UNITED STATES DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
NATIONAL MARINE FISHERIES SERVICE
Northwest Region
7600 Sand Point Way N.E., Bldg. 1
Seattle, WA 98115

Refr to NMFS No.:
2008/00148

August 14, 2012

Dennis McLerran
Regional Administrator
U.S. Environmental Protection Agency, Region 10
1200 Sixth Avenue, Suite 900
Seattle, Washington 98101-3140

Re: Jeopardy and Adverse Modification of Critical Habitat Biological Opinion for the Environmental Protection Agency's Proposed Approval of Certain Oregon Administrative Rules Related to Revised Water Quality Criteria for Toxic Pollutants

Dear Mr. McLerran: Dennis

Enclosed is a biological opinion (opinion) prepared by the National Marine Fisheries Service (NMFS) pursuant to section 7(a)(2) of the Endangered Species Act (ESA) on the Environmental Protection Agency's proposed approval of certain Oregon administrative rules related to revised water quality criteria for toxic pollutants.

In this opinion, NMFS concludes that the proposed action is likely to jeopardize the continued existence of LCR Chinook salmon (*Oncorhynchus tshawytscha*), UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon (*O. keta*), LCR coho salmon (*O. kisutch*), SONCC coho salmon, OC coho salmon, SR sockeye salmon (*O. nerka*), LCR steelhead (*O. mykiss*), UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon (*Acipenser medirostris*), eulachon (*Thaleichthys pacificus*), and Southern Resident killer whales (*Orcinus orca*).

NMFS also concludes that the proposed action will result in the destruction or adverse modification of designated critical habitats for LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, SONCC coho salmon, and OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, and eulachon.

NMFS concludes that the proposed action is not likely to adversely affect the following species: Steller sea lion (*Eumetopias jubatus*), blue whale (*Balaenoptera musculus*), fin whale, (*Balaenoptera physalus*), Sei whale (*Balaenoptera borealis*), sperm whale (*Physeter macrocephalus*), humpback whale (*Megaptera novaeangliae*), North Pacific right whale (*Eubalaena glacialis*), loggerhead turtle (*Caretta caretta*), green sea turtle (*Chelonia mydas*), leatherback turtle, (*Dermochelys coriacea*), and Olive Ridley turtle (*Lepidochelys olivacea*); or designated critical habitats for Steller sea lion, North Pacific right whale, green sea turtle, or leatherback turtle.

Section 7(b)(3)(A) of the ESA requires that, if jeopardy or destruction or adverse modification of critical habitat is found, NMFS must provide a Reasonable and Prudent Alternative (RPA), which is an alternative action that the Federal agency could take which would not violate section 7(a)(2). NMFS has developed an RPA, which, if implemented, will change the action such that NMFS would conclude no jeopardy or destruction or adverse modification of critical habitat.

This opinion assesses effects to listed species that occur in the State of Oregon pursuant to the ESA. It does not address EPA's obligation under the Magnuson-Stevens Fishery Conservation and Management Act to consult on effects to essential fish habitat (EFH) for Federally-managed species. Please contact the Oregon State Habitat Office regarding the EFH consultation process.

If you have questions regarding this consultation, please contact Robert Anderson, Fishery Biologist, NMFS Northwest Region, at 503.231.2226.

Sincerely,

William W. Stelle, Jr.
Regional Administrator

cc: Paul Henson, USFWS

Jeopardy and
Destruction or Adverse Modification of Critical Habitat
Endangered Species Act
Biological Opinion
for

Environmental Protection Agency's Proposed Approval of Certain Oregon Administrative Rules Related to Revised Water Quality Criteria for Toxic Pollutants

NMFS Consultation Number: 2008/00148

Federal Action Agency: U.S. Environmental Protection Agency

Affected Species and Determinations:

ESA-Listed Species	Status	Is Action Likely to Adversely Affect Species or Critical Habitat?	Is Action Likely to Jeopardize Species?	Is Action Likely to Destroy or Adversely Modify Critical Habitat?
Lower Columbia River Chinook salmon (*Oncorhynchus tshawytscha*)	Threatened	Yes	Yes	Yes
Upper Willamette River Chinook salmon (*Oncorhynchus tshawytscha*)	Threatened	Yes	Yes	Yes
Upper Columbia River spring-run Chinook salmon (*Oncorhynchus tshawytscha*)	Endangered	Yes	Yes	Yes
Snake River spring/summer run Chinook salmon (*Oncorhynchus tshawytscha*)	Threatened	Yes	Yes	Yes
Snake River fall-run Chinook salmon (*Oncorhynchus tshawytscha*)	Threatened	Yes	Yes	Yes
Columbia River chum salmon (*Oncorhynchus keta*)	Threatened	Yes	Yes	Yes
Lower Columbia River coho salmon (*Oncorhynchus kisutch*)	Threatened	Yes	Yes	Yes
Southern Oregon/Northern California Coasts coho salmon (*Oncorhynchus kisutch*)	Threatened	Yes	Yes	Yes
Oregon Coast coho salmon (*Oncorhynchus kisutch*)	Threatened	Yes	Yes	Yes
Snake River sockeye salmon (*Oncorhynchus nerka*)	Endangered	Yes	Yes	Yes
Lower Columbia River steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes
Upper Willamette River steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes
Middle Columbia River steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes

ESA-Listed Species	Status	Is Action Likely to Adversely Affect Species or Critical Habitat?	Is Action Likely to Jeopardize Species?	Is Action Likely to Destroy or Adversely Modify Critical Habitat?
Upper Columbia River steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes
Snake River Basin steelhead (*Oncorhynchus mykiss*)	Threatened	Yes	Yes	Yes
Green sturgeon Southern DPS (*Acipenser medirostris*)	Threatened	Yes	Yes	Yes
Eulachon (*Thaleichthys pacificus*)	Threatened	Yes	Yes	Yes
Southern Resident killer whale (*Orcinus orca*)	Endangered	No	Yes	No
Steller sea lion (*Eumetopias jubatus*)	Threatened	No	No	No
Blue whale (*Balaenoptera musculus*)	Endangered	No	No	N/A
Fin whale (*Balaenoptera physalus*)	Endangered	No	No	N/A
Sei whale (*Balaenoptera borealis*)	Endangered	No	No	N/A
Sperm whale (*Physeter macrocephalus*)	Endangered	No	No	N/A
Humpback whale (*Megaptera novaeangliae*)	Endangered	No	No	N/A
North Pacific Right whale (*Eubalaena glacialis*)	Endangered	No	No	No
Loggerhead turtle (*Caretta caretta*)	Threatened	No	No	N/A
Green sea turtle (*Chelonia mydas*)	Threatened	No	No	No
Leatherback turtle (*Dermochelys coriacea*)	Endangered	No	No	No
Olive Ridley turtle (*Lepidochelys olivacea*)	Threatened	No	No	N/A

Consultation Conducted By: National Marine Fisheries Service, Northwest Region

Issued by:

William W. Stelle, Jr.
Regional Administrator

Date: August 14, 2012

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1. INTRODUCTION

This Introduction section provides information relevant to the other sections of this document and is incorporated by reference.

1.1 Background

The biological opinion (opinion) and incidental take statement portions of this document were prepared by the National Marine Fisheries Service (NMFS) in accordance with section 7(b) of the Endangered Species Act (ESA) of 1973, as amended (16 U.S.C. 1531, *et seq.*), and implementing regulations at 50 CFR 402.

The opinion is in compliance with section 515 of the Treasury and General Government Appropriations Act of 2001 (Public Law 106-5444) ("Data Quality Act") and underwent pre-dissemination review.

1.2 Consultation History

On June 9, 2004, and September 15, 2004, NMFS, the U.S. Fish and Wildlife Service (FWS), and the U.S. Environmental Protection Agency (EPA) met to develop a work plan for the consultation on EPA's proposed approval of the 2004 Oregon revisions to state water quality standards for toxic pollutants.

Between September 2005 and February 2007, NMFS, EPA, and FWS participated in a series of technical and policy workgroup meetings, conference calls, and e-mail exchanges, and discussed and reviewed EPA's draft methodology for conducting biological evaluations (BE) of EPA's aquatic life criteria methods manual (Methods Manual, EPA 2005). Key events covered over this period are summarized below.

On August 9, 2005, EPA provided NMFS with a copy of the methods manual.

On October 3, 2005, EPA provided NMFS with a preliminary analysis for saltwater zinc and saltwater cadmium to review.

On November 9, 2005, November 10, 2005, and November 17, 2005, NMFS provided EPA several issue papers detailing technical issues with the methods manual and the preliminary analyses for saltwater zinc and saltwater cadmium.

On April 7, 2006, Northwest Environmental Advocates (NWEA) sent EPA a 60-day notice of intent to sue for violations of the Clean Water Act (CWA).

On August 21, 2006, EPA provided NMFS with a draft BE on the effects of its proposed approval of 39 freshwater and 16 saltwater criteria for toxics to review.

On November 2, 2006, NMFS provided EPA with detailed comments on the draft BE for toxics. In our letter, we identified several fundamental problems with the

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application of the methods manual and the draft BE. Subject areas that needed substantial revision or a new approach are summarized below by category.

- Median lethal concentration (LC_{50}) toxicity data interpretation and application
- No observable effect concentration (NOEC) toxicity data interpretation and application
- Exclusion of published toxicity data in the BE analysis
- Acute adjustment factor
- Sublethal effects analysis
- Chemical mixture analysis
- Scale of effect determinations—effects of the action as a whole versus effects based on individual criterion

On December 20, 2006, NMFS, FWS and EPA met to discuss issues with the draft BE and the methods manual.

On February 2, 2007, NMFS, FWS, and EPA developed a draft issues paper as a means to resolve outstanding issues with the BE.

On February 6, 2007, NMFS, FWS, and EPA met to discuss a path forward for resolving outstanding issues with the BE.

On January 16, 2008, EPA submitted a BE with a letter requesting formal consultation on its proposed approval of the Oregon revisions to state water quality standards for toxic pollutants.

On April 4, 2008, NMFS submitted a data request via letter to EPA.

On May 23, 2008, EPA and NWEA settled their lawsuit via consent decree.

October 3, 2008, EPA provided the last of the data requests to NMFS.

On May 26, 2009, NWEA sent NMFS a 60-day notice of intent to sue for failing to timely complete ESA section 7 consultation.

On August 23, 2010, NMFS and NWEA settled their lawsuit via a stipulated order of dismissal.

Between January 2012 through May 2012, NMFS and EPA participated in a series of meetings to discuss the findings in the draft opinion and develop the reasonable and prudent alternative, including meeting with EPA region 10 staff on April 19, 2012, to discuss the reasonable and prudent alternatives and reasonable and prudent measures.

On February 24, 2012, NMFS provided EPA with a preliminary draft opinion.

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On March 8, 2012, NMFS meet with representatives of the Columbia River Inter-Tribal Fish Commission for a technical-level meeting on the consultation.

On March 20, 2012, NMFS meet with representatives of the Yakama Nation for a technical-level meeting on the consultation.

On March 28, 2012, NMFS sent EPA a letter regarding the court-ordered deadline and key dates for interagency coordination to finalizing the opinion.

On April 11, 2012, NMFS received a letter from EPA recognizing the court-ordered deadline and key dates for interagency coordination to finalizing the opinion.

On May 7, 2012, NMFS received a letter from EPA with comments on the February 24, 2012, draft opinion.

On May 7, 2012, NMFS provided EPA with a final draft opinion.

Between May 17, 2012, and August 1, 2012, NMFS and EPA exchanged information on the development of the reasonable and prudent alternative (RPA).

On August 9, 2012, EPA sent NMFS a letter withdrawing their request for consultation on Oregon's acute and chronic aluminum criteria as "EPA has determined that the BE submitted to NMFS in January 2008 incorrectly described the proposed federal action under consultation for aluminum (*i.e.*, CW A § 303(c)(3) approval of Oregon's submission of aluminum criteria). Specifically, Oregon's submitted description of the pollutant refers to aluminum in waters with a pH of 6.5- 9.0, but a footnote in the criterion itself indicates that the criterion is meant to apply to waters with pH less than 6.6 and hardness less than 12 mg/L (as $CaCO_3$)." Due to the court-ordered deadline of August 14, 2012, NMFS did not have time to modify its opinion to exclude acute and chronic aluminum from the document. The NMFS acknowledges EPA's revision to the proposed action, however, and notes it does not anticipate EPA will carry out the RPA for aluminum in light of this change. The NMFS will await a further request from EPA relating to EPA's potential future actions regarding Oregon's aluminum criteria.

1.3 Proposed Action

The proposed action is EPA's, Region 10, proposed approval of portions of Oregon Administrative Rules (340-041-0033) related to revised water quality criteria for toxic pollutants for aquatic life (Table 1.1) under section 303(c) of the Clean Water Act (CWA), and 40 CFR 131. The CWA requires all states to adopt water quality standards (WQS) to restore and maintain the physical, chemical, and biological integrity the Nation's waters. Section 303(c) of the act requires states to adopt chemical-specific, numeric criteria for priority toxic pollutants. The criteria must protect state-designated beneficial uses of water bodies. Development of WQS is primarily the responsibility of the states, but adoption of the WQS is subject to approval by EPA. The EPA is proposing to approve or disapprove Oregon's proposed numeric water quality criteria for 20 toxic pollutants that include 39 freshwater criteria and 26 saltwater criteria.

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Oregon's proposed aquatic life criteria are listed in Table 1.1. The Oregon criteria are identical to the national criteria developed by EPA and recommended by EPA to states for adoption. Table 1.2 provides a comparison of the Oregon's existing numeric criteria with the proposed numeric criteria for aquatic life subject to this consultation. Table 1.3 lists all the toxic criteria with numeric criteria (regulated by Oregon) and those without numeric criteria (unregulated). In the BE, EPA evaluated the proposed criteria as continuous water quality conditions, *i.e.*, EPA assumed that listed species would be exposed to waters meeting the proposed water quality criteria listed in Table 1.1. The EPA assumed that the numeric criteria would be met outside the State's applicable mixing zone boundaries, *i.e.*, that the criteria represent ambient water quality conditions.

Proposed aquatic life criteria that are the same or more stringent than previously approved by EPA may be used prior to EPA approval in national pollution elimination system [NPDES and stormwater (MS4)] permits issued by the Oregon Department of Environmental Quality (ODEQ) unless they are (1) formula-based metals, (2) ammonia, (3) were previously total recoverable criteria, or (4) would discharge into a 303(d)-listed impaired water, and are otherwise not in effect until approved by EPA. Compounds subject to pre-approval use are lindane, dieldrin, endosulfan-alpha, endosulfan-beta, and heptachlor epoxide, all legacy compounds, *i.e.*, compounds that are either no longer in use or their use is highly restricted within the U.S.

The acute criterion is the Criterion Maximum Concentration (CMC) and is EPA's acute criterion recommendation. The CMC is set to one-half of the fifth percentile of the average acute toxicity values for the various genera tested. The EPA's technical support document (EPA 1991) recommends that the one-hour average exposure concentrations should not exceed the CMC more than once every three years on the average.

The chronic criterion is the Criterion Continuous Concentration (CCC), criterion for indefinite exposures, and is EPA's chronic criterion recommendation. The CCC is derived from a set of chronic toxicity values, which are the geometric mean of the highest no observed effect concentrations (NOEC) and lowest observed effect concentrations (LOEC) for survival, growth, or reproduction in tests which range from seven days to several months or more. The EPA's technical support document (EPA 1991) recommends that the four-day average exposure concentrations should not exceed the CCC more frequently than once every three years on the average.

For ammonia, the numeric criteria are based on the following equations (numeric criteria for ammonia are calculated based on site-specific pH and temperature):

1) Acute ammonia criterion, salmonid fishes present:

$$CMC = \frac{0.275}{1 + 10^{7.204 - pH}} + \frac{39.0}{1 + 10^{pH - 7.204}}$$

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2) Acute ammonia criterion, salmonid fishes absent:

$$CMC = \frac{0.411}{1 + 10^{7.204 - pH}} + \frac{58.4}{1 + 10^{pH - 7.204}}$$

3) Chronic ammonia criterion, early life stages present:

$$CCC = \frac{0.577}{1+10^{7.688 - pH}} + \frac{2.487}{1+10^{pH - 7.688}} * MIN (2.85, 1.45^{*} 10)^{0.028(25-T)}$$

4) Chronic ammonia criterion, early life stages not present:

$$CCC = \frac{0.577}{1+10^{7.688 - pH}} + \frac{2.487}{1+10^{pH - 7.688}} *1.45^{*} 10^{0.028 (25- (MAX T, 7))}$$

The freshwater criterion for cadmium, chromium (III), copper, lead, nickel, silver, and zinc are expressed as a function of hardness (CaCO3 mg/L) in the water column (refer to Appendix A in the BE, pages 16-26, for equations and conversion factors).

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Table 1.1 Proposed Oregon aquatic life criteria for toxics. All values are expressed as micrograms per liter (μg/L) except where noted. Shaded cells denote no criteria proposed for EPA approval.

Compounds	Freshwater Acute Criteria (μg/L)	Freshwater Chronic Criteria (μg/L)	Saltwater Acute Criteria (μg/L)	Saltwater Chronic Criteria (μg/L)
Aluminum	750	87		
Ammonia*	5.6 mg/L	1.7 mg/L		
Arsenic	340	150	69	36
gamma-BHC (Lindane)	0.95			
Cadmium	2.0	.25	40	8.8
Chromium (III)	570	74		
Chromium (VI)	16	11	1100	50
Copper	13	9.0	4.8	3.1
Dieldrin	0.24	0.056		
alpha- Endosulfan	0.22	0.056	0.034	0.0087
beta- Endosulfan	0.22	0.056	0.034	0.0087
Endrin	0.086	0.036		
Heptachlor epoxide	0.52	0.0038	0.053	0.0036
Lead	65	2.5	210	8.1
Nickel	470	52	74	8.2
Pentachlorophenol	19	15		7.9
Selenium	190	5.0	290	71
Silver	3.2	0.10	1.9	
Tributyltin	.46	.063	.37	.01
Zinc	120	120	90	81

* See equations 1, 2, 3, and 4.

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Table 1.2 Existing and proposed numeric criteria for aquatic life in Oregon.

Compound	Existing Acute Criteria	Proposed Acute Criteria	Existing Chronic Criteria	Proposed Chronic Criteria	Existing Acute Criteria	Proposed Acute Criteria	Existing Chronic Criteria	Proposed Chronic Criteria
	FW	FW	FW	FW	SW	SW	SW	SW
Ar	360	340	190	150	69	69	36	36
Cd	3.9	2	1.1	0.25	43	40	9.3	8.8
CrIII	1700	570	210	74				
CrVI	16	16	11	11	1100	1100	50	50
Cu	18	13	12	9	2.9	4.8	2.9	3.1
Pb	82	65	3.2	2.5	140	210	5.6	8.1
Ni	1400	470	160	52	75	74	8.3	8.2
Se	260	190	35	5	410	290	54	71
Ag	4.1	3.2	0.12	0.1	2.3	1.9		
Zn	120	120	110	120	95	90	86	81
PCP	20	19	13	15				7.9
Dieldrin	2.5	0.24	0.0019	0.056				
Endrin	0.18	0.086	0.0023	0.036				
Ammonia	6	5.6	0.76	1.7				
Lindane	2	0.95						
TEIT		0.36		0.063		0.37		0.31
Al		750		87				
Hept E		0.52		0.0038		0.053		0.0036
Endo-a		0.22		0.056		0.034		0.0087
Endo-b		0.22		0.056		0.034		0.0087
same	7							
more strict	30							
less strict	9							
previously unregulated	19							
	No criteria proposed							
Boldtype=legacy compounds								

Table 1.3 Regulated and unregulated toxic compounds in the State of Oregon (ODEQ 2003). Compounds considered in this opinion for approval by EPA are shaded.

Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
Antimony				
Arsenic *	360	190	69	36
Cadmium ***	3.9	1.1	43	9.3
Chromium III ***	1700	210		
Chromium VI *	16	11	1100	50
Copper ***	18	12	2.9	2.9
Lead ***	82	3.2	241	5.6
Mercury	2.4	0.012	2.1	0.025
Nickel ***	1400	160	75	8.3
Selenium *	260	35	410	54
Silver **	4.1	0.12	2.3	
Thallium				
Zinc ***	120	110	95	86
Cyanide	22	5.2	1	1
Asbestos				
Dioxin (2,3,7,8-TCDD)				
Acrolein				
Acrylonitrile				
Benzene				
Bromoform				
Carbon Tetrachloride				
Chlorobenzene				
Chlorodibromomethane				
Chloroform				
Dichlorobromomethane				
Dichloroethane 1,2-				
Dichloroethylene 1,1-				
Dichloropropane 1,2-				
Dichloropropene 1,3-				
Ethylbenzene				
Methyl Bromide				
Methylene Chloride				
Tetrachloroethane 1,1,2,2-				
Tetrachloroethylene				
Toluene				
Dichloroethylene 1,2-Trans-				
Trichloroethane 1,1,2-				
Trichloroethylene				
Vinyl Chloride				
Chlorophenol 2-				
Dichlorophenol 2,4-				

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Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
Dimethylphenol 2,4-				
Methyl-4,6-Dinitrophenol 2-				
Dinitrophenol 2,4-				
Pentachlorophenol	20	13	13	7.9
Phenol				
Trichlorophenol 2,4,6-				
Acenaphthene				
Anthracene				
Benzidine				
BenzoaAnthracene				
BenzoaPyrene				
BenzobFluoranthene				
BenzokFluoranthene				
ChloroethylEther, Bis2-				
ChloroisopropylEther, Bis2-				
EthylhexylPhthalate, Bis2-				
Butylbenzyl Phthalate				
Chloronaphthalene 2-				
Chrysene				
Dibenzoa,hAnthracene				
Dichlorobenzene 1,2-				
Dichlorobenzene 1,3-				
Dichlorobenzene 1,4-				
Dichlorobenzidine 3,3'-				
DiethylPhthalate				
Dimethyl Phthalate				
Di-n-Butyl Phthalate				
Dinitrotoluene 2,4-				
Diphenylhydrazine 1,2-				
Fluoranthene				
Fluorene				
Hexachlorobenzene				
Hexachlorobutadiene				
Hexachlorocyclopentadiene				
Hexachloroethane				
Ideno1,2,3-cdPyrene				
Isophorone				
Nitrobenzene				
Nitrosodimethylamine, N-				
Nitrosodi-n-Propylamine, N-				
Nitrosodiphenylamine, N-				
Pyrene				
Trichlorobenzene 1,2,4-				
Aldrin	3.0		1.3	

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Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
BHC, alpha-				
BHC, beta-				
BHC, gamma- (Lindane)	2	0.08	0.16	
Chlordane	2.4	0.0043	0.09	0.004
DDT 4,4'-	1.1	0.001	0.13	0.001
DDE 4,4'-				
DDD 4,4'-				
Dieldrin	2.5	0.0019	0.71	0.0019
Alpha-Endosulfan				
Beta-Endosulfan				
Endosulfan Sulfate				
Endrin	0.18	0.0023	0.037	0.0023
Endrin Aldehyde				
Heptachlor	0.52	0.0038	0.053	0.0036
Heptachlor Epoxide				
Polychlorinated biphenyls PCBs:	2	0.014	10	0.03
Toxaphene	0.73	0.0002	0.21	0.0002
Aluminum				
Ammonia (mg/L)	6	0.76		
Barium				
Chloride	860000	230000		
Chlorine	19	11	13	7.5
Chlorophenoxy Herbicide 2,4,5,-TP				
Chlorophenoxy Herbicide 2,4-D				
Chloropyrifos	0.083	0.041	0.011	0.0056
Demeton		0.1		0.1
Ether, Bis Chloromethyl				
Guthion		0.01		0.01
Hexachlorocyclo-hexane-Technical				
Iron		1000		
Malathion		0.1		0.1
Manganese				
Methoxychlor		0.03		0.03
Mirex		0.001		0.001
Nitrates				
Nitrosamines				
Dinitrophenols				
Nitrosodibutylamine,N				
Nitrosodiethylamine,N				
Nitrosopyrrolidine,N				
Parathion	0.065	0.013		
Pentachlorobenzene				
Phosphorus Elemental				0.1
Sulfide-Hydrogen Sulfide		2.0		2.0

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Aquatic Life Criteria				
	Freshwater	**Freshwater**	**Marine**	**Marine**
	Acute Criteria	**Chronic Criteria**	**Acute Criteria**	**Chronic Criteria**
Compound (µg/L)				
Tetrachlorobenzene,1,2,4,5				
Tributyltin TBT				
Trichlorophenol 2,4,5				

* all criteria expressed as dissolved metal
** all criteria expressed as dissolved metal. FW acute criteria are hardness dependent (concentration shown is hardness = 100 mg/L $CaCO_3$)
*** all criteria expressed as dissolved metal. FW criteria are hardness dependent (concentration shown is hardness = 100 mg/L $CaCO_3$)

1.4 Action Area

'Action area' means all areas to be affected directly or indirectly by the Federal action and not merely the immediate area involved in the action (50 CFR 402.02). The species occurring within the action area that are the subject of this consultation are listed in Table 1.4.1 and Table 1.4.2.

References for listing status and dates, ESA section 4(d) take prohibitions, and critical habitat designations are provided in Table 1.4.1 and Table 1.4.2.

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Table 1.4.1. Federal Register notices for final rules that list threatened and endangered species, designate critical habitats, or apply protective regulations to listed species considered in this consultation (anadromous fishes).

Species	Listing Status	Critical Habitat	Protective Regulations
Chinook salmon (*Oncorhynchus tshawytscha*)			
Lower Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Upper Willamette River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Upper Columbia River spring-run	E 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	ESA section 9 applies
Snake River spring/summer run	T 8/15/11; 76 FR 50448	10/25/99; 64 FR 57399	6/28/05; 70 FR 37160
Snake River fall-run	T 8/15/11; 76 FR 50448	12/28/93; 58 FR 68543	6/28/05; 70 FR 37160
Chum salmon (*O. keta*)			
Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Coho salmon (*O. kisutch*)			
Lower Columbia River	T 8/15/11; 76 FR 50448	Not applicable	6/28/05; 70 FR 37160
Southern Oregon/northern California coasts	T 8/15/11; 76 FR 50448	5/5/99; 64 FR 24049	6/28/05; 70 FR 37160
Oregon coast	T 2/11/08; 73 FR 7816	2/11/08; 73 FR 7816	2/11/08; 73 FR 7816
Sockeye salmon (*O. nerka*)			
Snake River	E 8/15/11; 76 FR 50448	12/28/93; 58 FR 68543	ESA section 9 applies
Steelhead (*O. mykiss*)			
Lower Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Upper Willamette River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Middle Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Upper Columbia River	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	2/1/06; 71 FR 5178
Snake River basin	T 8/15/11; 76 FR 50448	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160
Green sturgeon (*Acipenser medirostris*)			
Southern DPS	T 4/7/06; 71 FR 17757	10/9/2009: 74 FR 52300	6/2/10; 75 FR 30714
Eulachon (*Thaleichthys pacificus*)			
Eulachon	3/18/10; 75 FR 13012	10/20/11; 76 FR 65324	Not applicable

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Table 1.4.2. Federal Register notices for final rules that list threatened and endangered species, designate critical habitats, or apply protective regulations to listed species considered in this consultation (marine mammals and turtles).

Species	Listing Status	Critical Habitat	Protective Regulations
Southern Resident killer whale (*Orcinus orca*)	E 11/18/05; 70 FR 69903	11/29/06; 71 FR 69034	ESA section 9 applies
Steller sea lion (*Eumetopias jubatus*)	T 11/26/90; 55 FR 49204	8/27/93; 58 FR 45269	11/26/90; 55 FR 49204
Blue whale (*Balaenoptera musculus*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
Fin whale (*Balaenoptera physalus*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
Sei whale (*Balaenoptera borealis*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
Sperm whale (*Physeter macrocephalus*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
Humpback whale (*Megaptera novaeangliae*)	E 12/2/70; 35 FR 18319	Not applicable	ESA section 9 applies
North Pacific right whale (*Eubalaena glacialis*)	E 12/2/70; 35 FR 19319	7/6/06; 71 FR 38277	ESA section 9 applies
Loggerhead turtle (*Caretta caretta*)	T 7/28/78; 43 FR 32800	Not applicable	7/28/78; 43 FR 32800
Green sea turtle (*Chelonia mydas*)	T 7/28/78; 43 FR 32800	9/2/98; 63 FR 46693	7/28/78; 43 FR 32800
Leatherback turtle (*Dermochelys coriacea*)	E 12/2/70; 35 FR 18319	1/26/2012; 77 FR 4170	ESA section 9 applies
Olive Ridley turtle (*Lepidochelys olivacea*)	T 7/28/78; 43 FR 32800	Not applicable	7/28/78; 43 FR 32800

The fish considered in the opinion occur in the action area and use freshwater and marine habitats for multiple life history events, including incubation; emergence (residence in gravel); juvenile rearing, smoltification and migration; and adult migration, holding and spawning.

Marine mammals and sea turtles considered in this opinion occur in the marine portion of the below stated action area and use freshwater (Steller sea lions only) and marine habitats for multiple life history events, including foraging, rearing, and migration. Chinook salmon that originate from Oregon will disperse both north (to the coastal waters of Washington and the west coast of Vancouver Island), and south off the coast of California (Weitkamp 2010). Therefore, the action area for Southern Resident killer whales encompasses the whales' entire coastal range from California to Vancouver, British Columbia where the marine ranges of Southern Residents and affected Chinook salmon overlap.

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The action area for this consultation includes the freshwater, estuarine, and ocean areas subject to the jurisdiction of the State of Oregon, where the criteria apply, as well as areas beyond the state's jurisdiction where the regulated pollutants area likely to be transported. The action area includes the Pacific Ocean, limited to the entire coastal range from California to Vancouver, British Columbia, where the marine ranges of some of the species subject to this consultation (Southern Resident killer whales and Chinook salmon) overlap, and to which the particular compounds under consultation (Table 1.1) are transported beyond these limits by such biotic and abiotic factors as river runoff, tidal energy, topography, stratigraphy, biota trapping/assimilation), that may influence chemical transport processes beyond original areas of dispersion.

Based on the chemical processes (sources, transport, fate, transformation) of compounds listed in Table 1.1, which are described later in this opinion, the action area, in addition to the Pacific Ocean area delineated above, includes all inland basins that provide access to the species listed in Table 1.1 (Figure 1.4.1 and Figure 1.4.2), including the Columbia River, bank-to-bank, from the mouth to the Washington-Oregon border [river mile (RM) 292]; and the Snake River, from RM 169 to RM 247.5 (Figure 1.4.1 and Figure 1.4.2). The Klamath River originates in southwest Oregon. However, the Iron Gate dam prevents up-river migration of (southern Oregon/Northern California coasts) SONCC coho salmon across the Oregon-California border. Iron Gate dam is located on the Klamath River at river mile 190.2 in California. Based on the fact that no southern Oregon/Northern California coasts SONCC coho salmon from the Klamath Strata occur in Oregon, NMFS determined that individuals of populations in the Klamath, Trinity, or central strata are not at risk of direct exposure to the toxics listed in Table 1.1 in association with this action.

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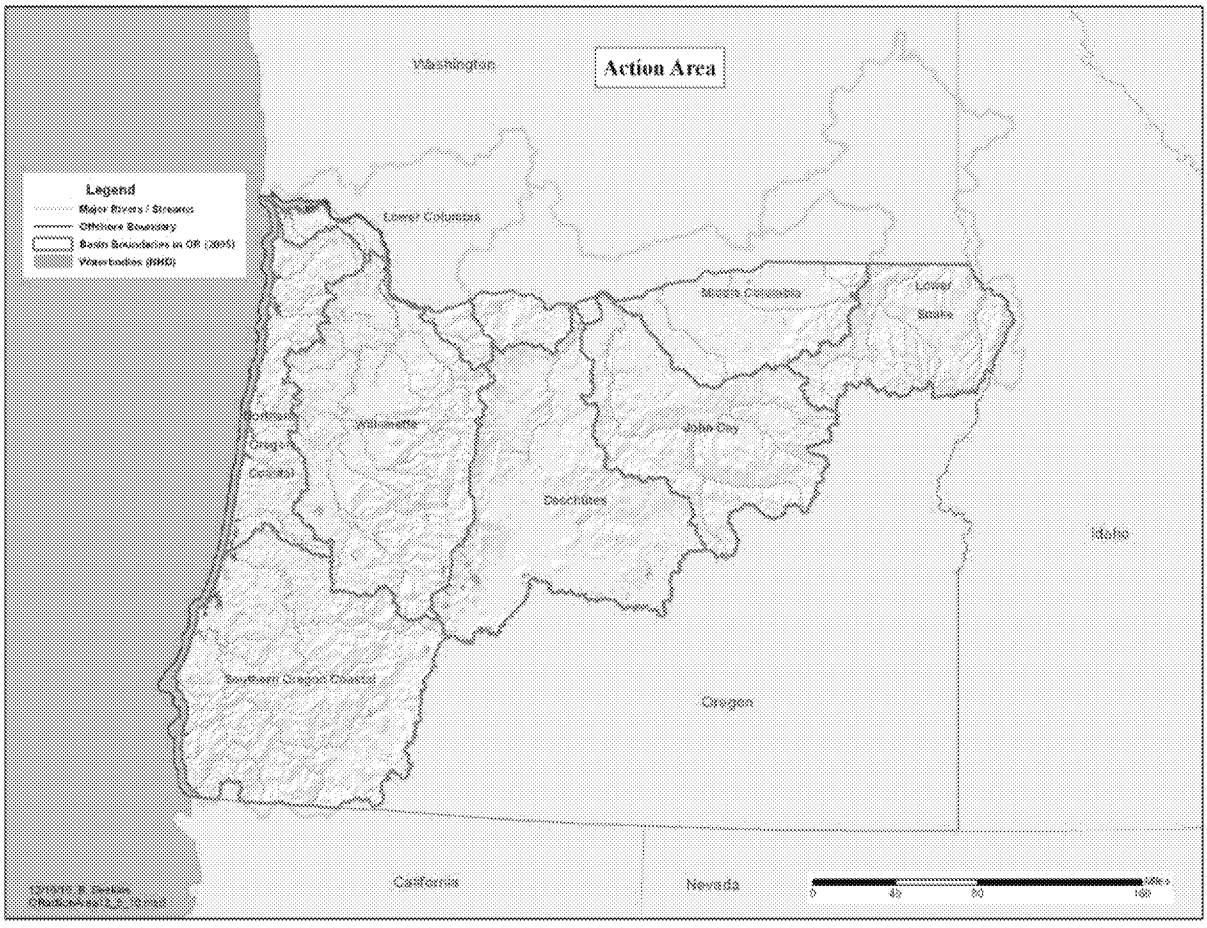

Figure 1.4.1. Overview of the of the action area (highlighted subbasins and the Pacific Ocean, not inclusive of the action area for Southern Resident killer whales).

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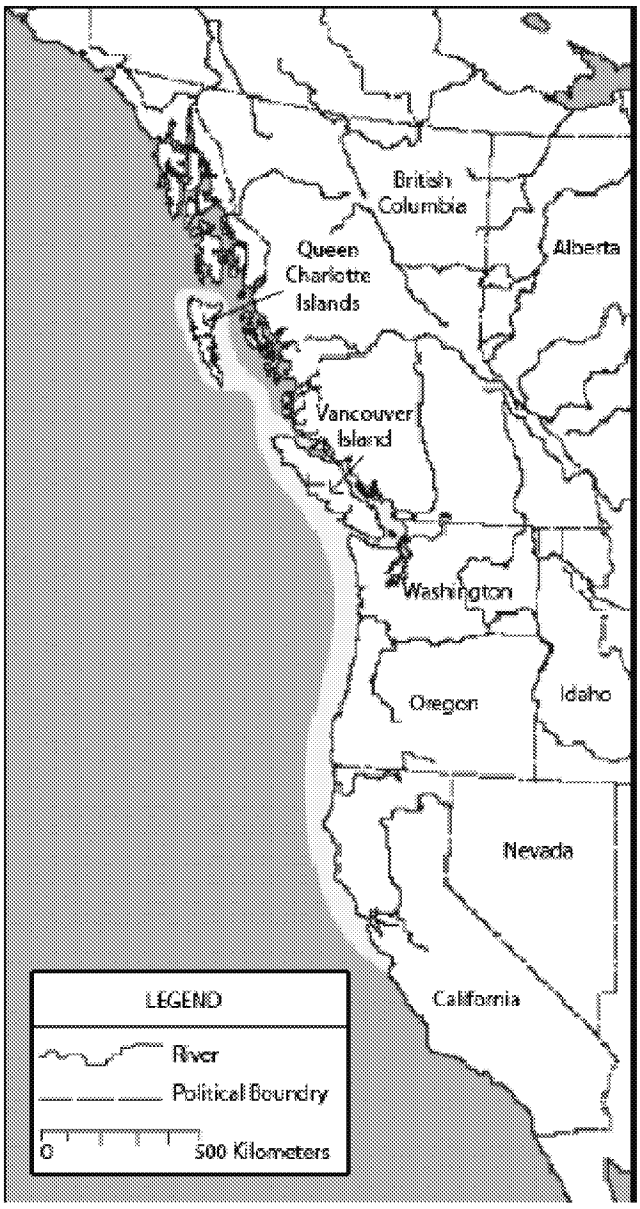

Figure 1.4.2. Action area (light shading) for southern resident killer whales. Reprinted from Wiles (2004).

2. ENDANGERED SPECIES ACT BIOLOGICAL OPINION AND INCIDENTAL TAKE STATEMENT

The ESA establishes a national program for conserving threatened and endangered species of fish, wildlife, plants, and the habitat on which they depend. Section 7(a)(2) of the ESA requires Federal agencies to consult with the U.S. Fish and Wildlife Service, NMFS, or both, to ensure that their actions are not likely to jeopardize the continued existence of endangered or threatened species or adversely modify or destroy their designated critical habitat. Section 7(b)(3) requires that at the conclusion of consultation, the Service provide an opinion stating how the agencies' actions will affect listed species or their critical habitat. If incidental take is expected, section 7(b)(4) requires the provision of an incidental take statement (ITS) specifying the impact of any incidental taking, and including reasonable and prudent measures to minimize such impacts.

2.1 Introduction to the Biological Opinion

Section 7(a)(2) of the ESA requires Federal agencies, in consultation with NMFS, to insure that their actions are not likely to jeopardize the continued existence of endangered or threatened species, or adversely modify or destroy their designated critical habitat. The jeopardy analysis considers both survival and recovery of the species. The adverse modification analysis considers the impacts to the conservation value of the designated critical habitat.

"To jeopardize the continued existence of a listed species" means to engage in an action that would be expected, directly or indirectly, to reduce appreciably the likelihood of both the survival and recovery of a listed species in the wild by reducing the reproduction, numbers, or distribution of that species (50 CFR 402.02).

This opinion does not rely on the regulatory definition of "destruction or adverse modification" of critical habitat at 50 CFR 402.02. Instead, we have relied upon the statutory provisions of the ESA to complete the following analysis with respect to critical habitat.[1]

2.2 Approach to the Assessment

We will use the following approach to determine whether the proposed action described in Section 1.4 is likely to jeopardize listed species or destroy or adversely modify critical habitat:

- *Identify the rangewide status of the species and critical habitat likely to be adversely affected by the proposed action.* This section describes the current status of each listed species and its critical habitat relative to the conditions needed for recovery. For listed salmon and steelhead, NMFS has developed specific guidance for analyzing the status of the listed species' component populations in a "viable salmonid populations" paper (VSP; McElhany *et al.* 2000). The VSP approach considers the abundance, productivity, spatial structure, and diversity of each population as part of the overall review of a species' status. For listed salmon and steelhead, the VSP criteria therefore encompass the

[1] Memorandum from William T. Hogarth to Regional Administrators, Office of Protected Resources, NMFS (Application of the "Destruction or Adverse Modification" Standard Under Section 7(a)(2) of the Endangered pecies Act) (November 7, 2005).

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species' "reproduction, numbers, or distribution" (50 CFR 402.02). In describing the range-wide status of listed species, we rely on viability assessments and criteria in technical recovery team documents and recovery plans, where available, that describe how VSP criteria are applied to specific populations, major population groups, and species. We determine the rangewide status of critical habitat by examining the condition of its physical or biological features (also called "primary constituent elements" or PCEs in some designations) – which were identified when the critical habitat was designated. Species and critical habitat status are discussed in Section 2.4 of this opinion.

- *Describe the environmental baseline for the proposed action.* The environmental baseline includes the past and present impacts of Federal, state, or private actions and other human activities *in the action area.* It includes the anticipated impacts of proposed Federal projects that have already undergone formal or early section 7 consultation and the impacts of state or private actions that are contemporaneous with the consultation in process. The environmental baseline is discussed in section 2.5 of this opinion.

- *Analyze the effects of the proposed actions.* In this step, NMFS considers how the proposed action would affect the species' reproduction, numbers, and distribution or, in the case of salmon and steelhead, their VSP characteristics.

- *Analyze the effects of the proposed actions.* In this step, NMFS considers how the proposed action would affect the conservation value of critical habitat for the affected species.

- *Describe any cumulative effects.* Cumulative effects, as defined in NMFS' implementing regulations (50 CFR 402.02), are the effects of future state or private activities, not involving Federal activities, that are reasonably certain to occur within the action area. Future Federal actions that are unrelated to the proposed action are not considered because they require separate section 7 consultation. Cumulative effects are considered in Section 2.6.8 of this opinion.

- *Integrate and synthesize the above factors to assess the risk that the proposed action poses to species and critical habitat.* In this step, NMFS adds the effects of the action (section 2.6) to the environmental baseline (section 2.5) and the cumulative effects (section 2.6.8) to assess whether the action could reasonably be expected to: (1) appreciably reduce the likelihood of both survival and recovery of the species in the wild by reducing its numbers, reproduction, or distribution; or (2) reduce the value of designated or proposed critical habitat for the conservation of the species. These assessments are made in full consideration of the status of the species and critical habitat (section 2.4). Integration and synthesis occurs in section 2.7 of this opinion.

- *Reach jeopardy and adverse modification conclusions.* Conclusions regarding jeopardy and the destruction or adverse modification of critical habitat are presented in section 2.9 of this opinion. These conclusions flow from the logic and rationale presented in the Integration and Synthesis section (2.7) of this opinion.

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- *If necessary, define a reasonable and prudent alternative to the proposed action.* If, in completing the last step in the analysis, NMFS determines that the action under consultation is likely to jeopardize the continued existence of listed species or destroy or adversely modify designated critical habitat, NMFS must identify a reasonable and prudent alternative (RPA) to the action. The RPA must not be likely to jeopardize the continued existence of ESA-listed species nor destroy or adversely modify their designated critical habitat, and it must meet other regulatory requirements.

2.3. Species and Critical Habitat not considered further in this Opinion

In this opinion NMFS concludes that the proposed action is not likely to adversely affect (NLAA) Steller sea lions, humpback whales, blue whales, fin whales, Sei whales, sperm whales, North Pacific Right whales, loggerhead sea turtles, green sea turtles, leatherback sea turtles, and Olive Ridley sea turtles. Refer to section 2.14 for NLAA determinations.

2.4 Rangewide Status of the Species and Critical Habitat

The summaries that follow describe the status of the listed species, and their designated critical habitats, that occur within the action area of this proposed action and are considered in this opinion. More detailed information on the status and trends of these listed resources, and their biology and ecology, can be found in the listing regulations and critical habitat designations published in the Federal Register (Table 1.4.1 and Table 1.4.2, above).

2.4.1 Climate Change

Climate change is likely to play an increasingly important role in determining the abundance of listed species, and the conservation value of designated critical habitats, in the Pacific Northwest. These changes will not be spatially homogeneous across the Pacific Northwest. Areas with elevations high enough to maintain temperatures well below freezing for most of the winter and early spring would be less affected. Low-lying areas that historically have received scant precipitation contribute little to total streamflow and are likely to be more affected.

During the last century, average regional air temperatures increased by 1.5°F, and increased up to 4°F in some areas (USGCRP 2009). Warming is likely to continue during the next century as average temperatures increase another 3 to 10°F (USGCRP 2009). Overall, about one-third of the current cold-water fish habitat in the Pacific Northwest is likely to exceed key water temperature thresholds by the end of this century (USGCRP 2009).

Precipitation trends during the next century are less certain than for temperature but more precipitation is likely to occur during October through March and less during summer months, and more of the winter precipitation is likely to fall as rain rather than snow (ISAB 2007, USGCRP 2009). Where snow occurs, a warmer climate will cause earlier runoff so stream flows in late spring, summer, and fall will be lower and water temperatures will be warmer (ISAB 2007, USGCRP 2009).

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Higher winter stream flows increase the risk that winter floods in sensitive watersheds will damage spawning redds and wash away incubating eggs (USGCRP 2009). Earlier peak stream flows will also flush some young salmon and steelhead from rivers to estuaries before they are physically mature, increasing stress and the risk of predation (USGCRP 2009). Lower stream flows and warmer water temperatures during summer will degrade summer rearing conditions, in part by increasing the prevalence and virulence of fish diseases and parasites (USGCRP 2009). Other adverse effects are likely to include altered migration patterns, accelerated embryo development, premature emergence of fry, variation in quality and quantity of tributary rearing habitat, and increased competition and predation risk from warm-water, non-native species (ISAB 2007).

The earth's oceans are also warming, with considerable interannual and inter-decadal variability superimposed on the longer-term trend (Bindoff *et al.* 2007). Historically, warm periods in the coastal Pacific Ocean have coincided with relatively low abundances of salmon and steelhead, while cooler ocean periods have coincided with relatively high abundances (Scheuerell and Williams 2005, Zabel *et al.* 2006, USGCRP 2009). Ocean conditions adverse to salmon and steelhead may be more likely under a warming climate (Zabel *et al.* 2006).

2.4.2 Status of the Species

The status of species and critical habitat sections below are organized under four recovery domains (Table 2.4.2.1) to better integrate recovery planning information that NMFS is developing on the conservation status of the species and critical habitats considered in this consultation. Recovery domains are the geographically-based areas that NMFS is using to prepare multi-species recovery plans. Southern green sturgeon are under the jurisdiction of NMFS' Southwest Region. The first meeting of the recovery team for this species was announced to be held in December, 2009. A recovery team has not yet been convened for eulachon, a species under the jurisdiction of NMFS' Northwest Region. Green sturgeon and eulachon may occur in multiple recovery domains.

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Table 2.4.2.1. Recovery planning domains identified by NMFS and their ESA-listed salmon and steelhead species.

Recovery Domain	Species
Willamette-Lower Columbia (WLC)	LCR Chinook salmon UWR Chinook salmon CR chum salmon LCR coho salmon LCR steelhead UWR steelhead
Interior Columbia (IC)	UCR spring-run Chinook salmon SR spring/summer Chinook salmon SR fall-run Chinook salmon SR sockeye salmon UCR steelhead MCR steelhead SRB steelhead
Oregon Coast (OC)	OC coho salmon
Southern Oregon/Northern California Coasts (SONCC)	SONCC coho salmon

For each recovery domain, a technical review team (TRT) appointed by NMFS has developed, or is developing, criteria necessary to identify independent populations within each species, recommended viability criteria for those species, and descriptions of factors that limit species survival. Viability criteria are prescriptions of the biological conditions for populations, biogeographic strata, and ESUs that, if met, would indicate that the ESU will have a negligible risk of extinction over a 100-year time frame.[2]

The definition of a population used by each TRT to analyze salmon and steelhead is set forth in the "viable salmonid population" document prepared by NMFS for use in conservation assessments of Pacific salmon and steelhead (McElhany *et al.* 2000). That document defines population viability in terms of four variables: abundance, population growth rate (productivity), population spatial structure, and genetic diversity.

Abundance is of obvious importance since, in general, small populations are at greater risk of extinction than large populations, primarily because many processes that affect population dynamics may operate differently in small populations than in large populations (Shaffer 1987, McElhany *et al.* 2000).

[2] For Pacific salmon, NMFS uses its 1991 ESU policy, that states that a population or group of populations will be considered a distinct population segment if it is an evolutionarily significant unit (ESU). An ESU represents a distinct population segment of Pacific salmon under the Endangered Species Act that 1) is substantially reproductively isolated from conspecific populations and 2) represents an important component of the evolutionary legacy of the species. The species *O. mykiss* is under the joint jurisdiction of NMFS and the Fish and Wildlife Service, so in making its listing January, 2006 determinations NMFS elected to use the 1996 joint FWS-NMFS DPS policy for this species.

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Population growth rate, the productivity over the entire life cycle, and factors that affect population growth rate provide information about how well a population is performing in the various habitats it occupies during the life cycle. Examining population growth rate allows one to assess if populations are able to replace themselves. Populations that consistently fail to replace themselves are at greater risk of extinction than populations that are consistently at or above replacement levels.

Spatial structure refers to the distribution of individuals within a population at a certain life stage throughout the available habitats, recognizing the abiotic and biotic processes that give rise to that structure. McElhany *et al.* (2000) gave two main reasons why spatial structure is important to consider when evaluating population viability: 1) overall extinction risk at longer time scales may be affected in ways not apparent from short-term observations of abundance and productivity, because there can be a time lag between changes in spatial structure and the resulting population-level effects, and 2) spatial population structure affects the ability of a population to respond to changing environmental conditions and therefore can influence evolutionary processes. Maintaining spatial structure within a population, and its associated benefits to viability, requires appropriate habitat conditions and suitable corridors linking the habitat and the marine environment to be consistently available.

Diversity relates to the variability of phenotypic characteristics such as life histories, individual size, fecundity, run timing, and other attributes exhibited by individuals and populations, as well as the genetic diversity that may underlie this variation. There are many reasons diversity is important in a spatially and temporally varying environment. Three key reasons are: (1) Diversity allows a species to use a wide array of environments; (2) diversity protects a species against short-term spatial and temporal changes in the environment; and (3) genetic diversity provides the raw material for surviving long-term environmental change (McElhany *et al.* 2000).

Although the TRTs operated from the common set of biological principals described in McElhany *et al.* (2000), they worked semi-independently from each other and developed criteria suitable to the species and conditions found in their specific recovery domains. All of the criteria have qualitative as well as quantitative aspects. The diversity of salmonid species and populations makes it impossible to set narrow quantitative guidelines that will fit all populations in all situations. For this and other reasons, viability criteria vary among species, mainly in the number and type of metrics and the scales at which the metrics apply (*i.e.*, population, major population group (MPG, or strata, or ESU) (Busch *et al.* 2008).

Overall viability risk scores (high to low) are based on combined ratings for the abundance and productivity (A/P) and spatial structure and diversity[3] (SS/D) metrics. WLC scores (Table 2.4.2.2) are based on population persistence established by McElhany *et al.* (2006). IC-TRT viability criteria were based on (McElhany *et al.* 2000 and 2006), as well as the results of previous applications in other TRTs and a review of specific information available relative to listed IC ESU populations (IC-TRT 2007). The A/P score considers the TRT's estimate of a populations' minimum threshold population, natural spawning abundance and the productivity of

[3] The WLC-TRT provided ratings for diversity and spatial structure risks. The IC-TRT provided spatial structure and diversity ratings combined as an integrated SS/D risk.

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the population. Productivity over the entire life cycle and factors that affect population growth rate provide information on how well a population is "performing" in the habitats it occupies during the life cycle. Estimates of population growth rate that indicate a population is consistently failing to replace itself are an indicator of increased extinction risk. The four metrics (abundance, productivity, spatial structure, and diversity) are not independent of one another and their relationship to sustainability depends on a variety of interdependent ecological processes (Wainwright *et al.* 2008).

Table 2.4.2.2. Population persistence categories from McElhany *et al.* (2006). A low or negligible risk of extinction is considered "viable" (Ford *et al.* 2011). Population persistence categories correspond to: 4 = very low (VL), 3 = low (L), 2 = moderate (M), 1 = high (H), and 0 = very high (VH) in Oregon populations, which corresponds to "extirpated or nearly so" (E) in Washington populations (Ford *et al.* 2011).

Population Persistence Category	Probability of population persistence in 100 years	Probability of population extinction in 100 years	Description
0	0-40%	60-100%	Either extinct or "high" risk of extinction
1	40-75%	25-60%	Relatively "high" risk of extinction in 100 years
2	75-95%	5-25%	"Moderate" risk of extinction in 100 years
3	95-99%	1-5%	"Low" (negligible) risk of extinction in 100 years
4	>99%	<1%	"Very low" risk of extinction in 100 years

Integrated SS/D risk combines risk for likely, future environmental conditions, and diversity (McElhany *et al.* 2000, McElhany *et al.* 2007, Ford *et al.* 2011). Diversity factors include:

- Life history traits: Distribution of major life history strategies within a population, variability of traits, mean value of traits, and loss of traits.
- Effective population size: One of the indirect measures of diversity is effective population size. A population at chronic low abundance or experiencing even a single episode of low abundance can be at higher extinction risk because of loss of genetic variability, inbreeding and the expression of inbreeding depression, or the effects of mutation accumulation.
- Impact of hatchery fish: Interbreeding of wild populations and hatchery origin fish can be a significant risk factor to the diversity of wild populations if the proportion of hatchery fish in the spawning population is high and their genetic similarity to the wild population is low.
- Anthropogenic mortality: The susceptibility to mortality from harvest or habitat alterations will differ depending on size, age, run timing, disease resistance or other traits.
- Habitat diversity: Habitat characteristics have clear selective effects on populations, and changes in habitat characteristics are likely to eventually lead to genetic changes through

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selection for locally adapted traits. In assessing risk associated with altered habitat diversity, historical diversity is used as a reference point.

The boundaries of each population were defined using a combination of genetic information, geography, life-history traits, morphological traits, and population dynamics that indicate the extent of reproductive isolation among spawning groups. The overall viability of a species is a function of the VSP attributes of its constituent populations. Until a viability analysis of a species is completed, the VSP guidelines recommend that all populations should be managed to retain the potential to achieve viable status to ensure a rapid start along the road to recovery, and that no significant parts of the species are lost before a full recovery plan is implemented (McElhany *et al.* 2000).

The size and distribution of the species and their component populations considered in this opinion generally have declined over the last few decades due to natural phenomena and human activity, including climate change (as described in section 2.4.1), the operation of hydropower systems, over-harvest, effects of hatcheries, and habitat degradation. Enlarged populations of terns, seals, California sea lions, and other aquatic predators in the Pacific Northwest may be limiting the productivity of some Pacific salmon and steelhead populations (Ford *et al.* 2011).

Southern distinct population segment (DPS) green sturgeon (southern green sturgeon) occur in all coastal recovery domains, although they only spawn in the Sacramento River system. Therefore, only subadults and adults may be present in recovery domains north of San Francisco Bay. Southern DPS eulachon (eulachon) also occur in all coastal recovery domains. However, the status of these species will only be presented once, with information presented for the Willamette and Lower Columbia (WLC) recovery domain. Each species consist of a single population.

Viability status is described below for each of the populations considered in this opinion.

Southern Green Sturgeon. Two DPSs have been defined for green sturgeon (*Acipenser medirostris*), a northern DPS (spawning populations in the Klamath and Rogue rivers) and a southern DPS (spawners in the Sacramento River). There are no empirical data on population size and trends for green sturgeon in the Southern DPS. The estimated abundance (based on the percent of viable spawners) was 1,500 (NMFS 2010). Southern green sturgeon includes all naturally-spawned populations of green sturgeon that occur south of the Eel River in Humboldt County, California. When not spawning, this anadromous species is broadly distributed in nearshore marine areas from Mexico to the Bering Sea. Although it is commonly observed in bays, estuaries, and sometimes the deep riverine mainstem in lower elevation reaches of non-natal rivers along the west coast of North America, the distribution and timing of estuarine use are poorly understood.

Southern green sturgeon occur in the Willamette and Lower Columbia (WLC), Oregon Coast (OC), and Southern Oregon/Northern California Coasts (SONCC) recovery domains. The principal factor for the decline of southern green sturgeon is the reduction of its spawning area to a single known population limited to a small portion of the Sacramento River. It is currently at risk of extinction primarily because of human-induced ''takes'' involving elimination of

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freshwater spawning habitat, degradation of freshwater and estuarine habitat quality, water diversions, fishing, and other causes (USDC 2010). Adequate water flow and temperature are issues of concern. Water diversions pose an unknown but potentially serious threat within the Sacramento and Feather Rivers and the Sacramento River Delta. Poaching also poses an unknown but potentially serious threat because of high demand for sturgeon caviar. The effects of contaminants and nonnative species are also unknown but potentially serious threats. Retention of green sturgeon in both recreational and commercial fisheries is now prohibited within the western states, but the effect of capture/release in these fisheries is unknown. There is evidence of fish being retained illegally, although the magnitude of this activity likely is small (NOAA Fisheries 2011).

The viability of this species is still under assessment.

Eulachon. The southern distinct population segment of eulachon occur in four salmon recovery domains: Puget Sound, the Willamette and Lower Columbia, Oregon Coast, and Southern Oregon/Northern California Coasts. The 5-year geometric mean abundance (2006-2010) for eulachon (based on converting fish landings per pound to numbers of fish at 10.8 fish per pound) was 879,669 (NMFS 2010a). The ESA-listed population of eulachon includes all naturally-spawned populations that occur in rivers south of the Nass River in British Columbia to the Mad River in California. Core populations for this species include the Fraser River, Columbia River and (historically) the Klamath River. Eulachon leave saltwater to spawn in their natal streams late winter through early summer, and typically spawn at night in the lower reaches of larger rivers fed by snowmelt. After hatching, larvae are carried downstream and widely dispersed by estuarine and ocean currents. Eulachon movements in the ocean are poorly known although the amount of eulachon bycatch in the pink shrimp fishery seems to indicate that the distribution of these organisms overlap in the ocean.

In the early 1990s, there was an abrupt decline in the abundance of eulachon returning to the Columbia River with no evidence of returning to their former population levels since then (Drake *et al.* 2008). Persistent low returns and landings of eulachon in the Columbia River from 1993 to 2000 prompted the states of Oregon and Washington to adopt a Joint State Eulachon Management Plan in 2001 that provides for restricted harvest management when parental run strength, juvenile production, and ocean productivity forecast a poor return (WDFW and ODFW 2001). Despite a brief period of improved returns in 2001–2003, the returns and associated commercial landings have again declined to the very low levels observed in the mid-1990s (JCRMS 2009), and since 2005, the fishery has operated at the most conservative level allowed in the management plan (JCRMS 2009). Large commercial and recreational fisheries have occurred in the Sandy River in the past. The most recent commercial harvest in the Sandy River was in 2003. No commercial harvest has been recorded for the Grays River from 1990 to the present, but larval sampling has confirmed successful spawning in recent years (USDC 2011a).

The primary factors responsible for the decline of the southern DPS of eulachon are changes in ocean conditions due to climate change (Gustafson *et al.* 2010, Gustafson *et al.* 2011), particularly in the southern portion of its range where ocean warming trends may be the most pronounced and may alter prey, spawning, and rearing success. Additional factors include climate-induced change to freshwater habitats, dams and water diversions (particularly in the

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Columbia and Klamath Rivers where hydropower generation and flood control are major activities), and bycatch of eulachon in commercial fisheries (NOAA Fisheries 2011). Other limiting factors include (Gustafson *et al.* 2010, Gustafson *et al.* 2011):

- Adverse effects related to dams and water diversions
- Artificial fish passage barriers
- Increased water temperatures, insufficient streamflow
- Altered sediment balances
- Water pollution
- Over-harvest
- Predation

Willamette-Lower Columbia Recovery Domain. Species in the Willamette-Lower Columbia (WLC) Recovery Domain include LCR Chinook salmon, UWR Chinook salmon, CR chum salmon, LCR coho salmon, LCR steelhead, UWR steelhead, southern green sturgeon, and eulachon. The WLC-TRT has identified 107 demographically independent populations of Pacific salmon and steelhead (Table 2.4.2.3). These populations were further aggregated into strata, groupings above the population level that are connected by some degree of migration, based on ecological subregions. All 107 populations use parts of the mainstem of the Columbia River and the Columbia River estuary for migration, rearing, and smoltification.

Table 2.4.2.3. Populations in the WLC recovery domain.

Species	Populations
LCR Chinook salmon	32
UWR Chinook salmon	7
CR chum salmon	17
LCR coho salmon	24
LCR steelhead	26
UWR steelhead	4

LCR Chinook Salmon. This species includes all naturally-spawned populations of Chinook salmon in the Columbia River and its tributaries from its mouth at the Pacific Ocean upstream to a transitional point between Washington and Oregon east of the Hood River and the White Salmon River; the Willamette River to Willamette Falls, Oregon, exclusive of spring-run Chinook salmon in the Clackamas River; and progeny of seventeen artificial propagation programs. LCR Chinook populations exhibit three different life history types base on return timing and other features: fall-run (a.k.a. "tules"), late-fall-run (a.k.a. "brights"), and spring-run. The WLC-TRT identified 32 historical populations of LCR Chinook salmon; seven in the Coast Range, six in the Columbia Gorge, and 19 in the Cascade Range (Table 2.4.2.4). The 5-year geometric mean abundance for LCR Chinook salmon (2005-2009) was 31,305 total spawners (NOAA 2011, CBFWA 2011).

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Table 2.4.2.4. LCR Chinook salmon strata, ecological subregions, run timing, populations, and scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH) in Oregon populations. VH corresponds to "extirpated or nearly so" (E) in Washington populations.

Stratum		Spawning Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Viability Risk
Ecological Subregion	Run Timing					
Coast Range	Fall	Grays River (WA)	E	E	L	E
		Elochoman River (WA)	E	H	L	E
		Mill, Germany, and Abernathy creeks (WA)	E	H	L	E
		Young Bay (OR)	H to VH	H	L	VH
		Big Creek (OR)	H to VH	H	L to M	VH
		Clatskanie River (OR)	H	M to H	L	VH
		Scappoose River (OR)	H to VH	M to H	L to M	VH
Columbia Gorge	Spring	White Salmon River (WA)	E	E	E	E
		Hood River (OR)	VH	VH	L	VH
	Fall	Upper Gorge (OR)	E	H	H	VH
		Upper Gorge (WA)	H to VH	H	L to M	E
		White Salmon River (WA)	E	H	H	E
		Lower Gorge (OR)	H to VH	H	L to M	VH
		Lower Gorge (WA)	E	H	H	E
		Hood River (OR)	H to VH	H to VH	L	VH
Cascade Range	Spring	Upper Cowlitz River (WA)	E	M	H	E
		Cispus River (WA)	E	M	H	E
		Tilton River (WA)	E	E	E	E
		Toutle River (WA)	E	H	L	E
		Kalama River (WA)	E	H	L	E
		Sandy River (OR)	M to H	L to M	M	M
		Lewis (WA)	E	M	H	E
	Fall	Lower Cowlitz River (WA)	E	M	M	E
		Upper Cowlitz River (WA)	E	M	E	E
		Lewis River (WA)	E	L	M	E
		Salmon Creek (OR)	E	M	M	E
		Sandy River (OR)	H to VH	H	L	VH
		Toutle River (WA)	E	M	M	E
		Coweeman River (WA)	E	L	M	E
		Kalama River (WA)	E	M	L	E
		Clackamas River (OR)	H to VH	H	L	H
		Washougal River (WA)	E	M	M	E
	Late Fall	Lewis River (WA)	VL	L	L	VL
		Sandy River (OR)	L	L to M	L	L

A/P ratings for most LCR Chinook salmon populations are currently "high" risk to "extirpated or nearly so." Spatial structure was generally rated "low" to "moderate" risk for most populations. Other than the Sandy River, Oregon LCR Chinook salmon populations were rated "high" or "very high" risk for diversity. In 2005, diversity risk for Clackamas River and Lower Gorge

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tributary fall Chinook salmon was rated "moderate"; now the risk is rated "high." Most Washington LCR Chinook salmon populations are currently at "moderate" or "high" risk for diversity (Table 2.4.2.4).

Of the 32 historical populations in the ESU, 28 are extirpated or at "very high" risk. Based on the recovery plan analyses, all of the tule populations are "very high" risk except one that is considered at "high" risk. The modeling conducted in association with tule harvest management suggests that three of the populations (Coweeman, Lewis and Washougal) are at a somewhat lower risk. However, even these more optimistic evaluations suggest that the remaining 18 populations are at substantial risk because of very low natural origin spawner abundance (<100/population), high hatchery fraction, habitat degradation and harvest impacts (Ford *et al.* 2011).

Limiting factors and threats to LCR Chinook salmon include (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and near-shore marine habitat resulting from cumulative impacts of land use and flow management by the Columbia River hydropower system Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas, stream substrate, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development.
- Reduced access to spawning and rearing habitat mainly as a result of tributary hydropower projects
- Hatchery-related effects
- Harvest-related effects on fall Chinook salmon
- An altered flow regime and Columbia River plume has altered the temperature regime and estuarine food web, and has reduced ocean productivity
- Reduced access to off-channel rearing habitat in the lower Columbia River
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Juvenile fish strandings that result from ship wakes
- Contaminants affecting fish health and reproduction

CR Chum Salmon. This species includes all naturally-spawned populations of chum salmon in the Columbia River and its tributaries in Washington and Oregon, and progeny of three artificial propagation programs. The WLC-TRT identified 17 historical populations of CR chum salmon and aggregated these into four strata (Myers *et al.* 2006; Table 2.4.2.5). Unlike other species in the WLC recovery domain, CR chum salmon spawning aggregations were identified in the mainstem Columbia River. These aggregations generally were included in the population associated with the nearest river basin. Three strata and eight historical populations of CR chum salmon occur within the action area (Table 2.4.2.5); of these, none are "viable" (McElhany *et al.* 2007). The 5-year geometric mean abundance for CR chum salmon (2005-2009) was 4,068 total spawners (NOAA 2011, CBFWA 2011).

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Table 2.4.2.5. CR chum salmon strata, ecological subregions, run timing, populations, and scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk (Ford *et al.* 2011). Risk ratings are very low (VL), low (L), moderate (M), high (H), and "extirpated or nearly so" (E).

Stratum		Spawning Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Viability Risk
Ecological Subregion	Run Timing					
Coast Range	Fall	Young's Bay (OR)	*	*	*	*
		Grays River (WA)	VL	L	M	M
		Big Creek (OR)	*	*	*	*
		Elochoman River (WA)	E	E	L	E
		Clatskanie River (OR)	*	*	*	*
		Mill, Abernathy and Germany creeks (WA)	E	E	L	E
		Scappoose Creek (OR)	*	*	*	*
Columbia Gorge	Fall	Lower Gorge (OR)	*	*	*	*
		Lower Gorge (WA)	VL	VL	L	L
		Upper Gorge (OR)	*	*	*	*
		Upper Gorge (WA)	E	E	H	E
Cascade Range	Summer	Cowlitz River (WA)	E	E	H	E
	Fall	Cowlitz River (WA)	E	E	L	E
		Kalama River (WA)	E	E	L	E
		Salmon Creek (WA)	E	E	H	E
		Lewis River (WA)	E	E	L	E
		Clackamas River (OR)	*	*	*	*
		Washougal River (WA)	E	E	L	E
		Sandy River (OR)	*	*	*	*

* No viability risk was completed for Oregon chum salmon populations. Oregon rivers have occasional reports of a few chum salmon. Populations are functionally extinct, or the risk of extinction is very high.

The vast majority (14 out of 17) chum salmon populations remain "extirpated or nearly so". The Grays River and Lower Gorge populations showed a sharp increase in 2002, but have since declined back to relatively low abundance levels in the range of variation observed over the last several decades. Chinook and coho salmon populations in the Lower Columbia and Willamette similarly increased in the early 2000s, then declined to typical recent levels, suggesting the increase in chum salmon may be related to ocean conditions. The Grays and Lower Gorge populations were rated "very low" risk for A/P, but all other populations were rated "extirpated or nearly so." Spatial structure was rated "low" for seven populations, one was has moderate risk and three have a "high" risk. Diversity risk was "high" for all populations except Grays ("moderate") and Lower Gorge ("very low"). Recent data on the Washougal/mainstem Columbia population are not available, but they likely follow a pattern similar to the Grays and Lower Gorge populations (Ford *et al.* 2011).

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Limiting factors and threats to CR chum salmon include (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and nearshore marine habitat resulting from cumulative impacts of land use and flow management by the Columbia River hydropower system
- Degraded freshwater habitat, in particular of floodplain connectivity and function, channel structure and complexity, stream substrate, and riparian areas and large wood recruitment as a result of cumulative impacts of agriculture, forestry, and development
- Degraded stream flow as a result of hydropower and water supply operations
- Loss of access and loss of some habitat types as a result of passage barriers such as roads and railroads
- Reduced water quality
- Current or potential predation from hatchery-origin salmonids, including coho salmon
- An altered flow regime and Columbia River plume has altered the temperature regime and estuarine food web, and has reduced ocean productivity
- Reduced access to off-channel rearing habitat in the lower Columbia River
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Juvenile fish strandings that result from ship wakes
- Contaminants affecting fish health and reproduction

LCR Coho Salmon. This species includes all naturally-spawned populations of coho salmon in the Columbia River and its tributaries in Washington and Oregon, from the mouth of the Columbia up to and including the Big White Salmon and Hood rivers; in the Willamette River to Willamette Falls, Oregon; and progeny of 25 artificial propagation programs. The WLC-TRT identified 24 historical populations of LCR coho salmon and divided these into two strata based on major run timing: early and late (Myers *et al.* 2006). Three strata and nine historical populations of LCR coho salmon occur within the action area (Table 2.4.2.6). Of these nine populations, Clackamas River is the only population characterized as "viable" (McElhany *et al.* 2007). The 5-year geometric mean abundance for LCR coho salmon (2004-2008) was 6,375 total spawners (NOAA 2011, CBFWA 2011).

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Table 2.4.2.6. LCR coho salmon strata, ecological subregions, run timing, populations, and scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH) in Oregon populations. VH corresponds to "extirpated or nearly so" (E) in Washington populations.

Stratum		Spawning Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Viability Risk
Ecological Subregion	Run Type					
Coast Range	N*	Young's Bay (OR)	VH	VH	L	VH
		Big Creek (OR)	VH	H	L to M	VH
		Clatskanie River (OR)	H to VH	M	L	H
		Scappoose River (OR)	M to H	M	L to M	M
		Grays River (WA)	E	E	L	E
		Elochoman Creek (WA)	E	E	L	E
		Mill, Germany, and Abernathy Creeks (WA)	E	H	L	E
Columbia Gorge	N	Lower Gorge Tributaries (OR)	VH	H	L to M	VH
		Lower Gorge Tributaries (WA)	E	E	M	E
	S**	Upper Gorge Tributaries (WA)	E	E	M	E
		Hood River (OR)	VH	H	L	H
Cascade Range	N	Lower Cowlitz River (WA)	E	M	M	E
		Coweeman River (WA)	E	M	L	E
		Salmon Creek (WA)	E	E	M	E
	N and S	Upper Cowlitz River (WA)	E	H	M	E
		Cispus River (WA)	E	H	M	E
		Tilton River (WA)	E	H	M	E
		South Fork Toutle River (WA)	E	M	L	E
		North Fork Toutle River (WA)	E	H	M	E
		Kalama River (WA)	E	M	L	E
		North Fork Lewis River (WA)	E	H	H	E
		East Fork Lewis River (WA)	E	M	L	E
		Washougal River (WA)	E	H	L	E
		Clackamas River (OR)	M	L to M	L	M
		Sandy River (OR)	H	L to M	M to H	H

*"Type N" are late-run fish that tend to undertake oceanic migrations to the north of the Columbia River, extending as far as northern British Columbia and southeast Alaska.
**"Type S" are early coho salmon that spawn in the upper reaches of larger rivers in the lower Columbia River and in most rivers inland of the Cascade Crest that tend to migrate to the south of the Columbia River.

Three status evaluations of LCR coho salmon status, all based on WLC-TRT criteria, have been conducted since the last NMFS status review in 2005 (McElhany *et al.* 2007, Beamesderfer *et al.* 2010, LCFRB 2010). Of the 27 historical populations in the ESU, 24 are at "very high" risk. The remaining three populations (Sandy, Clackamas and Scappoose) are at "moderate" or "high" risk (Ford *et al.* 2011).

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In Oregon, the Scappoose Creek and Clackamas River populations have "moderate" risk ratings for A/P, while the rest are rated "high" or "very high" risk. All of the Washington populations have "extirpated or nearly so" A/P ratings. Spatial diversity is rated "moderate" or "low" risk for all the populations, except the North Fork Lewis River, which has a "high" risk rating for spatial structure. All LCR coho salmon populations, except the Clackamas and Sandy river populations (low risk), are at "moderate" or "high" risk for diversity. All of the Washington side populations are at "very high" risk, although uncertainty is high because of a lack of adult spawner surveys. As was noted in the 2005 status review, smolt traps indicate some natural production in Washington populations, though given the high fraction of hatchery origin spawners suspected to occur in these populations it is not clear that any are self-sustaining (Ford *et al.* 2011).

Limiting factors and threats to LCR coho salmon include (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and near-shore marine habitat resulting from cumulative impacts of land use and flow management by the Columbia River hydropower system
- Fish passage barriers that limit access to spawning and rearing habitats
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Hatchery-related effects
- Harvest-related effects
- An altered flow regime and Columbia River plume has altered the temperature regime and estuarine food web, and has reduced ocean productivity
- Reduced access to off-channel rearing habitat in the lower Columbia River
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Juvenile fish strandings that result from ship wakes
- Contaminants affecting fish health and reproduction

LCR Steelhead. This species includes all naturally-spawned steelhead populations below natural and manmade impassable barriers in streams and tributaries to the Columbia River between and including the Cowlitz and Wind rivers, Washington; in the Willamette and Hood rivers, Oregon; and progeny of ten artificial propagation programs; but excluding all steelhead from the upper Willamette River basin above Willamette Falls, Oregon, and from the Little and Big White Salmon rivers, Washington. Summer steelhead return to freshwater long before spawning. Winter steelhead, in contrast, return from the ocean much closer to maturity and spawn within a few weeks. Summer steelhead spawning areas in the Lower Columbia River are found above waterfalls and other features that create seasonal barriers to migration. Where no temporal barriers exist, the winter-run life history dominates. Six strata and 23 historical populations of LCR steelhead occur within the action area (Table 2.4.2.7). The 5-year geometric mean abundance for LCR steelhead (2006-2010) was 5,863 total spawners (NOAA 2011, CBFWA 2011).

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Table 2.4.2.7. LCR steelhead strata, ecological subregions, run timing, populations, and scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH) in Oregon populations. VH corresponds to "extirpated or nearly so" (E) in Washington populations.

Stratum		Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Viability Risk
Ecological Subregion	**Run Timing**					
Columbia Gorge	Summer	Wind River (WA)	VL	L	VL	L
		Hood River (OR)	H	M	L	VH
	Winter	Lower Gorge (OR)	H	L	L	M to H
		Lower Gorge (WA)	H	M	VL	H
		Upper Gorge (OR)	M	M to H	L	VH
		Upper Gorge (WA)	H	M	M	E
		Hood River (OR)	M	M	L	M
West Cascade Range	Summer	Kalama River (WA)	L	M	VL	M
		North Fork Lewis River (WA)	E	E	E	E
		East Fork Lewis River (WA)	E	M	VL	E
		Washougal River (WA)	M	M	VL	M
	Winter	Cispus River (WA)	E	M	M	E
		Tilton river (WA)	E	H	M	E
		Upper Cowlitz River (WA)	E	M	M	E
		Lower Cowlitz River (WA)	H	M	M	H
		North Fork Toutle River (WA)	E	L	L	E
		South Fork Toutle River (WA)	M	L	VL	M
		Coweeman River (WA)	H	VL	VL	H
		Kalama River (WA)	H	L	VL	H
		North Fork Lewis River (WA)	E	M	M	E
		East Fork Lewis River (WA)	M	M	VL	M
		Salmon Creek (WA)	E	M	VL	E
		Washougal River (WA)	H	M	VL	H
		Sandy River (OR)	H	M	M to H	VH
		Clackamas River (OR)	L	L to M	L	L to M

All of the populations increased in abundance during the early 2000s, generally peaking in 2004. Most populations have since declined back to levels within one standard deviation of the long term mean. Exceptions are the Washougal summer-run and North Fork Toutle winter-run, which are still higher than the long term average, and the Sandy, which is lower (Ford *et al.* 2011).

Limiting factors and threats to LCR steelhead include (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and nearshore marine habitat resulting from cumulative impacts of land use and flow management by the Columbia River hydropower system
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and recruitment of large wood, stream substrate, stream flow,

and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development

- Reduced access to spawning and rearing habitat mainly as a result of tributary hydropower projects and lowland development
- Avian and marine mammal predation in the lower mainstem Columbia River and estuary.
- Hatchery-related effects
- An altered flow regime and Columbia River plume has altered the temperature regime and estuarine food web, and has reduced ocean productivity
- Reduced access to off-channel rearing habitat in the lower Columbia River
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Juvenile fish strandings that result from ship wakes
- Contaminants affecting fish health and reproduction

UWR Chinook Salmon. This species includes all naturally spawned populations of spring-run Chinook salmon in the Clackamas River; in the Willamette River and its tributaries above Willamette Falls, Oregon; and progeny of seven artificial propagation programs. All seven historical populations of UWR Chinook salmon identified by the WLC-TRT occur within the action area and are contained within a single ecological subregion, the western Cascade Range (Table 2.4.2.8); only the Clackamas population is characterized as "viable" (McElhany *et al.* 2007). The 5-year geometric mean abundance for UWR spring Chinook salmon (2004-2008) was 4,177 total spawners (NOAA 2011, CBFWA 2011).

Table 2.4.2.8. Scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk for UWR Chinook salmon (ODFW and NMFS 2011). All populations are in the Western Cascade Range ecological subregion. Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH).

Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Extinction Risk
Clackamas River	M	M	L	M
Molalla River	VH	H	H	VH
North Santiam River	VH	H	H	VH
South Santiam River	VH	M	M	VH
Calapooia River	VH	H	VH	VH
McKenzie River	VL	M	M	L
Middle Fork Willamette River	VH	H	H	VH

Consideration of data collected since the last status review in 2005 has confirmed the high fraction of hatchery origin fish in all of the populations of this species (even the Clackamas and McKenzie rivers have hatchery fractions above WLC-TRT viability thresholds). All of the UWR Chinook salmon populations have "moderate" or "high" risk ratings for diversity. The Clackamas and McKenzie river populations currently have the best risk ratings for A/P, spatial structure, and diversity. Clackamas River Chinook salmon have a "low" risk rating for spatial structure.

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The new data have also highlighted the substantial risks associated with pre-spawning mortality. Although recovery plans are targeting key limiting factors for future actions, there have been no significant on-the-ground-actions since the last status review to resolve the lack of access to historical habitat above dams nor have there been substantial actions removing hatchery fish from the spawning grounds (Ford *et al.* 2011).

Limiting factors and threats to UWR Chinook salmon include (ODFW and NMFS 2011, NOAA Fisheries 2011):

- Significantly reduced access to spawning and rearing habitat because of tributary dams
- Degraded freshwater habitat, especially floodplain connectivity and function, channel structure and complexity, and riparian areas and large wood recruitment as a result of cumulative impacts of agriculture, forestry, and development
- Degraded water quality and altered temperature as a result of both tributary dams and the cumulative impacts of agriculture, forestry, and urban development
- Hatchery-related effects
- Anthropogenic introductions of non-native species and out-of-ESU races of salmon or steelhead have increased predation on, and competition with, native UWR Chinook salmon
- Ocean harvest rates of approximately 20%

UWR Steelhead. This species includes all naturally-spawned steelhead populations below natural and manmade impassable barriers in the Willamette River, Oregon, and its tributaries upstream from Willamette Falls to the Calapooia River. The WLC-TRT identified five historical populations of UWR steelhead, all with winter-run timing (Myers *et al.* 2006). UWR steelhead are currently found in many tributaries that drain the west side of the upper Willamette River basin. Analysis of historical observations, hatchery records, and genetic analysis strongly suggested that many of these spawning aggregations are the result of recent introductions and do not represent a historical population. Nevertheless, the WLC-TRT recognized that these tributaries may provide juvenile rearing habitat or may be temporarily (for one or more generations) colonized during periods of high abundance. One stratum[4] and five historical populations of UWR steelhead occur within the action area (Table 2.4.2.9), although the west-side tributaries population was included only because it is important to the species as a whole, and not because it is independent. Summer steelhead have become established in the McKenzie River where historically no steelhead existed, although these fish were not considered in the identification of historical populations. Hatchery summer-run steelhead that are produced and released in the subbasins are from an out-of-basin stock and are not part of the DPS (ODFW and NMFS 2011). The 5-year geometric mean abundance for UWR steelhead (2004-2008) was 6,392 total spawners (NOAA 2011, CBFWA 2011).

[4] The WLC-TRT defined the hierarchy by grouping the independent populations into larger aggregates that share similar genetic, geographic (hydrographic and ecoregion), and/or habitat characteristics. They called these "major groupings" stratum (plural: strata).

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Table 2.4.2.9. Scores for the key elements (A/P, diversity, and spatial structure) used to determine current overall viability risk for UWR steelhead (ODFW and NMFS 2011). All populations are in the Western Cascade Range ecological subregion. Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH).

Population (Watershed)	A/P	Diversity	Spatial Structure	Overall Extinction Risk
Molalla River	VL	M	M	L
North Santiam River	VL	M	H	L
South Santiam River	VL	M	M	L
Calapooia River	M	M	VH	M

Since the last status review in 2005, UWR steelhead initially increased in abundance but subsequently declines and current abundance is at the levels observed in the mid-1990s when the DPS was first listed. The DPS appears to be at lower risk than the UWR Chinook salmon ESU, but continues to demonstrate the overall low abundance pattern that was of concern during the last status review. The elimination of winter-run hatchery release in the basin reduces hatchery threats, but non-native summer steelhead hatchery releases are still a concern for species diversity (Ford *et al.* 2011).

Limiting factors and threats to UWR steelhead include (ODFW and NMFS 2011, NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large wood recruitment, and stream flow have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Degraded water quality and altered temperature as a result of both tributary dams and the cumulative impacts of agriculture, forestry, and urban development
- Reduced access to spawning and rearing habitats mainly as a result of artificial barriers in spawning tributaries
- Hatchery-related effects: impacts from the non-native summer steelhead hatchery program
- Anthropogenic introductions of non-native species and out-of-ESU races of salmon or steelhead have increased predation and competition on native UWR steelhead.

Interior Columbia Recovery Domain. Species in the Interior Columbia (IC) recovery domain include UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, SR sockeye salmon, UCR steelhead, MCR steelhead, and SRB steelhead. The IC-TRT identified 82 populations of those species based on genetic, geographic (hydrographic), and habitat characteristics (Table 2.4.2.10). In some cases, the IC-TRT further aggregated populations into "major groupings" based on dispersal distance and rate, and drainage structure, primarily the location and distribution of large tributaries (IC-TRT 2003). All 82 populations identified use the lower mainstem of the Snake River, the mainstem of the Columbia River, and the Columbia River estuary, or part thereof, for migration, rearing, and smoltification.

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Table 2.4.2.10. Populations of ESA-listed salmon and steelhead in the IC recovery domain.

Species	Populations
UCR spring-run Chinook salmon	3
SR spring/summer-run Chinook salmon	31
SR fall-run Chinook salmon	1
SR sockeye salmon	1
UCR steelhead	4
MCR steelhead	17
SRB steelhead	25

The IC-TRT also recommended viability criteria that follow the VSP framework (McElhany *et al.* 2006) and described biological or physical performance conditions that, when met, indicate a population or species has a 5% or less risk of extinction over a 100-year period (IC-TRT 2007; see also NRC 1995).

UCR Spring-run Chinook Salmon. This species includes all naturally-spawned populations of Chinook salmon in all river reaches accessible to Chinook salmon in Columbia River tributaries upstream of the Rock Island Dam and downstream of Chief Joseph Dam in Washington (excluding the Okanogan River), the Columbia River upstream to Chief Joseph Dam in Washington, and progeny of six artificial propagation programs. The IC-TRT identified four independent populations of UCR spring-run Chinook salmon in the upriver tributaries of Wenatchee, Entiat, Methow, and Okanogan (extirpated), but no major groups due to the relatively small geographic area affected (IC-TRT 2003, Ford *et al.* 2011)(Table 2.4.2.11). The 5-year geometric mean abundance for UCR spring-run Chinook salmon (2005-2009) was 3,134 total spawners (NOAA 2011, CBFWA 2011). The current estimate (2003-2008 5-year average) of natural origin spawning abundance ranges from 29% to 46% across populations.

Table 2.4.2.11. Scores for the key elements (A/P, diversity, and SS/D) used to determine current overall viability risk for spring-run UCR Chinook salmon (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH).

Population	A/P	Diversity	Integrated SS/D	Overall Viability Risk
Wenatchee River	H	H	H	H
Entiat River	H	H	H	H
Methow River	H	H	H	H
Okanogan River	n/a	n/a	n/a	n/a

TUCR spring-run Chinook salmon is not currently meeting the viability criteria (adapted from the IC-TRT) in the Upper Columbia recovery plan. A/P remains at "high" risk for each of the three extant populations in this MPG/ESU (Table 2.4.2.11). The 10-year geometric mean abundance of adult natural origin spawners has increased for each population relative to the levels for the 1981-2003 series, but the estimates remain below the corresponding IC-TRT thresholds. Estimated productivity (spawner to spawner return rate at low to moderate

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escapements) was on average lower over the years 1987-2009 than for the previous period. The combinations of current abundance and productivity for each population result in a "high" risk rating. The composite SS/D risks for all three of the extant populations in this MPG are at "high" risk. The spatial processes component of the SS/D risk is "low" for the Wenatchee River and Methow River populations and "moderate" for the Entiat River (loss of production in lower section increases effective distance to other populations). All three of the extant populations in this MPG are at "high" risk for diversity, driven primarily by chronically high proportions of hatchery-origin spawners in natural spawning areas and lack of genetic diversity among the natural-origin spawners (Ford *et al.* 2011).

Increases in natural origin abundance relative to the extremely low spawning levels observed in the mid-1990s are encouraging; however, average productivity levels remain extremely low. Overall, the viability of UCR Chinook salmon has likely improved somewhat since the last status review, but the ESU is still clearly at "moderate-to-high" risk of extinction (Ford *et al.* 2011).

