A Toxics-focused Biological Observing System (T-BiOS)

Washington Department of FISH and WILDLIFE

Mission Statement:

Evaluate the effects of toxic contaminants on marine and anadromous species to:

- ***guide efforts to protect fish and shellfish health,***
- ***ensure seafood safety (supply data to DOH), and***
- ***promote ecosystem recovery.***

WDFW photo

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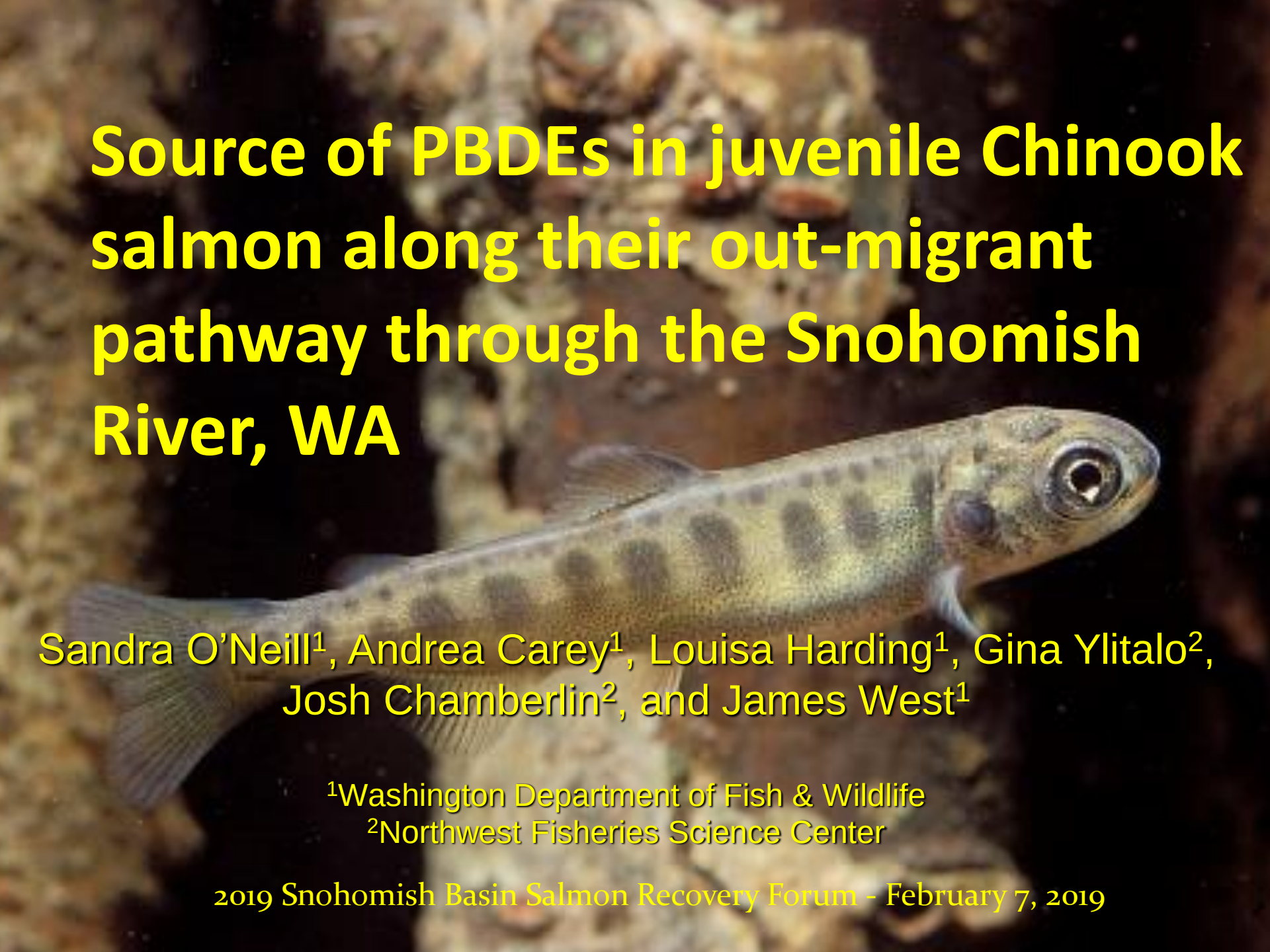

Source of PBDEs in juvenile Chinook salmon along their out-migrant pathway through the Snohomish River, WA

Sandra O'Neill[1], Andrea Carey[1], Louisa Harding[1], Gina Ylitalo[2], Josh Chamberlin[2], and James West[1]

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2019 Snohomish Basin Salmon Recovery Forum - February 7, 2019

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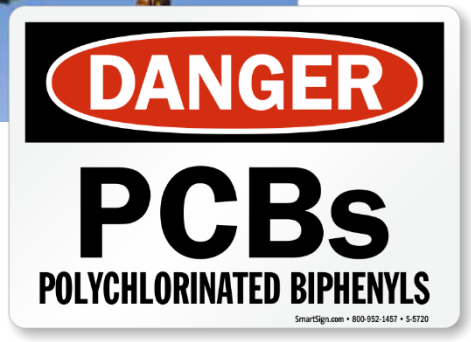
David Baldwin
Lyndal Johnson

Talk Outline

- Background – review results of previous studies
- 2016 Snohomish Survey Design
- Results- data types to investigate PBDE “source”
  - PBDE concentrations – where exposure occurs
  - Contaminant Fingerprints – wastewater vs. stormwater source
  - Stable Isotopes – altered nitrogen source – wastewater?
- Conclusions
- Next Steps

Background

Persistent Organic Pollutants (POPs)



Background

Effects of PBDEs on juvenile salmon are evaluated by laboratory exposure studies conducted by Arkoosh et al. 2010, 2018

Aquatic Toxicology 98 (2010) 51–59

Contents lists available at ScienceDirect

Aquatic Toxicology

journal homepage: www.elsevier.com/locate/aquatox

Disease susceptibility of salmon exposed to polybrominated diphenyl ethers (PBDEs)

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ABSTRACT

The health effects of the flame retardant polybrominated diphenyl ethers (PBDEs) in fish are not well understood. To determine the potential effects of this ubiquitous contaminant class on fish health, juvenile subyearling Chinook salmon (*Oncorhynchus tshawytscha*) were fed a diet that reflected the PBDE congeners found in the stomach contents of subyearling Chinook salmon collected from the highly urbanized and industrialized lower Willamette River in the Columbia River Basin of North America. The diet, consisting of five PBDE congeners (BDE-47, BDE-99, BDE-100, BDE-153 and BDE-154), was fed to the salmon at 2% of their body weight in food per day for 40 days. Two concentrations of the diet (1× and 10× PBDE) were fed to the salmon. The 1× PBDE diet reflected the concentration of PBDEs (190 ng PBDEs/g food) found in the stomach contents of juvenile subyearling Chinook salmon; the 10× diet was prepared at 10 times that concentration. The fish were then exposed to the marine bacterial pathogen *Listonella anguillarum* to assess susceptibility to infectious disease. Juvenile Chinook salmon fed the 1× PBDE diet were more susceptible to *L. anguillarum* than salmon fed the control diet. This suggests that juvenile salmonids in the lower Willamette River exposed to PBDEs may be at greater risk for disease than nonexposed juvenile salmonids. In contrast, salmon that consumed the 10× PBDE diet were not more susceptible to the pathogen than salmon fed the control diet. The mechanisms for the dichotomous results observed in disease susceptibility between salmon fed the 1× and 10× PBDE diets are currently not known but have also been observed in other species exposed to PBDEs with respect to immune function.

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1. Introduction

Activities that degrade the habitat and result in ecosystems that are contaminated with persistent organic pollutants (POPs) can influence the health of endangered salmonid populations (Loge et al., 2005; Spromberg and Meador, 2005). Earlier studies have determined that juvenile subyearling Chinook salmon (*Oncorhynchus tshawytscha*) are exposed to varying levels of the following legacy and nonlegacy organic pollutants in various industrialized waterways (McCain et al., 1990; Johnson et al., 2007a, 2007b; Sloan et al., 2010): polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), organochlorine pesticides (OCP) such as DDTs (dichloro-diphenyl-trichloroethane), and polybrominated diphenyl ethers (PBDEs). Juvenile subyearling Chinook salmon in industrialized waterways exposed to relatively high concentrations of ΣPCBs and ΣPAHs have been found to be immunosuppressed and more susceptible to infectious disease, as well as having reduced growth and survival compared to fish not exposed to organic pollutants (Arkoosh et al., 1991, 1998; Yaramasi et al., 1993). Therefore, contaminant exposure has the potential to alter the ability of salmon to respond to infectious agents as well as specifically altering immune function.

