, preference will generally be given to tissues that either represent or are closely linked to the site(s) of toxic action. However, the choice of tissue(s) upon which to base the effects characterization will have to balance proximity to the site(s) of action with the availability of data for that tissue and the ability to extrapolate chemical concentrations between tissues. For example, cursory examination of the tissue concentration-based toxicity data indicates that the preponderance of data exists in the form of whole body concentrations (Appendix A). Thus, it appears that whole body concentrations will need to be used as surrogates for concentrations at the site(s) of toxic action and/or integrated with models for estimating concentrations in specific tissues.

3.1.4 Minimum Data Requirements and Assemblages

The current view of the Subcommittee is that tissue concentration-based toxicity data will need to be evaluated against a set of "minimum data requirements" (MDRs) before a criterion could be derived. This concept is consistent with the 1985 Guidelines and the new proposal for deriving water-based criteria. Minimum data requirements are a defined set of taxonomic or ecologically-based species groups from which acceptable toxicological data must be available in order to derive a criterion. The Subcommittee believes some set of MDRs are needed in order to preserve a minimum level of reliability in tissue-based aquatic life criteria.

The Subcommittee has discussed the issue of MDRs but has not reached final consensus on an exact set of MDRs to propose. However, current thinking is that MDRs would be defined separately for three assemblages of aquatic organisms:

(1) vertebrates,
(2) invertebrates
(3) plants

These three assemblages are consistent with the current proposal for deriving water-based aquatic life criteria (see SAB Consultation Document on Water-based Criteria). Defining MDRs separately for different assemblages might allow for some flexibility when deriving criteria. For example, criteria might be derived only for those assemblages for which sufficient data are available. Although the exact composition of MDRs has not been specified, current thinking is that they would consider taxonomic diversity in addition to factors related to a species' "ecological niche" as defined by trophic status/feeding guild, habitat preference, life history, etc. Thus, MDRs could be defined as requiring data for a top predatory (piscivorous) fish, a benthic feeding carnivorous fish, an herbivorous fish, etc. Life history attributes such as generation time (an important influence on population recovery time) may also be considered so that both short-lived and long-lived species would be represented. The Subcommittee also recognizes practical constraints to defining MDRs. If MDRs are too onerous, few if any criteria could be derived in the near future. Based on a cursory review of the availability of tissue-based toxicity data for aquatic organisms, it appears that MDRs for deriving deterministically-based criteria would approximate 4-5 species per vertebrate and invertebrate assemblage. The Subcommittee has not discussed MDRs for plants.

3.1.5 Deciding Between a Deterministic or Probabilistic-based Effects Characterization

The MDRs discussed above would presumably apply to deriving criteria using a deterministic-based approach (i.e., that approach requiring the least amount of toxicity data). However, if sufficient data were available, probabilistic-based methods for characterizing effects would be considered (e.g., SSD, the TEA model described in the SAB Consultation Document on Water-based Criteria). In cases where the advantages of a probabilistic approach over a deterministic approach are not obvious, the Subcommittee can envision that criteria would be derived using both general approaches, with the approach that achieves the management goals with the least uncertainty becoming the preferred method. The Subcommittee has not discussed the quality or quantity of data required to apply a probabilistic-based approach such as a SSD but will address this issue in the future.

3.2 Characterization of Effects: Deterministic Criteria

The focus of the effects characterization is quantifying relationship between accumulated chemical concentrations in tissues and toxicological effects across multiple species in order to support the selection of a chemical concentration in tissue that would adequately protect a given assemblage. In the ideal situation, an abundance of toxicological data would be available from which quantitative relationships between tissue concentrations and adverse effects could be established. In the aggregate, the underlying toxicological database should ideally represent:

(1) a diverse array of aquatic species (e.g., multiple families of fish, invertebrates and perhaps plants) in order to capture variability in sensitivity among species within each assemblage,
(2) a diverse array of toxicological endpoints that can be closely linked to population-level effects (e.g., reproduction, mortality, growth, development to name a few),
(3) chemical measurements in tissues that represent, or are closely linked to, the site(s) of toxic action, and

(4) toxicity tests conducted under standardized protocols with regard to routes of exposure, duration, life stages tested, etc.

In reality, the situation appears to be far from this ideal. Cursory examination of two compilations of tissue-effects data (Jarvenin and Ankley, 1999; USACE, 2004) reveals that the majority of chemicals have relatively few species represented, a strong dominance of lethal endpoints over sublethal endpoints, a variety of routes of exposure, and most measurements in whole body vs. specific tissues (see Appendix A). Perhaps the greatest obstacle facing the successful derivation of tissue-based aquatic life criteria for bioaccumulative chemicals is the relative lack of appropriate, standardized, tissue-based toxicological data.

The following sections provide some insight into the thinking of the Subcommittee on how tissue concentration-based toxicity data would be synthesized for supporting a deterministically-based criterion.

3.2.1 Characterizing Effects on Organisms

The overall goal in this step is to define concentrations in tissue(s) below which unacceptable adverse effects on the test organisms are not likely to occur. Some of the Subcommittee's general preferences for synthesizing toxicity data among studies within a species are provided below.

- In general, determination of tissue-based effect concentrations using point estimation methods (e.g., ECxx based on regression analysis) is preferred over those determined by hypothesis testing (e.g., ANOVA-based NOAELs and LOAELs). All else being equal, point estimation methods enable interpolation between treatment levels to obtain a more precise estimate of the magnitude of effect compared to hypothesis testing methods.
- Studies with treatments (or observations in the case of field data) that bracket the onset of unacceptable adverse effects are preferred over those studies where either: (1) all treatments showed unacceptable adverse effects, or (2) no treatments showed unacceptable adverse effects.
- Defining what constitutes an unacceptable adverse effect (i.e., the magnitude of effect or EC_{xx}) will likely depend on the toxicological endpoint measured. Results of population modeling could conceivably help inform the selection of an appropriate EC_{xx} (see Section 3.2.3 and the population modeling discussion in the SAB Consultation Document on Water-based Criteria).
- Studies using a chronic exposure duration involving multiple life stages (or exposure to early or other critical life stages) are generally preferred over those of shorter exposure duration involving single life stages.
- If two or more acceptable tissue-based effect concentrations are available for a given species, life stage, and endpoint (e.g., mortality), the study that is considered to be of the highest quality and containing the least uncertainty in quantifying the threshold for unacceptable effects would be selected. Likely factors to consider in this evaluation include:
    a) environmental realism of exposure regime
    b) statistical power of the study

c) statistical uncertainty associated with the tissue-based effect concentrations
d) repeatability of the test results
e) accuracy and precision of the biological and chemical measurements
f) uncertainty associated with extrapolating results to the field.

- If no discernable difference exists between the quality and uncertainty associated with two or more studies involving the same species, life stage and endpoint, current thinking is that tissue-based effect concentrations would be averaged. This would help minimize the impact of inter-test variability on selecting a representative tissue-based effect concentration for a given species.

If two or more acceptable tissue-based effect concentrations are available for the same species and endpoint but for different life stages, preference would be given to the values from more sensitive life stage(s) for characterizing effects on that species (unless data are being used in population modeling where data for multiple life stages are preferred).

3.2.2 Toxicity Data Extrapolations

The Subcommittee expects that limitations in the scope and quantity of tissue-based toxicity data will require that various extrapolations be made in order to derive aquatic life criteria that can achieve an adequate level of protection. The Subcommittee has identified various types of toxicity data extrapolations that may be needed (below) but has had very little discussion to date on how to conduct such extrapolations. The Subcommittee invites SAB comment on the need and methods for conducting toxicity data extrapolations on a tissue concentration basis.

- **Extrapolating Across Magnitudes of Effect.** The Subcommittee envisions a potential need for extrapolating from higher magnitudes of effect to lower magnitudes of effect (e.g., LOAEL to NOAEL, EC_{50} to EC_{10}) in cases where tissue concentrations corresponding to lower magnitudes of effect are not quantified or are not reported for a given endpoint (e.g., mortality). Statistical modeling may be used in cases where the tissue concentration-response relationship has been adequately defined. In cases where the tissue concentration-response relationship has not been adequately defined, traditional approaches for human health and wildlife criteria have used uncertainty factors (UF). Methods for developing or selecting UFs have not been discussed by the Subcommittee.

- **Extrapolating Across Exposure Duration.** In cases where there is sufficient evidence to indicate the potency of a given chemical concentration in tissue is influenced by exposure duration, it is conceivable that some type of extrapolation may be needed to relate observed effects from shorter exposure durations to those expected from longer (chronic) exposure durations. There is some evidence of time-dependent toxicity of tissue concentrations in the literature for certain compounds (Lee et al., 2002a; 2002b; Landrum et al., 2004; 2005). However, these studies involve relatively short exposure durations (10 days or less), and their applicability to longer-term chronic and subchronic exposures (which is the general focus for bioaccumulative chemicals) is not clear. If chronic and subchronic tissue-based toxicity data are subject to time-dependency, the use of tissue concentration-based toxicokinetic modeling may be required.

- **Extrapolating Between Tissues.** The Subcommittee expects a need to extrapolate between tissues for expressing tissue concentrations associated with adverse effects (for example, from concentrations in whole body to concentrations in specific tissues). This may be required to place available tissue-based toxicity data on a common basis. For highly hydrophobic organic chemicals, current thinking is that information on lipid content of different tissues may be used for extrapolating tissue-based effect levels between tissues. In other situations, use of empirical relationships may be required for relating chemical concentrations between tissues.

- **Extrapolating Between Species.** Assuming that deterministic-based criteria could be derived with as few as 4-5 species within an assemblage, the current thinking of the Subcommittee is that some type of interspecies extrapolation of toxicity may be needed to account for untested species of an assemblage that may be substantially more sensitive than the most sensitive species tested. The assumption here is that we would likely be addressing specific modes of action where species sensitivity can differ substantially on a tissue concentration basis as opposed to nonspecific modes of action (narcosis) where effects may be more narrowly distributed on a tissue concentration basis. In the context of a cumulative frequency distribution, the most sensitive species among a dataset containing four species approximates the 25^{th} percentile, a level substantially larger than traditional aquatic life criteria which are set at the 5^{th} percentile. However, because aquatic life criteria that are derived using the 1985 Guidelines combine data from the aquatic invertebrate and vertebrate assemblages into a single SSD for as few as 8 species, this comparison is not entirely straightforward. The Subcommittee notes that methods for extrapolating toxicity between species have been derived from toxicity data expressed as concentrations in exposure media (e.g., ICE, Asfaw et al., 2003). However, we are not aware of methods for interspecies extrapolation of toxicity on a tissue-concentration basis. At this point in time, the Subcommittee has not discussed how such extrapolations would be conducted with tissue concentration-based toxicity data and solicits SAB comments on the issue.

3.2.3 Characterizing Effects on Populations

The Subcommittee on Tissue-based Criteria has not had detailed discussions on characterizing the effects of bioaccumulative chemicals at the population level for aquatic organisms, deferring instead to the expertise and work in this area being conducted by the Water-based Criteria Subcommittee. Conceptually, population models being considered for deriving water-based criteria should be applicable to tissue-based toxicity data available for bioaccumulative chemicals. For example, Munns et al. (1997) used a stage-specific, density independent model to estimate the effects of dioxin and PCB tissue concentrations on the intrinsic rate of population growth for the mumichog, *Fundulus heteroclitus.*

In practice, however, the feasibility and utility of population modeling appears ambiguous to this Subcommittee in the context of tissue-based criteria for bioaccumulative chemicals. Part of this ambiguity relates to the apparent limited availability of tissue concentration-based toxicity data for multiple life stages within a species. Lacking data to

characterize the differential sensitivity of different life stages would appear to significantly limit the ability to parameterize stage-specific population models. Furthermore, the availability of tissue-based toxicity data for reproductive endpoints appears to be extremely limited, based on a review of two databases containing tissue concentration (residue)-based toxicity information. Finally, the utility of population modeling in the context of constant (time invariant) exposure concentrations is also questionable to the Subcommittee. Part of the rationale for using population models for water-based criteria is to characterize effects resulting from fluctuating exposure concentrations and to integrate recovery time. If toxicity modeling for bioaccumulative chemicals is generally limited to constant concentrations in tissue (steady-state conditions) as discussed earlier in this proposal, the "value added" of population modeling appears, at least at this point, to be unclear. The Subcommittee plans to conduct additional analyses to clarify the role and utility of population modeling for setting aquatic life criteria for bioaccumulative chemicals.

3.2.4 Setting a Deterministically-based Tissue Criterion

The goal of a national tissue criterion for aquatic life would be to represent a concentration in tissue that at or below which the likelihood of unacceptable adverse effects on aquatic life would be appropriately low (i.e., as determined by risk management goals). Where multiple criteria are derived for different assemblages (e.g., invertebrates, vertebrates, plants), current thinking is that criteria for the most sensitive assemblage would apply due to the interdependence among assemblages in maintaining healthy ecosystems. Within an assemblage, current thinking is that the tissue criterion would be derived from data for a species that enables the protection goals to be met with the least uncertainty. Generally, this will be the most sensitive species. However, exceptions may exist in cases where uncertainty associated with basing the tissue criterion on the data for the most sensitive species is considered substantially higher than basing the criterion on data from a less sensitive species (e.g., the next most sensitive species). As discussed in Section 3.2.2, some type of toxicity data extrapolation may be needed to address concerns over the potential for greater sensitivity of untested species. The technical basis for conducting this extrapolation or evaluating uncertainty has not been discussed by the Subcommittee.

3.3 Characterizing Effects: Probabilistic-based Criteria

One option being explored by the Subcommittee for characterizing effects on a probabilistic basis involves the use of Species Sensitivity Distributions (SSD). Characterizing effects on the basis of SSDs forms the foundation of the 1985 Guidelines. One distinct advantage of an SSD approach over the deterministic approach described above is that the criterion can be selected to conform to a specified "risk level" or percentile (e.g., setting at a 5^{th} percentile to theoretically protect 95% of the tested species, per the 1985 Guidelines) via interpolation or extrapolation using statistical techniques. Aside from enabling consistency in the "risk level" selected across chemicals with heterogeneous datasets, the use of statistically-based interpolation or extrapolation techniques with the SSD approach enables one to mitigate

the influence of potential "outliers" on the derivation of the criterion, at least when compared to selecting the most sensitive species as the basis of the criterion.

A significant obstacle to the use of SSDs for tissue-based criteria is that they require data for a relatively large number of species in order to characterize species sensitivity with statistical rigor. To be statistically valid, SSDs should ideally be composed of data for the same or similar toxicological endpoints. If the underlying toxicity test data lack consistency in test design and endpoints measured, the SSD derived from such data would not only reflect true sensitivity differences but also differences related to test design. The Subcommittee has not discussed which specific SSD models it would recommend for use nor criteria for judging when to apply an SSD for deriving a tissue criterion.

The Subcommittee has also not discussed in detail the feasibility of applying the Toxic Effect Aggregation model (TEA) for characterizing effects, which is described in the SAB Consultation Document on Water-based Criteria but plans to do so in the future as details with the TEA model are resolved.

3.4 Setting a Probabilistically-based Criterion

Assuming that a valid SSD could be constructed using tissue-based toxicity data, then a criterion value could be selected to correspond to any desired level of 'risk' (i.e., any percentile of the SSD). For example, the 1985 Guidelines set criteria to correspond to the 5th percentile of the SSD. To date, we have not discussed a specific percentile or range of percentiles at which to set the national tissue criterion. However, in order to facilitate different risk management options to be considered, the Subcommittee is promoting transparency and flexibility in the selection of the percentile(s) for setting a national tissue criterion rather than setting it at a single percentile specified *a priori*.