Limiting factors and threats to the UCR spring-run Chinook salmon ESU include (UCSRB 2007, NOAA Fisheries 2011):

- Mainstem Columbia River hydropower–related adverse effects: upstream and downstream fish passage, ecosystem structure and function, flows, and water quality
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Degraded estuarine and nearshore marine habitat
- Hatchery related effects: including past introductions and persistence of non-native (exotic) fish species continues to affect habitat conditions for listed species
- Harvest in Columbia River fisheries

SR Spring/summer-run Chinook Salmon. This species includes all naturally-spawned populations of spring/summer-run Chinook salmon in the mainstem Snake River and the Tucannon River, Grande Ronde River, Imnaha River, and Salmon River subbasins; and progeny of fifteen artificial propagation programs. The IC-TRT identified 27 extant and 4 extirpated populations of SR spring/summer-run Chinook salmon, and aggregated these into major population groups (IC-TRT 2003, Ford *et al.* 2011). Each of these populations faces a "high" risk of extinction (Ford *et al.* 2011) (Table 2.4.2.12). The 5-year geometric mean abundance for SR Spring/Summer Chinook salmon (2005-2009) was 6,365 total spawners (Ford *et al.* 2011). The current estimate (2005-2009 5-year average) of natural origin spawning abundance ranges from 25% to 100% across populations.

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Table 2.4.2.12. SR spring/summer-run Chinook salmon ecological subregions, populations, and scores for the key elements (A/P, diversity, and SS/D) used to determine current overall viability risk for SR spring/summer-run Chinook salmon (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH) and extirpated (E).

Ecological Subregions	Spawning Populations (Watershed)	A/P	Diversity	Integrated SS/D	Overall Viability Risk
Lower Snake River	Tucannon River	H	M	M	H
	Asotin River				E
Grande Ronde and Imnaha rivers	Wenaha River	H	M	M	H
	Lostine/Wallowa River	H	M	M	H
	Minam River	H	M	M	H
	Catherine Creek	H	M	M	H
	Upper Grande Ronde R.	H	M	H	H
	Imnaha River	H	M	M	H
	Big Sheep Creek				E
	Lookingglass Creek				E
South Fork Salmon River	Little Salmon River	*	*	*	H
	South Fork mainstem	H	M	M	H
	Secesh River	H	L	L	H
	EF/Johnson Creek	H	L	L	H
Middle Fork Salmon River	Chamberlin Creek	H	L	L	H
	Big Creek	H	M	M	H
	Lower MF Salmon	H	M	M	H
	Camas Creek	H	M	M	H
	Loon Creek	H	M	M	H
	Upper MF Salmon	H	M	M	H
	Pistol Creek				E
	Sulphur Creek	H	M	M	H
	Bear Valley Creek	H	L	L	H
	Marsh Creek	H	L	L	H
Upper Mainstem Salmon	N. Fork Salmon River	H	L	L	H
	Lemhi River	H	H	H	H
	Pahsimeroi River	H	H	H	H
	Upper Salmon-lower mainstem	H	L	L	H
	East Fork Salmon River	H	H	H	H
	Yankee Fork	H	H	H	H
	Valley Creek	H	M	M	H
	Upper Salmon main	H	M	M	H
	Panther Creek				E
* Insufficient data.					

Population level status ratings remain at high risk across all MPGs within the ESU, although recent natural spawning abundance estimates have increased, all populations remain below

minimum natural origin abundance thresholds (Table 2.4.2.12). Spawning escapements in the most recent years in each series are generally well below the peak returns but above the extreme low levels in the mid-1990s. Relatively low natural production rates and spawning levels below minimum abundance thresholds remain a major concern across the ESU.

The ability of SR spring/summer-run Chinook salmon populations to be self-sustaining through normal periods of relatively low ocean survival remains uncertain. Factors cited by Good *et al.* (2005) remain as concerns or key uncertainties for several populations (Ford *et al.* 2011). Limiting factors and threats to the SR spring/summer-run Chinook salmon ESU include (NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, elevated water temperature, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Mainstem Columbia River and Snake River hydropower impacts
- Harvest-related effects
- Predation

SR Fall-run Chinook Salmon. This species includes all naturally-spawned populations of fall-run Chinook salmon in the mainstem Snake River below Hells Canyon Dam, and in the Tucannon River, Grande Ronde River, Imnaha River, Salmon River, and Clearwater River, and progeny of four artificial propagation programs. The IC-TRT identified three populations of this species, although only the lower mainstem population exists at present, and it spawns in the lower main stem of the Clearwater, Imnaha, Grande Ronde, Salmon and Tucannon rivers. The extant population of Snake River fall-run Chinook salmon is the only remaining population from an historical ESU that also included large mainstem populations upstream of the current location of the Hells Canyon Dam complex (IC-TRT 2003, Ford *et al.* 2011). The 5-year geometric mean abundance for SR fall-run Chinook salmon (2004-2008) was 11,321 total spawners. The current estimate (1999-2008 10-year geometric mean) of natural origin spawning abundance of SR fall-run Chinook is just over 2,200 (Ford *et al.* 2011).

The recent increases in natural origin abundance are encouraging. However, hatchery origin spawner proportions have increased dramatically in recent years – on average, 78% of the estimated adult spawners have been hatchery origin over the most recent brood cycle. The apparent leveling off of natural returns in spite of the increases in total brood year spawners may indicate that density dependent habitat effects are influencing production or that high hatchery proportions may be influencing natural production rates. The A/P risk rating for the population is "moderate." The population is at moderate risk for diversity and spatial structure. (Ford *et al.* 2011). Given the combination of current A/P and SS/D ratings summarized above, the overall viability rating for Lower SR fall Chinook salmon would be rated as "maintained."[5]

[5] "Maintained" population status is for populations that do not meet the criteria for a viable population but do support ecological functions and preserve options for ESU/DPS recovery.

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Limiting factors and threats to SR fall-run Chinook salmon include (NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, and channel structure and complexity have been degraded as a result of cumulative impacts of agriculture, forestry, and development
- Harvest-related effects
- Lost access to historic habitat above Hells Canyon and other Snake River dams
- Mainstem Columbia River and Snake River hydropower impacts
- Hatchery-related effects
- Degraded estuarine and nearshore habitat

SR Sockeye Salmon. This species includes all anadromous and residual sockeye salmon from the Snake River basin, Idaho, and artificially-propagated sockeye salmon from the Redfish Lake captive propagation program. The IC-TRT identified historical sockeye salmon production in at least five Stanley Basin and Sawtooth Valley lakes and in lake systems associated with Snake River tributaries currently cut off to anadromous access (*e.g.,* Wallowa and Payette Lakes), although current returns of SR sockeye salmon are extremely low and limited to Redfish Lake (IC-TRT 2007). The 5-year geometric mean abundance for SR sockeye salmon (2005-2009) was 166 total spawners (NOAA 2011, CBFWA 2011).

This species is still at extremely high risk across all four basic risk measures (abundance, productivity, spatial structure and diversity. Although the captive brood program has been successful in providing substantial numbers of hatchery produced *O. nerka* for use in supplementation efforts, substantial increases in survival rates across life history stages must occur in order to re-establish sustainable natural production (Hebdon *et al.* 2004, Keefer *et al.* 2008).

The key factor limiting recovery of SR sockeye salmon ESU is survival outside of the Stanley Basin. Portions of the migration corridor in the Salmon River are impeded by water quality and temperature (Idaho Department of Environmental Quality 2011). Increased temperatures may reduce the survival of adult sockeye returning to the Stanley Basin. The natural hydrological regime in the upper mainstem Salmon River Basin has been altered by water withdrawals. In most years, sockeye adult returns to Lower Granite suffer catastrophic losses (*e.g.*, > 50% mortality in one year; Reed *et al.* 2003) before reaching the Stanley Basin, although the factors causing these losses have not been identified. In the Columbia and lower Snake River migration corridor, predation rates on juvenile sockeye salmon are unknown, but terns and cormorants consume 12% of all salmon smolts reaching the estuary, and piscivorous fish consume an estimated 8% of migrating juvenile salmon (NOAA Fisheries 2011).

MCR Steelhead. This species includes all naturally-spawned steelhead populations below natural and artificial impassable barriers in streams from above the Wind River, Washington, and the Hood River, Oregon (exclusive), upstream to, and including, the Yakima River, Washington, excluding steelhead from the Snake River basin; and progeny of seven artificial propagation programs. The IC-TRT identified 17 extant populations in this DPS (IC-TRT 2003). The populations fall into four major population groups: the Yakima River Basin (four extant populations), the Umatilla/Walla-Walla drainages (three extant and one extirpated populations);

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the John Day River drainage (five extant populations) and the Eastern Cascades group (five extant and two extirpated populations) (Table 2.4.2.13) (NMFS 2009, Ford *et al.* 2011). The 5-year geometric mean abundance for MCR steelhead (2006-2010) was 15,723 total spawners (NOAA 2011, CBFWA 2011). The current estimate (2005-2009 5-year average) of natural origin spawning abundance ranges from 70% to 97% across populations.

Table 2.4.2.13. Ecological subregions, populations, and scores for the key elements (A/P, diversity, and SS/D) used to determine current overall viability risk for MCR steelhead (NMFS 2009, Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH). Maintained (MT) population status indicates that the population does not meet the criteria for a viable population but does support ecological functions and preserve options for recovery of the DPS.

Ecological Subregions	Population (Watershed)	A/P	Diversity	Integrated SS/D	Overall Viability Risk
Cascade Eastern Slope Tributaries	Fifteenmile Creek	L	L	L	Viable
	Klickitat River	M	M	M	MT?
	Eastside Deschutes River	L	M	M	Viable
	Westside Deschutes River	H	M	M	H*
	Rock Creek	H	M	M	H?
	White Salmon	Extinct	n/a	n/a	Extinct*
	Crooked River	Extinct	n/a	n/a	Extinct*
John Day River	Upper Mainstem	M	M	M	MT
	North Fork	VL	L	L	Highly Viable
	Middle Fork	M	M	M	MT
	South Fork	M	M	M	MT
	Lower Mainstem	M	M	M	MT
Walla Walla and Umatilla rivers	Umatilla River	M	M	M	MT
	Touchet River	M	M	M	H
	Walla Walla River	M	M	M	MT
Yakima River	Satus Creek	M	M	M	Viable (MT)
	Toppenish Creek	M	M	M	Viable (MT)
	Naches River	H	M	M	H
	Upper Yakima	H	H	H	H

* Re-introduction efforts underway (NMFS 2009).

There have been improvements in the viability ratings for some of the component populations, but the MCR steelhead DPS is not currently meeting the viability criteria (adopted from the IC-TRT) in the MCR steelhead recovery plan (NMFS 2009). In addition, several of the factors cited by Good *et al.* (2005) remain as concerns or key uncertainties. Natural origin spawning estimates of populations have been highly variable with respect to meeting minimum abundance thresholds. Straying frequencies into at least the Lower John Day River population are high. Returns to the Yakima River basin and to the Umatilla and Walla Walla Rivers have been higher

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over the most recent brood cycle, while natural origin returns to the John Day River have decreased. Out-of-basin hatchery stray proportions, although reduced, remain very high in the Deschutes River basin (Ford *et al.* 2011).

The limiting factors and threats to MCR steelhead include (NMFS 2009, NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas, fish passage, stream substrate, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, tributary hydro system activities, and development
- Mainstem Columbia River hydropower–related impacts
- Degraded estuarine and nearshore marine habitat
- Hatchery-related effects
- Harvest-related effects
- Effects of predation, competition, and disease

UCR Steelhead. This species includes all naturally-spawned steelhead populations below natural and manmade impassable barriers in streams in the Columbia River Basin upstream from the Yakima River, Washington, to the U.S.-Canada border, and progeny of six artificial propagation programs. Four independent populations of UCR steelhead were identified by the IC-TRT in the same upriver tributaries as for UC spring-run Chinook salmon (*i.e.*, Wenatchee, Entiat, Methow, and Okanogan; Table 2.4.2.14) and, similarly, no major population groupings were identified due to the relatively small geographic area involved (IC-TRT 2003, Ford *et al.* 2011). All extant populations are considered to be at high risk of extinction (Table 22; Ford *et al.* 2011). The 5-year geometric mean abundance for UCR steelhead (2005-2009) was 7,884 total spawners (Ford *et al.* 2011). The current estimate (2003-2008 5-year average) of natural origin spawning abundance ranges from 9% to 47% across populations.

Table 2.4.2.14. Summary of the key elements (A/P, diversity, and SS/D) and scores used to determine current overall viability risk for UCR steelhead populations (Ford *et al.* 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH).

Population (Watershed)	A/P	Diversity	Integrated SS/D	Overall Viability Risk
Wenatchee River	H	H	H	H
Entiat River	H	H	H	H
Methow River	H	H	H	H
Okanogan River	H	H	H	H

UCR steelhead populations have increased in natural origin abundance in recent years, but productivity levels remain low. The proportions of hatchery origin returns in natural spawning areas remain extremely high across the DPS, especially in the Methow and Okanogan River populations. The modest improvements in natural returns in recent years are probably primarily the result of several years of relatively good natural survival in the ocean and tributary habitats.

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With the exception of the Okanogan population, the Upper Columbia populations rated as "low" risk for spatial structure. The "high" risk ratings for SS/D are largely driven by chronic high levels of hatchery spawners within natural spawning areas and lack of genetic diversity among the populations (Ford *et al.* 2011).

The limiting factors and threats to the UCR steelhead DPS include (UCSRB 2007, NOAA Fisheries 2011):

- Mainstem Columbia River hydropower–related adverse effects.
- Impaired tributary fish passage.
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development.
- Effects of predation, competition, and disease mortality: Fish management, including past introductions and persistence of non-native (exotic) fish species continues to affect habitat conditions for listed species.
- Hatchery-related effects.
- Harvest-related effects.

SRB Steelhead. This species includes all naturally-spawned steelhead populations below natural and manmade impassable barriers in streams in the Snake River Basin of southeast Washington, northeast Oregon, and Idaho, and progeny of six artificial propagation programs. The IC-TRT identified 25 historical populations in five major groups (Table 2.4.2.15) (IC-TRT 2006, Ford *et al.* 2011). The IC-TRT has not assessed the viability of this species. The 5-year geometric mean abundance for SRB steelhead (2005-2009) was 3,546 total spawners (NOAA 2011, CBFWA 2011).

The level of natural production in the two populations with full data series and the Asotin Creek index reaches is encouraging, but the status of most populations in this DPS remains highly uncertain. Population-level natural origin abundance and productivity inferred from aggregate data and juvenile indices indicate that many populations are likely below the minimum combinations defined by the IC-TRT viability criteria. The relative proportion of hatchery fish in natural spawning areas near major hatchery release sites is highly uncertain. There is little evidence for substantial change in ESU viability relative to the previous BRT and IC-TRT reviews (Ford *et al.* 2011).

Limiting factors and threats to the SRB steelhead DPS include (IC-TRT 2006, NOAA Fisheries 2011):

- Mainstem Columbia River hydropower–related adverse effects
- Impaired tributary fish passage
- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, and development

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- Impaired water quality and increased water temperature
- Related harvest effects, particularly for B-run steelhead
- Predation
- Genetic diversity effects from out-of-population hatchery releases

Table 2.4.2.15. Ecological subregions, populations, and scores for the key elements (A/P, diversity, and SS/D) used to determine current overall viability risk for SRB steelhead (Ford *et al.* 2011, NMFS 2011). Risk ratings range from very low (VL), low (L), moderate (M), high (H), to very high (VH). Maintained (MT) population status indicates that the population does not meet the criteria for a viable population but does support ecological functions and preserve options for recovery of the DPS.

Ecological subregions	Spawning Populations (Watershed)	A/P	Diversity	Integrated SS/D	Overall Viability Risk*
Lower Snake River	Tucannon River	**	M	M	H
	Asotin Creek	**	M	M	MT
Grande Ronde River	Lower Grande Ronde	**	M	M	Not rated
	Joseph Creek	VL	L	L	Highly viable
	Upper Grande Ronde	M	M	M	MT
	Wallowa River	**	L	L	H
Clearwater River	Lower Clearwater	M	L	L	MT
	South Fork Clearwater	H	M	M	H
	Lolo Creek	H	M	M	H
	Selway River	H	L	L	H
	Lochsa River	H	L	L	H
Salmon River	Little Salmon River	**	M	M	MT
	South Fork Salmon	**	L	L	H
	Secesh River	**	L	L	H
	Chamberlain Creek	**	L	L	H
	Lower MF Salmon	**	L	L	H
	Upper MF Salmon	**	L	L	H
	Panther Creek	**	M	H	H
	North Fork Salmon	**	M	M	MT
	Lemhi River	**	M	M	MT
	Pahsimeroi River	**	M	M	MT
	East Fork Salmon	**	M	M	MT
	Upper Main Salmon	**	M	M	MT
Imnaha	Imnaha River	M		M	MT

* There is uncertainty in these ratings due to a lack of population-specific data.

** Insufficient data.

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Oregon Coast Recovery Domain. The OC recovery domain includes OC coho salmon, southern green sturgeon, and eulachon, covering Oregon coastal streams south of the Columbia River and north of Cape Blanco. Streams and rivers in this area drain west into the Pacific Ocean, and vary in length from less than a mile to more than 210 miles in length.

OC Coho Salmon. This species includes all naturally-spawned populations of coho salmon in Oregon coastal streams south of the Columbia River and north of Cape Blanco, including the Cow Creek population, which is stock #37 of Oregon Department of Fish and Wildlife's (ODFW) coho hatchery program. OC Coho salmon were first listed in February 2008. As part of a legal settlement agreement in 2008, NMFS completed a new status review for the ESU. In 2011, NMFS issued a final rule re-promulgating the threatened listing for Oregon Coast coho salmon (USDC 2011b).

The OC-TRT identified 56 populations — 21 independent and 35 dependent. The dependent populations were dependent on strays from other populations to maintain them over long time periods. The TRT also identified 5 biogeographic strata (Table 2.4.2.16) (Lawson *et al.* 2007). The 5-year geometric mean abundance for OC coho salmon (2006-2010) was 162,769 total spawners (ODFW 2011).

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Table 2.4.2.16. OC coho salmon populations. Dependent populations (D) are populations that historically would not have had a high likelihood of persisting in isolation for 100 years. These populations relied upon periodic immigration from other populations to maintain their abundance. Independent populations are populations that historically would have had a high likelihood of persisting in isolation from neighboring populations for 100 years and are rated as functionally independent (FI) and potentially independent (PI) (McElhany *et al.* 2000, Lawson *et al.* 2007).

Stratum	Population	Type	Stratum	Population	Type
North Coast	Necanicum	PI	Mid-Coast (cont.)	Alsea	FI
	Ecola	D		Big (Alsea)	D
	Arch Cape	D		Vingie	D
	Short Sands	D		Yachats	D
	Nehalem	FI		Cummins	D
	Spring	D		Bob	D
	Watseco	D		Tenmile	D
	Tillamook	FI		Rock	D
	Netarts	D		Big (Siuslaw)	D
	Rover	D		China	D
	Sand	D		Cape	D
	Nestucca	FI		Berry	D
	Neskowin	D		Sutton	D
Mid-Coast	Salmon	PI	Lakes	Siuslaw	FI
	Devils	D		Siltcoos	PI
	Siletz	FI		Tahkenitch	PI
	Schoolhouse	D		Tenmile	PI
	Fogarty	D	Umpqua	Lower Umpqua	FI
	Depoe	D		Middle Umpqua	FI
	Rocky	D		North Umpqua	FI
	Spencer	D		South Umpqua	FI
	Wade	D	Mid-South Coast	Threemile	D
	Coal	D		Coos	FI
	Moolack	D		Coquille	FI
	Big (Yaquina)	D		Johnson	D
	Yaquina	FI		Twomile	D
	Theil	D		Floras	PI
	Beaver	PI		Sixes	PI

Wainwright *et al.* (2008) determined that the weakest strata of OC coho salmon were in the North Coast and Mid-Coast of Oregon, which had only "low" certainty of being persistent. The strongest strata were the Lakes and Mid-South Coast, which had "high" certainty of being persistent. To increase certainty that the ESU as a whole is persistent, they recommended that restoration work should focus on those populations with low persistence, particularly those in the North Coast, Mid-Coast, and Umpqua strata.

A 2010 BRT (Stout *et al.* 2011) noted significant improvements in hatchery and harvest practices have been made. However, harvest and hatchery reductions have changed the population

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dynamics of the ESU. It has not been demonstrated that productivity during periods of poor marine survival is now adequate to sustain the ESU. Recent increases in adult escapement do not provide strong evidence that the century-long downward trend has changed. The ability of the OC coho salmon ESU to survive another prolonged period of poor marine survival remains in question.

Current concerns for spatial structure focus on the Umpqua River. Of the four populations in the Umpqua stratum, the North Umpqua and South Umpqua, were of particular concern. The North Umpqua is controlled by Winchester Dam and has historically been dominated by hatchery fish. Hatchery influence has recently been reduced, but the natural productivity of this population remains to be demonstrated. The South Umpqua is a large, warm system with degraded habitat. Spawner distribution appears to be seriously restricted in this population, and it is probably the most vulnerable of any population in this ESU to increased temperatures.

Current status of diversity shows improvement through the waning effects of hatchery fish on populations of OC coho salmon. In addition, recent efforts in several coastal estuaries to restore lost wetlands should be beneficial. However, diversity is lower than it was historically because of the loss of both freshwater and tidal habitat loss coupled with the restriction of diversity from very low returns over the past 20 years.

The BRT concluded that there is a moderate certainty of ESU persistence over the next 100 years and a low-to-moderate certainty that the ESU is sustainable for the foreseeable future, assuming no future trends in factors affecting the ESU. The NMFS issued a final determination to retain the ESA listing status, effective June 20, 2011. Thus, the February 2008 critical habitat designation and 4(d) regulations remain in effect (USDC 2011b).

Limiting factors and threats to the OC coho salmon ESU include (Stout *et al.* 2011, NOAA Fisheries 2011):

- Degraded freshwater habitat: Floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, stream flow, and water quality have been degraded as a result of cumulative impacts of agriculture, forestry, instream mining, dams, road crossings, dikes, levees, etc.
- Fish passage barriers that limit access to spawning and rearing habitats
- Adverse climate, altered past ocean/marine productivity, and current ocean ecosystem conditions have favored competitors and predators and reduced salmon survival rates in freshwater rivers and lakes, estuaries, and marine environments

Southern Oregon and Northern California Coasts Recovery Domain. The SONCC recovery domain includes coho salmon, southern green sturgeon, and eulachon. The SONCC recovery domain extends from Cape Blanco, Oregon, to Punta Gorda, California. This area includes many small-to-moderate-sized coastal basins, where high quality habitat occurs in the lower reaches of each basin, and three large basins (Rogue, Klamath and Eel) where high quality habitat is in the lower reaches, little habitat is provided by the middle reaches, and the largest amount of habitat is in the upper reaches.

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SONCC Coho Salmon. This species includes all naturally-spawned populations of coho salmon in coastal streams between Cape Blanco, Oregon, and Punta Gorda, California, and progeny of three artificial propagation programs. The SONCC-TRT identified 42 extant populations within this ESU, as well as 3 artificial propagation programs (Williams *et al.* 2011). In some cases, the SONCC-TRT also identified groups of populations referred to as "diversity strata" largely based on the geographical arrangement of the populations and basin-scale environmental and ecological characteristics. Of those populations, 13 strata and 17 populations occur in Oregon (Table 2.4.2.17).

The estimated abundance for SONCC coho salmon was 6,705 total spawners (ODFW 2010, Williams *et al.* 2011).

In most cases, populations appear to be well below the proposed viability thresholds, and the steps needed to move them toward viability will be similar, regardless of the specific recovery targets, which can be refined as more information becomes available. The SONCC-TRT developed a framework to assess the viability of this species and recommended: (1) Securing all extant populations, (2) collecting distribution and abundance data, (3) minimizing straying from hatcheries to natural spawning areas, and (4) beginning critical research on climate change and its potential impacts (Williams *et al.* 2008). Although long-term data on abundance of SONCC coho salmon are scarce, available evidence from shorter-term research and monitoring efforts indicate that conditions have worsened for populations since the last formal status review was published (Good *et al.* 2005, Williams *et al.* 2011). Many independent populations are well below low-risk abundance targets, and several are likely below the high-risk depensation thresholds specified by the TRT (Williams *et al.* 2011).

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Table 2.4.2.17. SONCC coho salmon populations in Oregon. Dependent populations (D) are populations that historically would not have had a high likelihood of persisting in isolation for 100 years. These populations relied upon periodic immigration from other populations to maintain their abundance. Independent populations are populations that historically would have had a high likelihood of persisting in isolation from neighboring populations for 100 years and are rated as functionally independent (FI) and potentially independent (PI). Two ephemeral populations (E) are defined as populations both small enough and isolated enough that they are only intermittently present (McElhany *et al.* 2000, Williams *et al.* 2011).

Population		Population Type
River Basin	**Subbasin**	
Elk River		FI
Mill Creek		D
Hubbard Creek		E
Brush Creek		D
Mussel Creek		D
Euchre Creek		E
Rogue River*	Lower Rogue River	PI
	Illinois River*	FI
	Mid Rogue/Applegate*	FI
	Upper Rogue River	FI
Hunter Creek		D
Pistol River		D
Chetco River		FI
Winchuck River		PI
Smith River*		FI
Klamath River*	Middle Klamath River	PI
	Upper Klamath River	FI

* Populations that also occur partly in California.

Limiting factors and threats to SONCC coho salmon include (NMFS 2012, NOAA Fisheries 2011):

- Lack of floodplain and channel structure
- Impaired water quality
- Altered hydrologic function due to altered amount and timing of river flows
- Degraded riparian forest conditions and large wood recruitment
- Altered sediment supply
- Degraded stream substrate
- Impaired estuarine function
- Impaired fish passage
- Hatchery-related adverse effects
- Effects of predation, competition, and disease mortality

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Threats from natural or man-made factors have worsened in the past 5 years, primarily due to four factors: small population dynamics, climate change, multi-year drought, and poor ocean survival conditions (NOAA Fisheries 2011).

2.4.3 Status of the Critical Habitats

We based our ratings of the status of critical habitat primarily on a watershed-scale analysis of conservation value that focused on the presence of listed ESA-listed species and physical features (*i.e.*, the primary constituent elements or PCEs) that are essential to their conservation. The physical or biological features of freshwater spawning and incubation sites include water flow, water quality, water temperatures, suitable substrate for spawning and incubation, and migratory access for adults and juveniles. These features are essential to conservation because without them the species cannot successfully spawn and produce offspring. The physical or biological features of freshwater migration corridors associated with spawning and incubation sites include water flow, water quality and water temperatures to support larval and adult mobility; abundant prey items to support larval feeding after the yolk sac is depleted; and free passage (*i.e.*, no obstructions) for adults and juveniles. These features are essential to conservation because they allow adult fish to swim upstream to reach spawning areas, and they allow juvenile fish to proceed downstream and reach the ocean.

The analysis for the 2005 designations of critical habitat for 12 species of listed salmon and steelhead species in the Columbia River basin was completed by interagency critical habitat analytical review teams (CHARTs). These teams focused on large geographical areas corresponding approximately to recovery domains (NOAA Fisheries 2005). A CHART also did an initial assessment of PCEs for coho salmon on the Oregon Coast (NOAA Fisheries 2005). The CHARTs ranked the conservation value of each watershed based on the quantity of stream habitat with PCEs, the present condition of those PCEs, the likelihood of achieving PCE potential (either naturally or through active restoration), support for rare or important genetic or life history characteristics, support for abundant populations, and support for spawning and rearing populations. In some cases, we have refined our understanding of these conservation values of these watersheds based on the work of TRTs and other recovery planning efforts that have better explained the habitat attributes, ecological interactions, and population characteristics important to each species.

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Salmon and Steelhead Critical Habitat. Tables 2.4.3.1 and 2.4.3.2 identify the PCEs (*i.e.*, site types, site attributes) and corresponding life history events for the critical habitats of listed salmon and steelhead.

Table 2.4.3.1. PCEs of critical habitats designated for listed salmon and steelhead species (except SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, SR sockeye salmon, and SONCC coho salmon), and corresponding species life history events.

Primary Constituent Elements		**Species Life History Event**
Site Type	**Site Attribute**	
Freshwater spawning	Substrate Water quality Water quantity	Adult spawning Embryo incubation Alevin growth and development
Freshwater rearing	Floodplain connectivity Forage Natural cover Water quality Water quantity	Fry emergence from gravel Fry/parr/smolt growth and development
Freshwater migration	Free of artificial obstruction Natural cover Water quality Water quantity	Adult sexual maturation Adult upstream migration and holding Kelt (steelhead) seaward migration Fry/parr/smolt growth, development, and seaward migration
Estuarine areas	Forage Free of artificial obstruction Natural cover Salinity Water quality Water quantity	Adult sexual maturation and "reverse smoltification" Adult upstream migration and holding Kelt (steelhead) seaward migration Fry/parr/smolt growth, development, and seaward migration
Nearshore marine areas	Forage Free of artificial obstruction Natural cover Water quantity Water quality	Adult growth and sexual maturation Adult spawning migration Nearshore juvenile rearing
Offshore marine areas	Forage Water quality	Adult growth and sexual maturation Adult spawning migration Subadult rearing

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Table 2.4.3.2. PCEs of critical habitats designated for SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, SR sockeye salmon, and SONCC coho salmon, and corresponding species life history events.

Primary Constituent Elements		Species Life History Event
Site	**Site Attribute**	
Spawning and juvenile rearing areas	Access (sockeye) Cover/shelter Food (juvenile rearing) Riparian vegetation Space (Chinook, coho) Spawning gravel Water quality Water temp (sockeye) Water quantity	Adult spawning Embryo incubation Alevin growth and development Fry emergence from gravel Fry/parr/smolt growth and development
Adult and juvenile migration corridors	Cover/shelter Food (juvenile) Riparian vegetation Safe passage Space Substrate Water quality Water quantity Water temperature Water velocity	Adult sexual maturation Adult upstream migration and holding Kelt (steelhead) seaward migration Fry/parr/smolt growth, development, and seaward migration
Areas for growth and development to adulthood	Ocean areas – not identified	Nearshore juvenile rearing Subadult rearing Adult growth and sexual maturation Adult spawning migration

We give descriptions of the status of critical habitat for each species of salmon and steelhead below.

LCR Chinook salmon. Designated critical habitat for LCR Chinook salmon includes all Columbia River estuarine areas and river reaches from the mouth to the confluence with the Hood River, as well as specific stream reaches in the following subbasins: Middle Columbia/Hood, Lower Columbia/Sandy, Lewis, Lower Columbia/Clatskanie, Upper Cowlitz, Cowlitz, Lower Columbia, Grays/Elochoman, Clackamas, and Lower Willamette (NMFS 2005b). There are 48 watersheds within the range of this ESU. Four watersheds received a low rating, 13 received a medium rating, and 31 received a high rating of conservation value for the species (*i.e.*, for recovery) (NOAA Fisheries 2005). The lower Columbia River has a high conservation value. It connects every population with the ocean, and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,655 miles of habitat eligible for designation, NMFS designated 1,311 miles as critical habitat.

The major factors affecting the condition of the PCEs for this species are (LCFRB 2010, NOAA Fisheries Service 2011):

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- Degraded estuarine and near-shore marine habitat resulting from the cumulative impacts of land use and flow management by the Columbia River hydropower system
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- In freshwater habitats, degradation of floodplain connectivity and function, channel structure and complexity, riparian areas, stream substrate, stream flow, and water quality, all as a result of the cumulative impacts of agriculture, forestry, and development
- Elevated concentrations of contaminants in sediments and water
- Reduced access to spawning and rearing habitats in tributaries, mainly as a result of hydropower projects
- Reduced access to off-channel rearing habitat in the Lower Columbia River

UWR Chinook salmon. Designated critical habitat for UWR Chinook salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence with the Willamette River, as well as specific stream reaches in the following subbasins: Middle Fork Willamette, Coast Fork Willamette, Upper Willamette, McKenzie, North Santiam, South Santiam, Middle Willamette, Molalla/Pudding, Clackamas, and Lower Willamette (NMFS 2005b). There are 60 watersheds within the range of this species. Nineteen watersheds received a low rating, 18 received a medium rating, and 23 received a high rating of conservation value for the species (NOAA Fisheries 2005). The lower Willamette/Columbia River rearing/migration has a high conservation value. It connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,796 miles of habitat eligible for designation, NMFS designated 1,472 miles as designated critical habitat.

The major factors affecting the condition of the PCES for this species are (ODFW and NMFS 2011, NOAA Fisheries 2011):

- Significantly reduced access to spawning and rearing habitat because of tributary dams
- Degraded freshwater habitat, especially floodplain connectivity and function, channel structure and complexity, and riparian areas and large wood recruitment as a result of the cumulative impacts of agriculture, forestry, and development
- Degraded water quality and altered water temperatures as a result of both tributary dams and the cumulative impacts of agriculture, forestry, and urban development

UCR spring-run Chinook salmon. Designated critical habitat for UCR spring Chinook includes all Columbia River estuarine areas and river reaches from the mouth upstream to Chief Joseph Dam, as well as specific stream reaches in the following subbasins: Chief Joseph, Methow, Upper Columbia/Entiat, and Wenatchee (NMFS 2005b). There are 31 watersheds within the range of this species. Five watersheds received a medium rating and 26 received a high rating of conservation value to the species. The Columbia River downstream of the specie's spawning range has a high conservation value and is the only habitat area designated in 15 of the high-value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,002 miles of habitat eligible for designation, NMFS designated 974 miles as critical habitat.

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The major factors affecting the condition of the PCES for this species are (UCSRB 2007, NOAA Fisheries 2011):

- Altered upstream and downstream fish passage, ecosystem structure and function, flows, and water quality, all due to the Columbia River hydropower system
- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development
- Degraded estuarine and nearshore marine habitats

SR SS Chinook salmon. Designated critical habitat for SR spring/summer-run Chinook salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence of the Columbia and Snake rivers, and all Snake River reaches from the confluence of the Columbia River upstream to Hells Canyon Dam (NMFS 1999a). Critical habitat also includes river reaches presently or historically accessible (except those above impassable natural falls, including Napias Creek Falls, and Dworshak and Hells Canyon dams) in the following subbasins: Hells Canyon, Imnaha, Lemhi, Little Salmon, Lower Grande Ronde, Lower Middle Fork Salmon, Lower Salmon, Lower Snake-Asotin, Lower Snake-Tucannon, Middle Salmon-Chamberlain, Middle Salmon-Panther, Pahsimeroi, South Fork Salmon, Upper Middle Fork Salmon, Upper Grande Ronde, Upper Salmon, and Wallowa.

Designated areas of critical habitat consist of the water, waterway bottom, and the adjacent riparian zone (defined as an area 300 feet from the normal high water line on each side of the river channel) (NMFS 1999a). Designation did not involve rating the conservation value of specific watersheds as was done in subsequent designations (NMFS 2005b). The lower Columbia River is among the areas of high conservation value to this species because it connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats.

The major factors affecting the condition of the PCES for this species are (NOAA Fisheries 2011):

- Degradation of floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, water temperatures, stream flows, and water quality, all as a result of the cumulative impacts of agriculture, forestry, and development
- Impacts from the mainstem Columbia River hydropower system

SR fall-run Chinook salmon. Designated critical habitat for SR fall-run Chinook salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence of the Columbia and Snake rivers; all Snake River reaches from the confluence of the Columbia River upstream to Hells Canyon Dam; the Palouse River from its confluence with the Snake River upstream to Palouse Falls; the Clearwater River from its confluence with the Snake River upstream to its confluence with Lolo Creek; and the North Fork Clearwater River from its confluence with the Clearwater River upstream to Dworshak Dam. Critical habitat also includes river reaches

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presently or historically accessible (except those above impassable natural falls and Dworshak and Hells Canyon dams) in the following subbasins: Clearwater, Hells Canyon, Imnaha, Lower Grande Ronde, Lower North Fork Clearwater, Lower Salmon, Lower Snake, Lower Snake-Asotin, Lower Snake-Tucannon, and Palouse. The lower Columbia River is among the areas of high conservation value to this species because it connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Designated areas consist of the water, waterway bottom, and the adjacent riparian zone (defined as an area 300 feet from the normal high water line on each side of the river channel).

The major factors affecting the condition of the PCES for this species are (NOAA Fisheries 2011):

- Degraded floodplain connectivity and function, and channel structure and complexity, as a result of the cumulative impacts of agriculture, forestry, and development
- Lost access to historical habitat above Hells Canyon and other Snake River dams
- Impacts of the mainstem Columbia River hydropower system
- Degraded estuarine and nearshore habitat

CR chum salmon. Designated critical habitat for CR chum salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence with the White Salmon River, as well as specific stream reaches in the following subbasins: Middle Columbia/Hood, Lower Columbia/Sandy, Lewis, Lower Columbia/Clatskanie, Cowlitz, Lower Columbia, and Grays/ Elochoman (NMFS 2005b). There are 20 watersheds within the range of this ESU. Three watersheds received a medium rating and 17 received a high rating for their conservation value to the ESU (*i.e.*, for recovery). The lower Columbia River has a high conservation value and is the only habitat area designated in one of the high value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 725 miles of habitat eligible for designation, NMFS designated 708 miles as critical habitat.

The major factors affecting the condition of the PCES for this species are (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and nearshore marine habitats resulting from the cumulative impacts of land use and flow management by the Columbia River hydropower system
- Degraded floodplain connectivity and function, channel structure and complexity, stream substrate, and riparian areas and large wood recruitment as a result of the cumulative impacts of agriculture, forestry, and development
- Altered stream flows as a result of hydropower and water supply operations
- Reduced access to off-channel rearing habitat
- Reduced water quality
- Alterations of the Columbia River's flow regime and the Columbia River plume that have altered the water temperature regime and estuarine food web, and have reduced ocean productivity
- Contaminants that have affected fish health and reproduction

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SONCC coho salmon. Critical habitat for SONCC coho salmon includes all accessible waterways, substrate, and adjacent riparian zones between the Mattole River in California, and the Elk River in Oregon, inclusive (USDC 1999). Excluded are: (1) areas above specific dams identified in USDC (1999), (2) areas above longstanding natural impassible barriers (*i.e.*, natural waterfalls), and (3) tribal lands.

The major factors affecting the condition of the PCES for this species are (NOAA Fisheries 2011, NMFS 2012):

- Lack of floodplain function and channel structure
- Impaired water quality
- Altered hydrologic function (timing of volume of water flow)
- Impaired estuary functioning
- Degraded riparian forest conditions
- Altered sediment supply
- Barriers to migration

Oregon Coast coho salmon. Critical habitat for OC coho salmon includes areas specified in USDC (2008) south of the Columbia River and north of Cape Blanco including the Nehalem River, Nestucca River, Siletz River, Yaquina River, Alsea River, Siuslaw River, Umpqua River, Coos River, and Coquille River.

The major factors affecting the condition of the PCES for this species are (Stout *et al.* 2011, NOAA Fisheries 2011):

- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large wood supply, stream substrate, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, instream mining, dams, road crossings, dikes, and levees
- Fish passage barriers that limit access to spawning and rearing habitats

SR sockeye salmon. Designated critical habitat for SR sockeye salmon includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence of the Columbia and Snake rivers; all Snake River reaches from the confluence of the Columbia River upstream to the confluence of the Salmon River; all Salmon River reaches from the confluence of the Snake River upstream to Alturas Lake Creek; Stanley, Redfish, Yellow Belly, Pettit, and Alturas lakes (including their inlet and outlet creeks); Alturas Lake Creek; and that portion of Valley Creek between Stanley Lake Creek and the Salmon River (USDC 1993).

Designated areas consist of the water, waterway bottom, and the adjacent riparian zone (defined as an area 300 feet from the normal high water line on each side of the river channel) (USDC 1993). Designation did not involve rating the conservation value of specific watersheds as was done in subsequent designations. The lower Columbia River is among the areas of high conservation value to this species because it connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a

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unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats.

The major factors affecting the condition of the PCES for this species are (NOAA Fisheries 2011):

- High water temperatures in portions of the migration corridor in the Salmon
- Alteration of the natural hydrological regime in the upper mainstem Salmon River Basin by water withdrawals
- Impacts of the mainstem Columbia River hydropower system

LCR steelhead. Designated critical habitat for LCR steelhead includes all Columbia River estuarine areas and river reaches from the mouth upstream to the confluence with the Hood River, as well as specific stream reaches in the following subbasins: Middle Columbia/Hood, Lower Columbia/Sandy, Lewis, Lower Columbia/Clatskanie, Upper Cowlitz, Cowlitz, Clackamas, and Lower Willamette (NMFS 2005b). There are 32 watersheds within the range of this DPS. Two watersheds received a low rating, 11 received a medium rating, and 29 received a high rating of conservation value to the DPS. The lower Columbia River has a high conservation value. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 2,673 miles of habitat areas eligible for designation, NMFS designated 2,324 miles as critical habitat.

The major factors affecting the condition of the PCES for this species are (LCFRB 2010, NOAA Fisheries 2011):

- Degraded estuarine and nearshore marine habitat resulting from the cumulative impacts of land use and flow management by the Columbia River hydropower system
- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and recruitment of large wood, stream substrate, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development
- Reduced access to spawning and rearing habitat as a result of tributary hydropower projects and lowland development
- Alterations of the Columbia River's flow regime and the Columbia River plume that have altered the water temperature regime and estuarine food web, and have reduced ocean productivity
- Reduced productivity resulting from sediment and nutrient-related changes in the estuary
- Contaminants that are affecting fish health and reproduction

UWR steelhead. Designated critical habitat for UWR steelhead includes all Columbia River estuarine areas and river reaches proceeding upstream to the confluence with the Willamette River, as well as specific stream reaches in the following subbasins: Upper Willamette, North Santiam, South Santiam, Middle Willamette, Molalla/Pudding, Yamhill, Tualatin, and Lower Willamette (NMFS 2005b). There are 38 watersheds within the range of this DPS. The lower Willamette/Columbia River has a high conservation value and is the only habitat area designated in one of the high value

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watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,830 miles of habitat eligible for designation, 1,276 miles of stream are designated critical habitat.

The major factors affecting the condition of the PCES for this species are (ODFW and NMFS 2011, NOAA Fisheries 2011):

- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large wood recruitment, stream substrate, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development
- Reduced access to spawning and rearing habitat as a result of tributary hydropower projects and lowland development
- Reduced access to spawning and rearing habitats, mainly as a result of artificial barriers in tributaries

MCR steelhead. Designated critical habitat for MCR steelhead includes all Columbia River estuarine areas and river reaches in the following subbasins: Upper Yakima, Naches, Lower Yakima, Middle Columbia/Lake Wallula, Walla Walla, Umatilla, Middle Columbia/Hood, Klickitat, Upper John Day, North Fork John Day, Middle Fork John Day, Lower John Day, Lower Deschutes, Trout, and Upper Columbia/Priest Rapids (NMFS 2005b). There are 114 watersheds within the range of this DPS. Nine watersheds received a low rating, 24 received a medium rating, and 81 received a high rating of conservation value to the DPS (see Chapter 4 for more detail). The lower Columbia River downstream of the specie's spawning range has a high conservation value and is the only habitat area designated in three of the high value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. Of the 6,529 miles of habitat areas eligible for designation, 5,815 miles of stream are designated critical habitat.

The major factors affecting the condition of the PCES for this species are (NMFS 2009, NOAA Fisheries 2011):

- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas, fish passage, stream substrate, stream flow, and water quality as a result of cumulative impacts of agriculture, forestry, tributary hydropower projects, and development
- Impacts from the mainstem Columbia River hydropower system
- Degraded estuarine and nearshore marine habitats

UCR steelhead. Designated critical habitat for UCR steelhead includes all Columbia River estuarine areas and river reaches from the mouth upstream to Chief Joseph Dam, as well as specific stream reaches in the following subbasins: Chief Joseph, Okanogan, Similkameen, Methow, Upper Columbia/Entiat, Wenatchee, Lower Crab, and Upper Columbia/Priest Rapids (NMFS 2005b). There are 42 watersheds within the range of this DPS. Three watersheds received a low rating, 8 received a medium rating, and 31 received a high rating of conservation

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value to the DPS. The Columbia River downstream of the specie's spawning range has a high conservation value and is the only habitat area designated in 11 of the high value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 1,332 miles of habitat areas eligible for designation, NMFS designated 1,262 miles as critical habitat.

The major factors affecting the condition of the PCES for this species are (UCSRB 2007, NOAA Fisheries 2011):

- Impacts from the mainstem Columbia River hydropower system
- Impaired tributary fish passage
- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development

SRB steelhead. Designated critical habitat for SRB steelhead includes all Columbia River estuarine areas and river reaches proceeding upstream to the confluence of the Columbia and Snake rivers as well as specific stream reaches in the following subbasins: Hells Canyon, Imnaha River, Lower Snake/Asotin, Upper Grande Ronde River, Wallowa River, Lower Grande Ronde, Lower Snake/Tucannon, Lower Snake River, Upper Salmon, Pahsimeroi, Middle Salmon-Panther, Lemhi, Upper Middle Fork Salmon, Lower Middle Fork Salmon, Middle Salmon-Chamberlain, South Fork Salmon, Lower Salmon, Little Salmon, Upper Selway, Lower Selway, Lochsa, Middle Fork Clearwater, South Fork Clearwater, and Clearwater (NMFS 2005b). There are 289 watersheds within the range of this DPS. Fourteen watersheds received a low rating, 44 received a medium rating, and 231 received a high rating of conservation value to the DPS. The lower Snake/Columbia River downstream of the specie's spawning range has a high conservation value and is the only habitat area designated in 15 of the high value watersheds identified above. This corridor connects every population with the ocean and is used by rearing/migrating juveniles and migrating adults. The Columbia River estuary is a unique and essential area for juveniles and adults making the physiological transition between life in freshwater and marine habitats. Of the 8,225 miles of habitat areas eligible for designation, NMFS designated 8,049 miles as critical habitat.

The major factors affecting the condition of the PCES for this species are (IC-TRT 2006, NOAA Fisheries 2011):

- Impacts from the mainstem Columbia River hydropower system
- Degraded floodplain connectivity and function, channel structure and complexity, riparian areas and large woody debris recruitment, stream flow, and water quality as a result of the cumulative impacts of agriculture, forestry, and development
- Increased water temperature

Green sturgeon. Critical habitat for green sturgeon includes: freshwater rivers, the bypasses, the Sacramento-San Joaquin Delta, coastal bays and estuaries, and coastal marine areas

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(within 110 m depth) extending from the California/Mexico border north to Monterey Bay, California, and from the Alaska/Canada border northwest to the Bering Strait; and certain coastal bays and estuaries in California, Oregon, and Washington (USDC 2009b).

For freshwater rivers north of and including the Eel River, NMFS did not consider the areas upstream of the head of the tide to be part of the geographical area occupied by southern DPS green sturgeon. However, the critical habitat designation recognizes not only the importance of natal habitats, but of habitats throughout their range. Critical habitat has been designated in coastal U.S. marine waters within 60 fathoms depth from Monterey Bay, California (including Monterey Bay), north to Cape Flattery, Washington, including the Strait of Juan de Fuca, Washington, to its United States boundary; the Sacramento River, lower Feather River, and lower Yuba River in California; the Sacramento-San Joaquin Delta and Suisun, San Pablo, and San Francisco bays in California; the lower Columbia River estuary; and certain coastal bays and estuaries in California (Humboldt Bay), Oregon (Coos Bay, Winchester Bay, Yaquina Bay, and Nehalem Bay), and Washington (Willapa Bay and Grays Harbor) and freshwater (USDC 2009b). Table 2.4.3.1 lists the PCEs of critical habitat for southern DPS green sturgeon and corresponding life history events.

Table 2.4.3.3. PCEs of critical habitat designated for southern DPS green sturgeon and corresponding species life history events.

Primary Constituent Elements		Species Life History Event
Site Type	**Site Attribute**	
Freshwater riverine system	Food resources Migratory corridor Sediment quality Substrate type or size Water depth Water flow Water quality	Adult spawning Embryo incubation, growth and development Larval emergence, growth and development Juvenile metamorphosis, growth and development
Estuarine areas	Food resources Migratory corridor Sediment quality Water flow Water depth Water quality	Juvenile growth, development, seaward migration Subadult growth, development, seasonal holding, and movement between estuarine and marine areas Adult growth, development, seasonal holding, movements between estuarine and marine areas, upstream spawning movement, and seaward post-spawning movement
Coastal marine areas	Food resources Migratory corridor Water quality	Subadult growth and development, movement between estuarine and marine areas, and migration between marine areas Adult sexual maturation, growth and development, movements between estuarine and marine areas, migration between marine areas, and spawning migration

The major factors affecting the condition of the PCEs for this species within freshwater rivers, bypasses, and the Sacramento-San Joaquin Delta (the Delta) are (USDC 2009b):

- Dams and diversions that obstruct migration, alter water flows and temperature, and modify substrate composition within the rivers
- Low water levels may obstruct passage through the bypasses, resulting in stranded fish

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- Pollution from agricultural runoff and water returns, as well as from other point- and non-point sources, degrades water quality within the rivers, bypasses and the Delta.
- Dredging and pile driving can adversely affect water quality and prey resources, and alter the composition and distribution of bottom substrates within the Delta

Within bays and estuaries, the major factors affecting the condition of the PCEs for this species are (USDC 2009b):

- The application of pesticides that adversely affects prey resources and water quality
- Disturbance of bottom substrates by dredging or certain other activities that adversely affects prey resources, or degrades water quality through re-suspension of contaminated sediments.
- Commercial shipping and other sources of point- and non-point source pollution that discharge contaminants
- Disposal of dredged materials that bury prey resources
- Bottom trawl fisheries that disturb the bottom and may result in beneficial or adverse effects on prey resources for green sturgeon

Within coastal marine areas, the major factors affecting the condition of the PCEs for this species are (USDC 2009b):

- Disturbance of bottom substrates by dredging or certain other activities that adversely affects prey resources, or degrades water quality through re-suspension of contaminated sediments.
- Commercial shipping and other sources of point- and non-point source pollution that discharge contaminants
- Disposal of dredged materials that bury prey resources
- Bottom trawl fisheries that disturb the bottom and may result in beneficial or adverse effects on prey resources for green sturgeon

Eulachon. Critical habitat for eulachon includes portions of 16 rivers and streams in California, Oregon, and Washington (USDC 2011c). All of these areas are designated as migration and spawning habitat for this species. In Oregon, NMFS designated 24.2 miles of the lower Umpqua River, 12.4 miles of the lower Sandy River, and 0.2 miles of Tenmile Creek as critical habitat. The NMFS also designated the mainstem Columbia River from the mouth to the base of Bonneville Dam, a distance of 143.2 miles, as critical habitat. Table 2.4.3.2 lists the designated Physical and Biological Features (PBFs) for eulachon and associated species life history events.

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Table 2.4.3.4. PBFs of critical habitats designated for eulachon and corresponding species life history events.

Essential Features		Species Life History Event
Site Type	**Site Attribute**	
Freshwater spawning and incubation	Flow, Water quality Water temperature Substrate	Adult spawning Incubation
Freshwater migration	Flow, Water quality Water temperature, Food	Adult and larval mobility Larval feeding

The major factors affecting the condition of the PCEs for this species include (Gustafson *et al.* 2010, Gustafson *et al.* 2011, NOAA Fisheries 2011):

- Changes in ocean conditions due to climate change
- Adverse effects related to dams and water diversions
- Artificial fish passage barriers
- Water pollution
- Increased water temperatures
- Insufficient stream flow
- Altered sediment balances

2.4.4 Marine Mammals

2.4.4.1 Southern Resident Killer Whales

Current Rangewide Status of the Species. The Southern Resident killer whale DPS, composed of J, K and L pods, was listed as endangered under the ESA on November 18, 2005 (70 FR 69903). Southern Residents are designated as "depleted" and "strategic" under the Marine Mammal Protection Act (MMPA)(68 FR 31980, May 29, 2003).

This section summarizes the status of the Southern Resident killer whales throughout their range. The final recovery plan for Southern Residents was issued in January 2008 (NMFS 2008a). This section summarizes information taken largely from the recovery plan and recent 5-year status review (NMFS 2011), as well as new data that became available more recently. For more detailed information about this population, please refer to NMFS (2008a).

Abundance, Productivity and Trends. Southern Resident killer whales are a long-lived species, with late onset of sexual maturity (review in NMFS 2008a). Females produce a low number of surviving calves over the course of their reproductive life span (Bain 1990, Olesiuk *et al.* 1990). Southern Resident females appear to have reduced fecundity relative to Northern Residents; the average interbirth interval for reproductive Southern Resident females is 6.1 years,

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which is longer than that of Northern Resident killer whales (Olesiuk *et al.* 2005). Mothers and offspring maintain highly stable social bonds throughout their lives, which is the basis for the matrilineal social structure in the Southern Resident population (Baird 2000, Bigg *et al.* 1990, Ford *et al.* 2000). Groups of related matrilines form pods. Three pods – J, K, and L – make up the Southern Resident community. Clans are composed of pods with similar vocal dialects and all three pods of the Southern Residents are part of J clan.

The historical abundance of Southern Resident killer whales is estimated from 140 to an unknown upper bound. The minimum historical estimate (~140) included whales killed or removed for public display in the 1960s and 1970s added to the remaining population at the time the captures ended. Several lines of evidence (*i.e.*, known kills and removals [Olesiuk *et al.* 1990], salmon declines [Krahn *et al.* 2002] and genetics [Krahn *et al.* 2002, Ford *et al.* 2011a]) all indicate that the population used to be much larger than it is now, but there is currently no reliable estimate of the upper bound of the historical population size. When faced with developing a population viability analysis for this population, NMFS' biological review team found it reasonable to assume an upper bound of as high as 400 whales to estimate carrying capacity (Krahn *et al.* 2004).

At present, the Southern Resident population has declined to essentially the same size that was estimated during the early 1960s, when it was considered as likely depleted (Olesiuk *et al.* 1990) (Figure 2.4.4.1). Since censuses began in 1974, J and K pods have steadily increased their sizes. However, the population suffered an almost 20 percent decline from 1996-2001 (from 97 whales in 1996 to 81 whales in 2001), largely driven by lower survival rates in L pod. Since then the overall population has increased slightly from 2002 to present (from 83 whales in 2002 to 88 whales in August, 2011). Over the last 28 years (1983-2010), population growth has been variable, with an average annual population growth rate of 0.3 percent and standard deviation of ± 3.2 percent. Seasonal mortality rates among Southern and Northern Resident whales may be highest during the winter and early spring, based on the numbers of animals missing from pods returning to inland waters each spring. Olesiuk *et al.* (2005) identified high neonate mortality that occurred outside of the summer season. At least 12 newborn calves (nine in the southern community and three in the northern community) were seen outside the summer field season and disappeared by the next field season. Additionally, stranding rates are higher in winter and spring for all killer whale forms in Washington and Oregon (Norman *et al.* 2004). Southern Resident strandings in coastal waters offshore include three separate events (1995 and 1996 off of Northern Vancouver Island and the Queen Charlotte Islands, and 2002 offshore of Long Beach, Washington State), but the causes of death are unknown (NMFS 2008a).

There are 26 whales in J pod, 20 whales in K pod and 42 whales in L pod. There are currently 2 adult males and one nearly matured male in J pod, three adult males in K pod, and 10 adult males in L pod. The population is 35.6 percent juveniles, 34.5 percent reproductive females, 10.3 percent post-reproductive females and 18.4 percent adult males. This age distribution is similar to that of Northern Residents that are a stable and increasing population (Olesiuk *et al.* 2005). However, there are several demographic factors of the Southern Resident population that are cause for concern, namely the small number of breeding males (particularly in J and K pods), reduced fecundity, sub-adult survivorship in L pod, and the total number of individuals in the population (review in NMFS 2008a). The current population abundance of 87 whales is small, at

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most half of its likely previous abundance (140 to an unknown upper bound that could be as high at 400 whales, as discussed above). The estimated effective size of the population (based on the number of breeders under ideal genetic conditions) is very small at approximately 26 whales or roughly 1/3 of the current population size (Ford *et al.* 2011a). The small effective population size and the absence of gene flow from other populations may elevate the risk from inbreeding and other issues associated with genetic deterioration, as evident from documented breeding within pods (Ford *et al.* 2011a). As well, the small effective population size may contribute to the lower growth rate of the Southern Resident population in contrast to the Northern Resident population (Ford *et al.* 2011a, Ward *et al.* 2009).

Because of this population's small abundance, it is also susceptible to demographic stochasticity – randomness in the pattern of births and deaths among individuals in a population. Several other sources of stochasticity can affect small populations and contribute to variance in a population's growth and extinction risk. Other sources include environmental stochasticity, or fluctuations in the environment that drive fluctuations in birth and death rates, and demographic heterogeneity, or variation in birth or death rates of individuals because of differences in their individual fitness (including sexual determinations). In combination, these and other sources of random variation combine to amplify the probability of extinction, known as the extinction vortex (Gilpin and Soule 1986, Fagen and Holmes 2006, Melbourne and Hastings 2008). The larger the population size, the greater the buffer against stochastic events and genetic risks. A delisting criterion for the Southern Resident killer whale DPS is an average growth rate of 2.3% for 28 years (NMFS 2008a). In light of the current average growth rate of 0.3%, this recovery criterion reinforces the need to allow the population to grow quickly.

Population growth is also important because of the influence of demographic and individual heterogeneity on a population's long-term viability. Population-wide distribution of lifetime reproductive success can be highly variable, such that some individuals produce more offspring than others to subsequent generations, and male variance in reproductive success can be greater than that of females (*i.e.*, Clutton-Brock 1988, Hochachka 2006). For long-lived vertebrates such as killer whales, some females in the population might contribute less than the number of offspring required to maintain a constant population size (n = 2), while others might produce more offspring. The smaller the population, the more weight an individual's reproductive success has on the population's growth or decline (*i.e.*, Coulson *et al.* 2006). This further illustrates the risk of demographic stochasticity for a small population like Southern Resident killer whales – the smaller a population, the greater the chance that random variation will result in too few successful individuals to maintain the population.

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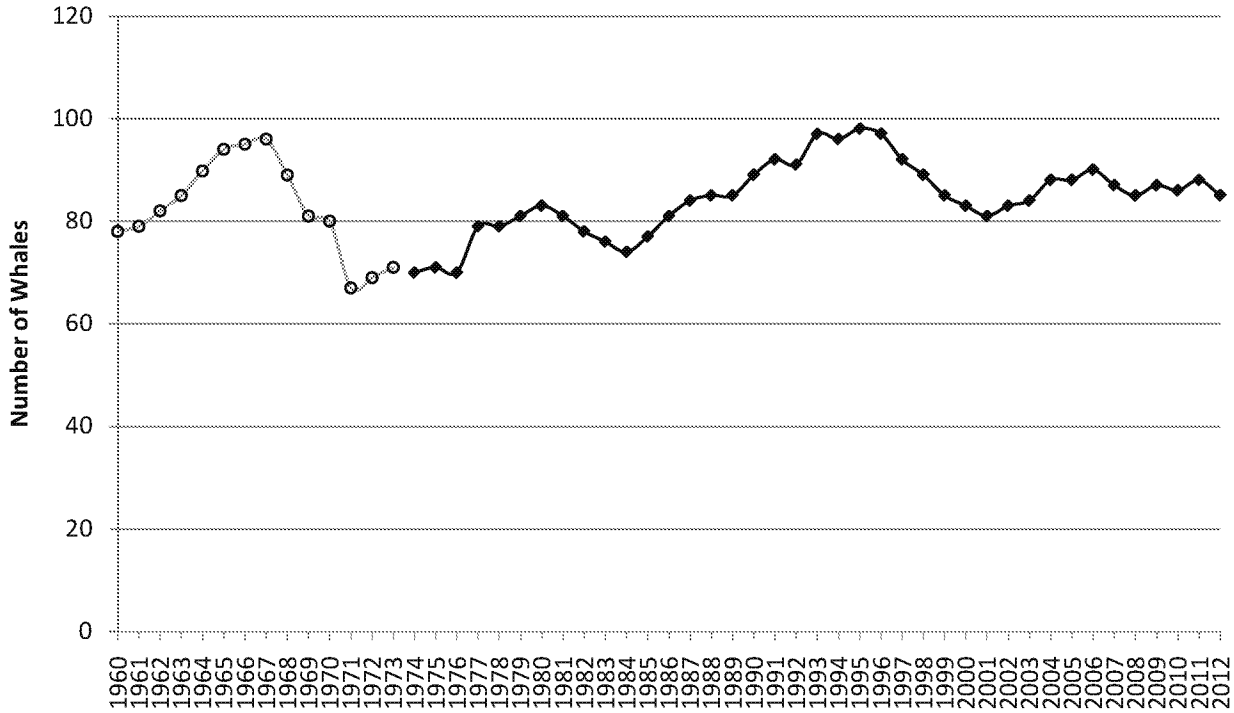

Figure 2.4.4.1.1. Population size and trend of Southern Resident killer whales, 1960-2012. Data from 1960-1973 (open circles, gray line) are number projections from the matrix model of Olesiuk *et al.* (1990). Data from 1974-2012 (diamonds, black line) were obtained through photo-identification surveys of the three pods (J, K, and L) in this community and were provided by the Center for Whale Research (unpubl. data) and NMFS (2008). Data for these years represent the number of whales present at the end of each calendar year, except for 2012, when data only extend to July.

Range and Distribution. Southern Residents occur throughout the coastal waters of Washington, Oregon, and Vancouver Island and are known to travel as far south as central California and as southeast Alaska (one sighting occurred in Chatham Strait, Alaska; Figure 2.4.4.1.2.). The Figure 2.4.4.1.2. does not reflect the recent sighting in Alaska. There is limited information on the distribution and habitat use of Southern Residents along the outer Pacific Coast.

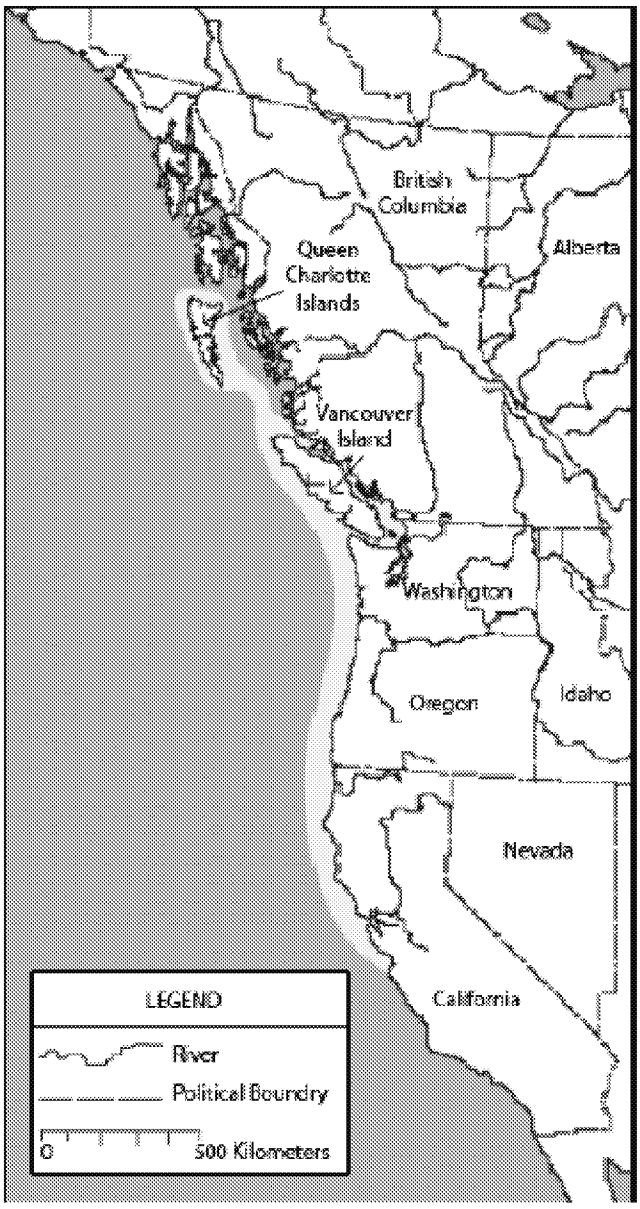

Figure 2.4.4.1.2. Geographic Range (light shading) of the Southern Resident Killer Whale DPS. Reprinted from Wiles (2004).

Southern Residents are highly mobile and can travel up to 86 miles in a single day (Erickson 1978, Baird 2000). To date, there is no evidence that Southern Residents travel further than 50 km offshore (Ford *et al.* 2005). Although the entire Southern Resident DPS has potential to occur in coastal waters at any time during the year, occurrence is more likely from November to May (Table 2.4.4.1.1).

Southern Residents spend a substantial amount of time from late spring to early autumn in inland waterways of Washington State and British Columbia (Strait of Georgia, Strait of Juan de Fuca, and Puget Sound. Bigg 1982, Ford *et al.* 2000, Krahn *et al.* 2002, Table 2.4.4.1.1). Typically, J,

K and L pods are increasingly present in May or June and spend considerable time in the core area of Georgia Basin and Puget Sound until at least September. During this time, pods (particularly K and L) make frequent trips from inland waters to the outer coasts of Washington and southern Vancouver Island, which typically last a few days (Ford *et al.* 2000).

Table 2.4.4.1.1. Average number of days spent by Southern Resident killer whales in inland and coastal waters by month[1], 2003-2007 (Hanson and Emmons 2010).

Months	Lpod		Jpod		Kpod	
	Days Inland	Days Coastal	Days Inland	Days Coastal	Days Inland	Days Coastal
Jan	5	26	3	29	8	23
Feb	0	28	4	24	0	28
March	2	29	7	24	2	29
April	0	30	13	17	0	30
May	2	29	26	5	0	31
June	14	16	26	5	12	18
July	18	13	24	7	17	14
Aug	17	15	17	15	17	14
Sep	20	10	19	11	17	13
Oct	12	19	14	17	8	24
Nov	5	25	13	17	7	23
Dec	1	30	8	23	10	21

[1]Hanson and Emmons report sightings in inland waters. For purposes of this consultation analysis, and because the population is highly visible when in inland waters, NMFS assumes that when not sighted in inland waters the whales are in their coastal range.

Late summer and early fall movements of Southern Residents in the Georgia Basin are consistent, with strong site fidelity shown to the region as a whole and high occurrence in the San Juan Island area (Hanson and Emmons 2010, Hauser *et al.* 2007). There is inter-annual variability in arrival time and days present in inland waters from spring through fall, with late arrivals and fewer days present during spring in recent years potentially related to weak returns of spring and early summer Chinook salmon to the Fraser River (Hanson and Emmons 2010). Similarly, recent high occurrence in late summer may relate to greater than average Chinook salmon returns to South Thompson tributary of the Fraser River (Hanson and Emmons 2010). During fall and early winter, Southern Resident pods, and J pod in particular, expand their routine movements into Puget Sound, likely to take advantage of chum and Chinook salmon runs (Hanson *et al.* 2010a, Osborne 1999). During late fall, winter, and early spring, the ranges and movements of the Southern Residents are less known. Sightings through the Strait of Juan de Fuca in late fall suggest that activity shifts to the outer coasts of Vancouver Island and Washington (Krahn *et al.* 2002).

The Southern Residents were formerly thought to range southward along the coast to about Grays Harbor (Bigg *et al.* 1990) or the mouth of the Columbia River (Ford *et al.* 2000). However, recent sightings of members of K and L pods in Oregon (in 1999 and 2000) and California (in 2000, 2003, 2005, 2006, 2007, 2008, and 2009) have considerably extended the southern limit of their known range (NMFS 2008a). There have been verified visual sightings or

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strandings of J, K or L pods along the outer coast from 1975 to present with most made from January through April (summarized in NMFS 2008a, and NWFSC unpubl. data). These include 16 records off Vancouver Island and the Queen Charlottes, 15 off Washington, four off Oregon, and 10 off central California. Most records have occurred since 1996, but this may be because of increased viewing effort along the coast for this time of year.

Sightings in Monterey Bay, California coincided with occurrence of salmon, with feeding witnessed in 2000 (Black *et al.* 2001). Southern Residents were also sighted in Monterey Bay during 2008, when salmon runs from California were expected to be near record lows (PFMC 2010). L pod was also seen feeding on unidentified salmon off Westport, Washington, in March 2004 during the spring Chinook salmon run in the Columbia River (M. B. Hanson, personal observation as cited in Krahn *et al.* 2004). In March, 2005 L pod was sighted working a circuit across the Columbia River plume from the North Jetty across to the South Jetty during the spring Chinook salmon run in the Columbia River (Zamon *et al.* 2007). Also in March of 2006, K and L pods were encountered off the Columbia River (Hanson *et al.* 2008). L pod was again seen feeding off Westport, Washington in March 2009, and genetic analysis of prey remains collected from two predation events identified one fish as spring Chinook salmon and the other as a summer/fall Chinook salmon from Columbia River stocks (Hanson *et al.* 2010b).

The Northwest Fisheries Science Center (NWFSC) also deploys and collects data from remote autonomous acoustic recorders in coastal waters of Washington State, and in 2009 alone documented 52 Southern Resident killer whale detections from this acoustic system (Emmons *et al.* 2009). The Department of Fisheries and Oceans (DFO), Canada also maintains acoustic recorders in British Columbia. When the NWFSC and DFO analyze these data, more information will be available about the seasonal distribution, movements and habitat use of Southern Resident killer whales, specifically in coastal waters off Washington and British Columbia.

Limiting Factors and Threats. Several factors identified in the final recovery plan for Southern Residents may be limiting recovery. These are quantity and quality of prey, toxic chemicals that accumulate in top predators, disturbance from sound and vessels. Oil spills are also a risk factor. It is likely that multiple threats are acting in concert to impact the whales. Although it is not clear which threat or threats are most significant to the survival and recovery of Southern Residents, all of the threats identified are potential limiting factors in their population dynamics (NMFS 2008a). Here we focus on the quantity and quality of prey, and the toxic chemicals in the whales because these are affected by the proposed action. The discussion in the Environmental Baseline and Cumulative Effects sections contain a thorough evaluation of all threats in the action area.

Prey. Healthy killer whale populations depend on adequate prey levels. First, we discuss the prey requirements of Southern Residents followed by an assessment of threats to the quantity and quality of their prey.

Prey Requirements. Southern Resident killer whales consume a variety of fish species (22 species) and one species of squid (Scheffer and Slipp 1948; Ford *et al.* 1998, 2000; Ford and Ellis 2006; Saulitis *et al.* 2000; Hanson *et al.* 2010c), but salmon are identified as their primary prey (*i.e.*, a high percent of prey consumed during spring, summer and fall, from long-term studies of resident killer whale diet; Ford and Ellis 2006, Hanson *et al.* 2010c). Feeding records

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for Southern and Northern Residents show a predominant consumption of Chinook salmon during late spring to fall (Ford and Ellis 2006). Chum salmon are also taken in significant amounts, especially in fall. Other salmon eaten include coho, pink, steelhead (*O. mykiss*), and sockeye (*O. nerka*). The non salmonids included Pacific herring, sablefish, Pacific halibut, quillback and yelloweye rockfish (*Sebastes maliger*), lingcod (*Ophiodon elongates*), and Dover sole (*Microstomus pacificus*) (Ford *et al.* 1998, Hanson *et al.* 2010c). Chinook salmon were the primary prey despite the much lower abundance of Chinook salmon in the study area in comparison to other salmonids (primarily sockeye), for mechanisms that remain unknown but factors of potential importance include the species' large size, high fat and energy content, and year-round occurrence in the area. Killer whales also captured older (*i.e.*, larger) than average Chinook salmon (Ford and Ellis 2006). Recent research suggests that killer whales are capable of detecting, localizing and recognizing Chinook salmon through their ability to distinguish Chinook salmon echo structure as different from other salmon (Au *et al.* 2010).

Southern Residents are the subject of ongoing research, including direct observation, scale and tissue sampling of prey remains, and fecal sampling. A recent publication by Hanson *et al.* (2010c) provides the best available scientific information on diet composition of Southern Residents in inland waters during summer months. The results provide information on (1) the percentage of Chinook in the whales' diet, and (2) the predominant river of origin of those Chinook. Other research and analysis provides additional information on the age of prey consumed (Hanson, unpubl. data, as summarized in Ward *et al.* 2010), indicating that the whales are consuming mostly larger (*i.e.*, older) Chinook.

Scale and tissue sampling in inland waters from May to September indicate that the Southern Residents' diet consists of a high percentage of Chinook, with an overall average of 88% Chinook across the timeframe and monthly proportions as high as >90% Chinook (*i.e.*, July: 98% and August: 92%, see S/T sample type in Table 2 Hanson *et al.* 2010c). Fecal samples are also available in Hanson *et al.* (2010c) but were not used to estimate proportion of the Southern Residents' diet, because the data from these samples represents presence or absence of prey species, but not proportion of diet. DNA quantification methods can be used to estimate the proportion of diet from fecal samples (*i.e.*, Deagle *et al.* 2005). This technique is still in the developmental stages. However, preliminary DNA quantification results from Hanson *et al.* (2010c) samples indicate that Chinook make up the bulk of the prey DNA in the fecal samples (Ford *et al.* 2011b).

Genetic analysis of the Hanson *et al.* (2010c) samples indicate that when Southern Resident killer whales are in inland waters from May to September, they consume Chinook stocks that originate from regions including the Fraser River (including Upper Fraser, Mid Fraser, Lower Fraser, N. Thompson, S. Thompson and Lower Thompson), Puget Sound (N. and S. Puget Sound), the Central British Columbia Coast and West and East Vancouver Island. Hanson *et al.* (2010c) find that the whales are likely consuming Chinook salmon stocks at least roughly proportional to their local abundance, as inferred by Chinook run-timing pattern and the stocks represented in killer whale prey for a specific area of inland waters, the San Juan Islands. Ongoing studies also confirm a shift to chum salmon in fall (Ford *et al.* 2010a, Hanson *et al.* 2010a).

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Although less is known about the diet of Southern Residents off the Pacific coast, the available information indicates that salmon, and Chinook salmon in particular, are also important when the whales occur in coastal waters. To date, there are direct observations of two different predation events (where the prey was identified to species and stock from genetic analysis of prey remains) when the whales were in coastal waters. Both were identified as Columbia River Chinook stocks (Hanson *et al.* 2010b). Chemical analyses also support the importance of salmon in the year round diet of Southern Resident killer whales (Krahn *et al.* 2002, 2007, 2009). Krahn *et al.* (2002), examined the ratios of DDT (and its metabolites) to various PCB compounds in the whales, and concluded that the whales feed primarily on salmon throughout the year rather than other fish species. The predominance of Chinook in their diet in inland waters, even when other species are more abundant, combined with information to date about prey in coastal waters (above), makes it reasonable to expect that Chinook salmon is equally predominant in the whales' diet when available in coastal waters. It is also reasonable to expect that the diet of Southern Residents is predominantly larger Chinook when available in coastal waters. The diet of Southern Residents in coastal waters is a subject of ongoing research.

Quantity of Prey. Human influences have had profound impacts on the abundance of many prey species in the northeastern Pacific during the past 150 years, including salmon. The health and abundance of wild salmon stocks have been negatively affected by altered or degraded freshwater and estuarine habitat, including numerous land use activities, from hydropower systems to urbanization, forestry, agriculture and development. Harmful artificial propagation practices and overfishing have also negatively affected wild salmon stocks. Section 2.4 provides a comprehensive overview of limiting factors for Puget Sound Chinook, as does the Puget Sound Salmon Recovery Plan (Shared Strategy 2007 and NMFS 2007). Predation also contributes to natural mortality of salmon. Salmonids are prey for pelagic fish, birds, and marine mammals including killer whales.

While wild salmon stocks have declined in many areas, hatchery production has supplemented additional prey. Currently, hatchery production contributes a significant component of the salmon prey base returning to watersheds within the range of Southern Resident killer whales (*i.e.*, review PFMC 2011 for Puget Sound, Barnett-Johnson *et al.* 2007 for Central Valley California, and NMFS 2008b for Columbia River Basin). Although hatchery production has contributed some offset of the historical declines in the abundance of wild salmon within the range of Southern Residents, hatcheries also pose risks to wild salmon populations (*i.e.*, Ford 2002, Nickelson *et al.* 1986, Levin and Williams 2002, Naish *et al.* 2007). In recent decades, managers have been moving toward hatchery reform, and are in the process of reducing risks identified in hatchery programs, through region-wide recovery planning efforts and hatchery program reviews. Healthy wild salmon populations are important to the long-term maintenance of prey populations available to Southern Resident killer whales, because it is uncertain whether a hatchery dominated mix of stocks is sustainable indefinitely.

Salmon abundance is also substantially affected by climate variability in freshwater and marine environments, particularly by conditions during early life-history stages of salmon (NMFS 2008b). Sources of variability include inter-annual climatic variations (*e.g.*, El Niño and LaNiña), longer term cycles in ocean conditions (*e.g.*, Pacific Decadal Oscillation, Mantua *et al.* 1997), and ongoing global climate change. For example, climate variability can affect ocean

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productivity in the marine environment and water storage (*e.g.* snow pack) and in-stream flow in the freshwater environment. Early life-stage growth and survival of salmon can be negatively affected when climate variability results in conditions that hinder ocean productivity (*e.g.*, Scheuerell and Williams 2005) and/or water storage (*e.g.*, ISAB 2007) in marine and freshwater systems, respectively. Severe flooding in freshwater systems can also constrain salmon populations (NMFS 2008c). The availability of adult salmon may be reduced in years following unfavorable conditions to the early life-stage growth and survival of salmon.

When prey is scarce, whales likely spend more time foraging than when it is plentiful. Increased energy expenditure and prey limitation can cause nutritional stress. Nutritional stress is the condition of being unable to acquire adequate energy and nutrients from prey resources and as a chronic condition can lead to reduced body size and condition of individuals and lower reproductive and survival rates of a population (*e.g.*, Trites and Donnelly 2003). The Center for Whale Research has observed the very poor body condition in 13 members of the Southern Resident population, and all but two of those whales subsequently died (Durban *et al.* 2009). Both females and males across a range of ages were found in poor body condition (Durban *et al.* 2009). Food scarcity could also cause whales to draw on fat stores, mobilizing contaminants stored in their fat that are at relatively high levels (Krahn *et al.* 2007, 2009; Mongillo 2009) and affecting reproduction and immune function (as discussed above).

Here we examine potential symptoms of chronic nutritional stress by considering the available data on poor body condition of individual Southern Residents and discussing demographic modeling conducted to date that identifies Chinook abundance as strongly correlated with changes in demographic rates of the Southern Resident killer whale population.

Body Condition of Whales. The Center for Whale Research is the primary source of data for body condition of Southern Resident killer whales and retains photographs of all individual Southern Resident killer whales identified during annual census. They document body condition with boat-based visual observation and photographs. This technique is not able to detect fine scale differences in condition, because from the dorsal vantage a detectable change is only visible when a whale's condition has become very poor (Durban *et al.* 2009). Very poor condition is detectable by a depression behind the blowhole that presents as a "peanut-head" appearance. The Center for Whale Research has observed the "peanut-head" condition in 13 members of the Southern Resident population, and all but two of those whales subsequently died (Table 2.4.3.2). Durban *et al.* (2009) are currently refining methods to detect changes in body condition at a finer scale with aerial photogrammetry. Ayres *et al.* (2012) also examined potential symptoms of nutritional stress in the whales by measuring fecal hormones.

None of the whales that died were subsequently recovered, and therefore definitive cause of death could not be identified. Both females and males across a range of ages were found in poor body condition (Table 2.4.4.1.2). Regardless of the cause(s) of death, it is possible that poor nutrition could contribute to mortality through a variety of mechanisms. To demonstrate how this is possible, we reference studies that have demonstrated the effects of energetic stress (caused by incremental increases in energy expenditures or incremental reductions in available energy) on adult females and juveniles, which have been studied extensively (*e.g.*, adult females: Gamel *et al.* 2005, Daan *et al.* 1996, juveniles: Noren *et al.* 2009, Trites and Donnelly 2003). Small,

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incremental increases in energy demands should have the same effect on an animal's energy budget as small, incremental reductions in available energy, such as one would expect from reductions in prey. Ford and Ellis (2006) report that resident killer whales engage in prey sharing about 76% of the time. Prey sharing presumably would distribute more evenly the effects of prey limitation across individuals of the population than would otherwise be the case (*i.e.*, if the most successful foragers did not share with other individuals). Therefore, although cause of death for these specific individuals is unknown, poor nutrition could contribute to additional mortality in this population.

Demographic Modeling. Ford *et al.* (2005 and 2010b) evaluated 25 years of demographic data from Southern and Northern Resident killer whales and found that changes in survival largely drive their population trends, and the populations' survival rates are strongly correlated with coast-wide availability of Chinook salmon (from Pacific Salmon Commission [PSC] abundance indices that estimate abundance between Southeast Alaska and Oregon). Ward *et al.* (2009) found that Northern and Southern Resident killer whale fecundity is highly correlated with Chinook abundance indices, and reported the probability of calving increased by 50 percent between low and high Chinook abundance years. PSC Chinook abundance indices from the West Coast of Vancouver Island (WCVI) were the most important predictor of the relationship. Recently, Ward (2010) considered new information to update the 2009 fecundity model with new birth data and a singular focus on the Southern Resident killer whale population. Ward (2010) also conducted the updated analysis for survival, where the survival of L pod was evaluated separately from the survival of J and K pods because of the apparent lower survival in L pod (Ward *et al.* 2011, Krahn *et al.* 2004). Best-ranked models all included one of the PSC Chinook indices (the Northern British Columbia indices performed best, and WCVI, Southeast Alaska and inland WCVI indices performed equally well at second best). The results are consistent with findings from Ford *et al.* 2010b.

Quality of Prey. The quality of Chinook salmon, Southern Resident killer whales' primary prey, is likely influenced by a variety of factors, including contaminant load, size of the fish, their fat content, and origin (natural vs. hatchery). Overall, Chinook have the highest lipid content (Stansby 1976, Winship and Trites 2003), largest size, and highest caloric value per kg of any salmonid species (Ford and Ellis 2006, Osborne 1999). Details about contaminant load, size, and origin are provided below.

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Table 2.4.4.1.2. Dates of observed "peanut-head" condition of individual Southern Resident killer whales and their fates (Durban *et al.* 2009).

Year	Whale ID	Whale Sex/Age	Description	Fate
1994	L42	M / 21	A slight depression behind the blowhole was first noticed in mid-June; a prominent depression by mid-July; the dorsal fin was drooping by mid August; the depression had become large by early September exposing the shape of the back of the cranium and vertebrae; last seen in late September.	Died
	K17	M / 28	A slight depression behind the blowhole was first noticed in mid July; prominent depression by mid August; last seen in mid September with the fin severely drooping.	Died
1995	J3	M / 43	A slight depression behind the blowhole noticeable by the end of March; moderate depression by mid May with the fin beginning to droop; last seen late May.	Died
	L63	M / 11	A prominent depression behind the blowhole noticeable by late July; last seen late July.	Died
	L68	M / 10	A moderate depression behind the blowhole was noticeable in mid May; depression prominent by mid June; last seen in late June.	Died
1996	J12	F / 24	A slight depression behind the blowhole first noticed in mid February; depression moderate by April with the base of the cranium apparent; prominent depression by early June, with ribs beginning to show on flanks; depression very prominent by early September, revealing the shape of the base of the cranium and vertebrae, and ribs visible on flanks showing; last seen late September.	Died
	L9	F / 65	A slight depression behind the blowhole noticeable in early July; depression prominent by mid August, exposing the shape of the base of the cranium; last seen mid August.	Died
1997	J5	F / 59	A slight depression noticeable in early April; last seen early April.	Died
2002	L102	Unk / Calf	Moderate depression behind the blowhole noticeable in early December- only time the calf was seen; last seen early December.	Died
2005	K25	M / 14	A moderate depression was noticeable behind the blowhole in late July, with a laceration on the whale's back following a collision with a whale-watch boat in early July; depression slight by early September; whale survived.	Survived
2006	K28	F / 12	A prominent depression behind the blowhole was noticeable in mid September; whale not seen afterward.	Died
2008	L106	M / 3	A prominent depression behind the blowhole was noticeable in mid June; depression just slight by mid July; depression barely noticeable by early August; whale survived the year, and seen in early 2009.	Survived
	L67	F / 23	A slight depression behind the blowhole was first noticeable in late June; depression still slight in early August; depression prominent by mid September; last seen mid September.	Died

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Contaminant Load. Levels of persistent organic pollutants (POPs) in killer whales are primarily determined by contaminant levels in their prey and the geographic region, although the age, gender, and birth order of the whale will also influence accumulation. Various studies have documented a range of concentrations of POPs in many populations of adult Pacific salmon (see Table 2.4.4.1.3). POP accumulation in Pacific salmon is primarily determined by geographic proximity to contaminated environments (Mongillo *et al.* in prep.). Because Chinook salmon are distributed in more coastal waters, they are more readily exposed to contaminants that are present in coastal waters than other species. In contrast, sockeye, pink, and chum salmon have lower POP concentrations because by the end of their first year, they have migrated through the coastal waters and are found in the open waters of the North Pacific, Gulf of Alaska, and Bering Sea (Quinn 2005). Measured average concentrations of PCBs and polybrominated diphenyl ethers (PBDEs) were highest for Chinook intermediate for coho, less for sockeye, and lowest for pink and chum salmon (see Table 2.4.4. 1.3). Similarly, average DDT values were higher in Chinook and coho salmon compared to sockeye and lowest for pink and chum salmon (see Table 2.4.4. 1.3). Intermediate levels of PCB and PBDEs were measured in California and Oregon populations and the lowest average levels were measured in populations off Alaska (Mongillo *et al.* in prep.). The biological traits in Pacific salmon (*e.g.* trophic status, lipid content, age, exposure duration, metabolism, and detoxification) may also affect the degree to which POPs accumulate (Mongillo *et al.* in prep.).

Size. Size of individual salmon is an aspect of prey quality that could affect the foraging efficiency of Southern Resident killer whales. As discussed above, available data suggests that Southern Residents consume larger prey. The degree to which this is a function of the availability of all sizes of fish in the coastal range of the whales, their ability to detect all sizes or a true preference of only large fish is unknown. It is possible although not conclusive that there has been a historical decrease in salmon age, size, or size at a given age (*i.e.*, Bigler *et al.* 1996, but also see PFMC data (PFMC 2011). Fish size is influenced by factors such as environmental conditions, selectivity in fishing effort through gear type, fishing season or regulations, and hatchery practices. The available information on size is also confounded by factors including inter-population difference, when the size was recorded, and differing data sources and sampling methods (review in Quinn 2005).

Origin. Southern Resident killer whales likely consume both natural and hatchery salmon (Hanson *et al.* 2010c). The best available information does not indicate that natural and hatchery salmon generally differ in size, run-timing, or ocean distribution (*e.g.*, Nickum *et al.* 2004, NMFS 2008c, Weitkamp and Neely 2002, regarding differences that could affect Southern Residents); however, there is evidence of size and run-timing differences between hatchery and natural salmon from specific river systems or runs (*i.e.*, size and run timing differences as described for Willamette River Chinook in NMFS 2008d). Potential run-specific differences in the quality of natural and hatchery salmon are evaluated where data are available.