Recently, a monitoring study determined that juvenile subyearling Chinook salmon from the heavily urbanized and industrialized lower Willamette River in the Columbia River Basin in North America are exposed to the emerging contaminant PBDE (Sloan et al., 2010). PBDEs are a class of flame retardants that are added to a number of household and commercial items including televisions, computers, electronic equipment, and furniture (de Wit, 2002). These compounds have been measured in air, water, fish, birds, marine mammals, and humans (Hites, 2004), with levels in the environment that have been increasing over time (Koizumi et al.,

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Dietary exposure to a binary mixture of polybrominated diphenyl ethers alters innate immunity and disease susceptibility in juvenile Chinook salmon (*Oncorhynchus tshawytscha*)

Mary R. Arkoosh[a,*], Ahna L. Van Gaest[b], Stacy A. Strickland[b], Greg P. Hutchinson[b], Alex B. Krupkin[b], Mary Beth Rew Hicks[c], Joseph P. Dietrich[a]

ARTICLE INFO

Keywords:
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ABSTRACT

Polybrominated diphenyl ethers (PBDEs) have been used as flame retardants in consumer products and are now found in the aquatic environment. The presence of PBDEs puts the health and survival of aquatic species at risk due to the various toxic effects associated with exposure to these compounds. The effects of a binary dietary mixture of PBDEs on innate immunity and disease susceptibility of juvenile Chinook salmon (*Oncorhynchus tshawytscha*) were examined in the present study. Salmon were fed roughly 1:1 mixtures of two environmentally predominant PBDE congeners, BDE-47 and BDE-99. The six resulting whole body total PBDE concentrations ranged from less than the limit of quantification to 184 ng/g, wet weight (ww). The innate immune system was assessed by using two in vitro macrophage function assays. Specifically, assays that examined the ability of head kidney macrophages to: (1) engulf sheep red blood cells (SRBCs); and (2) produce a respiratory burst, as determined by the production of a reactive oxygen species, superoxide anion. Macrophages from salmon fed the BDE-47/99 mixture diets engulfed more SRBCs and produced greater superoxide anion than salmon fed the control diet. An increase in macrophage function was observed in fish with whole body total PBDE concentrations ranging from 2.81 ng/g, ww to 18.4 ng/g, ww. The mechanism for this increase in macrophage function due to PBDE exposure is currently unknown, but may be due to the ability of PBDEs to act as an endocrine receptor agonist and/or antagonist. Salmon exposed to the BDE-47/99 mixture diets were also challenged with the pathogenic bacteria, *Vibrio* (*Listonella*) *anguillarum* to determine disease susceptibility. Kaplan-Meier survival curves of fish exposed to the BDE-47/99 mixture and control diets were significantly different. The Cox proportional hazard risk ratios of disease-induced mortality in juvenile Chinook salmon with whole body concentrations of total PBDEs of 10.9, 36.8, and 184 ng/g, ww were significantly greater than the fish fed the control diet by 1.56, 1.83 and 1.50 times, respectively. Not all concentrations of the binary mixture diets had significant hazard ratios relative to the control diet, due to a non-monotonic concentration response curve. The mixture of PBDE congeners resulted in interactive effects that were generally non-additive and dependent upon the congener concentrations and metric examined. Consequently, predicting the interactive effects in juvenile Chinook salmon exposed to mixtures of PBDE congeners on innate immunity and disease susceptibility cannot be readily determined from the adverse effects of individual PBDE congeners.

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Salmon with elevated PBDEs have increased susceptibility to disease and altered thyroid function.

Background

PBDEs in Snohomish Chinook at levels high enough to increase their susceptibility to disease and alter thyroid function

Sloan et a. 2010

- 2006 study
- Snohomish plus Skagit, Duwamish, Elliott Bay, Columbia River
- PBDEs highest in salmon from Snohomish and 3 of 6 sites in Columbia River

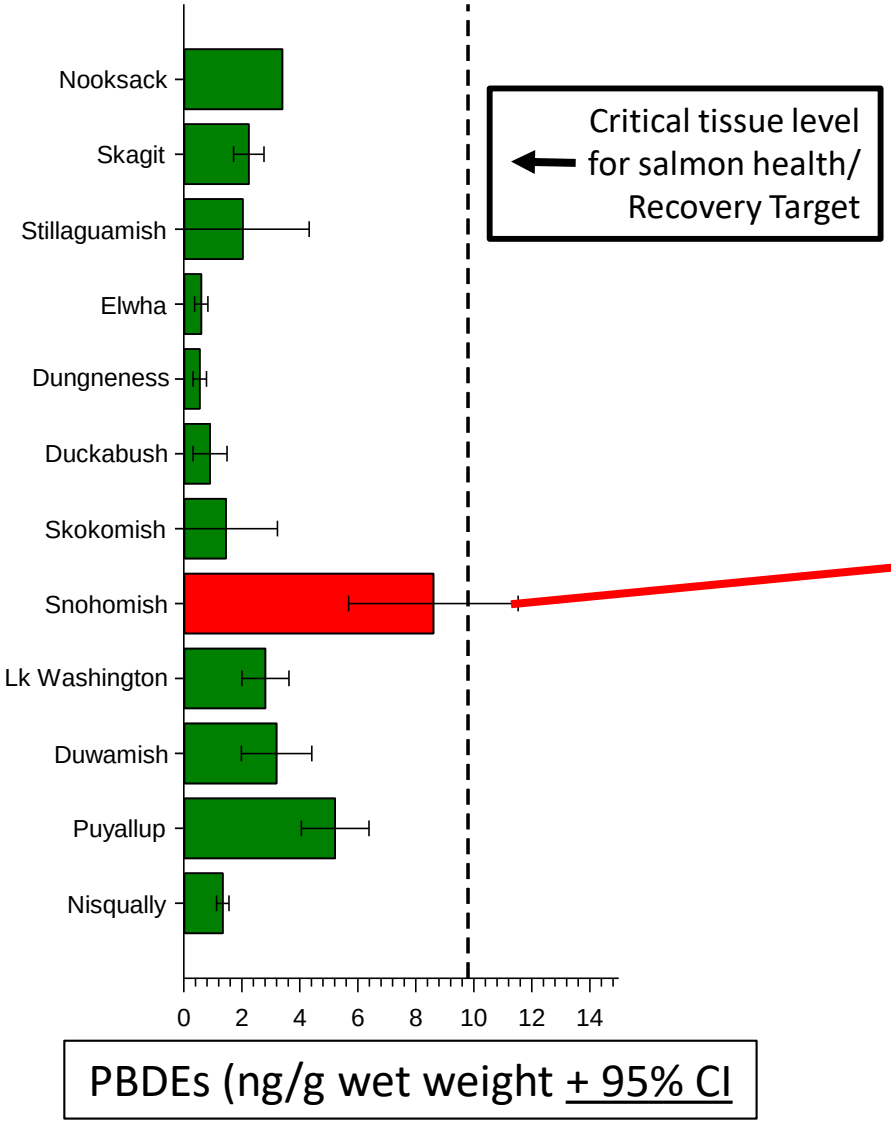
Sloan et al. 2010. Arch Environ Contam Toxicol 58:403–414

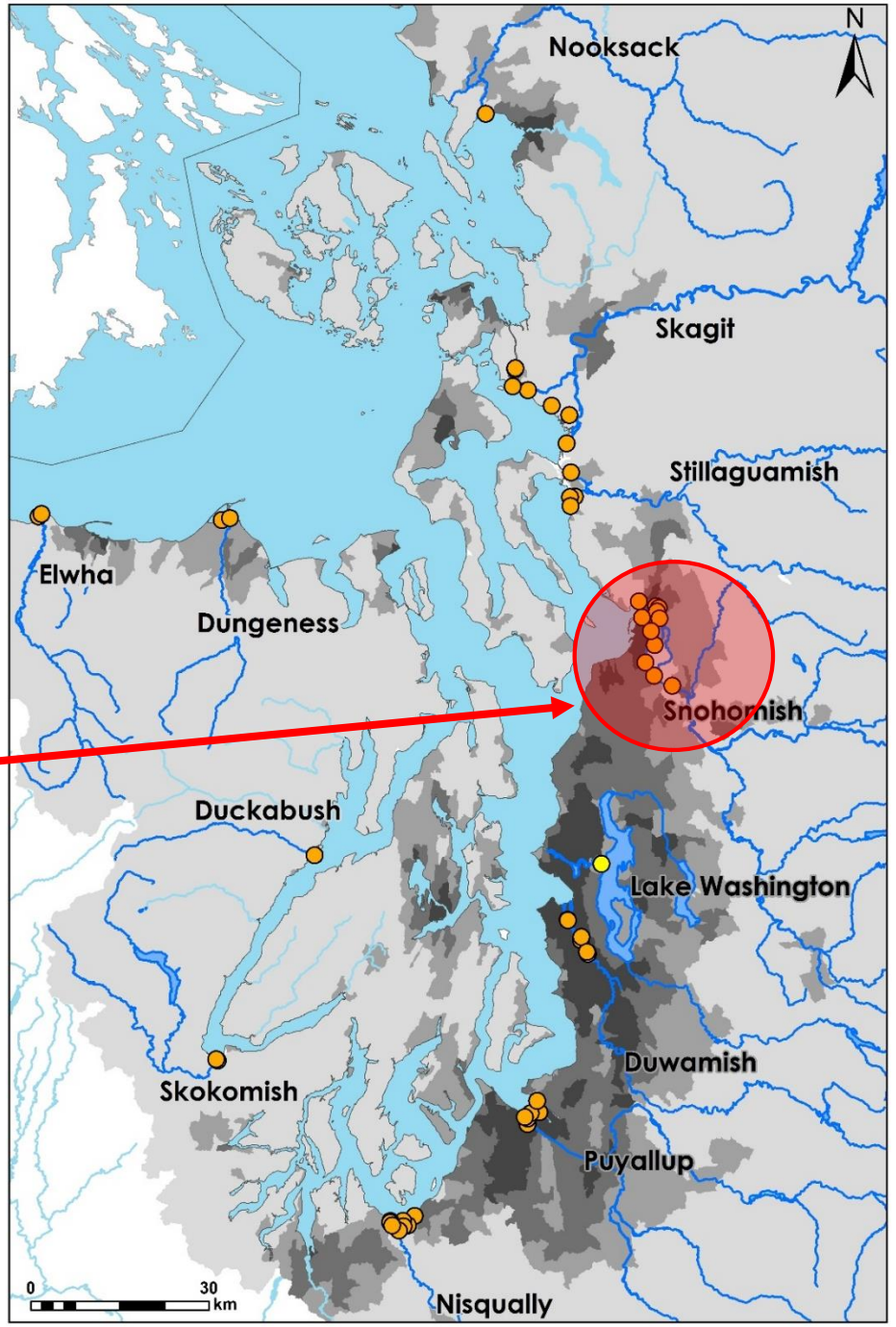
O'Neill et al. 2015

- 2013
- Snohomish plus Skagit, Duwamish, Comm. Bay, Nisqually
- PBDEs highest in fish from Snohomish