3.5 Translating Tissue Criteria to Concentrations in Water, Food Web

As discussed in Section 2, the Tissue-based Criteria Subcommittee anticipates the need to develop guidelines for translating tissue-based aquatic life criteria into corresponding concentrations in environmental media (e.g., water) and relevant components of the aquatic food web. Translating tissue-based criteria into concentrations in ambient environmental media is often required for implementing criteria through regulatory programs. Translating tissue-based criteria into corresponding concentrations in components of the aquatic food web may also be required to facilitate monitoring of tissue concentrations (e.g., monitoring chemical concentrations in the diet of fish may be more practical than direct monitoring of fish tissue in some cases). This section presents the thinking of the Subcommittee regarding how national tissue-based criteria for bioaccumulative chemicals might be translated to other compartments of the aquatic ecosystem.

Ideally, this translation would be conducted using data specific to the site(s) of concern because many attributes can affect bioaccumulation of chemicals on a site-specific basis (e.g.,

food web structure, organic carbon concentration, chemical disequilibrium between sediments and water, etc.). Therefore, consistent with other EPA guidelines on estimating chemical bioaccumulation (USEPA 2000; 2003), use of site-specific data for translating tissue criteria to media concentrations would be strongly encouraged. However, past experience indicates that site-specific data (and/or the resources to obtain such data) may not be available in some circumstances. Therefore, the Subcommittee is considering the possibility that a default set of conditions may have to be defined for translating tissue criteria into media concentrations for use in circumstances where site-specific data are unavailable. This approach is consistent with past EPA guidance on bioaccumulation.

3.5.1 Use of Representative Species

Translating a tissue-based aquatic life criterion to media concentrations would initially appear to be a straight-forward task that would involve the use of bioaccumulation models. However, the following two issues arose during the Subcommittee's discussion of this translation step.

- **Ambiguity in Species Identity Associated with a Tissue Criterion.** If the tissue criterion were derived with the use of extrapolation or interpolation techniques (e.g., uncertainty factors for deterministic criteria; at a specified SSD percentile for probabilistic criteria), the identity of the species that would correspond to the tissue criterion would not be obvious. Since bioaccumulation models require that components of the food web to be described (e.g., dietary composition, lipid fraction, growth rate, etc.), the parameterization of bioaccumulation models (or choice of bioaccumulation factors) would be ambiguous in such cases. Although tissue criteria for aquatic life would be derived within specified assemblages (e.g., vertebrates, invertebrates), the dietary habits and chemical exposure potential of species within these assemblages can vary widely (e.g., from herbivory to piscivory). This variation in chemical exposure potential would appear to introduce considerable uncertainty in the translation of a tissue criterion to concentrations in ambient media or the aquatic food web.

- **Relationship Between Chemical Sensitivity and Risk.** A second issue that surfaced relates to potential for discontinuity between a species exposure potential (as defined by dietary composition, chemical uptake rates, etc.) and its inherent sensitivity to the chemical as defined by tissue concentration-effect values. This is perhaps best considered in the context of a SSD composed of tissue-based toxicity data. Although this SSD represents the sensitivity differences among species based on intrinsic (internal) toxicity, this distribution does not necessarily correspond to the distribution of exposure potential (and risk) experienced by these species in response to a given exposure regime. Therefore, the relative differences in "risk" to a set of species could differ considerably from their relative differences in sensitivity as defined by tissue concentrations. In other words, the most sensitive species on a tissue concentration basis may not be the species at greatest risk due to variation in exposure potential among species.

To address these two issues, the Subcommittee is considering the use of "representative species" for translating a tissue criterion to corresponding concentrations in ambient media and components of the aquatic food web. A set of representative species could be selected for each aquatic life assemblage that would span a range of factors related to chemical exposure potential (e.g., different feeding guilds/trophic position). For translating national tissue criteria at a national scale, a set of representative species could be defined *a priori*. For translations at a regional or site-specific scale, the representative species could be defined using information specific to the region or site. Using region or local information to define the representative species may be particularly useful, for example, if certain feeding guilds of fish (e.g., large piscivores) are not found at a particular location. In considering the use of representative species, the Subcommittee notes the following feature that make this option attractive.

1. **Representative Species Can Readily be Defined.** Data related to defining chemical exposure potential (e.g., diet, growth rate, lipid content) are expected to be much more plentiful than tissue-based toxicity data. In some cases, available toxicity data may not encompass species that are among the highest exposed. Thus, the translation of tissue criteria to media (or food web) concentrations would be done on a consistent basis even when the composition of the toxicological data sets varied.

2. **Addresses Discontinuity Between Risk and Intrinsic Toxicity.** Representative species would be defined according to a range of exposure pathways, feeding guilds, and habitat preferences. This would enable one to address variation in exposure potential (and risk) that can occur as a function of food web composition, chemical properties, and chemical distribution (e.g., disequilibrium) between water and sediment.

3. **Maintains Consistency Between Criteria Methods.** The same set of representative species could be used for both the deterministic and probabilistic-based tissue criteria. This would maintain consistency among the criteria derivation methods and lead to prediction of media concentrations that would be less dependent on the nuances of the tissue concentration-effects dataset.

4. **Facilitates Translation to Concentrations in Food Web.** By using representative species, one could also translate the national tissue criterion into concentrations in the diet of the representative species (e.g., benthic macroinvertebrates for fish). This might facilitate easier monitoring on the basis of tissue concentrations.

5. **Amenable to Adjustment by Site or Region-Specific Attributes.** Representative species could be defined on a local or regional level which could help address site- or region-specific concerns regarding bioaccumulation potential.

It is worth noting that the approach above does not assume that the representative species are the species actually "at risk" near the tissue criterion. Rather, it assumes that species with intrinsic sensitivities at or near the tissue criterion (whose identity is unknown) could have a range of exposure potential as defined by the representative species.

3.5.2 Bioaccumulation

Once representative species have been defined for an assemblage, the next step in translating a tissue criterion to media concentrations would involve estimating bioaccumulation potential of the chemical in relation to the representative species. For estimating bioaccumulation potential, the Subcommittee proposes to use a framework similar that used by EPA to derive National Ambient Water Quality Criteria to protect human health (USEPA 2000; 2003). This methodological framework is based on the use of both empirical (e.g., bioaccumulation factors, biota-sediment accumulation factors) and mechanistically-based methods (e.g., food web bioaccumulation model; Gobas 1993) for characterizing chemical bioaccumulation in the aquatic diet of humans. Appropriate modifications of this methodology would need to be made to address the diet of representative aquatic life species, but the basic framework would still apply. Some of the salient features of this methodology include:

- Use of high quality measured data for characterizing bioaccumulation (e.g., BAFs, BSAFs) are generally preferred over modeled estimates in part because factors such as chemical metabolism by biota are addressed.
- A three-phase partitioning model is used to address the effect of dissolved and particulate organic carbon on the bioavailability of nonionic organic chemicals.
- Lipid normalization is used to address the effect of differences in lipid content that occur across different species. Accounting for chemical partitioning to organic carbon and lipids has been shown to reduce variability in BAFs measured for PCBs in the Fox River and Green Bay (Burkhard et al., 2003).
- BAFs are aggregated separately for organisms in different trophic levels in order to account for biomagnification and broad physiological differences that can affect bioaccumulation.
- A fugacity-based food web model (Gobas 1993) is used to estimate bioaccumulation in absence of measured data and when the effect of chemical metabolism is considered negligible or is not known.

Once bioaccumulation has been estimated for the representative species, translation to water would be accomplished by dividing the tissue criterion by the appropriate bioaccumulation factor for each representative species within each of the assemblages (e.g., aquatic vertebrates, invertebrates, plants).

$$\text{Water Criterion}_{i,j}\ (\text{mg/L}) = \frac{\text{Tissue Criterion}_{j}\ (\text{mg/kg})}{\text{BAF}_{i,j}\ (\text{L/kg})}$$

Where "i,j" = the "i^{th}" representative species for the "j^{th}" assemblage.

For each assemblage, conversion of a tissue criterion to corresponding concentrations in the aquatic food web (e.g., macroinvertebrates, zooplankton, algae) could be conducted using trophic transfer factors (TTFs) defined separately for each representative species.

$$\text{Concentration in Food Web Component}_{i,j,k}\ (\text{mg/kg}) = \frac{\text{Tissue Criterion}_{k}\ (\text{mg/kg})}{\text{TTF}_{i,j,k}\ (\text{unitless})}$$

Where "i,j,k" = the "i^{th}" food web component of the "j^{th}" representative species for the "k^{th}" assemblage.

According to this scheme, the end result would be a table of criterion values in environmental media (water, sediment) and applicable components of the aquatic food web (e.g., trophic levels 1, 2, 3, 4, etc.) that would vary according to each representative species defined for that assemblage. An example might look something like a table below, with actual chemical concentrations defined in each of the checked boxes.

	Aquatic Vertebrate Assemblage		
Translated Criterion Concentration	Representative Sp. A (piscivore)	Representative Sp. B (benthic carnivore)	Representative Sp. C (herbivore)
Water	✓	✓	✓
Sediment	✓	✓	✓
Algae/Macrophytes	✓	✓	✓
Zooplankton	✓	✓	
Macroinvertebrates	✓	✓	
Forage fish	✓		

The Subcommittee has not discussed <u>if (or how)</u> a final set of "default" criteria concentrations would be selected among the various possible values using the approach outlined above.

3.6 Thoughts on Site-Specific Criteria

Perhaps the most appropriate opportunity for adjusting tissue-based criteria to reflect site-specific differences would arise in their translation to media concentrations (summarized above). Representative species and bioaccumulation could be defined specifically for the site(s) of concern using site data. It is also conceivable that the specific composition of species used to derive the tissue criterion could be modified to more accurately reflect the occurrence of species at a particular site. Such a procedure (called the "recalculation procedure") currently exists for aquatic life criteria derived using the 1985 Guidelines. In using this approach, it would be important to demonstrate that data for any species that would be eliminated from the national tissue criterion database was <u>not</u> a reasonable surrogate for species occurring at the site. The Subcommittee plans to discuss methods for deriving site-specific aquatic life criteria in the near future.

4 Process for Deriving National Tissue-based Wildlife Criteria

4.1 Background on Development of Wildlife Criteria

Although aquatic-dependent wildlife may be protected by aquatic-life criteria, the procedures do not systematically incorporate information on the toxicological sensitivity or the unique exposure scenarios of wildlife species. In 1987, the Government Accounting Office (GAO) issued a report entitled "National Refuge Contamination is Difficult to Confirm and Clean Up" that documented the contaminant clean-up activities at the Kesterson National Wildlife Refuge and other refuges and the limited federal efforts to develop water quality criteria to protect wildlife and their habitats from the adverse effects of chemical contamination (USEPA 1989). The GAO report recognized that cleaning up contaminated sites is difficult when there is a lack of water quality criteria to determine when wildlife are threatened. EPA agreed to modify the criterion for selenium to include wildlife effects.

A workshop in 1988 entitled "Water Quality Criteria to Protect Wildlife Resources" co-chaired by EPA and USFWS focused on evaluating the need for wildlife criteria and developing a strategy for producing wildlife criteria (USEPA 1989). The recommendations from the workshop were that 1) the process for ambient water quality criteria should be modified to consider effects on aquatic-dependent wildlife and 2) chemicals should be prioritized based on their potential to adversely impact wildlife species. In 1989, a preliminary chemical screening was conducted to 1) evaluate whether existing water quality criteria would be protective of wildlife and 2) prioritize chemicals for their potential to adversely impact wildlife species. The approach for screening was derived from an approached developed by the State of Wisconsin for deriving criteria to protect wildlife and domestic animals, which was derived from non-cancer human health criteria. The screening approach considered toxicity and bioconcentration assuming oral ingestion via food and water consumption. The screening study identified the following classes of chemicals for which current water quality criteria may not be adequate to protect wildlife: chlorinated alkanes, chlorinated benzenes and chlorinated phenols, metals, dioxins, and DDT.

The EPA refined this approach in 1991 in an internal report developing interim wildlife criteria. The objective of this analysis was to assess the validity of the previous screening exercises and to evaluate the availability of high quality wildlife toxicity data for criteria development. The report identified chemicals where the interim wildlife criterion was lower than the aquatic life and human health criteria. It also acknowledged that generation of additional wildlife criteria will be difficult due to the lack of toxicity data.

The same approach to wildlife criteria development was being developed at the same time through collaboration with the Great Lakes Water Quality Initiative (GLWQI) for deriving criteria for protection of wildlife species in the Great Lakes. The basic approach used the following model for calculating a wildlife value expressed as the water concentration of a contaminant that, if not exceeded, should be protective of wildlife populations:

$$WV\ (mg/L) = \frac{TD\ (mg/kg\ bw/d) * (1/(UF_A * UF_S * UF_L)) * BW\ (kg)}{W\ (L/d) + \sum[FC_i\ (kg\ food/d) * BAF_i\ (L/kg)]}$$

where:

WV = wildlife value
TD = toxic daily dose
UF = uncertainty factors for interspecies variation (UF_A), subchronic to chronic (UF_S), and LOAEL to NOAEL (UF_L)
BW = body weight of species of concern
W = amount of daily water consumption
FC_i = amount of daily food consumption from the i^{th} trophic level
BAF_i = bioaccumulation factor for the i^{th} trophic level

The toxic daily dose (TD) was derived from an assessment of available toxicity data for a specific chemical. Historically, it was based on an endpoint from the study judged to represent the strongest scientific quality and highest relevance to the assessment. Typically, the TD is calculated using the no-observed-adverse-effect level (NOAEL) or, if necessary, the lowest-observed-adverse-effect level (LOAEL) converted to a daily dose (mg/kg body wt/day). Uncertainty factors are applied to address variation in species sensitivity to the chemical (i.e., UF_A) and deficiencies in study design (i.e., UF_S and UF_L).

Several GLWQI reports related to the process for developing wildlife criteria were published in 1995, including a report detailing the calculation of wildlife criteria for DDT, mercury, 2,3,7,8-TCDD, and PCBs (USEPA 1995c) and a technical support document that presented the rationale for the approach (USEPA 1995d). The wildlife value was calculated based on both drinking water and dietary routes of exposure and was expressed as the chemical concentration in water that would be protective of wildlife. Wildlife values were calculated for several bird and mammal species chosen to represent the Great Lakes aquatic-dependent wildlife, with the final wildlife value for each taxonomic class based on the geometric means of species-specific values.

While work on wildlife criteria for the Great Lakes was nearing completion, work continued on developing approaches for use in developing national wildlife criteria. The EPA Science Advisory Board (SAB) held a meeting in April of 1994 to review progress on development of a national wildlife criteria program. Their primary recommendations were that the program should 1) be guided by the agency's Ecological Risk Assessment Framework, 2) develop a national methodology that can be used to derive regional or site-specific wildlife criteria, 3) use case studies to validate models and methodologies, and 4) focus of protection of wildlife populations, as opposed to individuals (USEPA Science Advisory Board 1994).

The Mercury Study Report to Congress (USEPA 1997) used the GLWQI approach, with a few minor modifications, to develop a national wildlife value for methylmercury in water protective of birds and mammals. The modifications primarily involved changes in the list of representative species and estimates of their diets and the use of additional information to reinterpret toxicity information and the use of uncertainty factors. The report also demonstrated how the approach could be used to calculate the chemical concentration in dietary components representing various trophic levels.

The Canadian government developed a similar approach for national wildlife criteria, with a few notable differences compared to the GLWQI approach (CCME 1998). First, instead of using NOAEL values as the test dose, they use the geometric mean of NOAEL and LOAEL. Second, instead of basing a class-specific criterion on the geometric mean of the wildlife values for representative species, they use the lowest wildlife value calculated for a list of 28 avian or 9 mammalian species to calculate class-specific reference concentrations. Third, the reference concentrations are expressed as the chemical concentration in the diet of each representative species, though the approach does not address the relationship in concentrations among dietary items from the various trophic levels. Wildlife reference values have been developed for DDT, methylmercury, toxaphene, PCBs, and polychlorinated dibenzo-*p*-dioxins/polychlorinated dibenzofurans.