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Table 2.4.4. 1.3. Lipid and persistent organic pollutant concentrations (ng/g wet weight) of adult and subadult Pacific salmon sampled in terminal areas. Terminal areas include coastal marine water and river mouths through which fish migrate en route to their natal stream. From Mongillo *et al.* (in prep).

Species	Region	sub-region	Population	n	Tissue Analyzed	Lipid (%)	PCBs	DDTs	PBDEs	Citation
Chinook salmon	Alaska	unknown	unknown	2	muscle w/o skin	NR	5.6	NR	0.95	4
	Alaska	Aleutian Islands	unknown	3	muscle w/skin	7.6	5.0	22	0.71	13, 14*
	Alaska	SE Alaska/ Gulf of Alaska/ Bering Sea	unknown	35	muscle w/o skin	9.7	11	7.1	0.53	20
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	8.0	NR	0.50	5*, 6*
	Alaska	South Central	River	10	muscle w/o skin	NR	9.1	9.8	NR	12
		Alaskan Chinook salmon Average				8.7	7.7	13.0	0.67	
	British Columbia	BC North Coast	Skeena	30	whole body	NR	7.3	7.3	0.08	10
	British Columbia	Fraser River	Thompson	6	muscle w/o skin	10	9.1	1.5	NR	1
	British Columbia	Fraser River		13	whole body	NR	9.4	6.6	0.80	10
	British Columbia	Fraser River	Thompson	7	muscle w/o skin	12	8.6	7.7	1.54	16**
	British Columbia	Fraser River	Shuswap	2	muscle w/o skin	3.0	9.8	5.5	NR	16**
	British Columbia	Fraser River	Harrison	6	muscle w/o skin	5.4	47	4.3	17.7	1
		Fraser River Chinook salmon Average (excluding Harrison)				8.3	10	5.7	1.67	
		British Columbia Chinook salmon Average				7.6	15	5.5	4.87	
	Washington	Puget Sound	Nooksack River	28	muscle w/o skin	3.5	37	NR	NR	11
	Washington	Puget Sound	Skagit River	29	muscle w/o skin	4.8	40	NR	NR	11
	Washington	Puget Sound	Duwamish River	65	muscle w/o skin	7.3	56	NR	NR	11
	Washington	Puget Sound	Nisqually River	20	muscle w/o skin	3.8	41	NR	NR	11
	Washington	Puget Sound	Deschutes River	34	muscle w/o skin	1.7	59	NR	NR	11
	Washington	Puget Sound	PS mixed	28	muscle w/o skin	4.8	76	NR	NR	11
	Washington	Puget Sound	Duwamish River	3	whole body	6.4	35	18.3	6.43	1
	Washington	Puget Sound	Deschutes River	4	whole body	4.3	56	NR	NR	1
	Washington	Puget Sound	Deschutes River	10	muscle w/o skin	1.0	49	NR	NR	8
	Washington	Puget Sound	Issaquah Creek	10	muscle w/o skin	0.6	49	NR	NR	8
	Washington	Puget Sound	PS mixed	36	whole body	NR	43	29.1	18.9	10

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Species	Region	sub-region	Population	n	Tissue Analyzed	Lipid (%)	PCBs	DDTs	PBDEs	Citation
	Washington	Puget Sound	PS mixed	34	whole body	NR	91	16.4	42.2	10
	Washington	WA Coast	Makah	10	muscle w/o skin	1.5	19	NR	NR	8
	Washington	WA Coast	Quinault	10	muscle w/o skin	1.8	16	NR	NR	8
		Puget Sound Chinook salmon Average				3.8	53	21.3	22.5	
		Washington Coast Chinook salmon Average				1.7	17	NR	NR	
		Washington Chinook salmon Average				3.5	48	21.3	22.5	
	Oregon	unknown	unknown	3	muscle w/skin	NR	10	NR	2.10	5*, 6*
	Oregon	Columbia River	unknown Fall	17	whole body	NR	18	19.9	3.69	10
	Oregon	Columbia River	unknown Spring	20	whole body	NR	33	34.8	9.77	10
	Oregon	Columbia River	mixed fall Chinook	15	muscle w/skin	7.0	37	21.0	NR	17
	Oregon	Columbia River	mixed spring Chinook	24	muscle w/skin	9.0	38	22.0	NR	17
	Oregon	Columbia River	fall Chinook	4	whole body	9.4	15	NR	2.30	15
	Oregon	Columbia River	Clackamas River	3	muscle w/skin	8.8	13	NR	1.80	15
	Oregon	Columbia River	Clackamas River	3	muscle w/o skin	6.1	10	NR	1.50	15
		Oregon Chinook salmon average				8.1	22	24.4	3.53	
	California	Sacramento /San Joaquin	unknown	29	whole body	NR	14	33.6	2.56	10
	Chinook salmon Average					5.6	29	15.7	6.22	
Sockeye salmon	Alaska	unknown	Alaska	2	muscle w/o skin	NR	3.6	NR	0.21	4
	Alaska	Aleutian Islands	unknown	13	muscle w/o skin	5.8	130	6.9	NR	3
	Alaska	Kodiak	unknown	3	muscle w/skin	NR	5.0	NR	0.10	5*, 6*
	Alaska	Gulf of Alaska/ Berring Sea	unknown	24	muscle w/o skin	8.2	13	12.0	0.22	20
	Alaska	Gulf of Alaska/ Berring Sea	Copper River	97	muscle w/o skin	5.5	37	12.2	NR	18**
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	13.3	NR	0.10	5*, 6*
		Alaskan sockeye salmon average				6.5	14.4#	10.4	0.16	
	British Columbia	unknown	unknown	3	muscle w/skin	NR	8.0	NR	0.10	5*, 6*
	British Columbia	Fraser River	Early Stuart	3	soma	16	13	NR	NR	7**
	British Columbia	Fraser River	Early Stuart	5	muscle w/o skin	4.0	3.9	NR	NR	7**
	British Columbia	Fraser River	Early Stuart	6	muscle w/o skin	5.0	6.9	NR	NR	7**
	British Columbia	Fraser River	Adams	5	muscle w/o skin	8.8	7.7	6.6	NR	16**

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Species	Region	sub-region	Population	n	Tissue Analyzed	Lipid (%)	PCBs	DDTs	PBDEs	Citation
	British Columbia	Fraser River	Weaver Creek	3	muscle w/o skin	1.4	6.8	NR	NR	7**
	British Columbia	Fraser River	Weaver Creek	2	muscle w/o skin	1.1	3.6	NR	NR	7**
	British Columbia	Fraser River	Weaver Creek	2	muscle w/o skin	1.5	5.3	NR	NR	7**
	British Columbia	Fraser River	Weaver Creek	1	muscle w/o skin	1.1	4.0	NR	NR	7**
	British Columbia	Fraser River	Weaver	8	muscle w/o skin	3.9	6.8	5.4	NR	16**
	British Columbia	West Coast VI	Great Central Lk.	6	muscle	6.1	1.7	NR	NR	7**
	British Columbia	West Coast VI	Great Central Lk.	3	muscle	6.6	1.6	NR	NR	2**
	British Columbia	West Coast VI	Great Central Lk.	2	muscle	1.0	1.5	NR	NR	2**
	British Columbia	West Coast VI	Great Central Lk.	3	muscle	1.0	2.4	NR	NR	2**
	British Columbian sockeye salmon Average					4.4	5.2	6.00	0.10	
	Sockeye salmon Average					4.8	7.6#	8.6	0.15	
Steelhead	Oregon	Columbia River		21	muscle w/skin	6.0	34	21.0	NR	17
Coho Salmon	Alaska	unknown	unknown	2	muscle w/o skin	NR	1.6	NR	0.32	4
	Alaska	Kodiak	unknown	3	muscle w/skin	NR	4.0	NR	0.10	5*, 6*
	Alaska	seak/goa	unknown	14	muscle w/o skin	2.9	2.0	1.5	0.19	20
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	4.0	NR	0.10	5*, 6*
	Alaskan coho salmon Average					2.9	2.9	1.5	0.18	
	British Columbia	unknown	unknown	3	muscle w/skin	NR	6.0	NR	0.30	5*, 6*
	Washington	Puget Sound	unknown	32	muscle w/o skin	3.1	35	NR	NR	9
	Washington	Puget Sound	PS mixed	125	muscle w/o skin	3.1	27	NR	NR	9
	Washington	Puget Sound	PS mixed	266	muscle w/o skin	3.3	NR	11.7	NR	19
	Washington coho salmon Average					3.2	31	11.7	NR	
	Oregon	Columbia River	Umatilla River	3	muscle w/skin	2.5	35	41.0	NR	17
	Coho salmon Average					3.0	14	18.1	0.20	
Pink salmon	Alaska	Kodiak	unknown	3	muscle w/skin	NR	3.0	NR	0.10	5*, 6*
	Alaska	northern Alaska	unknown	7	canned	6.3	2.6	1.8	NR	21
	Alaska	SE Alaska/GOA	unknown	12	muscle w/o skin	3.5	1.3	0.6	0.22	20
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	2.0	NR	0.10	5*, 6*
	Alaskan pink salmon Average					4.9	2.2	1.2	0.14	
	British Columbia	unknown	unknown	3	muscle w/skin	NR	3.0	NR	0.30	5*, 6*

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Species	Region	sub-region	Population	n	Tissue Analyzed	Lipid (%)	PCBs	DDTs	PBDEs	Citation
	Pink salmon Average					4.9	2.4	1.2	0.18	
Chum salmon	Alaska	Kodiak	unknown	3	muscle w/skin	NR	2.0	NR	0.10	5*, 6*
	Alaska	SE Alaska	unknown	3	muscle w/skin	NR	3.0	NR	0.10	5*, 6*
	Alaska	Bering Sea	unknown	18	muscle w/o skin	4.8	3.2	1.9	0.16	20
	Alaskan chum salmon Average					4.8	2.7	1.9	0.12	
	British Columbia	unknown	unknown	3	muscle w/skin	NR	2.0	NR	0.20	5*, 6*
	Chum salmon Average					4.8	2.6	1.9	0.14	
(1) Cullon *et al.* 2009, (2) Debruyn *et al.* 2004, (3) Hardell *et al.* 2010, (4) Hayward *et al.* 2007, (5) Hites *et al.* 2004a, (6) Hites *et al.* 2004b,										
(7) Kelly *et al.* 2007, (8) Missildine *et al.* 2005, (9) O'Neill *et al.* 1998, (10) O'Neill *et al.* 2006, (11) O'Neill and West 2009,										
(12) Rice and Moles 2006, (13) Shaw *et al.* 2008, (14) Shaw *et al.* 2006, (15) Stone 2006, (16) Veldhoen *et al.* 2010,										
(17) US EPA 2002, (18) Ewald *et al.* 1998, (19) West *et al.* 2001, (20) ADEC 2011, (21) O'Hara *et al.* 2005										
* estimated values from figure										
** estimated value from reported lipid weight										
#excluded value as an outlier										

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Toxic Chemicals. Contaminants enter fresh and marine waters and sediments from numerous sources such as atmospheric transport and deposition, ocean current transport, and terrestrial runoff (Iwata *et al.* 1993, Grant and Ross 2002, Hartwell 2004), but are typically concentrated near populated areas of high human activity and industrialization. Oceans act as a repository for domestic and industrial wastes and significant contaminant concentrations have been measured in the sediment, water, and biota. Persistent contaminants can biomagnify or accumulate up the food chain in such a degree where levels in upper trophic-level mammals can have significantly higher concentrations than that found in the water column or in lower trophic-level species. Southern Resident killer whales are exposed to relatively high levels of persistent pollutants because they are long-lived, upper trophic-level predators that are in close proximity to industrial and agricultural areas. Consequentially, Southern Residents are a highly contaminated whale population.

Persistent pollutants are highly lipophilic (*i.e.*, fat soluble) and are primarily stored in the fatty tissues in marine mammals (O'Shea 1999, Reijnders and Aguilar 2002). Therefore, when killer whales consume contaminated prey they store the contaminants primarily in their blubber. However, some persistent contaminants (*e.g.*, the butyltins) are primarily stored in the liver and kidneys of marine mammals (Iwata *et al.* 1997). Persistent pollutants can resist metabolic degradation and can remain stored in the tissues or organs of an individual whale for extended periods of time. When prey is scarce and when other stressors reduce foraging efficiency (*e.g.*, as possible from vessel disturbance, disease, *etc.*), killer whales metabolize their blubber lipid stores and the contaminants can become mobilized to other organs or they can remain in the blubber and become more concentrated (Krahn *et al.* 2002). Nursing mothers can also transmit large quantities of contaminants to their offspring, particularly during lactation. The mobilized contaminants can reduce the whales' resistance to disease, can affect reproduction, disrupt the endocrine system, disrupt enzyme function and vitamin A physiology, induce developmental neurotoxicity, and cause skeletal deformities (see NMFS 2008a for a review).

There are several persistent pollutants of concern that have been highlighted in the Southern Resident killer whale Recovery Plan (Table 2.4.4. 1.4). Some of these pollutants do not need to be in high concentration in a species to be toxic and have long been recognized as problematic for the Southern Resident killer whales. The organochlorines (*e.g.*, PCBs and DDTs) are thought to pose the greatest risk to killer whales (Ross *et al.* 2000, Center for Biological Diversity 2001, Krahn *et al.* 2002). Organochlorines are a diverse group of lipophilic compounds. Designed for their stability, most are highly persistent in the environment and can resist metabolic degradation. These persistent pollutants can accumulate in the food webs and are at relatively high concentrations in upper trophic-level species such as killer whales. PCBs were designed for chemical stability and were historically used in paints and sealants, industrial lubricants and coolants, and flame-retardants. DDTs were primarily used to control insects in commercial and agricultural areas, forests, homes and gardens. PCBs and DDTs were banned in the 1970s and 1980s due to their toxicity in humans and wildlife. Although levels of PCBs and DDTs have dramatically decreased in environmental samples since the mid 1970s (Mearns *et al.* 1988, Lieberg-Clark *et al.* 1995, Calambokidis *et al.* 2001, Rigét *et al.* 2010), these compounds continue to be measured in marine biota around the world, including killer whales and their prey.

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Many studies have found organochlorines in marine mammal tissues (*e.g.*, Appendices 10-1 through 10-4, O'Shea 1999). Several marine mammal populations have high levels of organochlorines associated with adverse health effects. For example, the St. Lawrence beluga population contains high levels of organochlorines, as well as lead, mercury, and selenium (Martineau *et al.* 1987, Muir *et al.* 1990, Wagemann *et al.* 1990). This beluga whale population has a high prevalence for tumors, and lesions in the digestive tract and mammary glands, which are thought to be associated with the high levels of contaminants, particularly PCBs (Martineau *et al.* 1994, De Guise *et al.* 1995).

The majority of Southern Residents have high levels of PCBs (Ross *et al.* 2000, Krahn *et al.* 2007a, 2009) that exceed a health-effects threshold (17,000 ng/g lipid) derived by Kannan *et al.* (2000) and Ross *et al.* (1996) for PCBs in marine mammal blubber. The PCB health-effects threshold is associated with reduced immune function and reproductive failure in harbor seals (Reijnders 1986, de Swart *et al.* 1994, Ross *et al.* 1996, Kannan *et al.* 2000). Hickie *et al.* (2007) projected that it will take at least 50 years for the Southern Residents to drop below the threshold. Moreover, juvenile Southern Resident killer whales had blubber concentrations that were 2 to 3.6 times higher than the established health-effects threshold (Krahn *et al.* 2009). Similarly, Southern Residents also have high levels of measured DDTs in their blubber (Krahn *et al.* 2007a, 2009).

Recent decades have brought rising concern over a list of the so-called "emerging" contaminants and other pollutants, such as the PBDEs. PBDEs have been used as additive flame-retardants in many products including electronics, textiles, and plastics. Additive flame-retardants can readily disassociate from the products they are added to and discharge into the environment. Due to the increase in fire regulations in many countries, the use of PBDEs has increased in the last few decades. PBDEs have been identified as a growing concern and have a ubiquitous distribution with increasing levels found in various matrices including surface water, sewage sludge, sediment, air, and biota (Hale *et al.* 2003, Hites 2004). PBDEs are structurally comparable to PCBs and share some similar toxicological properties (Hooper and McDonald 2000). In January 2006, the Washington State Department of Ecology (DOE) and the Washington State Department of Health (DOH) issued a Final PBDE Chemical Action Plan (DOE and DOH 2006) that recommended the Legislature prohibit the three main types of PBDEs used in consumer products (*e.g.*, penta-, octa-, and deca-BDEs). The penta and octa forms are currently being phased out in Washington State because manufacturers agreed to voluntarily stop producing these two forms of PBDEs by the end of 2004, and following a bill (ESHB1024) that was passed in 2007. This bill banned the use of the penta and octa forms by 2008, banned the use of the deca form in mattresses by 2008, and banned the use of the deca form in televisions, computers, and furniture by 2011.

Although specific regional data is limited for PBDE levels, the environmental levels of a few PBDE congeners appear to have surpassed PCBs in some areas in North America (Hale *et al.* 2003, Ross *et al.* 2009). Recent studies have documented relatively high concentrations of PBDEs in Southern Resident killer whales (Krahn *et al.* 2007a, 2009, Mongillo 2009). Although PBDE levels in the whales are lower than PCBs or DDTs (Krahn *et al.* 2007a, 2009), concern is growing because PBDE exposure and accumulation will likely continue in the future increasing the risk to the health of the killer whales. Several other marine species have recently experienced

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an almost exponential increase in PBDE concentrations (*e.g.*, Ikonomou *et al.* 2002, Lebeuf *et al.* 2004).

Recent studies suggest that certain pharmaceuticals and personal care products (PPCPs) may also accumulate in killer whales. Synthetic musks and antibacterial chemicals (*e.g.* Triclosan) have been detected in dolphins and porpoises in coastal waters off Japan and the southeastern United States and in harbor seals off the California Coast (Fair *et al.* 2009, Kannan *et al.* 2005, Nakata 2005, Nakata *et al.* 2007). A wider range of PPCPs, including anti-depressants, cholesterol lowering drugs, antihistamines, and drugs affecting blood pressure and cholesterol levels have been detected in tissues of fish from urban areas and sites near wastewater treatment plants (Brooks *et al.* 2005, Ramirez *et al.* 2009), suggesting possible contamination of prey. As yet we have no data on concentrations of PPCPs in either killer whales or their prey species, but they could be a concern because of their widespread occurrence, potential for biomagnification, and biological activity.

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Table 2.4.4. 1.4. Persistent pollutants that may pose a risk to resident killer whales. From Table 1 in Killer Whale Recovery Team (2007). Updated from NMFS (2008a).

Pollutant	Use/Source	Persistent	Bio-accumulate	Risk
DDT (Dichlorodi-phenyl trichloroethane	pesticide used in some countries, banned in North America, persists in terrestrial runoff 30 years post ban, enters atmosphere from areas where still in use	yes	yes	Reproductive impairment, immunosuppression, adrenal and thyroid effects
PCBs Polychlorinated Biphenyls	electrical transformer and capacitor fluid, limited use in North America but enters environment from runoff, spills and incineration	yes	yes	reproductive impairment, skeletal abnormalities, immunotoxicity and endocrine disruption
Dioxins and Furans	by-product of chlorine bleaching, wood product processing and incomplete combustion. Mills less of a source now. Current sources include burning of salt-laden wood, municipal incinerators, and residential wood and wood waste combustion, in runoff from sewage sludge, wood treatment	yes	yes	thymus and liver damage, birth defects, reproductive impairment, endocrine disruption, immunotoxicity and cancer
PAHs Persistent Polycyclic aromatic hydrocarbons	by-product of fuel combustion, aluminum smelting, wood treatment, oil spills, metallurgical and coking plants, pulp and paper mills	yes	no	Carcinogenic
flame retardants, esp. PBBs and PBDEs Polybrominated diphenyl ethers	flame retardants; in electrical components and backings of televisions and computers, in textiles and vehicle seats, ubiquitous in environment. 2/3 product PBDEs banned in Europe. Same two products withdrawn from North American marketplace in 2005, but one (deca) product still used globally.	yes	yes	endocrine disruption, impairs liver and thyroid
PFOs Perfluro-octane sulfonate	stain, water and oil repellent (included in Scotchgard until recently), fire fighting foam, fire retardants, insecticides and refrigerants, ubiquitous in environment	yes	yes but in blood, liver, kidney and muscle	promotes tumor growth
TBT, DBT Tributyltin Dibutyltin	antifoulant pesticide used on vessels	yes	yes	unknown but recently associated with hearing loss

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Pollutant	Use/Source	Persistent	Bio-accumulate	Risk
PCPs (Polychlorinated paraffins)	flame retardants, plasticizers, paints, sealants and additives in lubricating oils	yes	yes	endocrine disruption
PCNs Polychlorinated napthalenes	ship insulation, electrical wires and capacitors, engine oil additive, municipal waste incineration and chlor-alkali plants, contaminant in PCBs	yes	yes	endocrine disruption
APEs Alkyl-phenol ethoxylates	detergents, shampoos, paints, pesticides, plastics, pulp and paper mills, textile industry found in sewage effluent and sediments	moderate	moderate	endocrine disruption
PCTs Polychlorinated terphenyls	fire retardants, plasticizers, lubricants, inks and sealants, enters environment in runoff	yes	yes	endocrine disruption and reproductive impairment
References: Primarily Grant and Ross 2002, but also Lindstrom *et al.* 1999, Hooper and MacDonald 2000, Kannan *et al.* 2001, Hall *et al.* 2003; Van de Vijver *et al.* 2003, Rayne *et al.* 2004, Song *et al.* 2005.				

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Below we highlight the available information about marine mammal toxicity, storage, concentration levels, and detoxification mechanisms for toxic chemicals considered in the proposed action, as introduced in Table 1.1. We first discuss the organic compounds: dieldrin, endrin, endosulfan, heptachlor epoxide, Lindane, pentachlorophenol (PCP), and tributyltin (TBT). Second, we discuss the metals and elemental pollutants: cadmium, lead, aluminum, ammonia, arsenic, copper, chromium (III and VI), nickel, selenium, silver, and zinc. Of all the chemicals described below that are a part of this action, the organic compounds are of highest concern, followed by the metals and elemental pollutants.

Dieldrin and Endrin. Dieldrin and endrin are organochlorine insecticides that are more acutely toxic than DDT. They are highly neurotoxic and can cause reproductive defects in laboratory mammals (O'Shea 1999). Reproductive effects can include reduced fertility, reduced litter size, and increased pup mortality in mice, rats, and dogs (AMAP 1998). Furthermore, dieldrin has shown to be estrogenic, cause immunosuppression in laboratory animals, and increase benign and malignant tumors in mice (AMAP 1998).

By the end of the 1960s, dieldrin had been reported in tissues of marine mammals (O'Shea and Tanabe 2003). Dieldrin is commonly found in marine mammals throughout the world, whereas endrin, which is more toxic, is reported less often (see Appendices 10-1 to 10-4, O'Shea 1999). In the late 1980s, dieldrin was measured in the tissues of killer whales of the west coast of North America (Jarman *et al.* 1996). Concentration values revealed a geometric mean of 340 μg/kg wet weight (ww); this average level was appreciably less than the total DDT (32,000 μg/kg ww) and total PCB (22,000 μg/kg ww) in the six killer whales that were sampled (Jarman *et al.* 1996). Similarly, in a separate study, dieldrin levels in stranded or dead North Atlantic killer whales were measurably less than PCBs and DDTs (McHugh *et al.* 2007). Ylitalo *et al.* (2009) measured persistent organic pollutant concentrations including dieldrin in the false killer whale from the Hawaiian Islands. Dieldrin measured in these whales were relatively low. Subadults had significantly higher mean dieldrin levels compared to those measured in other age classes. Concentrations of dieldrin measured in blubber of Southern Residents sampled from 2004-2007 ranged from 9.2 ng/g wet weight (ww) to 440 ng/g ww, whereas the lipid-normalized levels ranged from 32 ng/g lipid to 1,100 ng/g lipid (G. Ylitalo NWFSC, pers. comm.).

Endosulfan. Endosulfan is a semi-volatile and relatively persistent organochlorine. It has shown to be estrogenic and cause reproductive effects in laboratory animals (AMAP 1998). It has high acute oral and inhalation toxicity as well as moderate dermal toxicity in humans (http://www.epa.gov/oppsrrd1/REDs/factsheets/endosulfan_fs.htm). Small and Solomon (2005) concluded that risk from endosulfan in marine mammals was negligible because the range of exposure concentrations were lower than the no observed adverse effect level (NOAEL) doses in laboratory species (*e.g.*, rat and grey partridge, see Figure 2.4.4. 1.3).

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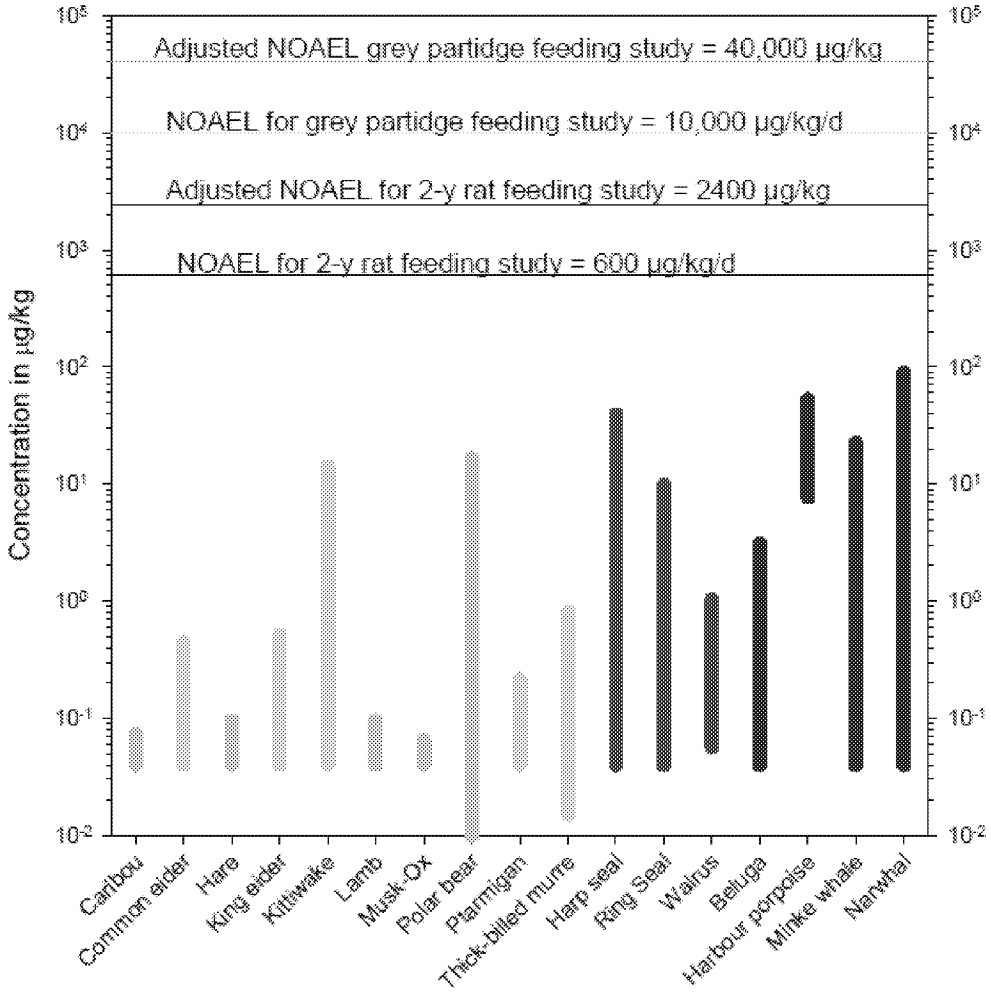

Figure 2.4.4. 1.3. Range of exposure concentrations measured in various polar marine and terrestrial wildlife species as compared to NOAEL doses in test species (reprinted from Small and Solomon 2005).

Endosulfan is present in several cetaceans such as the narwhal, beluga, and minke whales (Vorkamp *et al.* 2004, Small and Solomon 2005). The beluga whale appears to have varying levels depending on geographic location but no significant difference in concentration between sexes (Stern *et al.* 2005). Several studies focusing on the Arctic have shown the continued deposition of endosulfan from use at lower latitudes. Endosulfan is one of the few persistent organic pollutants that increased in concentration from the 1970s to the 1990s in the Canadian Arctic (Braune *et al.* 2005). However, there appears to be uncertainty in some of the datasets because of differences in analytical techniques (Weber *et al.* 2010). Endosulfan I (alpha endosulfan) levels in the blubber of false killer whales from the Hawaiian islands were below the limits of quantification (Ylitalo *et al.* 2009). Alpha endosulfan levels determined in blubber of the Southern Residents sampled between 2004 – 2007 were below the limits of quantification (< 2.2 - < 14 ng/g ww) for all samples analyzed and thus do not appear to currently pose a health risk (G. Ylitalo NWFSC, pers. comm.).

Heptachlor Epoxide. Heptachlor epoxide is a more toxic metabolite of heptachlor (which is prepared from chlordane and has a higher acute toxicity). Laboratory animals fed high levels in a short time period experienced tremors and convulsions (EPA 2008). Long term exposure can lead to liver and kidney tissue damage, enlarged liver, increased red blood cells, and liver cancer (EPA 2008).

Similar to dieldrin, heptachlor epoxide is found in marine mammals throughout the world but in relatively low concentrations (O'Shea 1999). Heptachlor epoxide can be offloaded from mother to offspring and is the primary metabolite of heptachlor found in marine mammals tissues (see Appendices 10-1 through 10-4, O'Shea 1999). In the late 1980s, heptachlor epoxide was measured in the tissues of killer whales of the west coast of North America (Jarman *et al.* 1996). Concentration values revealed a geometric mean of 120 μg/kg ww, respectively, which were appreciably less than DDTs and PCBs (Jarman *et al.* 1996). Blubber levels of heptachlor epoxide measured in Southern Residents sampled from 2004 – 2007 ranged from < 5.3 ng/g ww to 660 ng/g ww whereas the lipid-normalized values ranged from below the limits of quantification to 5,400 ng/g lipid (G. Ylitalo NWFSC pers. commun.).

Lindane. Hexachlorocyclohexane (HCH), also referred to as benzene hexachloride (BHC), is an organochlorine insecticide and consists of a number of isomers: γ-HCH (Lindane), α-HCH, and β-HCH. Lindane is the most biologically active isomer and is a neurotoxin; it affects the nervous system, liver and kidneys, and may act as an endocrine disruptor (http://www.epa.gov/oppsrrd1/REDs/factsheets/lindane_fs_addendum.htm). HCH isomers have caused tumors in laboratory mammals (O'Shea 1999). Lindane has shown to reduce immune responses in laboratory animals and may have both estrogenic and antiestrogenic effects (AMAP 1998).

Between 1986 and 1989, the average concentration of total HCHs (or the sum of Lindane, α-HCH, and β-HCH) measured in killer whales from the west coast of North America was 708 μg/kg ww, of that, the average lindane concentration was only 31 μg/kg ww (Jarman *et al.* 1996). More recently, total HCH was measured in Southern Resident killer whales (Krahn *et al.* 2007a, 2009). Similar to the previous study, total HCHs were measurably lower than PCBs or DDTs. The juvenile whales had significantly higher HCH levels than adult males and total HCH levels were strongly correlated with total PBDEs and did not correlate with age (Krahn *et al.* 2007a, 2009). Lindane concentrations in killer whales are relatively low, likely because it is less bioaccumulative than some of the other organochlorines, and it is potentially regulated by the whales' metabolic system (McHugh *et al.* 2007). Concentrations of total HCHs in the Southern Residents ranged from 62 ng/g to 1,700 ng/g lipid based on biopsy blubber samples collected from 2004 to 2007 (Table 2.4.4. 1.5).

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Table 2.4.4. 1.5. Persistent organic pollutants (ng/g lipid) and percent lipid in blubber of biopsy samples from Southern Resident killer whales (data from Krahn *et al.* 2007a, 2009).

Whale ID	Age	Sex	Lipid %	ΣPCBs	ΣDDTs	ΣPBDEs	ΣHCHs
J39	3	M	40.9	34,000	24,000	15,000	1,300
J38	4	M	20.9	41,000	24,000	14,000	1,000
J22	22	F	28.4	4,600	1,500	880	62
J19	27	F	29.4	45,000	26,000	7,500	310
K36	4	F	18.3	62,000	95,000	15,000	1,700
K34	6	M	22.3	39,000	61,000	10,000	1,200
K21	21	M	26.6	38,000	73,000	2,900	410
K13	35	F	22	8,900	11,000	1,200	300
K7	est 97	F	28.5	120,000	44,000	6,700	1,100
L78	15	M	15.2	22,000	38,000	2,600	630
L85	15	M	24.8	50,000	120,000	2,500	530
L87	15	M	25.6	24,000	44,000	2,600	410
L71	18	M	9.6	36,000	72,000	2,600	920
L74	18	M	18	45,000	86,000	3,100	720
L73	21	M	23.8	32,000	55,000	3,400	450
L67	22	F	29.2	5,600	4,300	680	150
L57	29	M	19.4	56,000	110,000	3,300	640
L26	est 51	F	22.1	17,000	27,000	4,400	580
L21	est 57	F	18.7	55,000	99,000	4,200	750

Total HCH levels in Southern Resident killer whales are generally higher than resident killer whales from Central Aleutian Islands, and less than transient killer whales from the Eastern Aleutian Islands (EAI) and from California (Krahn *et al.* 2007b). In fact, the transients from the EAI had significantly higher total HCHs than all other whale groups sampled (Krahn *et al.* 2007b). Herman *et al.* (2005) also found higher total HCH levels in transient killer whales from the eastern North Pacific (mean of 11,500 ng/g lipid) compared to residents (mean of 470 ng/g lipid) followed by the offshore ecotype (mean of 120 ng/g lipid). Relatively low levels of HCH are not uncommon in other killer whale populations. In a separate study, organochlorines were measured in live stranded or dead North Atlantic killer whales (McHugh *et al.* 2007). Similar to previous studies, lindane in individual blubber tissues were relatively low compared to PCBs and DDTs. Blubber levels of Lindane measured in Southern Residents sampled from 2004 – 2007 ranged from < 1.9 ng/g ww to 17 ng/g ww, whereas the lipid-normalized valued ranged from below the limits of quantification to 42 ng/g lipid (G. Ylitalo NWFSC pers. commun.).

Pentachlorophenol (PCP). Pentachlorophenol (PCP) is an organochlorine pesticide and disinfectant, however its greatest use is as a fungicide (wood preservative). PCP is still currently used, but to a lesser degree than in the 1990s. The use of chlorophenol-based chemicals for wood treatment was a major source of dioxins and furans to the Georgia Basin (Garrett and Ross 2010). Although adverse health effects are unknown in marine mammals, chlorophenols (such as

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PCP) can adversely affect the survival, reproduction, growth, and metabolism of fish and shellfish (Garrett and Ross 2010).

Data are limited on PCP concentrations in marine mammals, with no information available for Southern Residents. These compounds are less persistent than other organic compounds because they readily degrade in the environment, and there is no evidence of biomagnification in upper trophic-level species (Garrett and Ross 2010). However, PCP was measured in bowhead whale plasma and was relatively abundant compared to similar phenolic compounds (Hoekstra *et al.* 2003). Because long-range transport of PCPs is limited due to rapid photolysis, they do not readily bioaccumulate. It is assumed that PCPs found in these whales result from biotransformation of hexachlorobenzene or potentially a biotransformation of pentachloroanisole (Hoekstra *et al.*2003).

Tributyltin (TBT). Tributyltin has been used as an antifoulant on ships, buoys, nets and piers to restrict or retard growth of fouling organisms. It has been identified as a persistent organic pollutant that may pose a toxic threat to the Southern Resident killer whales (NMFS 2008a). However, bioaccumulation appears to be less than other persistent pollutants (*e.g.*, PCBs, DDTs, and PBDEs).

TBT acts as an endocrine disruptor and has shown to competitively inhibit aromatase cytochrome P450 activity (Heidrich *et al.* 2001). Aromatase plays a significant role in sustaining the ratio between male and female hormones during sexual differentiation during embryonic development. TBT inhibits the conversion of androgens to estrogens. TBT can also act synergistically with a PCB congener (PCB-126) known to induce P4501A, and produce opposite effects than when the chemicals are isolated at higher doses. For example, female mice exposed to high doses of TBT combined with PCB-126 inhibited P450 activity, whereas low doses of TBT combined with the PCB congener enhanced the activity (DeLong and Rice 1997). Although TBT can significantly inhibit P450 activities, the concentration levels in the liver at which this inhibition occurs is almost 25 times higher than that found in free-ranging marine mammals (Kim *et al.* 1998). However, some marine mammal populations are at or above TBT levels that cause immunotoxicity in laboratory species (Figure 2.4.4. 1.4).

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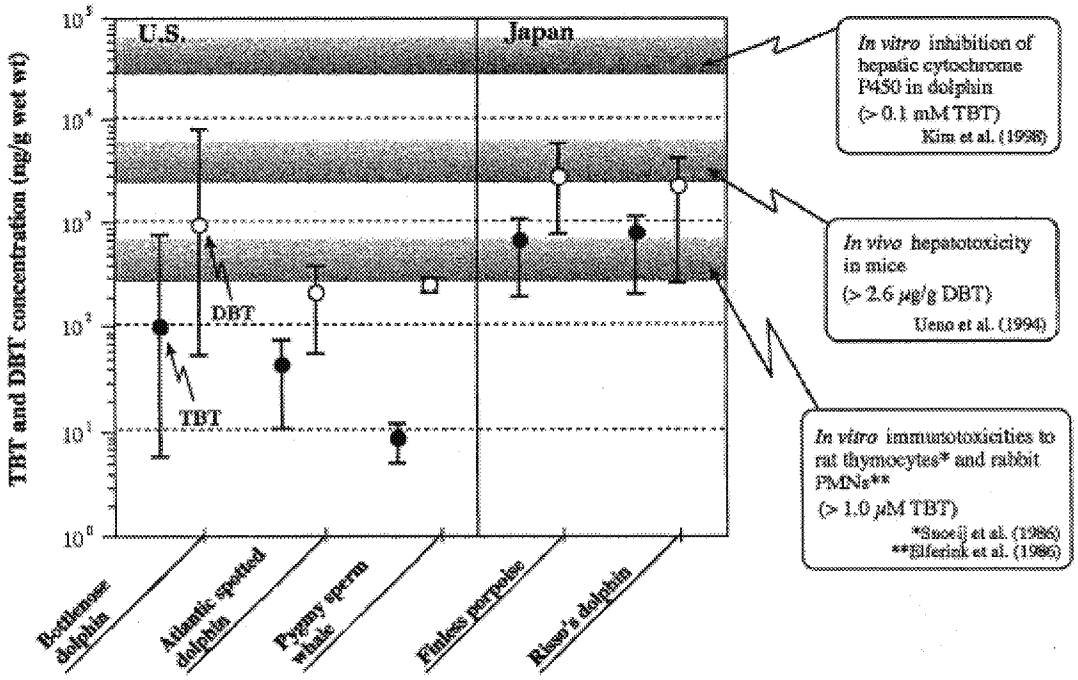

Figure 2.4.4. 1.4. Range of tributyltin (TBT) and a metabolite, dibutyltin (DBT), concentrations in the liver of cetaceans from the U.S. and Japanese coastal waters, and toxic effects threshold levels of TBT and the DBT metabolite. Reprinted from Tanabe (1999).

The distribution of TBT in the tissues and organs of marine mammals is similar to that of other species and are primarily in the liver and kidneys and lower in the muscles and blubber (Iwata *et al.* 1997, Tanabe 1999). Currently, butyltin concentrations in Southern Residents are unknown. Therefore, the extent of contamination relative to effect thresholds is unknown. Cetaceans distributed near more developed nations have elevated TBT levels compared to cetaceans adjacent to developed nations (Tanabe *et al.* 1998). Therefore, it is likely that the Southern Residents have relatively high TBT concentrations compared to cetaceans in less industrialized regions. Butyltin concentrations in cetaceans off of Japan and USA are similar. For example, the mean TBT liver concentration in killer whales off Japan (n=3) was 180 ng/g ww (Tanabe *et al.* 1998), and the mean TBT liver concentration in bottlenose dolphins off southeast Atlantic and Gulf coasts was 100 ng/g ww (Kannan *et al.* 1997). These levels are higher than concentrations in cetaceans near the Philippines, India, and China (Kannan *et al.* 1997, Tanabe *et al.* 1998). Transplacental transfer of TBT from mother to fetus is relatively low compared to other persistent pollutants. For example, TBT concentrations in the liver of a pregnant female killer whale (150 ng/g ww) was much higher compared to concentrations in the liver of the fetus (26 ng/g ww) (Tanabe *et al.* 1998). TBTs do not appear to differ between males and females, however increasing levels have been observed in immature stages of Risso's dolphins (Tanabe 1999).

Metals and Elemental Pollutants. Unlike the persistent pollutants described above, metals are naturally found in the environment and some are essential to an animals' nutrition. Heavy metals in marine mammals are primarily determined by the levels in prey and the geographic region, as well as age and gender of the individual. For example, marine mammals that feed on squid can be exposed to higher levels of cadmium, copper, and zinc because squid have the ability to retain these elements (Reijnders and Aguilar 2002). Human activities can increase the concentrations and metals can become toxic at certain exposure levels. Currently, there is little information on metals in killer whales or in their prey. Most metals, like persistent pollutants, settle to the ocean floor where they can accumulate in sediment. Therefore, areas with high human activity can become hotspots of multiple toxic chemicals.

The distribution or storage of heavy metals in marine mammals is dependent on the metal. In general, heavy metals are found in the liver, kidneys, muscles, and bones (O'Shea 1999, Reijnders and Aguilar 2002, Das *et al.* 2003). Some metals may transfer from mother to offspring during gestation and lactation, although not to the same degree as the persistent organic pollutants. For example, Honda *et al.* (1987) found the hepatic concentrations of iron, lead, nickel, and cobalt decreased in adult female southern minke whales with progress of gestation. Pregnant pilot whales had less mercury in the serum than non-pregnant females, indicating a potential transplacental transfer to the fetus (Nielsen *et al.* 2000). However, it may also be possible that a change in the diet of the pregnant pilot whales can explain the change in mercury levels (Nielsen *et al.* 2000).

Non-essential metals that can be toxic to marine mammals, even at low doses, include mercury, cadmium, and lead. Mercury, cadmium, and lead in the tissues of marine mammals have been the focus of several studies because of their known toxicity to humans and other wildlife, such as damage to the central nervous system, skeletal deformities, kidney lesions and kidney or liver damage, as well as carcinogenic, mutagenic, and teratogenic effects (O'Shea 1999, Das *et al.* 2003). However, little information is known about toxic effects of heavy metals in marine mammals. Essential metals that occur naturally in the environment can also be toxic and their concentrations can be elevated in areas of high human activities. These essential metals include copper, zinc, iron, and selenium. Below is a brief description of toxicity, storage, concentration levels, and detoxification mechanisms for the metals and elements discussed in this opinion.

Cadmium. Adverse health effects from high exposure to cadmium (or cadmium compounds) in mammals include reduced growth, impaired immunity, cancer, and renal dysfunction, whereas acute exposure can cause dystrophic changes in several organs including the liver, heart, and kidneys (Grant and Ross 2002 as cited in Government of Canada *et al.* 1993). Dietz *et al.* (1998) suggests that marine mammals in the Arctic regions may have habituated to naturally high levels of cadmium. For example, cadmium concentrations in ringed seals from Greenland are higher than the health-effects threshold for kidney damage (200 μg/g wet weight, WHO 1992). This health effects threshold has been more recently considered an overestimation, and that renal dysfunction from cadmium exposure has been observed at concentrations of only 50 μg/g wet weight (Elinder and Järup 1996). The ringed seals that had cadmium concentrations above both of the thresholds still displayed normal renal structure (Dietz *et al.* 1998). Despite the high levels of cadmium found in marine mammals (*e.g.*, Nielsen *et al.* 2000, O'Shea 1999 and Government of Canada *et al.* 1993), no toxic effect has been

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observed indicating a potential detoxification mechanism (described further below). Liver levels of cadmium in an adult female transient killer whale that stranded at Dungeness Spit in 2002 were < 0.15mg/kg ww (G. Ylitalo NWFSC, pers. comm.).

Lead. Chronic exposure to lead in mammals can cause disorders of the nervous system, renal system, and gastrointestinal tract, impaired or weakened mental function, anemia, and variable immunotoxic effects (O'Shea 1999, Grant and Ross 2002, De Guise *et al.* 2003). Exposure to high concentrations of lead in mammals has lead to hypertension, reproductive disorders, and metabolic and neurological issues (Grant and Ross 2002). Long-term storage of lead primarily occurs in the bone; however, lead can be released with calcium into the bloodstream (Grant and Ross 2002).

Only a limited number of studies have measured lead concentrations in the bone of marine mammals. The few studies that have measured lead in the bone reported negligible concentrations (O'Shea 1999, Das *et al.* 2003, O'Hara *et al.* 2003). One of the highest concentrations of lead measured in the bone of marine mammals was approximately 61.6 ppm (wet weight) in a bottlenose dolphin from an area known for emissions from a lead smelter (O'Shea 1999 as cited in Kemper *et al.* 1994). In most studies, levels in tissues of marine mammals have not been reported at levels that were a cause for concern and were within normal ranges and included concentrations less than 1ppm (O'Shea 1999). Liver levels of lead in an adult female transient killer whale that stranded at Dungeness Spit in 2002 were < 0.15mg/kg ww (G. Ylitalo NWFSC, pers. comm.).

Detoxification Mechanisms. Some marine mammals (particularly from the northern arctic regions) appear to tolerate high levels of mercury, lead, and cadmium and are able to detoxify them through several processes. Cadmium and mercury can combine with selenium or metallothionein (MT, a protein molecule) to mitigate the toxic effects of exposure (Rooney 2007, Klaassen *et al.* 2009). These new complexes (mercury and selenium or cadmium and MT) in the liver or kidneys mitigate toxic effects and change the metals into non-toxic forms (Klaassen *et al.* 2009). This detoxification mechanism appears to be species-specific. For example, unlike in sperm whales that did not show an obvious relationship between mercury and selenium, pilot whales demonstrated a strong correlation between mercury and selenium with an almost fourfold higher molar ratio than that found in the sperm whales (Nielsen *et al.* 2000).

Other Metals and Elements. Aluminum, ammonia, arsenic, copper, chromium (III and VI), nickel, selenium, silver, and zinc are not primary toxic chemicals of concern for marine mammals compared to mercury, cadmium, or lead, because they are either essential to the nutrition of the animal and are found at relatively low concentrations (*e.g.*, aluminum, nickel, selenium, and zinc), the available data does not support a health risk from exposure (O'Shea 1999, O'Hara *et al.* 2003), or because the element does not build up in the food chain (*e.g.*, ammonia). Arsenic has been measured in marine mammals, but not at levels considered to be toxic (O'Shea 1999). Concentrations of arsenic tend to be higher in lower trophic level species and there is no evidence that arsenic biomagnifies (Garrett and Ross 2010). Selenium, zinc, and copper are all essential elements for the nutrition of animals. Effects in mammals exposed to high copper concentrations include genetic and developmental abnormalities, and renal failure (Grant and Ross 2002). Although low concentrations of copper have been measured in marine

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mammals, chronic exposure to copper may be of concern to killer whales because anthropogenic activities can result in increased levels near urban and industrial areas (Grant and Ross 2002). Copper in the liver of marine mammals declines with age, however differences in copper concentrations in populations have been reported after accounting for age (Stein *et al.* 2003). For example, copper concentrations declined in the livers of bottlenose dolphins in Florida and Texas, however the dolphins from Florida had lower concentrations (Stein *et al.* 2003). In general, mammals are more sensitive to chromium (VI) than to chromium (III) and biomagnification factors are relatively low and increased concentrations up the food chain have not been observed (Garrett and Ross 2010). Recent evidence indicates chromium (VI) is cytotoxic and genotoxic to North Atlantic right whale lung and testes cells, indicating chromium (VI) may be a significant risk factor to these whales (Wise *et al.* 2008). They suggest inhalation is likely an important exposure route. Chromium (VI) was also cytotoxic and clastogenic to Steller sea lion lung cells (Wise *et al.* 2009). Lastly, research on selenium in marine mammals has been primarily focused on its ability to form a non-toxic complex with mercury.

Extinction Risk. In conjunction with the 2004 status review, NMFS conducted a population viability analysis (PVA) for Southern Resident killer whales (Krahn *et al.* 2004). Demographic information from the 1970s to fairly recently (1974-2003, 1990-2003, and 1994-2003) were considered to estimate extinction and quasi-extinction risk. The NMFS defined "quasi-extinction" as the stage at which 10 or fewer males or females remained a threshold from which the population was not expected to recover.

The model evaluated a range in Southern Resident survival rates, based on variability in mean survival rates documented from past time intervals (highest, intermediate, and lowest survival). The model used a single fecundity rate for all simulations. The study considered seven values of carrying capacity for the population ranging from 100 to 400 whales, three levels of catastrophic event (*e.g.*, oil spills and disease outbreaks) frequency ranging from none to twice per century, and three levels of catastrophic event magnitude in which 0, 10, or 20 percent of the animals died per event.

The analysis indicated that the Southern Resident killer whales have a range of extinction risk from 0.1 to 18.7 percent in 100 years and 1.9 to 94.2 percent in 300 years, and a range of quasi-extinction risk from 1 to 66.5 percent in 100 years and 3.6 to 98.3 percent in 300 years (Table 2.4.4. 1.6). The population is generally at greater risk of extinction as survival rate decreases and over a longer time horizon (300 years) than over a shorter time horizon (100 years) (as would be expected with long-lived mammals). There is a greater extinction risk associated with increased probability and magnitude of catastrophic events. The NWFSC continue to evaluate mortality rates and reproduction, and will complete work on a PVA similar to the analysis summarized above. Until these updated analyses are completed, the Krahn *et al.* (2004) analysis represents the best available science on extinction risk of Southern Resident killer whales.

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Table 2.4.4. 1.6. Range of extinction and quasi-extinction risk for Southern Resident killer whales in 100 and 300 years, assuming a range in survival rates (depicted by time period), a constant rate of fecundity, between 100 and 400 whales, and a range catastrophic probabilities and magnitudes (Krahn *et al.* 2004).

Time Period	Extinction Risk (%)		Quasi-Extinction Risk (%)	
	100 yrs	300 yrs	100 yrs	300 yrs
Highest survival	0.1 – 2.8	1.9 – 42.4	1.0 – 14.6	3.6 – 67.7
Intermediate survival	0.2 – 5.2	14.4 – 65.6	6.1 – 29.8	21.4 – 85.3
Lowest survival	5.6 – 18.7	68.2 – 94.2	39.4 – 66.5	76.1 – 98.3

2.5 Environmental Baseline

The 'environmental baseline' includes the past and present impacts of all Federal, state, or private actions and other human activities in the action area, the anticipated impacts of all proposed Federal projects in the action area that have already undergone formal or early section 7 consultation, and the impact of state or private actions which are contemporaneous with the consultation in process (50 CFR 402.02).

In this section, NMFS first provides information on water body segments in Oregon that currently fail to meet applicable water quality standards. Second, NMFS provides information on stormwater (MS4) and point-source (NPDES) permits in Oregon, in terms of spatial distribution and chemical-specific constituents, and species distribution, exposure potential via point-source discharges. And third, NMFS summarizes past and current human activities and describes how these activities influence current habitat conditions within the action area.

2.5.1 303(d)-Listed Waterbody Segments in Oregon

Under section 303(d) of the CWA, states and tribes are required to provide EPA a biennial list of water body segments that do not meet water quality standards. On its 2004/2006 303(d) list, the Oregon Department of Environmental Quality (ODEQ) identified more than 15,000 stream miles listed for at least one pollutant. Pollutants identified on the 303(d) list fall into several major groups which include sediment, nutrients, metals, bacteria, oxygen demand, and toxic organics. For this consultation NMFS focused on metals, toxic organics, and conventional pollutants, (*i.e.*, temperature, pH, and dissolved oxygen) as these pollutants can affect the toxicity of metal and organic pollutants. Figure 2.5.1.1.1 identifies toxics associated with those listed in Table 1.1 that were detected in one or more watersheds in Oregon by the USGS. Figures 2.5.1.1.2 through 2.5.1.1.19 identify 303(d)-listed waters in Oregon for toxins, temperature, dissolved oxygen, and pH.

A query by NMFS of the National Aquatic Water Quality Assessment (NAWQA) database (http://water.usgs.gov/nawqa/about.html) determined that all but three compounds listed in Table 1.1 were detected in one or more watersheds in Oregon (Figure 2.5.1.1.1).

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Compound	NAWQA Detection—Oregon
Aluminum	Yes
Ammonia	Yes
Arsenic	Yes
Lindane	Yes
Cadmium	Yes
Chromium III	Yes
Chromium VI	Yes
Copper	Yes
Dieldrin	Yes
Endosulfan-alpha	Yes
Endosulfan-beta	Yes
Endrin	No
Heptachlor Epoxide	No
Lead	Yes
Nickel	Yes
PCP	No
Selenium	Yes
Silver	Yes
TBT	Yes
Zinc	Yes

Figure 2.5.1.1.1 NAWQA database search results for compounds listed in Table 1.1.

ED_002991_00017299-00102

2.5.1.1 303(d)-Listed Waters in Oregon

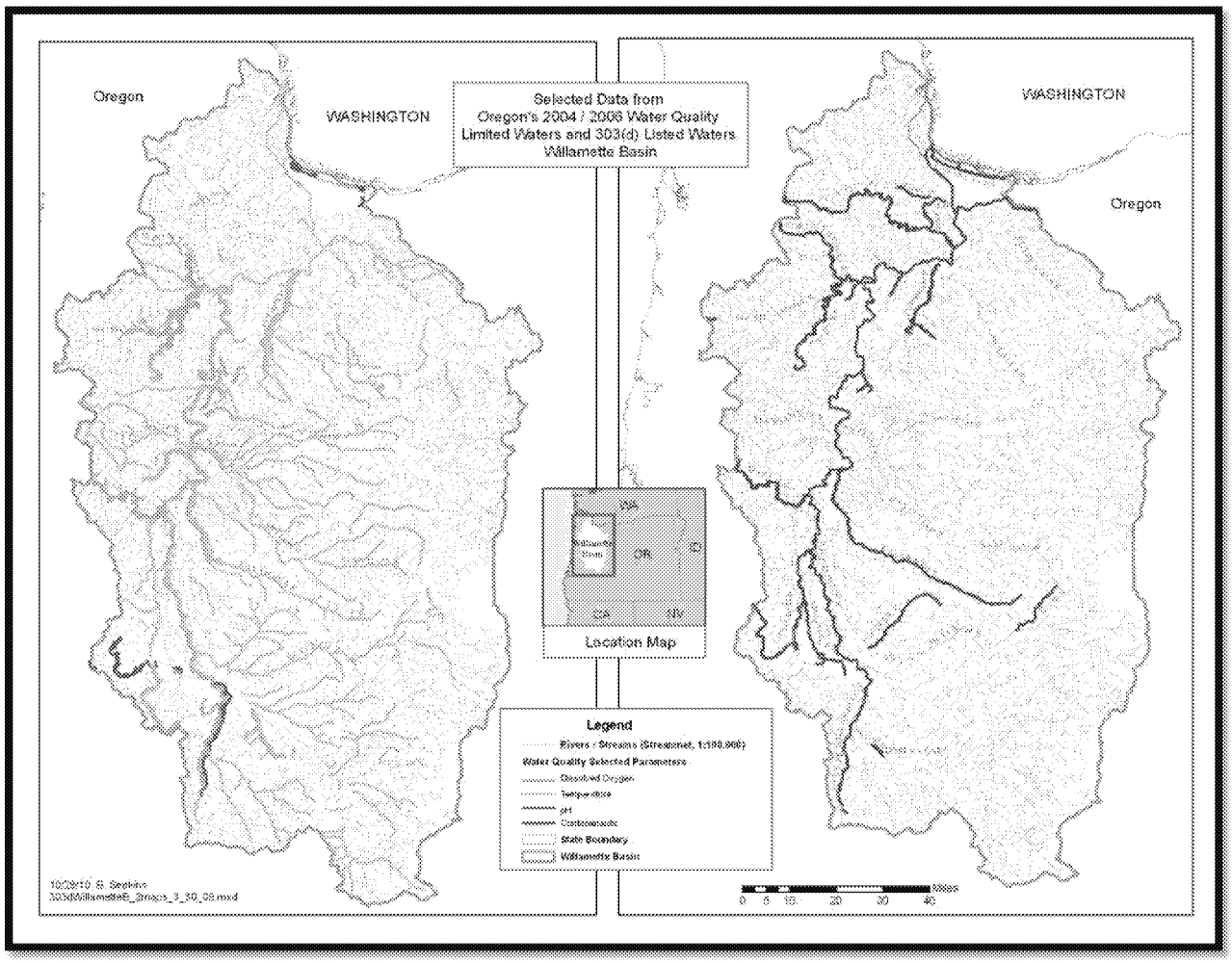

Figure 2.5.1.1.2 303(d) listed waters in the Willamette River Basin, Oregon for dissolved oxygen, pH, temperature, and non-specified toxins.

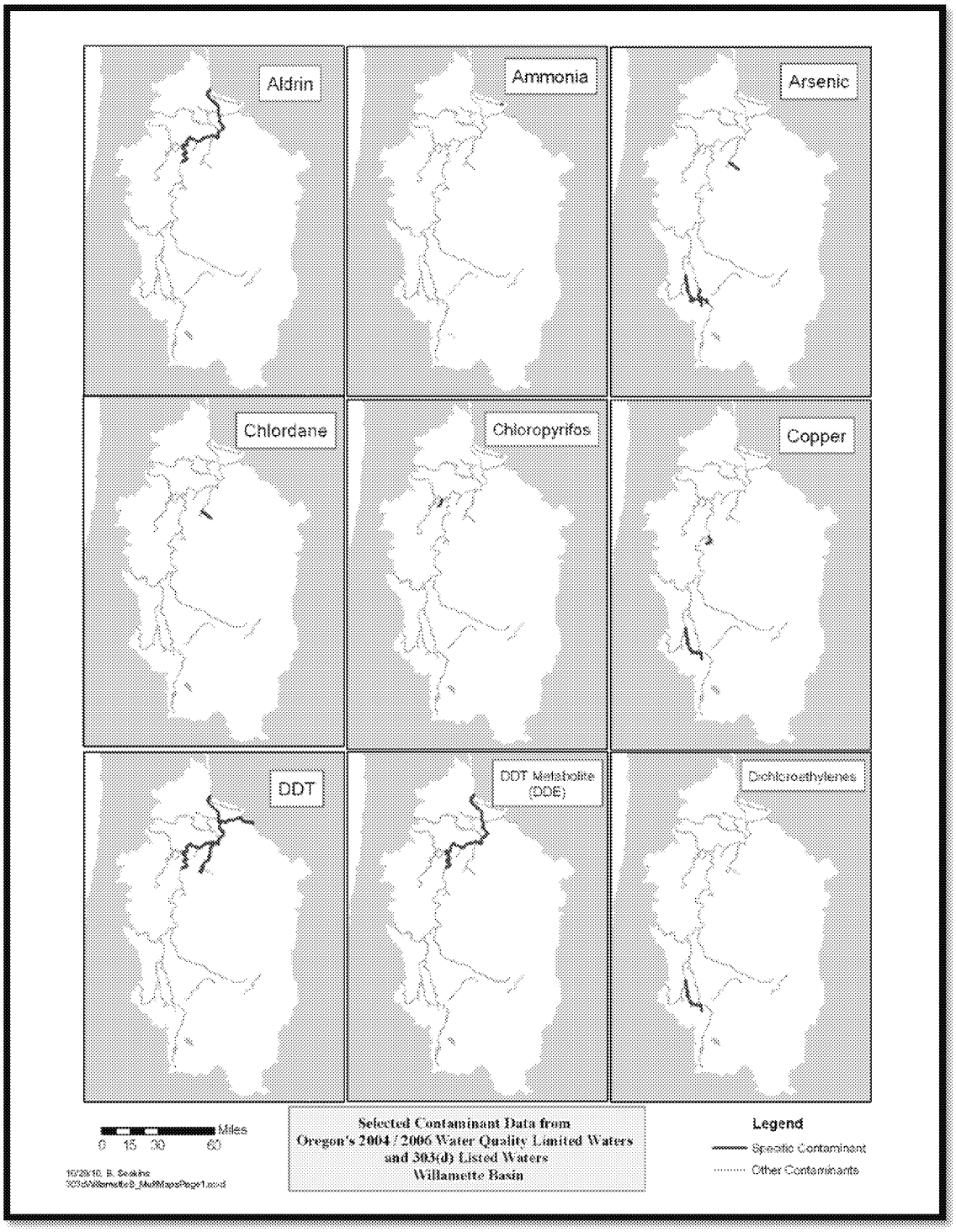

Figure 2.5.1.1.3 303(d) listed waters in the Willamette River Basin, Oregon for specified toxins.

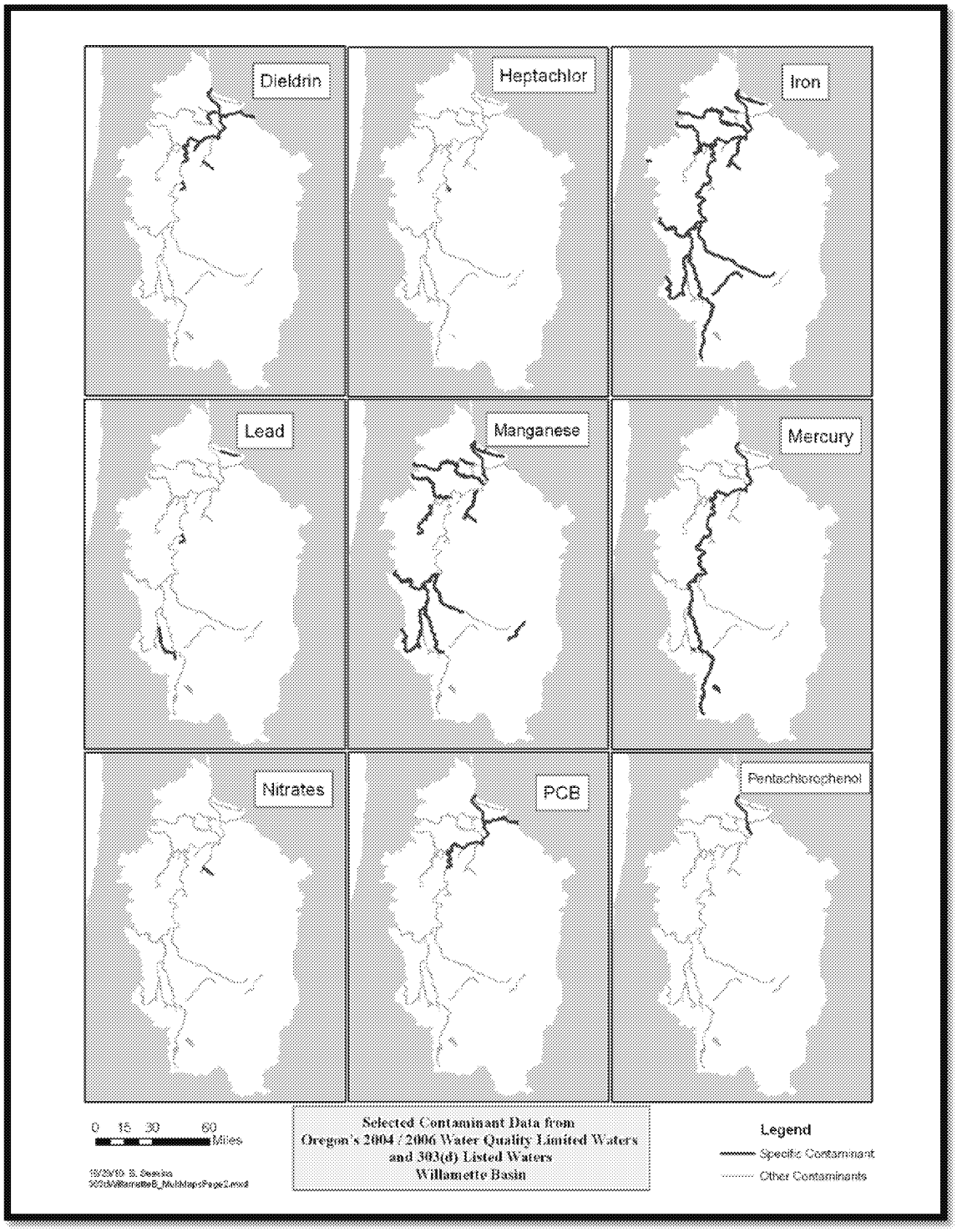

Figure 2.5.1.1.4 303(d) listed waters in the Willamette River Basin, Oregon for specified toxins.

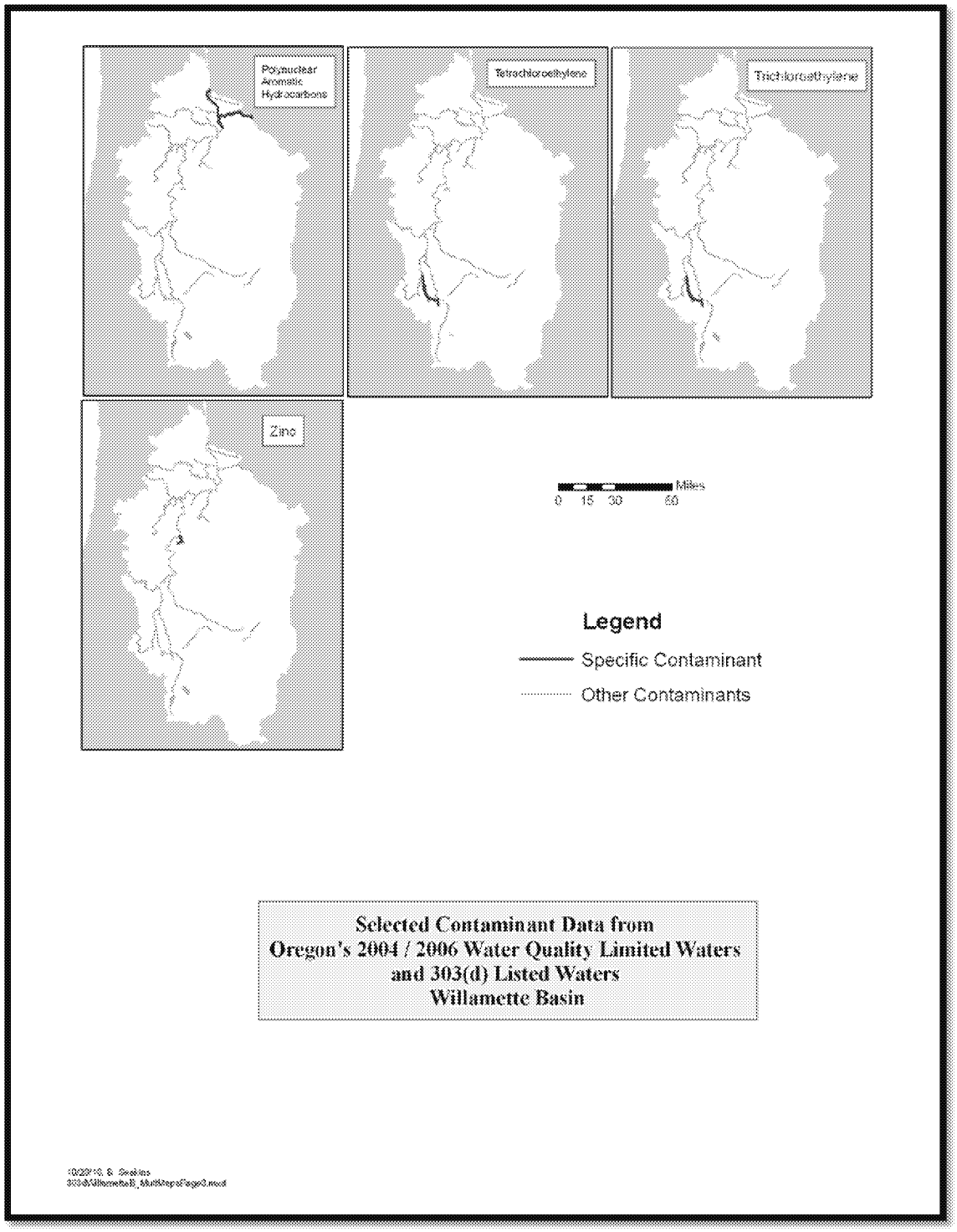

Figure 2.5.1.1.5 303(d) listed waters in the Willamette River Basin, Oregon for specified toxins.

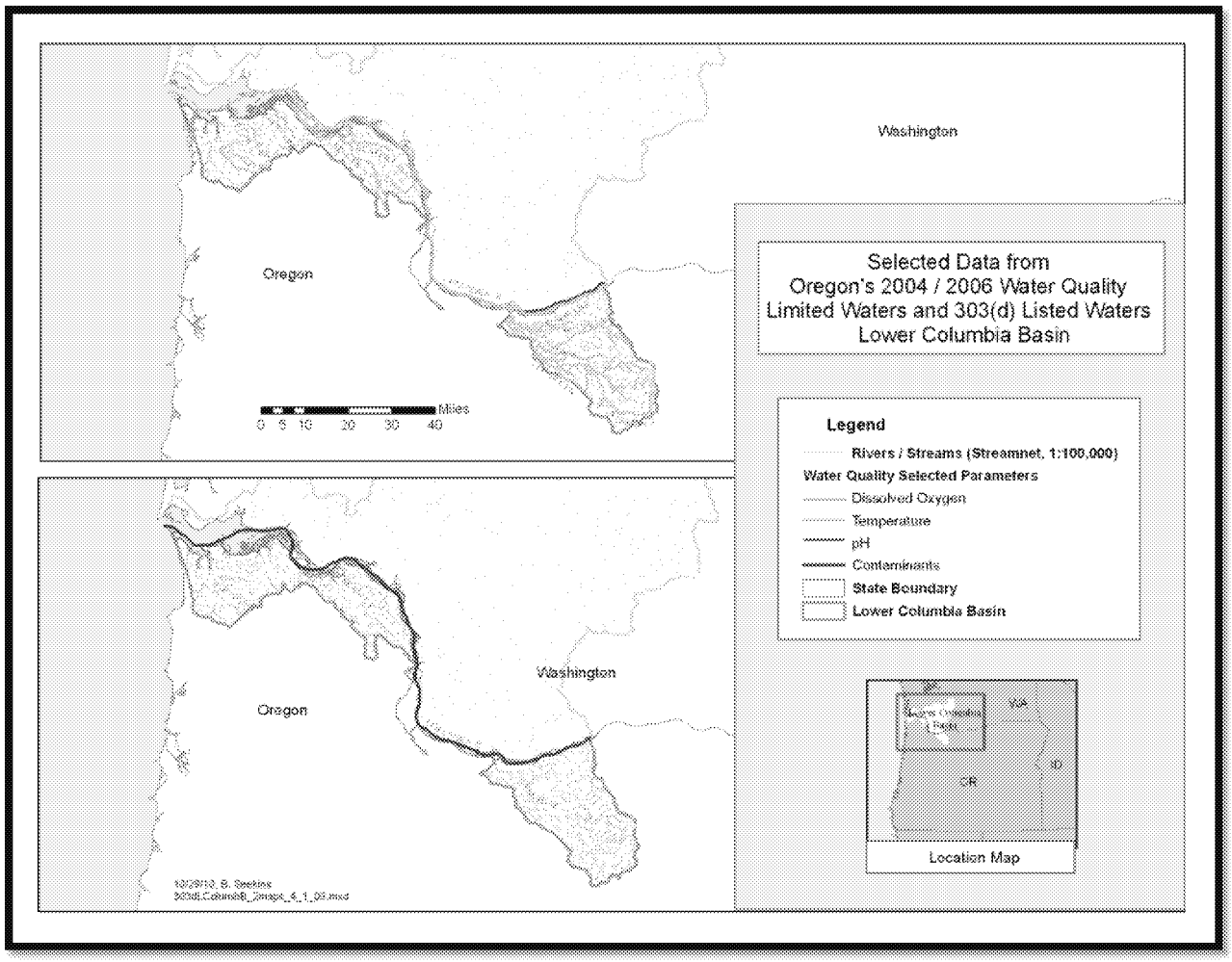

Figure 2.5.1.1.6 303(d) listed waters in the lower Columbia River and associated tributariy rivers in Oregon for dissolved oxygen, pH, temperature, and non-specified toxins.

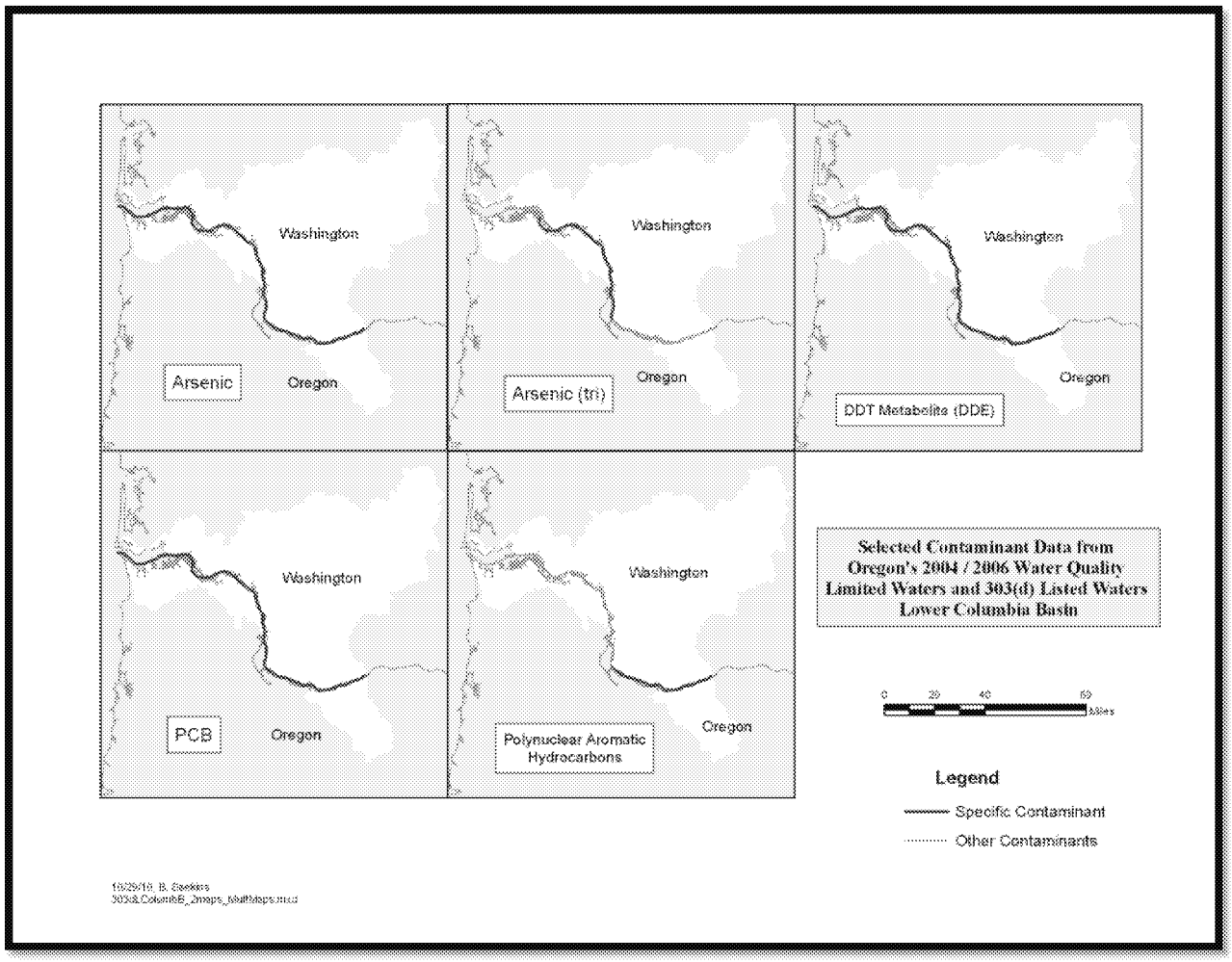

Figure 2.5.1.1.7 303(d) listed waters in the lower Columbia River in Oregon for specified toxins.

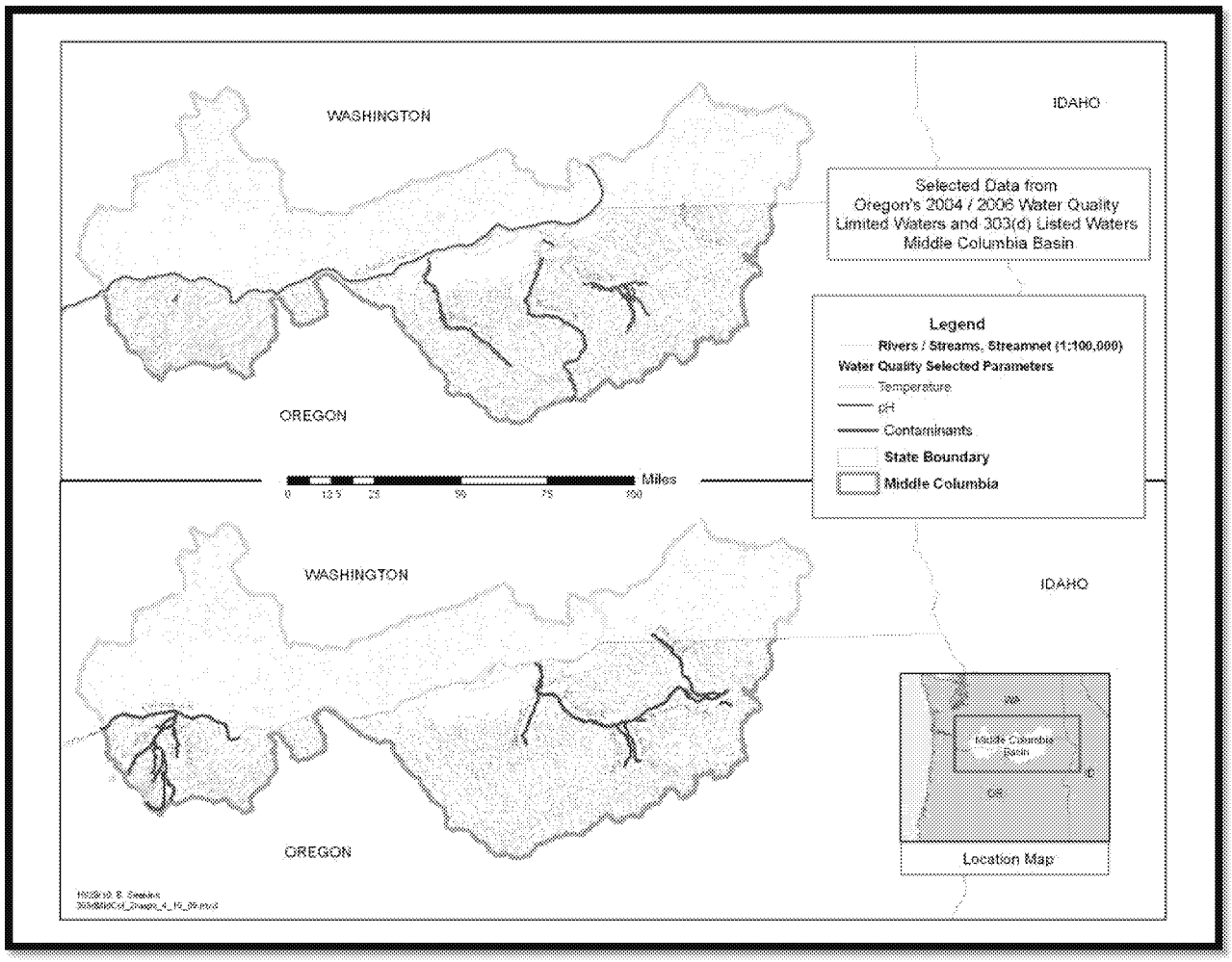

Figure 2.5.1.1.8 303(d) listed waters in the middle Columbia River and associated tributaries in Oregon for dissolved oxygen, pH, temperature, and non-specified toxins.

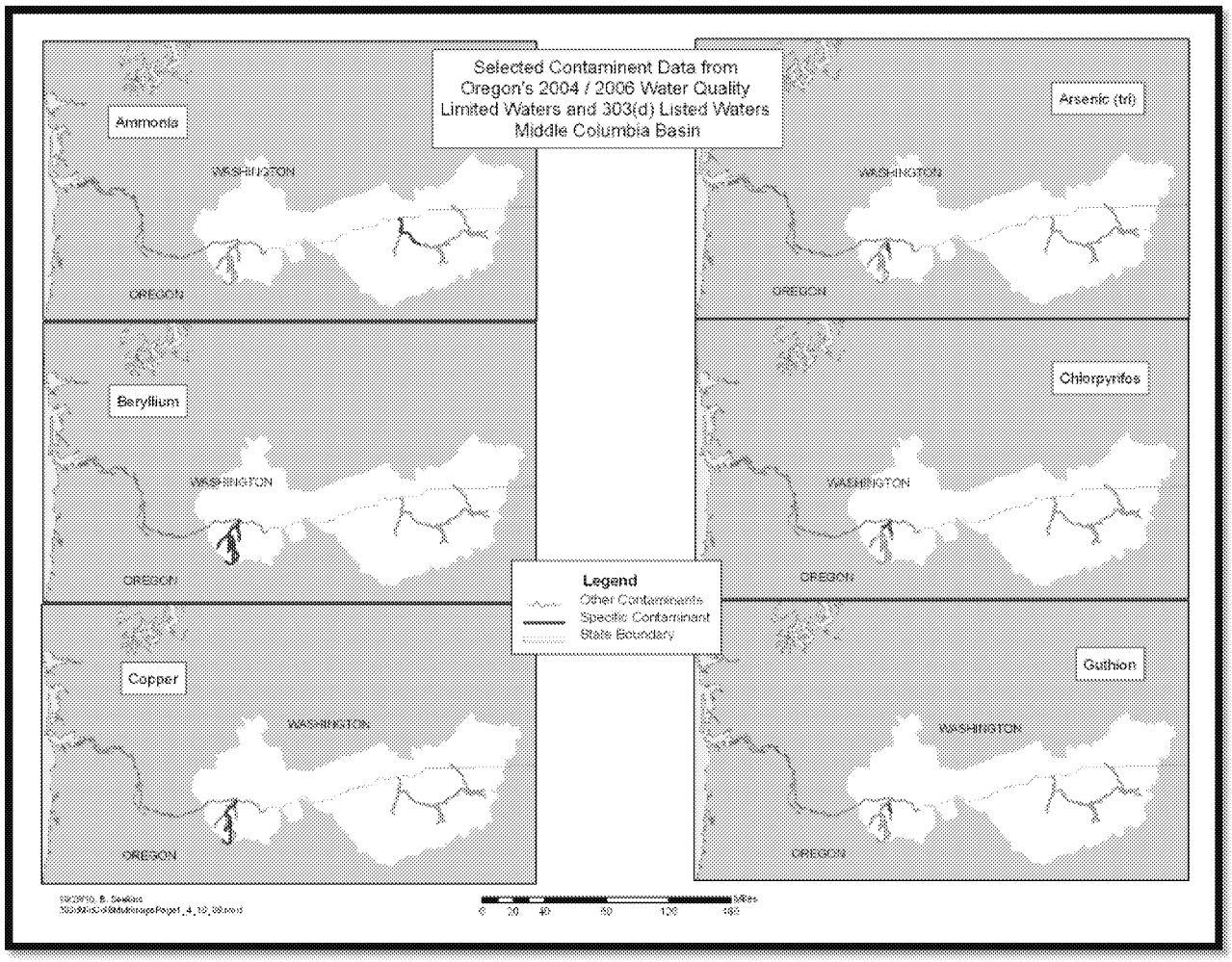

Figure 2.5.1.1.9 303(d) listed waters in the middle Columbia River and associated tributaries in Oregon for specified toxins.

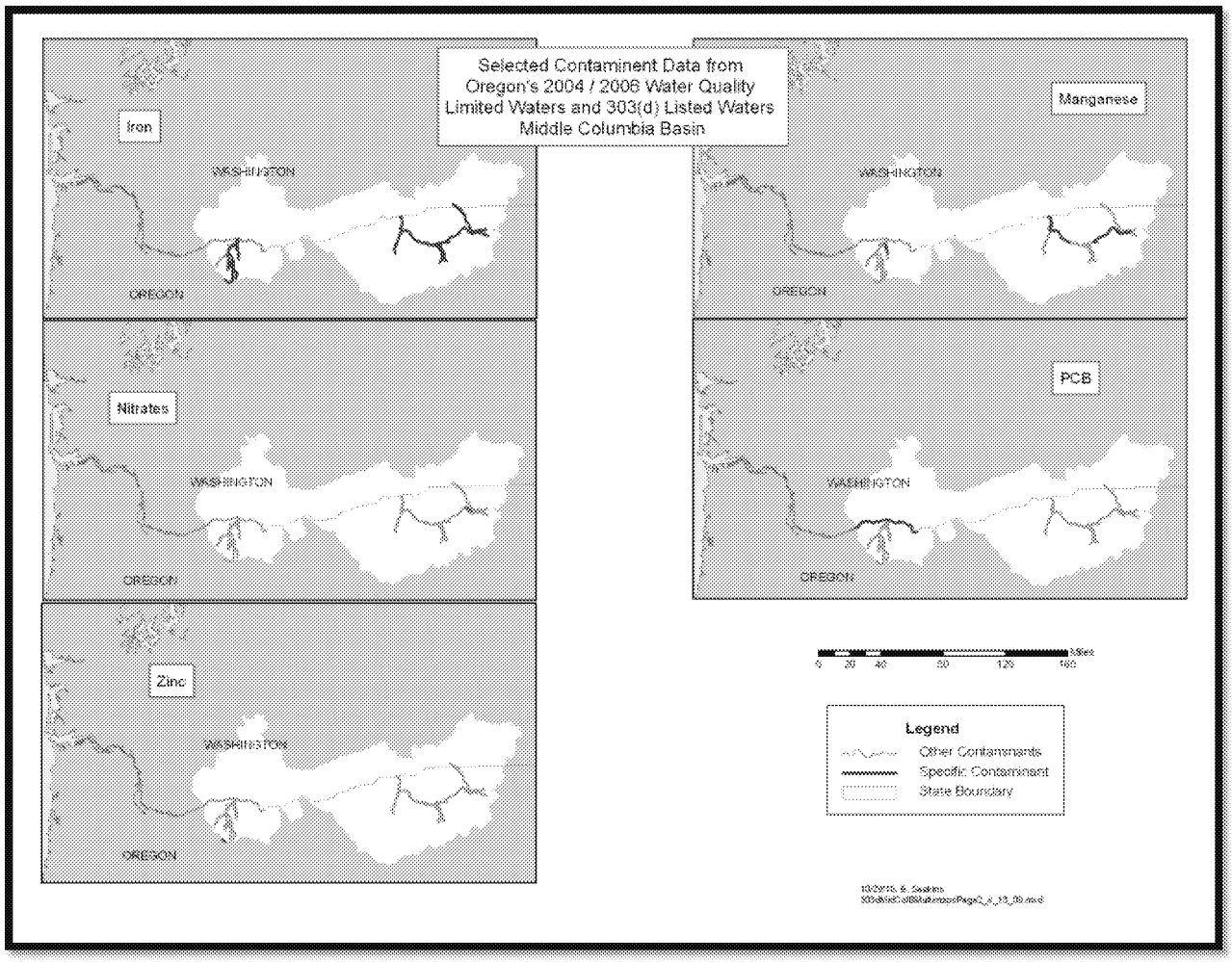

Figure 2.5.1.1.10 303(d) listed waters in the middle Columbia River and associated tributaries in Oregon for specified toxins.