O'Neill et al. 2015. WDFW Report No FTP 16-02, 116 pages

High PBDEs in Snohomish Chinook Salmon





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  - Stable Isotopes – altered nitrogen source – wastewater?
- Conclusions
- Next Steps

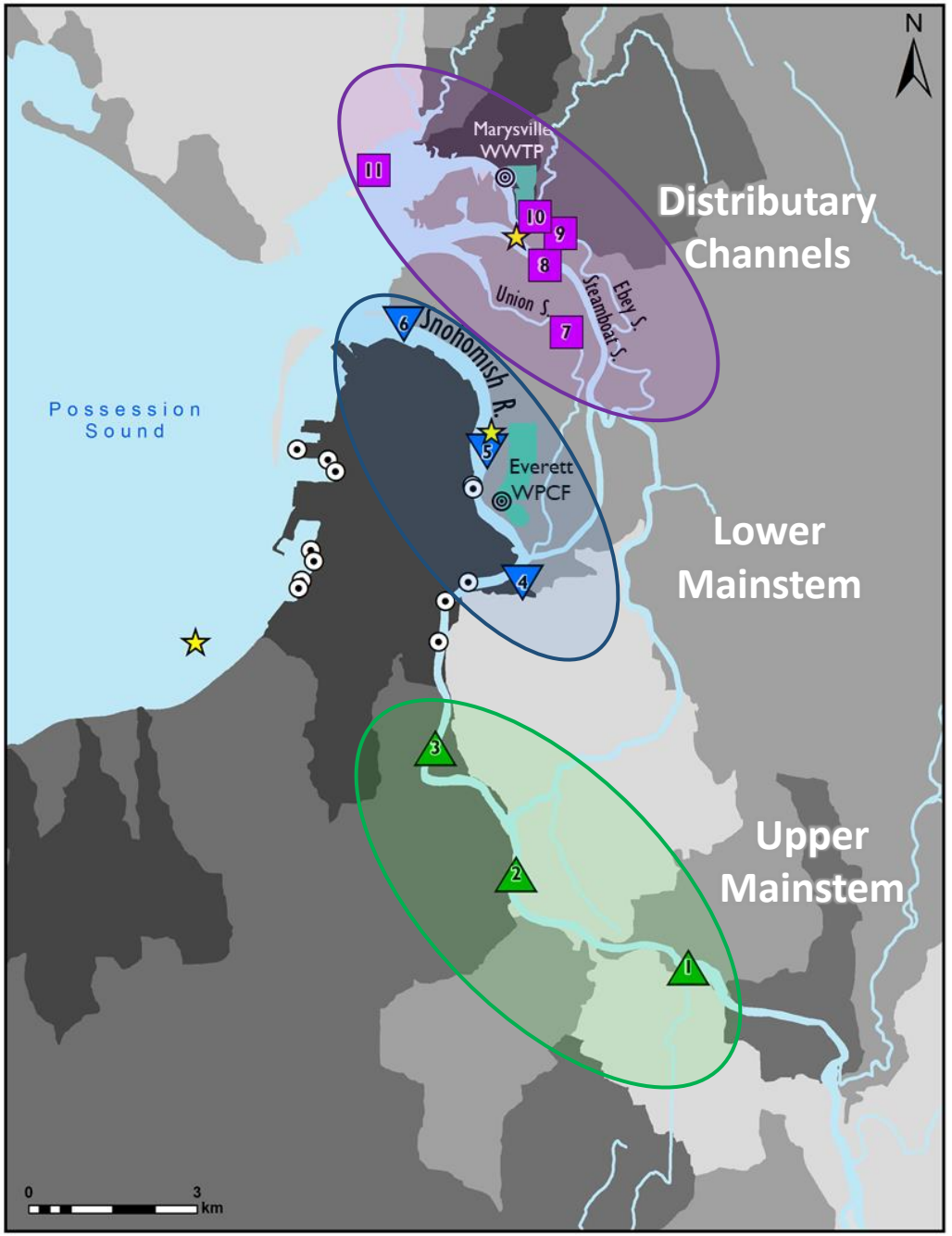
Snohomish River 2016 Study

Where are juvenile Chinook salmon exposed to and accumulating PBDEs?

- Upper Mainstem
- Lower Mainstem
- Distributary Channels

What is the "source" of PBDE inputs?

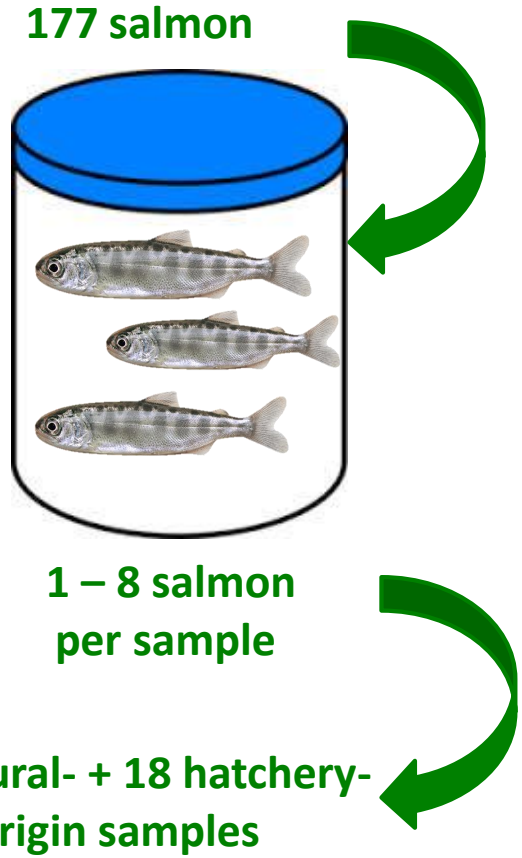
- wastewater (WWTP effluent, CSOs?)
- stormwater (storm drains, CSO, etc.?)



Snohomish River 2016 Study

Types of information collected for juvenile Chinook salmon

- Fish length, weight, origin, age, life history
- **Concentrations** of PBDEs, PCBs, DDTs
  - ✓ indicates where exposure occurs
- Contaminant **fingerprints**
  - ✓ indicates changes in contaminant source
- **Stable isotopes** of nitrogen
  - ✓ Indicates changes in nitrogen source



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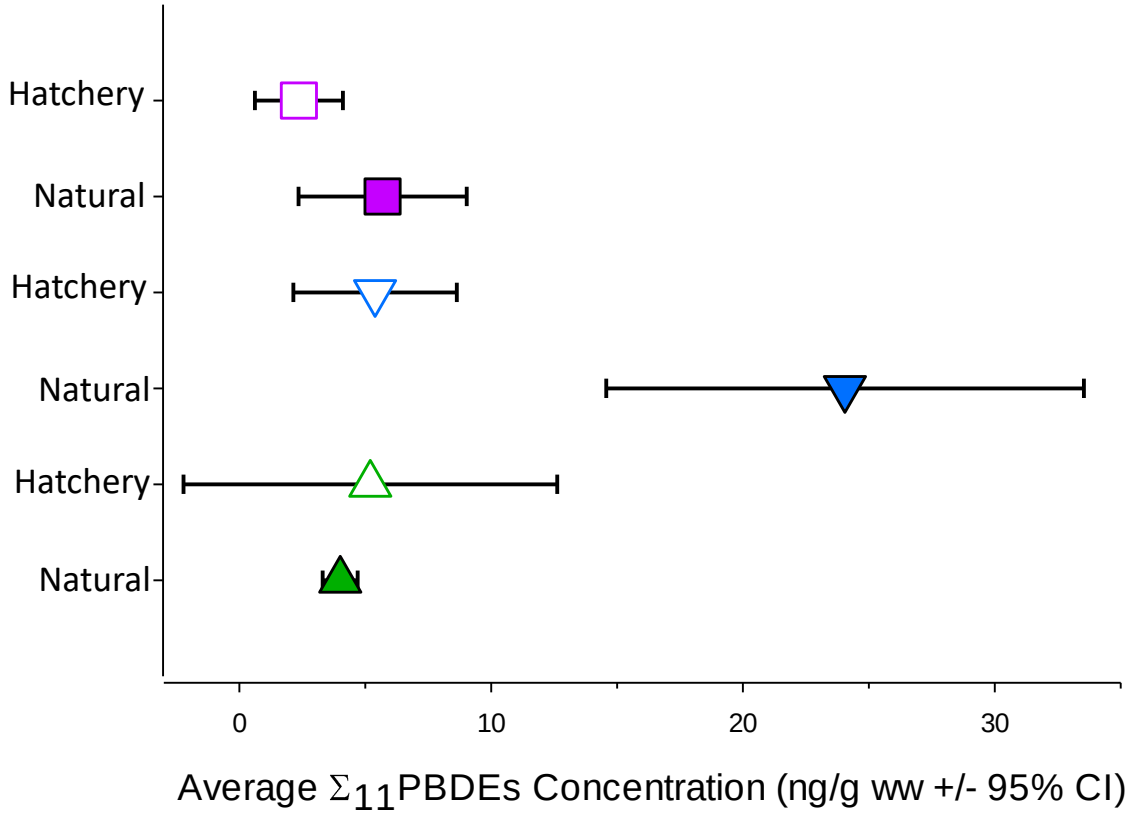
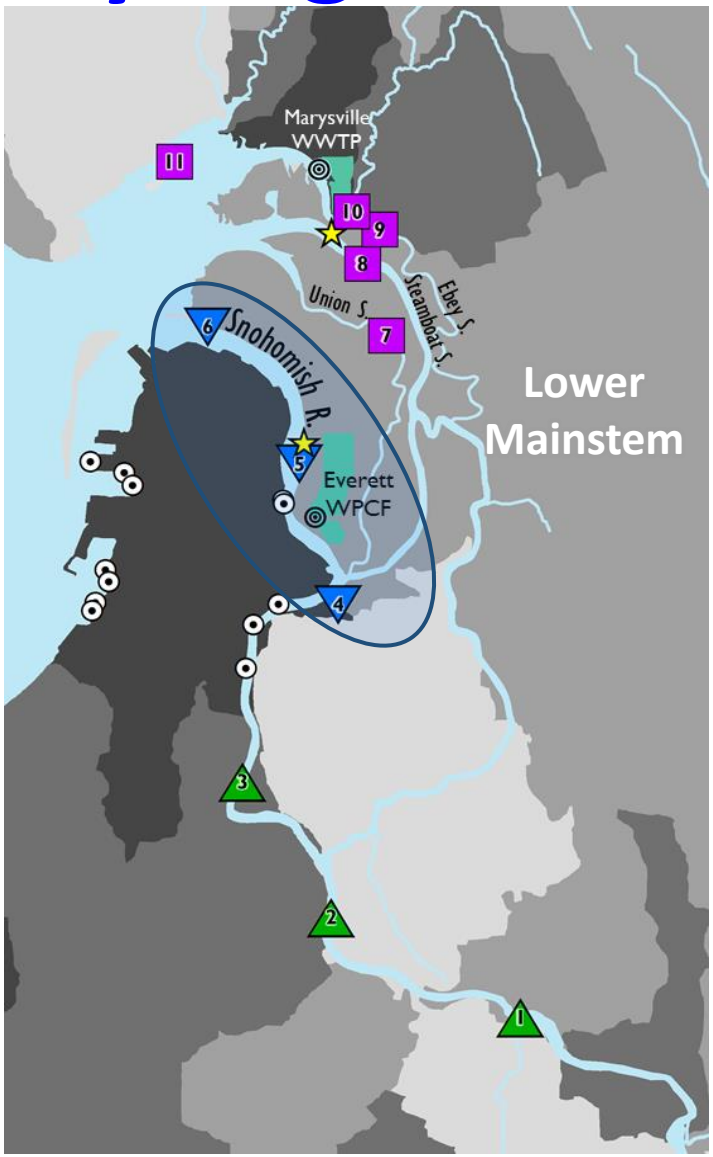
PBDE Concentrations by Region