Wildlife criteria also have been developed for specific locations to address questions about the risks of waterborne contaminants to wildlife species. In New Jersey, the GLWQI approach is being used for developing wildlife criteria for PCBs, DDT and mercury for addressing concerns for bald eagles (*Haliaeetus leucocephalus*) and peregrine falcons (*Falco peregrinus*) related to the Endangered Species Act of 1973 (Buchanan et al., 2001). A modification of the GLWQI approach was used in California for addressing a question about the extent to which the EPA proposed human health criterion for methylmercury in the diet was protective of the state's threatened and endangered species (USFWS 2003). The approach was modified to calculate a wildlife value based on dietary concentration of methyl mercury and to convert that value to the corresponding concentrations in dietary constituents from the various trophic levels.

4.2 Issues in Developing Tissue-based Criteria Protective of Wildlife

The process for developing tissue-based criteria for aquatic-dependent wildlife is conceptually the same as for aquatic organisms. However, there are several specific differences in methods that reflect differences in chemical exposure pathways for wildlife and the nature and availability of wildlife toxicity testing. Also, the process for wildlife criteria being discussed is conceptually the same as has been used in previous development of wildlife criteria, although we are revising or expanding some aspects of the process.

The next several sections will discuss the process envisioned for national-level wildlife criteria. This process also is intended to be flexible for modifying a criterion to incorporate site-specific information. Although our current focus has been on an overall national process, guidance will be provided in future versions for determining when and how a site-specific criterion may be developed.

The following sections are intended to describe the wildlife criteria process conceptually, rather than to provide detailed procedures with supporting technical documents. In describing the process, we will highlight those aspects that differ from previous uses of wildlife criteria. One of the primary changes compared to previous wildlife criteria efforts is that we are proposing that a criterion may be based on either a tissue concentration in species of concern or their diet, depending on the availability and quality of information. Another change is to promote the use of probabilistic methods in formulating a criterion when data are of appropriate quantity and quality to do so. For most chemicals with limited data it is recognized that deterministic methods may be more appropriate.

4.3 Screening Available Toxicity Data

The first step in determining how to proceed with a particular chemical is to screen the available toxicity information and collect studies that satisfy the minimum standards for acceptability. The term "study" may refer to a single experiment (or similar unit of research) that estimates a toxicological effect level of a species or a series of experiments that can be integrated to estimate an effect level for a species. Both laboratory and field studies are to be considered if they meet all of the following standards:

- Studies must be based on an experimental design or approach that provides a defensible, chemical-specific response on endpoints that could have implications at the population level, such as reproductive or developmental success, organism viability or growth, etc. For instance, a study must have suitable controls or reference conditions.

- Laboratory studies must contain sufficient information so that the form of the chemical tested is clearly stated, and the administered doses are either reported or can be calculated from information provided.

- Studies must include a subchronic or chronic exposure duration. Laboratory acute oral (i.e., LD50) and short-term (e.g., 5-day LC50) tests are not acceptable.

- Laboratory studies should be based on an oral route of exposure. Laboratory studies using non-oral routes of exposure (e.g., intravenous or subcutaneous injections, implants, etc.) are not acceptable. A possible exception is the use of egg injection studies when there is sufficient understanding of the comparable toxicity from maternally-transferred concentrations.

- Studies may be based on effects relative to a dietary exposure concentration OR to a tissue concentration (e.g., egg or liver concentration vs effect), where scientifically justified.

- Studies must exist in a written form that is available to the public (e.g., journal articles, book chapters, published reports) and that either have gone through a defined technical peer-review process or exist in sufficient detail that a technical quality review can be conducted prior to acceptance.

After reviewing the available studies for a specific chemical, if no study satisfies these standards for a particular taxonomic assemblage (i.e., three wildlife assemblages are birds, mammals, and reptiles), there is insufficient toxicity information to establish a wildlife value for that assemblage. If one or more studies satisfy the standards, they are further evaluated to determine the quality of the study and to document the species tested, endpoints measured, and how endpoints are expressed. This review provides a compilation of all toxicity information for making a preliminary assessment of the quality and quantity of data available for supporting different forms of criteria. It is important to determine how many species from each assemblage have been tested, the comparability of experimental designs and endpoints measured, and any deficiencies in designs that may be addressed through the use of uncertainty factors.

Where multiple studies for a chemical satisfy the standards, the studies need to be evaluated to determine if the quality of information is sufficient to calculate a species sensitivity distribution (SSD). If sufficient toxicity data exist to estimate a representative SSD, the toxicity value used in calculating a criterion would be selected from the distribution (e.g., 5^{th} or 10^{th} percentile from SSD) depending on the intended level of protection. If there is insufficient information for defining a SSD, studies are evaluated to select the one study (or series of related studies) for each taxonomic group that represents the most complete, scientifically-sound study on which to define the test dose (i.e., TD) or tissue concentration for use in a deterministic criterion.

Since a tissue-based wildlife criterion may be based either on chemical concentrations in specific animal tissues (e.g., mg/kg tissue) or in dietary items (e.g., mg/kg food type), the review of toxicity information will group relationships based on animal tissues separately from those based on diet and proceed to derive wildlife values using both types of relationships in parallel. Some studies will provide information on the relationship of effects to both dietary concentrations and tissues concentrations. Ultimately, the weight of the evidence will be used to determine the most scientifically sound means for expressing a wildlife criterion in terms of diet or tissues concentrations.

4.4 Wildlife Criteria Based on Diet Concentration

Wildlife criteria based on dietary concentrations differ somewhat from the tissue-based criteria described for aquatic organisms. The focus on studies that relate effects to chemical concentrations in the diet reflects that this is a commonly used experimental design in wildlife toxicity testing. The core of this approach for wildlife criteria is to determine a daily dietary dose of a chemical that is protective of the more sensitive species of concern and integrate this with information on exposure potential to estimate a concentration in the diet of representative species (also known as a wildlife value) that is intended to be protective (Figure 3). The primary difference in this process based on dietary concentrations compared to the more generalized tissue-based criterion process in Figure 1 is that the dietary toxicity information is integrated with exposure parameters for each representative species prior to calculating the wildlife values. The following sections describe the overall approach for determining a wildlife criterion based on dietary concentrations using deterministic or probabilistic methods.

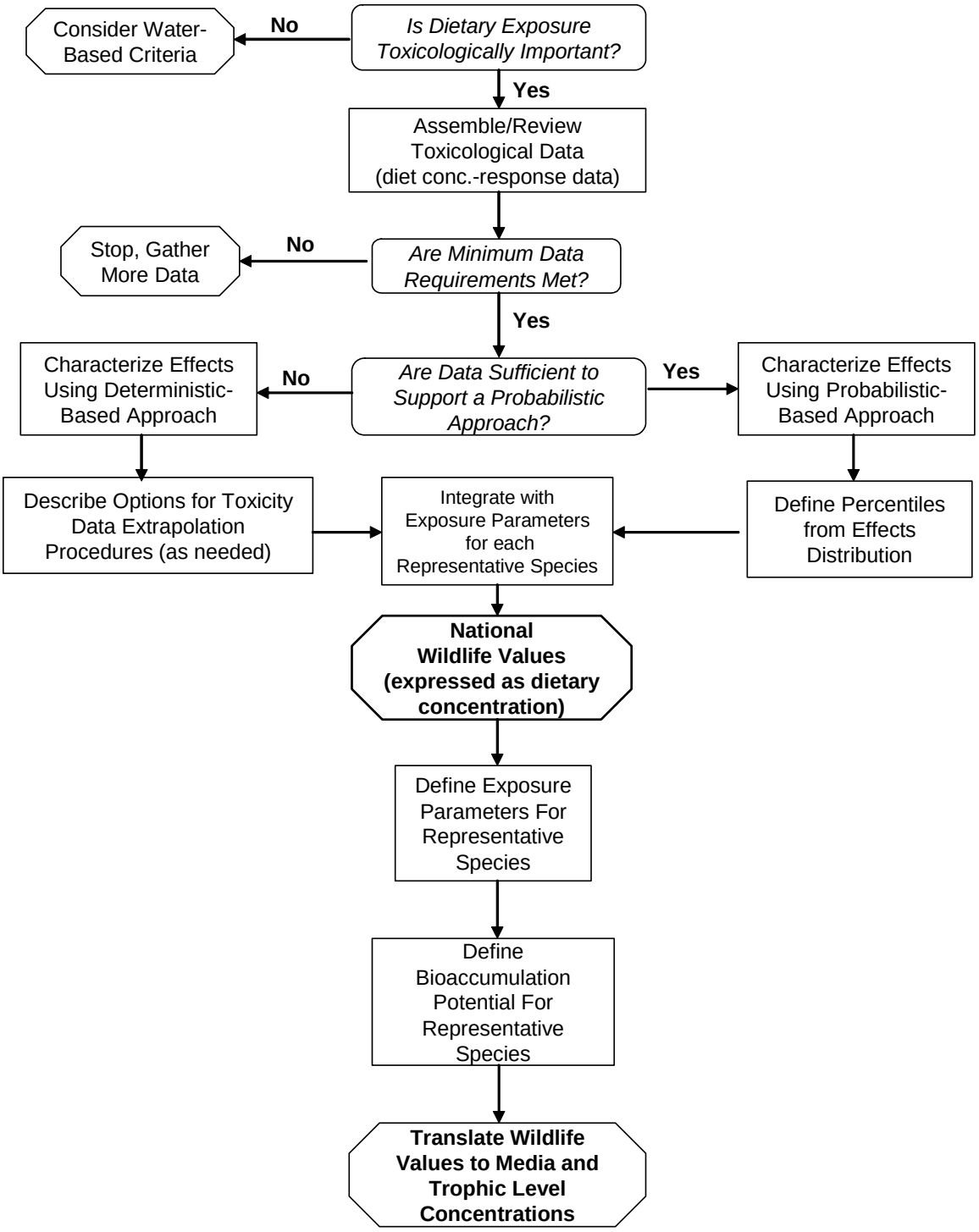


Figure 3. Schematic for Deriving Wildlife Criteria based on Dietary Concentrations

4.4.1 Characterizing effects for a deterministic criterion

For dietary studies that meet the minimum standards of acceptability, endpoints expressed as the chemical concentration in the diet need to be converted to an estimated daily dietary dose (mg/kg body wt/day). The test dose for deterministic criteria should be derived from the study (or series of related studies) for each taxonomic assemblage that represents the most complete, scientifically-sound study. The standards for selecting the most appropriate study are:

- Studies that were designed to measure effects on a suite of reproductive and/or developmental endpoints, as well as record effects on survival, are preferred over studies that are not designed to address reproductive effects.

- Integrative reproductive endpoints that most closely reflect measures of annual fecundity rates are preferred over reproductive endpoints reflecting specific aspects of the reproductive process. For example, while all endpoints may be useful, the number of fledglings produced per nesting attempt is preferred over endpoints such as eggshell quality or number of eggs laid.

- In general, the exposure duration of studies should result in the maximum severity of effects. For bioaccumulative chemicals, studies using a chronic exposure duration are preferred over studies with shorter durations (i.e., subchronic) which may underestimate the severity of effects observed at chronic exposures.

- Studies that clearly define the concentration or dose below which adverse effects are not observed (e.g., NOAELs, EC_{xx} from regression analyses) are preferred over studies where either all of the concentrations or doses cause significant adverse effects (i.e. provide an unbounded LOAEL) or none produce effects distinguishable from control responses (i.e., unbounded NOAEL).

- Laboratory studies that are designed to address the relationship of their results to field responses are preferred over laboratory studies that do not address the relationship to field responses.

- Studies using aquatic-dependent wildlife species would be preferred over studies using species that do not forage on aquatic organisms.

- Field studies that meet the above criteria would be preferred over laboratory studies assuming that relationship between exposure and effects can be accurately described. For populations exposed to multiple chemicals, an explanation is required addressing how observed effects can be assigned to the chemical of concern.

- If more than one study satisfies all of these standards, the study with the highest statistical power would be preferred.

Once the most complete study for selecting a test dose (TD) is determined for each assemblage, it needs to be evaluated to determine if it reflects a daily dietary dose that is protective of the more sensitive species within the assemblage. If not, the selected TD may need to be modified by uncertainty factors. There are two types of uncertainty factors. First, an interspecies uncertainty factor (UF_A) can be used address the uncertainty concerning the variability in toxicological sensitivity among species. Second, there are uncertainty factors used to compensate for deficiencies in the experimental designs of selected studies such as studies of insufficient duration (i.e., subchronic to chronic uncertainty factor or UF_S) or that do not estimate an effects threshold (i.e., LOAEL to NOAEL uncertainty factor or UF_L). By selecting the most complete study for determining the TD, we are striving to minimize the use of the second kind of factors.

During the GLWQI a technical basis for the use of uncertainty factors was developed from an analysis to toxicity studies (USEPA 1996). While this provides an empirical basis on which uncertainty factors can be parameterized, the selection of numerical uncertainty factors also is based on the management goals for the intended level of protection.

The selected study may not provide the most sensitive response among the studies available (i.e., may not result in the lowest TD). Even if the study selected provided the lowest TD among available studies, it is probable that there are additional untested species that are more sensitive to the chemical. Differences in toxicity among wildlife species can often exceed two orders of magnitude (Hart et al. 2001). Analyses of wildlife toxicity databases, primarily acute toxicity test data, have produced several statistical procedures for deriving extrapolation factors for estimating the sensitivity of untested species (Baril et al. 1994, Luttik and Aldenberg 1997, Mineau et al. 1996, 2001a). Although there is much less data to conduct similar analyses of reproduction data, Mineau et al. (2001b) considered that avian reproductive data would be at least as variable as acute toxicity. Luttik et al. (2005) review these methods and propose an approach for extrapolating long-term toxicity data among wildlife species. These methods provide an empirical basis for estimating a UF_A for use in deterministic criteria where there are insufficient data to calculate a chemical-specific SSD.

In order to develop a criterion that is protective of more sensitive aquatic-dependent species in a taxonomic class, the TD from the selected study is modified using a UF_A that integrates information available from the empirically-based methods above together with the toxicity data available from all species in studies evaluated under the second set of criteria. Although the empirically-based methods provide insights into the variation in sensitivity among species based on analysis of large datasets, a comparison of the TD from the selected study with endpoints from the other available studies provides insights into where the selected TD falls within the distribution.

The Subcommittee recognizes that for some chemicals the most complete study available may be of insufficient duration or may not clearly define an effects threshold and uncertainty factors may be appropriate to address these deficiencies. The guidance developed during the GLWQI on the use of uncertainty factors provides a basis for developing national-level wildlife criteria (USEPA 1996), but we have not fully discussed what modifications or additions are needed.

In characterizing the effects information for a deterministic wildlife criterion, the selected TD is divided by the product of the three uncertainty factors. Concern has been expressed that multiplying several uncertainty factors can result in criteria that are overprotective. In the process outlined above, it is recognized that the UF_A may be important to protect species that are thought to be more sensitive than the tested species, but the UF_L and UF_S are used to compensate for deficiencies in the experimental designs of available studies. The use of a UF_A may be appropriate for most chemicals unless there is evidence that the test species is also a relatively sensitive species with its taxonomic assemblage. On the other hand, studies requiring the use of UF_L and UF_S should be used only when no other studies are available. For chemicals where the only studies available would require use of both a UF_L and UF_S, the uncertainty in toxicity information may be so great that no criterion should be established.

We have additional work to do in providing guidance on when it is appropriate to consider uncertainty factors and how to parameterize them. We are striving to develop a process that minimizes the need for uncertainty factors, and when they are needed, to provide guidance for determining an empirically-based value or concluding that the uncertainty is too great for criterion development.