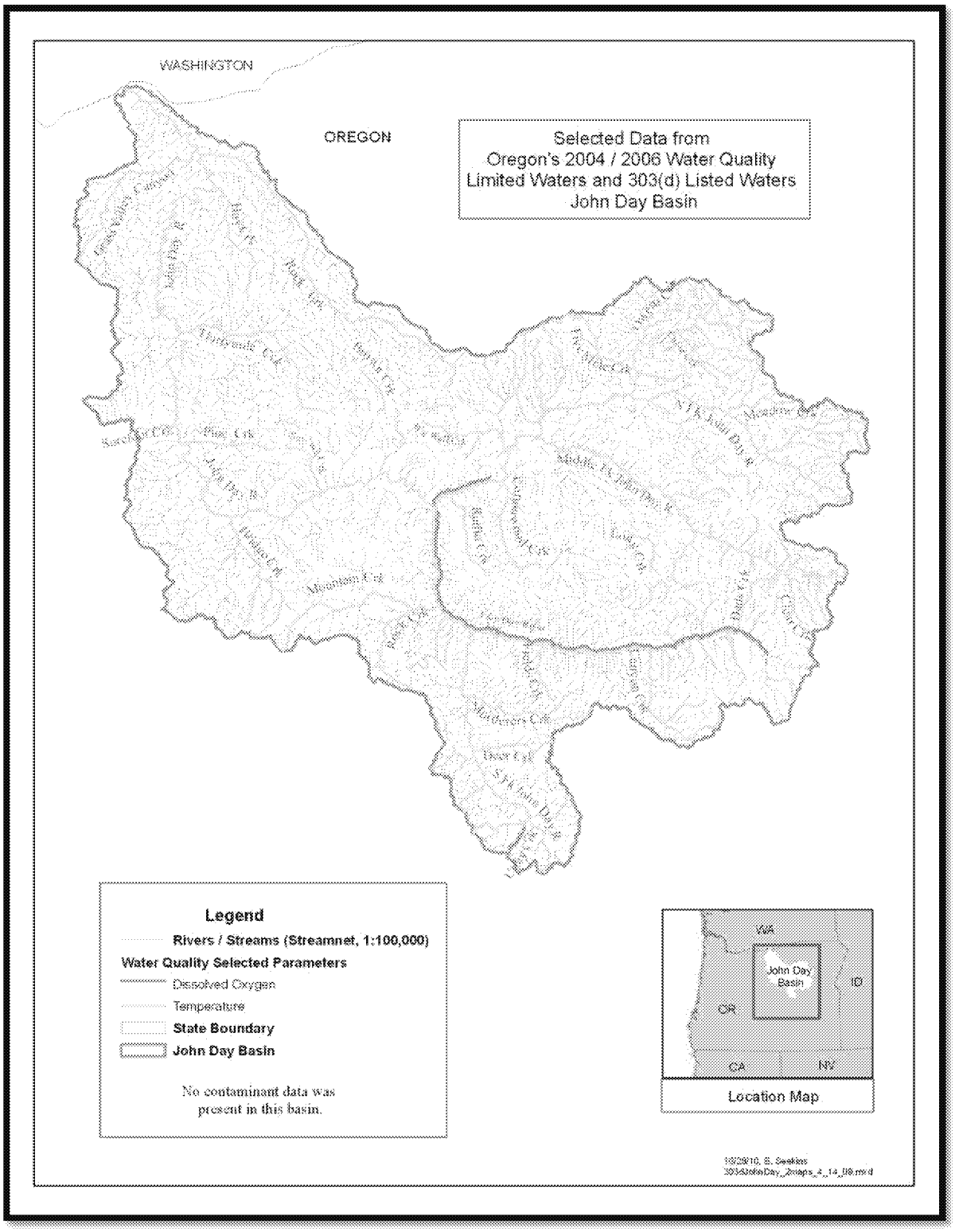

Figure 2.5.1.1.11. 303(d) listed waters in the John Day River Basin, Oregon for dissolved oxygen and temperature. No identified toxins.

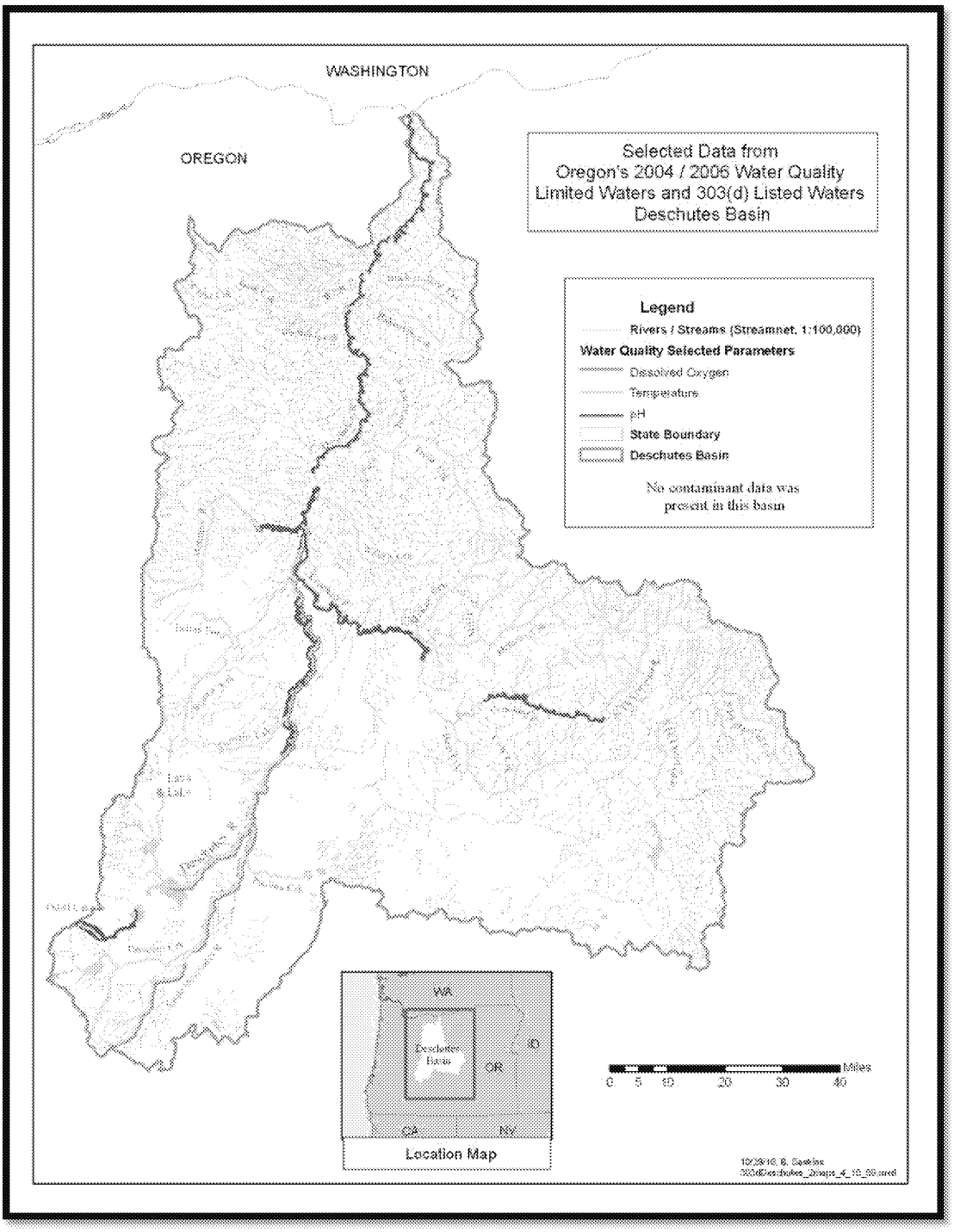

Figure 2.5.1.1.12 303(d) listed waters in the Deschutes River Basin, Oregon for dissolved oxygen, pH, and temperature. No identified toxins.

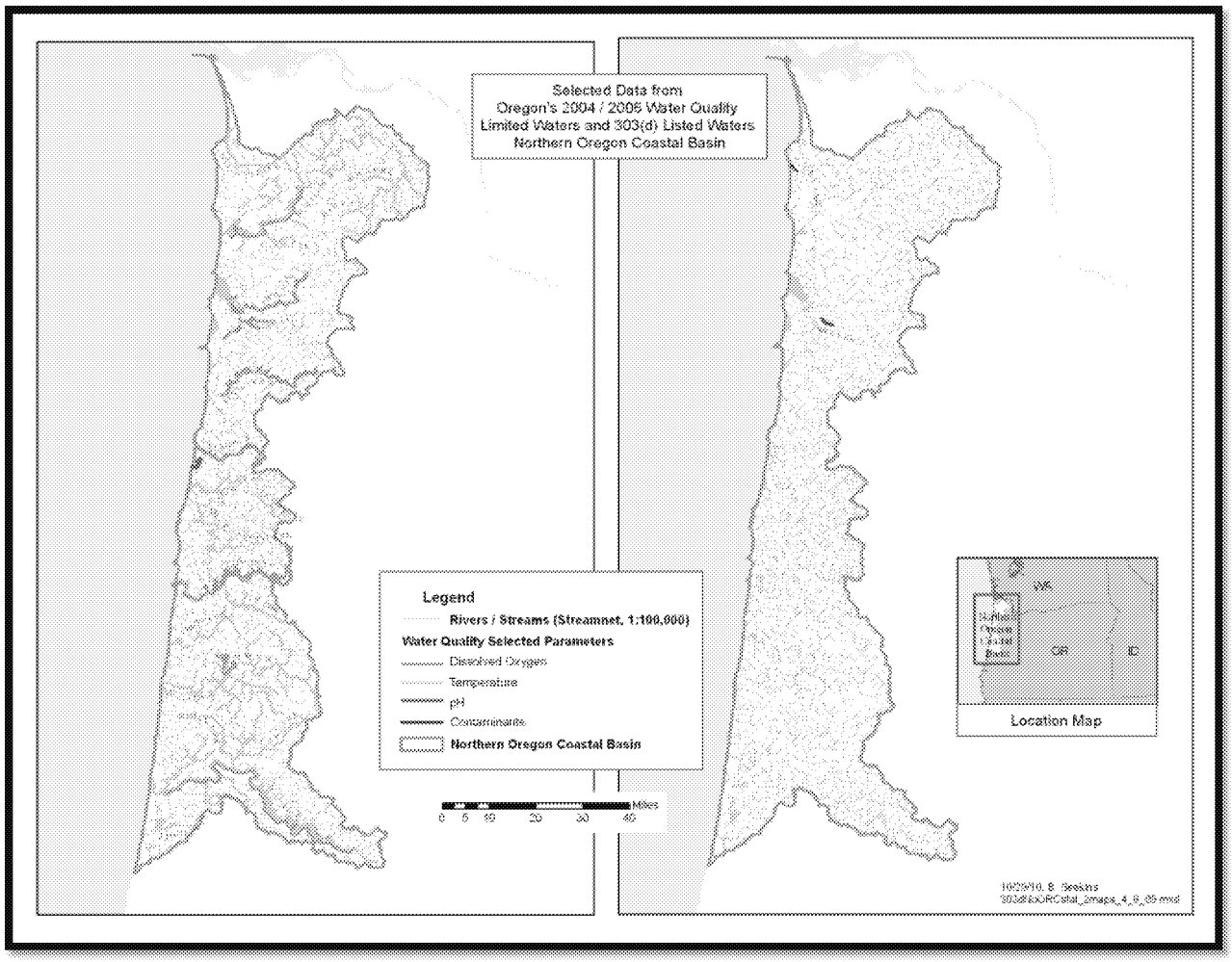

Figure 2.5.1.1.13 303(d) listed waters in the north coast river basins, Oregon for dissolved oxygen, temperature, and non-specified toxins.

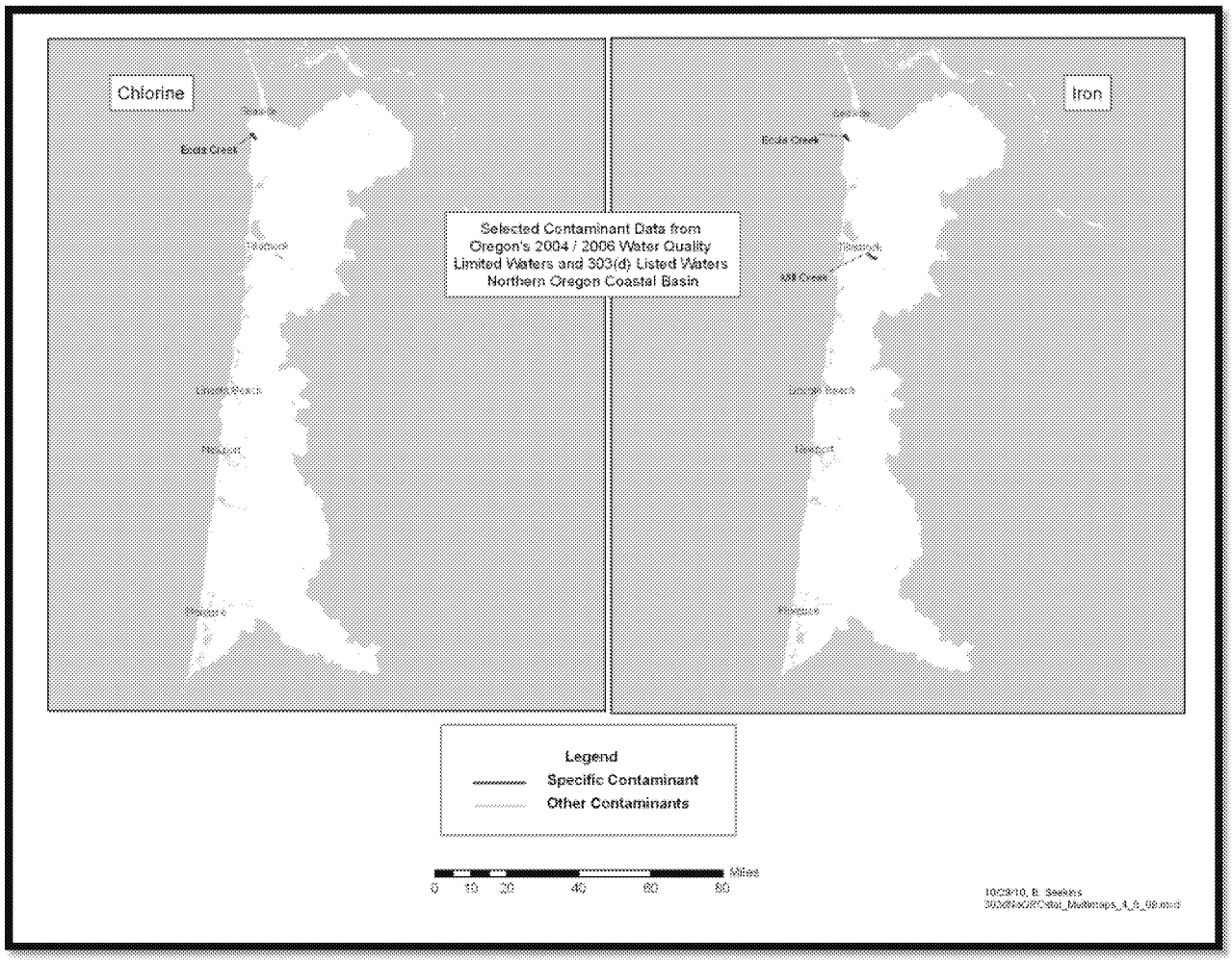

Figure 2.5.1.1.14 303(d) listed waters in the north coast river basins, Oregon for specified toxins.

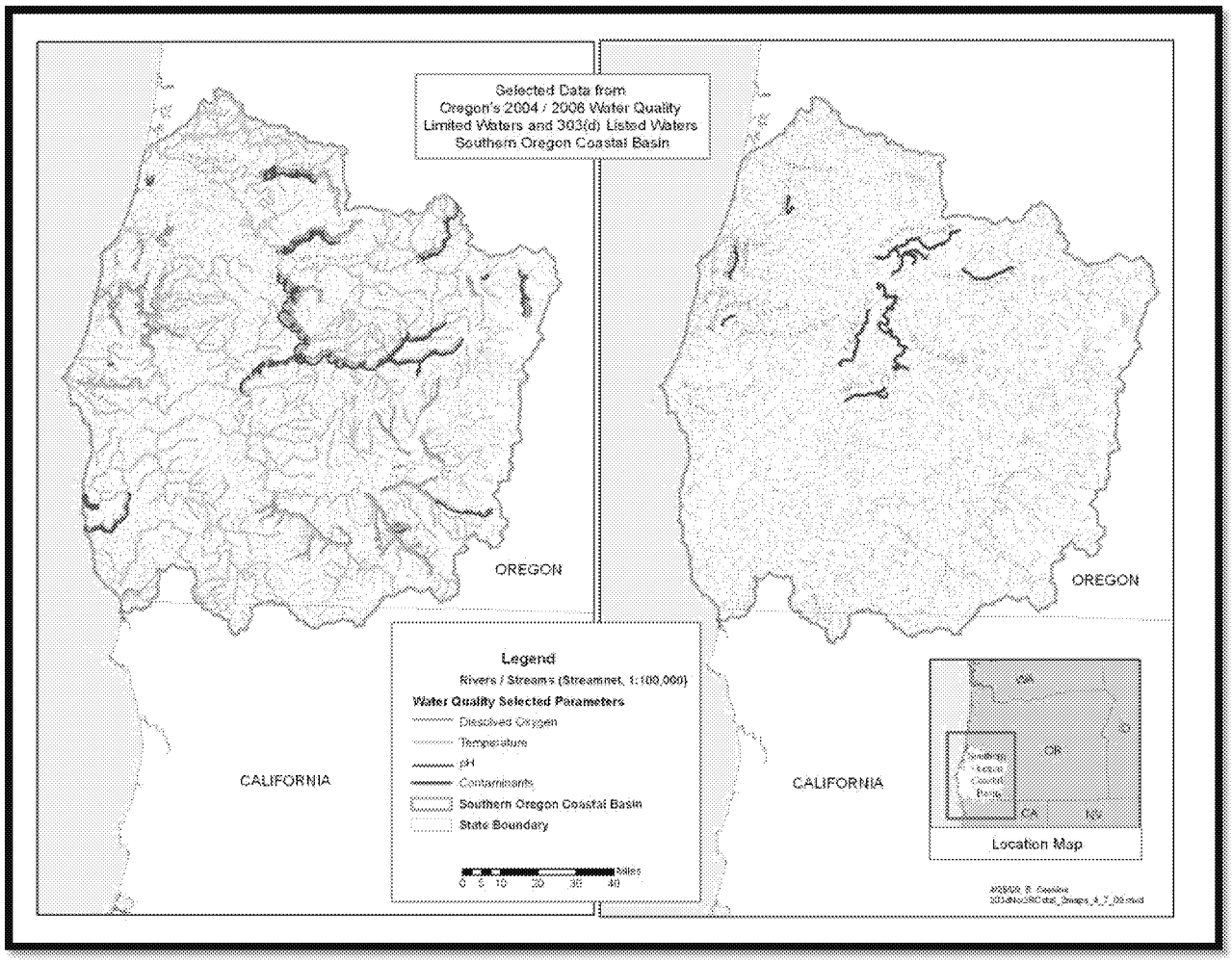

Figure 2.5.1.1.15 303(d) listed waters in the south coastal river basin, Oregon for dissolved oxygen, pH, and temperature, non-and specified toxins.

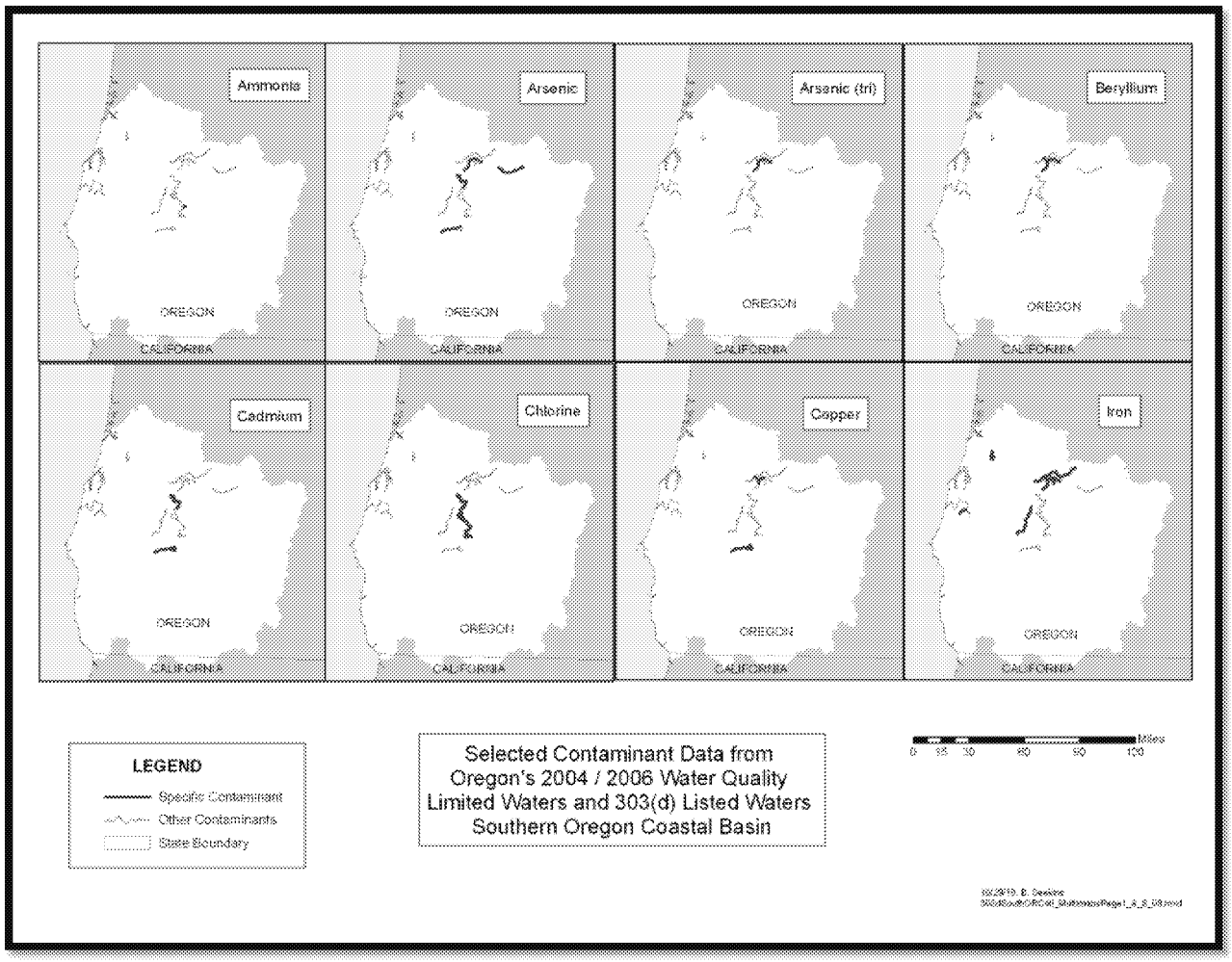

Figure 2.5.1.1.16 303(d) listed waters in the south coast river basins, Oregon specified toxins.

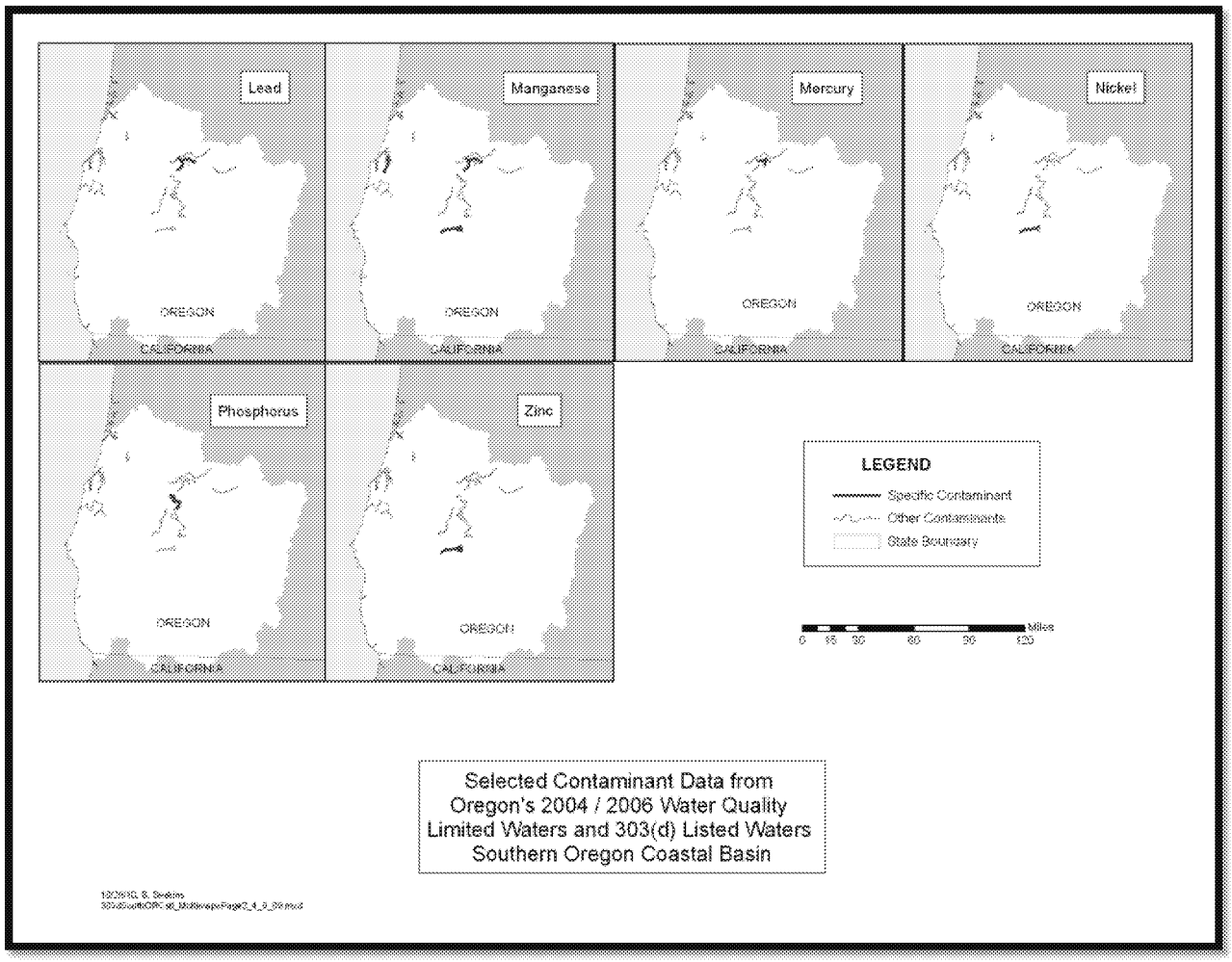

Figure 2.5.1.1.17 303(d) listed waters in the south coast river basins, Oregon for specified toxins.

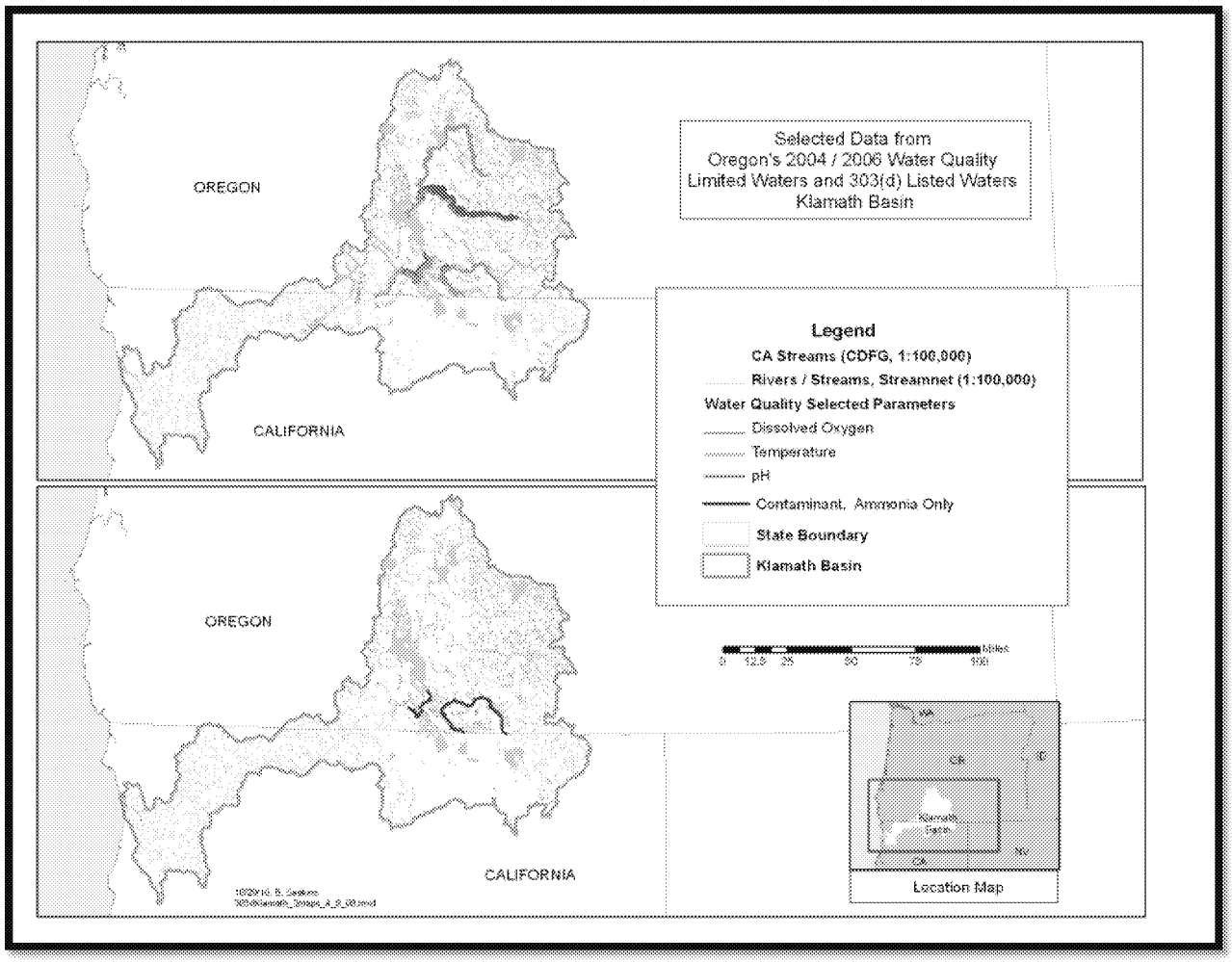

Figure 2.5.1.1.18 303(d) listed waters in the Klamath River Basin, Oregon for dissolved oxygen, pH, and temperature, and non-specified toxins.

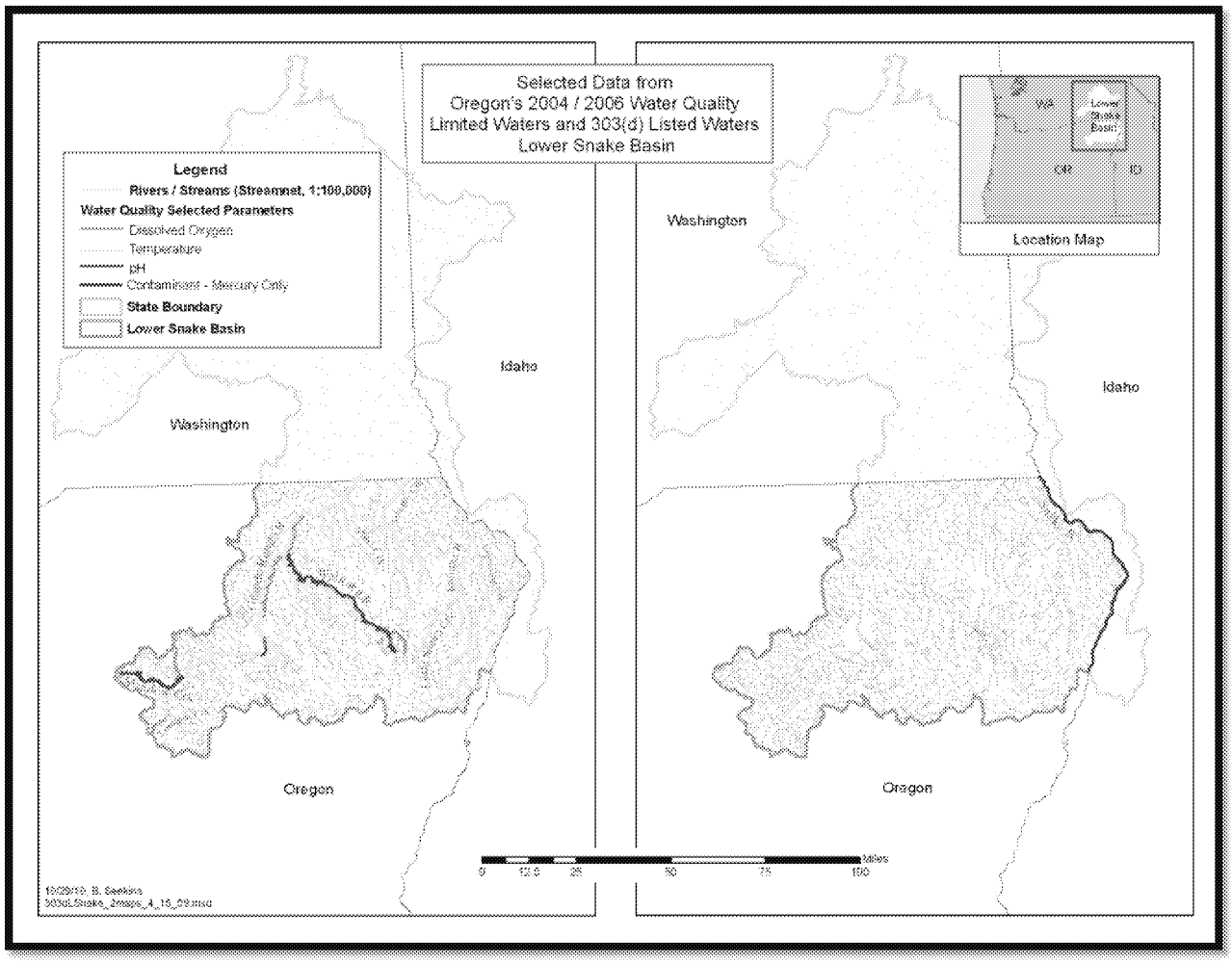

Figure 2.5.1.1.19 303(d) listed waters in the lower Snake River Basin, Oregon for dissolved oxygen, pH, and temperature, and specified toxins.

2.5.2. MS4 and NPDES Permits, Species Distribution, and Exposure Risk Potential

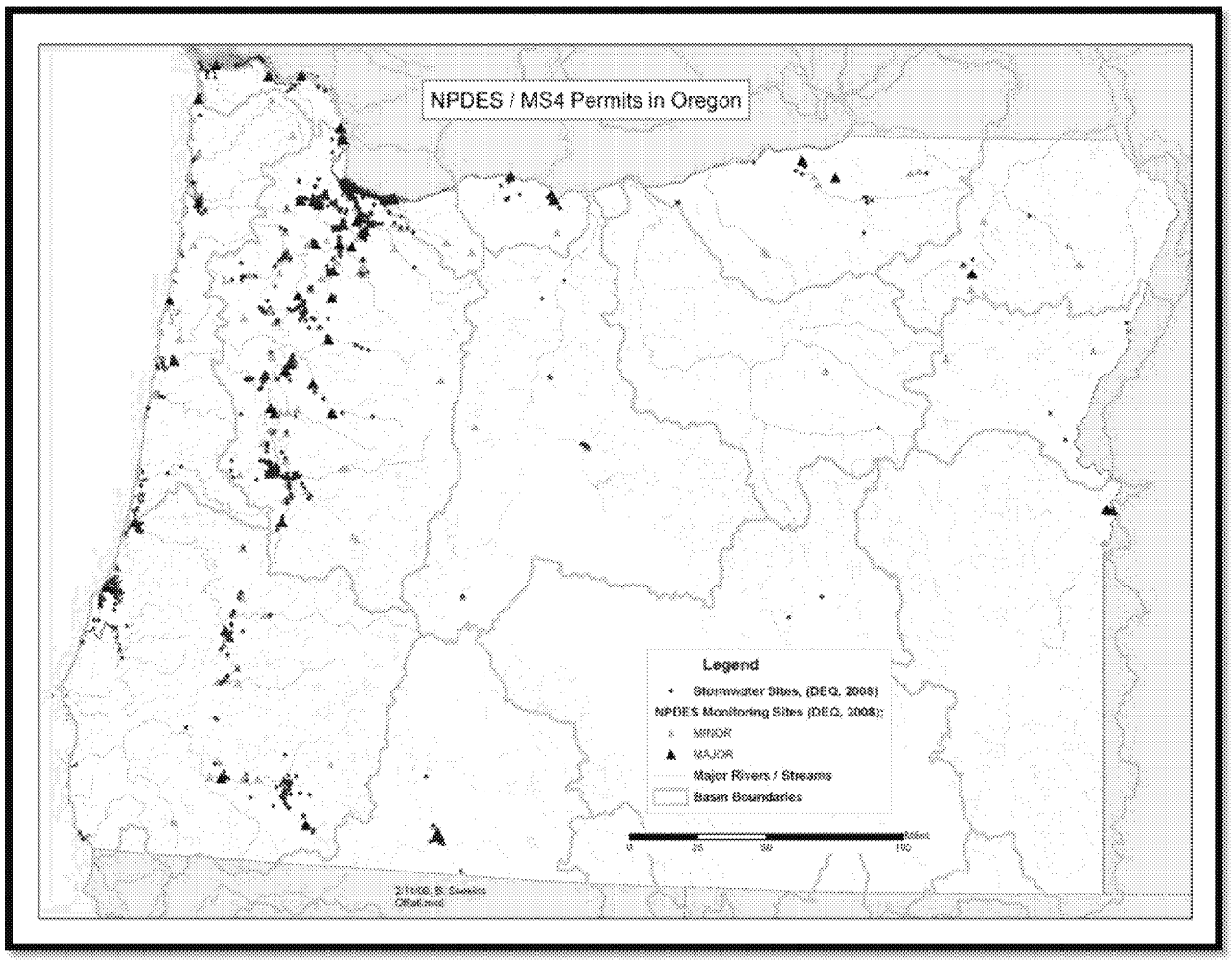

Figure 2.5.2.1 **Overview of the spatial distribution and intensity of point-source discharges in Oregon (MS4 and NPDES permits).**

Table 2.5.2.1.1 and Table 2.5.2.2.2 provide permit-specific information on pollutants for each class of stormwater (MS4) and NPDES permit (*i.e.*, industrial, domestic), where available. For MS4 permits, permit-specific parameters are listed where information was available. For unspecified MS4 permits, NMFS reviewed 91 MS4 permits with specific parameters and identified stormwater parameters common to all reviewed permits, and used this information as a surrogate for the unspecified MS4 permits. Industrial and domestic NPDES permits are categorized as either major (discharge greater than 1 million gallons per day) or minor (discharge less than 1 million gallons per day).

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Compounds that are discharged under existing MS4 and/or NPDES permits in Oregon that are listed in Table 1.1:

- Aluminum
- Ammonia
- Arsenic
- Cadmium
- Chromium (III)
- Chromium (VI)
- Copper
- Lead
- Nickel
- Pentachlorophenol
- Selenium
- Silver
- Tributyltin
- Zinc

Compounds listed in Table 1.1 that are associated with 303(d)-listed waters in Oregon:

- Ammonia
- Arsenic
- Cadmium
- Copper
- Dieldrin
- Heptachlor epoxide
- Lead
- Nickel
- Zinc

2.5.2.1 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution.

For SR sockeye salmon, UCR spring Chinook salmon, and UCR steelhead, the ESU/DPS boundaries are outside of the action area, and there are no NPDES or MS4 permits that occur in the action area that overlap with the ESU/DPS boundaries for these species. Therefore, MS4 and NPDES permit, and fish distribution data for these species are not reported in this section. However, smolts and adults will be exposed to stressors of the action as fish pass through the Columbia River, RM zero to RM 297, and in the Pacific Ocean from the mouth of the Columbia River to nautical mile 3.

Table 2.5.2.1.1 through Table 2.5.2.2.4 identify the ESU/DPS, number of populations in Oregon, the number of populations in Oregon without direct exposure to MS4 and/or NPDES point sources, the number of MS4 and/or NPDES point source discharges, and the compounds

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associated with each permit type. Figure 2.5.2.1.1 through Figure 2.5.2.1.17 identify the approximate location of each MS4 and/or NPDES permits in each watershed, fish habitat distribution, fish habitat use, and population.

Table 2.5.2.1.1 SR fall-run Chinook Salmon populations in Oregon. Three of eight spawning populations occur in Oregon.

ESU/DPS	Populations in Oregon
SR fall-run Chinook	Snake River—Major Population Group Grande Ronde Snake River Imnaha

Table 2.5.2.1.2 Type, number, and chemicals discharged for MS4 and NPDES permits within the SR fall-run Chinook salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	None	
NPDES	None	

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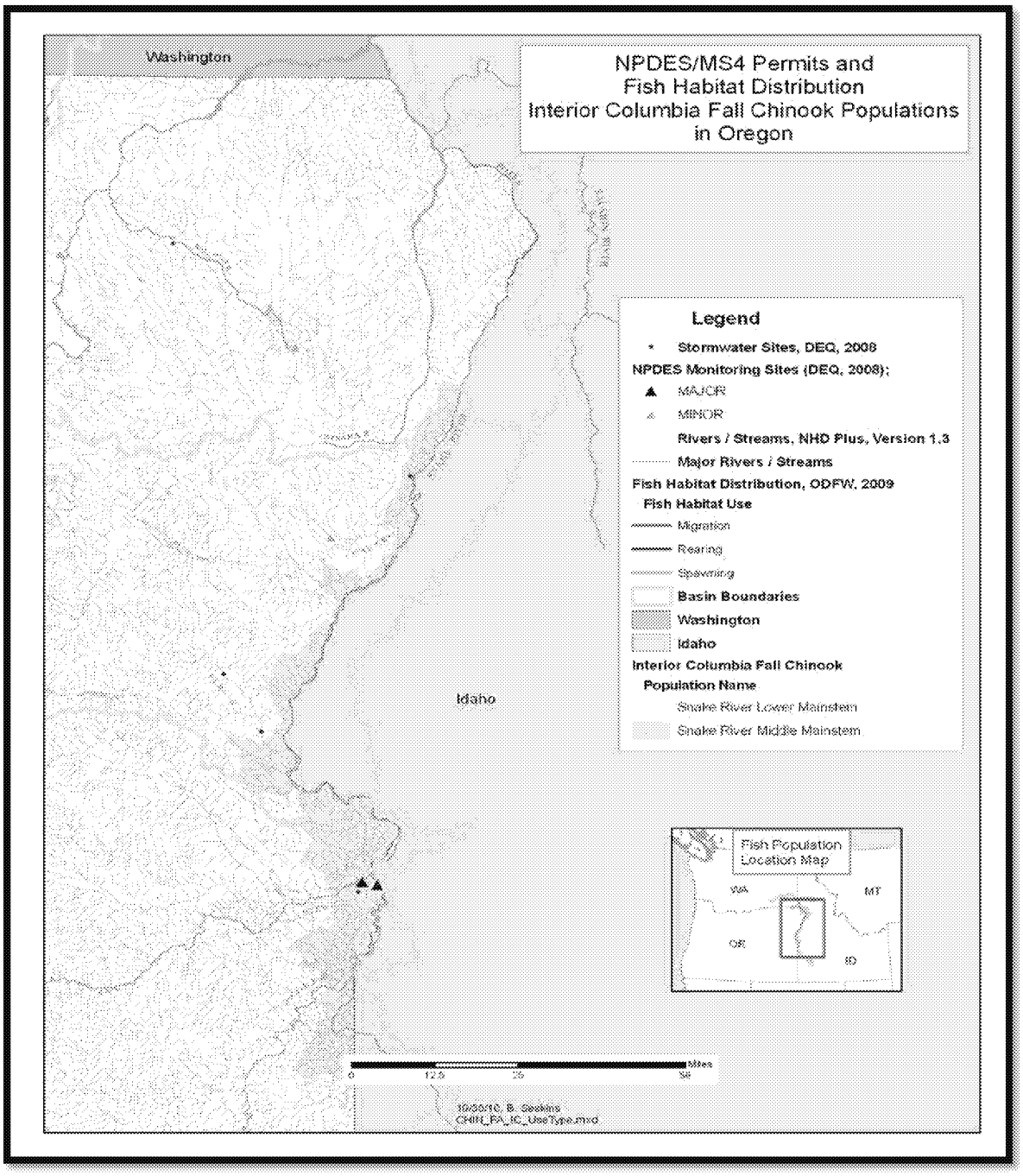

Figure 2.5.2.1.1 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for SR fall-run Chinook salmon.

Table 2.5.2.1.3 SRB steelhead populations in Oregon. Five of 24 populations occur in Oregon.

ESU/DPS	Populations in Oregon
SRB Steelhead	Wallowa River
	Grande Ronde River Upper Mainstem
	Imnaha River
	Joseph Creek
	Grande Ronde River Lower Mainstem

Table 2.5.2.1.4 Type, number, and chemicals discharged for MS4 and NPDES permits within the SRB steelhead DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	2	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	5	Ammonia, Zinc, Lead, Copper

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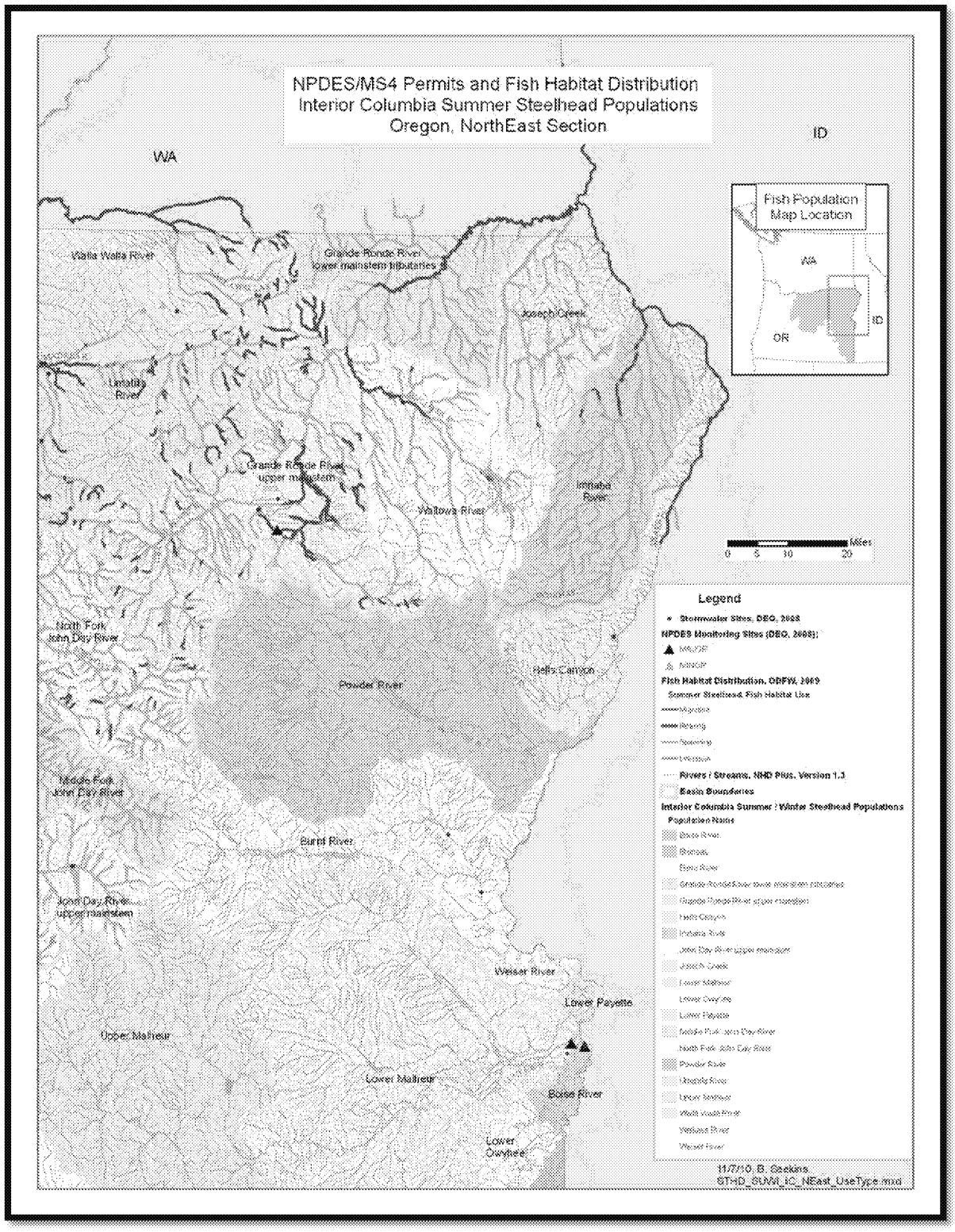

Figure 2.5.2.1.2 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for SRB steelhead.

Table 2.5.2.1.5 SR spring/summer Chinook salmon populations in Oregon. Eight of 27 populations occur in Oregon.

ESU/DPS	Populations In Oregon
SR Spring/Summer-Run Chinook	Grande Ronde UM
	Catherine Creek
	Lostine River
	Imnaha River
	Big Sheep Creek
	Minam River
	Looking Glass Creek
	Wenaha River

Table 2.5.2.1.6 Type, number, and chemicals discharged for MS4 and NPDES permits within the SR spring/summer Chinook salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	2	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	5	Ammonia, Zinc, Lead, Copper

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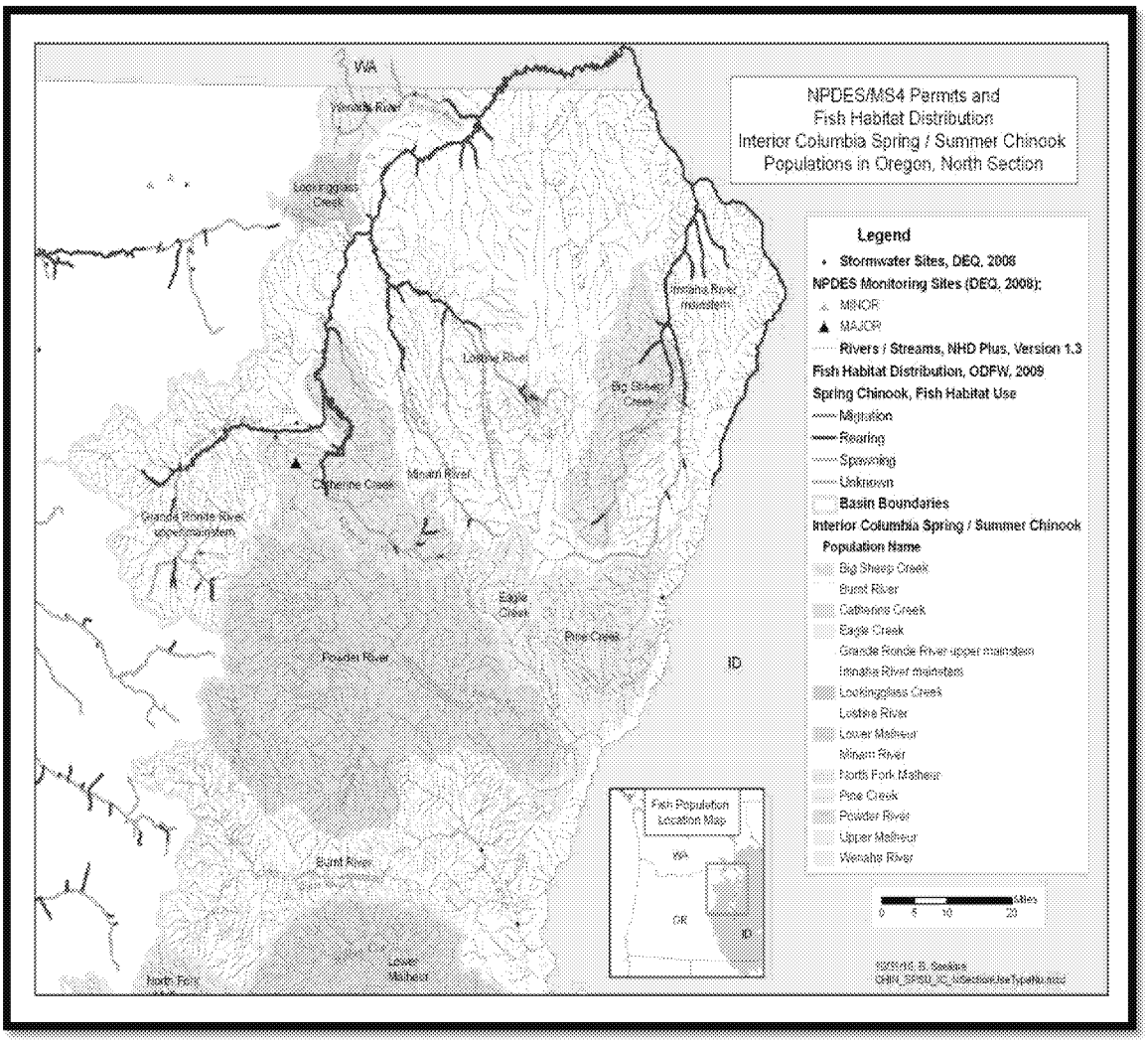

Figure 2.5.2.1.3 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for SR spring/summer-run Chinook salmon.

Table 2.5.2.1.7 MCR steelhead populations in Oregon. Ten of 17 populations occur in Oregon.

ESU/DPS	Populations In Oregon
MCR Steelhead	Walla Walla
	Umatilla River
	John Day Lower Mainstem
	John Day North Fork
	John Day Middle Fork
	John Day Upper Mainstem
	John Day South Fork
	Deschutes Westside
	Deschutes Eastside
	Fifteen Mile Creek

Table 2.5.2.1.8 Type, number, and chemicals discharged for MS4 and NPDES permits within the MCR steelhead DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	21	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	11	Ammonia, Lead, Copper, Zinc

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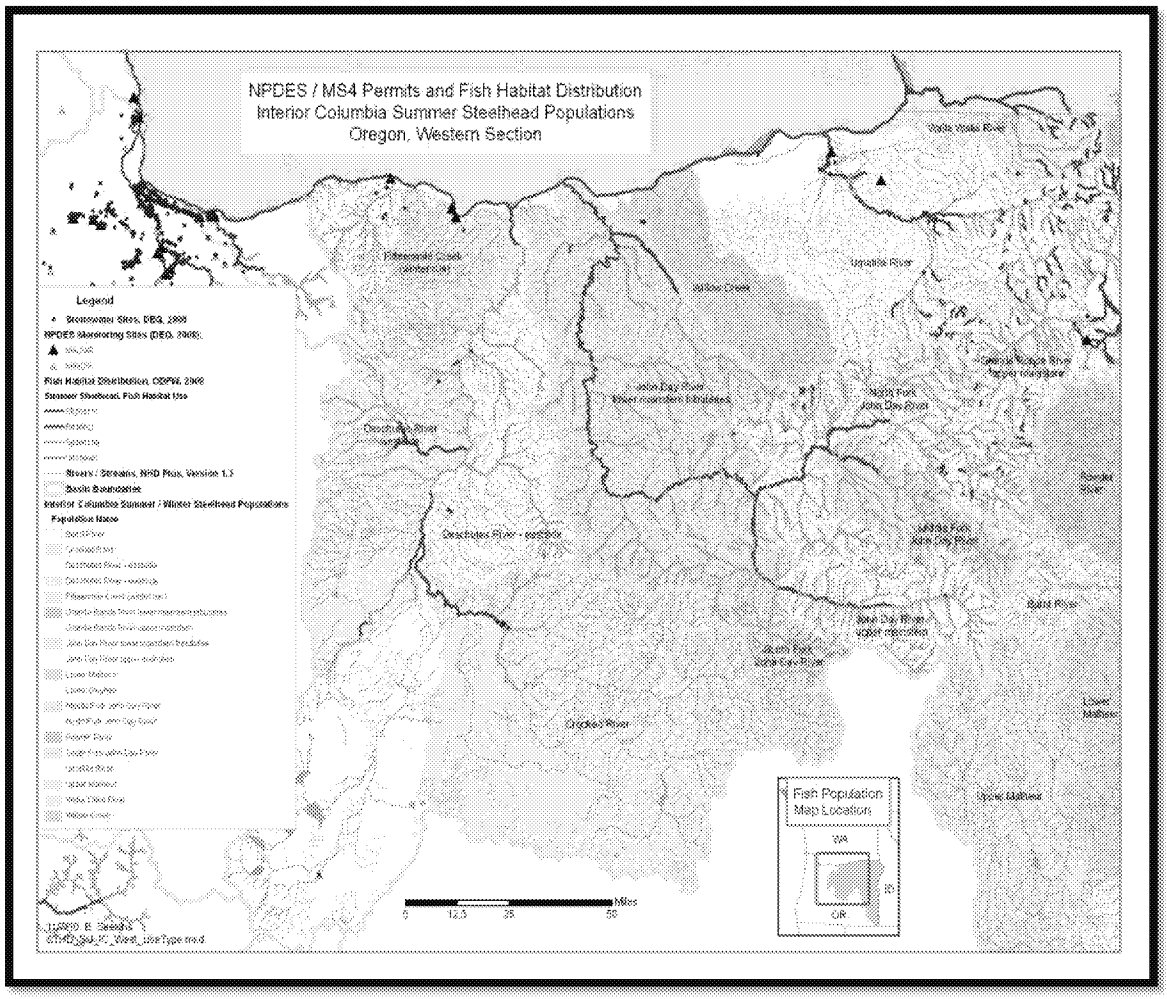

Figure 2.5.2.1.4 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for MCR steelhead.

Table 2.5.2.1.9 LCR Chinook salmon populations in Oregon. Nine of 32 populations occur in Oregon.

ESU/DPS	Populations In Oregon
LCR Chinook Salmon	Hood River (F+S)
	Sandy River (F+S)
	Lower Gorge Tributaries
	Clackamas
	Upper Gorge Tributaries
	Scappoose
	Clatskanine
	Big Creek
	Youngs Bay

Table 2.5.2.1.10 Type, number, and chemicals discharged for MS4 and NPDES permits within the LCR Chinook salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	654	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	48	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

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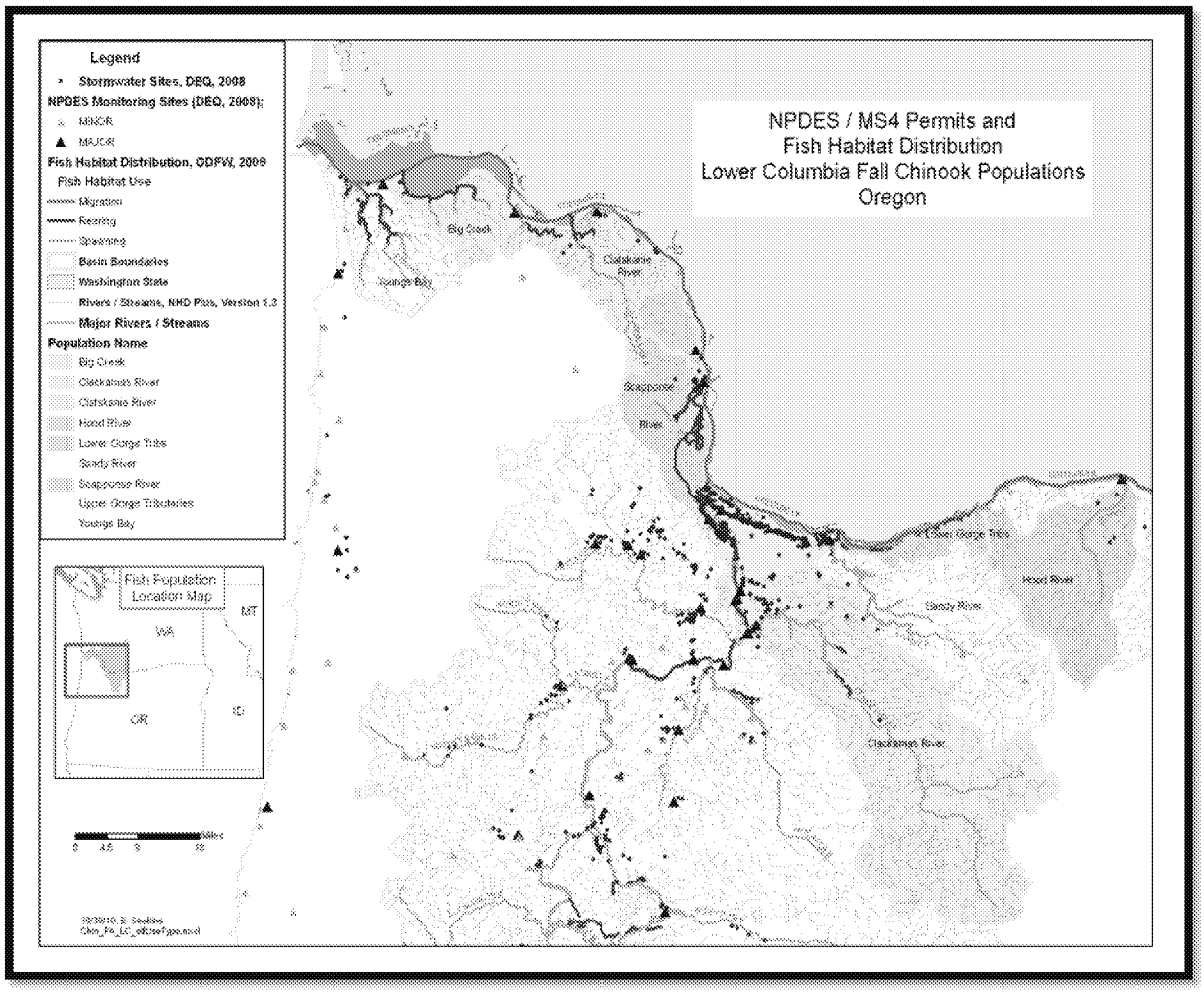

Figure 2.5.2.1.5 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR Chinook salmon.

Table 2.5.2.1.11 CR chum salmon populations in Oregon. One of 17 populations occurs in Oregon (14 of 17 chum populations remain extirpated or nearly so).

ESU/DPS	Populations In Oregon
CR Chum Salmon	Lower Gorge Tributaries/Mainstem
	Big Creek
	Clackamas
	Clatskanine
	Sandy
	Scappose
	Upper Gorge Tributaries
	Youngs Bay

Table 2.5.2.1.12 Type, number, and chemicals discharged for MS4 and NPDES permits within the CR chum salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	654	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	48	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

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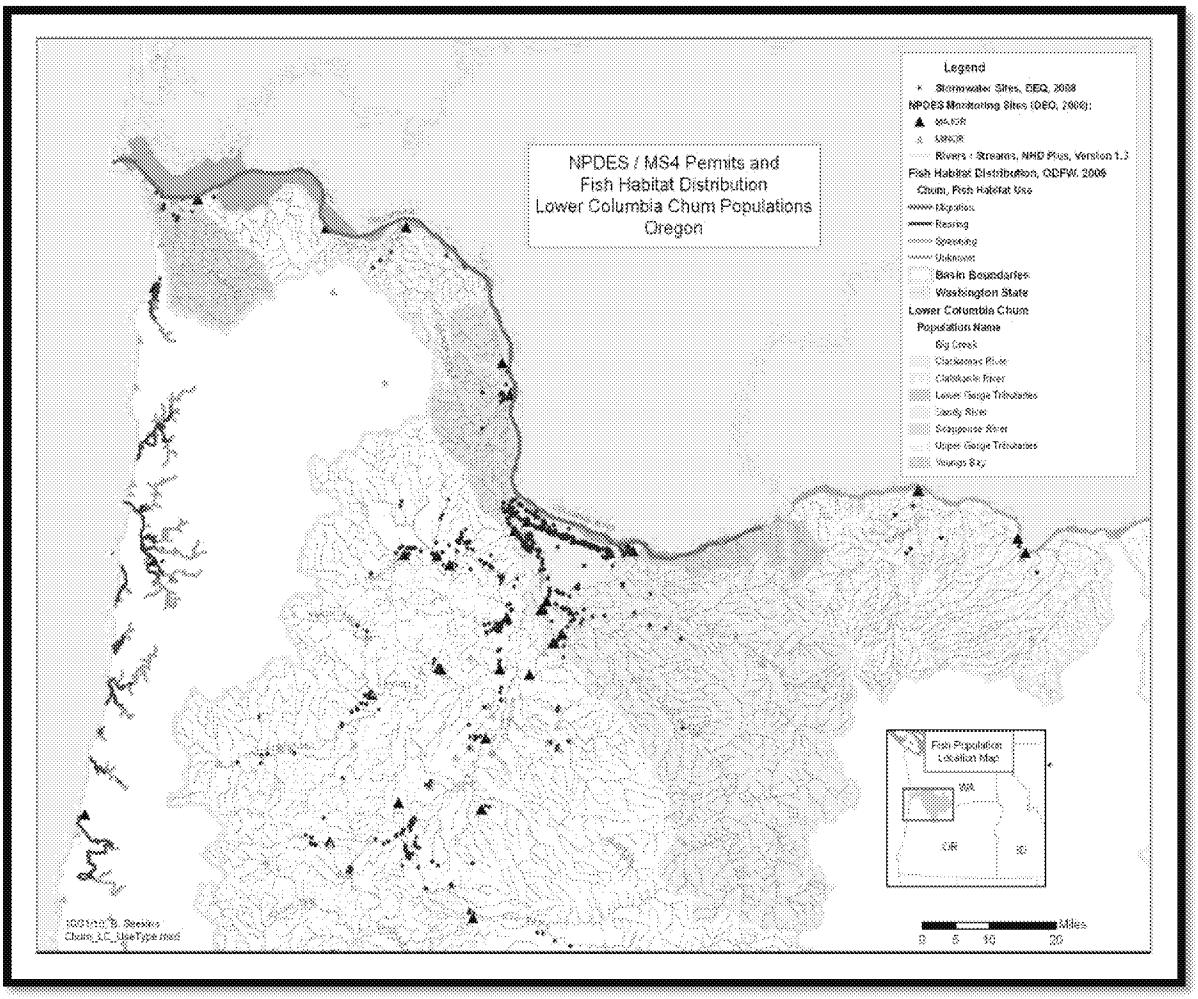

Figure 2.5.2.1.6 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for CR chum salmon.

Table 2.5.2.1.13 LCR coho salmon populations in Oregon. Eight of 27 populations occur in Oregon.

ESU/DPS	Populations In Oregon
LCR Coho Salmon	Big Creek
	Clackamas
	Clatskanie
	Lower Gorge Tributaries
	Upper Gorge and Hood River
	Sandy
	Scappose
	Youngs Bay

Table 2.5.2.1.14 Type, number, and chemicals discharged for MS4 and NPDES permits within the LCR coho salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	654	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	48	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

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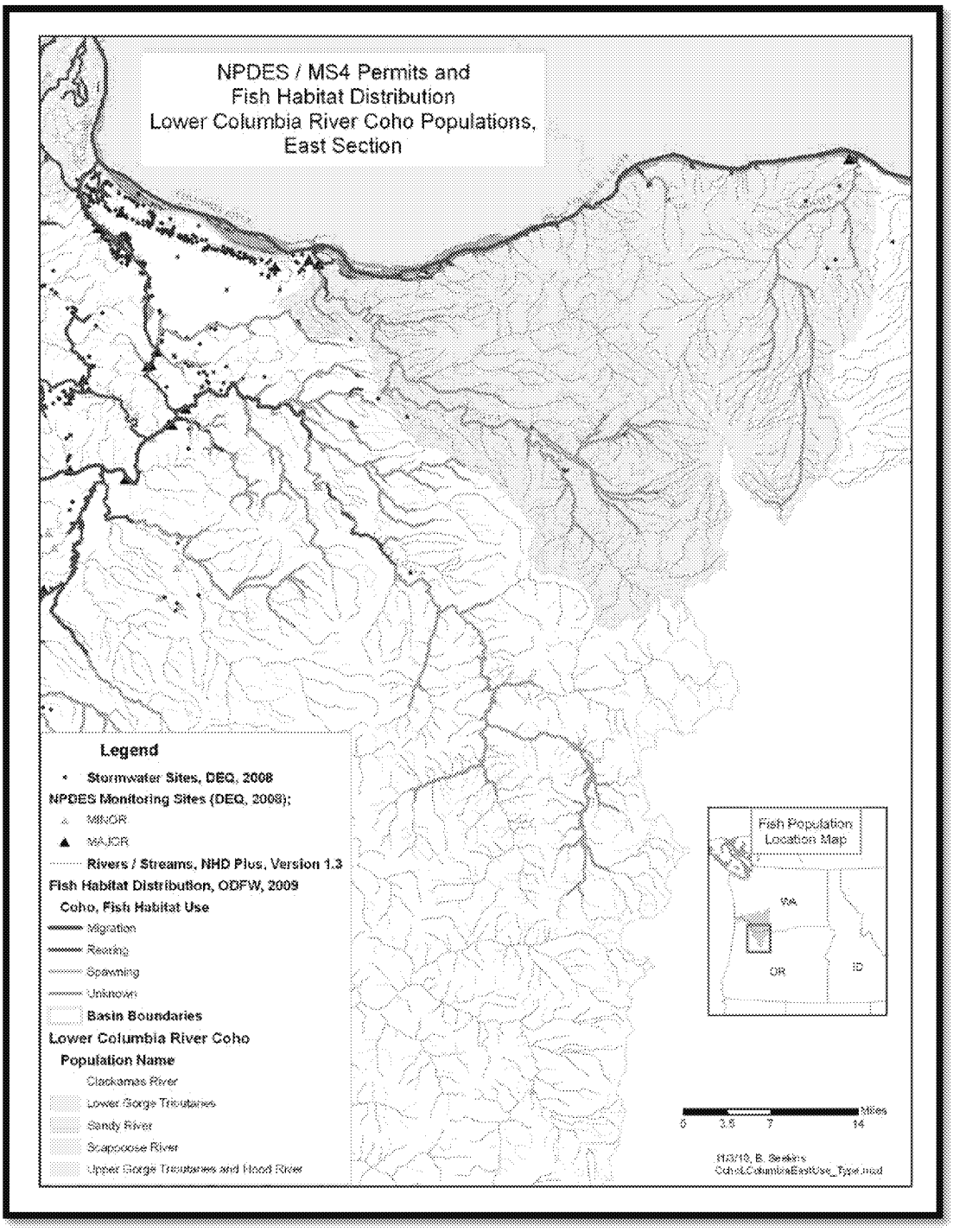

Figure 2.5.2.1.7 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR coho salmon (map 1 of 2).

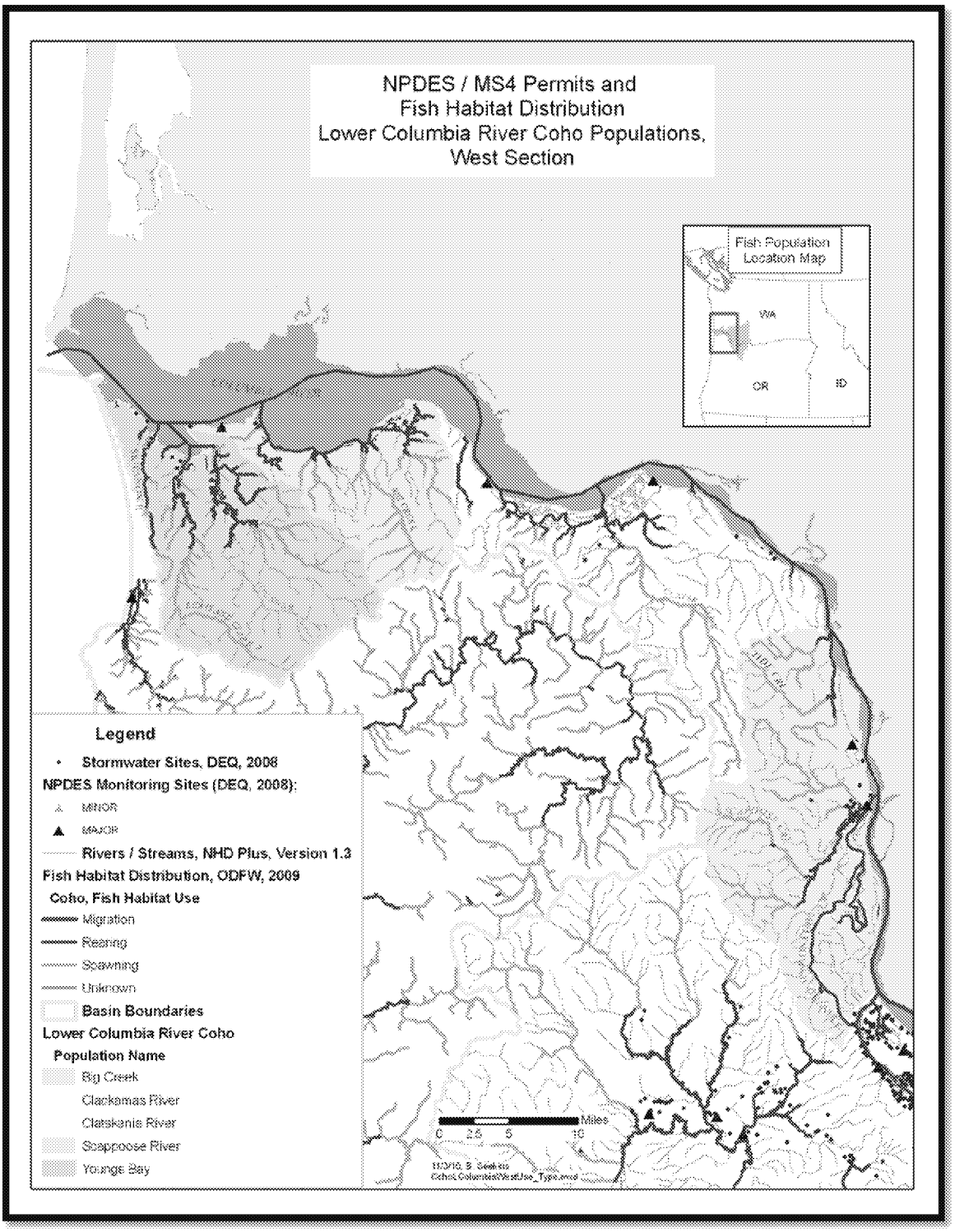

Figure 2.5.2.1.8 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR coho salmon (map 2 of 2).

Table 2.5.2.1.15 UWR steelhead populations in Oregon. All five populations occur in Oregon.

ESU/DPS	Populations In Oregon
UWR Steelhead	Calapooia River
	Molalla River
	North Santiam
	South Santiam
	Westside Tributaries
	Willamette River—Mainstem

Table 2.5.2.1.16 Type, number, and chemicals discharged for MS4 and NPDES permits within the UWR steelhead DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	118	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	50	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

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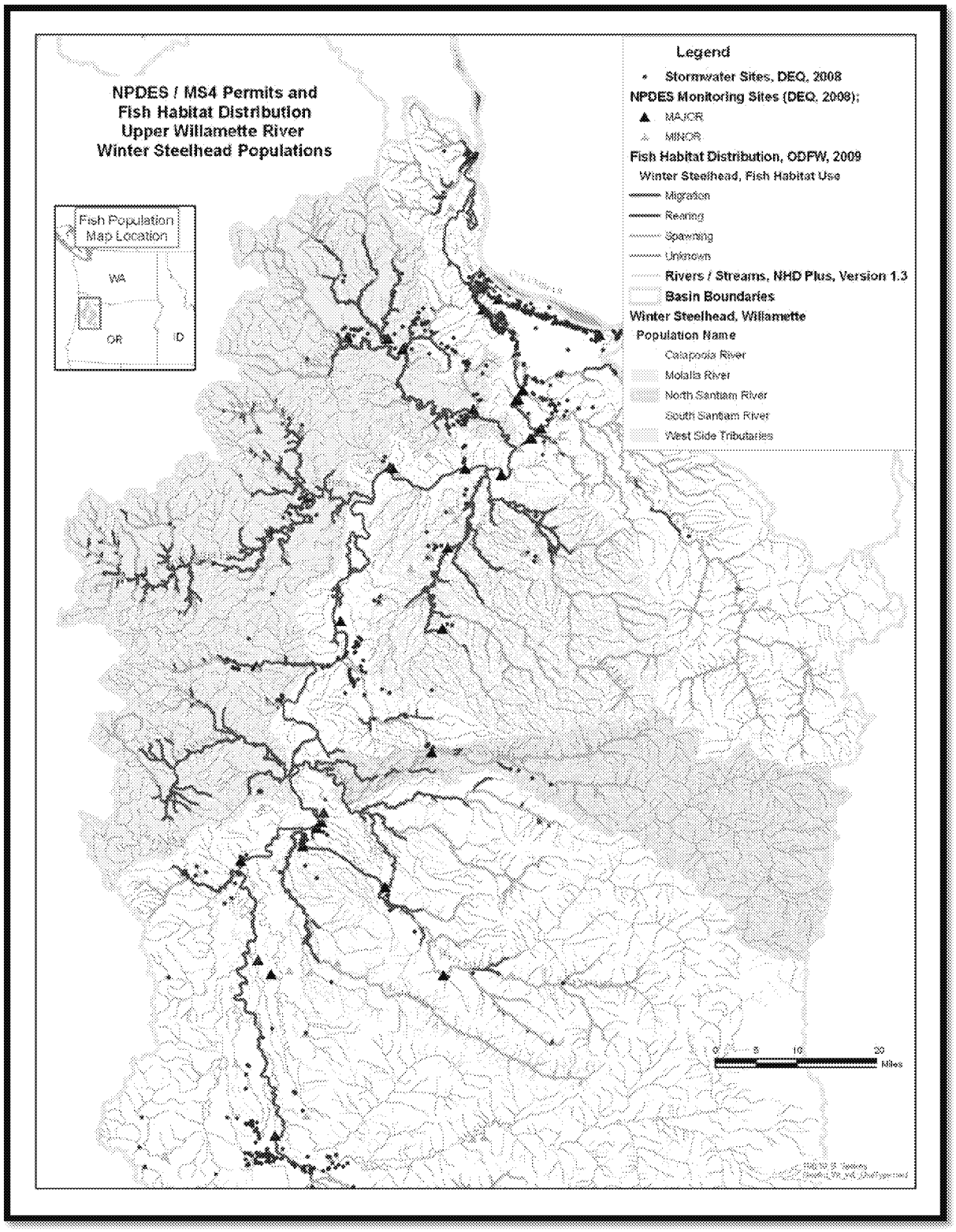

Figure 2.5.2.1.9 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for UWR steelhead.

Table 2.5.2.1.17 UWR Chinook salmon populations in Oregon. All seven populations occur in Oregon.

ESU/DPS	Populations In Oregon
UWR Chinook Salmon	Calapooia
	Clackamas
	McKenzie
	Middle Fork
	Molalla
	North Santiam
	South Santiam
	Willamette River—Mainstem

Table 2.5.2.1.18 Type, number, and chemicals discharged for MS4 and NPDES permits within the UWR Chinook salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	140	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	55	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

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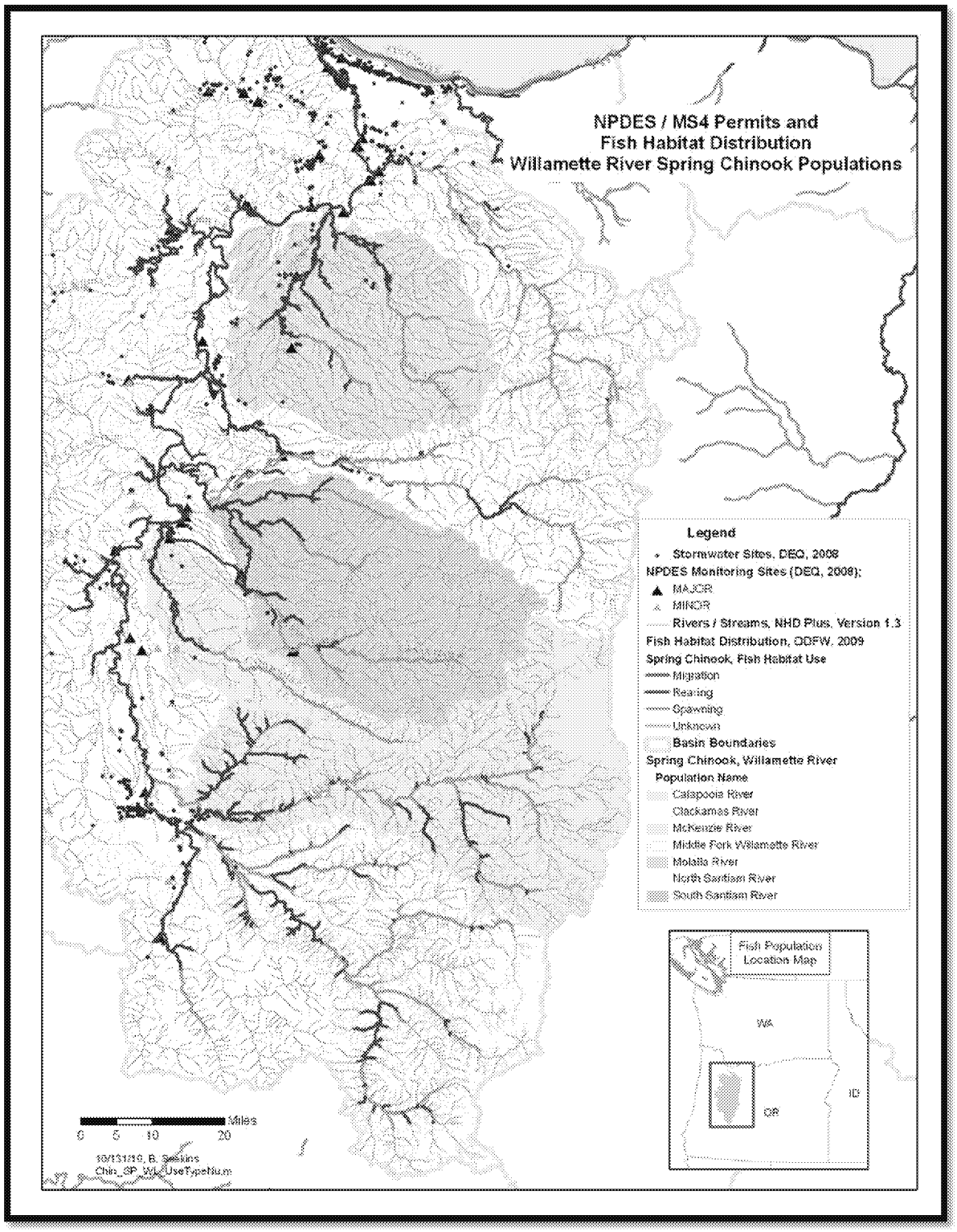

Figure 2.5.2.1.10 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for UWR Chinook salmon (map 1 of 2).

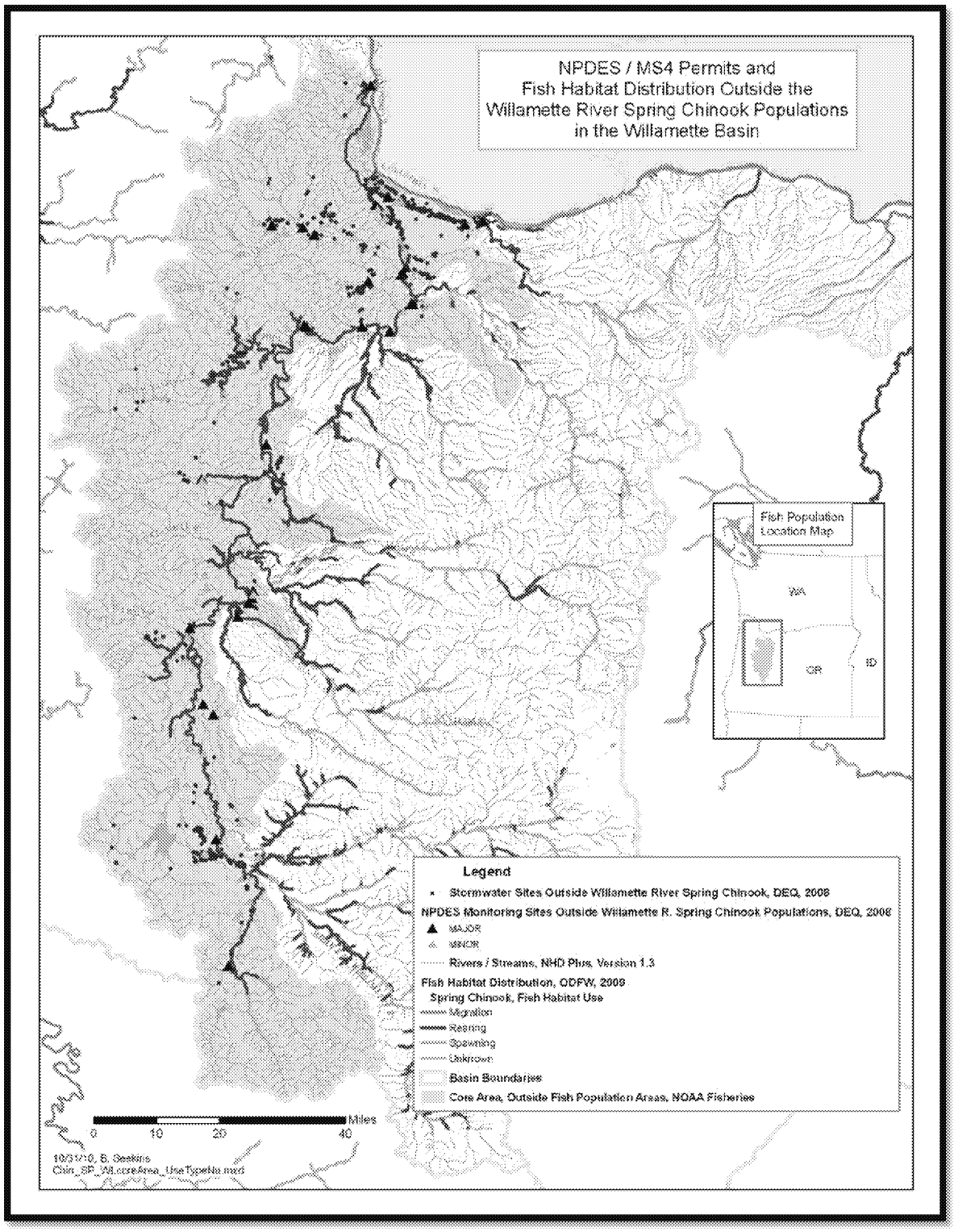

Figure 2.5.2.1.11 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for UWR Chinook salmon, non-core areas (map 2 of 2).