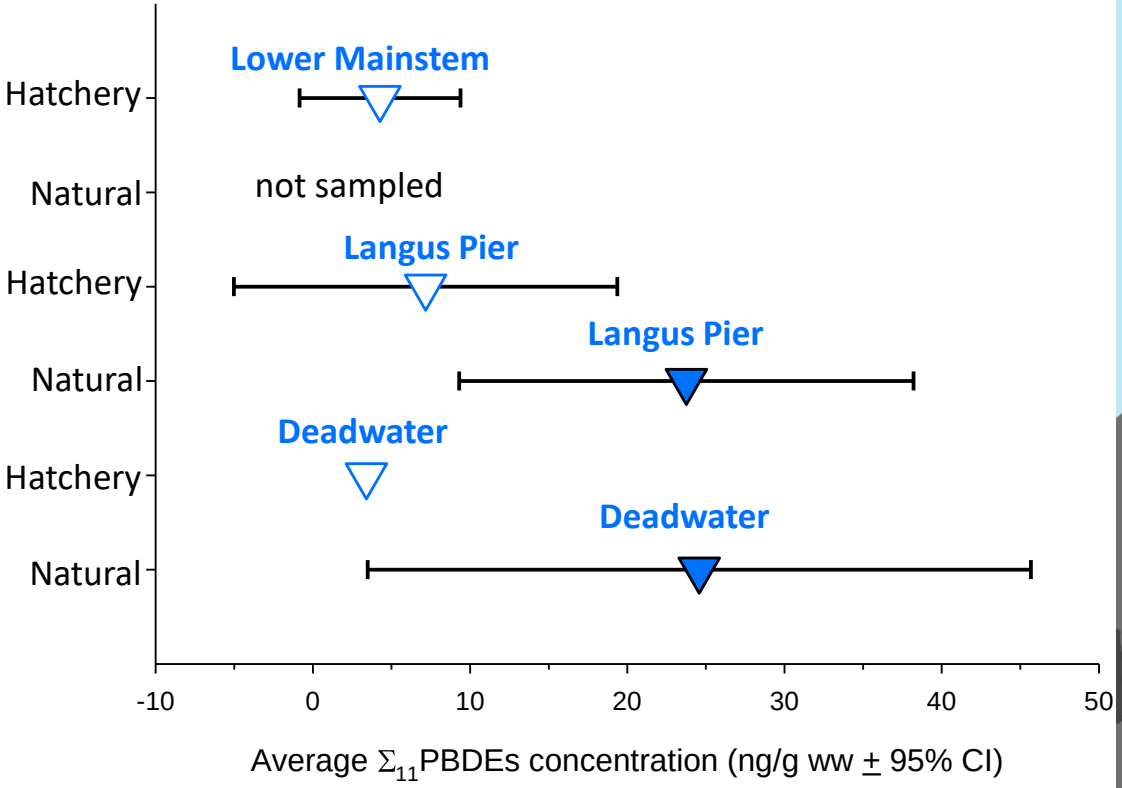
PBDEs were elevated in natural-origin Chinook salmon from the Lower Mainstem

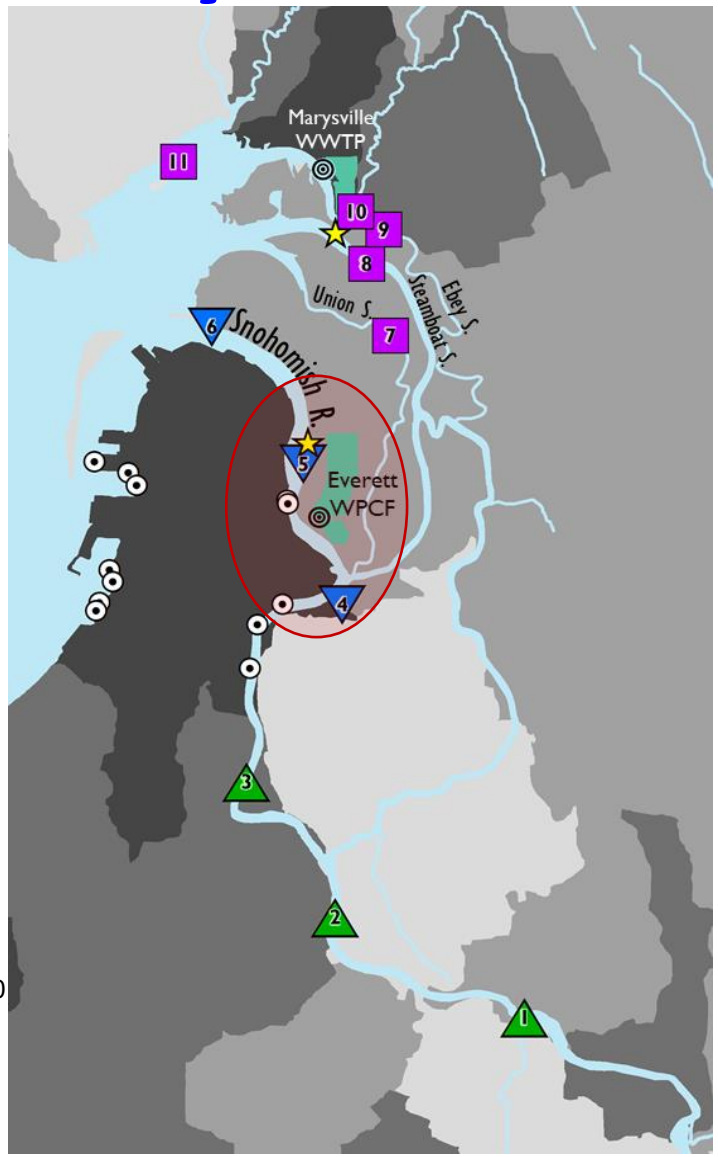




PBDE Concentrations by Site

PBDE concentrations are elevated in natural-origin Chinook from Langus Pier and Deadwater sites