4.4.2 Selecting toxicity information for a probabilistic criterion

When it is determined that there is a sufficient number of studies with different species in order to calculate an SSD, the studies are evaluated further to ensure that they are of comparable quality. An SSD is most useful if it accurately reflects the difference in chemical sensitivity among species. If studies vary too much in the endpoints measured, duration of exposure, statistical power, or other experimental design features, the calculated SSD may be confounded by these experimental differences that mask the true differences in sensitivity. It is also possible that some studies use exposure concentrations that do not result in a fully described dose-response relationship or identification of an effects threshold (i.e., only produce an unbounded LOAEL). In such cases, limited use of UF_L may be warranted to keep an adequate sample size of species tested. Similarly, for studies that are considered to be of insufficient exposure duration, it may be warranted to use UF_S to estimate what an effects threshold would be under chronic exposure scenarios. However, the more studies that need to be amended by UF_S or UF_L to compensate for deficiencies, the greater the uncertainty that the resulting SSD is an adequate reflection of the distribution of species sensitivities.

Criteria also can be expressed probabilistically when one or more studies quantify a dose-response relationship for a population-relevant endpoint. Instead of relying only on an estimate of an effects threshold, such as an NOAEL, a criterion derived using a dose-response relationship can be presented as an equation that estimates the exposure concentration associated with any level of effect.

The Subcommittee has not yet discussed the quantity or quality of data required to consider the use of probabilistic methods for criteria based on dietary concentrations.

4.4.3 Characterization of Exposure for National Criteria

Although there may be limited toxicological information for estimating the sensitivity of a specific wildlife species or the range of sensitivities among aquatic-dependent wildlife species,

the variation in exposure potential among aquatic-dependent wildlife species can be estimated where there is information about the dietary composition of species, their food consumptions rates (either measured or estimated as a function of body weight), and the relationship of chemical concentrations among various trophic levels of dietary items (i.e., trophic transfer factors). Aquatic-dependent wildlife species vary greatly in their dietary composition (e.g., aquatic vegetation vs higher trophic level fish, entirely aquatic diet vs partially aquatic diet), which results in great variation in exposure potential. The dietary composition of some wildlife species also can vary geographically due to differences in prey availability. Body size is important because food consumption rates tend to increase with decreasing body size. Also, trophic transfer factors vary depending on the bioaccumulation characteristics of a chemical, which affects the exposure potential among species.

For national-level criteria, trophic levels will be defined similarly to those used in the GLWQI assessment, with trophic level 1 (i.e., TL1) representing primary producers, TL2 representing primary consumers (i.e., many invertebrates and small fish), TL3 representing secondary consumers (e.g., forage fish, insectivorous birds), and TL4 representing top predators (e.g., carnivorous fish, fish-eating birds).

The wildlife criteria process is designed to determine which species have high exposure potential based on the factors above.

4.4.4 Representative species

There is a large number of wildlife species whose diet is derived entirely or partially from aquatic foodwebs. Many assessments will not estimate the exposure potential for every aquatic-dependent species, but will select a subset of species to represent the diversity of factors that determine exposure potential. Wildlife species identified as “representative species” are not necessarily the species of greatest concern or the only species being considered, but are chosen to represent the range of aquatic-dependent species. In other words, just because a selected representative species does not inhabit a certain location does not means it is not representing similar species that do. For each chemical the process is designed to identify which foraging strategies have high exposure potential.

For national-level wildlife criteria, a table of representative species is being developed that 1) reflects the diversity in body weights and diets among aquatic–dependent species, 2) includes species that have been studied sufficiently to quantify dietary composition and determine trophic level of dietary components, and 3) are relatively widely distributed and recognized and/or valued by the public. At a later stage, guidance will be provided for implementers that prefer to select species representative of their specific jurisdiction, including issues to address in providing a rationale for their selection.

A dietary composition and trophic level analysis was completed for 20 species for the GLWQI (USEPA 2002). The list includes 16 birds (including Osprey, Bald eagle, Belted kingfisher, Herring gull, Ring-billed gull, Great blue heron, Black-crowned night-heron, Common tern, Forster's tern, Caspian tern, Double-crested cormorant, Common merganser, American merganser, Red-breasted merganser, Lesser scaup, and Mallard,) and four mammals (including Mink, River otter, Raccoon, and Harbor seal). Work is currently underway to expand this analysis to additional species. Candidate species under consideration include Common loon,

Western grebe, Pied-billed grebe, Eared grebe, White pelican, Green heron, Little blue heron, King rail, Peregrine falcon, Least tern, American avocet, and Marsh wren. We have decided to initially focus on species feeding primarily in freshwater systems, though in the future the process will be expanded to address species feeding in marine and estuarine systems. We also have not decided how to address reptiles given the paucity of toxicological data.

4.4.5 Body weight, food ingestion rate, and diet composition of representative species for deterministic criteria

Smaller birds and mammals generally have higher food ingestion rates relative to their body mass than do larger ones. This suggests that small animals would be exposed to a larger quantity of contaminants relative to the body size (i.e., dose) than larger animals. However, small piscivores are generally size-limited predators, and feed on smaller fish in lower trophic levels than do larger piscivores. Because the concentration of bioaccumulative chemicals usually is lower in lower trophic level organisms, it is not clear that small animals always experience higher exposures than larger animals. Therefore, to identify species likely to experience the highest exposure levels, both relative food ingestion rates and the trophic level of prey must be considered. For highly bioaccumulative chemicals, the species feeding at highest trophic levels of the aquatic food chain may have the highest dietary exposure potential (i.e., result in lowest criterion) in the process. For chemicals with lower bioaccumulation potential, the smallest body mass (and consequently highest food ingestion rate) may have the highest dietary exposure potential.

For national-level deterministic wildlife criteria, default values representing female body weight, estimated food ingestion rate (FIR), and proportion of diet derived from each trophic level category will be selected for each representative species. The reference for each default body weight and FIR will be stated, as well as the background analysis for determining the trophic level proportions for each diet. Implementers at the state or site-specific level will be able to use locally-derived information for modifying these default values if they can provide a rationale for why that is an improvement over using the national default information.

4.4.6 Trophic transfer factors for deterministic criteria

Trophic transfer factors (TTF) represent the ratio of the estimated chemical concentration in one trophic level to the concentration in the trophic level below it. They may be calculated directly from measured concentrations in representatives from various trophic levels or indirectly from the ratio of BAFs. The Subcommittee has not discussed yet the requirements for determining TTF for a national-level criterion, although we recognize the need for establishing a transparent process.

4.4.7 Body weight, diet composition, and trophic transfer factors for probabilistic criteria

The point estimate for each of the exposure parameters used in a deterministic criterion is derived from a distribution of values. Some of these distributions are well-defined descriptions of the natural variability for a parameter, while we are more uncertain in our knowledge about others. In a probabilistic approach to developing criteria, each of the exposure parameters can be described as a distribution in order to better understand the variability and uncertainty in the exposure potential of individuals within a species. The subcommittee has not specifically discussed procedures for accomplishing this.

4.4.8 Calculating a deterministic wildlife value based on dietary concentration

The model used to calculate a wildlife value based on concentration of chemical in the diet is a modification of the model used in the GLWQI expressed as the concentration in diet rather than water (USEPA 1995b). It is the same as the model used in an assessment of mercury in California (USFWS 2003). A wildlife value is calculated for each representative species because exposure potential varies with body weight, food ingestion rate, and diet composition, even though the same test dose is applied to each representative species (Figure 3). The equation for calculating wildlife values is:

$$WV_{food}\ (mg/kg\ food) = \frac{TD\ (mg/kg\ bw/d) * (1/(UF_A * UF_S * UF_L)) * BW\ (kg)}{\sum[FC_i\ (kg\ food/d)]}$$

where:

WV = wildlife value expressed as the chemical concentration in the diet of each representative species,
TD = test dose expressed as daily dietary dose from selected study,
UF = uncertainty factors for interspecies variation (UF_A), subchronic to chronic (UF_S), and LOAEL to NOAEL (UF_L),
BW = estimated mean body weight of a representative species, and
FC_i = amount of daily food consumption for each species from the i^{th} trophic level.

Because it is based on the entire diet for each species, a wildlife value itself is not a criterion. The wildlife value of each species needs to be translated into the corresponding concentrations at each trophic level using the estimates for the amount of food consumed from each trophic level and the TTFs. For example, a wildlife species that feeds on both TL3 and TL4 fish will have a wildlife value that reflects the concentration in the entire diet which may correspond to a concentration in TL3 fish that is lower than the wildlife value and a concentration in TL4 fish that is higher. This is needed to compare the estimated risk among representative species regardless of diet composition by translating the wildlife value into a common currency, such the corresponding concentration in TL3 fish. Because of significant differences in diet composition among species, the species with the lowest wildlife value does not necessarily translate into the species with the lowest corresponding concentration in TL3 fish.

A table will be produced listing the wildlife value for each representative species within the bird and mammal assemblages and the corresponding concentrations in the various trophic levels and water that would result in an average dietary concentration equivalent to the wildlife value. This table would show how differences in diet composition and body weight among species influence the chemical concentrations in each trophic level considered to be protective. It should be remembered that the same daily dietary dose is used for each species within an assemblage in these calculations assuming that any one of these untested species could be among the more sensitive species in the assemblage distribution. Consequently, the table does not literally specify which species are at greatest risk, but it does indicate which types of exposure profiles may be at greatest risk if species with those exposure profiles are among the more sensitive species toxicologically.

The information presented in these tables will provide the basis for determining national wildlife criteria, but more detailed procedures for determining criteria values will not be decided until discussions with Office of Water management have occurred. The final criterion will reflect the management goals for the intended level of protection.

4.4.9 Calculating probabilistic wildlife values based on dietary concentration

There are a variety of ways that wildlife values could be expressed probabilistically. One or more of the parameters in the equation above could be expressed as a distribution or an equation. This could result in wildlife values describing a distribution of values (rather than a single value) or the probability of exceeding a specified value. The goal should be to improve the characterization of risks by more explicitly integrating natural variability and uncertainty into the calculation of wildlife values. This provides risk managers greater insight in the degree of uncertainty in calculating wildlife values and the ramifications for achieving the intended level of protection.

The Subcommittee has not yet discussed specific applications of probabilistic methods or the data needs for using these methods.

4.5 Wildlife Criteria Based on Tissue Concentrations

Wildlife criteria based on tissue concentrations are quite similar to the tissue-based criteria described for aquatic organisms. They focus on studies from the laboratory or field that relate effects to chemical concentrations in specific animal tissues. Beyer et al. (1996) reviewed the evidence for interpreting tissue concentrations in wildlife. The core of this approach for wildlife criteria is to determine a specific tissue concentration of a chemical that is protective of the more sensitive species of concern (Figure 4). The following sections describe the overall approach for determining a wildlife criterion based on tissue concentrations using deterministic or probabilistic methods.

4.5.1 Characterizing effects for a deterministic criterion

In some cases the relationship between a specific animal tissue concentration and population-relevant effects may be less uncertain and more repeatable that relationships between diet concentrations and the same effects. When this can be demonstrated, a wildlife criterion based on specific tissue concentrations may be more robust at defining an unacceptable risk to wildlife species than one based on dietary concentrations (Figure 4). This process would be the same conceptually as was described for aquatic organisms in Section 3 and as illustrated in Figure 1. The information on tissue concentration-response relationships may or may not be derived from studies that also provide diet concentration-response information. Some studies, especially field studies, may provide tissue concentration-response information with little or no information about corresponding dietary exposure. Tissue concentration studies that do not provide direct evidence of the relationship back to dietary exposure may be acceptable if there is an alternative method to estimate corresponding exposure concentrations. A method for translating tissue concentrations into corresponding dietary concentrations is needed in order to understand how the chemical concentrations in tissues relates to concentrations throughout the food web and in abiotic media.

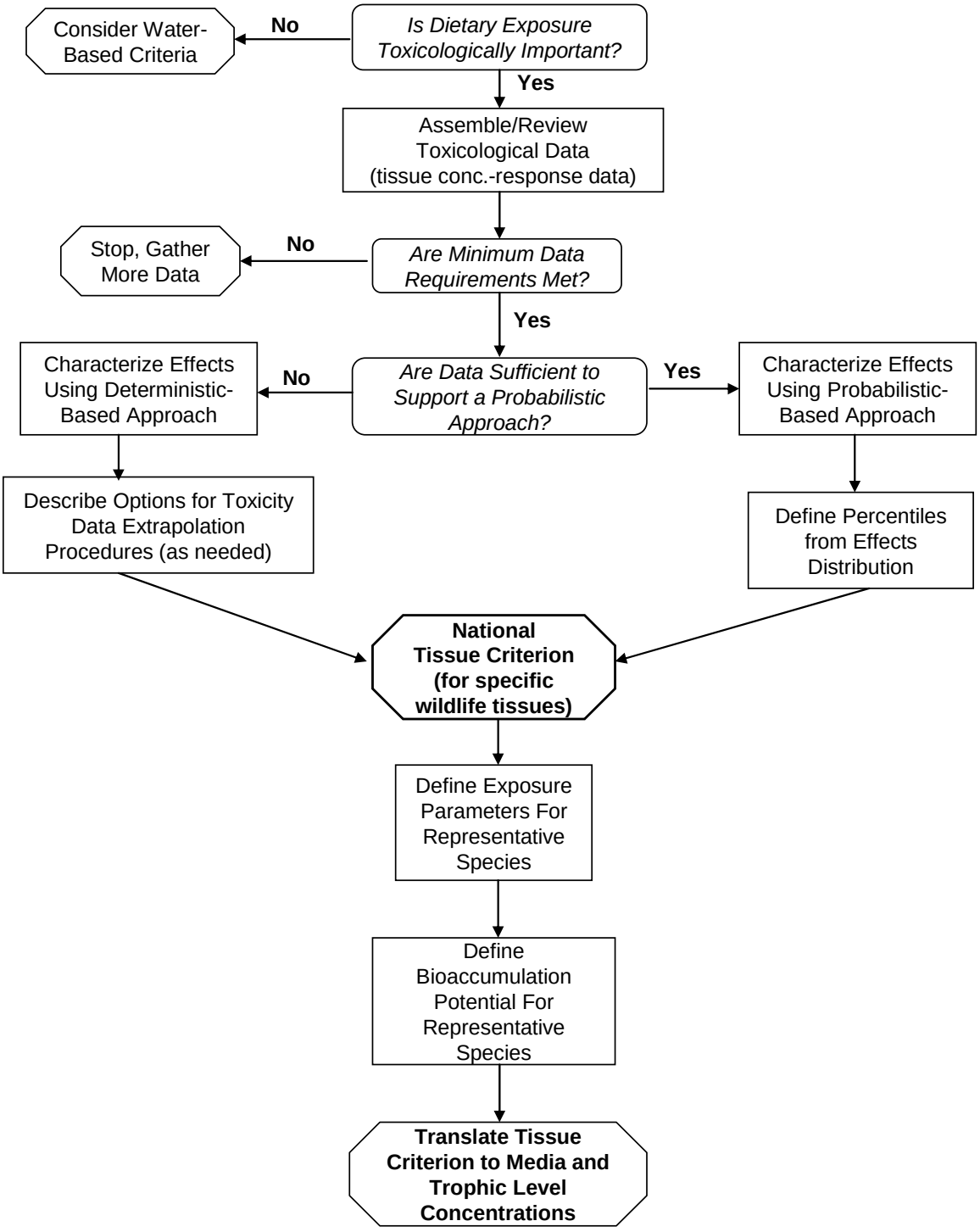


Figure 4. Schematic for Deriving Wildlife Criteria based of Tissue Concentrations

The standards outlined in section 5.4.1 for selecting the most appropriate study for each assemblage also apply here. An additional standard relates to the adequacy of the type of tissue used in the relationship.

- Studies with tissue concentration-response information based on tissue types with a direct causal relationship to the observed effects are preferred over studies based on tissue types with no clear causal connection. For example, if a major effect of a specific chemical is to interfere with embryo development and reduce hatchability, relationships based on whole egg concentrations may be the most appropriate tissue.