Table 2.5.2.1.19 LCR steelhead populations in Oregon. Five of 26 populations occur in Oregon.

ESU/DPS	Populations In Oregon
LCR Steelhead	Clackamas
	Hood River
	Lower Gorge Tributaries
	Upper Gorge Tributaries
	Sandy River

Table 2.5.2.1.20 Type, number, and chemicals discharged for MS4 and NPDES permits within the LCR steelhead DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	320	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	31	Aluminum, Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Pentachlorophenol, Selenium, Silver, Tributyltin, Zinc

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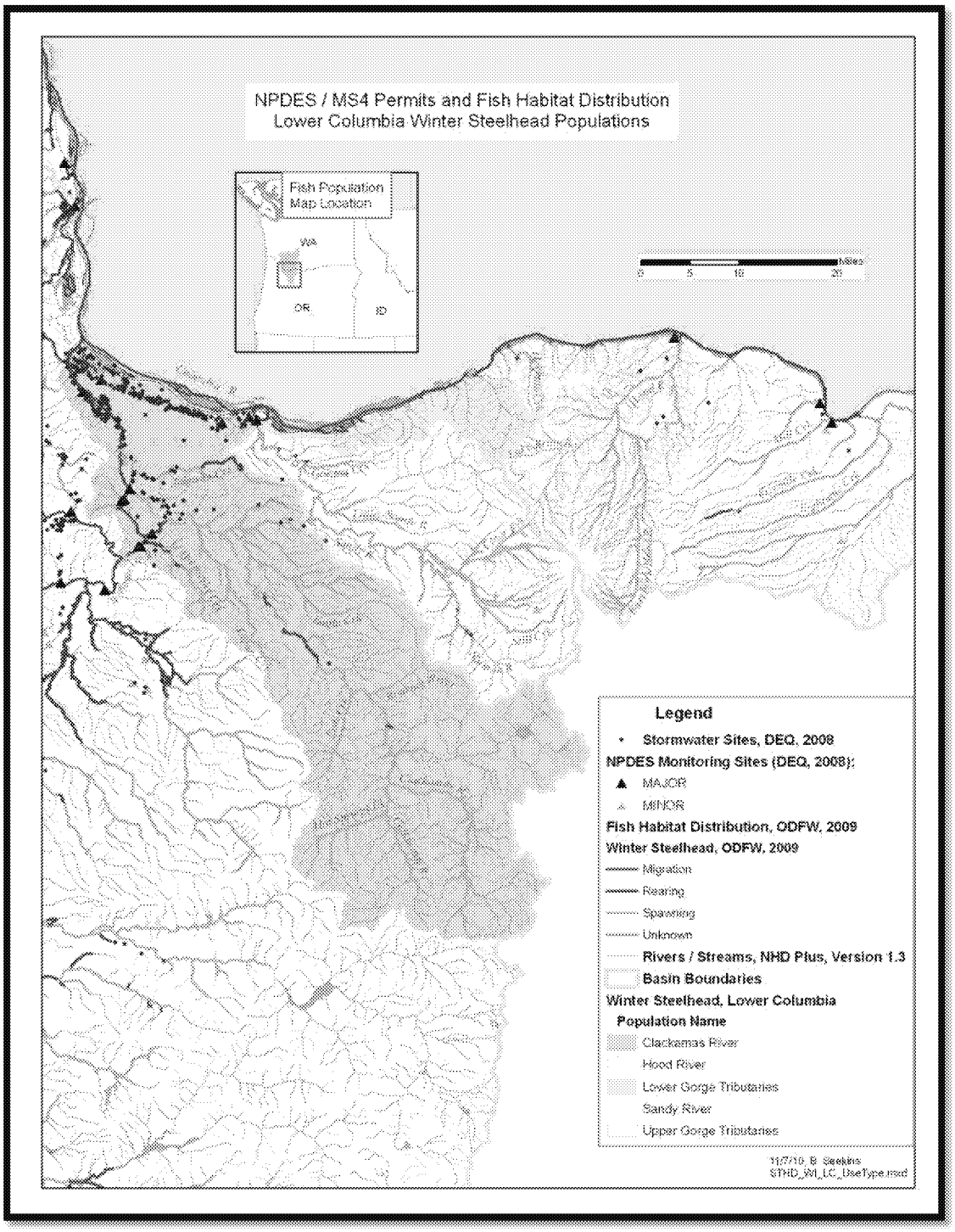

Figure 2.5.2.1.12 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR steelhead (winter).

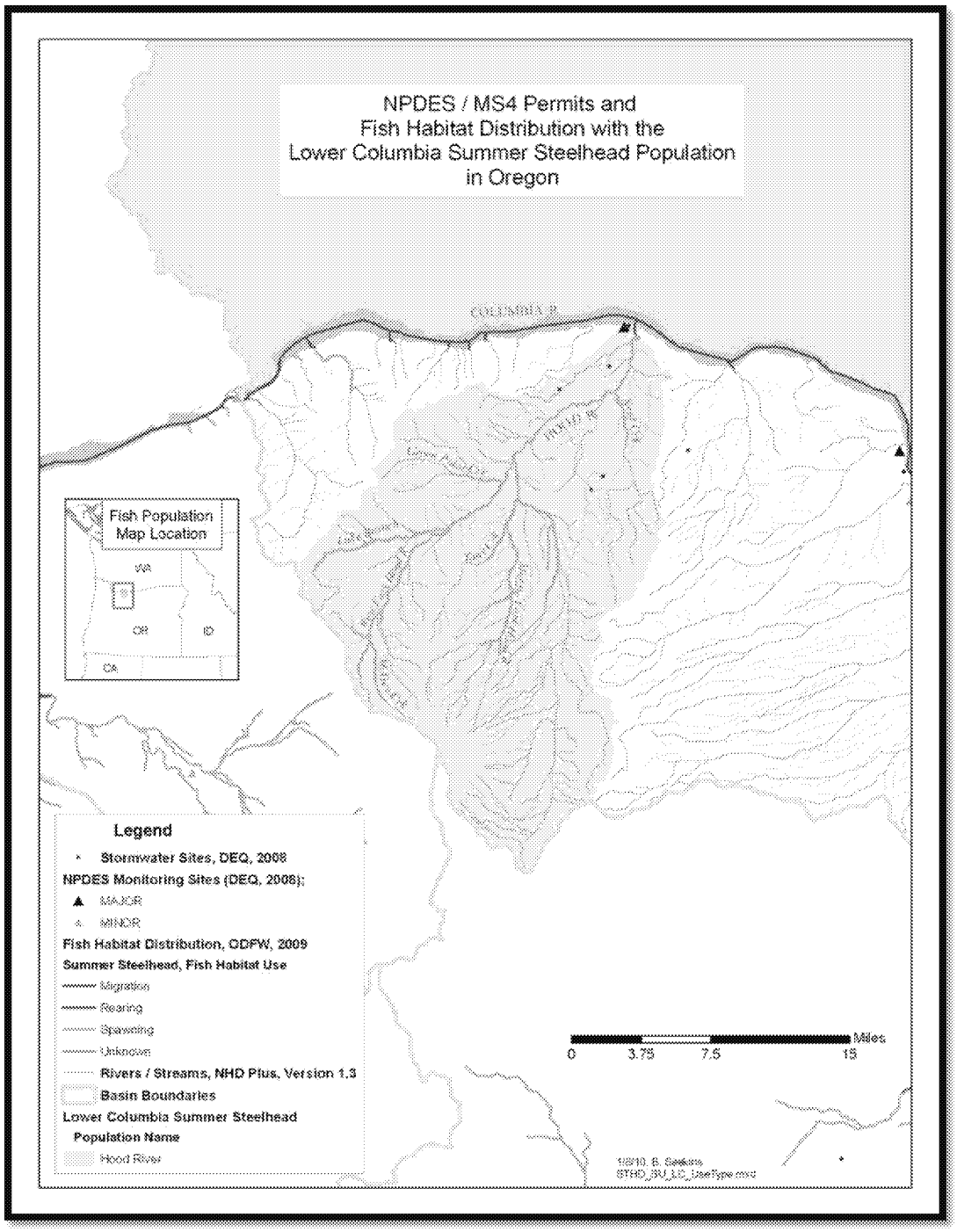

Figure 2.5.2.1.13 LCR Steelhead (summer). MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for LCR steelhead (summer).

Table 2.5.2.1.21 OC coho salmon populations in Oregon. All 56 populations occur in Oregon.

ESU/DPS	Populations In Oregon	
OC Coho Salmon	Necanicum	Devils Lake
	Ecola	Siltcoos
	Arch Cape	Siletz
	Short Sands	Tahkenitch
	Nehalem	Schoolhouse
	Spring	Threemile
	Watseco	Fogarty
	Netarts	Depoe Bay
	Rover	Lower Umpqua
	Sand	Middle Umpqua
	Nestucca	North Umpqua
	Neskowin	South Umpqua
	Alsea	Spencer
	Big (near Alsea)	Wade
	Rocky	Big
	Vingie	Coal
	Yachats	Tenmile
	Cummins	Moolack
	Bob	Coos
	Tenmile Creek	Big (near Yaquina)
	Tillamook Bay	Coquille
	Rock	Yaquina
	China	Johnson
	Cape	Theil
	Berry	Twomile
	Sutton (Mercer Lake)	Beaver
	Salmon	Floras/New
	Siuslaw	Sixes

Table 2.5.2.1.22 Type, number, and chemicals discharged for MS4 and NPDES permits within the OC coho salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	92	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	43	Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Selenium, Silver, Zinc

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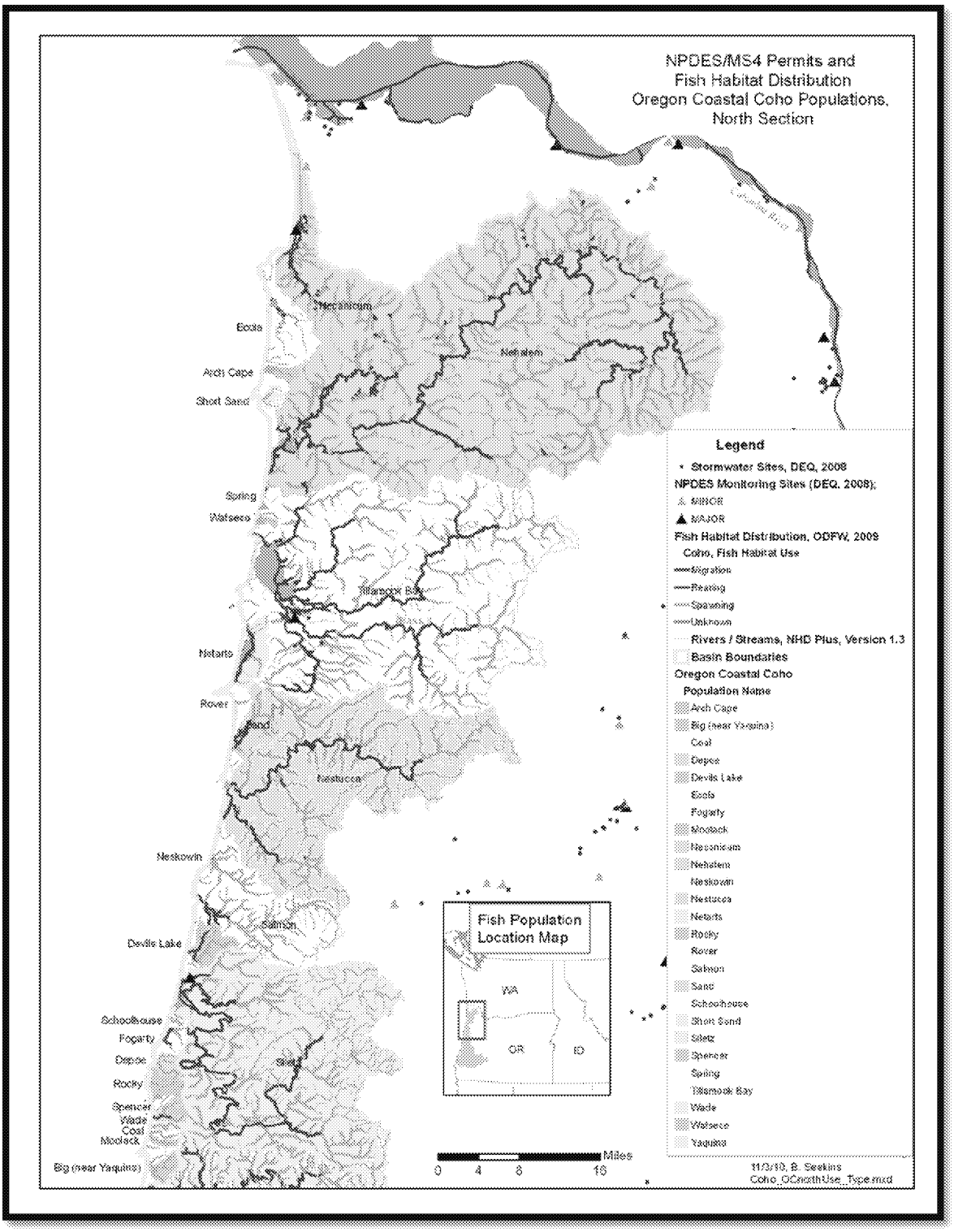

Figure 2.5.2.1.14 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for OC coho salmon (north coast).

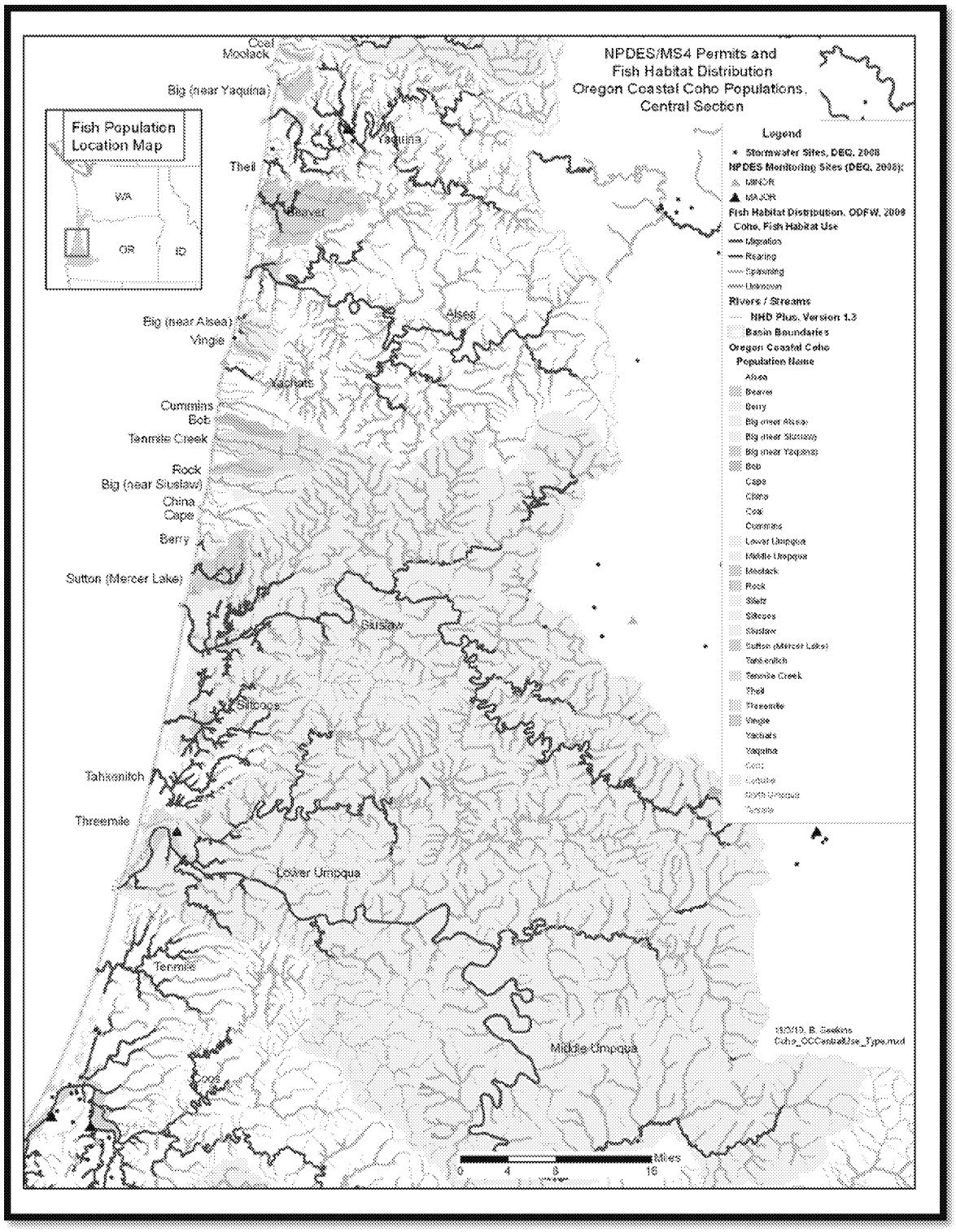

Figure 2.5.2.1.15 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for OC coho salmon (central coast).

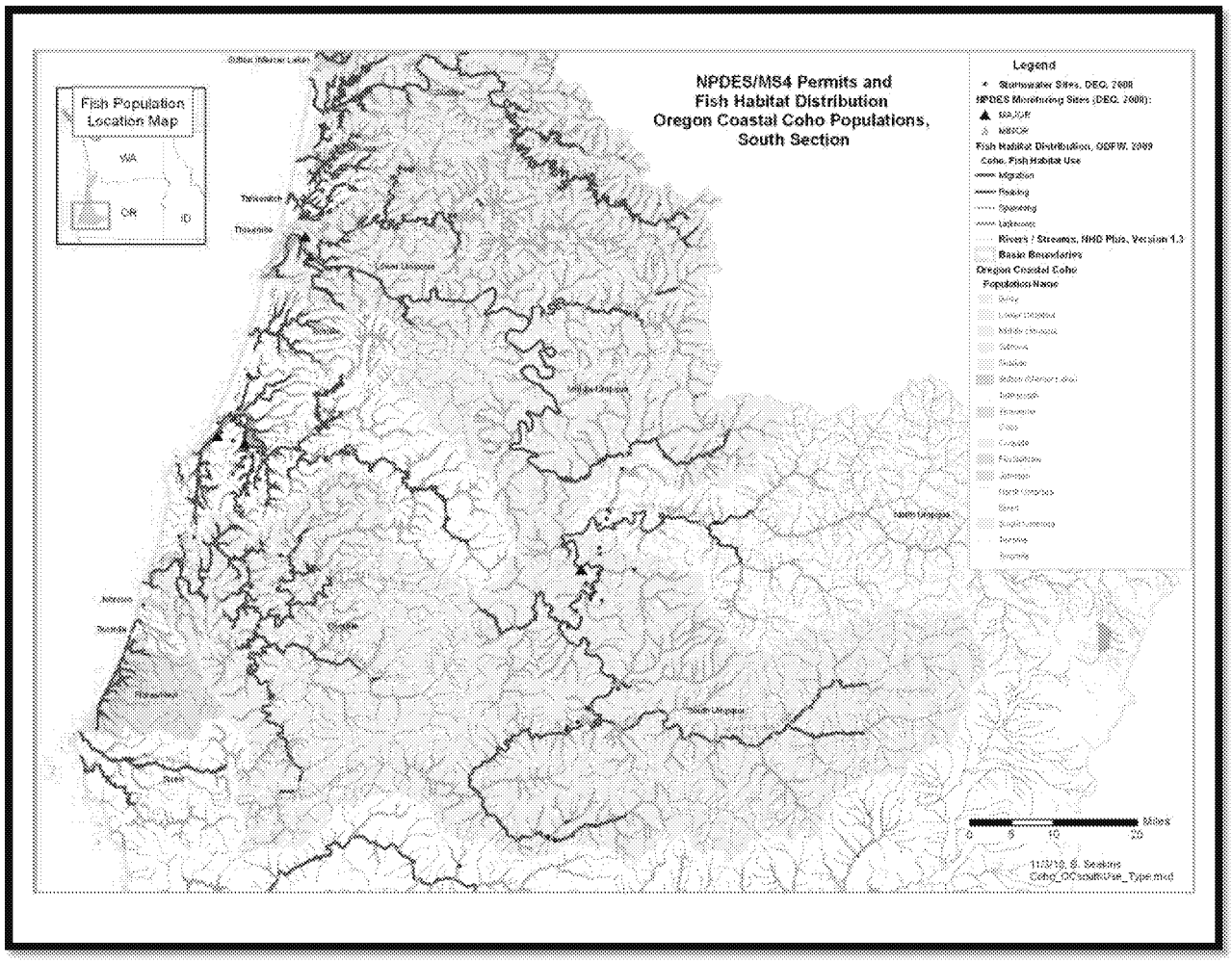

Figure 2.5.2.1.16 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for OC coho salmon (south coast).

Table 2.5.2.1.23 SONCC coho salmon populations in Oregon. Seventeen of 42 populations occur in Oregon.

ESU/DPS	Populations In Oregon
SONCC Coho Salmon	Bush Creek
	Chetco
	Elk
	Euchre
	Hubbard
	Hunter
	Illinois (OR and CA)
	Lower Rouge
	Middle Rouge and Applegate
	Mill Creek
	Mussel Creek
	Pistol
	Smith (OR and CA)
	Upper Klamath (OR and CA)
	Upper Rogue
	Winchuck River
	Brush Creek

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Table 2.5.2.1.24 Type, number, and chemicals discharged for MS4 and NPDES permits within the SONCC coho salmon ESU boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	62	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	12	Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Selenium, Silver, Zinc

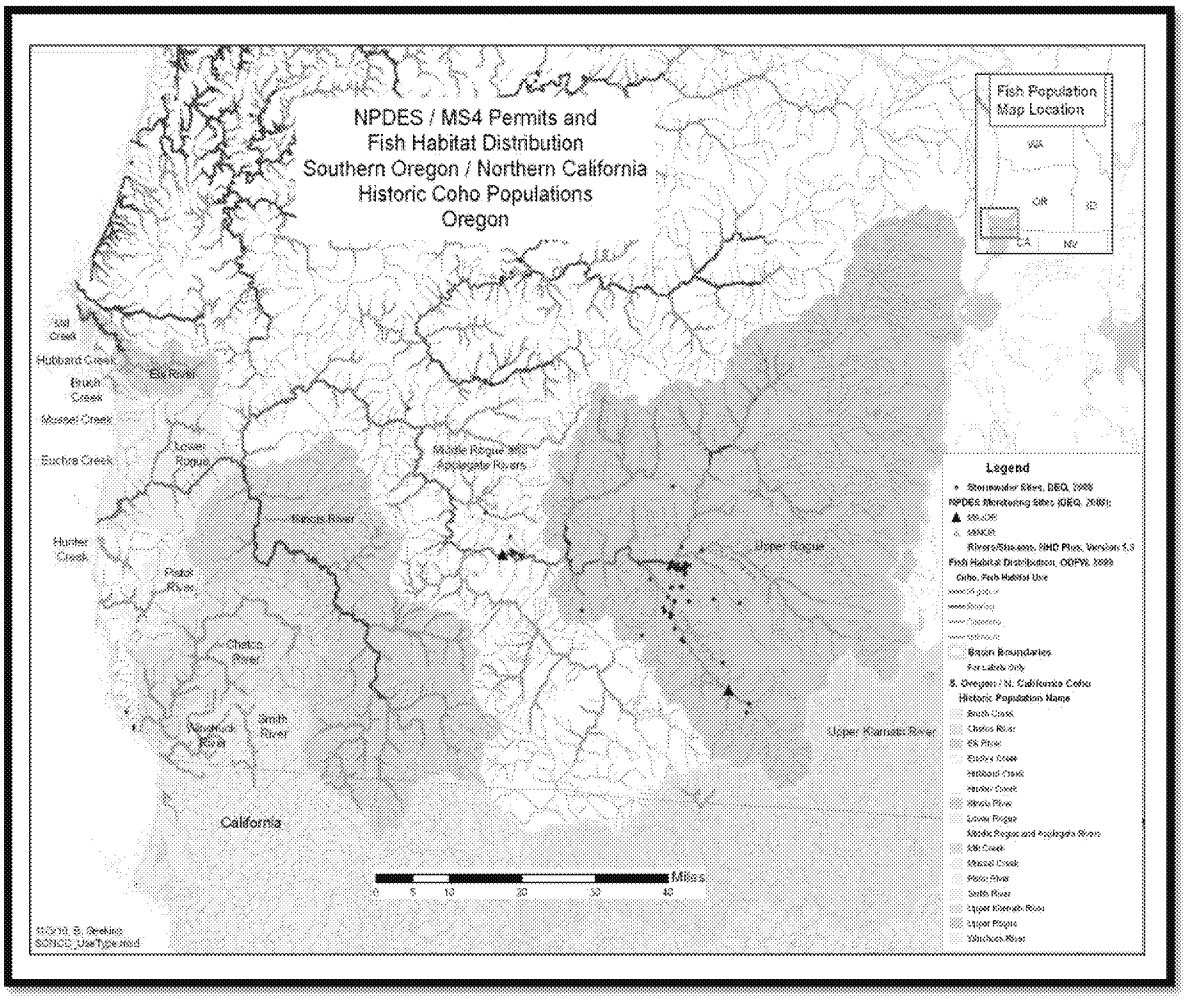

Figure 2.5.2.1.1.17 MS4 and NPDES permit/point-source discharge spatial distribution and fish distribution for SONCC coho salmon (Oregon populations).

2.5.2.2 Other Anadromous Fishes

2.5.2.2.1. Green Sturgeon

Table 2.5.2.2.1.1 No resident populations occur in Oregon.

ESU/DPS	Populations In Oregon
Green Sturgeon	NA

Table 2.5.2.2.1.2 Type, number, and chemicals discharged for MS4 and NPDES permits in Oregon that overlap with green sturgeon distribution (migratory).

Type of Permit	Number	Chemical(s)
MS4	324	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	23	Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Selenium, Silver, Tributyltin, Zinc

2.5.2.2.2. Eulachon

Table 2.5.2.2.2.1 Type, number, and chemicals discharged for MS4 and NPDES permits within the eulachon DPS boundary in Oregon.

Type of Permit	Number	Chemical(s)
MS4	327	Copper, Lead, Zinc, Cadmium, Chromium, Nickel, Ammonia, Arsenic, Silver, Iron, Mercury, Cyanide, Molybdenum, Selenium
NPDES	26	Ammonia, Arsenic, Cadmium, Copper, Chromium, Lead, Nickel, Selenium, Silver, Tributyltin, Zinc

Table 2.5.2.2.2.2. Eulachon populations in Oregon. Six of 24 populations occur in Oregon.

ESU/DPS	Populations In Oregon
Eulachon	Chetco
	Umpqua
	Ten Mile Creek
	Hood River
	Sandy River
	Columbia River

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Table 2.5.2.2.2.3 Regulated and unregulated toxics in the State of Oregon (ODEQ 2003). Compounds considered in this opinion for approval by EPA are shaded.

Aquatic Life Criteria				
	Freshwater	**Freshwater**	**Marine**	**Marine**
	Acute Criteria	**Chronic Criteria**	**Acute Criteria**	**Chronic Criteria**
Compound (µg/L)				
Antimony				
Arsenic *	360	190	69	36
Cadmium ***	3.9	1.1	43	9.3
Chromium III ***	1700	210		
Chromium VI *	16	11	1100	50
Copper ***	18	12	2.9	2.9
Lead ***	82	3.2	241	5.6
Mercury	2.4	0.012	2.1	0.025
Nickel ***	1400	160	75	8.3
Selenium *	260	35	410	54
Silver **	4.1	0.12	2.3	
Thallium				
Zinc ***	120	110	95	86
Cyanide	22	5.2	1	1
Asbestos				
Dioxin (2,3,7,8-TCDD)				
Acrolein				
Acrylonitrile				
Benzene				
Bromoform				
Carbon Tetrachloride				
Chlorobenzene				
Chlorodibromomethane				
Chloroform				
Dichlorobromomethane				
Dichloroethane 1,2-				
Dichloroethylene 1,1-				
Dichloropropane 1,2-				
Dichloropropene 1,3-				
Ethylbenzene				
Methyl Bromide				
Methylene Chloride				
Tetrachloroethane 1,1,2,2-				
Tetrachloroethylene				
Toluene				
Dichloroethylene 1,2-Trans-				
Trichloroethane 1,1,2-				
Trichloroethylene				
Vinyl Chloride				
Chlorophenol 2-				
Dichlorophenol 2,4-				

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Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
Dimethylphenol 2,4-				
Methyl-4,6-Dinitrophenol 2-				
Dinitrophenol 2,4-				
Pentachlorophenol	20	13	13	7.9
Phenol				
Trichlorophenol 2,4,6-				
Acenaphthene				
Anthracene				
Benzidine				
BenzoaAnthracene				
BenzoaPyrene				
BenzobFluoranthene				
BenzokFluoranthene				
ChloroethylEther, Bis2-				
ChloroisopropylEther, Bis2-				
EthylhexylPhthalate, Bis2-				
Butylbenzyl Phthalate				
Chloronaphthalene 2-				
Chrysene				
Dibenzoa,hAnthracene				
Dichlorobenzene 1,2-				
Dichlorobenzene 1,3-				
Dichlorobenzene 1,4-				
Dichlorobenzidine 3,3'-				
DiethylPhthalate				
Dimethyl Phthalate				
Di-n-Butyl Phthalate				
Dinitrotoluene 2,4-				
Diphenylhydrazine 1,2-				
Fluoranthene				
Fluorene				
Hexachlorobenzene				
Hexachlorobutadiene				
Hexachlorocyclopentadiene				
Hexachloroethane				
Ideno1,2,3-cdPyrene				
Isophorone				
Nitrobenzene				
Nitrosodimethylamine, N-				
Nitrosodi-n-Propylamine, N-				
Nitrosodiphenylamine, N-				
Pyrene				
Trichlorobenzene 1,2,4-				
Aldrin	3.0		1.3	

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Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
BHC, alpha-				
BHC, beta-				
BHC, gamma- (Lindane)	2	0.08	0.16	
Chlordane	2.4	0.0043	0.09	0.004
DDT 4,4'-	1.1	0.001	0.13	0.001
DDE 4,4'-				
DDD 4,4'-				
Dieldrin	2.5	0.0019	0.71	0.0019
Alpha-Endosulfan				
Beta-Endosulfan				
Endosulfan Sulfate				
Endrin	0.18	0.0023	0.037	0.0023
Endrin Aldehyde				
Heptachlor	0.52	0.0038	0.053	0.0036
Heptachlor Epoxide				
Polychlorinated biphenyls PCBs:	2	0.014	10	0.03
Toxaphene	0.73	0.0002	0.21	0.0002
Aluminum				
Ammonia (mg/L)	6	0.76		
Barium				
Chloride	860000	230000		
Chlorine	19	11	13	7.5
Chlorophenoxy Herbicide 2,4,5,-TP				
Chlorophenoxy Herbicide 2,4-D				
Chloropyrifos	0.083	0.041	0.011	0.0056
Demeton		0.1		0.1
Ether, Bis Chloromethyl				
Guthion		0.01		0.01
Hexachlorocyclo-hexane-Technical				
Iron		1000		
Malathion		0.1		0.1
Manganese				
Methoxychlor		0.03		0.03
Mirex		0.001		0.001
Nitrates				
Nitrosamines				
Dinitrophenols				
Nitrosodibutylamine,N				
Nitrosodiethylamine,N				
Nitrosopyrrolidine,N				
Parathion	0.065	0.013		
Pentachlorobenzene				
Phosphorus Elemental				0.1
Sulfide-Hydrogen Sulfide		2.0		2.0

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Aquatic Life Criteria				
	Freshwater	Freshwater	Marine	Marine
	Acute Criteria	Chronic Criteria	Acute Criteria	Chronic Criteria
Compound (µg/L)				
Tetrachlorobenzene,1,2,4,5				
Tributyltin TBT				
Trichlorophenol 2,4,5				

* all criteria expressed as dissolved metal
** all criteria expressed as dissolved metal. FW acute criteria are hardness dependent (concentration shown is hardness = 100 mg/L $CaCO_3$)
*** all criteria expressed as dissolved metal. FW criteria are hardness dependent (concentration shown is hardness = 100 mg/L $CaCO_3$)

The compounds listed in Table 2.5.2.3 that are not directly part of the proposed action (unshaded) are, however, part of EPA's overall approval of Oregon's water quality standards, and are compounds that are part of the environmental baseline. These compounds, either individually or in combination, are likely to adversely affect listed species considered in this opinion where exposure occurs. For example, concurrent exposure to cyanide and ammonia is likely to produce greater than additive effects to acute lethality in rainbow trout, salmon, and chub (Smith *et al.* 1979, Alabaster *et al,* 1983, and Douderoff 1976), and to sublethal effects to growth in rainbow trout (Smith *et al.* 1979). In rainbow trout and salmon, effects to acute lethality were 1.2 and 1.63 times greater than would be expected by additivity. Concurrent exposure to cyanide and zinc also resulted in synergistic effects to acute lethality in fathead minnows, where toxicity was 1.4 times that predicted by additivity (Smith *et al.* 1979).

Furthermore, Glubokoy (1990) reported increased mortality (0.7% to 10% above baseline) of coho salmon during early ontogeny when exposed to dichloro-diphenyl-trichloroethane (DDT) over the range of 0.1 µg/L to 10 µg/L, Niimi (1996) determined that 48 hour to 96 hour exposure to Polychlorinated biphenyls (PCB) concentrations on the order of 1 µg/L or more resulted in fish mortality, and Macek *et al.* (1969) reported a 96 hour LC_{50} value of 2.2 µg/L for rainbow trout exposed at 12.7EC, pH 7.1 in a static experiment with a 95% aldrin concentration.

2.5.2.2.3 Marine Mammals

Marine mammals are unlikely to be directly exposed to the subject pollutants, with the exception of Steller sea lions.

2.5.2.2.4 Sea Turtles

Sea turtles are unlikely to be directly exposed to the subject pollutants.

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2.5.2.3 General Environmental Baseline Conditions

Columbia River Basin. Major tributaries to the Columbia River include the Snake, Willamette, Salmon, Flathead, and Yakima Rivers; smaller rivers include the Owyhee, Grande Ronde, Clearwater, Spokane, Methow, Cowlitz, and the John Day Rivers. The Snake River is the largest tributary at more than 1,000 miles long; its headwaters originate in Yellowstone National Park, Wyoming. The second largest tributary is the Willamette River in Oregon (Kammerer 1990, Hinck *et al.* 2004). The average annual discharge at the mouth of the Columbia River is 265,000 cubic feet per second (Kammerer 1990). A saltwater wedge extends 23 miles upstream of the mouth, with tidal influences extending up to 146 miles up river (Hinck *et al.* 2004). Table 2.5.2.3.1 provides information on selected tributaries to the Columbia River.

Table 2.5.2.3.1. Select tributaries of the Columbia River

Watershed	Approx Length (mi)	Basin Size (mi^2)	Physiographic Provinces*	Mean Annual Precip. (in)	Mean Discharge (cfs)
Snake/Salmon Rivers	870	108,495	CU, NR, MR, B/R	14	55,267
Willamette River	143	11,478	CS, PB	60	32,384

Data from Carter and Resh 2005
*Physiographic Provinces: CU = Columbia-Snake River Plateaus, NR = Northern Rocky Mountains, MR = Middle Rocky Mountains, B/R = Basin & Range, CS = Cascade-Sierra Mountains, PB = Pacific Border

Human Activities and Their Impacts.

Land Use. More than 50% of the United States portion of the Columbia River Basin is in Federal ownership (most of which occurs in high desert and mountain areas), 39% is in private land ownership (most of which occurs in river valleys and plateaus), and the remainder is divided among tribes, state, and local governments (Hinck *et al.* 2004) (Table 2.5.2.3.2).

Table 2.5.2.3.2. Land uses and population density in select tributaries of the Columbia River Basin.

Watershed	Land Use Categories (%)				Density ($people/mi^2$)
	Agriculture	Forest	Urban	Other	
Snake/Salmon Rivers	30	10-15	1	54 scrub/rangeland/barren	39
Willamette River	19	68	5	--	171

Data from Stanford *et al.* 2005

The interior Columbia River basin has been altered substantially by humans, causing dramatic changes and declines in native fish populations. In general the basin supports a variety of mixed uses. Predominant human uses include logging, agriculture, ranching, hydroelectric power generation, mining, fishing, a variety of recreational activities, and urban uses. The decline of salmon runs in the Columbia River is attributed to loss of habitat, blocked migratory corridors, altered river flows, pollution, overharvest, and competition from hatchery fish. Critical ecological connectivity (mainstem to tributaries and riparian floodplains) has been disconnected by dams and associated activities such as floodplain deforestation and urbanization. The most

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productive floodplains of the watershed are either flooded by hydropower dams or dewatered by irrigation diversions. Portions of the basin are also subject to impacts from cattle grazing and irrigation withdrawals. In the Willamette River, riparian vegetation was greatly reduced by land conversion. By 1990, only 37 % of the riparian area within 120 meters was forested, 30% was agricultural fields and 16 % was urban or suburban lands.

Agriculture and Ranching. Roughly 6% of the annual flow from the Columbia River is diverted for the irrigation of 7.3 million acres of croplands within the basin. The vast majority of these agricultural lands are located along the lower Columbia River, the Willamette, Hood, and Snake rivers, and the Columbia Plateau (Hinck *et al.* 2004).

Agriculture and ranching increased steadily within the Columbia River basin from the mid to late 1800. By the early 1900s, agricultural opportunities began increasing at a much more rapid pace with the creation of more irrigation canals and the passage of the Reclamation Act of 1902 (NRC 2004). Today, agriculture represents the largest water user within the basin (>90%). Agriculture, ranching, and related services employ more than nine times the national average (19% of the households within the basin; NRC 2004).

Ranching practices have increased soil erosion and sediment loads within the Columbia' River's tributaries, the worst of these effects may have occurred in the late 1800s and early 1900s from deliberate burning to increase grass production (NRC 2004). Several measures are in use to reduce the impacts of grazing, including restricting grazing in degraded areas, reduced grazing allotments, and lower stocking rates. Today, agricultural impacts to water quality within the basin are second to large-scale influences of hydromodification projects for both power generation and irrigation. Water quality impacts from agricultural activities include alteration of the natural temperature regime, insecticide and herbicide contamination, and increased suspended sediments.

The USGS has a number of fixed water quality sampling sites throughout various tributaries of the Columbia River, many of which have been in place for decades. Water volumes, crop rotation patterns, crop type, and basin location are some of the variables that influence the distribution and frequency of pesticides within a tributary. Detection frequencies for a particular pesticide can vary widely. One study conducted by the USGS between May 1999 and January 2000 detected 25 pesticide compounds (Ebbert and Embrey 2001). Another study detected at least two pesticides or their breakdown products in 91% of the samples collected, with the median number of chemicals being eight, and a maximum of 26. The herbicide 2,4-D occurred most often in the mixtures, along with azinphos-methyl, the most heavily applied pesticide, and atrazine, one of the most mobile aquatic pesticides (Fuhrer *et al.* 2004). In addition to current-use chemicals, these legacy chemicals continue to pose a serious problem to water quality and fish communities despite their ban in the 1970s and 1980s (Hinck *et al.* 2004).

Fish and macroinvertebrate communities exhibit an almost linear decline in condition as the level of agriculture intensity increases within a basin (Cuffney *et al.* 1997, Fuhrer *et al.* 2004). A study conducted in the late 1990s examined 11 species of fish, including anadromous and resident fish collected throughout the Columbia River basin for a suite of 132 contaminants, including 51 semi-volatile chemicals, 26 pesticides, 18 metals, seven PCBs, 20 dioxins, and 10 furans. The

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study revealed PCBs, metals, chlorinated dioxins and furans (products of wood pulp bleaching operations) and other contaminants within fish tissues; white sturgeon tissues contained the greatest concentrations of chlorinated dioxins and furans (Hinck *et al.* 2004).

Urban and Industrial Development. The largest urban area in the basin is the greater Portland metropolitan area. Portland's population exceeds 500,000, and the next largest cities Salem and Eugene, OR have over 100,000 people (Hinck *et al.* 2004). Overall, the basin's population density is one-third the national average, and while the basin covers about 8% of United States land, only about 1.2% of the United States population lives within the basin (Hinck *et al.* 2004).

Discharges from sewage treatment plants, paper manufacturing, and chemical and metal production represent the top three permitted sources of contaminants within the lower basin according to discharge volumes and concentrations (Rosetta and Borys 1996). Rosetta and Borys (1996) review of 1993 data indicate that 52% of the point source waste water discharge volume is from sewage treatment plants, 39% from paper and allied products, 5% from chemical and allied products, and 3% from primary metals. However, the paper and allied products industry are the primary sources of the suspended sediment load (71%). Additionally, 26% of the point source waste water discharge volume comes from sewage treatment plants and 1% is from the chemical and allied products industry. Nonpoint source discharges (urban stormwater runoff) account for significant pollutant loading to the lower basin, including most organics and over half of the metals. Although rural nonpoint sources contributions were not calculated, Rosetta and Borys (1996) surmised that in some areas and for some contaminants, rural areas may contribute a large portion of the nonpoint source discharge. This is particularly true for pesticide contamination in the upper river basin where agriculture is the predominant land use. Water quality has been reduced by phosphorus loads and decreased water clarity, primarily along the lower and middle sections of the Columbia River Estuary. Although sediment quality is generally very good, benthic indices have not been established within the estuary. Fish tissue contaminant loads (PCBs, DDT, DDD, DDE, and mercury) are high and present a persistent and long lasting effect on estuary biology. Health advisories have been recently issued for people eating fish in the area that contain high levels of dioxins, PCBs, and pesticides. Morace (2012) reported waste water treatment plant samples containing anthropogenic organic compounds, pharmaceuticals, polybrominated diphenyl ether (PBDEs [brominated flame-retardants]), organochlorine or legacy compounds, currently used pesticides, mercury, and estrogenicity.

Habitat Modification. The mainstem habitats of the lower Columbia and Willamette rivers have been reduced primarily to a single channel. As a result, floodplain area is reduced, off-channel habitat features have been eliminated or disconnected from the main channel, and the amount of large woody debris in the mainstem has been reduced. Remaining areas are affected by flow fluctuations associated with reservoir management for power generation, flood control, and irrigation. Overbank flow events, important to habitat diversity, have become rare as a result of controlling peak flows and associated revetments. Portions of the basin are also subject to impacts from cattle grazing and irrigation withdrawals. Consequently, estuary dynamics have changed substantially.

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Habitat loss has fragmented habitat and human density increase has created additional loads of pollutants and contaminants within the Columbia River estuary (Anderson, Dugger, and Burke 2007). About 77 percent of swamps, 57 percent of marshes, and over 20 percent of tree cover have been lost to development and industry. The Willamette Basin Valley has been dramatically changed by modern settlement. The complexity of the mainstem river and extent of riparian forest have both been reduced by 80 percent (PNERC 2002). About 75 percent of what was formerly prairie and 60 percent of what was wetland have been converted to agricultural purposes. These actions, combined with urban development, bank stabilization, and in-river and nearshore gravel mining, have resulted in a loss of floodplain connectivity and off-channel habitat (PNERC 2002).

Hydromodification Projects. More than 400 dams exist in the basin, ranging from mega dams that store large amounts of water to small diversion dams for irrigation. Every major tributary of the Columbia River except the Salmon River is totally or partially regulated by dams and diversions. More than 150 dams are major hydroelectric projects, with 18 dams located on mainstem Columbia River and its major tributary, the Snake River. The Federal Columbia River Power System encompasses the operations of 14 major dams and reservoirs on the Columbia and Snake Rivers. These Federal projects are a major source of power in the region, and provide flood control, navigation, recreation, fish and wildlife, municipal and industrial water supply, and irrigation benefits.

Development of the Pacific Northwest regional hydroelectric power system, dating to the early 20th century, has had profound effects on the ecosystems of the Columbia River Basin (ISG 1996). These effects have been especially adverse to the survival of anadromous salmonids. The construction of the Federal power system modified migratory habitat of adult and juvenile salmonids, and in many cases presented a complete barrier to habitat access. Both upstream and downstream migrating fish are impeded by the dams, and a substantial number of juvenile salmonids are killed and injured during downstream migrations. Physical injuries and deaths occur as juveniles pass through turbines, bypasses, and spillways. Indirect effects of passage through all routes may include disorientation, stress, delays in passage, exposure to high concentrations of dissolved gases, warm water, and increased predation. Dams have also flooded historical spawning and rearing habitat with the creation of massive water storage reservoirs. More than 55 percent of the Columbia River Basin that was accessible to salmon and steelhead before 1939 has been blocked by large dams (NWPPC 1986).

The mainstem habitats of the lower Columbia and Willamette Rivers have been reduced primarily to a single channel. As a result, floodplain area has been reduced, off-channel habitat features have been eliminated or disconnected from the main channel, and the amount of large woody debris in the mainstem has been reduced. Remaining areas are affected by flow fluctuations associated with reservoir management for power generation, flood control and irrigation. Overbank flow events, important to habitat diversity, have become rare as a result of controlling peak flows and associated revetments. Consequently, estuary dynamics have changed substantially.

Artificial Propagation. There are several artificial propagation programs for salmon production within the Columbia River basin, many of which were instituted under Federal law to

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ameliorate the effects of lost natural salmon production within the basin from the dams. The hatcheries are operated by Federal, state, and tribal managers. For more than 100 years, hatcheries in the Pacific Northwest have been used to produce fish for harvest and replace natural production lost to dam construction, and have only minimally been used to protect and rebuild naturally produced salmonid population (*e.g.*, Redfish Lake sockeye salmon). In 1987, 95 percent of the coho salmon, 70 percent of the spring Chinook salmon, 80 percent of the summer Chinook salmon, 50 percent of the fall Chinook salmon, and 70 percent of the steelhead returning to the Columbia River Basin originated in hatcheries (CBFWA 1990). More recent estimates suggest that almost half of the total number of smolts produced in the basin come from hatcheries (Mann *et al.* 2005).

The impact of artificial propagation on the total production of Pacific salmon and steelhead has been extensive (Hard *et al.* 1992). Hatchery practices, among other factors, are a contributing factor to the 90 percent reduction in natural coho salmon runs in the lower Columbia River over the past 30 years (Flagg *et al.* 1995). Past hatchery and stocking practices have resulted in the transplantation of salmon and steelhead from nonnative basins, and the impacts of these practices are largely unknown. Adverse effects of these practices likely included loss of genetic variability within and among populations (Busack 1990 as cited in Hard *et al.* 1992, Riggs 1990, Reisenbichler 1997), disease transfer, increased competition for food, habitat, or mates, increased predation, altered migration, and displacement of natural fish (Steward and Bjornn 1990, Fresh 1997). Species with extended freshwater residence are likely to face higher risk of domestication, predation, or altered migration than are species that spend only a brief time in fresh water (Hard *et al.* 1992). Nonetheless, artificial propagation also may contribute to the conservation of listed salmon and steelhead although it is unclear whether or how much artificial propagation during the recovery process will compromise the distinctiveness of natural population (Hard *et al.* 1992).

Currently, NMFS is working on a hatchery reform project in the Columbia River Basin, which will include a collaborative review of how harvest and hatcheries (particularly Federally funded hatcheries) are affecting the recovery of listed salmon and steelhead in the basin. This effort was mandated by Congress in 2005, and is in its early stages. Eventually, the project team would create a management approach that allows tribal, state and Federal managers to effectively manage Columbia River Basin hatcheries to meet conservation and harvest goals consistent with their respective legal responsibilities.

Mining. Most of the mining in the basin is focused on minerals such as phosphate, limestone, dolomite, perlite, or metals such as gold, silver, copper, iron, and zinc. Many of the streams and river reaches in the basin are impaired from mining, and several abandoned, and former mining sites are designated as Superfund cleanup areas (Stanford *et al.* 2005, EPA 2007). According to the United States Bureau of Mines, there are about 14,000 inactive or abandoned mines within the Columbia River Basin of which nearly 200 pose a potential hazard to the environment (Quigley *et al.* 1997 as cited in Hinck *et al.* 2004). Contaminants detected in the water include lead and other trace metals. Mining of copper, cadmium, lead, manganese, and zinc in the upper Clark Fork River have contributed wastes to this basin since 1880 (Woodward *et al.* 1994). Benthic macroinvertebrates and fish within the basin have bioaccumulated metals,

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which are suspected of reducing their survival and growth (Farag *et al.* 1994, Woodward *et al.* 1994).

Commercial, Recreational, and Subsistence Fishing. During the mid-1800s, an estimated 10 to 16 million adult salmon and steelhead of all species entered the Columbia River each year. Large harvests of returning adult salmon during the late 1800s (20 to 40 million pounds of annually) significantly reduced population productivity (Mann *et al.* 2005). The largest known harvest of Chinook salmon occurred in 1883 when Columbia River canneries processed 43 million pounds of salmon (Lichatowich 1999). Commercial landings declined steadily from the 1920s to a low in 1993, when just over 1 million pounds were harvested (Mann *et al.* 2005).

Harvested and spawning adults reached 2.8 million in the early 2000s, of which almost half are hatchery produced (Mann *et al.* 2005). Most of the fish caught in the river are steelhead and spring/summer Chinook salmon, while ocean harvest consists largely of coho and fall Chinook salmon. Most ocean catches are made north of Cape Falcon, Oregon. Over the past five years, the number of spring and fall salmon commercially harvested in tribal fisheries has averaged between 25,000 and 110,000 fish (Mann 2004 in Mann *et al.* 2005). Recreational catch in both ocean and in-river fisheries varies from 140,000 to 150,000 individuals (Mann *et al.* 2005).

Interior Columbia River major subbasins: Deschutes, John Day, Umatilla, Walla Walla, Grande Ronde, and Imnaha Rivers. Habitat quality in tributary streams in the interior Columbia River subbasins varies from excellent in wilderness and roadless areas to poor in areas subject to heavy agricultural and urban development (Wissmar *et al.* 1994, Carmichael 2006).

Migratory habitat quality in this area has been severely affected by the development and operation of the FCRPS dams and reservoirs in the mainstem Columbia River, Bureau of Reclamation tributary projects, and privately owned dams in the Snake River. For example, construction of Hells Canyon Dam eliminated access to several likely production areas in Oregon and Idaho including the Burnt, Powder, Weiser, Payette, Malheur, Owyhee, and Boise river basins (Good *et al.* 2005). Hydroelectric development modified natural flow regimes, resulting in higher water temperatures, changes in fish community structure leading to increased rates of piscivorous and avian predation on juvenile salmon and steelhead, and delayed migration for both adult and juveniles. Physical features of dams such as turbines also kill migrating fish. In-river survival is inversely related to the number of hydropower projects encountered by emigrating juveniles.

Similarly, development and operation of extensive irrigation systems and dams for water withdrawal and storage in tributaries have drastically altered hydrological cycles. A series of large regulating dams on the middle and upper Deschutes River affect flow and block access to upstream habitat, and have extirpated one or more populations from the Cascades Eastern Slope major population (IC-TRT 2003). Similarly, operation and maintenance of large water reclamation systems such as the Umatilla Basin and Yakima Projects have significantly reduced flows and degraded water quality and physical habitat in this domain.

Many stream reaches are over-allocated under state water law, with more allocated water rights than existing streamflow conditions can support. Irrigated agriculture is common throughout this

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region and withdrawal of water increases summer stream temperatures, blocks fish migration, strands fish, and alters sediment transport (Spence *et al.* 1996). Reduced tributary stream flow has been identified as a major limiting factor for all listed salmon and steelhead species in this area except SR fall-run Chinook salmon (NMFS 2005).

North and Middle Oregon Coast. The historical disturbance regime in the central Oregon Coast Range was dominated by a mixture of high and low-severity fires, with a natural rotation of approximately 271 years. Old-growth forest coverage in the Oregon Coast Range varied from 25 to 75% during the past 3,000 years, with a mean of 47%, and never fell below 5% (Wimberly *et al.* 2000). Currently the Coast Range has approximately 5% old-growth, almost all of it on Federal lands. The dominant disturbance now is logging on a cycle of approximately 30 to 100 years, with fires suppressed.

The State of Oregon (2005) completed an assessment of habitat conditions in the range of OC coho in 2005. Oregon's assessment mapped how streams with high intrinsic potential for coho salmon rearing are distributed by land ownership categories. Agricultural lands and private industrial forests have by far the highest percentage of land ownership in high intrinsic potential areas and along all coho stream miles. Federal lands have only about 20% of coho stream miles and 10% of high intrinsic potential stream reaches. Because of this distribution, activities in lowland agricultural areas are particularly important to the conservation of Oregon coastal coho.

The coho assessment concluded that at the scale of the entire domain, pools are generally abundant, although slow-water and off-channel habitat (which are important refugia for coho during high winter flows) are limited in the majority of streams when compared to reference streams in minimally-disturbed areas. Amounts of large wood in streams are low in all four ODFW monitoring areas and land-use types relative to reference conditions. Amounts of fine sediment are high in three of the four monitoring areas, and were comparable to reference conditions only on public lands. Approximately 62 to 91% of tidal wetland acres (depending on estimation procedures) have been lost for functionally and potentially independent populations of coho.

As part of the coastal coho assessment, the Oregon Department of Environmental Quality (ODEQ) analyzed the status and trends of water quality in the range of OC coho using the Oregon water quality index, which is based on a combination of temperature, dissolved oxygen, biological oxygen demand, pH, total solids, nitrogen, total phosphates, and bacteria. Using the index at the species scale, 42% of monitored sites had excellent to good water quality, and 29% show poor to very poor water quality. Within the four monitoring areas, the North Coast had the best overall conditions (three sites in excellent or good condition out of nine sites), and the Mid-South coast had the poorest conditions (no excellent condition sites, and only two out of eight sites in good condition). For the 10-year period monitored between 1992 and 2002, no sites showed a declining trend in water quality. The area with the most improving trends was the North Coast, where 66% of the sites (six out of nine) had a significant improvement in index scores. The Umpqua River basin, with one out of nine sites (11%) showing an improving trend, had the lowest number of improving sites.

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Southern Oregon. Many large and small rivers supporting significant populations of coho salmon flow through this area, including the Elk, Rogue, Chetco, Smith and Klamath. The following summary of critical habitat information in the Elk, Rogue, and Chetco rivers is also applicable to habitat characteristics and limiting factors in other basins in this area. The Elk River flows through Curry County, and drains approximately 92 square miles (or 58,678 acres) (Maguire 2001). Historical logging, mining, and road building have degraded stream and riparian habitats in the Elk River basin. Limiting factors identified for salmon and steelhead production in this basin include sparse riparian cover, especially in the lower reaches, excessive fine sediment, high water temperatures, and noxious weed invasions (Maguire 2001).

The Rogue River drains approximately 5,160 square miles within Curry, Jackson and Josephine counties in southwest Oregon. The mainstem is about 200 miles long and traverses the coastal mountain range into the Cascades. The Rogue River estuary has been modified from its historical condition. Jetties were built by the Corps in 1960, which stabilized and deepened the mouth of the river. A dike that extends from the south shore near Highway 101 to the south jetty was completed in 1973. This dike created a backwater for the large shallow area that existed here, which has been developed into a boat basin and marina, eliminating most of the tidal marsh.

The quantity of estuary habitat is naturally limited in the Rogue River. The Rogue River has a drainage area of 5,160 square miles, but the estuary at 1,880 acres is one of the smallest in Oregon. Between 1960 and 1972, approximately 13 acres of intertidal and 14 acres of subtidal land were filled in to build the boat basin dike, the marina, north shore riprap and the other north shore developments (Hicks 2005). Jetties constructed in 1960 to stabilize the mouth of the river and prevent shoaling have altered the Rogue River, which historically formed a sill during summer months (Hicks 2005).

The Lower Rogue Watershed Council's watershed analysis (Hicks 2005) lists factors limiting fish production in tributaries to Lower Rogue River watershed. The list includes water temperatures, low stream flows, riparian forest conditions, fish passage and over-wintering habitat. Limiting factors identified for the Upper Rogue River basin include fish passage barriers, high water temperatures, insufficient water quantity, lack of large wood, low habitat complexity, and excessive fine sediment (Rogue Basin Coordinating Council 2006).

The Chetco River estuary has been significantly modified from its historical condition. Jetties were constructed by the Corps in 1957, which stabilized and deepened the mouth of the river. These jetties have greatly altered the mouth of the Chetco River and how the estuary functions as habitat for salmon migrating to the ocean. A boat basin and marina were built in the late 1950s and eliminated most of the functional tidal marsh. The structures eliminated shallow water habitats and vegetation in favor of banks stabilized with riprap. Since then, nearly all remaining bank habitat in the estuary has been stabilized with riprap. The factors limiting fish production in the Chetco River appear to be high water temperature caused by lack of shade, especially in tributaries, high rates of sedimentation due to roads, poor over-wintering habitat due to a lack of large wood in tributaries and the mainstem, and poor quality estuary habitat (Maguire 2001).

Summary of Environmental Baseline for Anadromous Fishes. Pacific salmon and steelhead, green sturgeon and eulachon are exposed to the impacts of a wide variety of past and

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present state, Federal or private actions and other human activities that comprise the action area, as well as Federal projects in this area that have already undergone formal section 7 consultation, and state or private actions that are contemporaneous with this consultation. Here we provide a review of major ESA section 7(a)(2) consultations where NMFS predicted effects would occur within in the action area.

The NMFS consulted on the effects of EPA's registration of pestidice products for chlorpyrifos, diazinon, and malathion (NMFS 2008); carbaryl, carbofuran, and methomyl (NMFS 2009); azinphos methyl, bensulide, dimethoate, disulfoton, ethoprop, fenamiphos, naled, methamidophos, methidathion, methyl parathion, phorate and phosmet (NMFS 2010); and 2,4-D, triclopyr BEE, diuron, linuron, captan, and chlorothalonil (NMFS 2011). These consultations concluded that registration of these pesticide products would jeopardize the continued existence of Pacific salmon and steelhead and/or result in the destruction or adverse modification of their critical habitats.

The NMFS consulted on the effects of fishery harvest actions, including 10-year terms of the Pacific Salmon Treaty (term of biological opinion from 2009-2018, NMFS 2008e) and the *United States v. Oregon* 2008 Management Agreement (term of biological opinion from 2008-2017; NMFS 2008f), and the Pacific Coast Salmon Plan fisheries (NMFS 2009a). In these past harvest opinions, NMFS characterized the short-term and long-term effects on reductions in Chinook abundance that occur during a specified year, and the long-term effects to whales that could result if harvest affected viability of the salmon stock over time by decreasing the number of fish that escape to spawn. The harvest biological opinions referenced above concluded that the harvest actions were not likely to jeopardize the continued existence of listed Chinook salmon.

The NMFS conducted additional consultations on the effects of hydro-power dams and flood control programs on all Columbia River basin salmon and steelhead, green sturgeon, and eulachon (NMFS 2008g, NMFS 2008h). As part of the proposed action for the Federal Columbia River Power System and the Willamette Flood Control Program, action agencies proposed funding hatchery programs in addition to their proposals for dam operations and maintenance. To mitigate for the harmful effects of hatchery production on long-term salmon and steelhead viability the action agencies committed to a schedule of future hatchery reforms.

2.5.2.4 Southern Resident Killer Whales

Prey Availability. Based on persuasive scientific information that the diet of Southern Residents is predominantly composed of Chinook salmon in inland waters (see further discussion in section 2.4.4), their diet may equally be predominantly composed of Chinook salmon when available in coastal waters of the action area. This analysis focuses on Chinook salmon abundance in coastal waters of the Southern Residents range. Focusing on Chinook salmon provides a conservative estimate of potential effects of the proposed action on Southern Residents because the total abundance of all salmon and other potential prey species is orders of magnitude larger than the total abundance of Chinook salmon.

When prey is scarce, whales likely spend more time foraging than when it is plentiful. Increased energy expenditure and prey limitation can cause nutritional stress. Nutritional stress is the

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condition of being unable to acquire adequate energy and nutrients from prey resources and as a chronic condition can lead to reduced body size and condition of individuals and lower birth and survival rates of a population. Ford *et al.* reported correlated declines in both the Southern Resident killer whales and Chinook salmon and suggested the potential for nutritional stress in the whales (Ford *et al.* 2005, Ford *et al.* 2010b). Food scarcity could also cause whales to draw on fat stores, mobilizing contaminants stored in their fat and potentially have the ability to alter thyroid homeostasis, reduce immune function, cause neurotoxicity, reproductive failure, and restrict the development and growth of the individual (see Table 9 in NMFS 2008a for a review of physiological effects resulting from exposure to toxic chemicals in marine mammals). Thus, nutritional stress may act synergistically with high contaminant burdens in the whales and result in contaminant-induced adverse health effects, higher mortality rates, or lower birth rates.

The availability of Chinook salmon to Southern Residents is affected by a number of natural and human actions. Climate effects from Pacific decadal oscillation and the El Nino/Southern oscillation conditions and events cause changes in ocean productivity which can affect natural mortality of salmon. Predation in the ocean also contributes to natural mortality of salmon. Salmonids are prey for pelagic fishes, birds, and marine mammals (including Southern Residents). Section 2.5 describes the baseline concentrations and sources (both natural and through human activities) of metal and elemental pollutants in Oregon waters and the potential adverse health effects to fish. Additional human activities and their impacts to salmon include land use such as logging, agriculture, ranching, hydroelectric power generation, mining, fishing, recreational activities, and urban uses (see section 2.5.2.5 above). Many of these activities have a federal nexus and have undergone section 7 consultation. Those actions have all met the standard of not jeopardizing the continued existence of the listed salmonids or adversely modifying their critical habitat, or if they did not meet that standard, we identified reasonable and prudent alternatives. Since the Southern Residents were listed, federal agencies have also consulted on impacts to the whales, including impacts to available prey. In addition, the environmental baseline is influenced by many actions that pre-date the salmonid listings and that have substantially degraded salmon habitat and lowered natural production of Chinook ESUs contemplated in this consultation.

Here we provide a review of Southern Resident killer whale determinations in previous ESA Section 7(a)(2) consultations where effects occurred in the action area, and where effects resulted in a significant reduction in available prey (*i.e.*, where prey reduction was likely to adversely affect or jeopardize the continued existence of the whales).

The NMFS consulted on the effects of fishery harvest actions on Southern Residents, including 10-year terms of the Pacific Salmon Treaty (term of biological opinion from 2009-2018, NMFS 2008e) and the *United States v. Oregon* 2008 Management Agreement (term of biological opinion from 2008-2017; NMFS 2008f), and the Pacific Coast Salmon Plan fisheries (NMFS 2009a). In these past harvest opinions, NMFS characterized the short-term and long-term effects on Southern Residents from prey reduction caused by harvest. We considered the short-term effects to whales resulting from reductions in Chinook abundance that occur during a specified year, and the long-term effects to whales that could result if harvest affected viability of the salmon stock over time by decreasing the number of fish that escape to spawn. These past analyses suggested that in the short term prey reductions were small relative to remaining prey

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available to the whales. In the long term, harvest actions have met the conservation objectives of harvested stocks, were not likely to appreciably reduce the survival or recovery of listed Chinook, and were therefore not likely to jeopardize the continued existence of listed Chinook. The harvest biological opinions referenced above concluded that the harvest actions cause prey reductions in a given year, but were not likely to jeopardize the continued existence of ESA-listed Chinook salmon or Southern Residents. New information about the relationship between Chinook salmon abundance and Southern Resident killer whale population growth is currently under scientific review and will inform future consultations and NMFS consideration of these previous conclusions.

NMFS also consulted on the effects of the long-term operations of the Central Valley Project (CVP) and State Water Project (SWP) (2008/09022). The NMFS found that the long-term operations of the CVP and SWP, as proposed, were likely to jeopardize the continued existence of Sacramento River winter-run Chinook salmon, Central Valley spring-run Chinook salmon, Central Valley steelhead, Southern DPS of North American green sturgeon, and Southern Resident killer whales. The increased risk of extinction of the winter- and spring-run Chinook salmon as a long-term consequence of the proposed action diminished the potential for Southern Residents to survive and recover. The involved action agencies are implementing actions identified as part of the reasonable and prudent alternative over specified time periods starting from issuance of the biological opinion.

NMFS conducted additional consultations on the effects of hydro-power dams and flood control programs on Southern Residents (NMFS 2008g, NMFS 2008h). As part of the proposed action for the Federal Columbia River Power System and the Willamette Flood Control Program, action agencies proposed funding hatchery programs in addition to their proposals for dam operations and maintenance. For both programs, the proposed actions did not result in a net decrease in Chinook salmon prey for Southern Residents in the short term. To mitigate for the harmful effects of hatchery production on long-term Chinook salmon viability (and thus killer whale prey availability) the action agencies committed to a schedule of future hatchery reforms.

Quality of Prey. As introduced in the above sections, contaminants enter marine waters from numerous sources throughout the action area, but are typically concentrated near populated areas of high human activity and industrialization. The majority of growth in salmon occurs while feeding in saltwater (Quinn 2005). Therefore, the majority (> 96 percent) of persistent pollutants in adult salmon are accumulated while feeding in the marine environment (Cullon *et al.* 2009, O'Neill and West 2009). Freshwater contamination is also a concern because it may contaminate salmon that are later consumed by the whales in marine waters. Only limited information is available for contaminant levels of Chinook in Oregon rivers; however, in general Chinook salmon contain higher levels of some contaminants than other salmon species (See Table 2.4.4.5 in the Status of the Species). As discussed in the Status of the Species, the marine distribution is an important factor affecting pollutant accumulation as is evident across the different salmon populations. For example, Chinook populations feeding in close proximity to land-based sources of contaminants have higher concentrations (O'Neill *et al.* 2006).

Vessel Activity and Sound. Commercial, military, recreational and fishing vessels traverse the coastal range of Southern Residents. Vessels may affect foraging efficiency, communication, and/or energy expenditure by their physical presence and by creating

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underwater sound (Williams *et al.* 2006, Holt 2008). Collisions of killer whales with vessels are rare, but remain a potential source of serious injury and mortality. Large ships that traverse coastal waters of the whales' range move at relatively slow speeds and are likely detected and avoided by Southern Residents.

Vessel sounds in coastal waters are most likely from large ships, tankers and tugs. Sound generated by large vessels is a source of low frequency (5 to 500 Hz) human-generated sound in the world's oceans (National Research Council 2003). While larger ships generate some broadband noise in the hearing range of whales, the majority of energy is below their peak hearing sensitivity. At close range large vessels can still be a significant source of background noise at frequencies important to the whales (Holt 2008). Commercial sonar systems designed for fish finding, depth sounding, and sub-bottom profiling are widely used on recreational and commercial vessels and are often characterized by high operating frequencies, low power, narrow beam patterns, and short pulse length (National Research Council 2003). Frequencies fall between 1 and 500 kHz, which is within the hearing range of some marine mammals, including killer whales, and may have masking effects.

Non-Vessel Sound. Anthropogenic (human-generated) sound in the range of Southern Residents is generated by other sources besides vessels, including oil and gas exploration, construction activities, and military operations. Natural sounds in the marine environment include wind, waves, surf noise, precipitation, thunder, and biological noise from other marine species. The intensity and persistence of certain sounds (both natural and anthropogenic) in the vicinity of marine mammals vary by time and location and have the potential to interfere with important biological functions (*e.g.*, hearing, echolocation, communication).

In-water construction activities are permitted by the Corps under section 404 of the CWA and section 10 of the Rivers and Harbors Act of 1899 and by the State of Washington under its Hydraulic Project Approval program. Consultations on these permits have been conducted and conservation measures have been included to minimize or eliminate potential effects of in-water activities, such as pile driving, on marine mammals. Military sonar also has the potential to disturb killer whales.

Oil Spills. Oil spills have occurred in the coastal range of Southern Residents in the past, and there is potential for spills in the future. Oil can be discharged into the marine environment in any number of ways, including shipping accidents, at refineries and associated production facilities, and pipelines. The magnitude of risk posed by oil discharges in the action area is difficult to precisely quantify, but improvements in oil spill prevention procedures since the 1980s likely provide some reduced risk of spill. New oil spill prevention procedures in the state of Washington likely positively contribute to the decrease in spill volume (WDOE 2007).

In marine mammals, acute exposure to petroleum products can cause changes in behavior and reduced activity, inflammation of the mucous membranes, lung congestion, pneumonia, liver disorders, neurological damage (Geraci and St. Aubin 1990), potentially death, and long-term effects on population viability (Matkin *et al.* 2008). In addition, oil spills have the potential to adversely impact habitat and prey populations, and, therefore, may adversely affect Southern Residents by reducing food availability.

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Scientific Research. Although research activities are typically conducted between May and October in inland waters, some permits include authorization to conduct research in coastal waters. In general, the primary objective of this research is population monitoring or data gathering for behavioral and ecological studies. In 2006, NMFS issued scientific research permits to seven investigators who intend to study Southern Residents (NMFS 2006). Additionally in 2008, NMFS issued another scientific permit to one investigator intending to study Southern Residents (NMFS 2008i). In the biological opinions NMFS prepared to assess the impact of issuing the permits, we determined that the effects of these disturbances on Southern Residents were likely to adversely affect, but not likely to jeopardize the continued existence of, the Southern Residents (NMFS 2006, 2008i). A small portion of the authorized take would occur in the coastal range of Southern Residents.

Summary of Southern Residents Environmental Baseline. Southern Residents are exposed to a wide variety of past and present state, Federal or private actions and other human activities in the coastal waters that comprise the action area, as well as Federal projects in this area that have already undergone formal section 7 consultation, and state or private actions that are contemporaneous with this consultation. All of the activities discussed in the above section are likely to have some level of impact on Southern Residents when they are in the action area.

No single threat has been directly linked to or identified as the cause of the recent decline of the Southern Residents, although the three primary threats are identified as prey availability, environmental contaminants, and vessel effects and sound (Krahn *et al.* 2002). Researchers are unsure about which threats are most significant. There is limited information on how these factors or additional unknown factors may be affecting Southern Residents when in coastal waters. For reasons discussed earlier, it is possible that two or more of these factors may act together to harm the whales. The small size of the population increases the level of concern about all of these risks (NMFS 2008a).

2.6 Effects of the Action

'Effects of the action' means the direct and indirect effects of an action on the species or critical habitat, together with the effects of other activities that are interrelated or interdependent with that action, that will be added to the environmental baseline (50 CFR 402.02).

EPA's approval of Oregon's revised water quality standards would have no direct effects to listed species or their habitat—that is, approving new water quality standards, by itself, will not directly affect listed species or designated critical habitat, or change the environmental baseline. However, there are significant indirect effects of approving the standards, because the approval allows the state to implement the standards. The analysis of effects of the proposed action assumes that the species of interest are exposed to waters meeting the water quality standards; however, there are many waters in Oregon that do not meet the current standards and would not meet the proposed standards. Implementation and attainment of the standards are key to improving the state's water quality, however, the only action under consideration in this consultation is EPA's proposed approval of Oregon's revised standards.

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2.6.1 Issues Common to All Criteria

The following discussion on acute and chronic toxicity data focuses on issues applicable to the development of all aquatic life criteria, and provides context for the toxicity data analyses on individual compounds provided in this section of the opinion.

Acute Toxicity Data. The acute criteria for aquatic life have been primarily based on compilations of toxicity study results reported in terms of the concentration resulting in 50 percent mortality over a fixed time period [usually 96 hours: *e.g.*, LC_{50}, effects concentration $(EC)_{50}$, EPA 1986a] using EPA's *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (Stephan *et al.* 1985) (Guidelines). Although there are a number of reasons why data are not included in the data sets used to develop criteria, some of the more common ones are that one or more pieces of information regarding study methodology or calculation of results needed to assess the reliability of the study is missing; data quality of the study is less than acceptable (*e.g.* unacceptably high control mortality); the test species was exposed to a chemical mixture or was previously exposed to the test chemical; the study reported effects on an endpoint other than survival, reproduction or growth; or the test duration was a non-standard test duration (*e.g.*, fish toxicity test reporting a 24-hr LC_{50} instead of the more standard 96-hr LC_{50}).

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, that indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias the magnitude of acute toxic effects. Theses factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that are protective against acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve, and challenge the notion that LC_{50} data that is above the acute criterion is protective against acute toxic effects based soley on a comparison of concentrations.

Acute water quality criteria are calculated by rank ordering the genus mean acute value (GMAV) values from the lowest LC_{50} to the highest LC_{50}, and using a formula given in Stephan *et al.* (1985) to estimate the 5th percentile of the resulting species sentitive distribution (SSD). This 5th percentile of measured GMAVs is termed the (final acute value) FAV in the EPA criteria development documents. As a criterion based on a concentration causing mortality to 50 percent of a test species would not be a protective criterion, EPA divides the FAV by a safety factor of 2.27 (rounded to a factor of 2 in the below analysis) to convert LC_{50} values into concentrations that EPA projects to be near or below lethality.

The database from which the safety factor was derived was published in the Federal Register in 1978. Table 10 from the Federal Register notice (43 FR 21506-21518) lumps data for freshwater and marine fish and invertebrates. The data are broken out by the chemicals tested. There are 219

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data points, but a large proportion of them aren't for a specific chemical, but rather for whole effluents of various sources—115 of the 219 data points used to derive the acute adjustment factor are based on effluent studies where individual pollutants are not measured. Interestingly, effluent studies are one of EPA's "not pertinent" or "reject" categories identified in EPA (2005).

The assumption that dividing an LC_{50} by 2 will result in effect concentrations near or below leathility rests on further assumptions of the steepness of the concentration-response slope. Several examples of tests with metals which had a range of response slopes are shown in Figure 2.6.1.1. These examples were selected from data sets that were relevant to salmonid species in Oregon and for which the necessary data to evaluate the range of responses could be located (Chapman 1975, 1978b, Marr *et al.* 1995, Marr *et al.* 1999, Mebane *et al.* 2010, Windward 2002). The citations given include both reports with detailed original data as well as the summarized, published forms of the same tests. The examples range from tests with some of the shallowest concentration-response slopes located to very steep response slopes. In the shallowest tests (panels A and E), an $LC_{50/2}$ concentration would still result in 15 to 20 percent mortality.

One challenge for deriving acute criteria for short-term exposures is that the great majority of available data is for mortality; that is, a concentration that kills 50 percent of a test population. A fundamental assumption of EPA's criteria derivation is that the FAV, which is the LC_{50} for a hypothetical species with a sensitivity equal to the 5th percentile of the SSD, may be divided by 2 in order to extrapolates from a concentration that would likely be extremely harmful to sensitive species in short-term exposures (*i.e.*, kill 50 percent of the population) to a concentration expected to kill few, if any, individuals. This assumption must be met for acute criteria to be protective of sensitive species. It is difficult to evaluate from published literature if this assumption is met because so few studies report the data behind an LC_{50} test statistic. While LC_{50}s are almost universally used in reporting short-term toxicity testing, they are not something that can be "measured," but are statistical model fits. An acute toxicity test is actually a series of 4 to 6 tests runs in parallel in order to test effects at these (usually) four to six different chemical concentrations. An LC_{50} is estimated by some statistical distribution or regression model, which generates an LC_{50} estimate, and some confidence interval, and then all other information is thrown away. Thus, while the original test data included valuable information on what were no, low and severe effects concentrations, that information is lost to reviewers unless the unpublished, raw, lab data are available. However, a more common pattern with the metals data was that an $LC_{50/2}$ concentration would probably result in about a 5 percent death rate (panels B and F), and in many instances, no deaths at all would be expected (panels C and D).

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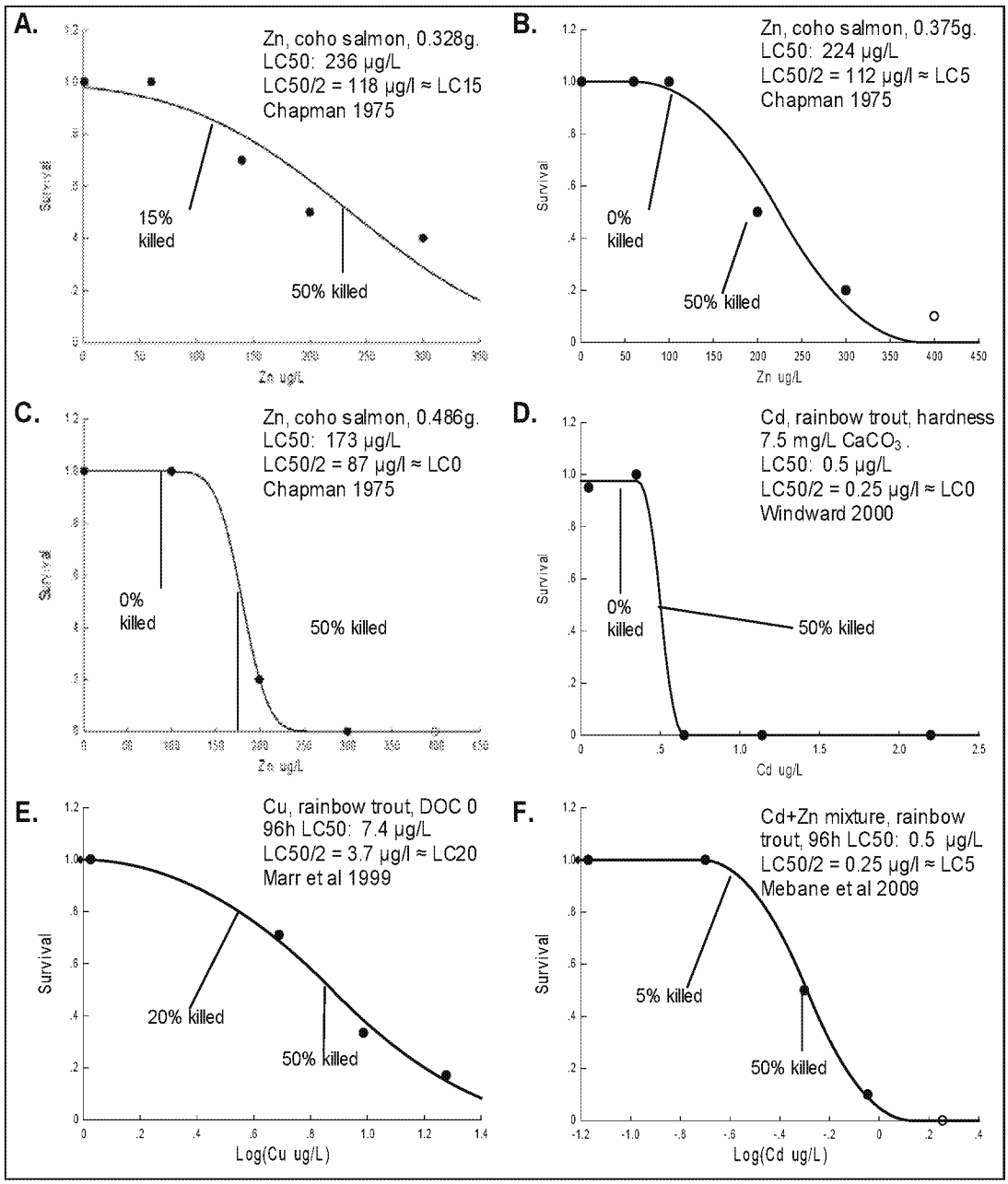

Figure 2.6.1.1 Examples of percentages of coho salmon or rainbow trout killed at one-half their LC_{50} concentrations and at LC_{50} concentrations with cadmium, copper, and zinc.

In one of the few additional published sources that gave relevant information, researchers happened to include effect-by-concentration information on the acute toxicity of chemical mixtures. Rainbow trout and the invertebrate zooplankton *Ceriodaphnia dubia* were exposed for 96 and 48 hours respectively to mixture of six metals, each at their presumptively "safe" acute CMC concentrations. In combination, the CMC concentrations killed 100% of rainbow trout and C. dubia, but 50% of the CMC concentrations killed none (Spehar and Fiandt 1986). This gives some support to the assumption that one-half the FAV divided by 2 is likely to kill a low

percentage of fish, although it raises questions about the overall protectiveness of criteria concentrations in mixtures.

Other relevant reviews include Dwyer *et al.* (2005b), who evaluated the $LC_{50/2}$ assumption with the results of the acute toxicity testing of 20 species with five chemicals representing a broad range of toxic modes of action. In those data, multiplying the LC_{50} by a factor of 0.56 resulted in a low (10%) or no-acute effect concentration. Testing with cutthroat trout and Cd, Pb, and Zn singly and in mixtures, Dillon and Mebane (2002) found that the $LC_{50/2}$ concentration corresponded with death rates of 0 to 15 percent.

Summary: Based on this analysis, acute criteria based on LC_{50} concentrations and the acute adjustment factor, instead of acute criteria that are based on an exposure-response curve, are likely to underestimate the magnitude of effects for field-exposed fishes. Therefore, the shortcomings identified in the above analysis are likely to result in mortality greater than the LC_{50} test predictions and the presumed protection from the acute adjustment factor in deriving acute criteria.

Chronic Toxicity Data. While the Guidelines give a great deal of advice on considerations for evaluating chronic or sublethal data (Stephan *et al.* 1985, at p. 39), those considerations were not usually reflected in the individual national EPA-recommended ambient water quality criteria documents NMFS reviewed. In practice, for most of the criteria documents we reviewed, "chronic values" were simply calculated as the geometric mean of the lowest tested concentration that had a statistically significant adverse effect at the 95 percent confidence level (LOEC), and the next lower tested concentration (NOEC). The "chronic value" as used in individual criteria documents is effectively the same thing as the maximum acceptable toxicant concentration[6] (MATC) used in much environmental toxicology literature, even though the MATC term is never used in the Guidelines. This MATC approach has the potential to seriously underestimate effects because the statistical power in typical toxicity tests is fairly low. A bias in many ecotoxicology papers is to focus on avoiding "false accusations" of a chemical with 95 percent accuracy (*i.e.*, Type I error or false positive, the risk of declaring an effect was present when in fact there was no effect). Often no consideration whatsoever is given to the companion problem, known as Type II error, or false negatives (*i.e.*, declaring no adverse effects occurred when in fact they did occur, but because of the limited sample size or variability, they were not significant with 95 percent confidence).

The magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be large (greater than 30 percent on average for some endpoints), and much higher for individual tests (Crane and Newman 2000). This problem is compounded when the "chronic value" or MATC is calculated in its most common form as the geometric mean of a NOEC and LOEC. For instance, in one study, 100 percent of juvenile brook died after being exposed to 17 µg/L copper for 8 months; this was considered the LOEC for the test. The next lowest concentration tested (9.5 µg/L) had no reduced survival relative to controls. (McKim and Benoit 1971). Therefore, the only thing that can be said about the geometric mean of these two effect concentrations (*i.e.*, the chronic value of 12.8 µg/L that was used in the chronic copper criteria, EPA 1985) is that it represents a concentration that can be expected to kill somewhere between

[6] The MATC is the range between the NOEC and LOEC.

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all and no brook trout in the test population. These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that are protective against chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

Suter *et al.* (1987) evaluated published chronic tests with fish for a variety of chemicals and found that, on average, the MATC represented about a 20 percent death rate and a 40% reduction in fecundity. They noted that "although the MATC is often considered to be the threshold for effects on fish populations, it does not constitute a threshold or even a negligible level of effect in most of the published chronic tests. It corresponds to a highly variable level of effect that can only be said to fall between 0 and 90 percent." Barnthouse *et al.* (1989) further extrapolated MATC-level effects to population-level effects using fisheries sustainability models and found that the MATC systematically undervalued test responses such as fecundity, which are both highly sensitive and highly variable.

One implication of this issue is that because the MATC chronic values typically used in the EPA water quality criteria documents for aquatic life criteria may cause a substantial adverse effect for that test species, the criteria on the whole will be less protective than the Guidelines' intended goal of protecting 95 percent of the species. How much less protective is unclear and probably varies among the criteria datasets. One dataset from which a hypothetical NOEC-based chronic criterion could readily be recalculated and compared with the usual MATC criteria was a 2006 cadmium criteria update (Mebane 2006). In this comparison, Mebane determined that the MATC-based chronic criteria would protect about 92 percent of the aquatic species in the dataset at the NOEC level. Because the NOEC statistic also can reflect a fairly sizable effect (Crane and Newman 2000) it may be that at least with cadmium, the true level of protection is closer to about 90 percent than the 95 percent intended by the guidelines.

Summary: Based on this analysis, chronic criteria based on hypothesis tests, instead of acute criteria that are based on an exposure-response curve, are likely to underestimate the magnitude of effects for field-exposed fishes. Therefore, the shortcomings identified in the above analysis are likely to result in sublethal greater than the NOEC/LOEC predictions.