Sampling locations of fish with elevated PBDEs and WWTP outfall and CSOs

Sampling locations of fish with elevated PBDEs and WWTP outfall and CSOs

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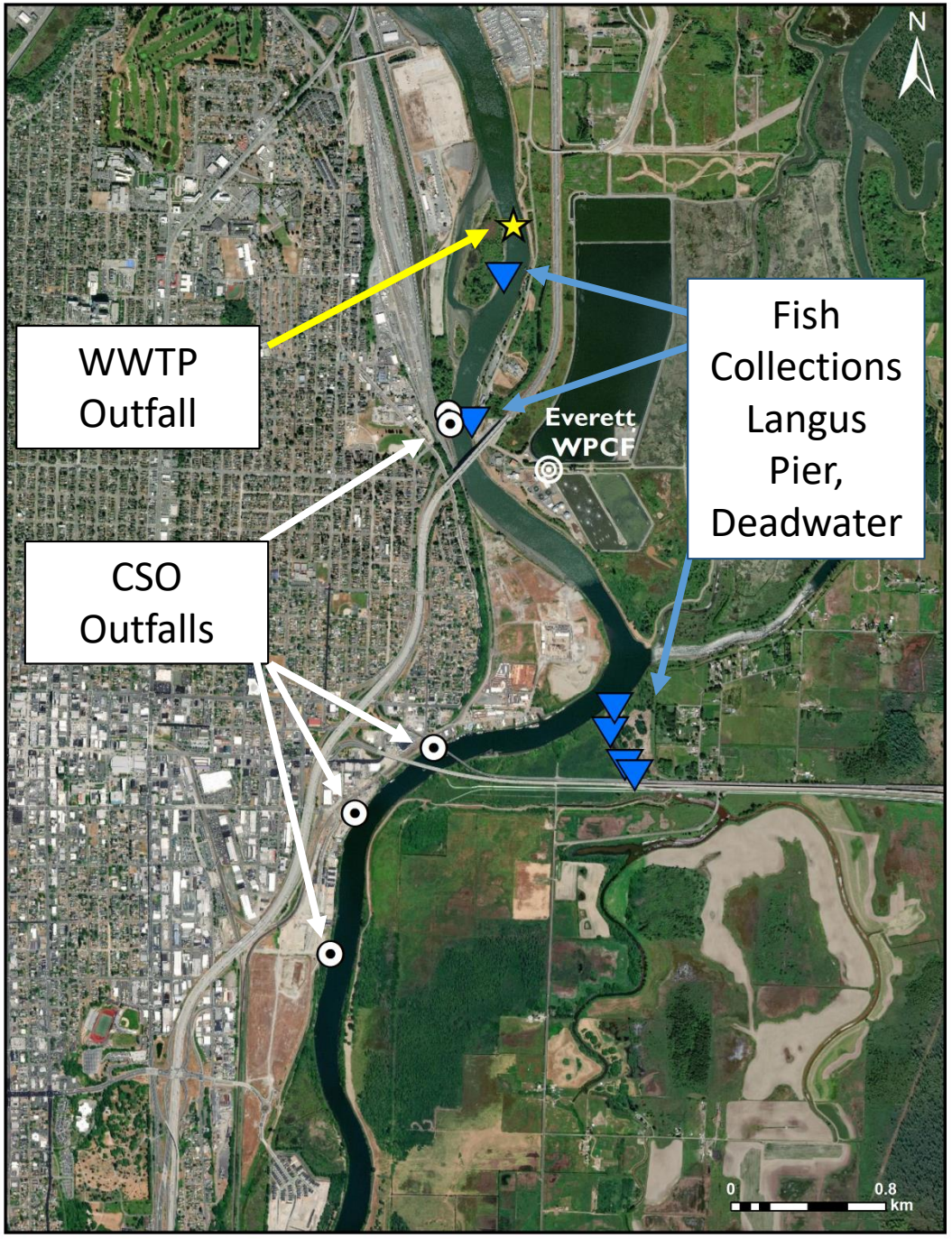
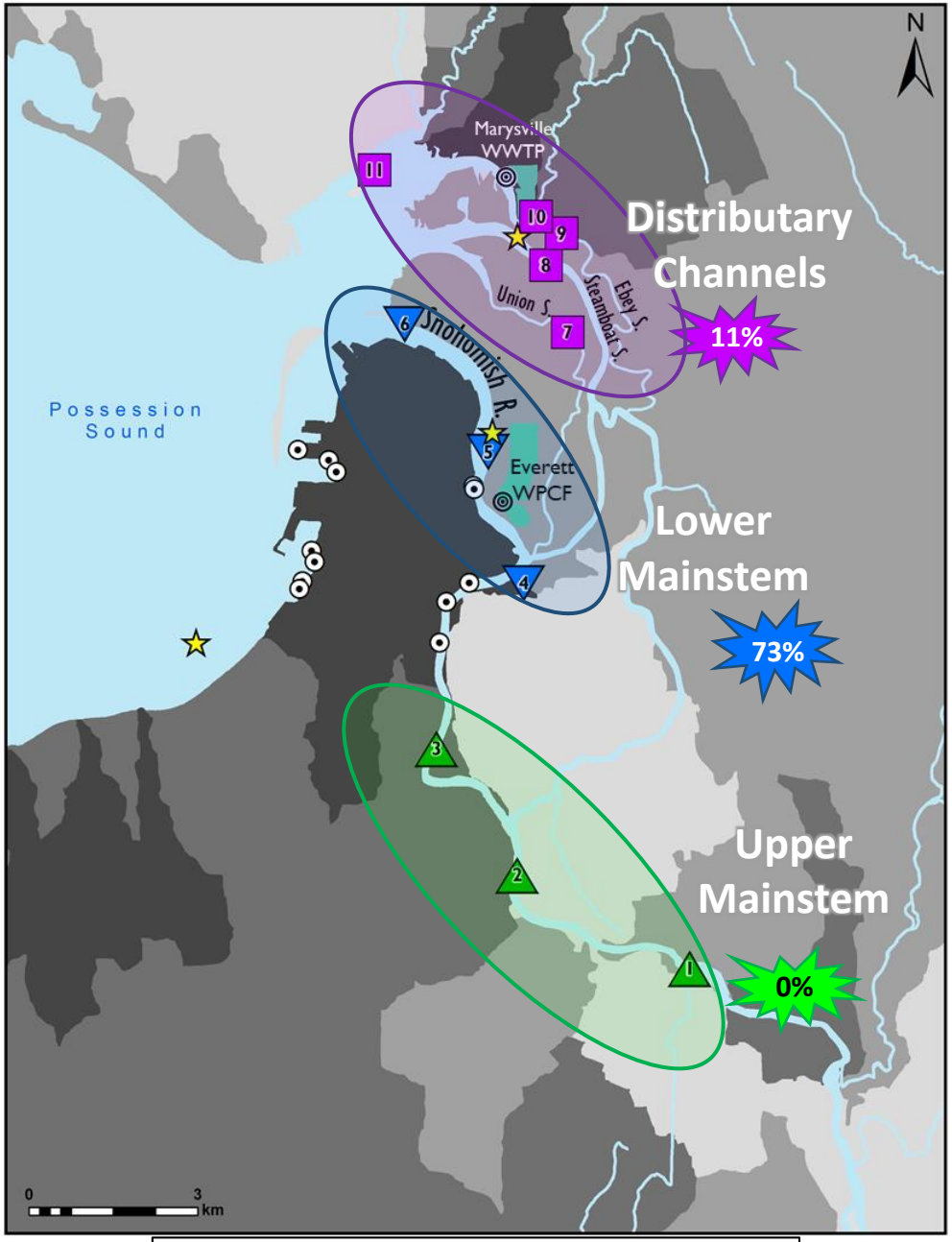


Adverse Effects of PBDEs: Juvenile Salmon Health

In dietary-exposure studies, juvenile Chinook with elevated PBDE concentrations had increased susceptibility to disease

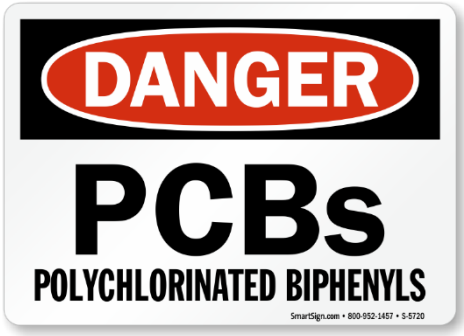
(Arkoosh et al. 2010, 2018)

In Snohomish River only natural-origin fish had PBDE concentration high enough to increase their susceptibility to disease!

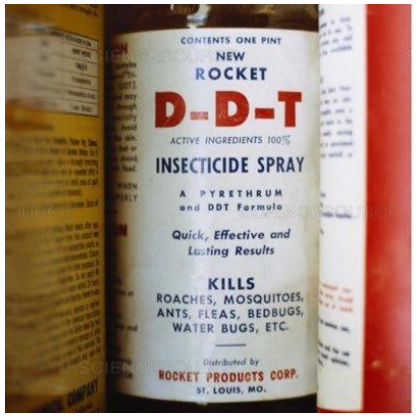


Based on wet weight concentrations

What about other Contaminants?



- elevated concentrations in salmon from Lower Mainstem region
- minor differences between natural- and hatchery-origin salmon



- low concentrations in salmon from all regions
- slightly higher concentrations in natural-origin salmon

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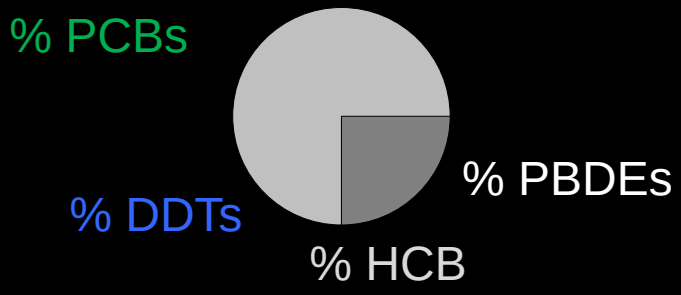
Source Identification
Using Contaminant Fingerprints

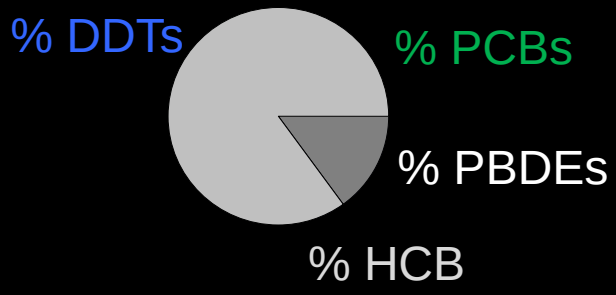
Aquatic environments have distinct patterns of persistent organic pollutants (POPs) based on inputs & environmental attributes