4.5.2 Characterizing effects for a probabilistic criterion

For some chemicals, there may be multiple studies that have determined the relationship between effects and the chemical concentrations in the same type of tissue. When it is determined that there is a sufficient number of studies with different species in order to calculate an SSD, the studies are evaluated further to ensure that they are of comparable quality. An SSD for tissue concentrations would provide additional information for determining a protective concentration commensurate with management goals.

The Subcommittee has not yet discussed the quantity or quality of data required to consider the use of probabilistic methods for criteria based on tissue concentrations.

4.5.3 Calculation of a wildlife value based on tissue concentration

Deterministic wildlife values based on tissue concentrations are calculated using the chemical concentration from the selected study modified by uncertainty factors, where necessary. Unlike the wildlife values based on dietary concentrations, the values based on tissue concentrations do not vary among representative species as a function of body weight and food ingestion rate. The equation for calculating wildlife values is:

$$WV_{tissue}\ (mg/kg\ tissue) = TC\ (mg/kg\ tissue) * (1/(UF_A * UF_S * UF_L))$$

where:

WV = wildlife value expressed as the chemical concentration in the specified tissue,
TC = tissue concentration from selected study, and
UF = uncertainty factors for interspecies variation (UF_A), subchronic to chronic (UF_S), and LOAEL to NOAEL (UF_L).

The uncertainty factors used to modify tissue concentrations are conceptually the same as those used for dietary concentrations. However, the empirical relationships based on tissue concentrations and effects may differ from those examined for dietary concentrations in the GLWQI guidance for use of uncertainty factors (USEPA 1996). Consequently, it should not be assumed that uncertainty factors selected for dietary studies apply equally well to tissue concentration information. Justification for the use of uncertainty factors should be based on a separate analysis of existing data. The Subcommittee has not specifically discussed the approach to using uncertainty factors for wildlife values based on tissue concentrations.

The wildlife values based on tissue concentrations may be used directly in determining a wildlife criterion. Additionally, there needs to be a method for translating these tissue concentrations into corresponding concentrations in lower trophic levels and abiotic media. This is straightforward when the relationship between effects and both diet and tissue concentrations are derived from the same study. When these relationships are based on different studies, we need to be able to compare wildlife values based on diet vs tissue concentrations to understand the relative degree of protection afforded. The Subcommittee is currently conducting an empirical analysis of the relationships between effects and concentrations in both diet and tissues to support methods to use in the criteria development process. In the future, guidance will be developed for determining wildlife criteria when there is sufficient data to calculate wildlife values based on both diet and tissue concentrations. The Saginaw Bay mink study series represents a good example of integrating both diet- and tissue-based data sets (Tillitt et al. 1996).

4.6 Role of population modeling

Under certain circumstances population modeling could be a valuable tool in the development of tissue-based wildlife criteria as a means of understanding the consequences of chemical exposures to wildlife populations. The primary use of population modeling would be in the development of site-specific criteria for data-rich chemicals where it is possible to improve the characterization of risks beyond that possible with the methods above. However, we do not envision the use of population modeling in national- or regional-level wildlife criteria development along the lines of that articulated by the Water-based Criteria Subcommittee (WCS). There are several important reasons for this difference in approach.

First, the WCS is using population models as a means of integrating effects data on survival, growth, and reproduction into a common metric of change in population size. However, the bulk of wildlife chronic effects data for bioaccumulative chemicals relates to reproduction endpoints, with little or no data on effects of chronic exposure to survival rates. Also, for many chemicals the effects to reproductive endpoints typically occur at environmental concentrations that are lower than would be expected to affect survival. Consequently, there is not the same possibility or need for integrating survival and reproduction data.

Second, the WCS is using population models to integrate population responses to varying exposure concentrations over time where there are periods of exposure causing declines in a population and periods of recovery. Wildlife exposed to bioaccumulative chemicals through an aquatic food web are expected to have less variation in exposure concentrations over time, and criteria are based on exposure concentrations deemed acceptable over the long-term. Wildlife criteria are intended to prevent the types of population-level effects that would require a recovery. Consequently, there is not the same need to integrate the effects of variable exposure or consider recovery rates.

Third, population models conceptually can be used to estimate the magnitude of effects to individuals that can be assimilated by a population, leading to estimates of the environmental concentration protective of the population rather than relying on the somewhat lower concentration that would protect against effects to individuals. However, wildlife toxicity information will often come from studies on species other than the species of concern and it will be from studies with less standardization than being required by the WCS. The toxicological

sensitivity of species of concern often has to be estimated from limited data on other species, resulting in an unknown degree of uncertainty in estimates. The amount of uncertainty in estimating the sensitivity of untested species can overshadow attempts to use population modeling to characterize effects in a population context.

Because of these reasons, we concluded that population modeling is unlikely to improve the characterization of risks at the national or regional level beyond what is possible with the deterministic or probabilistic approaches described above. However, we will be discussing the role population modeling could have in improving site-specific criteria development. An important consideration in those discussions is that populations are not exposed to one stressor (chemical or non-chemical) at a time, and population-level assessments in criteria development will need to address the cumulative impacts of co-occurring stressors.

5 Issues for SAB

5.1 Charge Questions

1. For chemicals with a high propensity to bioaccumulate in aquatic food webs and for which diet is a primary route of exposure, the Tissue-based Criteria Subcommittee proposes to develop tissue-based criteria expressed as the chemical concentrations in specific animal tissues or dietary concentrations, with a process for translating to corresponding water and sediment concentrations. Tissue-based criteria allow for integration of multiple exposure pathways (water, diet) and facilitate direct comparison with environmental tissue concentrations to determine if there is a risk of adverse effects. **Please comment on the rationale and conceptual approach used for the development of tissue-based criteria for this group of chemicals. Is the SAB aware of other approaches for deriving criteria for these bioaccumulative chemicals that EPA should consider?**

2. The proposed process for Tissue-based Criteria is intended to be flexible to maximize the use of available data and to accommodate certain limitations in the quality and quantity of data. National-level criteria may use deterministic approaches to characterize toxicity data when data are limited or probabilistic approaches (e.g., species sensitivity distributions) when data are sufficient. The process will also describe how a criterion may be refined on a site-specific basis when additional data are available. **Considering the strengths and limitations of the more flexible approach used to derive tissue-based criteria, please comment on the rationale and preference for allowing flexibility in the procedures used.**

3. Unlike the dynamic exposure scenarios being addressed by the Water-based Criteria Subcommittee, the Tissue-based Criteria Subcommittee is considering a steady-state approach for developing national criteria for bioaccumulative chemicals (i.e., modeling bioaccumulation and toxicity as a function of constant concentrations). Rationale for this approach is due in part to the much slower accumulation kinetics generally associated with these chemicals in higher trophic level fish and aquatic-dependent wildlife and concerns over their long-term bioaccumulation. In the context of population modeling, there appears to be much less residue-response information available for integrating responses of various demographic parameters over multiple life stages, such as fecundity and adult, juvenile, and larval survival. Consequently, the feasibility and utility of integrating population modeling into national-level tissue criteria for bioaccumulative chemicals is not clear to the Tissue-based Criteria Subcommittee. Current thinking is that where sufficient data exist to characterize exposure, bioaccumulation and toxicity on a dynamic basis, population modeling may evolve into an important tool in the development of site-specific criteria. **Please comment on the rationale used by the Tissue-based Criteria Subcommittee for determining if/when to use population modeling in the development of Tissue-Based Criteria.**

5.2 Additional Technical Issues: Aquatic Life Criteria

1. **Toxicity Data Extrapolations.** For deterministic-based aquatic life criteria, which might be derived with as few as 4-5 species per assemblage, the Subcommittee is considering the need for toxicity data extrapolations to account for potentially greater sensitivity of untested species. While methods have been developed conducting interspecies extrapolations of toxicity using water concentration-based toxicity data (e.g., ICE, adjustment factors for secondary Tier II criteria under the Great Lakes Initiative), the Subcommittee is not aware of analogous methods using tissue concentration-based toxicity data. **Please comment on: (1) the need for such toxicity data extrapolations and (2) available methods for conducting such extrapolations using tissue concentration-based toxicity data that the Subcommittee should consider.**

2. **Representative Species.** In order to address differential exposure potential among aquatic species and implementation of tissue-based criteria, the Subcommittee is considering the use of "representative species" (in conjunction with bioaccumulation methods) as described in Section 3.5.1. These representative species would reflect a range of exposure potential that might be experienced by aquatic species with tissue-based sensitivities at or near the tissue criterion. **Please comment on: (1) the rationale and approach presented by the Subcommittee for using representative species and (2) other methods the Subcommittee should consider for translating a tissue criterion into corresponding concentrations in media and the food web.**

3. **Bioaccumulation.** In the revision of the Aquatic Life Criteria guidelines to better address "bioaccumulative" chemicals, the Subcommittee proposes to use a framework for assessing bioaccumulation potential that is similar to that used by EPA in its National Ambient Water Quality Criteria to protect human health (USEPA 2000, 2003). The mechanistically-based portion of the bioaccumulation framework uses the fugacity based food web model of Gobas (1993) in cases where measured data is absent and when metabolism is considered negligible. Food web models have continued to evolve and improve since the publication of the Gobas 1993 model. **Have improvements in these models been significant enough to warrant EPA adopting an improved model into the bioaccumulation methodology of the revised guidelines? Do you agree with the idea of reserving the use of dynamic (time varying) bioaccumulation modeling for situations where short-term fluctuations in media concentrations are a concern with sensitive aquatic species?**

5.3 Additional Technical Issues: Wildlife Criteria

1. **Uncertainty Factors.** The standards for selecting wildlife toxicity studies emphasize the need to select the most complete studies in order to limit the need for uncertainty factors that compensate for deficiencies in experimental designs. When uncertainty factors are needed to maintain the desired level of protection, their selection should be based on an analysis of available information. **Based on the proposed procedures for selecting toxicity data, please comment on the rationale for use of uncertainty factors, where needed.**

6 References

Asfaw A, Ellersieck MR, and Mayer FL. 2003. *Interspecies Correlation Estimations (ICE) for Acute Toxicity to Aquatic Organisms and Wildlife. II. User Manual and Software*. EPA/600/R-03/106. U.S. Environmental Protection Agency, National Health and Environmental Effects Research Laboratory, Gulf Ecology Division, Gulf Breeze, FL. 14 p.

Baril A, Jobin B, Mineau P and Collins BT. 1994. *A consideration of interspecies variability in the use of the median lethal dose (LD50) in avian risk assessment.* Technical Report Series No. 216, Canadian Wildlife Service, Ottawa, Canada.

Barron MG, Hansen JA, and Lipton J. 2002. Association between contaminant tissue residues and effects in aquatic organsisms. *Rev. Environ. Contam. Toxicol.* 173:1-37.

Beyer WN, Heinz GH, and Redmon-Norwood AW. 1996. *Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations.* Lewis Publishers CRC Press, Boca Raton, FL. 494 pp.

Buchanan GA, Russell DW, and Thomas DA. 2001. *Derivation of New Jersey-Specific Wildlife Values as Surface Water Quality Criteria for: PCBs, DDT, Mercury*. Unpublished report.

Burkhard L.P. 2003. Factors influencing the design of bioaccumulation factors and biota-sediment accumulation factors field studies. *Environmental Toxicology and Chemistry.* 22:351-360.

Burkhard LP, Endicott DE, Cook PM, Sappington KG, and Winchester EL. 2003. Evaluation of two methods for prediction of bioaccumulation factors. *Environmental Science and Technology*. 37(20):4626-4634.

Cook PM, Carlson AR, Lee H. 1989. *Tissue Residue Approach*. Sediment Classification Methods Compendium, Chapter 7. EPA 823-R-92-006. Office of Water, Washington, D.C.

Cook PM, Erickson RJ, Spehar RL, Bradbury SP, and Ankley GT. 1993. *Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife.* EPA/600/R-93/055. Office of Research and Development, Duluth, MN 167pp.

Di Toro DM, McGrath JA, and Hansen DJ. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. I. Water And Tissue. *Environmental Toxicology and Chemistry*. 19:1951-1970.

Di Toro DM, and McGrath JA. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. II. Mixtures And Sediments. *Environmental Toxicology and Chemistry*. 19:1971-1982.

Di Toro DM, Allen HE, Bergman HL, Meyer JS, Paquin PR, and Santore RC. 2001. A biotic ligand model of the acute toxicity of metals I. Technical basis. *Environmental Toxicology and Chemistry* 20:2383-2396.

Escher BI and Hermens JLM. 2002. Modes of action in ecotoxicology: Their role in body burdens, species sensitivity, QSARs, and mixture effects. Environmental Science and Technology. 36(20): 4201-4217.

Gobas FAPC. 1993. A model for predicting the bioaccumulation of hydrophobic organic chemicals in aquatic food webs: Application to Lake Ontario. *Ecological Modeling*. 69:1-17.

Hart A, Balluff D, Barfknecht R, Chapman PF, Hawkes T, Joermann G, Leopold A, and Luttik R. 2001. *Avian effects assessment: A framework for contaminant studies*. SETAC, Pensacola, FL. 193 pp.

Jarvinen AW and Ankley JT. 1999. *Linkage of Effects to Tissue Residues: Development of a Comprehensive Database for Aquatic Organisms Exposed to Inorganic and Organic Chemicals*. SETAC Press, Pensacola, FL, 364 pp.

Könemann H. 1981. Quantitative structure-activity relationships in fish toxicity studies Part 1: Relationship for 50 industrial pollutants. *Toxicology*. 19(3): 209-221.

Landrum PF, Steevens JA, Gossiaux DC, McElroy M, Robinson S, Begnoche L, Chernyak S, and Hickey J. 2004. Time-dependent lethal body residues for the toxicity of pentachlorobenzene to Hyalella azteca. *Environmental Toxicology and Chemistry* 23:1335-1343.

Landrum PF, Steevens JA, McElroy M, Gossiaux DC, Lewis JS, and Robinson SD. 2005. Time-dependent toxicity of dichlorodiphenyldichloroethylene to Hyalella azteca. *Environmental Toxicology and Chemistry* 24(1): 211-218.

Lee J-H, Landrum PF, and Koh C-H. 2002a. Toxicokinetics and time-dependent PAH toxicity in the amphipod Hyalella azteca. *Environmental Science and Technology*. 36: 3124-3130.

Lee J-H, Landrum PF, and Koh C-H. 2002b. Prediction of time-dependent PAH toxicity in Hyalella azteca using a damage assessment model. *Environmental Science and Technology*. 36: 3131-3138.

Legierse KCHM, Verhaar HJM, Vaes WHJ, De Bruijn JHM, and Hermens JLM. 1999. Analysis of time-dependent acute aquatic toxicity of organophosphorus pesticides: The critical target occupation model. *Environmental Science and Technology*. 33:917-925.

Luttik R and Aldenberg T. 1997. Extrapolation factors for small samples of pesticide toxicity data: Special focus on LD50 values for birds and mammals. *Environmental Toxicology and Chemistry* 16: 1785-1788.

Luttik R, Mineau P, and Roelofs W. 2005. A review of interspecies toxicity extrapolation in birds and mammals and a proposal for long-term toxicity data. *Ecotoxicology* (in press).

McCarty LS and Mackay D. 1993. Enhancing ecotoxicological modeling and assessment: body residues and modes of toxic action. *Environmental Science and Technology* 27:1719-1728.

McCarty LS. 1986. The relationship between aquatic toxicity QSARS and bioconcentration for some organic chemicals. *Environmental Toxicology and Chemistry* 5:1071-1080.

Mineau P, Collins BT and Baril A. 1996. On the use of scaling factors to improve interspecies extrapolation of acute toxicity in birds. *Regulatory Toxicology and Pharmacology* 24: 24-29.

Mineau P, Baril A, Collins BT, Duffe JA, Joermann G and Luttik R. 2001a. Pesticide acute toxicity reference values for birds. *Reviews of Environmental Contamination and Toxicology* 170: 13-74.