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2.6.2 Freshwater Criteria Toxicity Analysis

The ESA directs that section 7 consultations use the best available scientific and commercial data. While EPA conducted an extensive data call and has developed a large database of toxicity (ECOTOX), thousands of toxicity studies were rejected by EPA for use in criteria development and formulation of the BE. A majority of these toxicity studies were rejected because the test duration was non-standard; EPA generally does not consider toxicity tests with non-standard durations (*e.g.*, 4-hr LC_{50} or 192-hr LC_{50}), or endpoint, *e.g.*, behavioral. However, these studies may still meet the standard of the "best available scientific data" as defined by the ESA and, as warranted, were intergrated into the analysis in this opinion.

NMFS also examined EPA's BE effects assessment methodology, but NMFS did not use the EPA effects assessment methodology or the analysis in the BE for its effects analysis as it included too many fundamental problems NMFS identified during preconsultation that EPA did not address in the BE submitted to NMFS. These problems include:

- LC_{50} toxicity data interpretation and application
- NOEC toxicity data interpretation and application
- Exclusion of published toxicity data in the BE analysis
- High uncertainty with use of the acute adjustment factor
- Lack of a sublethal effects analysis
- Lack of a chemical mixture analysis
- Scale of effect determinations—effects of the action as a whole verses effects based on individual criterion

Instead, NMFS used a much more extensive toxicity data set, including toxicity studies from the ECOTOX database that were excluded by EPA, for its analysis, and included an extensive sublethal effects analysis for each compound (where data was available), a chemical mixtures analysis, a direct mortality and population model for the freshwater acute criteria, and a synthesis of effects of the action as a whole.

In this opinion, NMFS also examined EPA's *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (Stephan *et al.* 1985), as it forms the basis for how EPA derives aquatic life criteria. That analysis is provided in Apprendix 1 of this opinion.

The analysis on freshwater criteria starts with a review of the chemical and toxicological concepts, principals, and factors that influence toxicity for each compound, and an assessment of critical exposure-response factors pertinent to the overall analysis. The data analysis in this section has five general components: (1) Available toxicity data presented in table format by endpoint; (2) a summary statistical analysis performed for each endpoint data set consisting of the arithmetic mean, the geometric mean, and the harmonic mean to assess the distribution of the data for each data set, and the statistical analysis is used later in the analysis on chemical mixtures; (3) a relative mortality analysis for the acute criteria; (4) a sublethal effects analysis on the chronic criteria, and (5) an analysis on food items (when data was available).

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The toxicity data for salmonid fishes includes data for listed and non-listed salmonid fishes, *e.g.*, rainbow trout are used to directly assess toxicity effects on steelhead as the resident form is indistinguishable from the anadromous form in juvenile life stages. Other salmonid fishes, *e.g.*, brook trout (*Salvelinus fontinalis*) and cutthroat trout (*Oncorhynchus clarki*), are used in addition to the species-specific toxicity data and/or as a surrogate for listed species where toxicity data is not available for listed species to analyze effects on additional endpoints. Our analysis of surrogate species toxicity data showed no difference in the range of concentrations when compared to the toxicity data for listed species. Furthermore, toxicity data for green sturgeon and eulachon was limited or non-existent for most of the compounds in Table 1.1. Therefore, NMFS used the salmonid fishes toxicity data as a surrogate for these two species, as salmonid fishes were the closest taxonomic group for which data were available.

The effects analysis on Southern Resident killer whales follows the analysis on salmon, steelhead, green sturgeon, and eulachon as the Southern Resident killer whale effects analysis is dependent upon the effects analysis and conclusions on salmon and steelhead addressed in this opinion

The summary conclusions provided in this section are based on an analysis of toxicity exposure-response potential for each listed species considered in this opinion and for each freshwater compound listed in Table 1.1. The NMFS based these analyses exclusively on an examination of the available toxicity data from exposure to a single compound. The NMFS also rated the magnitude of effects for each endpoint. The NMFS used a scale of *low intensity* increase in toxicity effects on listed species at the scale of individuals or groups of individuals, *moderate intensity* increase in toxicity effects on listed species at the scale of individuals or groups of individuals, *moderately-high-intensity* increase in toxicity effects on listed species at the scale of individuals or groups of individuals, but not at the scale of any population, and *high-intensity* increase in toxicity effects on listed species that affects one or more population attribute as a means to qualitatively assess the magnitude of acute or chronic toxics effects associated with the toxicity data. The summary conclusions do not take into account effects to the listed species considered in this opinion from exposure to multiple compounds. The issue of chemical mixtures, as well as criteria development issues, direct mortality population modeling, *etc.*, are examined in the *Integration and Synthesis*.

Toxicity Data Sources

The following is a list of data sources used in this opinion.

Data Set ECOTOX — all data are from ECOTOX and were provided to NMFS by EPA. The first data set provided to NMFS by EPA only included the rank ordered LC_{50} data and ranked ordered NOEC data. The NMFS also requested EPA provide the core data files for the compounds subject to this consultation, which were provided to NMFS. The core data files contain all toxicity data available in ECOTOX for the subject conmpounds at the time of the data requests. The EPA only used the rank ordered data for the analysis in their BE. On the other hand, NMFS used the core data files for its analysis in this opinion. Additionally, NMFS made several data requests to EPA for the reference sources listed in the core data files. The EPA only provided NMFS with the reference sources for the rank ordered data and did not provide the

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reference sources for the core data files. The NMFS cross-walked the rank ordered data with the references sources for data quality assurance. For the remainder of the core data, NMFS relied on the toxicity data as provided by EPA in the core data files. Reference sources for the ECOTOX data used in this opinion are provided in Appendix 2.

ECOTOX data selection: EPA used the concentration mean values (geometric mean) for the analysis in their BE. The NMFS used either the concentration mean value (geometric mean), the concentration minimum value (lower 95th percentile confidence interval), or the concentration maximum value (upper 95th percentile confidence interval). The NMFS also used statistically determined toxicity data, *e.g.*, LC_{50} values, as many toxicity tests results are based on a regression analysis. When available, NMFS selected the concentration minimum value, *i.e.*, lower 95th percentile confidence interval of the LC_{50}, as it is the best available statistical estimate of the actual reported LC_{50} value (in order to assess the uncertainty of the LC_{50} value as LC_{50} endpoints typically do not indicate the point at which listed fish could be killed or harmed) for a particular chemical-species combination and therefore represents the best available science in evaluating potential effects.

For the ECOTOX data set, the life stage (organism comment) information in each of the criterion-specific tables can be found in the ECOTOX code list document (EPA 2008).

Data Set 2 — all data indentified in tables with "Data Set 2" are from the NMFS' biological opinion (draft) for the proposed approval of Idaho's water quality criteria for toxic substances.

Data Set 3 — all data indentified in tables with "Data Set 3" are from NOAA Technical memorandums.

Data Set 4 — all data indentified in tables with "Data Set 4" are from the toxicity data for sturgeon (Section 4, Literature Cited).

Data Set BE — all data indentified in tables with "Data Set BE" are from the BE (saltwater data for cadmium, arsenic, heptachlor epoxide, nickel, pentachlorophenol, and lead).

Other data sources used in the opinion are cited directly in the text (Section 4, Literature Cited). The tables in section 2.6.2 and 2.6.3 provide information on compound concentration, life stage and exposure duration.

2.6.2.1 Organic Pollutants: Analysis of Individual Compounds

In this section, we identify the effects of each compound listed in Table 1.1 , and compare the proposed criteria with available toxicity data. The analysis identifies the potential effects on listed species and their critical habitats of each of the criteria that we would expect to occur if water concentrations were equal to the proposed criteria. Where possible, we also identify sublethal effects, effects related to bioaccumulation, and effects on the food sources of listed species.

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Organic Pollutants—Toxicity and Exposure

Eisler's series of synoptic reviews (1970), EPA's criteria documents, and the World Health Organization's environmental health criteria documents (*e.g.*, WHO 1984) were used to provide the following summary of sources, pathways, and toxic effects of organic pollutants. Most of the organic compounds considered in the proposed action are organochlorine pesticides (*e.g.*, dieldrin, lindane, heptachlor), used in the past for a variety of agricultural applications, as well as for controlling insects considered hazardous to human health. The remainder are industrial chemicals (*e.g.*, PCP, TBT) that have been used widely in the past but are now banned or restricted in the United States. Of the organic contaminants included in the proposed action, only lindane, endosulfan, heptachlor, and pentachlorophenol are still used at all United States, and permitted applications for lindane and heptachlor are very limited. They generally enter the aquatic environment attached to organic and inorganic particulate matter. However, because they are not highly water soluble and persistent in the environment, they remain sequestered in sediments and provide a continual source of potential exposure. This is of particular relevance when contaminated streambed sediments are disturbed as part of in-channel work. Organic pollutants may also enter the aquatic environment through non-point surface runoff from contaminated agricultural areas where they have been used in the past. Although the levels of most of these compounds have declined since their use was banned in the 1970s, they are still widely distributed in the environment and found in tissues of aquatic organisms.

Organic contaminants are rarely found alone in discharges or in the environment. Usually, several compounds are found together in areas where there has been extensive agricultural or industrial activity. In industrialized areas, other classes of contaminants (such as metals or aromatic hydrocarbons from petroleum products). For instance, the chemical forms of most organic pesticides and PCBs are mixtures that may contain a large number of isomers and congeners of each compound, of which the toxicity and persistence in the environment can vary considerably.

The most direct exposure pathway for dissolved organic compounds to aquatic organisms is via the gills. Dissolved organic compounds are also taken up directly by bacteria, algae, plants, and planktonic and benthic invertebrates. Organic pollutants can also adsorb to particulate matter in the water column and enter organisms through various routes. Planktonic and benthic invertebrates can ingest particulate-bound organic compounds from the water column and sediments and then be eaten by other organisms. Thus, dietary exposure may be a significant source of organic toxic pollutants for aquatic and aquatic-dependent organisms.

Although organic contaminants bound to sediments are generally less bioavailable to organisms, they are nonetheless present, and changes in the environment (*e.g.*, dredging, storm events, temperature, lower water levels, biotic activity) can significantly alter their bioavailability. Feeding habits of fish can determine the amount of uptake of certain organic contaminants; for example, where piscivorous fish are exposed to different levels of organics than are omnivorous or herbivorous fish.

Organic pollutants can have a wide variety of effects on organisms. Exposure to organochlorines can result in damage to gut tissues, disrupt nervous system operation, and alter liver and kidney

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functions, and impair the immune system. Elevated concentrations of many organochlorine compounds can cause growth inhibition, impaired reproduction, and developmental defects that may affect not only the target organisms themselves, but can also impact the growth and survival of predator species farther up the food chain. A number of these compounds are promoters that increase the risk of cancer. They may also disrupt immune function and increase the affected animal's susceptibility to infectious disease. Impacts from organic contamination can shift species composition and abundance towards more pollution-tolerant species. For each of the organic pollutants, we analyze these effects in subsequent sections.

2.6.2.1.1 Dieldrin

Dieldrin Criteria. The proposed acute and chronic criteria for dieldrin are 0.24 µg/L and 0.056 µg/L, respectively.

Tables 2.6.2.1.1.1 through 2.6.2.1.1.6 report toxicity data from the ECOTOX database for freshwater dieldrin, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.1.1 LC_{50} toxicity data for salmonid fishes, eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		**Data Set ECOTOX**
Criterion Concentration Acute 0.24 Micrograms Liter^{-1}	**Temperature 7.4-12° Celsius**	**Arithmetic Mean 635**
Criterion Concentration Chronic 0.056 Micrograms Liter^{-1}	**Hardness 40-272 mg/L $CaCO_3$**	**Geometric Mean 27**
Endpoint/Effect LC_{50}/Mortality	**pH 7.1-7.54**	**Harmonic Mean 5**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.56	UNDERYEARLING	96H
0.9	1.4G	96H
1	0.8G	96H
1.1		
1.4		
1.6	UNDERYEARLING	72H
1.8	0.8G	96H
2	EARLY FRY, 77 D	96H
2.3	UNDERYEARLING	24H
2.4		
4.55	1.1G	96H
4.55	1.1G	96H
5.3	JUVENILE	96H

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Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **635**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **27**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **7.1-7.54**	**Harmonic Mean** **5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
5.3	JUVENILE	24H
6.1	51-114 MM, 1.45-5 G	96H
9.9	51-79 MM, 3.2 G	72H
9.9		
9.9	51-79 MM, 3.2 G	96H
9.9	51-79 MM, 3.2 G	72H
10	UNDERYEARLING	48H
10.8	57-76 MM, 2.7-4.1 G	96H
10.8		
10.8	57-76 MM, 2.7-4.1 G	96H
11.5	1.1G	96H
13	51-79 MM, 3.2 G	48H
14.4	57-76 MM, 2.7-4.1 G	96H
15.3	57-76 MM, 2.7-4.1 G	96H
15.7	51-79 MM, 3.2 G	24H
17.5	57-76 MM, 2.7-4.1 G	96H
20	FINGERLING, 50.8 MM, 1.71 G	24H
20	FINGERLING, 52.6 MM, 1.87 G	96H
50	FINGERLING, 51.8 MM, 1.85 G	96H
50	FINGERLING, 50.8 MM, 1.71 G	96H
50	FINGERLING, 52.6 MM, 1.87 G	96H
50	FINGERLING, 51.8 MM, 1.85 G	24H
50	FINGERLING, 51.8 MM, 1.85 G	96H
98.4	SPERM	96H
100	FINGERLING, 53.1 MM, 1.86 G	24H
100	FINGERLING, 49.3 MM, 1.52 G	24H
100	FINGERLING, 49.2 MM, 1.55 G	96H
100	FINGERLING, 49.2 MM, 1.55 G	96H
100	FINGERLING, 49.2 MM, 1.55 G	24H
100	FINGERLING, 53.1 MM, 1.86 G	72H
100	FINGERLING, 53.1 MM, 1.86 G	48H
250	FINGERLING, 47.4 MM, 1.31 G	12D
250	FINGERLING, 50.4 MM, 1.64 G	24H

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Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute 0.24 Micrograms Liter^{-1}	**Temperature 7.4-12° Celsius**	**Arithmetic Mean 635**
Criterion Concentration Chronic 0.056 Micrograms Liter^{-1}	**Hardness 40-272 mg/L $CaCO_3$**	**Geometric Mean 27**
Endpoint/Effect LC_{50}/Mortality	**pH 7.1-7.54**	**Harmonic Mean 5**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
250	FINGERLING, 53.4 MM, 1.94 G	96H
250	FINGERLING, 50.4 MM, 1.64 G	96H
250	FINGERLING, 53.4 MM, 1.94 G	96H
500	FINGERLING, 52.5 MM, 1.91 G	24H
500	FINGERLING, 51.5 MM, 1.87 G	48H
1000	FINGERLING, 54.7 MM, 2.02 G	96H
1000	FINGERLING, 52.7 MM, 1.89 G	24H
10000	5-10 CM	96H
10000	5-10 CM	96H
10000	5-10 CM	96H

Table 2.6.2.1.1.2 Mortality toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute 0.24 Micrograms Liter^{-1}	**Temperature 7.4-12° Celsius**	**Arithmetic Mean 2509**
Criterion Concentration Chronic 0.056 Micrograms Liter^{-1}	**Hardness 40-272 mg/L $CaCO_3$**	**Geometric Mean 54**
Endpoint/Effect Mortality	**pH 7.1-7.54**	**Harmonic Mean 0.19**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.006	YEARLING, 29.5 G	24H
0.04	NR	24H
0.23	UNDERYEARLING	18D
0.55	NR	90D
0.9	1.4G	4H
0.91	NR	16H
0.97	NR	12H
1.3	0.8G	43D
1.8	0.8G	0.5H
2	EARLY FRY, 77 D	1D
2	6 MO, JUVENILE, 1.8 G	43D
3.3	0.8G	3.5H

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Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute **0.24 Micrograms Liter^{-1}**	**Temperature** **7.4-12° Celsius**	**Arithmetic Mean** **2509**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **40-272 mg/L $CaCO_3$**	**Geometric Mean** **54**
Endpoint/Effect **Mortality**	**pH** **7.1-7.54**	**Harmonic Mean** **0.19**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
6.1	51-114 MM, 1.45-5 G	12H
6.1	51-114 MM, 1.45-5 G	4H
6.4	JUVENILE	100D
6.7	51-114 MM, 1.45-5 G	4H
7.9	51-114 MM, 1.45-5 G	24H
9.4	0.8G	4H
43	ADULT, 175 G	1D
43	ADULT, 175 G	50D
100	JUVENILE, 1-1.5 YR	1D
125	JUVENILE, 1-1.5 YR	2D
250	JUVENILE, 1-1.5 YR	2D
250	JUVENILE, 1-1.5 YR	55D
250	JUVENILE, 1-1.5 YR	42D
250	JUVENILE, 1-1.5 YR	1D
500	FINGERLING, 7.6-10.2 CM	55D
1000	FINGERLING,7.6-10.2 CM	2D
5000	6 WK	30D
5000	6 WK	5D
5000	100-200 G	24H
10000	FERTILIZED EGG, 0 H	45D
10000	FERTILIZED EGG, 24 H	20D
10000	EARLY EYED EGG, 14 D	3D
10000	LATE-EYED EGG, 28 D	5D
10000	SAC FRY, 42 D	5D
10000	5-10 CM	12H
10000	5-10 CM	24H
10000	5-10 CM	4H

Table 2.6.2.1.1.3 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute 0.24 Micrograms Liter^{-1}	**Temperature 7.4-12° Celsius**	**Arithmetic Mean 0.3**
Criterion Concentration Chronic 0.056 Micrograms Liter^{-1}	**Hardness 40-272 mg/L $CaCO_3$**	**Geometric Mean 0.3**
Endpoint/Effect NOEC/Growth	**pH 7.1-7.54**	**Harmonic Mean 0.3**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.12		
0.55		90D

Table 2.6.2.1.1.4 Growth toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute 0.24 Micrograms Liter^{-1}	**Temperature 7.4-12° Celsius**	**Arithmetic Mean 0.4**
Criterion Concentration Chronic 0.056 Micrograms Liter^{-1}	**Hardness 40-272 mg/L $CaCO_3$**	**Geometric Mean 0.8**
Endpoint/Effect Growth	**pH 7.1-7.54**	**Harmonic Mean 0.09**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.04	7 MO, JUVENILE, 3.0-5.1 G	12M
0.087	7 MO, JUVENILE, 3.0-5.1 G	16W
0.19	6 MO, JUVENILE, 2.8 G	130D
1.2	1.4G	300D

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Table 2.6.2.1.1.5 Physiological toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute 0.24 Micrograms Liter^{-1}	**Temperature 7.4-12° Celsius**	**Arithmetic Mean 1.4**
Criterion Concentration Chronic 0.056 Micrograms Liter^{-1}	**Hardness 40-272 mg/L $CaCO_3$**	**Geometric Mean 0.8**
Endpoint/Effect Physiological	**pH 7.1-7.54**	**Harmonic Mean 0.2**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.04	7 MO, JUVENILE, 3.0-5.1 G	
1	0.8G	
1.3	0.8G	
2.2	0.8G	
2.3	0.8G	

Table 2.6.2.1.1.6 Reproductive toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater dieldrin.

Criterion Freshwater Dieldrin		Data Set ECOTOX
Criterion Concentration Acute 0.24 Micrograms Liter^{-1}	**Temperature 7.4-12° Celsius**	**Arithmetic Mean 7**
Criterion Concentration Chronic 0.056 Micrograms Liter^{-1}	**Hardness 40-272 mg/L $CaCO_3$**	**Geometric Mean 7**
Endpoint/Effect Reproductive	**pH 7.1-7.54**	**Harmonic Mean 7**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
7	JUVENILE	60MIN

Dieldrin Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and

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Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to dieldrin, NMFS added an additional step to its analysis for dieldrin to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.24 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.1.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.1.1, predicts a magnitude of

effect ranging from a low of an LC_{zero} at a concentration of 10,000 µg/L to a high of an LC_{21} at a concentration of 0.56 µg/L. In other words, the acute criterion of 0.24 µg/L has an equivalent toxicity potential predicted to kill zero percent to 21 percent, with a median toxicity potential of an $LC_{0.7}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for dieldrin, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for dieldrin, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Dieldrin is a synthetic cyclic chlorinated hydrocarbons called cyclodienes, and was used extensively in the 1950s and 1960s as a soil insecticide. At that time, dieldrin (and aldrin), were two of the most widely used domestic pesticides in the United States (EPA 1980a). However, the EPA cancelled the registration for both compounds in 1975 (Biddinger and Gloss 1984).

Once aldrin has been applied to any aerobic and biologically active soil, it rapidly undergoes a metabolic epoxidation reaction that converts it to dieldrin (EPA 1980a, and Wolfe and Seiber 1993). In fish, the epoxidation of aldrin to dieldrin occurs via a mixed-function oxidase system, which has been demonstrated in golden shiners, mosquitofish, green sunfish, bluegill sunfish and channel catfish (as cited in Chambers and Yarbrough 1976). Dieldrin can be further modified when exposed to sunlight, via cyclization to photodieldrin (Wolfe and Seiber 1993).

Dieldrin has extremely low volatility and low solubility in water. It is more environmentally stable than aldrin, and is probably the most stable of the cyclodiene insecticides (EPA 1980a, Wolfe and Seiber 1993). For this reason, dieldrin is more frequently observed in the environment than aldrin (Biddinger and Gloss 1984). One study, conducted on the environmental fate and transport of dieldrin in the Coralville Reservoir in eastern Iowa, revealed that 10% of the entire input of dieldrin into the reservoir was taken up by fish, 40% entered the sediment, and 50% was exported from the reservoir in the outflow. Moreover, of the portion of dieldrin that was present specifically in the water column, 74% occurred in fish, 25% was dissolved in water, and less than 1% was adsorbed to suspended solids (Schnoor 1981).

Acute toxicity of dieldrin reported in rainbow trout and other fish includes effects on cardiac muscles, as well as inhibition of oxygen uptake, the central respiratory center, bronchial muscles, and the central nervous system (Lunn *et al.* 1976). Aldrin and dieldrin are similarly toxic to fish, although aldrin is more toxic to cladocerans than dieldrin (EPA 1980a). Additionally, photodieldrin is more toxic than dieldrin (Wolfe and Seiber 1993).

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Because it is extremely a-polar, dieldrin that is present in fish has a particularly high affinity for fat. However, although it can be mobilized from tissue when the fish is placed in clean water, the dieldrin that has been eliminated then re-enters the water, making it available for subsequent uptake by other organisms (EPA 1980a). In channel catfish, approximately 50% of the dieldrin that had accumulated in dorsal muscle due to water-born exposure was eliminated after 14 days post-exposure, with total depuration by 28 days post-exposure. However, dieldrin that had accumulated in tissue due to dietary exposure was eliminated more slowly at 28 days post-exposure; approximately one third of the original dieldrin in muscle tissue was still present (Shannon 1977a). For rainbow trout, the predicted time to eliminate 50% of the dieldrin accumulated via dietary exposure is 40 days (Macek *et al.* 1970). In contrast, *Daphnia sp.* required four days to eliminate 50% of the photodieldrin that was accumulated in a water-born exposure study (Khan *et al.* 1975) and goldfish required less than 12 hours (Khan and Khan 1974). For the freshwater mussel *Lampsilis siliquoidea*, the half life of dieldrin was 4.7 days (Bedford and Zabik 1973). Khan and Khan (1974) noted that the initial elimination of dieldrin or photodieldrin from goldfish or *Daphnia* was due to excretion into the surrounding water.

A study by Van Leeuwen *et al.* (1985) examined the effects of water-borne dieldrin on rainbow trout at various early life stages, including fertilized eggs, early and late eye point eggs, sac fry and early fry. In the egg, the yolk acted as a temporary 'toxicant sink', but later in development, during the early sac fry stage, dieldrin was delivered from the yolk and began to accumulate in the fish tissue. The highest concentration in tissue was reached at the end of the sac fry stage. The second highest concentration in tissue was reached at the early fry stage, when susceptibility to dieldrin toxicity is most pronounced in early life stages.

The scope of the toxic properties of dieldrin is reinforced by the other studies reported above that involved other salmonid species for which lethality occurred at levels that were also below or slightly above the proposed acute criterion for dieldrin. Two of the trout studies (Van Leeuwen *et al.* 1985, Shubat and Curtis 1986) were more recent than the listed species studies. Also, two trout studies were done in flow-through experiments with measured dieldrin concentrations, which are likely to reflect more accurate estimates of toxicity than static experiments with nominal dieldrin concentrations (Chadwick and Shumway 1969, Shubat and Curtis 1986). The more recent and flow-through studies reported lethality concentrations that were below or near the proposed acute criterion for dieldrin, suggesting that this criterion could kill listed salmonid species.

Phillips and Buhler (1979) exposed fingerling rainbow trout to 0.18 µg/L dieldrin for 61 days under flow-through conditions and measured dieldrin concentrations. This resulted in a reduction in the rate of fat accumulation in fish that were fed a relatively high-fat diet (tubificid worms). Whole wet fish tissue concentration that corresponded to this effect was 0.82 or 1.32 mg/kg dieldrin. The effect of dieldrin exposure on fat accumulation was not apparent when fish were fed a relatively low fat diet (moist pellets), thus demonstrating that dieldrin toxicity can be affected by diet composition.

These limited results suggest that the proposed chronic criterion for dieldrin may avoid harming listed salmon subjected to short-term, water-borne exposure. However, they do not indicate whether the proposed chronic criterion is protective against bioaccumulation-related effects. To

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address this, several dietary exposure studies were evaluated that reported dieldrin tissue concentrations and chronic effects. If a specific chronic effect is associated with a specific tissue concentration and the BCF for dieldrin is known, then the tissue concentration and BCF can be used to back-calculate an estimate of the aqueous dieldrin exposure concentration resulting in an equivalent tissue concentration, and thus an equivalent chronic effect.

Two BCF values were identified: 1,700 whole body BCF for early fry rainbow trout (Van Leeuwen *et al.* 1985) and 8,875 whole body BCF for juvenile rainbow trout (calculated from Shubat and Curtis 1986). These BCF values are assumed to represent the low and high range for salmonid BCFs. Using these BCFs and data presented in the following studies, equivalent aqueous (*i.e.*, water-borne only) dieldrin concentrations NMFS estimated to be between 0.89 and 65 times the proposed chronic criterion of 0.056 µg/L for dieldrin.

Hendricks *et al.* (1979) reported repressed growth in juvenile rainbow trout exposed to 5 ppm dieldrin in their diet for 12 months at 12°C, with a corresponding tissue concentration of approximately 1.6 mg dieldrin/kg whole fish. The corresponding concentration for dieldrin in a water-borne-only exposure experiment was estimated here to be between 0.18 µg/L and 0.94 µg/L.

Mehrle *et al.* (1971) reported alteration of the serum concentration of 11 amino acids in rainbow trout exposed to 1 mg dieldrin/kg body weight per week in their diet for 140 days at 16°C, with a corresponding tissue concentration of 1.8 mg dieldrin/kg whole fish. The corresponding concentration for dieldrin in a water-borne-only exposure experiment was estimated here to be between 0.2 µg/L and 1.1 µg/L. The results suggested that the utilization of five of the amino acids was inhibited by dieldrin, possibly due to an effect on enzymes which are responsible for the utilization and energy transformation of these specific amino acids.

Kilbey *et al.* (1972) conducted a 300-day dietary exposure study using rainbow trout held at 17°C. Effects that were observed included increased blood phenylalanine levels, decreased liver phenylalanine hydroxylase activity, and increased concentration of urine phenylpyruvic acid when dieldrin was present in the diet at 14 µg/L to 430 µg/L dieldrin/kg body weight/day (0.36µg/L to 10.8µg/L dieldrin/g of food). The corresponding dieldrin tissue concentration was 0.41 mg/kg to 6.23 mg/kg wet weight. Based on these tissue concentrations, a corresponding concentration for dieldrin in a water-borne only exposure experiment was estimated to be between 0.05 µg/L and 3.66 µg/L. The three effects observed parallel those seen in phenylketonuria, an inherited defect in human phenylalanine metabolism that is also characterized by mental deficiency. Although the study did not address analogous effects, it is possible that fish adaptability, behavior, and survival may be compromised based on biochemical similarities.

There are numerous additional studies on tissue exposure of salmonids to dieldrin. However, they have low utility for the purpose of evaluating the proposed chronic criterion, either because necessary data and findings were not reported, whole body tissue concentration could not be

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estimated, or test specimens were exposed to a mixture of compounds (*e.g.*, Macek *et al.* 1970, Mehrle and Bloomfield 1974, Poels *et al.* 1980, Shubat and Curtis 1986).

Salmonid fishes and other freshwater fish species strongly bioaccumulated dieldrin from the water column in laboratory exposure studies. Van Leeuwen *et al.* (1985) exposed early fry rainbow trout to dieldrin for 24 hours and reported a steady state BCF of 1,700. Chadwick and Shumway (1969) reported a whole body BCF equal to approximately 3,200 for newly hatched steelhead trout alevins after 35 days of exposure.

Whole body or lipid BCF calculated from information provided in other studies on exposure concentration, duration, and tissue residue concentration are also indicative of the tendency of dieldrin to bioaccumulate. Shubat and Curtis (1986) exposed juvenile rainbow trout to 0.04 µg/L dieldrin for 16 weeks in a flow-through experiment with a measured dieldrin concentration, and indicated a whole body tissue residue level of 120 to 320 ng dieldrin/g fish tissue, or 7.1 ng to 11 ng dieldrin/mg lipid. This translates into a whole body BCF of approximately 3,000 to 8,000, or a lipid BCF of 178,000 to 275,000. For fish exposed to 0.08 µg/L, the calculated whole body BCF becomes 2,500 to 8,900, and the lipid BCF 225,000, indicating slightly higher bioaccumulation rates at higher water concentrations.

The only other freshwater fish for which laboratory-derived bioaccumulation information was found is the channel catfish *Ictalurus punctatus*. Shannon (1977a) conducted a 28-day exposure to 0.075 µg/L of an 87% dieldrin formulation in a flow-through experiment with measured concentrations of dieldrin. Based on reported tissue concentrations, the calculated dorsal muscle BCF is 2,333 for smaller fish and 3,653 for larger fish. Although Shannon (1977a) suggests that the higher bioaccumulation observed for the larger fish in this study could be due to a higher fat content, this notion was not supported by results from a field study where larger fish did not consistently harbor higher residue concentrations (Kellogg and Bulkley 1976). In another experiment, a 70-day exposure to 0.013 µg/L dieldrin resulted in a calculated dorsal muscle BCF of 2,385, with equilibrium being reached more rapidly at lower level exposures than at higher levels (Shannon 1977b). These laboratory BCF values for catfish are roughly comparable to BCFs determined for salmonids. However, they are approximately 10 fold below the BCF values reported in channel catfish from field studies. Leung *et al.* (1981) sampled fish and water from the Des Moines River in Iowa in June and August 1973, during a time when aldrin was being used on area cropland. The corresponding calculated muscle tissue BCF values range from 2,220 to 22,200. The authors did not discuss the possibility that the tissue residue levels could reflect dieldrin accumulation from food and sediment as well as water. However, Chadwick and Brocksen (1969 as cited in Shannon 1977a) noted that, when selected fish were tested for accumulation of dieldrin from food or water, most of the dieldrin in the tissue came from water. The reported information from additional field studies conducted in the Des Moines River can be used to calculate the BCF values for various other freshwater fish, yielding estimated BCFs of up to 1,600 for carpsucker, 10,200 for sand shiner, 15,500 for spotfin shiner, or 7,500 for bluntnose minnow (Kellogg and Bulkley 1976).

No laboratory derived BCF values were available for any aquatic insect species that are prey for salmonids. Reinert (1972) noted a BCF of approximately 14,000 for *Daphnia magna* exposed to dieldrin for 3 days. Kellog and Bulkley (1986) conducted a field study from which reported

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tissue and water concentrations of dieldrin can be used to calculate BCF values for various insect, crustacean, or fish prey species used by salmonids. Water samples contained 0.004 µg/L to 0.012 µg/L dieldrin, and aquatic organisms had tissue levels ranging from 2 ppb to 61 ppb from the Des Moines River in Iowa in 1973. Corresponding calculations result in BCF values that are on the order of 1,500 for the stonefly *Pteronarcys*, 5,100 for the mayfly *Potamanthus*, 3,500 for Chironomidae, 3,600 for Trichoptera, and 1,300 for the crayfish *Oronectes rusticus*.

For photodieldrin, BCF values derived from laboratory studies on various freshwater fish are approximately an order of magnitude lower than laboratory dieldrin BCF values determined for salmonids and catfish. For example, after a one 1-day exposure to 20 µg/L photodieldrin in a static experiment with measured dieldrin concentrations, BCF values were 133 for bluegill (*Lepomis machrochirus*), 150 for minnow (*Lebistes reticulata*), 609 for goldfish (*Carassius auratus*), and 820 for guppy (*Gambia affinis*) (Khan and Khan 1974). The data of Khan and Khan (1974) also indicated a BCF around 1,200 for a Gammarid exposed for four days at 10 µg/L.

Statham and Lech (1975) noted that dieldrin may interact synergistically with carbaryl. In a water-borne exposure study with fingerling rainbow trout, a 4-hour exposure to dieldrin at 1,000 µg/L caused 16% mortality, but when 1 mg/L carbaryl was added to the mixture, the resulting mortality level was 94%, which was greater than the sum of effects for either compound alone. No mechanism for this interaction was determined or suggested. Based on this information, natural freshwater areas that are known to contain both carbaryl (or other carbamate insecticides) and dieldrin may require special consideration with respect to synergistic toxicity to fish.

Interaction between dieldrin and DDT varies depending on the toxicity endpoint considered. Macek *et al.* (1970) conducted an experiment with rainbow trout fed dieldrin and DDT for 140 days. This was sufficient time for equilibrium to be reached with respect to tissue residue accumulation of the two compounds. A significant increase in lipogenesis was seen with either contaminant alone, but, after several months, an additive effect also was apparent in fish that were fed both contaminants. In the pyloric caecae, the accumulation rate of DDT was increased by the presence of dieldrin, while that of dieldrin decreased. Further, elimination of DDT decreased markedly, while elimination of dieldrin remained unchanged. The results from this study suggest the possibility of increased bioaccumulation of DDT when dieldrin and DDT are present together in the environment. In contrast, Mayer *et al.* (1972) noted an antagonistic effect in rainbow trout that were fed dieldrin at non-lethal levels and DDT at lethal levels for 6 days. The fish died at about half the rate as with DDT alone. The mechanism of this interaction was not determined in this study. From an environmental perspective, this observation may be important only when high (lethal) levels of DDT are bioavailable.

An antagonistic interaction also was suggested by Hendricks *et al.* (1979) between dieldrin and aflatoxin B_1. In juvenile rainbow trout fed with both compounds for 12 months, the observed growth inhibition was similar to that caused by dieldrin alone, thus indicating a reduction in the growth inhibitory effect of Aflatoxin B_1.

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Sublethal Effects Summary. The available evidence indicates that the chronic criterion is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Acute toxicity data available identified effects of dieldrin on aquatic invertebrates ranging from 0.5 µg/L to 3.7 µg/L:

- Sanders and Cope (1968) reported 96 hour LC_{50} values of 0.5 µg/L for the stonefly naiads *Pteronarcys californica* and *Pteronarcella badia*, and 0.58 µg/L for the stonefly naiad *Claassenia sabulosa*, in static experiments performed at around 15.5°C and pH 7.1.
- Karnak and Collins (1974) reported a 24 hour LC_{50} of 0.7 µg/L for the midge larvae *Chironomus tentans*, using 85% dieldrin at 22°C.
- Bowman *et al.* (1981) reported an 18-hour LD_{50} value of 3.7 µg/L for the glass shrimp *Palaemonetes kadiakensis* at 23°C in a static experiment.

Reports could not be found in the toxicological literature that indicate adverse effects from dieldrin occur to salmonid prey species at levels below the proposed chronic criterion of 0.056 µg/L. Results for three aquatic insects and three crustaceans demonstrate that adverse effects are manifest at the individual or population level only when dieldrin concentrations are much higher, ranging between 9 and 66 times the criterion (Jensen and Gaufin 1966, Adema 1978, Daniels and Allan 1981, Phipps *et al.* 1995).

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Dieldrin. The available evidence for dieldrin indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), reduced growth (moderate intensity), physiological trauma (moderate intensity), and reproduction (low intensity).

2.6.2.1.2 Endosulfan-alpha and Endosulfan-beta

Endosulfan Criteria. The proposed acute and chronic criteria for endosulfan-alpha and endosulfan-beta are 0.22 µg/L and 0.056 µg/L, respectively.

Tables 2.6.2.1.2.1 through 2.6.2.1.2.2 report toxicity data from the ECOTOX database for freshwater endosulfan, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.2.1.2.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endosulfan-alpha and endosulfan-beta.

Criterion Freshwater Endosulfan-alpha and Endosulfan-beta		Data Set ECOTOX
Criterion Concentration Acute **0.22 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **0.88**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **30-255 mg/L $CaCO_3$**	**Geometric Mean** **0.66**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **NR**	**Harmonic Mean** **0.51**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.17	NEWBORN	96H
0.24	NEWBORN	96H
0.26	NEWBORN	96H
0.26	NEWBORN	96H
0.27	NEWBORN	96H
0.29	NEWBORN	96H
0.3	NEWBORN	96H
0.3	NEWBORN	96H
0.32	NEWBORN	96H
0.41	NEWBORN	96H
0.42	NEWBORN	96H
0.49	NEWBORN	96H
0.63	NEWBORN	96H
0.69	NEWBORN	96H
0.79	NEWBORN	96H
0.8	NEWBORN	96H
0.8	NEWBORN	96H
0.81	NEWBORN	96H
0.86	NEWBORN	96H
0.94	NEWBORN	96H
1.21	NEWBORN	96H
1.3	NEWBORN	96H
1.34	NEWBORN	96H
1.5	NEWBORN	96H
1.63	NEWBORN	96H
1.69	NEWBORN	96H
1.7	NEWBORN	96H
2.43	NEWBORN	96H
2.6	NEWBORN	96H

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Table 2.6.2.1.2.2 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endosulfan-alpha and endosulfan-beta.

Criterion **Freshwater Endosulfan-alpha and Endosulfan-beta**		**Data Set BE**
Criterion Concentration Acute **0.22 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **0.88**
Criterion Concentration Chronic **0.056 Micrograms Liter^{-1}**	**Hardness** **30-255 mg/L $CaCO_3$**	**Geometric Mean** **0.66**
Endpoint/Effect **NOEC**	**pH** **NR**	**Harmonic Mean** **0.51**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.016		
0.02		
0.063		
0.075		
0.078		
0.17		

Water Quality Parameters as Predictors of Endosulfan Acute Toxicity. Schoettger (1970) tested various water quality parameters to determine their effect on the toxicity of endosulfan to several fish species. Variations in calcium and magnesium salts did not alter the acute toxicity to western white suckers, nor did changes in pH between 6.4 and 8.4. However, experiments with rainbow trout indicated that temperature changes did have an effect on toxicity. In three different studies, endosulfan toxicity increased with increasing temperature. Two other studies using rainbow trout also reported a temperature effect. Sunderam *et al.* (1992) determined that the 96-hour LC_{50} changed from 1.6 µg/L at 4°C to 0.7 µg/L at 12°C, using static conditions, pH 7.5, and measured concentrations of endosulfan. Macek *et al.* (1969) reported 96-hour LC_{50}s of 2.6 µg/L, 1.7 µg/L, and 1.5 µg/L at 1.6°C, 7.2°C, or 12.7°C, respectively, under static conditions at pH 7.1 and nominal endosulfan concentrations.

Endosulfan Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these

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studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to endosulfan-alpha and endosulfan-beta, NMFS added an additional step to its analysis for endosulfan-alpha and endosulfan-beta to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.22 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.2.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.2.1, predicts a magnitude of effect ranging from a

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low of an $LC_{4.2}$ at a concentration of 2.6 µg/L to a high of an LC_{65} at a concentration of 0.17 µg/L. In other words, the acute criterion of 0.24 µg/L has an equivalent toxicity potential predicted to kill 4.2 percent to 65 percent, with a median toxicity potential of an $LC_{13.9}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for endosulfan-alpha and endosulfan-beta, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for endosulfan-alpha and endosulfan-beta, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Endosulfan is a broad-spectrum polychlorinated cyclodiene insecticide. It is used to control over 100 agricultural pests and 60 food and non-food crops, and does not occur naturally in the environment. It was first developed in Germany by Hoechst in 1954 under the registered trade name Thiodan. In its pure form, endosulfan exists in two different conformations: I (alpha) and II (beta). Technical endosulfan, the form which is most often used in laboratory toxicity studies, is 94% to 96% pure, with an approximate ratio of 7:3 alpha:beta isomers (Naqvi and Vaishnavi 1993).

Endosulfan is virtually insoluble in water, but is readily dissolved in organic solvents before its addition to aqueous formulations (Geobel *et al.* 1982, Naqvi and Vaishnavi 1993). In alkaline water, hydrolysis is the primary process for degradation, with the beta isomer hydrolyzing more rapidly than the alpha isomer (Peterson and Batley 1993). Endosulfan diol is the main product of chemical hydrolysis, but it is also oxidized to endosulfan sulfate (Naqvi and Vaishnavi 1993). In solution, the alpha isomer is more abundant than the beta isomer or endosulfan sulfate. Also, in the aquatic environment, endosulfan beta and endosulfan sulfate are more likely to be bound to sediment and particulates than endosulfan alpha (Peterson and Batley 1993).

Endosulfan acts as a central nervous system poison (Naqvi and Vaishnavi 1993). Of the organochlorine insecticides, it is one of the most toxic to aquatic organisms (EPA 1976; EPA 1980g). In general, freshwater fish are more sensitive to endosulfan than freshwater invertebrates (EPA 1980g), and marine organisms are more sensitive than freshwater ones (Naqvi and Vaishnavi 1993). The toxicities of endosulfan and endosulfan sulfate are roughly equivalent (Naqvi and Vaishnavi 1993). However, comparisons of the toxicity of individual isomers of endosulfan indicate that the alpha form is generally more toxic than the beta. The other biological metabolites of endosulfan that do not contain sulfur, such as endosulfan diol, endosulfan ether, and endosulfan lactone, are considerably less toxic than either the sulfur-containing endosulfan sulfate or alpha or beta isomers.

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Most endosulfan toxicity studies on aquatic organisms have evaluated direct water-borne exposure. Studies reported by Barry *et al.* (1995) indicated that, for the cladoceran *Daphnia carinata*, water-borne exposure is the most toxic route. Toxicity towards *D. carinata* also increase at higher food concentrations. This may be due to a higher level of persistence of endosulfan in the water column, or increased uptake of the compound by the test organisms due to elevated metabolism. Similar toxicity studies that assessed food concentration or route of exposure for fish were not found in the literature. However, there are other aspects of study design that can influence toxicity outcome. Static flow or semi-static assay conditions are more likely to underestimate toxicity when compared with the more environmentally relevant constant flow assays. Studies that include nominal, or unmeasured, test compound concentrations during the exposure period also are more likely to underestimate toxicity compared with those with measured concentrations (Naqvi and Vaishnavi 1993). The toxic effects of endosulfan on fish are influenced by water temperature, with increased toxicity generally observed at higher temperatures. The influence of temperature is discussed further below.

The available information on the chronic effects of endosulfan on salmonids or other freshwater fish is limited. Arnold *et al.* (1996) observed sublethal effects at concentrations between 0.2 times and 1.8 times the proposed chronic criterion. Mature male rainbow trout that were exposed for 28 days to 0.01 µg/L endosulfan (measured) in a flow-through assay at 14.5°C developed qualitative hepatic cytological ultrastructural alterations. This dose was the LOEC. At 0.05 µg/L and 0.1 µg/L, degenerative subcellular effects such as dilation of intermembranous spaces in mitochondria and deformation of mitochondria were observed. Other subcellular effects included proliferation of smooth endoplasmic reticulum (SER), circular arrays of rough endoplasmic reticulum (RER), and an increase in lysosomal elements. The SER and RER effects were probably an indication of the activity of mixed-function oxygenases. These type of structural alterations have been shown by many investigators to be highly selective and sensitive biomarkers of chronic toxicity, although specific effects on fish health have not been elucidated.

Toxicity studies on other freshwater fish species have indicated adverse effects when exposure concentrations ranged between 0.8 times and 3.6 times the chronic criterion:

- Verma *et al.* (1981) exposed the freshwater catfish *Mystus vittatus* to 0.045, 0.067, and 0.13 u/L endosulfan for 30 days at 24°C in a nominal, static renewal assay. This treatment caused alterations in acid phosphatase, alkaline phosphatase, and glucose-6-phospatase in liver, kidney, and gills. Although the reason for these alterations is not clear, they may be due to uncoupling of oxidative phosphorylation or structural alterations of lysosomes.
- Sastry and Siddiqui (1982) exposed the freshwater murrel *Channa punctatus* to 0.2 µg/L endosulfan for 15 and 30 days at 20°C, pH 7.4 in a static renewal assay. This resulted in a reduction in the rate of glucose absorption by the intestine, possibly due to structural damage to the intestinal mucosa, or a decrease in the activity of enzymes that are involved in nutrient absorption, such as Na^+-K^+ ATPase and alkaline phosphatase.

The results of several studies indicate adverse effects can occur when concentrations are below or near the proposed chronic criterion after an exposure period less than 96 hours. Effects were evident at concentrations that were between 0.9 times and 1.8 times the proposed chronic criterion, suggesting that chronic toxic effects could occur to salmonids under the proposed

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criterion, assuming effects are equal among species. These studies are described below:

- Murty and Devi (1982) exposed the freshwater snakehead fish *Channa punctata* (Bloch) to 0.05 µg/L endosulfan alpha for 4 days at 27°C in a nominal, continuous flow assay. The lipid content and glycogen concentration of liver, muscle, and brain were significantly altered, as was the protein content of muscle and kidney.
- Nowak (1996) exposed the freshwater catfish *Tandanus tandanus* to 0.1 µg/L endosulfan for 24 hours in a nominal, static assay. Effects observed included dark atrophied hepatocytes (usually a sign of cell necrosis resulting from chronic injury); structural (necrotic) changes in liver tissue; proliferation, dilation, and vesiculation of the RER (possibly due to inhibition of protein synthesis); concentric bodies (a possible sign of cytologic regeneration); and residue levels in liver tissue up to 80 ppb.
- Nowak (1992) exposed *Tandanus tandanus* to 0.1 µg/L endosulfan for 24 hours in a measured, static assay. This resulted in edema and lifting and hyperplasia of lamellar epithelium in the gills, and also increased in respiratory diffusion distance. Although this may allow separation of blood from the toxicant, it can also damage gills, having deleterious effects on fish physiology.
- Rao *et al.* (1980) exposed the Indian major carp *Labeo rohita* to 0.1 µg/L endosulfan for 1 hour at 28°C, pH 8.4 in a nominal, static assay. An increase in oxygen consumption was observed.

Information on uptake, metabolism, and elimination of endosulfan was not available for salmonid fishes. However, the following is a brief overview of information available for other freshwater fish species, including the spotted snakehead *Channa punctata* (Devi *et al.* 1981), the rohi *Labeo rohita* (Rao *et al.* 1980), the Indian carp *Catla catla* (Rao 1989), the climbing perch *Anabus testudineus* (Rao and Murty 1980), and goldfish and western white sucker (Schoettger 1970).

The unaltered alpha and beta forms of endosulfan were detected in *Channa punctata*, *Anabus testudineus*, and *Catla catla* in one or more tissues, including brain, gills, kidney, liver, and muscle. In *Catla catla* in particular, muscle was found to be the principle storage site of unaltered endosulfan.

The principal metabolites of endosulfan in *Catla catla*, *Channa punctata*, or *Labeo rohita* were reported to be endosulfan alcohol, endosulfan ether, or endosulfan lactone. Other metabolites that were detected in various fish included endosulfan alpha-hydroxyether and endosulfan sulfate. The liver was cited as either the principal detoxifying organ or the site where uptake appeared to be considerably higher than for other tissues in *Labeo rohita*, the western white sucker *Catostomus commersoni*, and the goldfish *Carassius auratus auratus*. This differed somewhat from the climbing perch, in which both the liver and kidneys were reported as being the principal sites of detoxification.

Both Endosulfan and endosulfan sulfate are known to bioconcentrate, and thought to bioaccumulate (EPA 1999), which is in accord with log K_{ow} values of 4.10, 3.83, and 4.52 for technical endosulfan, isomer I and isomer II, respectively (Karickhoff and Long 1995). Toxicity of endosulfan to aquatic biota is influenced by water temperature (increased toxicity with

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increased temperature), and type of isomer (EPA 1999). Of the organochlorine insecticides, it is one of the most toxic to aquatic organisms (EPA 1980f). The primary mode of action of endosulfan is disruption of nerve function in the central nervous system (Casarett and Doull 2001). In general, freshwater fish are more sensitive to endosulfan than freshwater invertebrates (EPA 1980f). Effects of endosulfan toxicity to freshwater organisms include anoxic stress, altered calcium deposition, blood disease, altered gill structure, and reduced survival (EPA 1999).

Reports on the bioconcentration of endosulfan in salmonids were not available, although limited information for other freshwater fish was found, indicating that the BCF can vary greatly between species. Ramaneswari and Rao (2000) exposed *Channa punctata* to 0.141 µg/L endosulfan (alpha or beta isomers) for 1 month and measured a whole body BCF of 13. A similar exposure of *Labeo rohita* yielded a BCF of 37 for alpha endosulfan and 55 for beta endosulfan. The exposure concentration used (0.141 µg/L) was 2.5 times the proposed chronic criterion. These BCF values were much lower than those obtained for yellow tetra (*Hyphessobrycon bifasciatus*), in which the whole body BCF was 11,600 after a 21 day exposure to 0.3 µg/L endosulfan at 22°C, pH 7.1 under static-renewal conditions (Jonsson and Toledo 1993). In this study, the total residues in fish increased with increasing time, and the authors indicated that a steady state had not been reached. The biological half-life was estimated at 1.8 days, which is similar to the half-life in goldfish (Oeser *et al.* 1971 as cited in Geobel *et al.* 1982).

Only two reports of endosulfan bioaccumulation were found for salmonid prey species. Sabaliunas *et al.* (1998) exposed the lake mussel *Anodonta piscinalis* to 1.5 µg/L endosulfan in a continuous flow experiment at 10°C with measured contaminant concentration. They noted a whole BCF of 750 under conditions that may not have reached steady state. Finally, a field study was conducted using paired oyster whole body tissue samples and water samples from the Patuxent River, which discharges into the Chesapeake Bay in Maryland (Lehotay *et al.* 1999). In oyster tissue, more endosulfan sulfate was present compared to the alpha or beta isomers. In the water samples, more of the beta isomer was present than the alpha isomer or endosulfan sulfate (even though beta is less soluble than alpha and constitutes only 30% of the endosulfan mixture that is commonly used). Based on the average concentration of endosulfan alpha, beta, or sulfate in oyster tissue (0.037 ng/g to 0.13 ng/g) or in water samples (0.5 ng/L to1.0 ng/L), one can calculate the BCF range as 37 to 260.

Sublethal Effects Summary. Although the data regarding sublethal effects on fishes exposed to endosulfan-alpha and endosulfan-beta is available, there are no chronic toxicity studies available for juvenile salmonid fishes. If the mechanism and mode of actions are similar for salmonid fishes, salmonid fishes will suffer chronic toxic effects.

Toxicity to Food Organisms. Most toxicity studies indicate lethal effects do not occur on salmonid prey species until concentrations are between 19 and 2,232 times the proposed acute criterion. These species include the freshwater scud *Gammarus lacustris*, with 96-hour LC_{50} values of 4.1 µg/L or 5.8 µg/L (Johnson and Finley 1980; Sanders 1969 as cited in EPA 1980g); the cladoceran *Daphnia magna*, with LC_{50} values of 56 µg/L to 271 µg/L (Schoettger 1970, Nebeker *et al.* 1983, EPA 1976); damselfly naiad 96-hour LC_{50} of 71.8 µg/L to 107 µg/L

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(Schoettger 1970); and a 48 hour LC_{50} of 215 µg/L for *Moinodaphnia macleayi* or 491 µg/L for *Ceriodaphnia dubia*.

Chronic exposure studies reported in the scientific literature appear to include only cladocerans, and all of these studies report chronic effects at concentrations well above the proposed chronic criterion. For example, *D. magna* exhibited reduced survival after 22 days of exposure to 7 µg/L endosulfan or reduced reproduction in the second generation at 37.7 µg/L (EPA 1976), the LOEC for decrease in number of young for *C. dubia* was 20 µg/L after 14 days exposure, or 40 µg/L for *M. macleay* (Sunderam *et al.* 1994), and reduction of brood size and body length for *Daphnia carinata* was observed after 6 days at 320 µg/L (Barry *et al.* 1995).

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Endosulfan-alpha and Endosulfan-beta. The available evidence indicates that listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects including mortality (moderate intensity). There are no chronic toxicity studies available for juvenile salmonid fishes. However, the NOEC analysis suggests that salmonid fishes will suffer chronic toxic effects—sublethal effects— (moderate intensity). Furthermore, if the mechanism and/or mode of actions for the fish species with sublethal toxicity data are similar for salmonid fishes, salmonid fishes will suffer sublethal effects (moderate intensity).

2.6.2.1.3 Endrin

Endrin Criteria. The proposed acute and chronic criteria for endrin are 0.086 µg/L and 0.036 µg/L, respectively.

Tables 2.5.2.1.3.1 through 2.5.2.1.3.5 report toxicity data from the ECOTOX database for freshwater endrin, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.2.1.3.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion Freshwater Endrin		Data Set ECOTOX
Criterion Concentration Acute 0.086 Micrograms Liter^{-1}	**Temperature 1.6-20° Celsius**	**Arithmetic Mean 167**
Criterion Concentration Chronic 0.036 Micrograms Liter^{-1}	**Hardness 44-272 mg/L $CaCO_3$**	**Geometric Mean 1.1**
Endpoint/Effect LC_{50}/Mortality	**pH 6-7.95**	**Harmonic Mean 0.3**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.02	22 D, 32.3 MM, PTERYGIO LARVA	72H
0.02	29 D, 34.1 MM, PTERYGIO LARVA	48H
0.06	29 D, 34.1 MM, PTERYGIO LARVA	72H
0.089	FINGERLING	96H
0.095	.37 G	96H
0.113		
0.117	.37 G	72H
0.12	22 D, 32.3 MM, PTERYGIO LARVA	48H
0.12	71 D, 46.2 MM, JUVENILE	48H
0.12	71 D, 46.2 MM, JUVENILE	72H
0.167	1.30 G	96H
0.192	.37 G	48H
0.192		
0.218	1.30 G	48H
0.25	15 D, 31.0 MM, PROTOPTERYGIO LARVA	48H
0.25	15 D, 31.0 MM, PROTOPTERYGIO LARVA	72H
0.27	1.9 G, 2.5 IN	96H
0.27		
0.3	1.9 G, 2.5 IN	72H
0.3	1.44 G	96H
0.317	1.15 G	96H
0.327	1.24 G	96H
0.343	1.15 G	72H
0.355		
0.4	8 D, 29.2 MM, ELEUTER EMBRYO	72H
0.405		
0.432	1.15 G	48H
0.451	1.24 G	72H
0.464	2.04 G	96H
0.5	22 D, 32.3 MM, PTERYGIO LARVA	24H
0.5	2.04 G	72H
0.51		

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Criterion Freshwater Endrin		Data Set ECOTOX
Criterion Concentration Acute **0.086 Micrograms Liter^{-1}**	**Temperature** **1.6-20° Celsius**	**Arithmetic Mean** **167**
Criterion Concentration Chronic **0.036 Micrograms Liter^{-1}**	**Hardness** **44-272 mg/L $CaCO_3$**	**Geometric Mean** **1.1**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6-7.95**	**Harmonic Mean** **0.3**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.52	57-76 MM, 2.7-4.1 G	72H
0.55	29 D, 34.1 MM, PTERYGIO LARVA	24H
0.56	1.9 G, 2.5 IN	48H
0.568	1.24 G	48H
0.58	51-79 MM, 3.2 G	48H
0.58	51-79 MM, 3.2 G	72H
0.58	51-79 MM, 3.2 G	96H
0.58		
0.63	1G	96H
0.64	1G	96H
0.64	1.4G	96H
0.643	1.50 G	96H
0.674	1.50 G	72H
0.7	15 D, 31.0 MM, PROTOPTERYGIO LARVA	24H
0.7	22 D, 32.3 MM, PTERYGIO LARVA	12H
0.7	71 D, 46.2 MM, JUVENILE	24H
0.76	FINGERLING	24H
0.76		
0.79	57-76 MM, 2.7-4.1 G	96H
0.79	51-79 MM, 3.2 G	24H
0.8	57-76 MM, 2.7-4.1 G	48H
0.9	1 G, 1.625-2.25 IN	96H
0.9	1G	24H
0.9		
0.906	2.04 G	48H
0.92	6-8 G	96H
0.92		
0.97	1.4G	96H
1	1G	24H
1	1G	96H
1	1G	24H
1.01	6-8 G	72H
1.02	1.15 G	24H
1.1		
1.116	1.50 G	48H

Criterion Freshwater Endrin		Data Set ECOTOX
Criterion Concentration Acute **0.086 Micrograms Liter^{-1}**	**Temperature** **1.6-20° Celsius**	**Arithmetic Mean** **167**
Criterion Concentration Chronic **0.036 Micrograms Liter^{-1}**	**Hardness** **44-272 mg/L $CaCO_3$**	**Geometric Mean** **1.1**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6-7.95**	**Harmonic Mean** **0.3**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
1.12	1 G, 1.625-2.25 IN	72H
1.2	1.4G	96H
1.2	51-114 MM, 1.45-5 G	48H
1.2	51-114 MM, 1.45-5 G	72H
1.2	51-114 MM, 1.45-5 G	96H
1.2		
1.3	8 D, 29.2 MM, ELEUTER EMBRYO	48H
1.3	15 D, 31.0 MM, PROTOPTERYGIO LARVA	12H
1.3	1.4G	24H
1.3	57-76 MM, 2.7-4.1 G	24H
1.45	1 G, 1.625-2.25 IN	48H
1.5	6-8 G	48H
2	71 D, 46.2 MM, JUVENILE	12H
2	1.4G	96H
2	51-114 MM, 1.45-5 G	24H
2.17	1 G, 1.625-2.25 IN	24H
2.2	0.6-1.5 G	96H
2.355	1.50 G	24H
2.6	1.4G	24H
2.7	29 D, 34.1 MM, PTERYGIO LARVA	12H
2.9	8 D, 29.2 MM, ELEUTER EMBRYO	24H
4.6	1.4G	24H
5.2	2 D, 25.5 MM, ELEUTER EMBRYO	72H
6.3	8 D, 29.2 MM, ELEUTER EMBRYO	12H
7.7	1 D, 25.3 MM, ELEUTER EMBRYO	72H
11.9	1.4G	24H
12	1.9 G, 2.5 IN	24H
14.5	2 D, 25.5 MM, ELEUTER EMBRYO	48H
16.8	1 D, 25.3 MM, ELEUTER EMBRYO	48H
32.7	2 D, 25.5 MM, ELEUTER EMBRYO	24H
36.1	1 D, 25.3 MM, ELEUTER EMBRYO	24H
206	2 D, 25.5 MM, ELEUTER EMBRYO	12H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H

Table 2.6.2.1.3.2 Mortality toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion Freshwater Endrin		**Data Set ECOTOX**
Criterion Concentration Acute 0.086 Micrograms Liter^{-1}	**Temperature 2-20° Celsius**	**Arithmetic Mean 6364**
Criterion Concentration Chronic 0.036 Micrograms Liter^{-1}	**Hardness 44-272 mg/L $CaCO_3$**	**Geometric Mean 283**
Endpoint/Effect Mortality	**pH 6-7.95**	**Harmonic Mean 1.4**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.317	1.15 G	96H
0.464	2.04 G	96H
0.7		48H
0.906	2.04 G	48H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H

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Table 2.6.2.1.3.3 Physiological toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion Freshwater Endrin		Data Set ECOTOX
Criterion Concentration Acute 0.086 Micrograms Liter^{-1}	**Temperature 1.6-20° Celsius**	**Arithmetic Mean**
Criterion Concentration Chronic 0.036 Micrograms Liter^{-1}	**Hardness 44-272 mg/L $CaCO_3$**	**Geometric Mean**
Endpoint/Effect Physiological	**pH 6-7.95**	**Harmonic Mean**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.043	430-520 G	26H
0.12	55-80 G, 12-18 CM	30D
0.12	12-15 CM, 55-80 G	30D
0.343	1.15 G	72H
0.432	1.15 G	48H
0.5	2.04 G	72H
1.02	1.15 G	24H
120	NR	30D

Table 2.6.2.1.3.4 Reproductive toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion Freshwater Endrin		Data Set ECOTOX
Criterion Concentration Acute 0.086 Micrograms Liter^{-1}	**Temperature 2-20° Celsius**	**Arithmetic Mean 0.22**
Criterion Concentration Chronic 0.036 Micrograms Liter^{-1}	**Hardness 44-272 mg/L $CaCO_3$**	**Geometric Mean 0.22**
Endpoint/Effect Reproductive	**pH 6-7.95**	**Harmonic Mean 0.22**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.218	1.30 G	48H

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Table 2.6.2.1.3.5 Cellular toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater endrin.

Criterion Freshwater Endrin		Data Set ECOTOX
Criterion Concentration Acute **0.086 Micrograms Liter^{-1}**	**Temperature** **1.6-20° Celsius**	**Arithmetic Mean** **10**
Criterion Concentration Chronic **0.036 Micrograms Liter^{-1}**	**Hardness** **44-272 mg/L $CaCO_3$**	**Geometric Mean** **4.3**
Endpoint/Effect **Cellular**	**pH** **6-8**	**Harmonic Mean** **1.6**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.92	6-8 G	96H
20	FINGERLING, 7 MO, 7.5-8.0 G	0.5H

Endrin Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

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The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to endrin, NMFS added an additional step to its analysis for endrin to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.086 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.3.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.3.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 10,000 µg/L to a high of an LC_{100} at a concentration of 0.02 µg/L. In other words, the acute criterion of 0.086 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{5.4}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the criterion concentration for endrin, which implies that listed species exposed to waters equal to criterion concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for endrin, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute toxic effects, but may not suffer chronic toxic effects.

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Sublethal Effects. Endrin is a chlorinated pesticide that is a stereoisomer of dieldrin. It is no longer manufactured in the United States. Endrin ketone and endrin aldehyde are variants that occur as impurities or degradation products of endrin in commercial preparations of the insecticide. Endrin was first used in 1951 to control insects and rodents on cotton, apples, sugarcane, tobacco, and grain (IARC 1974, EPA 1980h, HSDB 1995). Its toxicity to migrant populations of migratory birds was the main reason for its cancellation as a pesticide in 1986 (EPA 1992b). It was still used as a toxicant on bird perches for several years, but this use was also banned in 1991 (EPA 1992b). There are no current releases of endrin in the United States

Exposure to endrin has been noted to result in adverse neurologic, liver, kidney, and miscellaneous endocrine and tissue weight effects (Treon *et al.* 1955 as cited in EPA 1980; Deichmann *et al.* 1970 as cited in EPA 1980, NCI 1978 as cited in HHS 1996). There are some indications that endrin may have genotoxic effects, including increased DNA damage in hepatocytes due to oxidative injury (Bagchi *et al.* 1992a, 1993a,1993c as cited in HHS 1996; Hassoun *et al.* 1993 as cited in HHS 1996). However, most studies suggest that endrin is not carcinogenic (NCI 1978 as cited in HHS 1996; EPA 1980h).

There is limited data available regarding chronic effects of water-borne exposure to endrin in salmonids (Tables 2.6.2.1.3.5 to 2.6.2.1.3.9). In other species, adverse effects have not been reported unless water concentrations were more than 10 times the proposed chronic criterion of 0.036 µg/L (*e.g.*, Hansen *et al.* 1977, Jarvenen and Tyo 1978, Jarvenin *et al.* 1988). However, there are some data available on tissue concentrations of endrin associated with a variety of sublethal adverse effects in rainbow trout, which is the non-anadromous form of steelhead trout. Grant and Mehrle (1973) determined that tissue levels associated with effects in rainbow trout included: alteration of plasma parameters, suppression of cortisol secretion and inhibited carbohydrate metabolism after a swim challenge at 0.01 mg/kg to 0.02 mg/kg, hyperexcitability at 0.12 mg/kg, and hyperglycemia and reduction in growth at 0.12 mg/kg to 0.22 mg/kg. No effects were seen at tissue concentrations at or below 0.00025 mg/kg (Grant and Mehrle 1973).

Laboratory exposure studies also suggest that exposure to endrin may affect immune responsiveness in rainbow trout. Bennet and Wolke (1987a,b) exposed rainbow trout for 30 days to sublethal concentrations of endrin (0.12 µg/L to 0.15 µg/L) and found that several immune responses (migration inhibition factor assay (MIF), plaque forming cell assay (PFC), and serum agglutination titres (SAG) were inhibited when fish were exposed to the bacterium *Yersinia ruckeri* O-antigen. Serum cortisol concentrations were found to be significantly elevated in endrin-exposed fish. Fish receiving cortisol in the di*et al.*so showed reduced immune responsiveness, suggesting that elevated serum cortisol concentration obtained in endrin-exposed fish has a central role in repression of the immune response. Fish were exposed to only one dose of endrin in this experiment, however, so there is no information on the threshold endrin concentration for immunosuppressive effects. Exposure to water-borne endrin from agricultural runoff has been associated with an increased prevalence of parasitic infections in cultured sand goby (Supamataya 1988), but the fish were also exposed at the same time to dieldrin, DDTs, and possibly stress due to changes in dissolved oxygen and water temperature.

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Singh and Singh (1980) reported total lipid levels in ovary and liver and cholesterol concentrations in ovary, liver and blood serum in the fossil catfish *Heteropneustes fossilis* after 4 weeks exposure to endrin at concentrations of 0.0006 µg/L and 0.008 µg/L during different phases of the annual reproductive cycle. Even the lower concentrations of endrin induced a significant decrease in liver lipid during the preparatory and late post-spawning phases. An appreciable increase in ovarian cholesterol was noticed during the pre-spawning and spawning. Serum cholesterol values demonstrated a significant increase in the preparatory and late post-spawning phases after exposure to endrin at all concentrations. This study suggests that exposure to endrin concentrations below the proposed chronic criterion could affect lipid and cholesterol balance in gravid salmon.

Studies show that endrin is bioaccumulated significantly by fish and other aquatic organisms (ASTDR 1996, EPA 1980h, Metcalf *et al.* 1973). Although specific BCFs are not available for salmonids, for other fish they range from 1,640 to 15,000 (EPA 1980h, Hansen *et al.* 1977). Endrin is also taken up by invertebrate prey species of salmonids, although bioconcentration factors are typically lower than those for fish. Anderson and DeFoe (1980) report pesticide accumulation in stoneflies, an invertebrate prey species, of 350 to 1150 times greater than the water concentrations after a 28-day exposure. However, biomagnification of endrin with increasing trophic level is less than that for some other chlorinated pesticides (Leblanc 1995, Metcalf *et al.* 1973).

Endrin in the diet may be an important source of uptake for fish species. Jarvinen and Tyo (1978) found that endrin in the food at a concentration of 0.63 mg/kg significantly reduced survival of fathead minnows in whole life cycle exposure tests, and residues contributed by food-borne endrin appeared to be additive to those contributed by water. Based on available BCF estimates for endrin, however, prey items would not accumulate endrin at this level under the proposed criterion.

Because endrin is no longer in use in the United States, the major source of this compound will be not through point source discharges into surface water bodies, but from repositories of the contaminant that are persistent in sediments. This means that endrin can occur through the water column, through direct contact with sediments, or through the diet. Thus, studies evaluating the effects of water-borne exposure alone are likely to underestimate actual exposure of organisms in the field.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for endrin is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Invertebrates tend to be more tolerant of endrin than fishes. Anderson and DeFoe (1980) exposed stoneflies, caddis-flies, isopods, and snails to endrin in a flowing-water test system for 28 days, increased mortality was observed at concentration in the 30,000 µg/L to 150,000 µg/L range. These values are at least two orders of magnitude above the acute criterion and at least four orders of magnitude above the chronic criterion. However, the available information is limited and may not account for exposure through other routes of exposure, such as sediments, or other invertebrate taxa.

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Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Endrin. The available evidence for endrin indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), cellular trauma (low intensity), physiological trauma (low intensity), and reproductive failure (low intensity).

2.6.2.1.4 Heptachlor Epoxide

Heptachlor Criteria. The proposed acute and chronic criteria for heptachlor are 0.52 µg/L and 0.0038 µg/L, respectively.

Tables 2.6.2.1.4.1 through 2.6.2.1.4.3 report toxicity data from the ECOTOX database for freshwater heptachlor, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.4.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater heptachlor epoxide.

Criterion **Freshwater Heptachlor Epoxide**		**Data Set** **ECOTOX**
Criterion Concentration Acute **0.52 Micrograms Liter^{-1}**	**Temperature** **13° Celsius**	**Arithmetic Mean** **14.7**
Criterion Concentration Chronic **0.0038 Micrograms Liter^{-1}**	**Hardness** **44 mg/L $CaCO_3$**	**Geometric Mean** **13.6**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **7.1**	**Harmonic Mean** **12.3**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
6.7	0.8G	96H
16	1.2G	96H
16	1.2G	96H
20	1.2G	96H

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Table 2.6.2.1.4.2 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater heptachlor epoxide.

Criterion Freshwater Heptachlor Epoxide		**Data Set BE**
Criterion Concentration Acute 0.52 Micrograms Liter^{-1}	**Temperature 13° Celsius**	**Arithmetic Mean 0.5**
Criterion Concentration Chronic 0.0038 Micrograms Liter^{-1}	**Hardness 44 mg/L $CaCO_3$**	**Geometric Mean 0.47**
Endpoint/Effect NOEC	**pH 7.1**	**Harmonic Mean 0.44**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.25		96H
0.46		96H
0.47		96H
0.53		96H
0.81		96H

Heptachlor Epoxide Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the

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criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to heptachlor epoxide, NMFS added an additional step to its analysis for heptachlor epoxide to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.52 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.4.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.4.1, predicts a magnitude of effect ranging from a low of an $LC_{1.3}$ at a concentration of 20 µg/L to a high of an LC_4 at a concentration of 6.7 µg/L. In other words, the acute criterion of 0.52 µg/L has an equivalent toxicity potential predicted to kill 1.3 percent to 4 percent, with a median toxicity potential of an $LC_{1.6}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, the available evidence for heptachlor epoxide indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute toxic effects, but may not suffer chronic toxic effects.

Sublethal Effects. Heptachlor is an organochlorine cyclodiene insecticide first isolated from technical chlordane in 1946 (ATSDR 1993). During the 1960s and 1970s, it was commonly used for crop pest control and by exterminators and home owners to kill termites. In 1976, it was prohibited from home and agricultural use, although commercial applications to control insects continued. In 1988, its use for termite control was banned, and currently its only permitted

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commercial use in the United States is fire ant control in power transformers (ATSDR 1993, Leber and Benya 1994 as cited in EPA 2008).

The principal metabolite of heptachlor is heptachlor epoxide, an oxidation product formed by many plant and animal species and through breakdown of heptachlor in the environment. The epoxide degrades more slowly and, as a result, is more persistent than heptachlor. Both heptachlor and heptachlor epoxide adsorb strongly to sediments, and both are bioconcentrated in terrestrial and aquatic organisms (EPA 1980i, ATSDR 1993).

In fishes heptachlor is readily taken up through the skin, lungs or gills, and gastrointestinal tract (ATSDR 1993). Once absorbed, it is distributed systemically and moves into body fat and is readily converted to its most persistent and toxic metabolite, heptachlor epoxide, in mammalian livers (Smith 1991, ATSDR 1993). Heptachlor is also metabolized to some extent by fish, although most evidence points to it being stored in the body predominantly as heptachlor rather than heptachlor epoxide (Feroz and Khan 1979).

Heptachlor and heptachlor epoxide are considered highly to moderately toxic to mammals, birds, and fish. The primary adverse health effects associated with acute exposure are central nervous system and liver effects (Smith 1991, ATSDR 1993, Akay and Alp 1981, Buck *et al.* 1959). Chronic exposure to heptachlor may cause some of the same neurological effects as acute exposure. An increased prevalence of neurological symptoms in humans has been associated with environmental exposure to heptachlor in epidemiological studies (Dayal *et al.* 1995), and in laboratory exposure where effects were noted on functional observational ability and motor activity (Moser *et al.* 1995). There is also evidence from epidemiological and laboratory studies that heptachlor alters the expression and function of dopamine transporters (Miller *et al.* 1999). Heptachlor may also affect immune function by inhibiting normal chemotactic responses of neutrophils and monocytes (Miyagi *et al.* 1998) or promoting necrosis of lymphocytes in the spleen and thymus (Berman *et al.* 1995).

Heptachlor does not appear to be a primary carcinogen, and laboratory tests indicate that neither heptachlor nor heptachlor epoxide are mutagenic (WHO 1984, ATSDR 1993). Heptachlor toxicity can be influenced by the presence of other compounds in the environment, but its interactions with other contaminants have not been well-studied.

As part of our data search, NMFS did not find any chronic toxicity data on salmonid fishes exposed to heptachlor epoxide, therefore we used the available toxicity for fishes as an surrogate for potential adverse effects on listed species considered in this opinion. Carr *et al.* (1999) reported that in channel catfish, heptachlor epoxides, and to a lesser extent heptachlor, bind to the gamma-aminobutyric acid (GABA) receptor and may thus suppress the activity of inhibitory neurons in the central nervous system. However, because this was an in vitro study, the exposure concentrations associated with this effect in live animals are not clear. Hiltibran (1982) investigated the effects heptachlor on the metal-ion-activated hydrolysis of ATP by liver mitochondria in by bluegill (*Lepomis macrochirus*) and found that it significantly inhibited ATP hydrolysis in an in-vitro assay. The lowest effective concentration was 0.00056 g/ml of reaction medium, but how that would compare to water concentrations affecting a live animal is not clear.

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Chronic toxicity data are correspondingly lacking for evaluating the protectiveness of the chronic criterion for salmonids. Exposure studies conducted with other species generally report effects at concentrations well above the proposed chronic criterion. For example, a study conducted on fathead minnow (Macek *et al.* 1976) showed 100% mortality after 60 days at 1.84 µg/L, with effects on sublethal endpoints at 0.86 µg/L. Similarly, Goodman *et al.* (1976) found effects of heptachlor on growth and survival of embryos and fry of the saltwater sheepshead minnow to occur when heptachlor concentrations exceeded 1.2 µg/L. Hansen and Parrish (1977) tested the chronic toxicity of heptachlor to sheepshead minnow in an 18-week partial life cycle exposure begun with juveniles, and observed decreased embryo production at 0.71 µg/L, but dose-response relationships were not consistent for this study so the data may not be accurate. The histological studies revealed conspicuous pathological changes in the liver. Other studies with non-salmonids report pathological effects on the liver and kidney, altered enzyme levels, inhibited fin regeneration, and mortality at higher concentrations (3 µg/L to 70 µg/L) with exposures ranging from 5 to 60 days (EPA 1980g, Azharbig *et al.* 1990, Rao *et al.* 1980).

In contrast to studies involving strictly water-borne exposure, other evidence suggests that adverse effects may occur when tissue concentrations are below the 0.34 mg/kg limit used to develop the chronic criterion. For example, Bishop *et al.* (1995) reported increased rearing mortality with heptachlor concentrations of 0.0279 mg/kg in Chinook salmon eggs. However, this was a field study, concentrations were measured in the eggs versus whole body tissues, and other contaminants may have been present. Tests with other species also suggest that some effects could occur at tissue residue levels in the 0.016 mg/kg to 0.3 mg/kg range. In spot (*Leistomus xantharus*), tissue concentrations of 0.654 mg/kg were associated with 25% mortality in test fish, and there are reports of increased long-term mortality at concentrations as low as 0.022 mg/kg in sheepshead minnow and 0.01 mg/kg in spot (Schimmel *et al.* 1976). It should be noted that there are some problems with analyses on which fish tissue heptachlor concentrations associated with the chronic criterion were based, particularly with respect to uncertainty about the applicability of a standardized BCF of 5,220 to salmonids.

Heptachlor is lipophilic, log K_{ow} of 6.26 (Karickhoff and Long 1995 as cited in BE), bioconcentrates and bioaccumulates in fish, animals, and milk (EPA 1999b as cited in BE). Heptachlor epoxide, log K_{ow} of 5.00 (Karickhoff and Long 1995 as cited in BE), would likewise be expected to bioconcentrate and bioaccumulate. Toxicity of heptachlor may be altered by a number of factors including temperature, duration of exposure (Johnson and Finley 1980), and presence of mixtures. Heptachlor is readily taken up in fish through the skin, lungs, gills, and gastrointestinal tract (ATSDR 1993). Heptachlor and its primary metabolite are considered to be moderately to highly toxic to fish (ATSDR 1993). Effects of heptachlor toxicity to freshwater organisms include reduced growth, inhibited ATPase activity, and reduced survival (EPA 1999b as cited in BE).

Both heptachlor and heptachlor epoxide have been shown to bioconcentrate in aquatic organisms such as fish, mollusks, insects, plankton, and algae (ATSDR 1989). They have been found in the fat of fish, mollusks, and other aquatic species at concentrations of 200 to 37,000 times the concentration of heptachlor in the surrounding waters (WHO 1984, ATSDR 1989). A wide range of BCFs have been determined in laboratory studies using fish (EPA 1980i). No BCF values are available for salmonids, but values for fathead minnow range from 9,500 to 14,400 (Veith *et al.*

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1979, EPA 1980i), and Goodman *et al.* (1976) reported average bioconcentration factors for heptachlor of 3,600 for sheepshead minnow. Because heptachlor is no longer in use in the United States, except for selected special applications, the major source of this compound will be not through point source discharges into surface water bodies, but from repositories of the contaminant that are persistent in sediments. This means that heptachlor and heptachlor epoxide will be taken up not only through the water column, but also through direct contact with sediments or through the diet. Thus, studies evaluating the effects of water-borne exposure alone are likely to under-estimate actual exposure of organisms in the field.

If it is assumed that sediments are a major source of heptachlor, the sediment-heptachlor concentrations that would result in heptachlor concentrations in the water column at or below the criteria are: For heptachlor, $\log_{10}(K_{ow}) = 6.26$, $\log_{10}(K_{oc}) = 6.15$, and $F_{cv} = 0.0038$, resulting in $SQC_{oc} = 5.37$ mg/kg organic carbon[7]. This would mean that for sediment total organic carbon (TOC) levels of 1% to 5% percent, the sediment heptachlor concentrations would range from 54 ng/g to 269 ng/g sediment. These levels bracket the sediment screening guideline of 10 ng/g dry wet established by the U.S. Corps of Engineers (Corps) for in-water disposal of dredged sediment (Corps 1998), and are above the interim Canadian freshwater sediment guidelines of 0.6 ng/g to 2.74 ng/g dry wet sediment. The higher of these values is a probable effect level, based on spiked sediment toxicity testing and associations between field data and biological effects (CCREM 2001b). This indicates a potential for adverse effects on aquatic life.

Because there has been very little research on the toxicity of sediment-associated heptachlor to salmonids, the sediment concentrations that cause adverse effects are not well defined. The BSAFs have not been determined for salmonids, so it is difficult to estimate the likely tissue concentrations of heptachlor that would be associated with sediment heptachlor concentrations permissible under the proposed criteria.

Sublethal Effects Summary. Although the data regarding sublethal effects on fishes exposed to endosulfan-alpha and endosulfan-beta is available, there are no chronic toxicity studies available for fishes subject to this consultation. If the mechanism and modes of actions are similar for fishes subject to this consultation to those described above, then fishes considered in this opinion may not be protected from chronic toxic effects.

Toxicity to Food Organisms. Heptachlor epoxide is acutely toxic to freshwater aquatic invertebrates at concentrations comparable to those that are lethal to fish (Johnson and Finley 1980). Reported LC_{50} values for freshwater invertebrate species have include 0.9 to 2.8 µg/L for stoneflies (Sanders and Cope 1968), 29 mg/kg to 47 mg/kg for gammarid amphipods (Sanders 1969, 1972), and 42 µg/L to 78 µg/L for *daphnid cladocerans* (Macek *et al.* 1976, Sanders and Cope 1966). These values were derived from static tests in which heptachlor concentrations were unmeasured. Tests using saltwater species using flow-through tests yielded lower LC_{50} values for grass shrimp and pink shrimp (0.03 µg/L to 0.11 µg/L) than static tests for shrimp and crayfish (1.8 µg/L to 7.8 µg/L; Sanders 1972; Schimmel *et al.* 1976), suggesting that the static tests underestimate the toxicity of heptachlor to aquatic invertebrates.

[7] SQCoc SQC stands for sediment quality criteria and oc stands for organic carbon content.

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Sublethal effects of acute exposure have also been reported for some invertebrate species at concentrations close to the proposed criteria, although these studies were not conducted in salmonid prey. When the criteria for heptachlor were developed (EPA 1980i), no data were available on chronic effects of this compound on invertebrate species, and little additional information has been generated since that time. Lowest heptachlor concentrations at which effects are reported have been above 0.01 µg/L. For example, a concentration of 0.04 µg/L was associated with increased mortality in the pink shrimp, *Penaeus duoraum* (Schimmel *et al.* 1976), which is well above the proposed chronic criterion.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Heptachlor Epoxide. The available evidence for heptachlor epoxide indicates that listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects including mortality (moderate intensity). As part of our data search, NMFS did not find any chronic toxicity data on salmonid fishes exposed to heptachlor epoxide. However, the NOEC analysis suggests that listed species exposed to waters equal to the chronic criterion concentration will suffer chronic toxic effects (low intensity). Furthermore, if the mechanism and modes of actions are similar for fishes subject to this consultation to those described above in the *Sublethal Effects* analysis, then fishes considered in this opinion will suffer sublethal effects (low intensity).

2.6.2.1.5 Lindane (gamma-BHC)

Lindane Criteria. The proposed acute criterion for lindane is 0.95 µg/L.

Tables 2.6.2.1.5.1 through 2.6.2.1.5.4 report toxicity data from the ECOTOX database for freshwater lindane, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.2.1.5.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater lindane.

Criterion Freshwater Lindane		Data Set ECOTOX
Criterion Concentration Acute 0.95 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 757**
	Hardness 40-314 mg/L $CaCO_3$	**Geometric Mean 17**
Endpoint/Effect LC_{50}/Mortality	**pH 6.8-8.1**	**Harmonic Mean 0.04**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.0022	312 G	96D
0.0022	175-312 G	96D
0.019	175 G	24D
0.019	183 G	96D
0.019	277 G	24D
0.019	284 G	96D
0.019	262 G	24D
0.019	288 G	48D
1	NR	96H
16	1.1G	96H
16	1G	24H
18	FINGERLING	96H
19	0.6G	96H
20	1.1G	96H
20	1G	24H
20	1G	24H
22	FRY, 3.0 CM	96H
22	0.5G	96H
22	FRY, 3.0 CM	96H
23	FRY,3 CM	96H
23	FRY, 3.0 CM	96H
24	0.7G	96H
24	JUVENILE, 0.69 G	96H
27	1G	96H
27	1G	96H
27	1G	96H
29	1G	96H
30	FRY,3 CM	96H
30	YEARLING,107.8 G,22.4 CM	96H
30	FRY,3 CM	24H
32	1G	96H

Criterion Freshwater Lindane		Data Set ECOTOX
Criterion Concentration Acute 0.95 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 757**
	Hardness 40-314 mg/L $CaCO_3$	**Geometric Mean 17**
Endpoint/Effect LC_{50}/Mortality	**pH 6.8-8.1**	**Harmonic Mean 0.04**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
32.7	5.2 G	96H
34	1G	96H
34	1G	24H
37	YEARLING,107.8 G,22.4 CM	48H
37	FRY,3 CM	24H
37	FRY, 3.0 CM	96H
38	51-79 MM, 3.2 G	96H
38	51-79 MM, 3.2 G	24H
39	JUVENILE, 0.69 G	48H
39	51-79 MM, 3.2 G	96H
40	51-114 MM, 1.45-5 G	96H
41	51-79 MM, 3.2 G	24H
42	51-79 MM, 3.2 G	96H
42	51-114 MM, 1.45-5 G	96H
42	51-114 MM, 1.45-5 G	24H
42	51-114 MM, 1.45-5 G	48H
44	1G	96H
50	57-76 MM, 2.7-4.1 G	96H
50	ADULT, 175-250 G	48H
56	YEARLING,107.8 G,22.4 CM	24H
56	51-114 MM, 1.45-5 G	72H
56	86 D, 77 MM	48H
500	YOUNG, 9-11 CM	24H
1000	YOLK SAC FRY, STAGE 30-31, 33-34/	11D
1000	ALEVIN	24H
1000	YOLK SAC FRY, STAGE 30-31, 33-34/	96D
1000	8 H POST HATCH,FRY	24D
10000	5-10 CM	72H
10000	5-10 CM	96H
10000	5-10 CM	96H

Table 2.6.2.1.5.2 Mortality toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater lindane.

Criterion Freshwater Lindane		Data Set ECOTOX
Criterion Concentration Acute 0.95 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 19**
	Hardness 40-314 mg/L $CaCO_3$	**Geometric Mean 13**
Endpoint/Effect Mortality	**pH 6.8-8.1**	**Harmonic Mean 5.8**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1	YEARLING	1D
1	YEARLING	24D
4.1	YEARLING	72D
8.8	YEARLING	24D
16	1.1G	NR
16.6	YEARLING	24D
18	FINGERLING	72H
19	5.2 G	24D
19	FINGERLING	2H
20	1.1G	24H
22	0.5G	25H
24	0.7G	25H
26	0.5G	NR
30	1 G, 3.0-4.0 CM, JUVENILE	24H
30	1.1G	24H
30	0.7G	72H
32.7	5.2 G	24H

Table 2.6.2.1.5.3 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater lindane.

Criterion Freshwater Lindane		Data Set ECOTOX
Criterion Concentration Acute 0.95 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 10000**
	Hardness 40-314 mg/L $CaCO_3$	**Geometric Mean 10000**
Endpoint/Effect NOEC/Mortality	**pH 6.8-8.1**	**Harmonic Mean 10000**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
10000	5-10 CM	3H

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Table 2.6.2.1.5.4 Physiological toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater lindane.