PCBs

DDTs

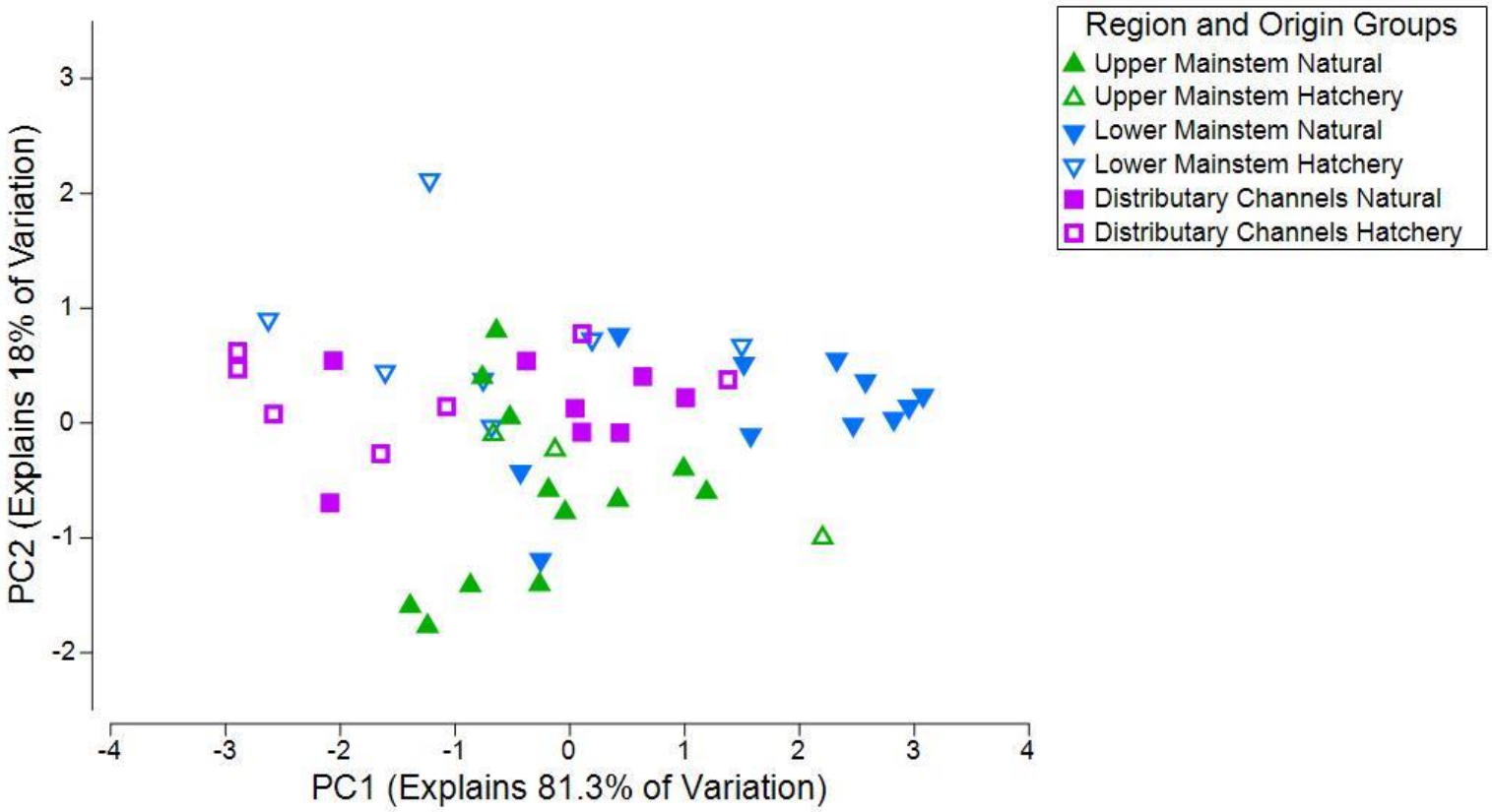
Biota foraging in regions with distinct POPs patterns accumulate specific POPs in proportion to their availability





POP Fingerprints in Chinook salmon

Higher proportion of PBDEs compared to PCBs and DDTs suggests a wastewater input ("source").



Increasing proportion PBDEs

POP Fingerprints in Chinook salmon

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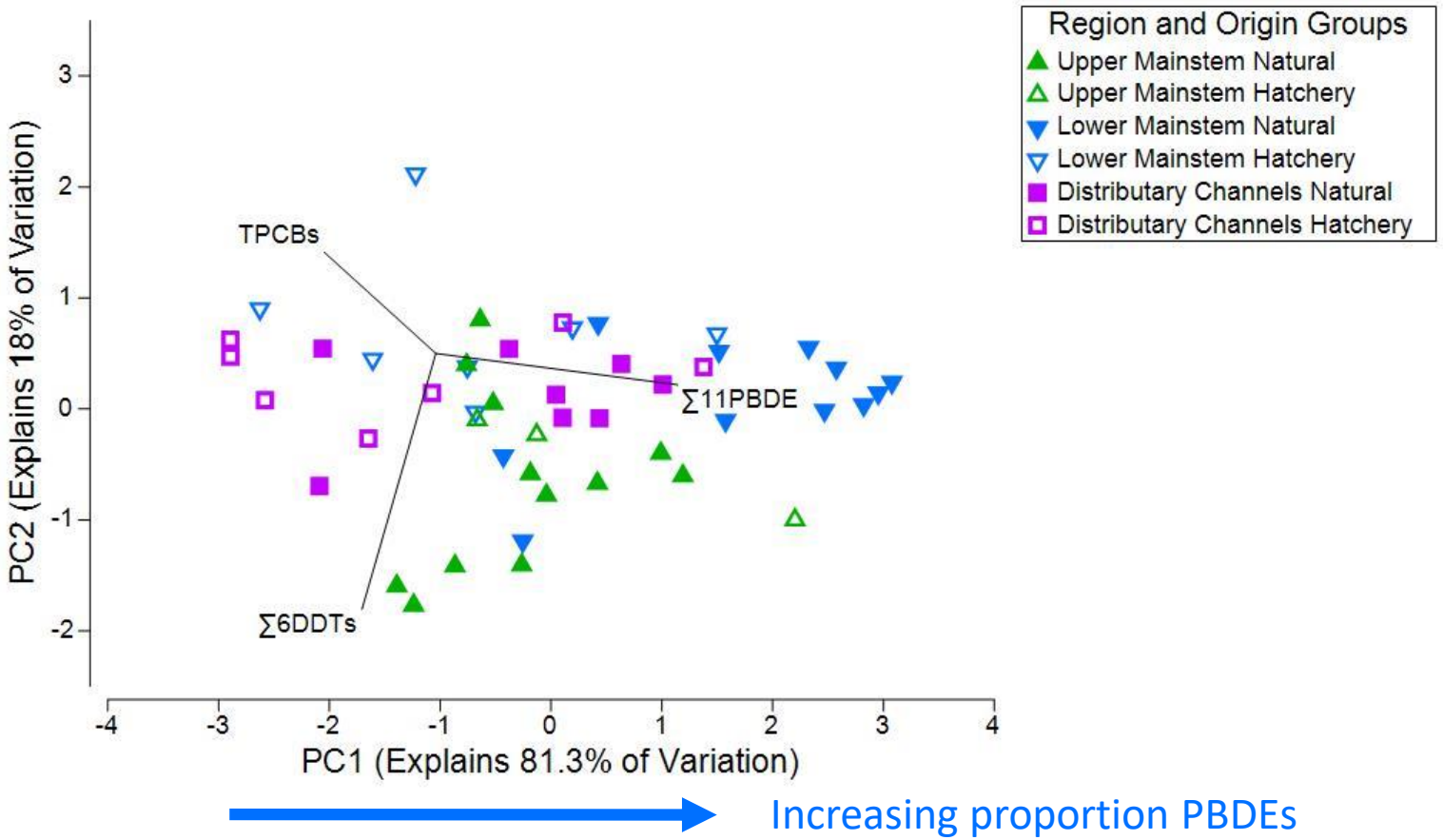

Increasing proportion PBDEs

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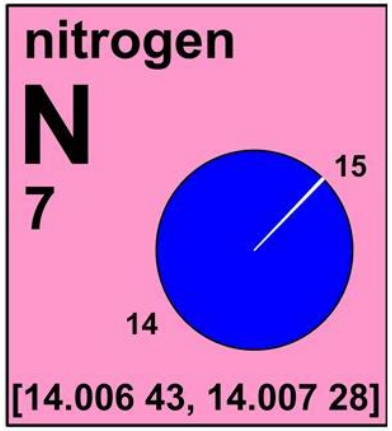
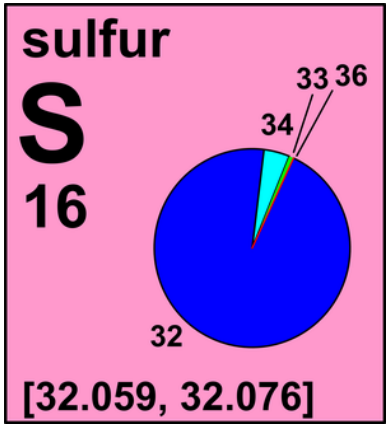
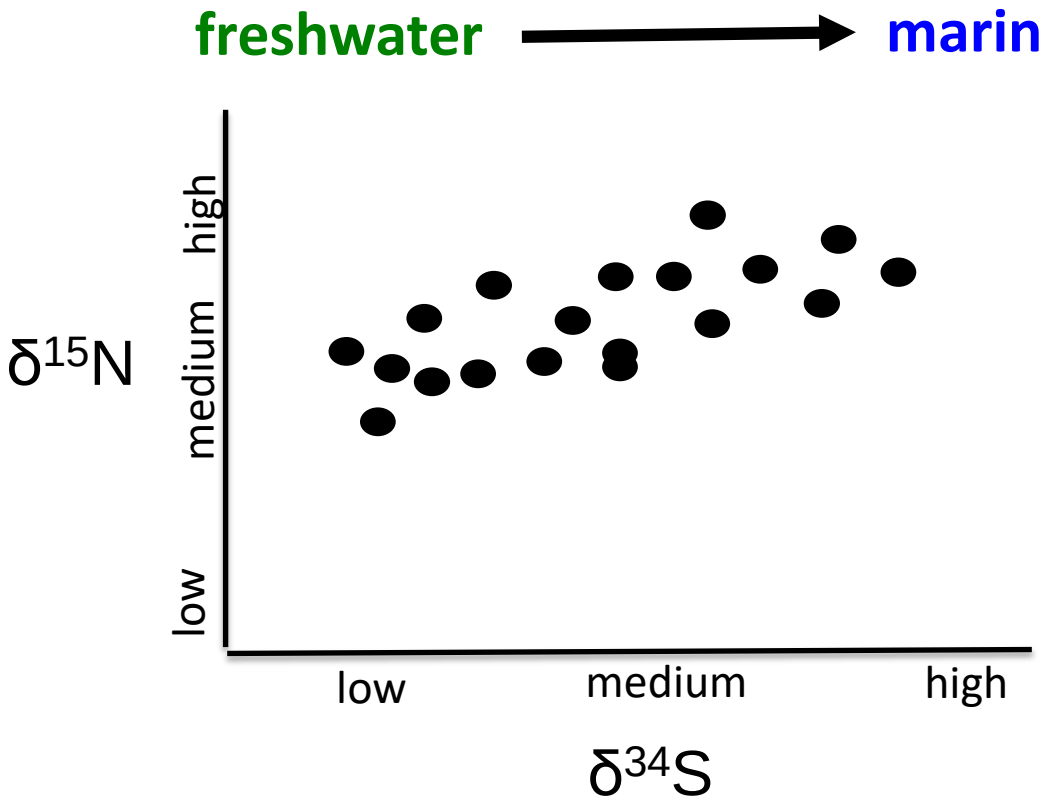
Stable Isotopes: Tools to infer food sources, habitat use & migrations