Mineau P, Hooper M, Elmegaard N, Grau R, Luttik R and Ringer R. 2001b. Case study 5: Foliar insecticide II. In Hart A, Balluff D, Barfknecht R, Chapman PF, Hawkes T, Joermann G, Leopold A, and Luttik R (eds). 2001. *Avian effects assessment: A framework for contaminant studies.* SETAC, Pensacola, FL. 193 pp.

Munns WR, Black DE, Gleason TR, Salomon K, Bengtson D, and Gutjahr-Gobell R. 1997. Evaluation of the effects of dioxin and PCBs on *Fundulus heteroclitus* populations using a modeling approach. *Environmental Toxicology and Chemistry*. 16(5):1074-1081.

Paquin PR, Gorsuch JW, Apte S, Batley GE, Bowles KC, Cambell PGC, Delos CG, Di Toro DM, Dwyer RL, Galvez R, Gensemer RW, Goss GG, Hogstrand C, Janssen CR, McGeer JC, Naddy RB, Playle RC, Santore RC, Schneider U, Stubblefield WA, Wood CM, Wu KB. 2002. The Biotic Ligand Model: a Historical Overview. *Comparative Biochemistry and Physiology Part C* 133:3-35.

Peterson JA and Nebeker AV. 1992. Estimation of waterborne selenium concentrations that are toxicity thresholds for wildlife. *Archives of Environmental Contamination and Toxicology* 23: 154-162.

Rainbow PS. 2002. Trace metal concentrations in aquatic invertebrates: why and so what? *Environmental Pollution* 120:497-507.

Stephan CE, Mount DI, Hansen DJ, Gentile JH, Chapman GA, and Brungs WA. 1985. *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses.* U.S. Environmental Protection Agency, Office of Research and Development, Duluth, MN, 98 pp.

Steevens JA, Reiss MR, and Pawlisz AV. 2005. A methodology for deriving tissue residue benchmarks for aquatic biota: A case study for fish exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin and equivalents. *Integrated Environmental Assessment and Management*. 1(2): 142-151.

Tillitt DE, Gale RW, Meadows JC, Zajicek JL, Peterman PH, Heaton SN, Jones PD, Bursian SJ, Kubiak TJ, Giesy JP, and Aulerich RJ. 1996. Dietary exposure of mink to carp from Saginaw Bay. 3. Characterization of dietary exposure to planar halogenated hydrocarbons, dioxin equivalents and biomagnification. *Environmental Science and Technology* 30:283-291.

USACE. 2004. *Environmental Residue-Effects Database*. U.S. Army Corps of Engineers, Waterways Experiment Station. Database available on line at: http://el.erdc.usace.army.mil/ered/

USEPA. 1989. *Water Quality Criteria to Protect Wildlife Resources*. Office of Research and Development. EPA/600/3-89/067. Corvallis, Oregon.

USEPA. 1995a. *Final Water Quality Guidance for the Great Lakes System; Final Rule*. Federal Register. V. 60, No. 56, pp. 15366-15425. March 23.

USEPA. 1995b. *Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors*. Office of Water. EPA-820-B-95-005.

USEPA. 1995c. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, Mercury, 2,3,7,8-TCDD, PCBs*. Office of Water. EPA/820/B-95/008. Washington, D.C.

USEPA. 1995d. *Great Lakes Water Quality Initiative Technical Support Document for Wildlife Criteria*. Office of Water. EPA/820/B-95/009. Washington, D.C.

USEPA. 1996. *Use of Uncertainty Factors in Toxicity Extrapolations Involving Terrestrial Wildlife*. Office of Research and Development, Office of Water. Peer Reviewed Draft, Washington, DC

USEPA. 1998. *Guidelines for Ecological Risk Assessment.* U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, DC, EPA/630/R095/002F. (address as of 1-31-05: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=12460)

USEPA. 2000. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000).* Office of Water, EPA-822-B-00-004. Washington, DC. (available at: http://www.epa.gov/waterscience/criteria/humanhealth/method/index.html)

USEPA. 2002. *Trophic Level and Exposure Analyses for Selected Piscivorous Birds and Mammals: Volumes 1, 2 and 3*. Office of Water, Washington, DC.

USEPA. 2003. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000), Technical Support Document Volume 2: Development of National Bioaccumulation Factors*. Office of Water. EPA-822-R-03-030. Washington, D.C.

USEPA. 2004. *Draft Aquatic Life Water Quality Criteria for Selenium-2004*. Office of Water, EPA-822-D-04-001. Washington, DC. November. (available at: http://www.epa.gov/waterscience/criteria/selenium/index.htm)

USEPA Science Advisory Board. 1994. Advisory on the development of a national wildlife criteria program. USEPA Science Advisory Board, Washington, DC. EPA-SAB-EPEC-ADV-94-001.

U.S. Fish and Wildlife Service. 2003. *Evaluation of the Clean Water Act Section 304(a) human health criterion for methylmercury: protectiveness for threatened and endangered wildlife in California*. U.S. Fish and Wildlife Service, Sacramento Fish and Wildlife Office, Environmental Contaminants Division. Sacramento, California. 96 pp + appendix.

Veith GD, DeFoe, DL, Bergstedt, BV. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. *Journal of the Fisheries Research Board Canada* 36:1040-1048.

Veith GD, Call DJ, and Brooke L. 1983. Structure-toxicity relationships for the fathead minnow, Pimephales promelas: Narcotic industrial chemicals. *Canadian Journal of Fisheries and Aquatic Science* 40:743-748.

Verhaar, HJM., De Wolf, W, Dyer, S, Legierse, KCHM, Seinen, W, Hermens, JLM. 1999. An LC_{50} vs time model for aquatic toxicity of reactive and receptor-mediated compounds. Consequences for bioconcentration kinetics and risk assessment. *Environmental Science and Technology*. 33:758-763.

7 Appendix A

Cursory Review of Tissue Concentration-Response Data for Aquatic Organisms Contained in Two Databases

This appendix contains results from initial queries made of two databases containing tissue concentration-response data for aquatic organisms. These databases are:

- Jarvinen and Ankley (1999)
- Environmental Residue-Effects Database (USACE) (downloaded on September 27, 2004)

The primary purpose of these queries was simply to characterize basic attributes of the available tissue concentration-response data that have been coded to date. For example:

- How many species are represented by various chemical?
- How frequent are different endpoint classes represented? (e.g., mortatlity, growth, reproduction)
- How frequent are different types of tissues represented?
- What are the most commonly tested species?
- What exposure routes are most commonly tested?

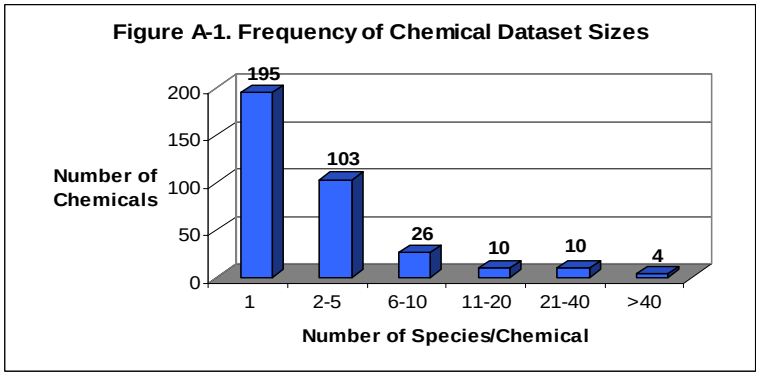
Both databases were available in electronic formats and were merged into a single MS Access™ database for further analysis. Duplicate records were removed when unambiguous determinations could be made. The ERED database contained significantly fewer fields than the Jarvinen and Ankley database, thus a number of fields in the merged database were unpopulated.

A few important caveats should be noted:

1. No attempt was made to review or screen the data for quality purposes.
2. In many cases, records reflect multiple effect levels of a given endpoint from the same test (e.g., NOAELs, LOAELs, and ECxx were recorded as separate records). The database structure did not enable unambiguous identification of "paired" NOAELs and LOAELs.
3. Nomenclature for classifying data between the two databases differed in some cases. Original classification was retained in situations were interpretations of nomenclature differed.
4. As a result of these and other factors, the actual amount of data that would be useful for criteria derivation purposes in the merged database would likely be significantly less than represented here (i.e., data were not screened). However, newer data not captured by these databases may mitigate the reduction in useable data to some extent.

Based on these simple queries, the following statements can be made regarding the status of the coded tissue concentration-response data:

1. The vast majority of chemicals are represented by 5 or fewer aquatic species (about 85%). Only about 7% of chemicals coded in the database contain more than 10 aquatic species represented (Figure A-1).



2. Organic chemicals with 6 or more species represented include:

Organochlorine Pesticides:

aldrin, DDE, DDT, chlordane, endrin, endosulfan, heptachlor, kepone, lindane, methoxychlor, mirex, toxaphene

Organophosphate Pesticides:

chlorpyrifos, diazinon

Pyrethroids:

fenvalerate, permethrin

PAHs:

benzo(a)pyrene, flouranthene, naphthalene, phenanthrene, pyrene

Metals/metalloids:

arsenic, cadmium, chromium, copper, lead, mercury, nickel, selenium, silver, vanadium, zinc

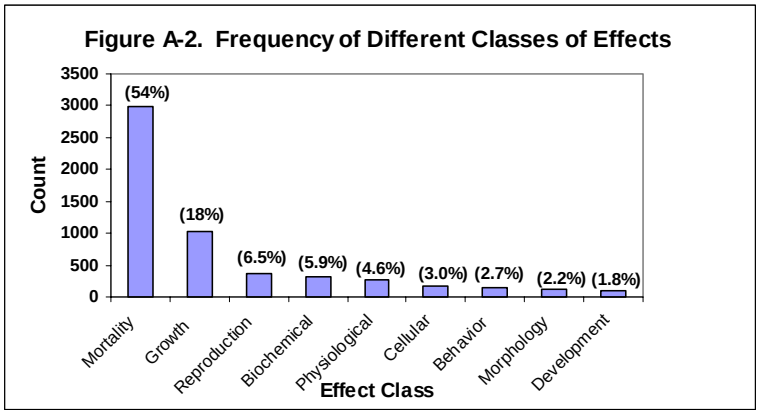
PCBs/Dioxins:

2,3,7,8-TCDD, various aroclors

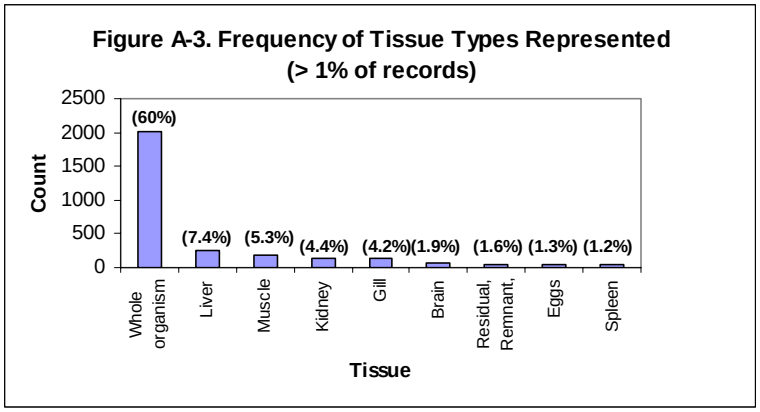
Other:

PCP, 2,4,6-trichlorophenol, 2,3,4,6-trichlorophenol, hexachlorobenzene, pentachlrobenzene, TBT, dibutyltin, di-2-ethylhexyl phthalate

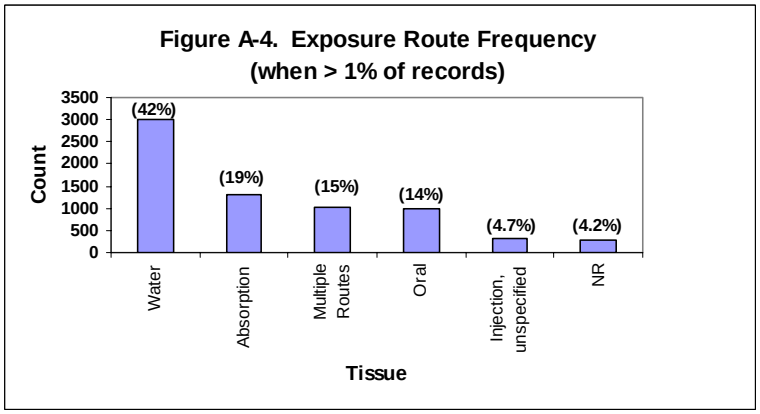
3. Mortality is by far the most common endpoint measured (over half of the coded data). Reproductive endpoints constitute a relatively small fraction of the data (about 6%; Figure A-2)



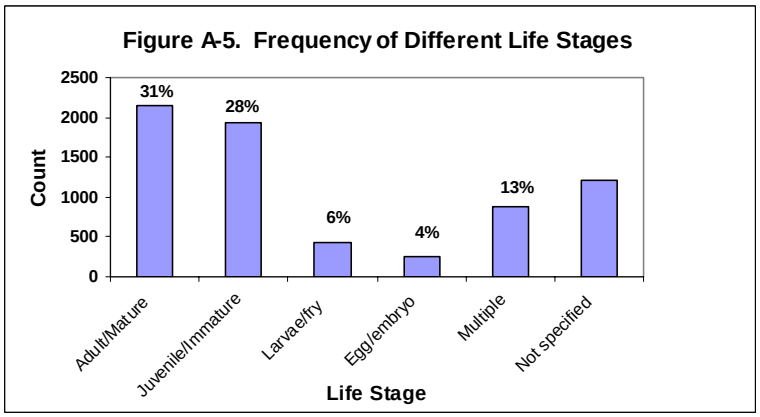
4. Whole organism measurements are by far the most common tissue sampled (Figure A-3).



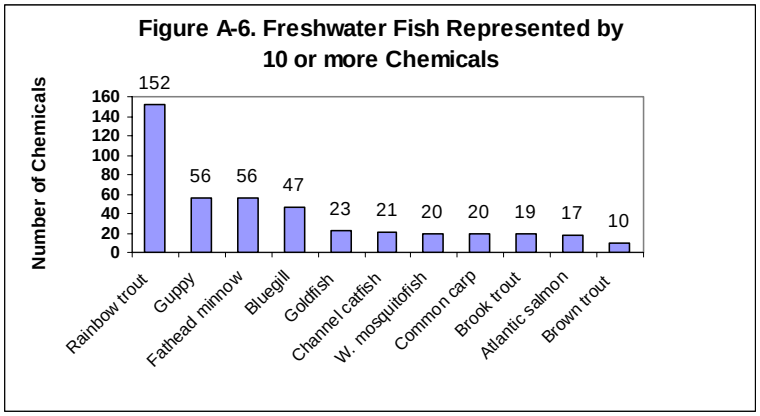
5. Water only exposures are most common, followed by multiple routes and oral (presumably food ingestion; Figure A-4).



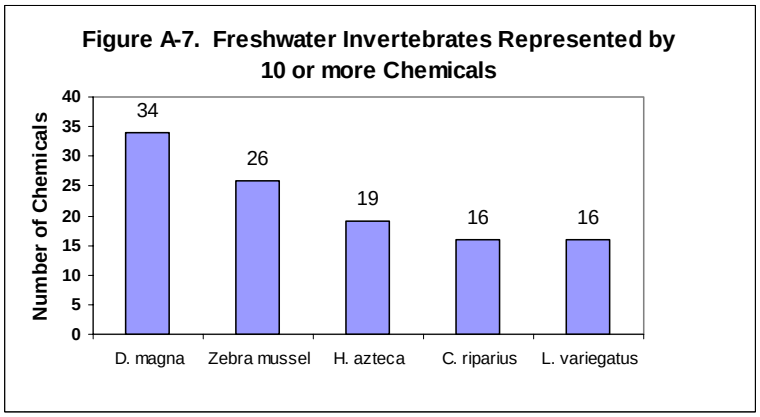
6. Adult and juvenile/immature life stages are most commonly represented (about 60% of the records), with early life stages (larval/fry and egg/embryo) and multiple life stages constituting about 10% and 13% of the records, respectively (Figure A-5).