Criterion Freshwater Lindane		Data Set ECOTOX
Criterion Concentration Acute **0.95 Micrograms Liter^{-1}**	**Temperature** **12-20° Celsius**	**Arithmetic Mean** **16**
	Hardness **40-314 mg/L $CaCO_3$**	**Geometric Mean** **7.9**
Endpoint/Effect **Physiological**	**pH** **6.8-8.1**	**Harmonic Mean** **3.9**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
2.1	YEARLING	2D
30	1.1G	NR

Lindane Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

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The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to lindane, NMFS added an additional step to its analysis for lindane to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.95 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.5.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.5.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 10,000 µg/L to a high of an LC_{100} at a concentration of 0.0022 µg/L. In other words, the acute criterion of 0.95 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{1.5}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for lindane, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute criterion concentration for lindane, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects.

Sublethal Effects. Lindane is one of the few chlorinated hydrocarbon insecticides considered in the proposed action that is still in use for pharmaceutical products (EPA 2002). It is used primarily for treating wood-inhabiting beetles and seeds, and in a more restricted manner

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for soil treatment and as an insecticide on fruit and vegetable crops, timber, and ornamental plants. It is also used as a dip for fleas and lice on pets, and in lotions, creams, and shampoos for the control of lice and mites in humans. It is rated as a "moderately toxic (toxicity class II)" compound by EPA. Labels for products containing it must bear warning labels, and some formulations are classified as RUPs that may only be purchased and used by certified pesticide applicators. Lindane is no longer manufactured, but is still formulated, in the United States, and aerial application of the pesticide has been prohibited. Lindane has been listed as a pollutant of concern to EPA's Great Waters Program due to its persistence in the environment, potential to bioaccumulate, and toxicity to humans and the environment.

Lindane has been sold under a number of trade names, including gamma-Hexachlorocyclohexane, Exagamma, Forlin, Gallogamma, Gammaphex, Inexit, Kwell, Lindagranox, Lindaterra, Lovigram, and Silvanol . Technical-grade lindane is comprised of the gamma-isomer of hexachlorocyclohexane (HCH). Five other isomers (molecules with a unique structural arrangement, but identical chemical formulas) of HCH are commonly found in technical lindane, but the gamma-isomer is the predominant one, comprising at least 99% of the mixture of isomers.

Lindane is moderately water soluble and may accumulate in sediments. It is relatively persistent and experiences significant degradation only under anaerobic conditions. Lindane is readily absorbed into the body, but in mammals is metabolized to some extent through conversion to tri- and tetra-chlorophenols, and conjugation with sulfates or glucuronides. Other pathways involve the ultimate formation of mercapturates. These water soluble end-products are eliminated via the urine (Smith 1991). Of the isomers, g-HCH is stored to the greatest extent in fat (Smith 1991).

Few chronic toxicity data are available for salmonids exposed to lindane in the water column. Macek *et al.* (1976) exposed brook trout for 261 days to 16.6 µg/L lindane. While survival was not affected, a reduction was observed in fish weight and length. Some disruption in reproductive activity was also recorded during the same experiment (Macek *et al.* 1976). Mendiola *et al.* (1981) determined decreased efficiency of protein utilization in rainbow trout exposed to lindane at concentrations of 1 µg/L to 10 µg/L for 21 days.

Some additional information is available on the effects of lindane associated with specific measured tissue residues in test fish. For example, in immature brook trout, Macek *et al.* (1976) found that growth rates were decreased, and observed abnormal spawning behavior in females, when muscle tissue concentrations were 1.2 mg/kg. However, there was no effect on survival. Other fish species also show effects of lindane at relatively low tissue concentrations. For example, in the gudgeon (*Gobio gobio*) the lowest tissue concentration at which a significant increase in mortality could be observed within 96 hours was 0.19 mg/kg in muscle (Marcelle and Thorne 1983). Similarly, in bluegill, the proposed no observable effect level (NOEL) for growth and mortality was 0.297 mg/kg (Macek *et al.* 1976). For other fish species, adverse biological effects occur at somewhat higher levels. Macek *et al.* (1976) observed decreased growth and increased mortality of fathead minnow at a concentration of 9.53 mg/kg in the carcass. In pinfish, the effective dose $(ED)_{50}$ for growth effects was 5.22 mg/kg (Schimmel *et al.* 1976).

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The likely tissue concentrations of lindane in fish exposed to the concentrations of lindane in the water column specified by the criteria can be calculated from EPA's estimated BCFs for lindane. Multiplying the proposed chronic criterion by the geometric mean of BCF values for lindane of 1400 (EPA 1980q) and a percent lipid of 15% (default value for freshwater fish) results in an estimated maximum allowable tissue concentration of 1.68 mg/kg lindane. For lower lipid values (5% to 10%) the values would be on the order of 0.56 mg/kg to 1.12 mg/kg. It should be noted that the normalized BCF value is based primarily on data for fathead and sheepshead minnow, not on studies with salmonids, so it may not reflect uptake in the species of concern. Also, because these BCFs were determined in the laboratory, they may underestimate lindane uptake by animals in the field. Assuming that the BCF values are in a reasonable range, it appears that tissue concentrations of lindane associated with biological effects (Macek *et al.* 1976, Marcelle and Thorne 1983) are relatively close to those predicted based on the proposed chronic criterion (1.68 mg/kg).

Some studies have also been conducted in which lindane was administered through feeding or injection studies. For example, Dunier *et al.* (1994, 1995) report that lindane modified non-specific immune responses in rainbow trout fed lindane for 30 days at a dose of 1 mg/kg. Aldegunde *et al.* (1999) observed lower body weights, increased serum cortisol levels and changes in the serotonergic brain activity after 18 days in rainbow trout implanted with 0.005 mg/kg body weight of lindane in coconut oil. These studies suggest the potential for sublethal effects on growth, metabolism, and immune function at tissue concentrations comparable or lower than those associated with the water quality criteria, but more information on the uptake ratio of lindane would be needed to evaluate these studies.

Lindane will accumulate slightly in fish and shellfish. Uptake of lindane by aquatic organisms is influenced by a number of environmental and water quality factors, including concentrations of organic particulate matter in the water column, turbidity, pH, and season of the year. Residue concentrations may also vary considerably between fish species. However, biological accumulation and persistence of lindane are low when compared to compounds such as DDT or dieldrin (Wilson 1965, Gakstatter and Weiss 1967). Lindane bioconcentrates to some extent in aquatic organisms such as fish, mollusks, insects, plankton, and algae (ATSDR 1989). Lindane has been found in the fat of fish, mollusks, and other aquatic species at concentrations up to 1400 times the concentration in the surrounding waters (WHO 1984, ATSDR 1989, Ulman 1972). Bioconcentration factors determined in laboratory studies with fish have ranged from 35 to 486, with the 486 value determined for rainbow trout (EPA 1980q).

Because lindane use in the United States is limited, one of the sources of this compound will be from repositories of the contaminant that are persistent in sediments. These means that lindane will be taken up not only through the water column, but also through direct contact with sediments or through the diet. Thus, studies evaluating the effects of water-borne exposure alone are likely to under estimate actual exposure of organisms in the field. However, because the value of the octanol/water partitioning coefficient of lindane ($\log_{10}(K_{ow}) = 3.3$) is relatively low in comparison to compounds such as DDTs and PCBs, adsorption and accumulation in sediments is also generally lower.

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The quantity and quality of available data raise concerns about the validity of the proposed acute criteria. Based on testing procedures and results from available studies that are not specific to listed species considered in this opinion and their prey, it is possible that mortality could result to both listed species and invertebrate prey under the proposed acute criterion, and adverse effects in listed fish, such as increased long-term mortality, growth reduction, increased cortisol levels, and changes in immune function. There are also a few studies suggesting that increased long-term mortality or sublethal effects could take place at lindane tissue concentrations close to those that might be expected in fish exposed to lindane at levels allowed under the acute aquatic life criteria.

Sublethal Effects Summary. The available evidence indicates that the acute criterion for lindane is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Available data on the acute toxicity of Lindane to aquatic invertebrates suggest that the proposed acute criterion of 0.95 µg/L may be protective of most types of salmonid invertebrate prey. Reported 96-hour LC_{50} values are on the order of approximately 5 to 7 times the criteria, including 4.5 µg/L for stoneflies *Pteronarcys*, and 6.3 µg/L for mysids (*Mysidopsis bahia*; Johnson and Finley 1980). For other prey species, such as Daphnia, LC_{50} values are substantially higher, *e.g.*, 460 µg/L to1460 µg/L (Fernando *et al.* 1995), or as high as 20,000 µg/L for rotifers (Janssen *et al.*1994). For amphipods, reported LC_{50} values have ranged from 5 µg/L to 80 µg/L (*Gammarus pulix*, McLoughlin *et al.* 2000, Abel 1980, Stephenson 1983, Taylor *et al.* 1991; *Gammarus lacutris* and *G. fasciatus*, Sanders 1972, *Hyalella azteca,* Blockwell *et al.* 1998).

Only one study was found that reported effects on aquatic macroinvertebrates at lindane concentrations that were below the chronic criterion; Schulz and Liess (1995) reported reduced emergence of caddisfly larvae after 90 days of exposure to concentrations of lindane as low as 0.0001 µg/L. However, most studies of the chronic effects of lindane exposure on aquatic invertebrates have reported effects occurring at levels that ranged from 2 to 28 times the proposed criterion of 0.95 µg/L. For example, for the amphipod, *Hyalella azteca*, Blockwell *et al.* (1998) reported 240-hour LC_{50}s of 26.9 µg/L and 9.8 µg/L for adults and neonates, respectively. In the amphipod *Gammarus pulix*, growth was reduced after a 14 day exposure to concentrations between 2.7 µg/L and 6.1 µg/L (Blockwell *et al.* 1996). Taylor *et al.* (1998) reported alterations in haeme biosynthesis in *Gammarus pulex* after a 240 hour exposure to lindane at 4.5 µg/L. Similarly, in mesocosm experiments involving exposures of 2 to 4 weeks, some zooplankton species, such as copepod and cyclopod nauplii and midge larvae, experienced significant mortality at lindane concentrations in the 2 µg/L to 12 µg/L range (Fliedner and Klein 1996, Peither *et al.* 1996). In contrast, effects were not observed on survival, reproduction and growth of *Daphnia magna* after 21 days of exposure until concentrations were 250 µg/L or higher (Ferrando *et al.*1995). Available data suggest that the proposed chronic criterion for lindane could adversely affect selected sensitive life stages of certain salmonid prey species.

Summary on Toxicity to Food Organisms. The available evidence indicates that the acute criterion is likely to adversely affect invertebrate productivity and abundance.

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Summary of Effects: Lindane. The available evidence for lindane indicates that listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects, *i.e.*, mortality (moderately-high-intensity).

2.6.2.1.6 Pentachlorophenol (PCP)

Pentachlorophenol Criteria. To determine the freshwater criteria as a function of pH the following equation is used:

CMC = exp (1.005 x pH – 4.83 (µg/L)
CCC = exp (1.005 x pH – 5.29 (µg/L)

At a pH of 7.8, the corresponding proposed criteria are 19 µg/L and 15 µg/L for acute and chronic criteria, respectively.

Tables 2.6.2.1.6.1 through 2.6.2.1.6.3 report toxicity data from the ECOTOX database for freshwater pentachlorophenol, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.1.6.1 LC_{50} toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater pentachlorophenol.

Criterion Freshwater Pentachlorophenol		**Data Set ECOTOX pH-adjusted**
Criterion Concentration Acute 19 Micrograms Liter^{-1}	**Temperature 6-16.5° Celsius**	**Arithmetic Mean 103**
Criterion Concentration Chronic 15 Micrograms Liter^{-1}	**Hardness 5-272 mg/L $CaCO_3$**	**Geometric Mean 87**
Endpoint/Effect LC_{50}/Mortality	**pH 5.7-8.19**	**Harmonic Mean 64**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
10	0.31 G	96H
11	1.3G	96H
11	1.3G	96H
11	1.3G	96H
32	YOLK-SAC FRY, 0.3G	96H
33	0.3G	96H
35	2.14 G, 5.80 CM	96H
36	1G	96H
41	2.14 G, 5.80 CM	96H
49	1 g	96H

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Criterion Freshwater Pentachlorophenol		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **19 Micrograms Liter^{-1}**	**Temperature** **6-16.5° Celsius**	**Arithmetic Mean** **103**
Criterion Concentration Chronic **15 Micrograms Liter^{-1}**	**Hardness** **5-272 mg/L $CaCO_3$**	**Geometric Mean** **87**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **5.7-8.19**	**Harmonic Mean** **64**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
49	.81 g	96H
53	1 g	96H
54	0.68 G	96H
54	0.68 G	96H
55	1G	96H
56	1.90 G, 5.80 CM	96H
56	1.90 G, 5.80 CM	96H
58	1G	96H
60	1 g	96H
61	1G	96H
64	1.39 G, 4.84 CM	96H
66	1.39 G, 4.84 CM	96H
67	0+ PARR	96H
68	0+ PARR	96H
68	0+ PARR	96H
69	1 g	96H
70	FRY, 10 WK, 264 MG, 33 MM	96H
70	JUVENILE, 2.7 G	96H
71	FINGERLING, 1G	96H
72	1G	96H
72	YEARLING, UNDER YEARLING	96H
75	0+ PARR	96H
83	1.0 G, 32 MM	96H
84	1.31 G	96H
87	0+ PARR	96H
93	0+ PARR	96H
95	1.0 G, 32 MM	96H
102	4.61 G, 7.40 CM	96H
103	2.84 G, 5.98 CM	96H
103	0+ PARR	96H
107	4.61 G, 7.40 CM	96H

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Criterion Freshwater Pentachlorophenol		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **19 Micrograms Liter^{-1}**	**Temperature** **6-16.5° Celsius**	**Arithmetic Mean** **103**
Criterion Concentration Chronic **15 Micrograms Liter^{-1}**	**Hardness** **5-272 mg/L $CaCO_3$**	**Geometric Mean** **87**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **5.7-8.19**	**Harmonic Mean** **64**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
107	0.87 G, 4.28 CM	96H
107	0.87 G, 4.28 CM	96H
107	0.62 G	96H
108	0+ PARR	96H
108	0+ PARR	96H
108	0.3-0.4 G FINGERLING	96H
110	2.84 G, 5.98 CM	96H
111	1.52 G, 5.24 CM	96H
114	2.48 G	96H
118	1.52 G, 5.24 CM	96H
118	0+ PARR	96H
118	0+ PARR	96H
118	0+ PARR	96H
121	2.2G	96H
122	0+ PARR	96H
124	0+ PARR	96H
124	0+ PARR	96H
127	ADULT, 18 MO, 218.0 MM, 101.0 G	152H
128	YOLK-SAC FRY	96H
129	0+ PARR	96H
132	1.38 G, 5.05 CM	96H
132	1.38 G, 5.05 CM	96H
132	0+ PARR	96H
132	0+ PARR	96H
133	0+ PARR	96H
135	1.9G	96H
136	ADULT, 18 MO, 218.0 MM, 101.0 G	96H
139	0+ PARR	96H
139	0+ PARR	96H
141	0+ PARR	96H
146	0+ PARR	96H

Criterion Freshwater Pentachlorophenol		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute 19 Micrograms Liter^{-1}	**Temperature 6-16.5° Celsius**	**Arithmetic Mean 103**
Criterion Concentration Chronic 15 Micrograms Liter^{-1}	**Hardness 5-272 mg/L $CaCO_3$**	**Geometric Mean 87**
Endpoint/Effect LC_{50}/Mortality	**pH 5.7-8.19**	**Harmonic Mean 64**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
156	0.71 G	96H
158	1.2-3.8 G, 4.6-6.4 CM, STD LENGTH	96H
161	0+ PARR	96H
166	0.46 G	96H
169	YOLK-SAC FRY	96H
174	SWIMUP FRY	96H
174	0.3G	96H
179	9G	96H
192	3.09 G, 6.3 CM	96H
220	1.2-7.9 G	96H
264	SWIMUP FRY, 0.5G	96H
316	EYED EGG	96H

Table 2.6.2.1.6.2 LC_{50} toxicity data for green sturgeon for freshwater pentachlorophenol.

Criterion Freshwater Pentachlorophenol		Data Set 4 pH-adjusted
Criterion Concentration Acute 19 Micrograms Liter^{-1}	**Temperature 22° Celsius**	**Arithmetic Mean 135**
Criterion Concentration Chronic 15 Micrograms Liter^{-1}	**Hardness 160-180 mg/L $CaCO_3$**	**Geometric Mean 134**
Endpoint/Effect LC_{50}	**pH 8.4**	**Harmonic Mean 134**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
149	JUVENILE	12H
121	JUVENILE	24H

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Table 2.6.2.1.6.3 NOEC toxicity data for salmonid fishes, Eulachon and green sturgeon for freshwater pentachlorophenol.

Criterion Freshwater Pentachlorophenol		**Data Set BE pH-adjusted**
Criterion Concentration Acute 19 Micrograms Liter^{-1}	**Temperature 6-16.5° Celsius**	**Arithmetic Mean 26**
Criterion Concentration Chronic 15 Micrograms Liter^{-1}	**Hardness 5-272 mg/L $CaCO_3$**	**Geometric Mean 21**
Endpoint/Effect NOEC/Growth	**pH 7.22-7.54**	**Harmonic Mean 16**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
6.27		
11.6	EGG	72D
12.8		
24		
25		
31		
31		
67		

Pentachlorophenol Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50}

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data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to pentachlorophenol, NMFS added an additional step to its analysis for pentachlorophenol to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 19 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.1.6.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.6.1, predicts a magnitude of effect ranging from a low of an LC_3 at a concentration of 319 µg/L to a high of an LC_{95} at a concentration of 10 µg/L. In other words, the acute criterion of 19 µg/L has an equivalent toxicity potential predicted to kill 3 percent to 95 percent, with a median toxicity potential of an $LC_{0.09}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for pentachlorophenol, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for pentachlorophenol, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available

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information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Pentachlorophenol (PCP) is a chlorinated hydrocarbon that is used primarily as an insecticide and fungicide, but also secondarily as an herbicide, molluscicide, and bactericide (Eisler 1989 as cited in EPA 2008). Technical grade PCP is approximately 86% pure and historically has been contaminated with dioxins and hexachlorobenzene. Pentachlorophenol does not occur naturally in the environment. It is produced by the chlorination of phenol. In pure form, it exists as colorless crystals and has a very sharp characteristic odor when hot. Impure pentachlorophenol is a dark gray to brown dust, beads, or flakes.

Pentachlorophenol rapidly degrades in air, on land, and in water. Pentachlorophenol is teratogenic but evidence of its mutagenic or carcinogenic properties is incomplete (Williams 1982 as cited in EPA 2008). It bioconcentrates, and bioaccumulates in predatory species (Eisler 1989 as cited in EPA 2008). Toxicity of PCP may be altered by a number of factors including pH, temperature, chemical composition (which congeners are present), organic matter, and presence of mixtures (Eisler 1989 as cited in EPA 2008). Toxicity of pure, reagent grade PCP is less than that of commercial PCP due to toxicity of some of the impurities present in commercial formulations (Cleveland *et al.* 1982). Many of the available toxicity tests have been conducted with reagent grade PCP and may thus underestimate toxic effects of commercial PCP releases into the environment. In general, fish are more sensitive to PCP than are other aquatic organisms (FWS 2000 as cited in EPA 2008). Coldwater species are generally more sensitive than warmwater species in acute lethal toxicity tests (EPA 1995 as cited in EPA 2008). Effects of PCP toxicity to algae include chlorosis inhibition, reduced cell numbers, reduced or inhibited growth, and reduced survival (Eisler 1989). Effects of PCP toxicity to freshwater invertebrates include reduced populations, reduced locomotion or immobilization, abnormal larvae development, reduced reproduction (decreased production of eggs or young), decrease in periphyton biomass, larval drift, and suppression of community metabolism in invertebrates (Eisler 1989 as cited in EPA 2008). Effects of PCP toxicity to freshwater fish include reduced growth, increased alevin mortality, reduced food conversion efficiency, reduced ability to capture and consume prey, fin erosion, cranial malformations, reduced activity, reduced egg survival, rapid swimming at the water surface and increased opercular movements, loss of balance, and reduced survival (Eisler 1989 as cited in EPA 2008).

Like other organic pollutants, PCP exhibits a tendency to be bioaccumulated by fish. Van den Heuvel *et al.* (1991) reported BCFs for rainbow trout exposed to PCP (pH 7.6) to be between 411 and 482. Similar values (350 to 764) were reported by Servizi *et al.* (1988) for pink salmon (*Oncorhynchus gorbuscha*) exposed to PCP at pH 7.75. Metabolism of PCP is relatively rapid in rainbow trout (McKim *et al.* 1986; Glickman *et al.* 1977), and this is likely true in other salmonids as well. Nevertheless, the elimination rate of this compound is sufficiently slow that it takes 11.7 days for tissue concentrations to reach 95% steady state (McKim *et al.* 1986). According to the data provided in McKim *et al.* (1986) a 96-hour exposure will produce tissue concentrations that are only 63% of steady state. Therefore, any assessment of the maximum

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attainable tissue concentration and resulting biological response for a given exposure concentration must consider a longer time period (*e.g.*, 12 days) to reach that level. An estimate of the steady-state wet-weight BCF for salmonids is 4,600 using the octanol-water partition coefficient for PCP ($\log_{10}$ (K_{ow}) = 5). Bioaccumulation of PCP is pH dependant, because pH determines the proportions of ionized and unionized PCP, which is directly related to bioaccumulation potential. The ionic form of PCP is less likely to bioaccumulate in organisms in large part because it is less likely to be taken up in the first place (Spehar *et al.* 1985).

PCP has a strong propensity to associate with the organic carbon of sediment and the lipids of organisms, as represented by a relatively high value octanol-water partition coefficient ($\log_{10}$ (K_{ow}) = 5; Eisler 1989). One of the primary toxicity mechanisms of PCP is inhibition of oxidative phosphorylation, which causes a decrease in the production of ATP in plants and animals. One consequence of this impairment is increased basal metabolism, resulting in increased oxygen consumption and high fat utilization. The effects of PCP may reduce the availability of energy for maintenance and growth, thus reducing survival of larval fish and ability of prey to escape from a predator (Brown *et al.* 1985, Johansen *et al.* 1985, Eisler 1989). PCP is known to cause several types of adverse effects in animals including dysfunction of the reproductive, nervous, and immune systems, hormone alterations, and impaired growth. In general, fish growth and behavioral endpoints have been shown to be sensitive indicators of PCP exposure (Webb and Brett 1973, Hodson and Blunt 1981, Dominquez and Chapman 1984, Brown *et al.* 1985).

The criteria for pentachlorophenol established by the EPA are pH dependent. In general, the toxicity of PCP increases with decreasing pH. At pH 4.74, half of PCP molecules are ionized (anions) and half are non-ionized. At pH 6, the ratio between the ionic and non-ionized forms is 18 (*i.e.*, the concentration of the ionized form is 18 times greater than the non-ionized form), and at pH 7 the ratio is 182. Studies have concluded that the ionic form of PCP is less toxic, primarily because it is less likely to cross membranes (Spehar *et al.* 1985). A correction factor is therefore needed for assessing bioaccumulation and toxicity to account for the effect of pH on the speciation of PCP.

Iwama *et al.* (1986) exposed juvenile Chinook salmon to 3.9 µg/L of PCP and found altered blood urea and glucose levels. Nagler *et al.* (1986) found oocyte impairment at 22 µg/L (pH 7.5). There is also evidence of sublethal effects occurring during relatively long-term exposures to PCP concentrations that are below the chronic criterion. Webb and Brett (1973) determined that juvenile sockeye salmon experienced decreased growth rates and food conversion efficiencies at PCP EC_{50}s of approximately 1.8 µg/L at pH 6.8 when exposed for 2 to 8 weeks. Hodson and Blunt (1981) also observed reduced weight, growth rate, and biomass in rainbow trout exposed over 4 weeks from embryo to fry stages. Mortality of rainbow trout eggs has also been observed at levels below the PCP chronic criterion when dissolved oxygen fell to low levels of 3 mg/L to 5 mg/L (Chapman and Shumway 1978).

Little *et al.* (1990) examined post-exposure behavioral effects in rainbow trout at exposure concentrations that were from 10 to 100 times less than the acute criterion of 19 µg/L. A statistically significant reduction in the percent survival of trout that were preyed on by largemouth bass occurred at an exposure concentration of 0.2 µg/L. A similar response may be

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expected for salmon if the mode of action is similar between species. Survival of trout was 32% to 55% in these predation studies compared to the control at 72%. This equals reductions in fish numbers of 28% to 55% in treatments compared to the control condition. Statistically significant reductions were also observed in the number of *Daphnia sp.* consumed and swimming activity when fish were exposed to a PCP concentration of 2 µg/L and a significant decrease in the strike frequency by trout on *Daphnia sp.* occurred at 20 µg/L. The exposures in Little *et al.* (1990) were conducted for 96 hours under static test conditions, and were based on nominal concentrations. The authors also expressed some concern about contaminants in the formulation used (technical grade PCP). Acetone was used as a carrier for PCP exposure in treatments and controls, which is very common in such experiments, but it is not likely to have contributed to toxicity; the concentration of acetone was 41 µg/L, which is very low. Acetone produces very low toxicity in salmonids (Majewski *et al.* 1978) and it is volatized or biodegraded in a matter of hours (Rathbun *et al.* 1982), implying that acetone was not likely a factor in the observed results.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for pentachlorophenol is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Eisler (1989) reviewed the effects of PCP on invertebrate growth, survival, and reproduction and reported adverse effects in the range of 3µg/L to 100 µg/L. It appears that most invertebrates are less sensitive than fish to PCP concentrations in water. There are, however, studies showing adverse effects to invertebrates exposed to water concentrations below the chronic criterion. Hedtke *et al.* (1985) determined reproductive impairment in a daphnid at 4 µg/L and pH 7.3. Tagatz *et al.* (1981) found a reduction in the number of species and organism abundance at PCP concentrations of 16 µg/L. The pH was not stated for this study but was likely between 7.5 and 8 because seawater was used.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is likely to adversely affect invertebrate productivity and abundance.

Summary of Effects: Pentachlorophenol. The available evidence for pentachlorophenol indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderatel-high-intensity) and reduced growth (moderate intensity).

2.6.2.1.7 Ammonia

Ammonia Criteria. At a pH of 8.0, the corresponding proposed criteria are 5.6 mg/L and 1.7 mg/L as N (NH_3-nitrogen) for acute and chronic criteria, respectively.

Tables 2.6.2.1.7.1 through 2.6.2.1.7.14 report toxicity data from the ECOTOX database for freshwater ammonia, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.2.1.7.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **2.1-18.7° Celsius**	**Arithmetic Mean** **34**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **32**
Endpoint/Effect **LC_{50}**	**pH** **6.00-9.46**	**Harmonic Mean** **29**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	**Duration**
7.3	40.0 G; SWIMMING FISH	NR
12.6	22.4 G	NR
14.0	LARVAE	NR
18.4	1.42 G	NR
22.4	10.9 G	NR
22.4	JUVENILE (4.8-9.2 CM)	NR
22.7	3.3 G	NR
23.0	JUVENILE (40 D)	NR
23.6	JUVENILE	NR
23.7	LARVAE	NR
24.4	1.30 G	NR
25.0	10.3 G	NR
25.6	1.30 G	NR
26.0	JUVENILE	NR
27.0	1 D OLD SAC FRY	NR
27.0	1 D OLD SAC FRY	NR
27.0	JUVENILE	NR
27.2	1.01 G	NR
27.7	JUVENILE	NR
27.8	1.11 G	NR
27.9	1.26 G	NR
28.7	0.90 G	NR
28.8	1.13 G	NR
30.6	1.44 G	NR
31.6	0.40 G	NR
32.1	14.0 G	NR
32.2	0.78 G	NR
32.6	JUVENILE (4.8-9.2 CM)	NR
32.7	0.60 G	NR

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Criterion Freshwater Ammonia		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **2.1-18.7° Celsius**	**Arithmetic Mean** **34**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **32**
Endpoint/Effect **LC_{50}**	**pH** **6.00-9.46**	**Harmonic Mean** **29**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	**Duration**
33.7	1.50 G	NR
33.7	1.40 G	NR
33.8	1.64 G	NR
33.8	0.90 G	NR
34.0	1.00 G	NR
34.8	0.63 G	NR
35.5	LARVAE	NR
36.1	1.38 G	NR
36.5	0.80 G	NR
37.0	1.60 G	NR
37.4	0.80 G	NR
37.7	0.80 G	NR
37.8	JUVENILE	NR
39.4	0.90 G	NR
39.4	1.30 G	NR
40.5	JUVENILE (4.8-9.2 CM)	NR
41.0	2.01 G	NR
42.6	1.26 G	NR
43.3	LARVAE	NR
46.4	JUVENILE	NR
47.0	40.0 G; RESTING FISH	NR
48.8	JUVENILE (4.8-9.2 CM)	NR
49.5	JUVENILE	NR
56.1	JUVENILE (4.8-9.2 CM)	NR
65.8	JUVENILE (4.8-9.2 CM)	NR
68.6	JUVENILE (4.8-9.2 CM)	NR
89.3	JUVENILE (4.8-9.2 CM)	NR

For Tables 2.6.2.1.7.2 through 2.6.2.1.7.10 NMFS only selected toxicity data in the core data file with a reported concentration type of total ammonia. Since total ammonia is the sum of the two forms of ammonia (NH_4^+ and NH_3), NMFS assumes that the data with a reported concentration type of total ammonia were normalized by EPA. For these toxicity studies, temperature and pH were not reported in the core data files; therefore verification regarding normalization was not possible (note: the acute criterion is not temperature-dependent). In Tables 2.6.2.1.7.5 through 2.6.2.1.7.9 NMFS reported the toxicity data as no other toxicity data was available for an analysis of chronic endpoints for ammonia, and therefore serves as the best available data. Table 2.6.2.1.7.10 through Table 2.6.1.7.13 are the ACR-NOEC analysis for the chronic criterion.

Table 2.6.2.1.7.2 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		**Data Set ECOTOX**
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 0.55**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 0.53**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 0.51**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.380		8H
0.460		8H
0.560		8H
0.790		8H

Table 2.6.2.1.7.3 LD_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		**Data Set ECOTOX**
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 22**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 22**
Endpoint/Effect LD_{50}	**pH NR**	**Harmonic Mean 22**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
22		2D

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Table 2.6.2.1.7.4 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 3.3**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 1.2**
Endpoint/Effect Mortality	**pH NR**	**Harmonic Mean 0.3**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.05		21D
0.2		2.5D
0.3		120D
0.4		2.4H
1.6		289D
4.9		2D
6		4D
6.3		1D
10		90D

Table 2.6.2.1.7.5 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 1.5**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 1.2**
Endpoint/Effect Growth	**pH NR**	**Harmonic Mean 0.9**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.3		120D
0.9		365D
1.2		365D
1.3		365D
1.6		365D
3.5		85D

Table 2.6.2.1.7.6 Biochemical toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 0.6**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 0.1**
Endpoint/Effect Biochemical	**pH NR**	**Harmonic Mean 0.004**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.001		1D
0.22		84D
0.7		4H
1.6		4H

Table 2.6.2.1.7.7 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 27.1**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 8.4**
Endpoint/Effect Behavioral	**pH NR**	**Harmonic Mean 1.7**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.4		4.8H
4.5		2.4H
6		2D
62.3		NR
62.3		NR

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Table 2.6.2.1.7.8 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 0.3**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 0.3**
Endpoint/Effect Cellular	**pH NR**	**Harmonic Mean 0.3**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.3		120D

Table 2.6.2.1.7.9 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature NR**	**Arithmetic Mean 0.23**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Geometric Mean 0.23**
Endpoint/Effect Physiological	**pH NR**	**Harmonic Mean 0.23**
Concentration Milligrams Liter^{-1}	**Life-Stage**	**Duration**
0.23		42D
0.23		42D

As mentioned above, NMFS only selected chronic toxicity data in the core data file with a reported concentration type of total ammonia. Since total ammonia is the sum of the two forms of ammonia (NH_4^+ and NH_3), NMFS assumes that the data with a reported concentration type of total ammonia were normalized by EPA. For these toxicity studies, temperature and pH were not reported in the core data files; therefore verification regarding normalization was not possible and creates uncertainty. Therefore, as an additional step to address this uncertainty and to assess the potential for chronic toxic effects of ammonia to the listed species considered in this opinion using an additional line of evidence, NMFS used four ACRs to estimate a NOEC for ammonia:

(1) The rank ordered ACR of 3.26 for ammonia used in EPA's BE, Table 2.6.2.1.7.10.

Based on the ACR used in EPA's BE, and using the minimum species mean salmonid fish LC_{50} test concentration for ammonia in Table 2.6.2.1.7.1 and divided that concentration to derive an estimated NOEC concentration to

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assess the potential for chronic toxic effects, NMFS calculated an estimated NOEC of 2.2 mg/L.

(2) The EPA reassessment of the 3.26 ACR used in the BE of 4.26 for ammonia, Table 2.6.2.1.7.11.

Based on the EPA reassessment ACR of 4.26, and using minimum species mean salmonid fish LC_{50} test concentration for ammonia in Table 2.6.2.1.7.1 and divided that concentration to derive an estimated NOEC concentration to assess the potential for chronic toxic effects, NMFS calculated an estimated NOEC of 1.7 mg/L.

(3) The ranked ordered data only for fishes—instead of the fish and invertebrate rank ordered data EPA used to calculate the ammonia ACR of 3.26 in the BE as NMFS considers a fish-based ACR the best scientific surrogate to estimate a NOEC for fishes for ammonia, Table 2.6.2.1.7.12.

Based on the adjusted ACR calculation, NMFS calculated an ACR of 5.8. The NMFS then selected minimum species mean salmonid fish LC_{50} test concentration for ammonia in Table 2.6.2.1.7.1 and divided that concentration by the adjusted ACR to derive an estimated NOEC concentration to assess the potential for chronic toxic effects, NMFS calculated an estimated NOEC of 1.3 mg/L.

(4) The ranked ordered data for fishes, without the catfish ACR value, instead of the fish and invertebrate rank ordered data EPA used to calculate the ammonia ACR of 3.26 in the BE as NMFS considers a fish-based ACR the best scientific surrogate to estimate a NOEC for fishes for ammonia, Table 2.6.2.1.7.13.

Based on the adjusted ACR calculation, without the catfish ACR value, NMFS calculated an ACR of 3.6. The NMFS then selected minimum species mean salmonid fish LC_{50} test concentration for ammonia in Table 2.6.2.1.7.1 and divided that concentration by the adjusted ACR to derive an estimated NOEC concentration to assess the potential for chronic toxic effects, NMFS calculated an estimated NOEC of 1.3 mg/L.

NMFS selected the minimum species mean value from the salmonid fishes LC_{50} test concentration for ammonia as it represents the lowest acute toxicity concentration that predicts the greatest risk of adverse toxic effects to field-exposed fishes, predicted at 38.4 percent (Table 2.6.2.1.7.14),), and therefore permits an assessment that considers the "worst case" exposure scenario.

The results of the ACR-NOEC analysis produced one NOEC below the chronic criterion, one NOEC equal to the chronic criterion, and two NOECs above the chronic chronic criterion.

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Table 2.6.2.1.7.10 ACR-NOEC toxicity analysis for salmonid fishes, eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set BE pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **16.6**	**ACR** **3.26**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Salmonid LC_{50}** **7.3 Milligrams Liter^{-1}**
Endpoint/Effect **ACR-NOEC**	**pH** **6.97**	**ACR EPA BE**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	
2.2	40.0 G; SWIMMING FISH	

Table 2.6.2.1.7.11 ACR-NOEC toxicity analysis for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set BE pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **16.6**	**ACR** **4.26**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Salmonid LC_{50}** **7.3 Milligrams Liter^{-1}**
Endpoint/Effect **ACR-NOEC**	**pH** **6.97**	**ACR EPA Reassessment**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	
1.7	40.0 G; SWIMMING FISH	

Table 2.6.2.1.7.12 ACR-NOEC toxicity analysis for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set BE pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **16.6**	**ACR** **5.8**
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	**Salmonid LC_{50}** **7.3 Milligrams Liter^{-1}**
Endpoint/Effect **ACR-NOEC**	**pH** **6.97**	**ACR Fish Only**
Concentration **Milligrams Liter^{-1}**	**Life-Stage**	
1.3	40.0 G; SWIMMING FISH	

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Table 2.6.2.1.7.13 ACR-NOEC toxicity analysis for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		**Data Set BE pH-adjusted**
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature 16.6**	**ACR 3.6**
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	**Salmonid LC_{50} 7.3 Milligrams Liter^{-1}**
Endpoint/Effect ACR-NOEC	**pH 6.97**	**ACR Fish Only (without catfish ACR value)**
Concentration Milligrams Liter^{-1}	**Life-Stage**	
2	40.0 G; SWIMMING FISH	

Ammonia Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

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The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, at face value, none of toxicity studies reported LC_{50} concentrations that are less than the acute criterion concentration for ammonia, which implies that listed species exposed to waters equal to criterion concentrations may not suffer acute toxic effects. However, since some of the LC_{50} data had concentrations near the acute criterion concentration, NMFS added an additional step to its analysis for ammonia to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality (Table 2.6.2.1.7.14). This assessment involved taking the acute criterion of 5.6 mg/L and dividing it by each LC_{50} concentration in Table 2.6.2.1.7.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.1.7.1, predicts a magnitude of effect ranging from a low of an $LC_{3.2}$ at a concentration of 89.3 mg/L to a high of an $LC_{38.4}$ at a concentration of 7.3 mg/L. In other words, the acute criterion of 5.6 mg/L has an equivalent toxicity potential predicted to kill 3.2 percent to 38.4 percent, with a median toxicity potential of an $LC_{8.6,}$ of the exposed test population, and therefore by inference, field-exposed individuals.

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Table 2.6.2.1.7.14 Relative percent mortality analysis for salmonid fishes, Eulachon, and green sturgeon for freshwater ammonia.

Criterion Freshwater Ammonia		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute 5.6 Milligrams Liter^{-1}	**Temperature 2.1-18.7° Celsius**	
Criterion Concentration Chronic 1.7 Milligrams Liter^{-1}	**Hardness NR**	
Endpoint/Effect LC_{50}	**pH 6.00-9.46**	
Concentration Milligrams Liter^{-1}	**Relative Percent Mortality (acute criterion/LC_{50})**	
7.3	38.4	
12.6	22.5	
14.0	20.0	
18.4	15.2	
22.4	12.5	
22.4	12.5	
22.7	12.3	
23.0	12.2	
23.6	11.9	
23.7	11.8	
24.4	11.5	
25.0	11.2	
25.6	11.0	
26.0	10.8	
27.0	10.4	
27.0	10.4	
27.0	10.4	
27.2	10.3	
27.7	10.1	
27.8	10.1	
27.9	10.1	
28.7	9.8	
28.8	9.7	
30.6	9.2	
31.6	8.9	
32.1	8.7	
32.2	8.7	
32.6	8.6	
32.7	8.6	

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Criterion Freshwater Ammonia		Data Set ECOTOX pH-adjusted
Criterion Concentration Acute **5.6 Milligrams Liter^{-1}**	**Temperature** **2.1-18.7° Celsius**	
Criterion Concentration Chronic **1.7 Milligrams Liter^{-1}**	**Hardness** **NR**	
Endpoint/Effect **LC_{50}**	**pH** **6.00-9.46**	
Concentration **Milligrams Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
33.7	8.3	
33.7	8.3	
33.8	8.3	
33.8	8.3	
34.0	8.3	
34.8	8.1	
35.5	7.9	
36.1	7.8	
36.5	7.7	
37.0	7.6	
37.4	7.5	
37.7	7.5	
37.8	7.4	
39.4	7.1	
39.4	7.1	
40.5	6.9	
41.0	6.9	
42.6	6.6	
43.3	6.5	
46.4	6.1	
47.0	6.0	
48.8	5.8	
49.5	5.7	
56.1	5.0	
65.8	4.3	
68.6	4.1	
89.3	3.2	

For the chronic criterion assessment, a number of chronic toxicity studies reported concentrations that are less than the chronic criterion concentration for ammonia, which implies that listed species exposed to waters equal to criteria concentrations will suffer chronic toxic effects. The NMFS only selected chronic toxicity data in the core data file with a reported

concentration type of total ammonia. For these toxicity studies, temperature and pH were not reported in the core data file, therefore verification regarding normalization was not possible and creates uncertainty. Nonetheless, the toxicity assessments in Table 2.6.2.1.7.10, which produced a concentration less than the chronic criterion concentration, through Table 2.6.2.1.7.13, with one NOEC equal to the chronic criterion, and two NOECs above the chronic criterion, indicates that listed species exposed to waters equal to chronic criterion concentrations will suffer chronic toxic effects.

When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle, the considerations of the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes, the relative percent mortality analysis, and the chronic toxicity assessment, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. The chemical form of ammonia in water consists of two species, a larger component which is the ammonium ion (NH_4^+) and a smaller component which is the non-dissociated or un-ionized ammonia (NH_3) molecule. The sum of the two forms is usually expressed as total ammonia-nitrogen. The ratio of un-ionized ammonia to ammonium ion, dependent upon both pH and temperature, generally increases 10-fold for each rise of a single pH unit, and approximately 2-fold for each 10°C rise in temperature over the 0 to 30°C range (Erickson 1985 as cited in EPA 2008). Toxicity of ammonia to aquatic life was initially thought to arise largely from the small uncharged NH_3 molecule (Wuhrmann and Woker 1948, Downing and Merkens 1955 as cited in EPA 2008), however more recent information indicates that ammonia is more toxic as the hydrogen ion concentration [H^+] increases (pH decreases), at least below a pH of 7.3 (Armstrong *et al.* 1978, Tomasso *et al.* 1980 as cited in EPA 2008).

Acute effects likely are primarily neurological in origin resulting from severe metabolic alterations of the central nervous system (Smart 1978, Levi *et al.* 1974 as cited in EPA 2008). The toxic symptoms observed in fish acutely exposed to ammonia include hyper-excitability, coma, convulsions and hyperventilation. Sublethal effects can be quite extensive, and include reduced food uptake and growth inhibition, diuresis and ion imbalance, inflammation and degeneration of the gills and other tissues, changes in the oxygen-carrying capacity of the blood, and increased susceptibility to disease (Russo 1985 as cited in EPA 2008).

Physiological effects on salmonid fishes has been reported to occur at concentrations as low as 0.005 mg/L (42-day exposure) (Burrows 1964), but other studies on mortality recorded thresholds as varied as 0.03 mg/L (2-day exposure) (Herbert 1956) and 5 mg/L (3-day exposure) (Holland *et al.* 1960). The physiological harm recorded in Burrows' study (1964) was gill hyperplasia that may additionally result in bacterial gill disease. Gill hyperplasia is a response by epithelial cells and lamellae in the gills of fishes to irritations that may include uncontrolled cell growth, thinning, and fusion of lamellae (Burrows 1964, Post 1971, Dauba *et al.* 1992).

Reductions in growth on rainbow trout may occur as low as 0.0023 mg/L (120-day exposure) (Soderberg *et al.* 1983) or as high as 1.3 mg/L (365-day exposure) (Smith 1972). The NMFS assumes that growth reductions occurred throughout the exposure during the Soderberg *et al.*

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study (1983) and that gill hyperplasia occurred throughout the exposure in Burrows' study (1964).

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for ammonia is likely to result in sublethal effects to listed species considered in this opinion.

Summary of Effects: Ammonia. The available evidence for indicates that listed species exposed to water equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (high-intensity), reduced growth (high-intensity), impairment of essential behaviors related to successful rearing and migration (moderately-high-intensity), cellular trauma (high-intensity), physiological trauma (high-intensity), impairment of biochemical processes (high-intensity), and sublethal effects—ACR-NOEC analysis—(moderately-high-intensity to high-intensity).

2.6.2.2 Metal and Elemental Pollutants: Analysis of Individual Compounds

In this section, the effects of each metal and elemental toxic substance listed in Table 1.1 are identified, and the proposed criteria are compared with available toxicity data that describe the results of toxicity tests. The analysis identifies potential effects on listed species and their critical habitat of each of the criteria that would be expected to occur if water concentrations were equal to or less than the proposed criteria. Where possible, effects on the food sources of listed species, and effects related to bioaccumulation, are also identified. The following analysis focuses on each parameter individually.

2.6.2.2.1 Aluminum[8]

Aluminum Criteria. The proposed criteria concentrations of aluminum are 750 µg/L and 87 µg/L for acute and chronic criteria, respectively.

Tables 2.6.2.2.1.1 through 2.6.2.2.1.9 report toxicity data from the ECOTOX database for freshwater aluminum, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

[8] On August 9, 2012, EPA sent NMFS a letter withdrawing their request for consultation on Oregon's acute and chronic aluminum criteria as "EPA has determined that the BE submitted to NMFS in January 2008 incorrectly described the proposed federal action under consultation for aluminum (*i.e.*, CW A § 303(c)(3) approval of Oregon's submission of aluminum criteria). Specifically, Oregon's submitted description of the pollutant refers to aluminum in waters with a pH of 6.5- 9.0, but a footnote in the criterion itself indicates that the criterion is meant to apply to waters with pH less than 6.6 and hardness less than 12 mg/L (as $CaCO_3$)." Due to the court-ordered deadline of August 14, 2012, NMFS did not have time to modify its opinion to exclude acute and chronic aluminum from the document. The NMFS acknowledges EPA's revision to the proposed action, however, and notes it does not anticipate EPA will carry out the RPA for aluminum in light of this change. The NMFS will await a further request from EPA relating to EPA's potential future actions regarding Oregon's aluminum criteria.

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Table 2.6.2.2.1.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		**Data Set ECOTOX**
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 12-15.7° Celsius**	**Arithmetic Mean 4684**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 6.6-115.8 mg/L $CaCO_3$**	**Geometric Mean 2247**
Endpoint/Effect LC_{50}	**pH 6.5-8.58**	**Harmonic Mean 867**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
170	FERTILIZATION THROUGH 4 DAY POST/	28D
400	EGGS	28D
400	EGGS	28D
445	ALEVINS	96H
510	EGG	28D
1620	JUVENILE, 1-3 G	96H
2860	JUVENILE, 1-3 G	96H
3600	JUVENILE	
5310	JUVENILE, 1-3 G	96H
5330	JUVENILE, 1-3 G	96H
6220	JUVENILE, 1-3 G	96H
7400		24H
7900		
9600	5.52 CM, 33 G	24H
18500	NR	48H

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Table 2.6.2.2.1.2 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 1-15° Celsius**	**Arithmetic Mean 2870**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 17-280 mg/L $CaCO_3$**	**Geometric Mean 408**
Endpoint/Effect Mortality	**pH 6.5-8.7**	**Harmonic Mean 134**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
20	EYED EGG STAGE	8D
20	CLEAVAGE EMBRYO, EYED	8D
50	CLEAVAGE EMBRYO, EYED	8D
57	EYED EGG	15D
57	EYED EMBRYO - LARVAE	30D
57	FRY	45D
57	FRY	60D
88	EYED EMBRYO - LARVAE	15D
90	118-355 G, 22-31 CM FORK LENGTH	96H
100	CLEAVAGE EMBRYO, EYED	8D
100	CLEAVAGE EMBRYO, EYED	8D
100	SMOLT, 1 YR, 65 G, 195 MM	23D
169	EYED EMBRYO - LARVAE	15D
169	EYED EMBRYO - LARVAE	30D
169	FRY	45D
242	EYED EGG	15D
242	EYED EGG	15D
242	EYED EGG	15D
242	EYED EGG	30D
242	37 D, JUVENILE	15D
268	0.2 G, 30 D	56H
283	EYED EMBRYO - LARVAE	60D
330	ADULT, 1518 G, 51.5 CM TL	48H
350	EYED EGG	15D
350	EYED EMBRYO - LARVAE	30D
350	FRY	45D
350	FRY	60D
500	CLEAVAGE EMBRYO, EYED	8D
720	JUVENILE, 1-3 G	16D
910	118-355 G, 22-31 CM FORK LENGTH	24H
910	118-355 G, 22-31 CM FORK LENGTH	48H
910	118-355 G, 22-31 CM FORK LENGTH	72H

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 1-15° Celsius**	**Arithmetic Mean 2870**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 17-280 mg/L $CaCO_3$**	**Geometric Mean 408**
Endpoint/Effect Mortality	**pH 6.5-8.7**	**Harmonic Mean 134**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
910	118-355 G, 22-31 CM FORK LENGTH	96H
1000	CLEAVAGE EMBRYO, EYED	8D
1680	JUVENILE, 1-3 G	16D
9100	118-355 G, 22-31 CM FORK LENGTH	24H
9100	118-355 G, 22-31 CM FORK LENGTH	48H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
10000	5-10 CM	24H
50000	50-80 MM	96H

Table 2.6.2.2.1.3 LT_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 4245**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness NR**	**Geometric Mean 3261**
Endpoint/Effect LT_{50}	**pH 6.52-8.99**	**Harmonic Mean 1837**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration/Days**
513	11 WK	43.9
5140	FINGERLINGS, 6 WK	7.5
5140	11 WK	38.9
5200	FINGERLINGS, 6 WK	2.98
5230	6 MO	31.96

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Table 2.6.2.2.1.4 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		**Data Set ECOTOX**
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 182**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 245-255 mg/L $CaCO_3$**	**Geometric Mean 148**
Endpoint/Effect NOEC/Growth/Behavioral	**pH 6.5-6.6**	**Harmonic Mean 121**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
57	EYED EMBRYO - LARVAE	30D
88	FRY	45D
88	FRY	60D
169	FRY EYED EMBRYO - LARVAE	30D
169	FRY	60D
350	EYED EMBRYO - LARVAE	30D
350	FRY	60D

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Table 2.6.2.2.1.5 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 11-19° Celsius**	**Arithmetic Mean 191**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 15-280 mg/L $CaCO_3$**	**Geometric Mean 103**
Endpoint/Effect Growth	**pH 6.52-8.99**	**Harmonic Mean 1.1**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.05	FINGERLINGS, 6-24 WK	222H
38.1	JUVENILE, 7.5-8.5 G	34D
52	6 WK-6 MO	113D
57	FRY	30D
57	FRY	45D
57	FRY	60D
88	FRY	30D
88	FRY	45D
88	FRY	60D
100	SMOLT, 1 YR, 65 G, 195 MM	16D
169	FRY	30D
169	FRY	45D
169	FRY	60D
242	EYED EGG	15D
242	EYED EGG	30D
242	37 D, JUVENILE	15D
268	0.2 G, 30 D	3D
283	EYED EMBRYO - LARVAE	45D
350	FRY	30D
350	FRY	45D
350	FRY	60D
740	JUVENILE, 1-3 G	16D

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Table 2.6.2.2.1.6 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 11-13° Celsius**	**Arithmetic Mean 270**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 15-103.5 mg/L $CaCO_3$**	**Geometric Mean 200**
Endpoint/Effect Behavioral	**pH 6.5-8.14**	**Harmonic Mean 148**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
57	FRY	60D
88	FRY	60D
169	FRY	60D
242	EYED EGG	30D
242	37 D, JUVENILE	15D
350	FRY	60D
740	JUVENILE, 1-3 G	16D

Table 2.6.2.2.1.7 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 11.5-19° Celsius**	**Arithmetic Mean 100**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness NR**	**Geometric Mean 100**
Endpoint/Effect Cellular	**pH 7.2**	**Harmonic Mean 100**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
100	SMOLT, 1 YR, 65 G, 195 MM	16D
100	SMOLT, 1 YR, 65 G, 195 MM	16D
100	SMOLT, 1 YR, 65 G, 195 MM	16D

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Table 2.6.2.2.1.8 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 1-19° Celsius**	**Arithmetic Mean 149**
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness NR**	**Geometric Mean 105**
Endpoint/Effect Physiological	**pH 6.5-7.1**	**Harmonic Mean 81**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
59	SMOLT, 30 G	48H
59	SMOLT, 30 G	2D
330	ADULT, 1518 G, 51.5 CM TL	48H

Aluminum Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

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The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to aluminum, NMFS added an additional step to its analysis for aluminum to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality (Table 2.6.2.2.1.9). This assessment involved taking the acute criterion of 750 µg/L and dividing it by each 24H, 48H, and 96H duration LC_{50} concentrations in Table 2.6.2.2.1.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.1.1, predicts a magnitude of effect ranging from a low of an LC_2 at a concentration of 18,500 µg/L to a high of an LC_{84} at a concentration of 445 µg/L. In other words, the acute criterion of 750 µg/L has an equivalent toxicity potential predicted to kill 2 percent to 84 percent, with a median toxicity potential of an $LC_{15,}$ of the exposed test population, and therefore by inference, field-exposed individuals.

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Table 2.6.2.2.1.9 Relative percent mortality analysis for salmonid fishes, eulachon, and green sturgeon for freshwater aluminum.

Criterion Freshwater Aluminum		Data Set ECOTOX
Criterion Concentration Acute 750 Micrograms Liter^{-1}	**Temperature 12° Celsius**	
Criterion Concentration Chronic 87 Micrograms Liter^{-1}	**Hardness 6.6-115.8 mg/L $CaCO_3$**	
Endpoint/Effect LC_{50}	**pH 6.5-8.58**	
Concentration Micrograms Liter^{-1}	**Relative Percent Mortality (acute criterion/LC_{50})**	
445	84	
1620	23	
2860	26	
5310	7	
5330	7	
6220	6	
7400	5	
9600	4	
18500	2	

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for aluminum, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for aluminum, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Aluminum is one of the most abundant elements in the earth's crust and occurs in many rocks and ores, but never as a pure metal. The presence of aluminum ions in streams may result from industrial wastes but is more likely to come from the wash water of drinking water treatment plants. Many aluminum salts are readily soluble; however, there are some that are very insoluble. Those that are insoluble will not exist long in surface water, but will precipitate and settle. Waters containing high concentrations of aluminum can become toxic to aquatic life if the pH is lowered (as in acid rain).

Aluminum, like other metals, generally acts as a surface active toxicant, exerting its damage by binding to anionic sites on respiratory surfaces of aquatic animals, such as a fish gill (Wood *et al.* 1997 as cited in EPA 2008). The physiological manifestation of these deleterious surface effects at the gill include both ionoregulatory and respiratory effects. Ionoregulatory effects of

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aluminum predominate at low pH (*e.g.*, less than pH 5.0) and include a mechanism similar to hydrogen ion toxicity alone, *i.e.*, sodium uptake blockade (Playle *et al.* 1989 as cited in EPA 2008). In moderately acidic water, it is generally the respiratory effects of aluminum that predominate. Respiratory effects are likely the result of the physical coating of the gills which occurs when aluminum-rich water passes into the more basic gill microenvironment (Gensemer and Playle 1999 as cited in EPA 2008). Overall, chronic aluminum toxicity to fish species is substantially greater at low pH, particularly for salmonids. For many fish, aluminum toxicity increases with early life stage such that eggs and endogenously-feeding alevins are generally less sensitive than exogenous-feeding swim-up larvae (Buckler *et al.* 1985, DeLonay *et al.* 1993 as cited in EPA 2008). Holtze (1984) concluded that rainbow trout were most sensitive to aluminum during the yolk sac and swim-up fry stages and least sensitive to aluminum during the cleavage stage. Holtze (1984) also concluded that aluminum was beneficial to the survival of cleavage embryos at pH 4.5. Therefore, aluminum at extreme low pH (pH <5) can protect against the direct toxic effects, and aluminum criteria based on higher pH values may undermine embryo survival. Several factors ameliorate aluminum toxicity at low pH, including, but probably not limited to: calcium ion (Brown 1983, Ingersoll *et al.* 1990 as cited in EPA 2008), silicic acid (Birchall *et al.* 1989 as cited in EPA 2008), fluoride (Wilkinson *et al.* 1990 as cited in EPA 2008), and dissolved and natural organic matter (Parkhurst *et al.* 1990; Roy and Campbell 1997 as cited in EPA 2008).

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for aluminum is likely to result in sublethal effects to listed species considered in this opinion.

Summary of Effects: Aluminum. The available evidence for indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (high-intensity), reduced growth (high-intensity), impairment of essential behaviors related to successful rearing and migration (moderately-high-intensity), cellular trauma (moderate intensity), and physiological trauma (moderately-high-intensity).

2.6.2.2.2 Arsenic

Arsenic Criteria. The proposed criteria for dissolved concentrations of trivalent arsenic equal 340 µg/L and 150 µg/L for acute and chronic criteria, respectively.

Tables 2.6.2.2.2.1 through 2.6.2.2.2.5 report toxicity data from the ECOTOX database for freshwater arsenic, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.2.2.2.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion Freshwater Arsenic		Data Set ECOTOX
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 57845**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 16698**
Endpoint/Effect LC_{50}	**pH 7.4-10.2**	**Harmonic Mean 342**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
10	NR	96H
25	NR	24H
25	57 G	24H
170	FERTILIZATION THROUGH 4 DAY POST	28H
420	EGGS	28H
420	EGGS	144H
490	NR	24H
490	EGG	4H
1400	FINGERLING, 5.7 G	22H
3510	FRY	96H
3830	JUVENILE, 7-8 WK, 0.20 G	96H
4050	JUVENILE, 7-8 WK, 0.34 G	96H
5000	EGG	96H
7500	NR	96H
8200	FINGERLING, 5.7 G	96H
8200	FINGERLING, 5.7 G	30H
10800	YY, 2 mo, 51-76 MM TL	96H
10800	YY, 2 mo, 51-76 MM TL	96H
11600	JUVENILE, 45.5 MM, 0.51 G	96H
12200	3.5 G	144H
12200	3.5 G	96H
12700	JUVENILE, 64.3 MM, 2.49 G	96H
12700	JUVENILE, 64.3 MM, 2.49 G	28H
13500	2.6G	96H
14500	JUVENILE, 39.0 MM, 0.41 G	96H
14500	JUVENILE, 39.0 MM, 0.41 G	24H
17700	FINGERLING, 5.7 G	24H
18100	FRY, 1.99 G	96H
18100	FRY, 1.99 G	96H
19300	FRY, 0.50 G	96H
19300	FRY, 0.50 G	96H

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Criterion Freshwater Arsenic		Data Set ECOTOX
Criterion Concentration Acute **340 Micrograms Liter^{-1}**	**Temperature** **5.4-15.1° Celsius**	**Arithmetic Mean** **57845**
Criterion Concentration Chronic **150 Micrograms Liter^{-1}**	**Hardness** **44-343 mg/L $CaCO_3$**	**Geometric Mean** **16698**
Endpoint/Effect **LC_{50}**	**pH** **7.4-10.2**	**Harmonic Mean** **342**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
21900	JUVENILE, 7-11 WK, 1.85 G	96H
23700	ADULT, 18 MO, 200.0 MM, 84.7 G	96H
25300	JUVENILE, 7-11 WK, 0.97 G	96H
25600	3.5 G	144H
25600	3.5 G	144H
32500	JUVENILE, 10-12 WK, 0.41 G	96H
32500	JUVENILE, 10-12 WK, 0.41 G	28H
34000	YOUNG OF YR, 0.5-3.0 G	24H
35000	JUVENILE, 5-6 WK, 0.85 G	96H
42100	ALEVIN, 29.8 MM, 0.24 G	96H
46000	FRY, 1.99 G	24H
47000	FRY, 1.03 G	24H
49400	JUVENILE, 18-22 WK, 0.47 G	96H
49400	JUVENILE, 18-22 WK, 0.47 G	24H
50300	FRY, 0.50 G	24H
55400	FRY, 0.50 G	96H
55400	FRY, 0.50 G	96H
56000	JUVENILE, 18-22 WK, 0.47 G	96H
56100	JUVENILE, 7-10 WK, 1.04 G	96H
56100	JUVENILE, 7-10 WK, 1.04 G	24H
62900	FRY	24H
69900	ALEVIN, 20.8 MM, 0.10 G	96H
70000	FRY, 0.50 G	96H
70000	FRY, 0.50 G	96H
70600	2.6G	96H
74000	JUVENILE, 10-12 WK, 0.41 G	96H
118000	JUVENILE, 7-10 WK, 1.04 G	96H
120000	FRY, 1.03 G	96H
120000	FRY	96H
120000	FRY, 1.03 G	96H
120000	FRY	96H
130000	FRY, 0.50 G	24H
216000	ALEVIN	24H
224000	FRY	24H

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Criterion Freshwater Arsenic		Data Set ECOTOX
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 57845**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 16698**
Endpoint/Effect LC_{50}	**pH 7.4-10.2**	**Harmonic Mean 342**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
360000	ALEVIN	96H
360000	ALEVIN	24H
547000	ALEVIN	96H

Table 2.6.2.2.2.2 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion Freshwater Arsenic		Data Set ECOTOX
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 69883**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 62625**
Endpoint/Effect Mortality	**pH 7.4-10.2**	**Harmonic Mean 57167**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
35000	JUVENILE, 5-6 WK, 0.85 G	11W
43300	JUVENILE, 7-11 WK, 0.97 G	4D
60000	ALEVIN	11W
61000	JUVENILE, 5-6 WK, 0.85 G	40D
75000	ALEVIN	10D
145000	ALEVIN	4D

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Table 2.6.2.2.2.3 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion Freshwater Arsenic		Data Set ECOTOX
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 31332**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 14894**
Endpoint/Effect Growth	**pH 7.4-10.2**	**Harmonic Mean 9305**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3510	FRY	11W
3830	JUVENILE, 7-8 WK, 0.20 G	12W
4050	JUVENILE, 7-8 WK, 0.34 G	12W
6630	JUVENILE, 7-8 WK, 0.34 G	8W
9200	JUVENILE, 7-8 WK, 0.20 G	12W
11600	JUVENILE, 45.5 MM, 0.51 G	8W
17100	ADULT, 18 MO, 200.0 MM, 84.7 G	8W
21100	FRY	11W
23500	ALEVIN, 15.0 MM, 0.02 G	2W
23900	ADULT, 18 MO, 200.0 MM, 84.7 G	4D
25300	JUVENILE, 7-11 WK, 0.97 G	2W
41600	JUVENILE, 7-11 WK, 1.85 G	8W
216000	ALEVIN	8W

Table 2.6.2.2.2.4 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion Freshwater Arsenic		Data Set ECOTOX
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 19933**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 19764**
Endpoint/Effect Behavioral	**pH 7.4-10.2**	**Harmonic Mean 19605**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
17800	ADULT, 18 MO, 200.0 MM, 84.7 G	8W
18300	ADULT, 18 MO, 200.0 MM, 84.7 G	8W
23700	ADULT, 18 MO, 200.0 MM, 84.7 G	12W

Table 2.6.2.2.2.5 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater arsenic.

Criterion Freshwater Arsenic		**Data Set ECOTOX**
Criterion Concentration Acute 340 Micrograms Liter^{-1}	**Temperature 5.4-15.1° Celsius**	**Arithmetic Mean 21900**
Criterion Concentration Chronic 150 Micrograms Liter^{-1}	**Hardness 44-343 mg/L $CaCO_3$**	**Geometric Mean 21900**
Endpoint/Effect Physiological	**pH 7.4-10.2**	**Harmonic Mean 21900**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
21900	JUVENILE, 7-11 WK, 1.85 G	1D

Arsenic Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

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The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to arsenic, NMFS added an additional step to its analysis for arsenic to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 340 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.2.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.2.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 547,000 µg/L to a high of an LC_{100} at a concentration of 10 µg/L. In other words, the acute criterion of 340 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{0.7}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for arsenic, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for arsenic, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute toxic effects, but may not suffer chronic toxic effects.

Sublethal Effects. Arsenic occurs naturally in aquatic environments in trace amounts. Background concentrations in freshwater streams are usually less than 1 µg/L (Moore and

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Ramamoorthy 1984). Mining, smelting, manufacturing, electric power plants, pesticides, agricultural defoliants, and battery manufacturing and reclamation plants are all significant anthropogenic sources of arsenic (Sorensen 1991).

Arsenic is a suspected carcinogen in fish. It is associated with necrotic and fibrous tissues and cell damage, especially in the liver. Arsenic can result in immediate death through increased mucus production and suffocation. Other effects include anemia and gallbladder inflammation. The toxicity of arsenic is influenced by a number of factors including fish size, water temperature, pH, redox potential, organic matter, phosphate content, suspended solids, presence of other toxicants, speciation of the chemical itself, and the duration of exposure (Dabrowski 1976, Eisler 1988a, McGeachy and Dixon 1989, Sorensen 1991, Cockell *et al.* 1992, Rankin and Dixon 1994, Woodward *et al.* 1994). Juvenile salmonids have been determined to be more sensitive to arsenic toxicity than alevins (Buhl and Hamilton 1990, 1991). Trivalent arsenic (arsenite) tends to be more toxic than other forms of arsenic, and inorganic forms of arsenic (including pentavalent) are typically more toxic than organic forms (EPA 1985b, Eisler 1988a, Sorensen 1991). Chronic toxicity in fish appears to be inversely proportional to water temperature under certain experimental conditions (McGeachy and Dixon 1990). Relatively little data exists that would allow establishment of separate standards for the multiple forms of arsenic that can occur in the aquatic environment.

Arsenic is bioconcentrated by organisms but is not biomagnified through the food chain (Eisler 1988a). Toxic effects of arsenic to aquatic life are significantly modified by numerous biological and abiotic factors (EPA 1985b as cited in EPA 2008) such as water temperature, hardness, pH, organic content, phosphate concentration, suspended solids, *etc.* (Eisler 1988a as cited in EPA 2008). In general, inorganic forms of arsenic are more toxic than organic forms to aquatic biota (EPA 1999). Early life stages are most sensitive, and large interspecies differences are recorded, even among those closely related taxonomically (Eisler 1988a as cited in EPA 2008). In fish, tolerance of arsenic appears to increase with temperature (McGeachy and Dixon 1990 as cited in EPA 2008), whereas in invertebrates the opposite is true (Bryant *et al.* 1985 as cited in EPA 2008). Effects of arsenic toxicity to aquatic biota include: avoidance and immobility in freshwater snails; and anemia, gall bladder inflammation, liver degeneration, reduced hemoglobin, and reduced success in seaward migration of fish.

Birge *et al.* (1981) reported an LC_{10} of 134 µg/L for rainbow trout embryos after a 28-day exposure (Birge *et al.* 1981). However, it is likely that the corresponding 4-day (the longest duration that a concentration can be between the acute and chronic criteria) LC_{10} would be higher, because in general test organisms mortality increases with exposure duration. Also, those results could have been influenced by bioaccumulation, such that the toxicity response was chronic rather than acute in nature. The studies reviewed indicate that acute toxicity, including to alevins, occurs at concentrations that are significantly higher than the proposed acute criterion (*e.g.*, Buhl and Hamilton 1990).

The results of Birge *et al.* (1978, 1981) suggests that chronic arsenic toxicity occurs to developing embryos of salmonids at concentrations below the proposed chronic criterion. For example, rainbow trout embryos exposed to arsenic for 28 days (4 days post-hatching) at 12°C to13°C and a hardness of 93 mg/L to 0.5 mg/L $CaCO_3$ in static tests (Birge *et al.* 1978, 1981) at

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concentrations of 40 µg/L to 42 µg/L were associated with the onset of embryo mortality. Acclimation appears to enhance resistance to chronic arsenic toxicity (Dixon and Sprague 1981, EPA 1985b), which may explain in part why no studies were found by NMFS that indicate chronic toxicity occurs to juvenile and adult salmonids at concentrations near or below the proposed chronic criterion. Studies reviewed in Eisler (1988) and EPA (1985a) indicate that chronic effects do not occur in other life stages until concentrations are at least about an order of magnitude higher than the levels determined by Birge *et al.* (1978, 1981) to be detrimental to developing embryos.

Chronic exposure results in bioaccumulation of arsenic to toxic levels in fish, with most accumulating in the liver, pancreas, spleen, and kidneys, and relatively little in muscle tissues. Trivalent arsenic appears to bioaccumulate more readily than pentavalent, but there is no consistent relation with fish size or condition (EPA 1985b, Sorensen 1991). The inorganic pentavalent form appears to be the most stable in aquatic systems (Eisler 1988a). Bioaccumulation rates vary with fish species, where planktivorous fish are more likely to concentrate arsenic than omnivorous or piscivorous fishes (Hunter *et al.* 1981, Sorensen 1991). Diet appears to be a significant pathway for arsenic accumulation in salmonids (Oladimeji *et al.* 1984), although developing embryos have also been documented to uptake arsenic (Dabrowski 1976). Spehar *et al.* (1980) determined that rainbow trout did not accumulate arsenic significantly at concentrations above the proposed criteria. Similarly, Robinson *et al.* (1995) found no evidence of arsenic uptake or accumulation from water in rainbow and brown trout.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for arsenic is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Data on arsenic toxicity to aquatic macroinvertebrates are limited. What information does exist (EPA 1980b, 1985b; Eisler 1988a; Canivet *et al.* 2001) suggests that the proposed criterion should not result in acute or chronic toxicity to most aquatic macroinvertebrate taxa. Results reported in Eisler (1988a) suggest that gammarid amphipods may experience acute toxicity at concentrations of trivalent arsenic that are below the chronic criterion. Canivet *et al.* (2001) similarly determined greater sensitivity of a gammarid amphipod compared with other taxa tested, with a 240-hour LC_{50} of 200 µg/L, which is higher than the proposed chronic criterion. There is evidence that benthic invertebrate communities respond to elevated chronic arsenic levels by shifting community composition to pollution-tolerant taxa, while overall biomass does not change significantly (Canfield *et al.* 1994; Beltman *et al.* 1999). A shift to pollution tolerant taxa could change the availability of forage items. Primary aquatic invertebrate taxa used for food by rearing juvenile Chinook and steelhead (*e.g.*, stoneflies, mayflies, and caddisflies; EPA 1980b, 1985b; Canivet *et al.* 2001) do not appear to exhibit chronic effects at concentrations below the proposed chronic criterion. Irving *et al.* (2008) exposed mayfly nymphs to tri- and pentavalent arsenic in water-only exposures for 12 days. For trivalent arsenic, the threshold of growth effects was about 100 µg/L. However, arsenic levels accumulated by the mayfly nymphs in their study (1.2 to 4.6 µg/g dry wt) were far lower than those reported from stream locations with far lower water concentrations of arsenic but that had elevated arsenic in diet or sediments, suggesting that the water-only exposures may have underrepresented likely environmental exposures.

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Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Arsenic. The available evidence for arsenic indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), interference in physiochemical processes (moderate intensity), interruption of ecological interactions (low intensity), and changes in pathological stress (low intensity).

2.6.2.2.3 Cadmium

Cadmium Criteria. The proposed acute and chronic criteria for cadmium are 2.0 µg/L and 0.25 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.3.1 through 2.6.2.2.3.7 report toxicity data from the ECOTOX database for freshwater cadmium, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.2.2.3.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 9.6-17.3° Celsius**	**Arithmetic Mean 18**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 9.2-410.5 mg/L $CaCO_3$**	**Geometric Mean 9**
Endpoint/Effect LC_{50}	**pH 6.84-7.63**	**Harmonic Mean 5.5**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.16	45 MM, 36 G	96H
1.32	3 MO, 0.21 G	96H
1.62	3 MO, 0.21 G	96H
1.64	50 MM	96H
1.77	50 MM	96H
1.84	3 MO, 0.21 G	72H
2.2	45 MM, 36 G	96H
2.29	45 MM, 36 G	96H
2.31	45 MM, 36 G	96H
2.51	3 MO, 0.21 G	72H
2.69	3 MO, 0.21 G	72H
2.71	3 MO, 0.21 G	24H
2.78	JUVENILE, 5 MO, 3.0 G, 7.0 CM	120H
2.81	1-2 G, JUVENILE	96H
2.89	50 MM	96H
3.08	PARR, 6.96 G, 8.6 CM	200H
3.16	ALEVIN, 20.8 MM, 0.10 G	96H
3.3	3 MO, 0.21 G	48H
3.35	50 MM	96H
3.68	2.36-3.01 G	96H
3.68	2.36-3.01 G	168H
4.06	3.9-6.8 CM FORK LENGTH	96H
4.45	SWIM-UP, 0.17 G	96H
4.45	SWIM-UP, 0.17 G	200H
4.62	0.5 G, JUVENILE	96H
4.66	130 MM	96H
4.77	3 MO, 0.21 G	96H
4.97	45 MM, 36 G	96H
5.06	45 MM, 36 G	96H

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Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	**Arithmetic Mean** **18**
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	**Geometric Mean** **9**
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	**Harmonic Mean** **5.5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
5.17	3 MO, 0.21 G	48H
5.36	50 MM	96H
5.47	SWIM-UP, 0.23 G	200H
5.47	SMOLT, 68.19 G, 18.8 CM	200H
5.54	3 MO, 0.21 G	48H
5.59	50 MM	96H
5.92	8.8 G	96H
5.92	8.8 G	72H
5.96	3 MO, 0.21 G	72H
6.16	SWIM-UP, 0.23 G	96H
6.84	PARR, 11.58 G, 9.6 CM	200H
7.1	ALEVINS-BUTTONED-UP FRY	96H
7.17	JUVENILE, 41.6-45.8 MM/	96H
7.87	SMOLT, 32.46 G, 14.4 CM	200H
7.89	8.8 G	48H
7.99	136 MM	96H
8.21	135 MM	96H
8.43	JUVENILE, 6.42-6.66 MM/	96H
8.71	NR	408H
9.2	2.36-3.01 G	96H
9.92	SMOLT, 68.19 G, 18.8 CM	96H
9.92	SMOLT, 32.46 G, 14.4 CM	96H
10.46	NR	96H
11.97	PARR, 11.58 G, 9.6 CM	96H
12.12	ALEVIN, 14.3 MM, 0.01 G	96H
12.65	ALEVIN 29.8 MM, 0.24 G	96H
13.13	NR	215H
14.26	0.5 G, JUVENILE	96H
15.5	3 MO, 0.21 G	24H
15.54	40 MM	96H
16.85	1.0 G, 32 MM	96H

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Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	**Arithmetic Mean** **18**
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	**Geometric Mean** **9**
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	**Harmonic Mean** **5.5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
21	3.9-6.8 CM FORK LENGTH	96H
23	PARR, 6.96 G, 8.6 CM	96H
23	SWIM-UP, 0.17 G	96H
23	SWIM-UP, 0.23 G	96H
23	PARR	96H
23	SMOLT, 32.46 G,	96H
23	PARR, 11.58 G, 9.6 CM	96H
23		96H
23	ADULT	96H
23	ALEVIN, 0.05 G	96H
23	ALEVIN	96H
25		96H
25.84	3 MO, 0.21 G	48H
31	130 MM	96H
41	ALEVIN, 20.8 MM, 0.10 G	96H
41	JUVENILE,	96H
41	ALEVIN 29.8 MM, 0.24 G	96H
43.5	1-2 G, JUVENILE	96H
43.5	0.5 G, JUVENILE	96H
44	3 MO, 0.21 G	96H
44	ALEVIN, 14.3 MM, 0.01 G	96H
44.4	8.8 G	96H
83.1	FRY, 0.14 G	7D
90	YEARLING	96H
140	JUVENILE	96H
211	FRY, 1.03 G	96H

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Table 2.6.2.2.3.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 9.6-17.3° Celsius**	**Arithmetic Mean 5**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 29-410.5 mg/L $CaCO_3$**	**Geometric Mean 3**
Endpoint/Effect NOEC/Mortality/Growth/Reproduction	**pH 6.84-7.63**	**Harmonic Mean 2**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.80	50 MM	100D
1.25	JUVENILE	100D
1.29	50 MM	100D
2.10	JUVENILE	100D
2.15	50 MM	100D
2.34	L. Superior	
2.74	JUVENILE	100D
3.06	YEARLING, 50-70 G	
4.29	2 YR, FEMALE ADULT	60W
6.83	2 YR, FEMALE ADULT	
7.37	West Coast	100D
26.66	NR	10D

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Table 2.6.2.2.3.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 5-20° Celsius**	**Arithmetic Mean 27**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 9.2-427 mg/L $CaCO_3$**	**Geometric Mean 4**
Endpoint/Effect NOEC/Mortality	**pH 6.6-8.28**	**Harmonic Mean 2**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.58	50 MM	100D
0.94	JUVENILE	100D
1.14	50 MM	100D
1.55	JUVENILE	100D
2.29	136 MM	1M
2.29	130 MM	96H
2.37	NR	1M
2.75	50 MM	100D
2.95	136 MM	1M
3.63	130 MM	96H
3.69	EGG	2M
3.83	YEARLING, 50-70 G	33M
3.86	JUVENILE	100D
5.17	1.0 G, 32 MM	96H
5.43	1.0 G, 32 MM	96H
11.5	EGGS	19M
12.8	EGGS	1M
41.55	NR	10D
407.7	NR	10D

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Table 2.6.2.2.3.4 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **5-20° Celsius**	**Arithmetic Mean** **21**
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **20-390 mg/L $CaCO_3$**	**Geometric Mean** **1.8**
Endpoint/Effect **Growth**	**pH** **6.6-8.28**	**Harmonic Mean** **0.3**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.02	EMBRYO	
0.10	NR	84D
0.47	ALEVIN	46D
0.59	18.2-23.5 CM, 51.2-114.9 G	112D
0.71	JUVENILE, 59 G	30D
0.71	JUVENILE, 59 G	30D
0.98	NR	84D
1	24 H, ALEVIN	13W
1.38	ALEVIN	46D
1.98	JUVENILE	30D
2.82	EGG-FRY	12W
3.59	EGG-FRY	12W
4	FINGERLING, 7.8 G	10W
4	FINGERLING, 7.8 G	10W
6.16	ALEVIN, 21 D	21D
6.4	ADULT, 375 G, 31.0 CM	178D
7.15	ADULT, 582 G	30D
7.15	ADULT, 582 G	30D
341	80 G	1W

Table 2.6.2.2.3.5 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 5-20° Celsius**	**Arithmetic Mean 79**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 10.1-320 mg/L $CaCO_3$**	**Geometric Mean 24**
Endpoint/Effect Physiological	**pH 6.6-8.28**	**Harmonic Mean 2**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.27	200-250 G	120D
1.98	JUVENILE	30D
12.7	NR	24H
67	20.01 CM FL, 101.54 G	48H
77.9	3-4 YR	7D
77.9	3-4 YR	24H
128	15-20 CM	24H
267	56 G	24H

Table 2.6.2.2.3.6 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 5-20° Celsius**	**Arithmetic Mean 1**
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 44-250 mg/L $CaCO_3$**	**Geometric Mean 0.9**
Endpoint/Effect Reproductive	**pH 6.6-8.28**	**Harmonic Mean 0.8**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.56	270 D, ADULT, FEMALE	65W
0.63	270 D, ADULT, FEMALE	65W
1.13	YEARLING, 50-70 G	33M
1.96	270 D, ADULT, FEMALE	80W

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Cadmium Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

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than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to cadmium, NMFS added an additional step to its analysis for cadmium to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 2 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.3.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.3.1, predicts a magnitude of effect ranging from a low of an $LC_{0.5}$ at a concentration of 211 µg/L to a high of an LC_{86} at a concentration of 1.16 µg/L (Table 2.6.2.2.3.7). In other words, the acute criterion of 2 µg/L has an equivalent toxicity potential predicted to kill 0.5 percent to 86 percent, with a median toxicity potential of an $LC_{12.7}$, of the exposed test population, and therefore by inference, field-exposed individuals.

Table 2.6.2.2.3.7 Relative percent mortality analysis for salmonid fishes, eulachon, and green sturgeon for freshwater cadmium.

Criterion Freshwater Cadmium		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 2 Micrograms Liter^{-1}	**Temperature 9.6-17.3° Celsius**	
Criterion Concentration Chronic 0.25 Micrograms Liter^{-1}	**Hardness 9.2-410.5 mg/L $CaCO_3$**	
Endpoint/Effect LC_{50}	**pH 6.84-7.63**	
Concentration Micrograms Liter^{-1}	**Relative Percent Mortality (acute criterion/LC_{50})**	
1.16	86.2	
1.32	75.8	
1.62	61.7	
1.64	61.0	
1.77	56.5	
1.84	54.3	
2.2	45.5	
2.29	43.7	
2.31	43.3	
2.51	39.8	
2.69	37.2	
2.71	36.9	
2.78	36.0	
2.81	35.6	

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Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
2.89	34.6	
3.08	32.5	
3.16	31.6	
3.3	30.3	
3.35	29.9	
3.68	27.2	
3.68	27.2	
4.06	24.6	
4.45	22.5	
4.45	22.5	
4.62	21.6	
4.66	21.5	
4.77	21.0	
4.97	20.1	
5.06	19.8	
5.17	19.3	
5.36	18.7	
5.47	18.3	
5.47	18.3	
5.54	18.1	
5.59	17.9	
5.92	16.9	
5.92	16.9	
5.96	16.8	
6.16	16.2	
6.84	14.6	
7.1	14.1	
7.17	13.9	
7.87	12.7	
7.89	12.7	
7.99	12.5	

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Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
8.21	12.2	
8.43	11.9	
8.71	11.5	
9.2	10.9	
9.92	10.1	
9.92	10.1	
10.46	9.6	
11.97	8.4	
12.12	8.3	
12.65	7.9	
13.13	7.6	
14.26	7.0	
15.5	6.5	
15.54	6.4	
16.85	5.9	
21	4.8	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
23	4.3	
25	4.0	
25.84	3.9	
31	3.2	
41	2.4	
41	2.4	

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Criterion Freshwater Cadmium		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **2 Micrograms Liter^{-1}**	**Temperature** **9.6-17.3° Celsius**	
Criterion Concentration Chronic **0.25 Micrograms Liter^{-1}**	**Hardness** **9.2-410.5 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}**	**pH** **6.84-7.63**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
41	2.4	
43.5	2.3	
43.5	2.3	
44	2.3	
44	2.3	
44.4	2.3	
83.1	1.2	
90	1.1	
140	0.7	
211	0.5	

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for cadmium, which implies that listed species exposed to waters equal to criteria concentrations will not be protected from acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for cadmium, which implies that listed species exposed to waters equal to criteria concentrations will be protected from acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Cadmium occurs naturally in the aquatic environment, and is considered one of the most toxic of metals to fish (Sorensen 1991). Uses of cadmium include electroplating, pigments, plastic stabilizers, batteries, and electronic components. In aquatic systems, cadmium is taken up quickly by sediments but is readily remobilized through a variety of physical, chemical, and biological processes, and can even be transported from aquatic to terrestrial food webs by emerging insects (Currie *et al.* 1997). Cadmium is a known teratogen, carcinogen and a probable mutagen to which freshwater organisms are considered the most sensitive. Effects of cadmium toxicity on freshwater organisms include spinal deformities; inhibited respiration; blood plasma and other hematological changes, decreased growth, inhibited reproduction and immune response; temporary immobility; and population alterations. Salmonid species are particularly sensitive to cadmium compared to other fish species (Sorensen 1991,

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Brent and Herricks 1998, Sanchez-Dardon *et al.* 1999). Chronic sublethal exposure to cadmium does not appear to significantly influence growth in juvenile salmonids (Hollis *et al.* 2000b).

Toxicity of cadmium to aquatic organisms varies with the type and life stage of organisms, presence of other toxicants, duration of exposure, and hardness. Acute mechanisms of cadmium toxicity to fish do not appear to be the same as chronic mechanisms. In acute tests cadmium accumulates in gill tissue to a greater extent than elsewhere, whereas in chronic tests at lower concentrations, cadmium accumulates more in liver and kidney tissue. The principal acute effect is gill toxicity leading to an aquatic organism's inability to breathe. Cadmium toxicity increases with water temperature (Moore and Ramamoorthy 1985, Eisler 1985a, EPA 1985c, Sorensen 1991), which is known to also stress listed species in many parts of Oregon. The presence of zinc, which has similar chemical properties, and selenium have been shown to antagonize cadmium toxicity, whereas other metals do not appear to compete with cadmium for enzyme receptors in aquatic organisms.

Stubblefield *et al.* (1999) determined that adult rainbow trout that were acclimated to elevated cadmium levels would survive sudden increases to higher concentrations at a higher rate than fish that were not acclimated. The non-acclimated fish exhibited an incipient lethal level (ILL: threshold level of exposure to toxic substances beyond which 50% of a test population of organisms cannot survive) of 6.1µg/L at a hardness of 280 mg/L, which is below the proposed acute criterion. However, the ILL was determined to occur after 187 hours of exposure, which is more than the maximum permitted under the proposed criterion (96 hours under the chronic criterion). On the basis of this study, therefore, an adverse effect would be expected at the proposed concentration if the concentrations occurred unmonitored or uncorrected for more than 7 days in waters where background concentrations are well below the chronic criterion. Young-of-year rainbow trout fared better and were determined to be less sensitive than adults (Stubblefield *et al.* 1999). Older (age 1+) fish were not tested, but could exhibit a response between that of the young of year and adult test fish, and thus also be susceptible to acute toxicity at cadmium levels below the proposed acute criterion when they are not suitably acclimated to background levels.

Birge *et al.* (1981) determined reduced survival (52% vs. 90% for control) of 4 day old larvae of rainbow trout after their parents were exposed to a concentration of 0.2 µg/L at 102 mg/L hardness for 18 months, which is well below the proposed chronic criterion. The exposed parents had tissue concentrations that were roughly seven times that of the control fish, indicating the potential for bioaccumulative effects on subsequent reproductive success.

Cadmium has been shown to cause neurotoxic effects in fish. These neurotoxic effects may manifest themselves through altered behavior, which in turn may predict more serious effects including reduced growth, reproductive failure, and death. Hyperactivity probably is the most widely observed maladaptive behavior reported from cadmium exposed fish, with several reports involving a variety of fish species during long-term cadmium exposures. Most fish that exhibited hyperactive behavior in long-term exposures ultimately died. Hyperactivity is detrimental to small fish because it makes them more likely to be seen and attacked by predatory fish. Similarly, hyperactive predatory fish have lower success rates in detecting, orienting to, attacking, and swallowing prey.

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Cadmium is bioconcentrated by organisms but is not biomagnified through the food chain (Eisler 1985a as cited in EPA 2008). Toxicity of cadmium to aquatic organisms varies with water hardness, alkalinity, the type and life stage of organisms, presence of organic matter, presence of other toxicants, and the duration of exposure (EPA 1999 as cited in EPA 2008). Cadmium is a known teratogen, carcinogen, and a probable mutagen to freshwater organisms (Eisler 1985a as cited in EPA 2008). Effects of cadmium toxicity to freshwater organisms include spinal deformities, inhibited respiration, immune response, temporary immobility, decreased growth, inhibited reproduction, decreased survival, and population alterations (Sorensen 1991, Eisler 1985a, Brent and Herricks 1998, Sanchez-Dardon *et al.* 1999 as cited in EPA 2008). A known mechanism of cadmium toxicity to fish is suppression of calcium uptake (Verbost *et al.* 1987 as cited in EPA 2008). Calcium is vital for growth in fish (Pelgrom *et al.* 1997) as cited in EPA 2008, and bone repair mechanisms are probably inhibited due to the hypocalcemic effect of cadmium (DWAF, 1996 as cited in EPA 2008).