- Elements occur in various forms (isotopes).
- Stable isotopes of predators reflect characteristics and habitats of their prey.
- Heavier nitrogen isotopes enriched with trophic position but also varies with nitrogen source (fertilizers & wastewater).
- Heavier sulfur isotopes only slightly enriched with trophic levels but vary lots with types of producers.

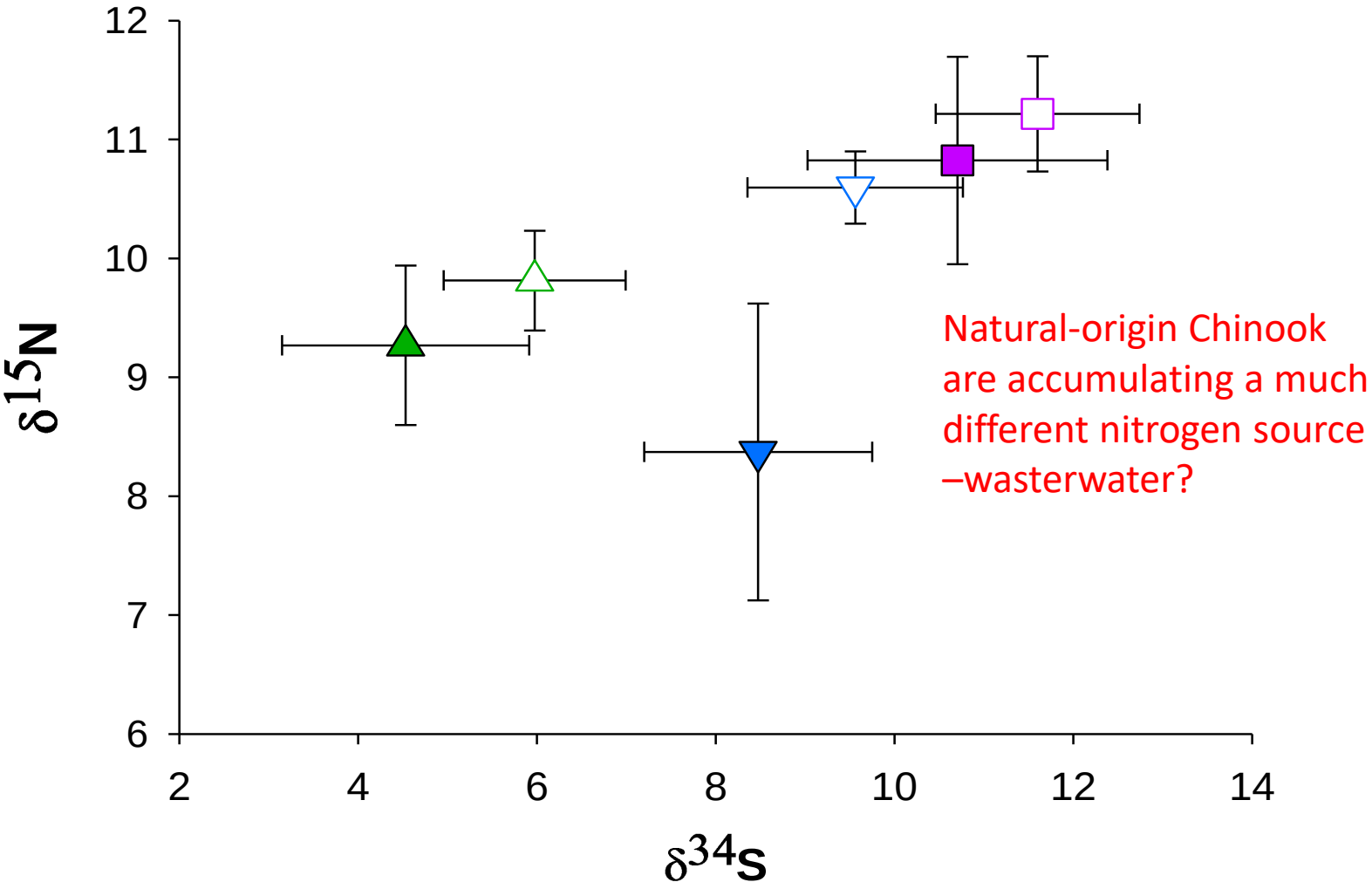
Typical Stable Isotope Signatures in Migrating Juvenile Chinook



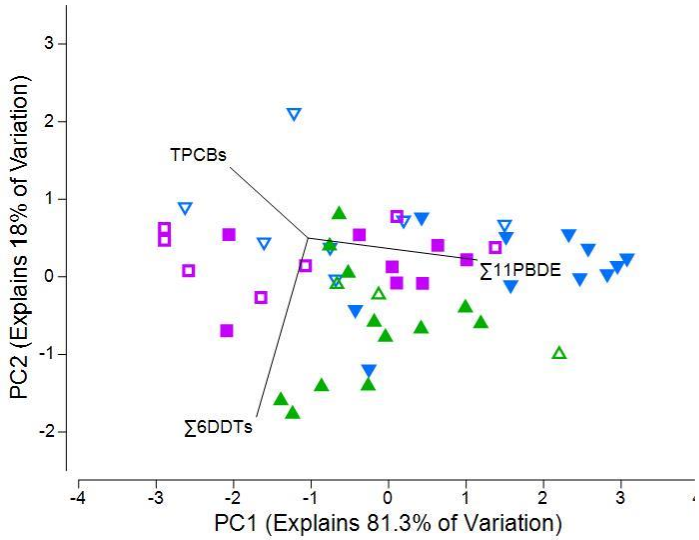
δ^{34}S becomes greatly enriched in salmon as the migrate towards marine waters but δ^{15}N is only slightly enriched

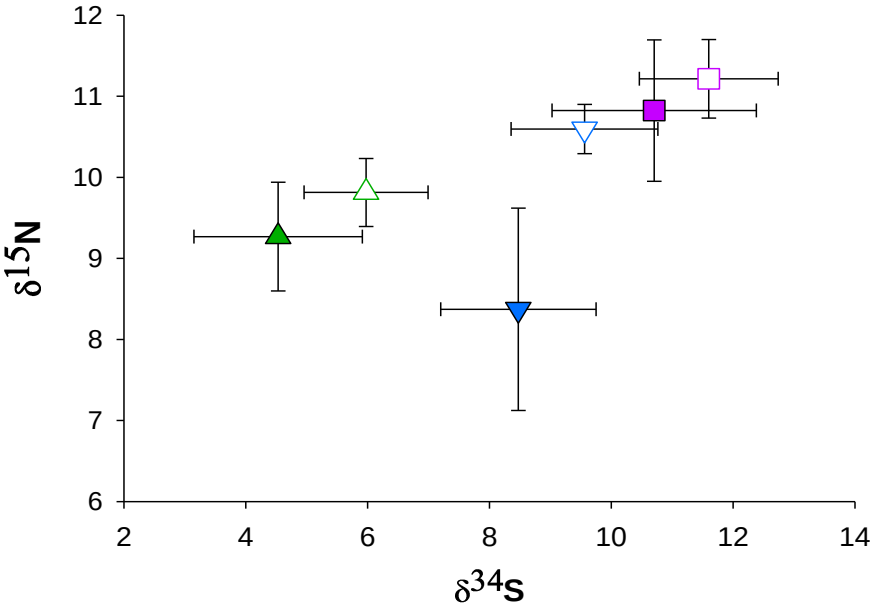
Adapted from Moore et al. 2016 MEPS 559:201-215