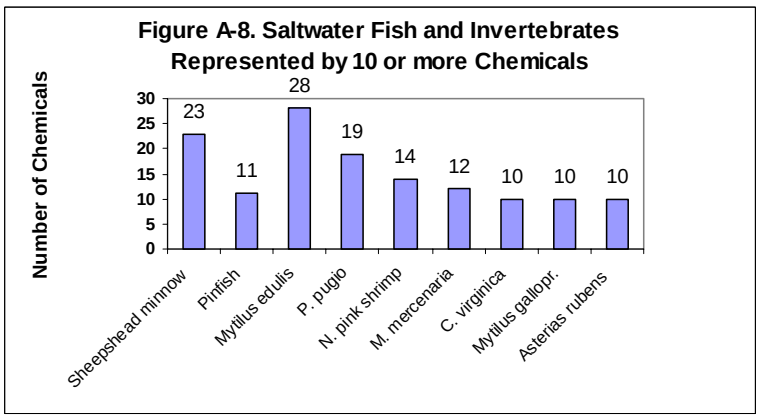
7. Most common groups of freshwater fish species represented are salmonids (rainbow trout, brook trout, brown trout, atlantic salmon), followed by cyprinids (fathead minnow, goldfish, common carp) and Poeciliidae (guppy, mosquitofish; Figure A-6).



8. The most common freshwater invertebrates represented include a cladoceran (*D. magna*), an amphipod (*H. azteca*), a mollusk (zebra mussel), an insect (*C. riparius*) and an oligochaete (*L. variegates*; Figure A-7).



9. Few saltwater fish species are represented broadly in the database (i.e., for 10 or more chemicals) while shrimp and bivalve mollusks are among most commonly tested invertebrate species (Figure A-8).



References for Appendix A

Jarvinen, AW, Ankley, JT. 1999. *Linkage of Effects to Tissue Residues: Development of a Comprehensive Database for Aquatic Organisms Exposed to Inorganic and Organic Chemicals*. SETAC Press, Pensacola, FL, 364 pp.

USACE. 2004. Environmental Residue-Effects Database. U.S. Army Corps of Engineers, Waterways Experiment Station. Database available on line at: http://el.erdc.usace.army.mil/ered/

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF WATER

May 25, 2023

Laura Watson, Director
Washington State Department of Ecology
Post Office Box 47600
Olympia, Washington 98504-7600

Dear Director Watson,

This letter constitutes the U.S. Environmental Protection Agency (EPA) Administrator's Determination (Determination), pursuant to Clean Water Act (CWA) Section 303(c)(4)(B), that new and revised water quality standards (WQS) in Washington are necessary to satisfy the requirements of the CWA.[1] Specifically, EPA has determined that new and revised aquatic life criteria are necessary to protect against adverse aquatic life impacts related to the following nine pollutants: acrolein, aluminum, arsenic, cadmium, copper, cyanide, mercury, nickel, and selenium. This Determination is made in accordance with a court order directing EPA to determine whether new or revised aquatic life criteria for these nine pollutants are necessary to meet the requirements of the CWA. *Nw. Envtl. Advocates v. EPA*, No. 2:20-cv-1362-MJP, Dkt. 84 (W.D. Wash.).

As explained further below, this Determination is based on EPA's evaluation of available information for these nine pollutants indicating that Washington needs new and revised criteria for these nine pollutants in order to protect Washington's designated uses. EPA has determined that new data and information have become available since Washington last adopted new or revised aquatic life criteria on how these nine pollutants may impact Washington's aquatic life designated uses. New and revised aquatic life criteria for these nine pollutants that account for new data and information will ensure that the State's WQS adequately protect aquatic life in Washington's waters.

EPA appreciates that the Washington State Department of Ecology (Ecology) identified updates to Washington's aquatic life criteria as a priority action in its April 2022 triennial review, and that in June 2022, Ecology announced plans to conduct rulemaking to adopt new or revised aquatic life criteria for certain pollutants.[2] As discussed below, CWA Section 303(c)(4)(B) provides the opportunity for Washington to adopt and submit new and revised aquatic life criteria to EPA prior to EPA taking final action to promulgate any such criteria. Accordingly, EPA encourages Ecology to continue its work to update the aquatic life criteria for Washington.

[1] 33 U.S.C. 1313(c); *see* 40 CFR 131.22(b).
[2] Department of Ecology. April 2022. Triennial Review of Water Quality Standards for Surface Waters of the State of Washington. Publication 22-10-002. https://apps.ecology.wa.gov/publications/documents/2210002.pdf

I. Statutory and Regulatory Background

Under the CWA, states have the primary responsibility for reviewing, establishing, and revising WQS applicable to their waters (CWA Section 303(c)). WQS define the desired condition of a water body, in part, by designating the use or uses to be made of the water (40 CFR 131.2 and 131.10) and by setting the numeric or narrative water quality criteria to protect those uses (40 CFR 131.2 and 131.11). There are two primary categories of water quality criteria: human health criteria and aquatic life criteria. Human health criteria protect designated human uses of a water body, such as public water supply, recreation, and fish and shellfish consumption. Aquatic life criteria protect designated aquatic life uses of a water body, such as survival, growth, and reproduction of fish, invertebrates, and other aquatic species. Regardless of their category, water quality criteria "must be based on sound scientific rationale and must contain sufficient parameters or constituents to protect the designated use. For waters with multiple use designations, the criteria shall support the most sensitive use" (40 CFR 131.11(a)(1)).

Section 304(a) of the CWA directs EPA to periodically develop and publish recommended water quality criteria "accurately reflecting the latest scientific knowledge" on the effects of pollutants on human health and welfare, including effects on aquatic life, as well as information on those pollutants, including their concentration and dispersal and how the pollutants affect receiving waters (CWA Section 304(a)(1)). EPA's Section 304(a) recommendations are one option available to states to use in developing their own water quality criteria (CWA Section 304(a)(3)). When establishing criteria, states should establish numeric criteria based on: (1) EPA's CWA Section 304(a) recommended criteria, (2) modified 304(a) recommended criteria that reflect site-specific conditions, or (3) other scientifically defensible methods (40 CFR 131.11(b)). States can also establish narrative criteria or criteria based on biomonitoring methods where numeric criteria cannot be established or to supplement numeric criteria. *Id.*

CWA Section 303(c)(2)(B), added in the 1987 amendments to the CWA,[3] requires states to adopt numeric criteria, where available, for all toxic pollutants listed pursuant to CWA Section 307(a)(1) (i.e., priority toxic pollutants[4]) for which EPA has published CWA Section 304(a) recommended criteria, the discharge or presence of which could reasonably be expected to interfere with the states' designated uses.

States are required to hold a public hearing to review applicable WQS at least once every three years and, if appropriate, revise or adopt new WQS (CWA Section 303(c)(1); 40 CFR 131.20(a)). This includes adopting criteria for additional toxic pollutants and revising existing toxic pollutant criteria as appropriate based on new information. Any new or revised WQS must be submitted to EPA for review and approval or disapproval (CWA Section 303(c)(2)(A) and (c)(3)). In addition, if a state does not adopt new or revised criteria for parameters for which EPA has published new or updated CWA Section 304(a) criteria recommendations, then the state shall provide an explanation when it submits the results of its triennial review to EPA (CWA Section 303(c)(1); 40 CFR 131.20(a)).

CWA Section 303(c)(4)(B) independently authorizes the Administrator to determine that a new or revised standard is necessary to meet CWA requirements. The authority to make a determination under

[3] Water Quality Act Amendments of 1987, Pub. L. 100-4, 101 Stat. 7.
[4] See 40 CFR part 423, Appendix A – 126 Priority Pollutants.

CWA Section 303(c)(4)(B) is discretionary and resides with the Administrator, unless delegated by the Administrator (40 CFR 131.22(b)). For the purposes of this Determination, the Administrator has delegated this authority to EPA's Assistant Administrator for the Office of Water.

II. Background on Washington's Aquatic Life Criteria and Relevant Litigation

On February 9, 1988, Washington submitted freshwater and marine aquatic life criteria for 26 priority toxic pollutants, which EPA approved on March 4, 1988.[5] At that time, EPA also determined under CWA Section 303(c)(4)(B) that some additional aquatic life criteria were necessary in Washington to comply with CWA Section 303(c)(2)(B) and promulgated aquatic life criteria for Washington in the 1992 National Toxics Rule – acute and chronic freshwater and marine arsenic and selenium criteria, chronic marine copper criteria, and chronic marine cyanide criteria.[6] Following the 1992 National Toxics Rule promulgation, EPA approved new and revised aquatic life criteria for toxic pollutants submitted by Washington on three occasions (1993, 1998, and 2007) and took subsequent actions to withdraw Washington from the National Toxics Rule. As a result of those actions, the only aquatic life criteria applicable in Washington are State-adopted and EPA-approved criteria; Washington is no longer in the National Toxics Rule for aquatic life criteria.[7] Washington's last update to its aquatic life criteria for toxic pollutants was approved by EPA in 2007.

This Determination relates to a 2013 Administrative Procedure Act rulemaking petition from Northwest Environmental Advocates (NWEA) requesting that EPA "update the State of Washington's water quality standards for the protection of . . . aquatic life from toxic contaminants."[8] The petition requested, in pertinent part, that EPA "determine that the State of Washington has failed to adopt such . . . aquatic life criteria as are required by Section 303(c)(2)(B) in each triennial review of its water quality standards conducted since 1992" and that EPA "promulgate new federal regulations applicable to Washington, pursuant to Section 303(c)(4), setting forth new and revised water quality standards as necessary to meet the requirements of the CWA."[9] EPA denied NWEA's petition in 2017, explaining that it was not determining that new or revised criteria were *not* necessary to meet CWA requirements.[10] Rather, in declining to undertake the time and resource-intensive evaluation to determine whether new or revised aquatic criteria were in fact necessary, EPA stated that federal rulemaking authority was not the most effective or practical means of addressing the concerns raised in the petition and that it was exercising its discretion to allocate Agency resources to other regional and national water quality efforts.[11] EPA further explained its strong preference to support states in their development of WQS to protect state waters, rather than to promulgate federal WQS, and noted that Washington's strategic plan identified aquatic life criteria updates as a future action.[12]

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[5] *See* U.S. EPA. (Dec. 22, 1992). *Establishment of Numeric Criteria for Priority Toxic Pollutants*, 57 FR 60848, 60857.
[6] *Id*.
[7] Washington has since been withdrawn from the National Toxics Rule for human health criteria as well (see 40 CFR 131.45).
[8] Northwest Environmental Advocates, Petition for CWA Section 303(c) Determinations and Rulemaking on Washington Water Quality Criteria (Oct. 28, 2013), at 1.
[9] *Id.* at 2.
[10] "Re: Final Response to Petition for Rulemaking on Water Quality Standards for Toxics in the State of Washington." Letter from Michael H. Shapiro, Acting Assistant Administrator for the Office of Water, to Nina Bell, Executive Director Northwest Environmental Advocates (May 31, 2017), at 6.
[11] *Id.*
[12] *Id.* at 1, 3.

In 2020, NWEA filed a Complaint in the Western District of Washington challenging EPA's denial of its petition.[13] In the ensuing litigation, the District Court found that EPA's denial was arbitrary and capricious, vacated that denial, and initially remanded the petition back to EPA "to make a necessity determination" pursuant to the petition, which covered numerous pollutants beyond the nine subject to this Determination.[14] On August 30, 2022, the court issued a modified order directing EPA to grant NWEA's petition with respect to only nine pollutants: acrolein, aluminum, arsenic, cadmium, copper, cyanide, mercury, nickel, and selenium, no later than September 1, 2022.[15] The order further provided that EPA would make an Administrator's Determination with respect to the nine pollutants no later than June 1, 2023.[16]

On August 30, 2022, EPA granted the petition for the nine pollutants specified in the court's order. In its letter to NWEA, EPA explained that it based its decision to grant the petition on the potential on-the-ground environmental impact of discharges of these pollutants into Washington waters and an initial review of readily available data.[17] By granting the petition for these nine pollutants, EPA agreed to evaluate whether new or revised criteria were necessary for these pollutants. EPA is now issuing an Administrator's Determination for these pollutants consistent with the court's modified order.[18]

III. Washington's Current Aquatic Life Criteria Do Not Protect Washington's Designated Uses With Respect to These Pollutants

Washington has CWA-effective aquatic life criteria for seven of the nine pollutants for which EPA granted the petition to evaluate whether new or revised criteria are necessary (arsenic, cadmium, copper, cyanide, mercury, nickel, and selenium). These pollutants are each naturally occurring but may also be found in aquatic systems as a result of anthropogenic sources.[19] For the remaining two pollutants – acrolein and aluminum – available data and information suggest that those pollutants are present in Washington's waters and can reasonably be expected to interfere with Washington's aquatic life designated uses. Since Washington does not currently have aquatic life criteria for acrolein or aluminum, these two pollutants are less likely than the others to be captured in a review of Washington's water

[13] *Nw. Envtl. Advocates v. EPA*, No. 2:20-cv-1362-MJP, Dkt. 1 (W.D. Wash.).
[14] *Id.* at Dkt. 57 p. 22; *id.* at Dkt. 72 p. 3-4 (noting the "numerous toxic pollutants" covered by the petition, including a dozen banned chemicals).
[15] *Id.*
[16] *Id.*
[17] "Re: Revised Response to Petition for Rulemaking on Water Quality Criteria for Toxics in the State of Washington." Letter from Radhika Fox, Assistant Administrator for the Office of Water, to Nina Bell, Executive Director Northwest Environmental Advocates (August 30, 2022).
[18] *Id*.
[19] *See* US EPA 2022. TRI Explorer (2020 National Analysis Dataset (October 2021, released October 2021)) [Internet database]. Accessed January 26, 2023. Retrieved from https://enviro.epa.gov/triexplorer/tri_release.chemical (indicating releases of arsenic, copper, mercury, and nickel compounds in Washington); Washington State Department of Ecology. n.d. Washington State Water Quality Assessment 303(d)/305(b) List [Internet Database]. Accessed January 26, 2023. Retrieved from https://apps.ecology.wa.gov/ApprovedWQA/ApprovedPages/ApprovedSearch.aspx (indicating that certain waters in Washington are impaired due to arsenic, cadmium, copper, mercury, and nickel); Department of Ecology 2016. Final Cost-Benefit and Least Burdensome Alternative Analyses. Chapter 173-201A WQC Water Quality Standards for Surface Waters of the State of Washington. Publication no. 16-10-019, at 25-27 (Washington's 2016 permit and effluent review indicates arsenic, copper, cyanide, mercury, nickel, and selenium are among the five most detected chemicals across various types of municipal and industrial facilities in Washington).

quality assessments and data from permitted dischargers.[20] Therefore, EPA evaluated other data and information to examine if these pollutants may be present in Washington's surface waters.

Acrolein is an aquatic herbicide often used in irrigation canals to control for weeds and algae. Washington's "Irrigation System Aquatic Weed Control" general permit (both the existing permit[21] and the draft permit reissuance[22]) lists acrolein as a permitted pollutant. The general permit "conditionally authorizes the use" of acrolein and includes mention of application plans, monitoring requirements, and a maximum concentration at the point of compliance.[23] Aluminum is found in most rocks and soils and can enter surface water through weathering and erosion of rock.[24] Given the natural abundance of aluminum, it is highly likely that the element is already present in Washington's surface waters. Additionally, as discussed further below, in its April 14, 2022, triennial review report,[25] Ecology indicated that it would consider future adoption of aquatic life criteria, including acrolein, aluminum, arsenic, cadmium, copper, mercury, nickel, and selenium (among other pollutants).