Altered Nitrogen Isotopes
in natural-origin Chinook from Lower Mainstem

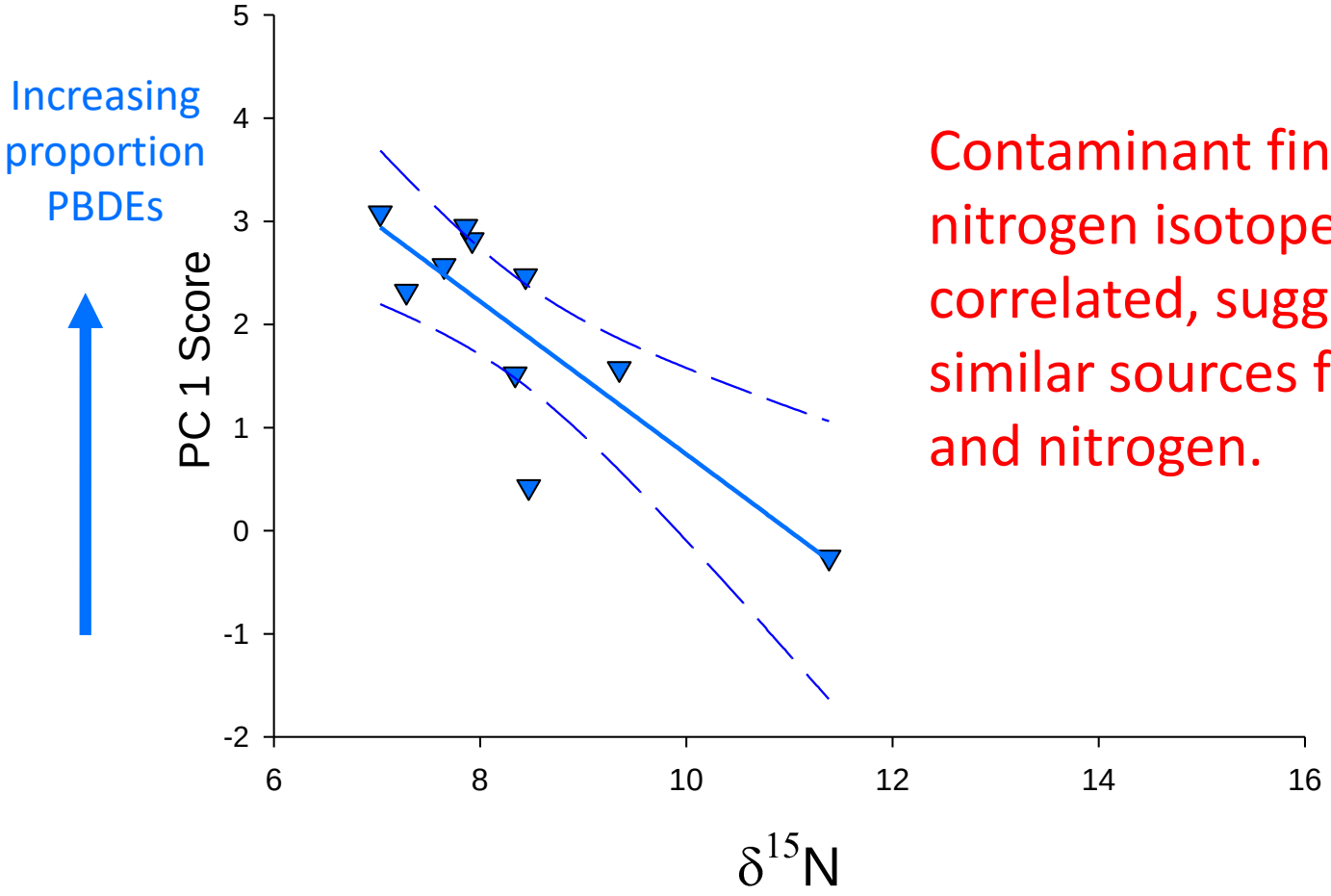


Is the nitrogen source related to contaminant fingerprint?





Sample with higher proportions of PBDES (PC1) have lower $\delta^{15}N$ (more altered nitrogen source)



Contaminant fingerprint and nitrogen isotope ratios are correlated, suggesting similar sources for PBDEs and nitrogen.

Possible Wastewater Sources

- WWTP effluent
  - Frequent discharge (avg 6.2 – 14.4 MGD)
  - nitrogen released as mostly as ammonium compared to nitrate and nitrite
  - other studies with similar release also show depleted $\delta^{15}N$
- CSOs
  - sporadic discharge (range 0.013 – 1.1 MGD)
  - nitrogen released as?

English Sole in Port Gardner

Arch Environ Contam Toxicol
DOI 10.1007/s00244-017-0383-z

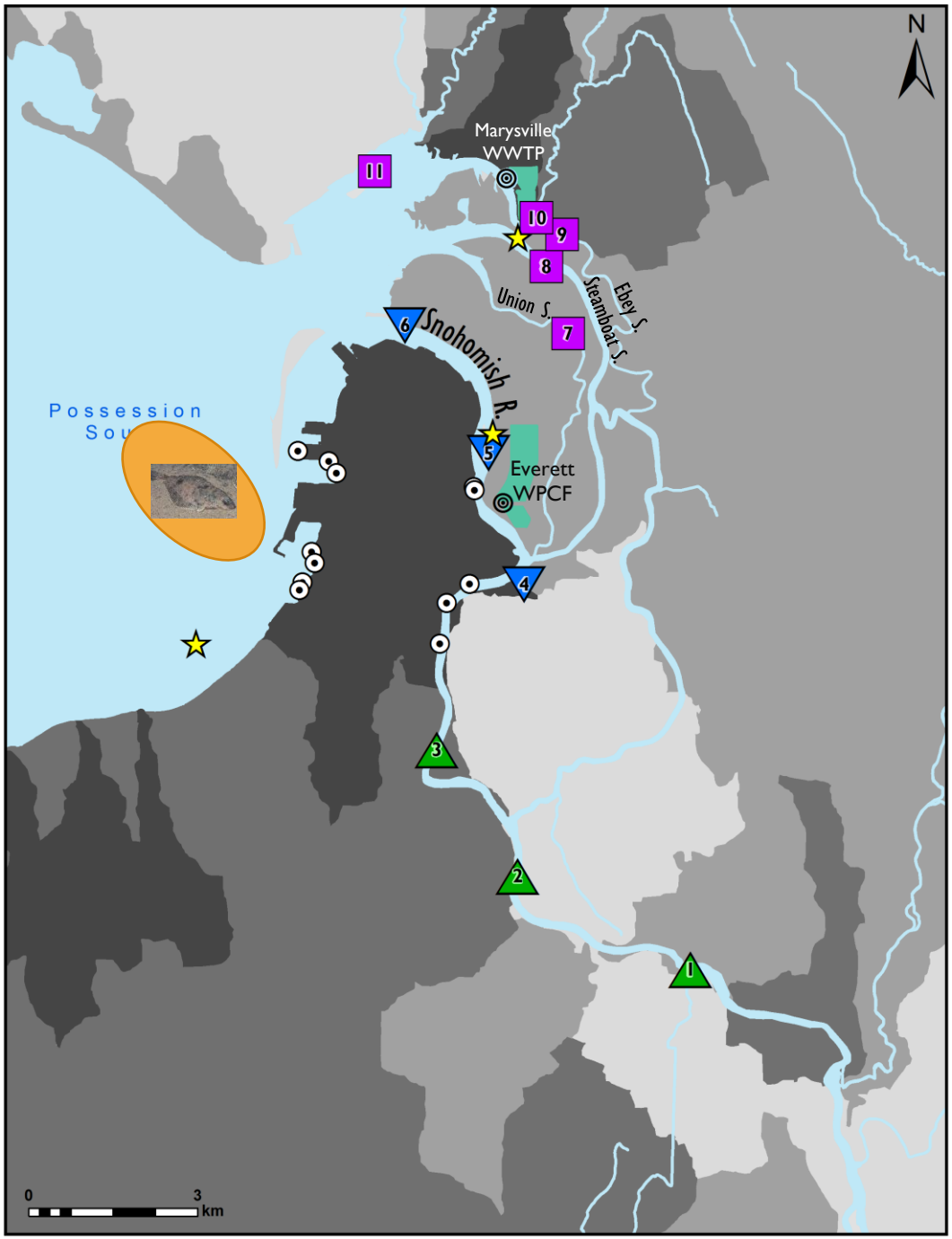
CrossMark

SPECIAL ISSUE: INDICATORS OF OCEAN POLLUTION

Time Trends of Persistent Organic Pollutants in Benthic and Pelagic Indicator Fishes from Puget Sound, Washington, USA

James E. West[1] · Sandra M. O'Neill[1] · Gina M. Ylitalo[2]

English sole from Port Gardner have elevated PBDE levels .



Conclusions ….

- Snohomish River is a PBDE hotspot for juvenile Chinook salmon.
- Highest PBDE exposure occurs in Lower Mainstem, in vicinity of WWTP outfall and CSOs.
- Natural-origin have higher PBDE levels than hatchery-origin Chinook, likely due to longer residence time of natural-origin fish.
- PBDE concentration in juvenile Chinook salmon are high enough to increase their susceptibility to disease, and possibly their marine survival.

Conclusions …

- Wastewater in the Lower Mainstem is likely source (pathway) of PBDEs to salmon:
  - Natural-origin Chinook from the Lower Mainstem have distinct contaminant fingerprints characterized by higher proportions of PBDEs than other POPs, consistent with input from wastewater source.
  - Natural-origin Chinook from the Lower Mainstem also have a distinct isotopic nitrogen ratio, suggesting of a different nitrogen source relative to other locations.
  - Contaminant fingerprint and nitrogen isotope ratios are correlated, suggesting similar sources for PBDEs and nitrogen.

- Loads from WWTP xx to xx times greater than CSOs but additional study needed to confirm which is a greater sources of PBDEs

Next Steps…

1. Proposed NTA - basin-wide evaluation of PBDE in water (SPMDs) and biofilms/sediment to further define PBDE inputs:
   - sample during the first low-flow period (while WWTPs would be discharging),
   - follow-up sampling during high flow period with SPMDs and possibly other media.

2. Measure PBDEs, other POPs, and nitrogen stable isotopes in WWTP effluent and CSO discharges??


Questions?