After reviewing the evidence indicating that aquatic life in Washington may be exposed to all nine toxic pollutants subject to this Determination, EPA relied primarily on two main sources of available information to assess whether Washington needs new or revised aquatic life criteria for those nine toxic pollutants to protect applicable aquatic life designated uses.[26] First, EPA compared Washington's existing criteria to EPA's CWA Section 304(a) national recommended criteria. Second, EPA evaluated whether recent Endangered Species Act (ESA) consultations for relevant species in neighboring states support a conclusion that new or revised criteria might be necessary to protect Washington's aquatic life designated uses, which include threatened and endangered species listed under the ESA.

Washington's Existing Aquatic Life Criteria Compared to EPA's CWA Section 304(a) National Recommended Criteria

As noted above, Washington has existing aquatic life criteria for seven of the nine pollutants subject to this Determination (arsenic, cadmium, copper, cyanide, mercury, nickel, and selenium) and does not have aquatic life criteria for the remaining two pollutants (acrolein and aluminum). EPA has published national recommended criteria for all nine pollutants under CWA Section 304(a). EPA periodically updates the national recommended criteria as new science and data become available. Of the nine pollutants relevant to this Determination, EPA has published updates to five of the corresponding CWA Section 304(a) national recommended criteria in the past 14 years. Table 1 provides a list of each of the

[20] Washington does, however, have human health criteria for acrolein, and has used aquatic life criteria from the neighboring State of Oregon to derive specific permit limits, when appropriate. *See:* https://fortress.wa.gov/ecy/ezshare/wq/permits/ISAWC-FactSheetforDraftPermit.pdf

[21] Existing general permit as of May 2023: Department of Ecology. Irrigation System Aquatic Weed Control. National Pollutant Discharge Elimination System (NPDES) and State Waste Discharge General Permit WA0991000. May 16, 2012. Accessed January 30, 2023. https://ecology.wa.gov/DOE/files/6b/6b9e466a-139b-4fdb-834c-2b1262cf25c0.pdf

[22] Draft general permit as of May 2023: Department of Ecology. Irrigation System Aquatic Weed Control General Permit. National Pollutant Discharge Elimination System and State Waste Discharge General Permit. n.d. Accessed January 30, 2023. https://fortress.wa.gov/ecy/ezshare/wq/permits/ISAWC-GeneralPermit-Draft.pdf

[23] Department of Ecology. Draft permit page 18. Existing permit page 6.

[24] US EPA. 2022. Aquatic Life Criteria – Aluminum. https://www.epa.gov/wqc/aquatic-life-criteria-aluminum

[25] Department of Ecology. April 2022. Triennial Review of Water Quality Standards for Surface Waters of the State of Washington. Publication 22-10-002. https://apps.ecology.wa.gov/publications/documents/2210002.pdf

[26] EPA notes that the analysis conducted to support this Determination is specific to these pollutants in Washington waters and is based on readily available information.

nine pollutants, the most recent EPA publication of CWA Section 304(a) national recommended criteria for that pollutant, and the year in which Washington most recently adopted or updated criteria for that pollutant.

Table 1 – History of Washington Criteria Adoption and EPA's National Recommended Criteria for Toxic Pollutants Relevant to this Determination.

Pollutant	Latest Update by EPA	Latest Update by Washington
Acrolein	2009	None
Aluminum	2018	None
Arsenic	1995	1992
Cadmium	2016	1997
Copper	2007	1997
Cyanide	1985	2003
Mercury	1995	1997
Nickel	1995	1997
Selenium	2016	1997

States are required to adopt criteria "that protect the designated use . . . based on sound scientific rationale." 40 CFR 131.11(a)(1). EPA's regulations also provide that states should adopt criteria based on EPA's 304(a) national recommended criteria, the 304(a) recommended criteria modified to reflect site-specific conditions, or other scientifically defensible methods. 40 CFR 131.11(b)(1). Updates to EPA's CWA Section 304(a) national recommended criteria reflect the latest scientific knowledge on the effects of those pollutants on aquatic life.[27]

New scientific information has been developed since Washington's adoption of its currently effective aquatic life criteria and that information is reflected in EPA's latest 304(a) national recommended criteria. Nonetheless, as explained further below, for some of the nine pollutants, Washington's criteria are not based on EPA's latest 304(a) criteria, nor are they based on modifications of EPA's 304(a) criteria to reflect site-specific conditions or other scientifically defensible methods. See 40 CFR 131.11(b)(1). For others, the State's criteria are based on recommendations that EPA is updating due to advances in the relevant science.

For acrolein and aluminum, Washington lacks any aquatic life criteria, despite recent updates to EPA's 304(a) national recommended criteria and evidence that those pollutants are present in Washington's waters. EPA's most recent updates to the CWA Section 304(a) national recommended criteria for cadmium, copper and selenium rely on the best available science and supersede prior recommendations for these chemicals. Washington's criteria for the remaining four pollutants – mercury, nickel, cyanide, and arsenic – are based on 304(a) criteria recommendations that the agency is in the process of updating based on the best available science. For mercury and nickel, Washington's current criteria are based on EPA's 1995 304(a) recommendations. EPA is currently evaluating data on mercury toxicity from dietary

[27] Section 304(a)(1) directs EPA to publish criteria "accurately reflecting the latest scientific knowledge[.]" EPA's water quality criteria published under Section 304(a)(1) of the CWA are not legally binding requirements, but rather serve as recommendations for states.

exposures for the purpose of developing protective mercury criteria for the State of Idaho and anticipates the Idaho work will help inform a future update to the CWA Section 304(a) national recommendation for mercury.[28] Nickel is one of the metals currently being studied as part of EPA's Cooperative Research and Development Agreement (CRADA).[29] As part of the CRADA, EPA plans to update the modeling approach for nickel criteria derivation and subsequently develop updated CWA Section 304(a) national recommended criteria for nickel. EPA is similarly in the process of evaluating the best available science regarding the impacts of cyanide and arsenic to aquatic species. Although EPA has not yet completed updates to these national criteria recommendations, as explained below, the agency has evidence indicating that Washington's existing criteria for these four pollutants are not protective of aquatic life designated uses in Washington's waters based on ESA consultations completed in neighboring Pacific Northwest states.

Review of Endangered Species Act Consultations for Relevant Species in Neighboring States

EPA evaluated data and information compiled in recent ESA Section 7 consultations with the National Marine Fisheries Service (NMFS) and U.S. Fish and Wildlife Service (FWS) – the agencies for determining jeopardy under the ESA – regarding EPA actions on aquatic life criteria in neighboring states (Oregon and Idaho).[30] If NMFS and/or FWS find that a criterion would likely jeopardize the continued existence of an ESA-listed species or cause an adverse modification of critical habitat, that is a factor that EPA may consider in evaluating whether new or revised criteria are necessary to protect the applicable designated uses. While aquatic conditions and species vary within and between states, several species reside in or travel between multiple states and evaluations of the potential effects of a certain pollutant on a species in one state may be scientifically relevant to how the same pollutant can affect the same species in a neighboring state. For example, in the Pacific Northwest, numerous salmonid species travel within and between Oregon, Idaho, and Washington. Therefore, it is technically appropriate to evaluate ESA consultations for criteria in neighboring states to inform whether the same or less stringent[31] aquatic life criteria in Washington could reasonably be expected to impact the same species or interfere with other aquatic life designated uses.

When reviewing the results of relevant ESA consultations, EPA evaluated whether NMFS or FWS concluded that a criterion in Oregon or Idaho would jeopardize a threatened or endangered species, or cause adverse modification of critical habitat, that is also present in Washington and is thus covered by Washington's aquatic life use, and whether that criterion was equal to or more stringent than Washington's existing aquatic life criterion for the pollutant. In 2012, NMFS concluded that EPA's proposed approval of Oregon's freshwater acute cadmium criterion, freshwater acute and chronic copper

[28] *See Nw. Envtl. Advocates v. EPA*, No. 1:13-cv-00263-DCN, Dkt. 119 (D. Id.).
[29] US EPA. "Aquatic Life Criteria and Methods for Toxics." February 7, 2023. https://www.epa.gov/wqc/aquatic-life-criteria-and-methods-toxics. Accessed March 3, 2023.
[30] Under Section 7 of the ESA, Federal agencies must consult with either FWS and/or NMFS, depending on the species at issue, to insure that any action the agency carries out, funds, or authorizes is not likely to jeopardize the continued existence of any ESA-listed species or result in the destruction or adverse modification of critical habitat.
[31] EPA notes that stringency alone does not dictate whether a criterion is or is not protective. As science advances, it may reveal that a criterion less stringent than the previously adopted criterion (or 304(a) recommendation) is protective of the applicable designated use. However, for the purposes of this Determination, EPA is using stringency as a surrogate metric because the data and information indicating that more stringent criteria are necessary to protect Washington's aquatic life uses all post-dated Washington's most recent update to its aquatic life criteria and therefore Washington could not have considered those data and information when concluding that their less stringent criteria are protective.

criteria, and freshwater acute and chronic aluminum criteria would jeopardize the continued existence of several salmonids, green sturgeon, eulachon, and Southern Resident killer whales.[32] These species are also present in Washington, and Washington's corresponding criteria for cadmium and copper are higher (less stringent) than the values EPA was proposing to approve in Oregon. Washington lacks aquatic life criteria for aluminum. In 2014 and 2015 respectively, NMFS[33] and FWS[34] found that EPA's approval of Idaho's freshwater chronic arsenic criterion, freshwater acute and chronic copper criteria, freshwater acute and chronic cyanide criteria, freshwater chronic mercury criterion, and freshwater chronic selenium criterion would jeopardize several salmonids. These species are also present in Washington, and Washington's corresponding criteria for arsenic, copper, cyanide, mercury, and selenium are higher (less stringent) than the values in Idaho. In summary, Washington's criteria for these pollutants are equal to or less stringent than criteria that NMFS and FWS found would likely jeopardize the survival of certain species in Oregon and Idaho that are also present in Washington. Here, in EPA's view, this indicates these criteria are not protective of Washington's aquatic life designated uses.

IV. Clean Water Act Section 303(c)(4)(B) Determination

EPA has reviewed available information regarding (1) how Washington's existing criteria (or lack thereof) for nine pollutants – acrolein, aluminum, arsenic, cadmium, copper, cyanide, mercury, nickel, and selenium – compare to EPA's CWA 304(a) national recommended criteria that reflect updated science, and (2) whether Washington's existing criteria for those pollutants protect aquatic life designated uses. EPA has concluded that Washington's existing aquatic life criteria for arsenic, cadmium, copper, cyanide, mercury, nickel, and selenium are not protective of the applicable designated uses and based on sound scientific rationale, as required by EPA's regulation at 40 CFR 131.11, and that Washington lacks aquatic life criteria for acrolein and aluminum where available information indicates that Washington needs criteria for those pollutants to protect applicable designated uses.

Accordingly, EPA is determining, pursuant to CWA Section 303(c)(4)(B) and 40 CFR 131.22(b), that new aquatic life criteria are needed for acrolein and aluminum, and revised aquatic life criteria are needed for arsenic, cadmium, copper, cyanide, mercury, nickel, and selenium to meet the requirements of the CWA for Washington.

V. Washington's Current Efforts to Update its Aquatic Life Criteria

On April 14, 2022, Ecology submitted to EPA its triennial review report for Chapter 173-201A of the Washington Administrative Code (WAC) for WQS for surface waters of the State.[35] The triennial review report evaluated EPA's aquatic life 304(a) national criteria recommendations and the aquatic life criteria currently in effect for CWA purposes in Washington's WQS. For each of EPA's 304(a) criteria

[32] National Marine Fisheries Service. August 14, 2012. 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. p. 536, 547.
[33] National Marine Fisheries Service. May 7, 2014. Final Endangered Species Act Section 7 Formal Consultation and Magnuson-Stevens Fishery Conservation and Management Act Essential Fish Habitat Consultation for Water Quality Toxics Standards for Idaho. p. 297
[34] Fish and Wildlife Service. June 25, 2015. Biological Opinion for the Idaho Water Quality Standards for Numeric Water Quality Criteria for Toxic Pollutants. p. 258
[35] Department of Ecology. April 2022. Triennial Review of Water Quality Standards for Surface Waters of the State of Washington. Publication 22-10-002. https://apps.ecology.wa.gov/publications/documents/2210002.pdf

recommendations, Washington made one of three determinations: *Future Action*, *Already Addressed*, or *Not Scheduled for Adoption*. *Future Action* indicates that Ecology will consider adoption of EPA's 304(a) criteria recommendations in upcoming rulemaking efforts. *Already Addressed* indicates that the currently adopted criteria in Washington's standards are either equal to or are more stringent than EPA's 304(a) national recommendations and therefore Washington does not intend to prioritize revisions to those criteria. *Not Scheduled for Adoption* indicates that Ecology does not intend to update these criteria in the near future for other reasons, despite any lack of alignment between those criteria and EPA's CWA Section 304(a) recommendations. Of the nine pollutants in this Determination, Ecology categorized eight (acrolein, aluminum, arsenic, cadmium, copper, mercury, nickel, and selenium) for *Future Action*. Ecology categorized cyanide as *Already Addressed* because Washington's existing statewide criteria are consistent with EPA's existing 304(a) national recommendations.

On June 23, 2022, Ecology announced its plans to move forward with a rulemaking to amend WAC 173-201A-240, toxic substances, specifically the aquatic life criteria.[36] EPA appreciates Washington's ongoing commitment to updating its aquatic life criteria.

This Determination does not preclude Washington from proceeding with its own rulemaking effort, and EPA encourages Washington to continue its work to update and adopt aquatic life criteria for toxic pollutants. Nevertheless, CWA Section 303(c)(4) requires that the Administrator promptly prepare and publish proposed regulations setting forth new or revised WQS following a Determination. However, if Washington adopts, and EPA approves, new or revised WQS that meet the requirements of the CWA before EPA proposes or promulgates federal WQS, then EPA would no longer be obligated to propose or promulgate those federal WQS.

VI. Next Steps

Following this Determination, the next step is for EPA to propose new and revised aquatic life criteria for these nine pollutants. For some of the nine pollutants, EPA's existing CWA Section 304(a) national recommended criteria are likely appropriate for proposal in Washington. However, for other pollutants in this Determination, EPA is still in the process of evaluating the latest science available – as well as Washington-specific information on surface water conditions and the presence of sensitive aquatic organisms, where applicable – to derive aquatic life criteria for Washington which are protective of designated uses and based on sound scientific rationale. After these analyses are completed, EPA will then develop proposed federal regulations setting forth such criteria for Washington. EPA will seek feedback from Washington, as well as interested stakeholders, on EPA's proposed rulemaking(s) in accordance with 40 CFR 131.22(c) and 131.20(b). After any federal rule is proposed, EPA plans to give full consideration to all comments received before proceeding to the final rule stage. As indicated above, CWA Section 303(c)(4)(B) provides the opportunity for Washington to adopt and submit new and revised aquatic life criteria to EPA prior to EPA taking final action to promulgate any such criteria. Accordingly, EPA encourages Washington to continue its work to update its aquatic life criteria.

[36] Department of Ecology. Chapter 173-201A WAC (Aquatic Life Toxics Criteria). Webpage. Accessed February 1, 2023. https://ecology.wa.gov/Regulations-Permits/Laws-rules-rulemaking/Rulemaking/WAC-173-201A-Aquatic-Life-Toxics-Criteria

EPA is committed to working closely and collaboratively with Washington to ensure that its aquatic life criteria are protective of applicable designated uses, based on sound scientific rationale, and consistent with the requirements of the CWA.

Sincerely,

Radhika Fox
Assistant Administrator

cc:

Casey Sixkiller, Regional Administrator, EPA Region 10
Dan Opalski, Director, Water Division, EPA Region 10