Cadmium bioaccumulates in numerous fish species including salmonids, where tissue concentrations reflect exposure levels and duration, hardness, and presence of other ions (*e.g.*, zinc). Besser *et al.* (2001) determined a mean bioaccumulation factor of 3.4 from aquatic macroinvertebrates to trout. Omnivorous fish tend to accumulate higher levels of cadmium than carnivorous fish, such as salmonid fishes, and bottom-feeding fish tend to accumulate more cadmium than free-swimming fish feeding in the water column. Evidence suggests that significant biomagnification is exhibited predominantly by species at lower trophic levels in aquatic ecosystems, whereas fish are able to depurate cadmium rapidly (Eisler 1985a, Sorensen 1991). Uptake occurs through both dissolved and particulate forms (Enk and Mathis 1977, Sorensen 1991). Cadmium tends to form stable complexes with metallothionein that have long half-lives and a tendency to accumulate with age in exposed organisms. Accumulation appears to occur primarily in the gills, liver, kidneys, and gastrointestinal tract (Sorenson 1991, Besser *et al.* 2001. Hollis *et al.* 2001). As such, long lived species tend to be at a higher risk from chronic low-level dietary cadmium exposure. Rainbow trout exposed to cadmium have been determined to contain residues in kidney, spleen, gill, muscle, and bone tissues that increase in concentration with duration of exposure (Camusso and Balestrini 1995). In contrast, Saiki *et al.* (1995) found no evidence of cadmium biomagnification in steelhead on the Upper Sacramento River. McGeer *et al.* (2000) reported evidence that cadmium accumulates inside rainbow trout continuously over time with continued exposure, because it not as actively regulated as copper and zinc are by the organism. McGeer used concentrations below the proposed criteria. It is unknown whether bioaccumulation also occurs when concentrations are below the proposed criteria for extended periods, but the possibility appears to exist.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for cadmium is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Amphipods are sometimes abundant in lakes and slow-moving rivers. Amphipods are benthic crustaceans that occupy an intermediate position in aquatic food webs between detritus and predators, such as salamanders and salmonids (Mathias 1971). Aquatic macroinvertebrates, which serve as significant food sources for early life stages of listed species as well as for other aquatic organisms that are in turn prey items, are sensitive to both dissolved and particulate cadmium. Invertebrate communities in rivers appear to respond to

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elevated cadmium levels in sediments and water by changing composition to pollution-tolerant taxa, rather than by reducing overall biomass (Canfield *et al.* 1994, Clements and Kiffney 1994). Hare and Shooner (1995) determined that population densities of the two most abundant colonizing insects (chironomidae) in a small lake were unrelated to cadmium gradients in sediments, even though they accumulated the metal in proportion to its concentration in the sediment. Interstitial water cadmium concentrations ranged up to 17 µg/L, suggesting that the two taxa were relatively insensitive to exposure to cadmium levels less than that. Larvae of another chironomid were negatively correlated with cadmium gradient. These tests suggest that the lower abundance at high concentrations is more likely due to toxicity effects than avoidance of cadmium-rich sediments. It is not clear if these effects also occur at water-borne cadmium levels that are below the proposed chronic criterion, although this possibility should not be discounted because of the potential for bioaccumulation.

Cadmium contained in bed sediments appears to be bioavailable to benthic invertebrates, was found to be elevated in benthic invertebrates in field studies conducted in metals-contaminated streams (*e.g.*, Enk and Mathis 1977, Woodward *et al.* 1994). Kiffney and Clements (1996) determined an inverse relation existed between aquatic macroinvertebrate body size and survival at water-borne cadmium levels in excess of the proposed acute criterion, which could partially counter the effects of bioaccumulation when invertebrates are exposed to contaminated sediments. Indirect effects of elevated cadmium levels to listed species therefore include reduced production of larger invertebrate taxa that could influence the availability of food for larger juvenile salmonids, and ingestion of bioconcentrated cadmium by fry and juveniles of all sizes. It is unknown if similar effects occur at concentrations below the proposed chronic criterion.

Salmonids and other fish readily prey upon amphipods, probably consuming them in rough proportion to their abundance relative to other vulnerable invertebrates. For example, in the lower Snake River in Washington and Idaho, amphipods contributed 2.7 and 7.9 percent of identifiable prey categories found in the stomachs of juvenile Chinook salmon and steelhead, respectively from Lower Granite Reservoir, (7th and 5th most important prey categories, respectively) (Karchesky and Bennett 1999).

One invertebrate, the amphipod *Hyalella azteca*, seems particularly sensitive to cadmium. It is the only species with a species mean chronic value that is lower than the NTR of 2.2 µg/L. Six chronic tests with *Hyalella* were analyzed by Mebane (2006). In all six tests, adverse effects would be expected at a concentration of 1 µg/L. Mebane (2006) attempted to evaluate several lines of evidence to evaluate if the predicted effects to this species would have appreciable adverse effects on fish populations or other indirect effects on aquatic ecosystems in the Pacific Northwest. These efforts included (1) reviews of role of *Hyalella azteca* in aquatic food chains, (2) occurrences of *Hyalella azteca* in waters with elevated cadmium concentrations, and (3) simulating effects of cadmium to a natural, coldwater *Hyalella azteca* population.

Potential effects of cadmium at chronic criteria concentrations on wild populations of *Hyalella azteca* were also estimated using mathematical population models that integrate toxicity testing results with ecological theory. The modeling predicted that at the NTR chronic criteria (2.2 µg/L at the scenario hardness of 280 mg/L), quasi-extinction of the population was highly likely, with >80% probability of a >98% population decline occurring during the 6-year modeling scenario.

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Applying these modeling results to the Oregon chronic criterion (0.25 µg/L) results in a marginal increased extinction risk.

Toxicity to Food Organisms Summary. The available evidence indicates that the chronic criterion for cadmium is likely to result in sublethal effects to listed species considered in this opinion.

Summary of Effects: Cadmium. The available evidence for indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (high intensity), reduced growth (moderately-high-intensity), impairment of essential behaviors related to successful rearing and migration (moderate intensity), physiological trauma (moderate intensity), and reproductive failure (moderate intensity).

2.6.2.2.4. Chromium (III)

Chromium (III) Criteria. The proposed acute and chronic criteria for chromium (III) are 570 µg/L and 74 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.4.1 through 2.6.2.2.4.2 report toxicity data from the ECOTOX database for freshwater CR (III), except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.4.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater chromium III.

Criterion Freshwater Chromium III		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 570 Micrograms Liter^{-1}	**Temperature 11.9-14.5° Celsius**	**Arithmetic Mean 10099**
Criterion Concentration Chronic 74 Micrograms Liter^{-1}	**Hardness 25-44 mg/L $CaCO_3$**	**Geometric Mean 9825**
Endpoint/Effect LC_{50}	**pH 5.45-7.33**	**Harmonic Mean 9558**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
7762	NR	96H
12436	NR	96H

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Table 2.6.2.2.4.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater chromium III.

Criterion Freshwater Chromium III		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **570 Micrograms Liter^{-1}**	**Temperature** **11.9-14.5° Celsius**	**Arithmetic Mean** **53**
Criterion Concentration Chronic **74 Micrograms Liter^{-1}**	**Hardness** **25 mg/L $CaCO_3$**	**Geometric Mean** **53**
Endpoint/Effect **NOEC/Growth/Mortality**	**pH** **5.45-7.33**	**Harmonic Mean** **53**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
53	NR	72H

2.6.2.2.5 Chromium (VI)

Chromium (VI) Criteria. The proposed acute and chronic criteria for chromium (VI) are 570 µg/L and 74 µg/L, respectively.

Tables 2.6.2.2.5.1 through 2.6.2.2.5.2 report toxicity data from the ECOTOX database for freshwater CR (VI), except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.5.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater chromium VI.

Criterion Freshwater Chromium VI		Data Set ECOTOX
Criterion Concentration Acute **16 Micrograms Liter^{-1}**	**Temperature** **3.5-19° Celsius**	**Arithmetic Mean** **98129**
Criterion Concentration Chronic **11 Micrograms Liter^{-1}**	**Hardness** **34-46 mg/L $CaCO_3$**	**Geometric Mean** **68333**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **7-8**	**Harmonic Mean** **44884**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
12079	NR	96H
27201	NR	96H
27496	NR	96H
37905	NR	96H
69722	NR	96H
74239	NR	96H

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Criterion Freshwater Chromium VI		Data Set ECOTOX
Criterion Concentration Acute **16 Micrograms Liter^{-1}**	**Temperature** **3.5-19° Celsius**	**Arithmetic Mean** **98129**
Criterion Concentration Chronic **11 Micrograms Liter^{-1}**	**Hardness** **34-46 mg/L $CaCO_3$**	**Geometric Mean** **68333**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **7-8**	**Harmonic Mean** **44884**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
98200	NR	96H
109002	NR	96H
141408	NR	96H
201310	NR	96H
280852	NR	96H

Table 2.6.2.2.5.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater chromium VI.

Criterion Freshwater Chromium VI		Data Set ECOTOX
Criterion Concentration Acute **16 Micrograms Liter^{-1}**	**Temperature** **3.5-19° Celsius**	**Arithmetic Mean** **100**
Criterion Concentration Chronic **11 Micrograms Liter^{-1}**	**Hardness** **34-46 mg/L $CaCO_3$**	**Geometric Mean** **52**
Endpoint/Effect **NOEC/Growth**	**pH** **7-8**	**Harmonic Mean** **24**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
9.6	EG-JV	7M
10	EG-JV	7M
10	EG-JV	7M
13	LV-JV	110D
13	LV-JV	110D
49	NR	
49	NR	
192	NR	
192	NR	
192	NR	
192	NR	
192	NR	
192	NR	

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Chromium III and Chromium VI Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

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than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to chromium (III) and chromium (VI), NMFS added an additional step to its analysis for chromium (III) and chromium (VI) to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 570 µg/L for chromium (III) and 16 µg/L for chromium (VI) and dividing it by each LC_{50} concentrations in Table 2.6.2.2.4.1 and Table 2.6.2.2.5.1, respectively, to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.4.1 and Table 2.6.2.2.5.1, respectively, predicts a magnitude of effect ranging from a low of an $LC_{2.3}$ at a concentration of 12,436 µg/L to a high of an $LC_{3.7}$ at a concentration of 7,762 µg/L for chromium (III), and a magnitude of effect of an LC_{zero} at a concentration of 12,074 µg/L and 280,852 µg/L for chromium (VI). In other words, the acute criterion of 570 µg/L for chromium (III) has an equivalent toxicity potential predicted to kill 2.3 percent to 3.7 percent, with a median toxicity potential of an LC_3, of the exposed test population, and therefore by inference, field-exposed individuals. The acute criterion of 16 µg/L for chromium (VI) has an equivalent toxicity potential predicted to kill zero percent.

In summary, none of toxicity studies reported concentrations that are less than the acute criterion concentration for chromium (III), which implies that listed species exposed to waters equal to criterion concentrations may not suffer acute toxic effects. Conversely, the single toxicity data reported for chronic effects is less than the chronic criterion concentration for chromium (III), which implies that listed species exposed to waters equal to criteria concentrations will suffer chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute and chronic toxic effects.

None of the toxicity studies reported concentrations that are less than the acute criterion for chromium (VI), which implies that listed species exposed to waters equal to the acute criterion concentration may not suffer acute toxic effects. A number of toxicity studies reported concentrations that are less than the chronic criteria for chromium (VI), and a number of toxicity studies reported concentrations that are greater than the chronic criterion for chromium (VI), which implies that listed species exposed to waters equal to the chronic criterion concentration will suffer chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration may not suffer acute toxic effects, but will suffer chronic toxic effects.

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Sublethal Effects (Chromium III and Chromium VI). Chromium (III) (the trivalent form) is much less toxic than chromium (VI) (the hexavalent form), which is a strong oxidizing agent and reduces readily to the former. Younger life stages of aquatic biota tend to be more sensitive to the toxic effects of chromium (VI). Effects of toxicity include abnormal enzyme activities, altered blood chemistry, lowered resistance to disease, reduced growth, behavioral modifications, disrupted feeding, cell damage in the gills and other tissues, and osmoregulatory upset in outmigrating smolts. The toxicity of chromium is influenced by pH, water temperature, concentrations of other contaminants, and fish age and sex (EPA 1980d, Eisler 1986).

chromium (III) toxicity is influenced by water hardness. It is unclear is the same if true for chromium (VI), which is significantly more toxic. Hexavalent chromium exists in solution in an anionic rather than cationic form, and therefore does not precipitate in an alkaline solution.

The acute standards for chromium (III) are unique from analogous standards for the other metals of concern because the total recoverable to dissolved conversion factor (0.316) is substantially smaller. Depending on the sampling location and the receiving water characteristics (that may promote dissolution of particulate chromium), this means that the proposed criterion could permit discharge of total recoverable chromium (III) at levels that result in higher than assumed, and potentially toxic, dissolved levels downstream.

Chromium may be present in the environment in both inorganic and organic forms. Inorganic forms do not biomagnify; it is unknown whether organic forms of chromium biomagnify (Eisler 1986). Chromium toxicity to aquatic biota is significantly influenced by abiotic variables such as water hardness, temperature, pH, salinity, species, life stage, and presence of mixtures (Eisler 1986). Sensitivity to chromium varies widely, even among closely related species (Eisler 1986). Effects of chromium toxicity to freshwater organisms include reduced survival in freshwater invertebrates (including molluscs), and reduced growth, reduced disease resistance, behavioral modifications, disrupted feeding, cell damage in the gills, osmoregulatory upset in outmigrating smolts, and reduced reproduction and survival in freshwater fish (Anestis and Neufeld 1986, Eisler 1986 and EPA 1999).

Hexavalent chromium is more toxic than the trivalent form because its oxidizing potential is high and it easily penetrates biological membranes (Steven *et al.* 1976, Taylor and Parr 1978 as cited in EPA 2008). At high concentrations, both forms of chromium can be a mutagen, teratogen, and carcinogen (Eisler 1986b as cited in EPA 2008). Although CrIII is the most common form found in nature, the known harmful effects of chromium is speculated to be related to the reduction of hexavalent chromium (chromium VI) to chromium III intracellularly as it crosses the cell membrane and forms complexes with intracellular macromolecules (Danielsson *et al.* 1982, R.O.W. Sciences, 1997 as cited in EPA 2008).

There are more toxicity test data available for the hexavalent form of chromium (VI), probably reflecting its greater toxicity. Insufficient data are available to evaluate the potential harm of the chromium (III) criterion for salmonids specifically. Toxicity data for salmonid fishes indicate that acute and chronic toxicity of chromium (VI) is likely to occur to juvenile salmonids when dissolved concentrations are at or below the chromium (VI) numeric criteria.

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Billard and Roubaud (1985) determined that the viability of rainbow trout sperm (but not ova) were adversely affected when exposed directly to a chromium (VI) concentration equal to 5 µg/L, which is well below the chronic criterion of 11 µg/L. Reproductive effectiveness is likely to be reduced if this water concentration occurs during spawning.

There is evidence that invertebrates and fishes bioaccumulate hexavalent chromium when exposed to ambient water concentrations that are above the chronic criterion. Uptake is influenced by water temperature, pH, other contaminant concentrations, fish age and sex, and tissue type (EIFAC 1983, Eisler 1986). Calamari *et al.* (1982) determined that liver, kidney, and muscle tissue concentrations of chromium were elevated in rainbow trout after 30, 90, and 180 days of exposure to 200 µg/L. The fish subsequently were able to depurate some, but not all, of the accumulated chromium within 90 days after exposure ended. At higher concentrations (>2000 µg/L), chromium is known to also accumulate in gill and digestive tract tissues of rainbow trout (Eisler 1986). Gill accumulation appears to continue with exposure, whereas the other tissues may achieve equilibrium in 2 to 4 days. Residues tend to remain high in the liver and kidneys in test fish during post-exposure periods. Eisler (1986) reported that tissue concentrations in excess of 4 mg/kg dry weight were presumptive evidence of chromium contamination, but the biological significance was not clear.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for chromium (III) and chromium (VI) is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Aquatic invertebrates other than cladocerans have been determined in a limited number of studies to experience acute and chronic effects at concentrations below the acute and chronic criterion, respectively, for both chromium (III) and (VI). Data in EPA (1980d) indicate reduced survival and reproductive impairment of daphnids at chromium (III) and (VI) concentrations as low as 4 and 10 µg/L, respectively. These concentrations are less than the proposed chronic criterion for each respective valency. Most studies have determined toxicity to daphnids occurs at higher concentrations than the criterion, however. Data summarized in EPA (1980d), EIFAC (1983), and Eisler (1986) suggest that other invertebrate taxa that juvenile fishes may feed on generally died at chromium (III) and (VI) concentrations that are well above the acute criterion. More recently, Canivet *et al.* (2001) determined 240-hour chromium (VI) LC_{50}s for larvae of a trichopteran and an ephemeropteran that were well above the proposed acute and chronic criteria.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for chromium (III) and chromium (VI) are unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Chromium (III) and Chromium (VI). The available evidence for chromium (III) and chromium (VI), respectively, indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity, for chromium III, and low intensity for chromium VI) and reduced growth (moderately-high-intensity, for chromium III and chromium VI).

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2.6.2.2.6 Copper

Copper Criteria. The proposed acute and chronic criteria for copper are 13 µg/L and 9 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.6.1 through 2.6.2.2.6.11 report toxicity data from the ECOTOX database for freshwater copper, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters, the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.6.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-16° Celsius**	**Arithmetic Mean 145**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 8-495 mg/L $CaCO_3$**	**Geometric Mean 96**
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.0**	**Harmonic Mean 59**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
5.70	4.2 G, 7.4 CM	96H
5.96	4.2 G, 7.4 CM	96H
9.14	YEARLING, 10-18 MO	96H
9.14	LARVAE	96H
11.56	PA	4D
12.85	10 G	96H
18.03	2.6 G	96H
19.32	1.7 G	96H
20.62	YEARLING, 10-18 MO	96H
21.20	LARVAE	96H
23.90	4.3 G	96H
25.45	PA	4D
25.49	3 MO, 1.35 G	96H
25.65	25.6 G, 13.4 CM	96H
27.55	FRY, 0.139 G, 2.87 CM	96H
30.13	2-3 YR	96H
30.48	176 MM	96H
31.26	FRY, 0.66 G	96H
31.61	2.2 G	96H
32.86	ALEVIN	96H

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Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
33.35	2.7 G	96H
33.41	2.5 G, 6.1 CM	96H
34.31	1.0 G	96H
35.15	ALEVIN	96H
36.39	FRY, 0.138 G, 2.96 CM	96H
37.88	4.4 G, 7.7 CM	96H
38.18	YEARLING, 10-18 MO	96H
38.58	160 MM	96H
39.63	3.1 G	96H
40.66	FRY, 0.87 G	96H
42.63	1.4 G	96H
42.83	1.0 G	96H
43.86	FY	4D
43.88	SMOLT, 5.5 G	96H
44.23	0.71 G	96H
45.86	9.7 G, 8.8 CM	96H
45.87	5.2 G, 8.5 CM	96H
46.38	3 MO, 1.35 G	96H
47.01	AD, MALE	96H
48.10	EM	96H
48.36	SMOLT, 4.69 G, 8.35 CM	96H
50.59	9.4 G, 9.2 CM	96H
51.40	9.4 G, 9.2 CM	96H
52.79	3 MO, 1.35 G	96H
52.79	24.9 G, 13.5 CM	96H
52.86	FRY, 1 G	96H
52.96	ALEVIN	96H
53.76	3.9-6.8 CM FORK LENGTH	96H
56.10	SWIM-UP, 0.17 G	96H
56.39	FRY, 1 G	96H
59.23	SMOLT, 4.8 G	96H

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Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-16° Celsius**	**Arithmetic Mean 145**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 8-495 mg/L $CaCO_3$**	**Geometric Mean 96**
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.0**	**Harmonic Mean 59**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
59.70	FRY, 0.132 G, 2.95 CM	96H
59.89	FRY, 0.136 G, 2.97 CM	96H
61.06	ALEVIN	96H
61.68	ALEVIN	96H
61.87	PA	4D
63.79	4.4 G, 8.1 CM	96H
64.68	3.2 G, 7.0 CM	96H
65.18	FY	4D
65.54	PA	4D
65.81	PA	4D
66.26	1.8 G	96H
67.63	YEARLING, 10-18 MO	96H
68.31	22.6 G, 11.8 CM	96H
69.01	4.0 G, 7.3 CM	96H
70.11	AD, MALE, ~2.7 KG	96H
70.46	JUVENILE, 5-6 WK, 0.85 G	96H
70.53	5.7 G, 8.9 CM	96H
71.12	SU, <3 mo, 32.1 MM, 0.23 G	96H
71.23	2.2 G	96H
71.38	JUVENILE, 7-8 WK, 0.20 G	96H
72.13	FRY, 1 G	96H
72.85	SMOLT, 4.63 G, 8.07 CM	96H
73.87	SU, <3 mo, 29.1 MM, 0.23 G	96H
73.96	167 MM	96H
74.56	1.1 G	96H
75.30	SMOLT, 68.19 G, 18.8 CM	96H
79.51	FINGERLING, 2.31 G, 6.61 CM	96H
81.10	JV, 14 mo	96H
84.84	PA	4D
86.51	YEARLING, 10-18 MO	96H
86.89	SMT	4D

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
87.12	3 MO, 1.35 G	96H
87.55	ALEVIN	96H
88.37	11.3 G, 9.7 CM	96H
88.91	ALEVIN	96H
90.44	3 MO, 1.35 G	96H
92.43	4.3 G	96H
92.74	4.4 G, 7.7 CM	96H
93.28	ALEVIN	96H
95.28	9.7 G, 8.8 CM	96H
99.44	PARR, 6.96 G, 8.6 CM	96H
99.68	2.7 G, 6.8 CM	96H
99.68	FINGERLING, 3.90 G, 7.17 CM	96H
99.68	25.6 G, 13.4 CM	96H
101.29	PA	4D
107.35	SMT	4D
108.15	0.80 G	96H
108.89	24.9 G, 13.5 CM	96H
111.19	FY, 2.36-3.01 G	96H
112.21	PARR, 11.58 G, 9.6 CM	96H
113.63	JV, 14 mo	96H
113.77	SU, <3 mo, 30.4 MM, 0.26 G	96H
114.29	11.5 G, 9.9 CM	96H
122.21	3.2 G	96H
123.91	4.9 CM	96H
124.94	2.1 G, 6.0 CM	96H
128.87	1.5 G	96H
130.72	JUVENILE, 18-22 WK, 0.87 G	96H
133.67	4.4 G, 8.1 CM	96H
138.04	1.6 G	96H
138.78	FRY, 1 G	96H
140.88	5.2 G, 8.5 CM	96H

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Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
145.69	11 CM, 13 G	96H
147.81	FRY	96H
148.58	1 G	96H
149.08	100.4(90-115)MM TL,10.6(7.5-14.5) G	96H
150.03	ALEVIN, NEWLY HATCHED	96H
150.52	ALEVINS-BUTTONED-UP FRY	96H
155.59	3 MO, 1.35 G	96H
163.37	16.47 CM FL, 53.85 G	96H
163.44	SU, <3 mo, 30.1 MM, 0.25 G	96H
171.44	2.7 G, 6.8 CM	96H
174.10	3.2 G, 7.0 CM	96H
174.36	JUVENILE	96H
177.75	JUVENILE, 7-10 WK, 0.60 G	96H
179.14	SU, <3 mo, 34.4 MM, 0.29 G	96H
179.91	3 MO, 1.35 G	96H
181.82	6.6 G	96H
183.34	FRY, 1 G	96H
184.58	JUVENILE, 6 G	96H
185.37	SU, <3 mo, 28.4 MM, 0.23 G	96H
189.35	ALEVIN	96H
194.30	3.2 G, 6.9 CM	96H
194.76	SU, <3 mo, 33.4 MM, 0.25 G	96H
199.96	JUVENILE, 7-8 WK, 0.34 G	96H
201.19	SMOLT, 32.46 G, 14.4 CM	96H
210.45	JUVENILE, 10-12 WK, 0.41 G	96H
212.83	FRY	96H
217.16	JUVENILE,29.1G WET WT,6.76 G DRY WT	96H
217.16	SMOLT, 5.5 G	96H
222.22	0.90 G	96H
227.44	SWIM-UP, 0.23 G	96H
228.59	ALEVIN, NEWLY HATCHED	96H

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Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **145**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	**Geometric Mean** **96**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	**Harmonic Mean** **59**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
229.06	FRY	96H
233.38	FINGERLING, 2.13 G, 6.67 CM	96H
240.00	ADULT, 16-18 CM	96H
240.02	18.7 G, 11.8 CM	96H
244.76	2.36-3.01 G	96H
250.22	5.7 G, 8.9 CM	96H
254.62	ALEVIN	200H
255.80	3 MO, 1.35 G	96H
264.28	PA	4D
266.36	FY, 2.36-3.01 G	96H
271.32	2.1 G, 6.0 CM	96H
274.31	3.2 G, 6.9 CM	96H
288.82	SU, <3 mo, 30.0 MM, 0.25 G	96H
289.33	12-16 CM	96H
301.90	3.2 G	96H
310.51	JUVENILE, 18-22 WK, 0.47 G	96H
313.32	FINGERLING, 3.28 G, 7.26 CM	96H
322.75	3 MO, 1.35 G	96H
326.37	3300 MG	96H
333.58	11.5 G, 9.9 CM	96H
346.63	JUVENILE, 10-12 WK, 0.81 G	96H
355.82	1.4 G	96H
376.54	YEARLING, 10-18 MO	96H
404.21	ALEVIN	96H
447.01	1.5 G	96H
447.48	ALEVIN, 0.05 G	96H
467.01	JUVENILE,3.9 G WET WT,0.94 G DRY WT	96H
475.90	1 G	96H
489.25	ALEVIN	96H
533.72	3 MO, 1.35 G	96H
533.72	JUVENILE,176 G WET WT,46.0 G DRY WT	96H

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-16° Celsius**	**Arithmetic Mean 145**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 8-495 mg/L $CaCO_3$**	**Geometric Mean 96**
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.0**	**Harmonic Mean 59**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
599.98	FRY, 1.60 G	96H
600.44	SMOLT, 5.5 G	96H
1160.10	2.6 G	96H

Table 2.6.2.2.6.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-16° Celsius**	**Arithmetic Mean 58**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 16-405 mg/L $CaCO_3$**	**Geometric Mean 35**
Endpoint/Effect NOEC/Growth	**pH 4.7-8.0**	**Harmonic Mean 25**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
6.57		
8		
9.5	MX, EG-YE, EXPOSED OR UNEXPOSED PAR	8M
11.4		
12	EGGS	6M
12	SACFRY, 9-11 D, 102.4-110.3 MG WT	15D
12	NR	24M
13.14		
14	FY OR SMT	30D
16	FY OR SMT	10D
16		
17	PA	29D
17.91	MX, EG-YE, EXPOSED OR UNEXPOSED PAR	8M
18	PA	8D
18	PA	29D

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Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	**Arithmetic Mean** **58**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **16-405 mg/L $CaCO_3$**	**Geometric Mean** **35**
Endpoint/Effect **NOEC/Growth**	**pH** **4.7-8.0**	**Harmonic Mean** **25**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
18	SWIM-UP, 0.23 G	96H
20	3 MO, 1.35 G	96H
20	3 MO, 1.35 G	96H
20.8		
21	PA	8D
21	PA	29D
21	PA	30D
21	FY OR SMT	60D
21.49		
22	PA	60D
22		
22.3		
23	SMOLT, 32.46 G, 14.4 CM	96H
24	ALEVIN, 0.05 G	96H
25	SACFRY, 9-11 D, 102.4-110.3 MG WT	15D
28	PA	60D
30	3 MO, 1.35 G	96H
30	3 MO, 1.35 G	96H
35	PARR, 11.58 G, 9.6 CM	96H
38	PA	9D
39.21		
40	PA	8D
40	FRY, 0.87 G	96H
41	FRY, 0.66 G	96H
41.47		
42.04		
50	3 MO, 1.35 G	96H
50	3 MO, 1.35 G	96H
54.69	FY OR SMT	60D
70.5	PA	60D
75	8 mo	10D
75	8 mo	10D

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-16° Celsius**	**Arithmetic Mean 58**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 16-405 mg/L $CaCO_3$**	**Geometric Mean 35**
Endpoint/Effect NOEC/Growth	**pH 4.7-8.0**	**Harmonic Mean 25**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
78.1	PA	60D
79	8 mo	10D
95	SMOLT, 4.69 G, 8.35 CM	96H
100	3 MO, 1.35 G	96H
100	3 MO, 1.35 G	96H
150	3 MO, 1.35 G	96H
200	FRY, 0.136 G, 2.97 CM	96H
200	3 MO, 1.35 G	96H
202	FINGERLING, 3.90 G, 7.17 CM	96H
213	FRY, 0.132 G, 2.95 CM	96H
216	SMOLT, 4.63 G, 8.07 CM	96H
240	SMOLT, 4.8 G	96H
312	8 mo	10D

Table 2.6.2.2.6.3 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-18° Celsius**	**Arithmetic Mean 91**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 135 mg/L $CaCO_3$**	**Geometric Mean 91**
Endpoint/Effect Behavioral	**pH 4.7-8.54**	**Harmonic Mean 91**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
65.8	SACFRY,9-11 D,102.4-110.3 MG WET WT	15D

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Table 2.6.2.2.6.4 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set 3
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 6.9-16.5° Celsius**	**Arithmetic Mean 6**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 20-240 mg/L $CaCO_3$**	**Geometric Mean 2**
Endpoint/Effect Behavioral/Olfaction	**pH 7.2-7.6**	**Harmonic Mean 0.98**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.18	JUVENILE	3H
0.59	JUVENILE	3H
0.75	JUVENILE	20MIN
0.79	JUVENILE	3H
1.6	JUVENILE	20MIN
2	JUVENILE	21D
2.1	JUVENILE	3H
2.4	JUVENILE	20MIN
5	JUVENILE	6D
10	ADULT	INDEFINITE
20	ADULT	INDEFINITE
25	ADULT	INDEFINITE

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Table 2.6.2.2.6.5 Sublethal toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set 2**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4-21° Celsius**	**Arithmetic Mean 4**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 20-120 mg/L $CaCO_3$**	**Geometric Mean 2**
Endpoint/Effect Sublethal/Olfaction	**pH 6.9-8.0**	**Harmonic Mean 1**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.18	JUVENILE	3H
0.59	JUVENILE	
0.6	JUVENILE	3H
0.75	JUVENILE	20 MIN
0.79	JUVENILE	
1.1	JUVENILE	60D
1.6	JUVENILE	20 MIN
1.9	JUVENILE	120D
2	JUVENILE	21D
2	JUVENILE	
2.1	JUVENILE	3H
2.8	JUVENILE	60D
3.1	JUVENILE	23W
5	JUVENILE	6D
8.5	JUVENILE	3M
17	JUVENILE	3M
17	JUVENILE	22M

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Table 2.6.2.2.6.6 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-18° Celsius**	**Arithmetic Mean 136**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 20-306 mg/L $CaCO_3$**	**Geometric Mean 58**
Endpoint/Effect Cellular	**pH 4.7-8.54**	**Harmonic Mean 21**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
29.2	YEARLING	15D
30.6	YEARLING, 140 MM	5W
32.2	ALEVIN	37W
32.2	EMBRYO, 14 D POST-FERTILIZATION	41W
45	17.8 CM TL, 65.0 G	96H
60.4	16.47 CM FL, 53.85 G	24H
167.3	FINGERLING, 4.1 G, 6.2 CM	2H
171.8	YEARLING	25H
217	15.5-20.0 CM	24H
1492.4	21.5 CM, 126 G	1H

Table 2.6.2.2.6.7 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-18° Celsius**	**Arithmetic Mean 110**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 16-380 mg/L $CaCO_3$**	**Geometric Mean 18**
Endpoint/Effect Growth	**pH 4.7-8.54**	**Harmonic Mean 6**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.1	EM	96H
2.2	FRY, 83.3-91.5 MG WET WT	10D
3.3	SWIM UP FRY, 0.120 G, 25.7 MM	20D
3.5	JUVENILE, 8 G	42D
3.6	YE, YEAR-CLASS I, 15 CM, 27 G MALE	8M
3.6	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
3.6	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-18° Celsius**	**Arithmetic Mean** **110**
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **16-380 mg/L $CaCO_3$**	**Geometric Mean** **18**
Endpoint/Effect **Growth**	**pH** **4.7-8.54**	**Harmonic Mean** **6**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
3.6	EG, FROM 8 MO COPPER EXPOSED PARENT	100D
3.6	EG, FROM 8 MO COPPER EXPOSED PARENT	100D
3.6	YE, YEAR-CLASS I, 15 CM, 27 G	8M
3.6	EG, UNEXPOSED PARENTS	1W
5.1	YEARLING, 10-18 MO	37D
8.3	1.7-3.3 G	21D
12.1	EGG, 0-1 D	95D
16.1	1.7-3.3 G	21D
19.6	YEARLING, 14-16 CM, 30-42 G/	720D
25.5	5.6 G, 7.8 CM	100D
25.8	EGG-FRY	14W
25.8	MX, EGG-FRY	14W
30.6	YEARLING, 140 MM	40W
37.2	EMBRYO, 6 H POST-FER	85D
40	ALEVINS-BUTTONED-UP FRY	96H
45	5.74 G, 8.4 CM	30D
63.8	55.5 G	40D
217	15.5-20.0 CM	20.5H
356.8	8 mo	10D
476.7	8 mo	10D
818	8 mo	10D
930	8 mo	10D

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Table 2.6.2.2.6.8 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set 3
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 6.9-16.5° Celsius**	**Arithmetic Mean 18**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 20-240 mg/L $CaCO_3$**	**Geometric Mean 8**
Endpoint/Effect Growth	**pH 7.2-7.6**	**Harmonic Mean 4**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.9	NR	120D
2.8	NR	120D
21	NR	60D
45	NR	60D

Table 2.6.2.2.6.9 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-18° Celsius**	**Arithmetic Mean 114**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 10.1-320 mg/L $CaCO_3$**	**Geometric Mean 36**
Endpoint/Effect Physiological	**pH 4.7-8.54**	**Harmonic Mean 9**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.3	200-250 G	120D
11.2	17 G	42D
33.1	NR	24H
36.4	8 MO, 3-8 G	7D
44.9	5.74 G, 8.4 CM	30D
60.4	20.01 CM FL, 101.54 G	96H
65.8	SACFRY, 9-11 D, 102.4-110.3 MG WT	15D
94.1	YEARLING	2H
99.8	YEARLING	78H
100	8 MO, 3-8 G	7D
313.6	75-100 G	8H
500	56 G	24H

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Table 2.6.2.2.6.10 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-18° Celsius**	**Arithmetic Mean 1724**
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 40-48 mg/L $CaCO_3$**	**Geometric Mean 57**
Endpoint/Effect Reproductive	**pH 4.7-8.54**	**Harmonic Mean 4**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3.5	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
3.5	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
3.5	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
3.5	YE, YEAR-CLASS I, 15 CM, 27 G FEMAL	8M
8.8	YEARLING, 14-16 CM, 30-42 G/	720D

Copper Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the

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criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to copper, NMFS added an additional step to its analysis for copper to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 13 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.6.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.6.1, predicts a magnitude of effect ranging from a low of an $LC_{0.6}$ at a concentration of 1160 µg/L to a high of an LC_{100} at a concentration of 5.7 µg/L (Table 2.6.2.2.6.11). In other words, the acute criterion of 13 µg/L has an equivalent toxicity potential predicted to kill 0.6 percent to 100 percent, with a median toxicity potential of an LC_7, of the exposed test population, and therefore by inference, field-exposed individuals.

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Table 2.6.2.2.6.11 Relative percent mortality analysis for salmonid fishes, eulachon, and green sturgeon for freshwater copper.

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-16° Celsius**	
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 8-495 mg/L $CaCO_3$**	
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.0**	
Concentration Micrograms Liter^{-1}	**Relative Percent Mortality (acute criterion/LC_{50})**	
5.70	114.0	
5.96	109.1	
9.14	71.1	
9.14	71.1	
11.56	56.2	
12.85	50.6	
18.03	36.1	
19.32	33.6	
20.62	31.5	
21.20	30.7	
23.90	27.2	
25.45	25.5	
25.49	25.5	
25.65	25.3	
27.55	23.6	
30.13	21.6	
30.48	21.3	
31.26	20.8	
31.61	20.6	
32.86	19.8	
33.35	19.5	
33.41	19.5	
34.31	18.9	
35.15	18.5	
36.39	17.9	
37.88	17.2	
38.18	17.0	
38.58	16.8	
39.63	16.4	

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Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
40.66	16.0	
42.63	15.2	
42.83	15.2	
43.86	14.8	
43.88	14.8	
44.23	14.7	
45.86	14.2	
45.87	14.2	
46.38	14.0	
47.01	13.8	
48.10	13.5	
48.36	13.4	
50.59	12.8	
51.40	12.6	
52.79	12.3	
52.79	12.3	
52.86	12.3	
52.96	12.3	
53.76	12.1	
56.10	11.6	
56.39	11.5	
59.23	11.0	
59.70	10.9	
59.89	10.9	
61.06	10.6	
61.68	10.5	
61.87	10.5	
63.79	10.2	
64.68	10.0	
65.18	10.0	
65.54	9.9	

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 13 Micrograms Liter^{-1}	**Temperature 4.4-16° Celsius**	
Criterion Concentration Chronic 9 Micrograms Liter^{-1}	**Hardness 8-495 mg/L $CaCO_3$**	
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.0**	
Concentration Micrograms Liter^{-1}	**Relative Percent Mortality (acute criterion/LC_{50})**	
65.81	9.9	
66.26	9.8	
67.63	9.6	
68.31	9.5	
69.01	9.4	
70.11	9.3	
70.46	9.2	
70.53	9.2	
71.12	9.1	
71.23	9.1	
71.38	9.1	
72.13	9.0	
72.85	8.9	
73.87	8.8	
73.96	8.8	
74.56	8.7	
75.30	8.6	
79.51	8.2	
81.10	8.0	
84.84	7.7	
86.51	7.5	
86.89	7.5	
87.12	7.5	
87.55	7.4	
88.37	7.4	
88.91	7.3	
90.44	7.2	
92.43	7.0	
92.74	7.0	
93.28	7.0	
95.28	6.8	

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Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
99.44	6.5	
99.68	6.5	
99.68	6.5	
99.68	6.5	
101.29	6.4	
107.35	6.1	
108.15	6.0	
108.89	6.0	
111.19	5.8	
112.21	5.8	
113.63	5.7	
113.77	5.7	
114.29	5.7	
122.21	5.3	
123.91	5.2	
124.94	5.2	
128.87	5.0	
130.72	5.0	
133.67	4.9	
138.04	4.7	
138.78	4.7	
140.88	4.6	
145.69	4.5	
147.81	4.4	
148.58	4.4	
149.08	4.4	
150.03	4.3	
150.52	4.3	
155.59	4.2	
163.37	4.0	
163.44	4.0	

Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
171.44	3.8	
174.10	3.7	
174.36	3.7	
177.75	3.7	
179.14	3.6	
179.91	3.6	
181.82	3.6	
183.34	3.5	
184.58	3.5	
185.37	3.5	
189.35	3.4	
194.30	3.3	
194.76	3.3	
199.96	3.3	
201.19	3.2	
210.45	3.1	
212.83	3.1	
217.16	3.0	
217.16	3.0	
222.22	2.9	
227.44	2.9	
228.59	2.8	
229.06	2.8	
233.38	2.8	
240.00	2.7	
240.02	2.7	
244.76	2.7	
250.22	2.6	
254.62	2.6	
255.80	2.5	
264.28	2.5	

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Criterion Freshwater Copper		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **13 Micrograms Liter^{-1}**	**Temperature** **4.4-16° Celsius**	
Criterion Concentration Chronic **9 Micrograms Liter^{-1}**	**Hardness** **8-495 mg/L $CaCO_3$**	
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.0**	
Concentration **Micrograms Liter^{-1}**	**Relative Percent Mortality** **(acute criterion/LC_{50})**	
266.36	2.4	
271.32	2.4	
274.31	2.4	
288.82	2.3	
289.33	2.2	
301.90	2.2	
310.51	2.1	
313.32	2.1	
322.75	2.0	
326.37	2.0	
333.58	1.9	
346.63	1.9	
355.82	1.8	
376.54	1.7	
404.21	1.6	
447.01	1.5	
447.48	1.5	
467.01	1.4	
475.90	1.4	
489.25	1.3	
533.72	1.2	
533.72	1.2	
599.98	1.1	
600.44	1.1	
1160.10	0.6	

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for copper, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for copper, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

Sublethal Effects. Copper toxicity is influenced by chemical speciation, hardness, pH, alkalinity, total and dissolved organic content in the water, previous exposure and acclimation, fish species and life stage, water temperature, and presence of other metals and organic compounds that may interfere with or increase copper toxicity. Synergistic toxicity is suggested for mixtures of copper and aluminum, iron, zinc, mercury, anionic detergents, or various organophosphorus insecticides (Eisler 1998a).

The distinction between copper deficiency and toxicity is small in organisms such as algae and invertebrates that lack effective mechanisms to control absorption (EPA 1999 as cited in EPA 2008). Copper is not strongly bioconcentrated in vertebrates but is more strongly bioconcentrated in invertebrates (EPA 1999 as cited in EPA 2008). Toxicity of copper to aquatic organisms is dependent on pH, temperature, alkalinity, hardness, and concentrations of bicarbonate, sulfide, and organic ligands (EPA 1980b as cited in EPA 2008), as well as the type and life stage of exposed organism (EPA 1999 as cited in EPA 2008). Copper is among the most toxic of the heavy metals to freshwater biota (Schroeder *et al.* 1966, Betzer and Yevich 1975 as cited in EPA 2008). In general, mortality of tested aquatic species is greatest under conditions of low water hardness, starvation, elevated water temperatures, and among early developmental stages (Eisler 1998a as cited in EPA 2008). Effects of copper toxicity to freshwater organisms include valve closure, reduction in filtration rates, impaired structure and function of cellular membranes, and cardiac inhibition in mussels. Impaired disease resistance, disrupted migration (via avoidance behavior of copper-contaminated areas), hyperactivity, impaired respiration, disrupted osmoregulation, pathology of kidneys, liver, and gills, impaired function of olfactory organs and brain, altered blood chemistry, and enzyme activity have been documented in fish (Eisler 1998a as cited in EPA 2008).

Biological copper toxicity has a diversity of systemic effects including reduced growth and survival rates and altered hematology, respiratory, and cardiac physiology. Reproductive effects, including reduced frequency of spawning, reduced egg production, reduced survival of young, and increased deformity of fry, have been reported (Sorensen 1991, Eisler 1998a). Elevated copper levels also influence the immune system and vulnerability to disease. For example, Carballo *et al.* (1995) determined that rainbow trout were more susceptible to the microbial parasite, *Saprolegnia parasitica*, and Dethloff and Bailey (1998) determined physiological changes in immune system characteristics at elevated copper concentrations . Hansen *et al.*

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(1999b) determined that cellular damage occurred to the olfactory system of juvenile Chinook salmon and rainbow trout that were exposed to high concentrations of copper.

Copper toxicity appears to be inversely related to the tendency of the metal to bind with the external gill surface via ionic interactions. In other words, a lower affinity of the gill surface to copper leads to a greater likelihood of disruption of intracellular processes, which may lead to gill dysfunction (Reid and McDonald 1991). Some studies have examined the disruption of gill processes by copper. For example, gill Na^+, K^+- ATPase activity in Chinook salmon parr was unaffected after an 18-hour exposure to stream water with elevated copper levels of 48 µg/L (hardness = 13.3 mg/L as $CaCO_3$). With the same exposure, significant inhibition of gill Na^+, K^+-ATPase activity was observed in smolts. Significant increases in hematocrit and plasma glucose were also observed in both parr and smolts resulting from the same 18-hour exposure (Beckman and Zaugg 1988). Sola *et al.* (1995) determined that divalent copper (Cu^{2+}) totally suppressed gill Na^+, K^+- ATPase activity and produced significant cell damage, edema, mucus production, smoothing of apical membranes, swelling of tubular system and destruction of mitochondria in rainbow trout at high concentrations of $CuCl_2$ (3.5 and 134.5 mg/L). They concluded that bioavailable copper, such as divalent copper, immediately damages the hydromineral balance of rainbow trout and causes morphological modifications that are irreversible.

Sauter *et al.* (1976) determined reduced growth in brook trout fry occurred between 3 µg/L and 5 µg/L, at a hardness of approximately 38 mg/L. The resulting chronic value from that study was 3.9 µg/L, which is below the proposed chronic criterion (4.9 µg/L). At a hardness of 187 mg/L, the effect occurred between 5 µg/L and 8 µg/L with a resulting chronic value of 6.3 µg/L, which is well below the proposed chronic criterion of 19 µg/L.

Munoz *et al.* (1991) observed rapid elevations of plasma cortisol, an indicator of stress, in rainbow trout after a 1-hour exposure to approximately 0.2 µg/L of copper at a hardness of 12 mg/L. The elevated plasma cortisol levels were maintained throughout the experiment's duration of 21 days. This concentration is 45 times the chronic criterion, with no corresponding adverse physiological effects detected in association with the elevated cortisol levels. However, elevated plasma cortisol levels are indicative of stress, and potentially represent a diversion of energy from normal physiological processes that may render salmonids more vulnerable to disease. Dethloff *et al.* (2001) also determined that exposure to copper concentrations below the proposed chronic criterion was associated with decreased levels of hematocrit, leukocrit, and lymphocyte percentage in the blood in wild rainbow trout, but condition factors and other biochemical parameters tested did not show a significant difference compared with fish from reference sites.

There is tremendous variation between fish species in the amount of copper that is accumulated for a given exposure. Copper is more strongly bioconcentrated in invertebrates than in fish, and is more commonly found in tissues of herbivorous fish than in carnivorous fish from the same location. In salmonids, copper has been determined to accumulate in liver, gill, muscle, kidney, pyloric caecae, and spleen tissues and the concentrations of copper in fish tissues reflect the amount of bioavailable copper in the environment (Peterson *et al.* 1991, Farag *et al.* 1994, Camusso and Balestrini 1995, Saiki *et al.* 1995, Sorensen 1991). The kidneys and gills are not thought to play a significant role in copper detoxification (Sorensen 1991). Both dissolved and dietary pathways have been associated with bioaccumulation in salmonids, whereas the case for

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particulate copper pathways is less clear. However, rainbow trout appear to be able to ingest more copper than cadmium, lead, or zinc without significant effects to survival or growth, and elevated copper levels in their gills and livers have been found to be measures of chronic exposure but not of significant toxic effects (Mount *et al.* 1994, Dethloff and Bailey 1998, Taylor *et al.* 2000).

Chemosensory and Behavioral Effects. In aquatic systems, chemoreception is one of oldest and most important sensory systems used by animals to collect information on their environment and generate behaviors involved in growth, reproduction, and survival (Pyle and Mirza 2007). These behaviors include recognition of conspecifics, mates and predators, food search, defense, schooling, spawning and migration. Stimuli are perceived by sensory structures and converted to electrical signals that are conducted to the central nervous system where the information is integrated and appropriate behavioral responses are generated (Baatrup 1991). Detection of chemical signals involves not only recognition of a spectrum of unique compounds or mixtures but also their spatial and temporal distribution in the medium (Atema 1995). Sensory receptors are in direct contact with the environment, and therefore pollutants may disrupt normal chemosensory function by masking or counteracting biologically relevant chemical signals or by causing direct morphological and physiological damage to the receptors (Baatrup 1991).

Impairment of olfaction can be measured by electrophysiological techniques called electroolfactograms (EOGs) (*e.g.*, Evans and Hara 1985, Baldwin *et al.* 2003) or electroencephalograms (EEGs) (*e.g.*, Hansen *et al.* 1999a, Sandahl *et al.* 2004). In fish, EOGs measure the response along the midline of a rosette within the fish's olfactory chamber (nose), EEGs record the response from the olfactory bulb (forebrain) (Sandahl *et al.* 2004, p. 406). Each rosette contains ciliated olfactory receptor neurons (ORNs) that respond to stimuli as water passes through the olfactory chamber and over the rosette. The EOG measures responses of an assemblage of ORNs. Reductions in or elimination of the EOG and EEG amplitude of exposed fish compared to unexposed fish reflect the in sensory ability.

Copper has been known to disrupt the normal function of the olfactory system in salmonids for over 45 years (Sprauge *et al.* 1965, Hara *et al.* 1976). More recent studies using EOGs and EEGs have shown disruption at concentrations of dissolved copper at or slightly above background concentrations (Baldwin *et al.* 2003, Sandahl *et al.* 2004). Hecht *et al.* (2007) defines background as surface waters equal to 3 µg/L dissolved copper, since experimental waters had background concentrations as high as 3 µg/L dissolved copper. There have been mixed results as to whether certain fish species are more sensitive than others to the olfactory neurotoxicity of copper. In experiments using EEG recordings, Hansen *et al.* (1999a) found that rainbow trout (*O. mykiss*) were more vulnerable than juvenile Chinook salmon (*O. tshawytscha*). Thus, while there may be modest differences in sensitivity for some species, the available evidence suggests that copper is a general olfactory toxicant for all freshwater fish. Although chemoreception is probably a fundamental function in most, if not all, fishes (Tierney *et al.* 2010), many of these studies evaluated copper avoidance or copper-induced olfactory impairment in salmonid fishes (*e.g.*, Hansen *et al.* 1999a,b; Baldwin *et al.* 2003, 2011; Sandahl *et al.* 2007; McIntyre *et al.* 2008a).

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Most behavioral studies on toxicity to chemoreception (*i.e.*, avoidance, food attraction, and alarm response) are problematic because it is difficult to separate olfactory toxicity from other forms of toxicity (Tierney *et al.* 2010). Behavioral responses can integrate many inputs, which may introduce uncertainty when attributing olfactory impairment to altered behavioral responses (Tierney *et al.* 2010). A few olfactory toxicological studies have related effects across organizational levels and these can be divided into two categories: 1) those that relate changes in electrochemical responses to physiological responses or to behavioral responses; and 2) those that relate olfactory-mediated physiologic responses to behavioral responses (Tierney *et al.* 2010). For copper, Sandahl *et al.* (2007) demonstrated that the relationship between loss of sensory function (EOG) and behavioral impairment was highly correlated. Alarm pheromone (a substance released during fish injuries) triggered an average reduction in swimming speed of 74% and elicited a mean EOG response of 1.2 mV in unexposed salmon. Salmon exposed to 2 to 20 µg/L copper exhibited reductions in both EOG (50-92%) and in alarm response (Hecht *et al.* 2007, Sandahl *et al.* 2007). Statistically significant reductions in EOG response to skin extract occurred at all concentrations tested (2, 5, 10, and 20 µg/L copper), while no significant reductions in swimming speed (majority of fish did not become motionless) occurred at higher copper concentrations (5, 10, and 20 µg/L; Sandahl *et al.* 2007). In fish, direct exposure to dissolved copper can impair and destroy ORNs, although the precise mechanism remains unknown (Hecht *et al.* 2007).

Given the importance of sensory perception, impaired olfaction may in many cases be of more immediate survival concern than other physiological impairments (Tierney *et al.* 2010). The studies reviewed in this section illustrate several important aspects of copper toxicity to the olfactory system: 1) neurotoxic effects of copper can occur within minutes of exposure; 2) low concentrations can elicit responses; 3) at low concentrations, inhibition is transient and recovery can be seen within hours or when the toxicant is removed; and 4) incomplete or time-sensitive recovery of olfactory system to food-based, conspecific and predator-related odors, and reproductive pheromones.

Several studies indicate that thresholds exist between neurological, physiological and behavioral responses, and more than sufficient information exists to indicate that for fishes, olfaction is indispensible and sensitive to contaminants. Tierney *et al.* (2010) reviewed the ramifications for extrapolating neurological and physiological data to behavioral and ecological impacts as straightforward: lower order measures (*e.g.*, EOG) may underestimate the impact of toxicity to higher order biological responses (*e.g.*, mating). Tierney *et al.* (2010) report that setting regulations below where negative responses are observed in olfactory-based systems is not warranted until effects relevant to populations are better established.

Acute copper toxicity is known to disrupt osmoregulation in fishes by interfering with sodium uptake in the gill. Metal toxicity varies due to various physicochemical characteristics of the exposure water (*e.g.*, either laboratory or field), namely hardness, alkalinity, pH, and dissolved organic matter (Niyogi and Wood 2004). These constituents can protect against toxicity either by competing at the binding sites of the sodium transporter or by reducing the bioavailability of copper by complexation (McIntyre *et al.* 2008a). In 2007, the EPA updated the ambient water quality criteria for copper and employed a biotic ligand model (BLM) to derive copper criteria (EPA 2007). The BLM differs from the previous hardness-based criterion by incorporating the

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water chemistry parameters (*e.g.*, pH, temperature, cations, and dissolved organic carbon) to predict lethality caused by copper binding to the gill (EPA 2007).

Due to the differences in structure and physiological function between the gill and olfactory epithelium, the extent to which the BLM can be used to estimate sublethal, neurobehavioral toxicity is unclear (McIntyre *et al.* 2008a). McIntyre *et al.* (2008a) used electrophysiological recordings from juvenile coho salmon to investigate the impacts of copper on the olfactory epithelium in freshwater with different chemical properties. Results showed olfactory function was 1) not affected by change in pH (8.6-7.6), 2) slightly protected by increasing water hardness (0.2-1.6 mM Ca) and alkalinity (0.2-3.2 mM HCO_3^-), and 3) partially restored by increasing dissolved organic carbon (0.1-6 mg/L; McIntyre *et al.* 2008a).

Since olfactory and behavioral endpoints were not used while deriving either the BLM- or hardness-based criteria, concerns have arisen that existing state water quality criteria for copper may not be protective of olfactory impairment especially in the western U.S. (McIntyre *et al.* 2008a). Using data from McIntyre *et al.* (2008a,b), Meyer and Adams (2010) parameterized an olfactory-based BLM and calculated IC_{20}s to evaluate whether the USEPA's BLM-based criteria for copper would be protective of neurological impairment in juvenile salmon. Of the 16 different laboratory test waters (data from Green *et al.* 2010; Hansen *et al.*, 1999a,b; and McIntyre *et al.* 2008a,b), the acute and chronic BLM-based copper criteria protected against at least 20% avoidance of copper and 20% olfactory impairment while the hardness-based criteria were considerably under protective in many of the same exposure waters (Meyer and Adams 2010).

McIntyre *et al.* (2012) calculated survival probabilities for copper exposures relative to controls for coho salmon that ranged from 10 percent at 20 µg/L to 17 percent at 5 µg/L. McIntyre *et al.* (2012) also determined that relatively brief (3 hours) exposures to copper ranging from 5 to 20 µg/L eliminated the behavioral alarm response in coho salmon prey, leading in turn to increased detection, reduced evasion, and reduced survival during predation trials.

Experimental data suggests that significant amelioration of olfactory toxicity due to hardness is unlikely in typical Pacific salmonid freshwater habitats (Hecht *et al.* 2007). The experiment showed that hardness at 20, 120, and 240 mg/L Ca (experimentally introduced as $CaCl_2$) did not significantly protect juvenile coho salmon from olfactory toxicity following 30 minute laboratory exposures to 10 µg dCu/L above an experimental background of 3 µg/L (Baldwin *et al.* 2003).

Hecht *et al.* (2007) calculated an acute CMC using the Biotic Ligand Model (BLM) (EPA 2007). Interestingly, the estimated acute CMC based on the BLM using measured and estimated water quality parameters from Sandahl *et al.* (2007) was 0.63 µg/L with a range from 0.34 to 3.2 µg/L, while the EPA hardness-based acute CMC (EPA 2002) was 6.7 µg/L. Because the BLM-based acute criterion is sensitive to pH and DOC, the range of measured test pH values (6.5–7.1) and the range of estimated DOC values (0.3–1.5 mg/L) produced this range of BLM-based acute criterion values. It is also interesting that the acute CMC range (0.34–3.2 µg/L) overlapped with the olfactory-based BMC range (0.18–2.1 µg/L).

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Sublethal Effects Summary. The available evidence indicates that the chronic criterion for copper is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Copper is highly toxic to most freshwater invertebrates (Moore and Ramamoorthy 1984). Aquatic macroinvertebrates are sensitive to both dissolved and particulate copper, and some taxa can be more sensitive than salmonids (*e.g.*, Kemble *et al.* 1994). Data in EPA (1985d) indicate that the proposed criteria are usually protective of invertebrates that juvenile listed species feed on, although in one case (Dave 1984 as cited in EPA 1985d) a cladoceran exhibited an LC_{50} that was lower than the acute and chronic criteria at high hardness. Invertebrate communities in rivers appear to respond to elevated copper in the sediments by changing composition to pollution-tolerant taxa, rather than by reducing overall biomass (Canfield *et al.* 1994, Clements and Kiffney 1994, Beltman *et al.* 1999). The biological significance of such species change to listed species is unknown.

Copper contained in bed sediments was elevated in benthic invertebrates in field studies conducted in metals-contaminated streams (*e.g.*, Ingersoll *et al.* 1994, Woodward *et al.* 1994, Beltman *et al.* 1999, Besser *et al.* 2001). Uptake by invertebrates is strongly influence by the presence of acid-volatile sulfide in the sediments (Besser *et al.* 1995). However, Kiffney and Clements (1996) determined an inverse relationship existed between aquatic macroinvertebrate body size and survival at copper levels in excess of the proposed chronic criterion, which may partially counter the effects of bioaccumulation. Indirect effects of elevated copper levels on listed species therefore likely include reductions in the availability of larger invertebrates as food for larger juvenile fishes, and ingestion of bioconcentrated copper by fry and juveniles of all sizes.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for copper is likely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Copper. The available evidence for copper indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (high-intensity), impairment of essential behaviors related to successful rearing and migration (high-intensity), cellular trauma (moderate intensity), physiological trauma (moderately-high-intensity), reproductive failure (high-intensity), and sublethal effects (high-intensity).

2.6.2.2.7 Lead

Lead Criteria. The proposed acute and chronic criteria for lead are 65 µg/L and 2.5 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.7.1 through 2.6.2.2.7.8 report toxicity data from the ECOTOX database for freshwater lead, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.2.2.7.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 78742**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 40-314 mg/L $CaCO_3$**	**Geometric Mean 14675**
Endpoint/Effect LC_{50}/Mortality	**pH 6.8-8.1**	**Harmonic Mean 2277**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
320	ALEVIN	96H
1000	FRY	96H
1700	JUVENILE, 7-11 WK, 0.97 G	96H
2100	JUVENILE, 18-22 WK, 0.94 G	24H
2670	72 WK, 102 G	96H
4100	JUVENILE, 7-10 WK, 0.60 G	96H
4500	145 MM	96H
12000	JUVENILE, 7-8 WK, 0.34 G	96H
170000	JUVENILE, 18-22 WK, 0.94 G	96H
170000	ALEVIN	96H
170000	ALEVIN	96H
170000	JUVENILE, 10-12 WK, 0.41 G	96H
170000	ALEVIN	96H
224000	JUVENILE, 5-6 WK, 0.85 G	96H

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Table 2.6.2.2.7.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 2-20.5° Celsius**	**Arithmetic Mean 113**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 23.95-385 mg/L $CaCO_3$**	**Geometric Mean 29**
Endpoint/Effect Growth	**pH 6.5-8.1**	**Harmonic Mean 9**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1	NR	19M
6	SEXUALLY MATURING MALES 2 YR	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
13	NR	141D
14	JUVENILE, 0.38 G WET WT/	29D
16	NR	19M
16	NR	19M
18	EGGS	19M
21	EYED EGGS	19M
36	FRY, 25 MM	19MIN
38	EGGS	7M
39	EMBRYO-ADULT, SPAWNING, F1, 2, 3	38W
77	EGGS/	7M
134	ALEVIN, 21 D	21D
149	F2, EMBRYO-12 WK JUVENILE	6M
154	EMBRYO-ADULT, SPAWNING, F1, 2, 3	38W
213	EMBRYO-ADULT, SPAWNING, F1, 2, 3	38W
305	F2, EMBRYO-12 WK JUVENILE	6M
1216	F1, EMBRYO-ADULT SPAWNING	2.25Y

Table 2.6.2.2.7.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **65 Micrograms Liter^{-1}**	**Temperature** **2-20.5° Celsius**	**Arithmetic Mean** **14011**
Criterion Concentration Chronic **2.5 Micrograms Liter^{-1}**	**Hardness** **16-350 mg/L $CaCO_3$**	**Geometric Mean** **1575**
Endpoint/Effect **NOEC/Mortality/Growth**	**pH** **6.5-8.1**	**Harmonic Mean** **75**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
18	EGGS	19M
32	NR	19M
150	NR	19M
13526	NR	10D
21811	NR	10D
25461	NR	10D
37079	NR	10D

Table 2.6.2.2.7.4 Behavioral toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **65 Micrograms Liter^{-1}**	**Temperature** **2-20.5° Celsius**	**Arithmetic Mean** **4**
Criterion Concentration Chronic **2.5 Micrograms Liter^{-1}**	**Hardness** **50-135 mg/L $CaCO_3$**	**Geometric Mean** **4**
Endpoint/Effect **Behavioral**	**pH** **6.5-8.1**	**Harmonic Mean** **3**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
3	NR	1200S
3	NR	1200S
3	NR	1200S
6	EGG	210D

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Table 2.6.2.2.7.5 Biochemical toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 2-20.5° Celsius**	**Arithmetic Mean 501**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 42.3-95 mg/L $CaCO_3$**	**Geometric Mean 190**
Endpoint/Effect Biochemical	**pH 6.5-8.1**	**Harmonic Mean 45**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
9	6-18 MO	2W
12	NR	28D
25	JUVENILE, 0.38 G WET WT/	1D
157	YEARLING	14D
157	YEARLING	56D
83	6-18 MO	2W
367	ALEVIN, 21 D	21D
1438	ALEVIN, 21 D	21D
762	6-8 MO	20D
1000	240 G	3D
1000	240 G	6D
1000	240 G	11H

Table 2.6.2.2.7.6 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 2-20.5° Celsius**	**Arithmetic Mean 414**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 121-150 mg/L $CaCO_3$**	**Geometric Mean 65**
Endpoint/Effect Cellular	**pH 6.5-8.1**	**Harmonic Mean 17**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
6	SEXUALLY MATURING MALES 2 YR	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
6	SEXUALLY MATURING, 2 YR, FEMALE	12D
454	28 CM, 240 G, FEMALE	26D

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Table 2.6.2.2.7.7 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 12-20° Celsius**	**Arithmetic Mean 38**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 40-314 mg/L $CaCO_3$**	**Geometric Mean 15**
Endpoint/Effect Physiological	**pH 6.8-8.1**	**Harmonic Mean 6**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3	NR	191D
72	NR	191D

Table 2.6.2.2.7.8 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater lead.

Criterion Freshwater Lead		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 65 Micrograms Liter^{-1}	**Temperature 2-20.5° Celsius**	**Arithmetic Mean 395**
Criterion Concentration Chronic 2.5 Micrograms Liter^{-1}	**Hardness 17-314 mg/L $CaCO_3$**	**Geometric Mean 375**
Endpoint/Effect Reproductive	**pH 6.5-8.1**	**Harmonic Mean 354**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
751	F1, EMBRYO-ADULT SPAWNING	2.25Y
1514	F1, EMBRYO-ADULT SPAWNING	2.25Y
1517	YEARLING, 50-70 G, ADULT SPAWNING	38W

Lead Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and

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Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to lead, NMFS added an additional step to its analysis for lead to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 65 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.7.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.7.1, predicts a magnitude of effect

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ranging from a low of an LC_{zero} at a concentration of 224,000 µg/L to a high of an LC_{10} at a concentration of 320 µg/L. In other words, the acute criterion of 65 µg/L has an equivalent toxicity potential predicted to kill zero percent to 10 percent, with a median toxicity potential of an $LC_{0.5}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, none of the toxicity studies reported concentrations that are less than the acute criterion for lead, which implies that listed species exposed to waters equal to the acute criterion concentration may not suffer acute toxic effects. A number of toxicity studies reported concentrations that are less than the chronic criteria for lead, and a number of toxicity studies reported concentrations that are greater than the chronic criterion for lead, which implies that listed species exposed to waters equal to the chronic criterion concentration will suffer chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects, but will suffer chronic toxic effects.

Sublethal Effects. Lead toxicity is influenced by species and life stage, metal speciation including whether in organic or inorganic form, hardness, pH, water temperature, and the presence of other metals that act either synergistically or antagonistically depending on the element. Elevated lead concentrations are associated with long-term effects including: spinal curvature and other deformities; anemia; caudal chromatophore degeneration (black tail); caudal fin degeneration; destruction of spinal neurons; aminolevulinic acid dehydratase (ALAD) inhibition in blood cells, spleen, liver, and renal tissues; reduced swimming ability; increased mucus formation and coagulation over body and gills and destruction of respiratory epithelium; scale loss; elevated lead in blood, bone and kidney; muscular atrophy and paralysis; teratogenic effects; inhibition of growth; retardation of maturity; changes in blood chemistry; testicular and ovarian histopathology; and death. Fish embryos appear to be more sensitive to lead than older fry and juvenile stages (Hodson *et al.* 1982, EPA 1985f, Eisler 1988b, Sorensen 1991; Farag *et al.* 1994). Organic lead compounds are generally more toxic than inorganic. Aquatic organisms are influenced more by dissolved than by total lead, because lead characteristically precipitates out to bed sediments in aqueous environments (Eisler 1988b, Sorensen 1991).

Although some of the available data suggest that toxic effects of inorganic lead on salmonids occurs above the proposed chronic criterion, the data exhibit wide variation, and there are limited lead toxicity test data available for salmonids, particularly for sublethal or indirect effects. Results for the early life stage are less conclusive than for adults, and there is conflicting evidence regarding the effects. Fish embryos and fry are more sensitive to lead in terms of effects to development than older life stages (Sorenson 1991). The results of Birge *et al.* (1978, 1981) indicate that salmonid embryos exposed for more than 4 days can begin to die when inorganic lead concentrations are between 2.5 µg/L and 10.3 µg/L, and hardness is 100 mg/L as $CaCO_3$.

Other studies were identified in this analysis that indicate the chronic criterion is at or below the NOEC level for the early life stage, as suggested by available data. For example, Sauter *et al.*

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(1976) determined that the threshold for adverse chronic effects to rainbow trout eggs and fry occurred at a lead concentration between 71 µg/L and 146 µg/L, both of which are above the chronic criterion. Davies *et al.* (1976) determined that in soft water (hardness ~30 mg/L), adverse developmental effects occurred to eggs and sac-fry when exposure concentrations were between 4.1 µg/L and 7.6 µg/L, which are below the proposed chronic criterion. When the eggs were not exposed, effects to sac-fry were determined to occur when exposure concentrations were between 7.2 µg/L and 14 µg/L in soft water, and between 190 µg/L and 380 µg/L in hard water (300 mg/L). Other bioassays involving adult trout and their offspring in soft water indicated that there were no adverse reproductive effects occurring when lead concentrations were around 6 µg/L (Davies *et al.* 1976); this level is also above the proposed chronic criterion.

The bioavailability of lead increases in environments with low pH, low organic content, and low metal salt content (Eisler 1988b as cited in EPA 2008). Toxicity of lead to aquatic organisms varies with water temperature, pH, water hardness, metal salt concentrations, organic matter, and suspended solid concentration (EPA 1999 as cited in EPA 2008). Invertebrates tend to have higher bioconcentration factors than vertebrates (EPA 1999 as cited in EPA 2008). Effects of lead toxicity to freshwater organisms include reduced growth, spinal curvature and other deformities, anemia, caudal fin degeneration, destruction of spinal neurons, enzyme inhibition, reduced swimming ability, increased mucus formation and coagulation over body and gills and destruction of respiratory epithelium, scale loss, muscular atrophy and paralysis, impaired reproduction, and reduced survival (Hodson *et al.* 1982, Eisler 1988b, Sorensen 1991, Farag *et al.* 1994 as cited in EPA 2008). Organic lead compounds are generally more toxic than inorganic (Eisler 1988b as cited in EPA 2008).

Fish do not accumulate lead extensively and the results and interpretations of lead accumulation studies vary. Farag *et al.* (1994) determined that adult and juvenile rainbow trout accumulated lead in their gut through their diet, and in gill and kidney tissues, when exposed to dissolved lead at concentrations slightly in excess of the proposed chronic criteria. In contrast, Mount *et al.* (1994) determined that much higher levels of dietary lead exposure than that tested by Farag *et al.* (1994) did not result in reduced survival or growth of rainbow trout fry. Fish excrete lead rapidly, and depuration generally reduces levels in tissues and organs (Sorensen 1991).

Lead accumulation is influenced by age, diet, particle size ingested, hardness, pH, water temperature, metal speciation, and presence of other compounds in the water (Eisler 1988b; Sorensen 1991). Bioavailability of lead increases with decreasing pH, organic content, hardness, and metal salt content (Eisler 1988b). Lead precipitation with increasing hardness leads to decreased bioavailability, although the potential for accumulation from precipitated lead still exists (Sorensen 1991). Fish do not accumulate lead extensively, and the results and interpretations of lead accumulation studies consequently vary. Farag *et al.* (1994) determined that adult and juvenile rainbow trout accumulated lead in their gut through their diet, and in gill and kidney tissues when exposed to dissolved lead at concentrations slightly in excess of the chronic criterion. In contrast, Mount *et al.* (1994) determined that much higher levels of dietary lead exposure than that tested by Farag *et al.* (1994) did not result in reduced survival or growth of rainbow trout fry. Fish excrete lead rapidly and depuration generally reduces levels in tissues and organs (Sorensen 1991).

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Sublethal Effects Summary. The available evidence indicates that the chronic criterion for lead is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Lead toxicity varies considerably among aquatic macroinvertebrates (EPA 1985f, Eisler 1988b). Results reviewed in EPA (1985f) and Eisler (1988b) indicate that amphipods are more sensitive than other taxa, and that some freshwater isopods are tolerant of elevated lead levels. However, the data indicate that mortality of the more sensitive taxa occurs at concentrations that are well above the acute criterion.

Invertebrates generally have higher bioconcentration factors than vertebrates (Enk and Mathis 1977; Eisler 1988b). Ingersoll *et al.* (1994) determined that while the amphipod *Hyalella azteca* accumulated lead from bed sediments, the level of accumulation was not related to concentration gradient in the riverbed. Because lead occurs in association with copper, cadmium, and zinc in the field studies reviewed, it is difficult to ascribe a direct adverse chronic effect of lead to aquatic invertebrates at exposure concentrations that are below the chronic criterion.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for lead is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Lead. The available evidence for lead indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), reduced growth (moderate) intensity, impairment of essential behaviors related to successful rearing and migration (moderately-high-intensity), cellular trauma (moderately-high-intensity), physiological trauma (moderate intensity), impairment of biochemical processes (moderate intensity), and reproductive failure (low intensity).

2.6.2.2.8 Nickel

Nickel Criteria. The proposed acute and chronic criteria for nickel are 470 µg/L and 52 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.8.1 through 2.6.2.2.8.5 report toxicity data from the ECOTOX database for freshwater nickel, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.2.2.8.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater nickel.

Criterion Freshwater Nickel		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 470 Micrograms Liter^{-1}	**Temperature 8-13.3° Celsius**	**Arithmetic Mean 92062**
Criterion Concentration Chronic 52 Micrograms Liter^{-1}	**Hardness 27-39 mg/L $CaCO_3$**	**Geometric Mean 18793**
Endpoint/Effect LC_{50}/Mortality	**pH 6.1-8.3**	**Harmonic Mean 1146**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
107	4 H POST-FER	85D
244	4 H POST-FER	85D
588	LARVAE	96H
8826	ADULT, 16-18 CM	96H
15571	JUVENILE, 43.4 MM, 0.60 G	96H
16390	ALEVIN, 14.3 MM, 0.01 G	96H
17390	JUVENILE, 62.4 MM, 1.44 G	96H
20652	15.4 G, 116 MM, 12 MO	96H
22691	16.4 G, 119 MM, 12 MO	96H
25496	0.37 G, 36 MM, 3 MO	96H
27790	0.58 G, 40 MM, 3 MO	96H
33380	ALEVIN, 29.8 MM, 0.24 G	96H
35978	JUVENILE, 45.8 MM, 0.63 G	96H
50170	ALEVIN, 20.8 MM, 0.10 G	96H
155928	NR	48H
161455	8 MO	4D
503126	NR	48H
561339	NR	48H

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Table 2.6.2.2.8.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater nickel.

Criterion Freshwater Nickel		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 470 Micrograms Liter^{-1}	**Temperature 4-20° Celsius**	**Arithmetic Mean 4824**
Criterion Concentration Chronic 52 Micrograms Liter^{-1}	**Hardness 11-52 mg/L $CaCO_3$**	**Geometric Mean 631**
Endpoint/Effect Growth	**pH 6.1-8.3**	**Harmonic Mean 183**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
60	4 H POST-FER	85D
61	4 H POST-FER	75D
108	4 H POST-FER	75D
413	4 H POST-FER	75D
672	8 MO	75D
672	EGGS	75D
748	4 H POST-FER	75D
9041	EYED EGGS-SWIM UP FRY	75H
31645	EGGS-SACK FRY	75D

Nickel Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on

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fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to nickel, NMFS added an additional step to its analysis for nickel to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 470 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.8.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.8.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 561,339 µg/L to a high of an LC_{100} at a concentration of 107 µg/L. In other words, the acute criterion of 470 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an LC_1, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for nickel, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for nickel, which implies that listed species exposed to waters equal to criteria concentrations may

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not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute toxic effects, but may not suffer chronic toxic effects.

Sublethal Effects. Nickel poisoning in fish can cause respiratory stress, convulsions, and loss of equilibrium prior to death. In fishes, adverse respiratory effects occur through destruction of gill tissues by ionic nickel and subsequent blood hypoxia. Other effects include decreased concentrations of glycogen in muscle and liver tissues and simultaneous increases in lactic acid and glucose in the blood, and interference with metabolic oxidation-reduction processes (Eisler 1998b). In general, the egg and embryo stages of salmonids are the most, and older stages the least, sensitive to nickel toxicity (Nebeker *et al.* 1985 as cited in Eisler 1998b). In contrast with other metals, alevins and juveniles appear to have a similar sensitivity to nickel (Buhl and Hamilton 1991).

Salmonid fishes accumulate nickel through both dietary and water-borne exposure routes (EIFAC 1984, Eisler 1998b). Bioconcentration factors vary substantially both within and between species, with age of organism, and with exposure concentration, and have been determined to range between 2 inch and 52 inch fish. Bioconcentration has been noted to occur in kidney, liver, and muscle tissues of rainbow trout exposed to ambient water concentrations of nickel equal to 1000 µg/L for 6 months, but the test fish were able to depurate much of the accumulated nickel within 3 months after exposure was terminated and were not visibly affected during the experiment (Calamari *et al.* 1982). Studies of saltwater and freshwater fish species have determined that piscivorous fish bioaccumulate greater levels of nickel in muscle tissues than other fish, indicating the potential for biomagnification to occur (albeit to a limited extent according to most studies; EIFAC 1984, Eisler 1998b). There is evidently a risk of bioaccumulation from chronic nickel exposure, but it remains to be determined to what extent this is a significant hazard for listed species.

Nickel can be carcinogenic, may be mutagenic, and is not teratogenic. It is bioconcentrated and bioaccumulated by aquatic organisms (Eisler 1998b). Toxicity of nickel to aquatic organisms is dependent on water hardness, pH, ionic composition, chemical form, type and concentration of ligands, presence of mixtures, and availability of solid surfaces for adsorption (Eisler 1998b). Nickel interacts with many compounds to produce altered patterns of accumulation, metabolism, and toxicity (Eisler 1998b). Mixtures of metals containing nickel salts are more toxic to daphnids and fishes than are predicted on the basis of individual components (Enserink *et al.* 1991). Effects of nickel toxicity to freshwater invertebrates include reduced growth, impaired reproduction, reduced population biomass, increased respiration rate, and reduced survival (see Eisler 1998b). Effects of nickel toxicity to freshwater fish include delayed hatching time, reduced swimming activity, behavioral alterations (avoidance), disrupted protein metabolism in gills and kidneys, loss of equilibrium, destruction of gill lamellae resulting in decreased ventilation rate, decreased concentrations of glycogen in muscle and liver, and reduced survival in fish (Eisler 1998b).

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Several studies have determined that mortality of salmonid embryos occurs over longer-term exposures to concentrations that are below the chronic criterion. For example, Birge *et al.* (1978) determined a 30-day LC_{50} for rainbow trout embryos of 50 µg/L at a water hardness between 93 mg/L and 105 mg/L. The corresponding lethal threshold (LC_1) was estimated to be approximately 0.6 µg/L. Birge and Black (1980; as cited in Eisler 1998, hardness not reported) determined an LC_{10} of 11 µg/L for rainbow trout embryos exposed from fertilization through hatching. In Eisler's (1998b) review, LC_{50}s were reported of 60 µg/L and 90 µg/L at water hardness of 125 and 174 mg/L, respectively, for rainbow trout embryos that were exposed from fertilization through hatching. These results and the review by Birge *et al.* (1981) suggest that adverse effects are likely to occur to embryos exposed to nickel concentrations that are lower than the proposed chronic criterion.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for nickel is likely to result in sublethal effects to listed species considered in this opinion.

Summary of Effects: Nickel. The available evidence for nickel indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity) and reduced growth (moderately-high-intensity).

2.6.2.2.9 Selenium

Selenium Criteria. The proposed acute and chronic criteria for selenium (VI) are 190 µg/L and 5.0 µg/L, and for selenium (IV), 12.8 µg/L and 5.0 µg/L, respectively.

Tables 2.6.2.2.9.1 through 2.6.2.2.9.5 report toxicity data from the ECOTOX database for freshwater selenium, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.9.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium.

Criterion Freshwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 51334**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 2850**
Endpoint/Effect LC_{50}/Mortality	**pH 6.1-9.6**	**Harmonic Mean 7**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.4	NR	96H
0.4	NR	96H

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Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 51334**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 2850**
Endpoint/Effect LC_{50}/Mortality	**pH 6.1-9.6**	**Harmonic Mean 7**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.4	NR	96H
0.4	NR	96H
0.4	NR	96H
0.4	NR	24H
0.4	NR	96H
0.4	NR	24H
0.4	NR	96H
1	NR	96D
3.78	4.40 CM, 0.69 G	96H
3.98	4.40 CM, 0.69 G	96H
5	60 MM	96H
7	60 MM	96H
40	EGGS	96M
40	EGG	96M
40	EGG	96M
40	EGG-FRY	96H
45.6	NR	24H
45.6	NR	96H
45.6	NR	24H
45.6	NR	96H
45.6	NR	48H
45.6	NR	96H
45.6	NR	6H
45.6	NR	7H
45.6	NR	24H
50	2.78(2.4-3.0) CM	96D
50	2.78(2.4-3.0) CM	120D
100	EGG, LATE-EYED STAGE	96D
100	EGG, LATE-EYED STAGE	96D
150	3.10(2.4-3.7) CM	43D
170	FERTILIZATION THROUGH 4 DAY POST	28D
170	FERTILIZATION THROUGH 4 DAY POST	28D

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 51334**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 2850**
Endpoint/Effect LC_{50}/Mortality	**pH 6.1-9.6**	**Harmonic Mean 7**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
220	6.57(5.1-10.1) CM	120D
260	6.57(5.1-10.1) CM	96D
260	6.57(5.1-10.1) CM	96D
300	EGG, LATE-EYED STAGE	96D
300	EGG, LATE-EYED STAGE	96D
310	NR	24D
310	NR	24D
310	NR	96D
310	NR	96D
310	NR	96D
310	NR	96D
430	2.78(2.4-3.0) CM	21D
430	2.78(2.4-3.0) CM	120D
470	6.57(5.1-10.1) CM	48D
470	6.57(5.1-10.1) CM	96D
1000	EGG, LATE-EYED STAGE	96D
1000	EGG, LATE-EYED STAGE	96D
1000	EGG, LATE-EYED STAGE	96D
1100	60 MM	24D
1290	NR	96H
1800	NR	96H
1800	NR	24H
2200	NEWLY FERTILIZED EGG, <48 H	24D
2200	NEWLY FERTILIZED EGG, <48 H	24D
2200	NEWLY FERTILIZED EGG, <48 H	24D
2350	4.40 CM, 0.69 G	96H
2350	4.40 CM, 0.69 G	120H
2350	4.40 CM, 0.69 G	16H
2350	4.40 CM, 0.69 G	96H
2570	4.40 CM, 0.69 G	96H
2570	4.40 CM, 0.69 G	120H
2570	4.40 CM, 0.69 G	96H
2570	4.40 CM, 0.69 G	384H

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Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute **190 Micrograms Liter^{-1}**	**Temperature** **5-30° Celsius**	**Arithmetic Mean** **51334**
Criterion Concentration Chronic **5 Micrograms Liter^{-1}**	**Hardness** **17-340 mg/L $CaCO_3$**	**Geometric Mean** **2850**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.1-9.6**	**Harmonic Mean** **7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
2820	EGGS	28D
2820	EGGS	21D
3000	EGG, LATE-EYED STAGE	96D
3680	0.8 G	28D
3680	0.8 G	28D
3780	4.40 CM, 0.69 G	96H
3780	4.40 CM, 0.69 G	120H
3780	4.40 CM, 0.69 G	96H
3980	4.40 CM, 0.69 G	96H
3980	4.40 CM, 0.69 G	120H
3980	4.40 CM, 0.69 G	24H
4150	NR	4D
4150	EGG	28D
4150	NR	96D
4990	0.8 G	9D
4990	0.8 G	9D
5000	60 MM	16D
5000	60 MM	384H
5000	60 MM	24D
5170	EGG	28D
5330	0.8 G	9D
5330	0.8 G	9D
6280	JUVENILE, 41.6 MM, 0.47 G	96H
6280	JUVENILE, 41.6 MM, 0.47 G	96H
6300	NEWLY FERTILIZED EGG, <48 H	96D
6700	FRY, 0.5 G	96H
7000	JUVENILE, 49.6 MM, 1.04 G	96H
7200	0.8 G	96H
7200	0.8 G	96H
8200	0.8 G	96H
8200	0.8 G	96H
8600	FRY, 0.5 G	96H

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Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 51334**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 2850**
Endpoint/Effect LC_{50}/Mortality	**pH 6.1-9.6**	**Harmonic Mean 7**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
8800	0.8 G	96H
8800	0.8 G	9H
10000	EGG, LATE-EYED STAGE	96D
10000	EGG, LATE-EYED STAGE	96D
10400	60 MM	96D
10600	125 MM	96H
10600	125 MM	24H
10800	FRY, 0.46 G	96H
11500	60 MM	96H
11500	60 MM	96H
11500	60 MM	96H
11500	60 MM	96H
11600	FRY, 2.6 G	96H
12500	125 MM	96H
12500	125 MM	96H
13100	ADULT, 1.8 MO, 210.8 MM, 99.6 G	96H
13400	FRY, 0.7 G	96H
14800	FRY, 0.7 G	96H
17000	FRY, 0.5 G	96H
18300	FRY, 2.6 G	24H
18500	FRY, 0.5 G	96H
18600	FRY, 0.5 G	96H
19200	FRY, 0.31 G	96H
19600	FRY, 2.6 G	96H
23000	FRY, 0.5 G	24H
23800	ADULT, 1.8 MO, 210.8 MM, 99.6 G	48H
23900	FRY, 2.6 G	24H
25000	JUVENILE, 49.6 MM, 1.04 G	96H
25300	FRY, 0.5 G	96H
28200	FRY, 2.6 G	24H
29000	JUVENILE, 51.5 MM, 0.81 G	96H
29000	FRY, 1.7 G	96H

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 51334**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 2850**
Endpoint/Effect LC_{50}/Mortality	**pH 6.1-9.6**	**Harmonic Mean 7**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
35800	FRY, 0.5 G	96H
36100	FRY, 2.6 G	24H
36300	ADULT, 1.8 MO, 210.8 MM, 99.6 G	24H
38000	NR	96H
38000	NR	24H
38200	FRY, 0.7 G	24H
39000	NR	96H
39000	NR	96H
39300	FRY, 0.5 G	96H
48300	FRY, 0.5 G	24H
50500	FRY, 0.46 G	24H
53000	JUVENILE, 41.6 MM, 0.47 G	96H
53000	JUVENILE, 41.6 MM, 0.47 G	96H
56000	ALEVIN, 15.0 MM, 0.02 G	96H
57100	FRY, 0.6 G	96H
61000	ALEVIN, 29.8 MM, 0.24 G	96H
61000	ALEVIN, 29.8 MM, 0.24 G	96H
63700	ADULT, 1.8 MO, 210.8 MM, 99.6 G	7H
66500	FRY, 0.5 G	96H
74000	FRY, 0.5 G	96H
74200	ADULT, 1.8 MO, 210.8 MM, 99.6 G	6H
78000	ALEVIN, 14.3 MM, 0.01 G	96H
79000	ALEVIN, 20.8 MM, 0.10 G	96H
84000	FRY, 0.31 G	24H
85000	FRY, 0.31 G	96H
85000	FRY, 0.31 G	43H
86000	FRY, 0.7 G	96H
87000	ALEVIN	96H
138000	JUVENILE, 62.4 MM, 1.44 G	96H
151000	ALEVIN	24H
171000	FRY, 0.5 G	24H
274000	ALEVINE, 29.8 MM, 0.24 G	96H

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute **190 Micrograms Liter^{-1}**	**Temperature** **5-30° Celsius**	**Arithmetic Mean** **51334**
Criterion Concentration Chronic **5 Micrograms Liter^{-1}**	**Hardness** **17-340 mg/L $CaCO_3$**	**Geometric Mean** **2850**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.1-9.6**	**Harmonic Mean** **7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
274000	ALEVINE, 29.8 MM, 0.24 G	96H
320000	ALEVIN	24H
320000	ALEVIN	96H
360000	FRY, 0.7 G	24H
361000	FRY, 0.5 G	24H
369000	FRY, 1.7 G	96H
374000	ALEVIN, 20.8 MM, 0.10 G	96H
381000	FRY, 0.31 G	24H
560000	EYED EGG	24H
560000	EYED EGG	96H
1000000	EYED EGG	24H
1000000	EYED EGG	96H

Table 2.6.2.2.9.2 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium

Criterion Freshwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 68398**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 10953**
Endpoint/Effect Mortality	**pH 6.1-9.6**	**Harmonic Mean 417**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
40	EGG	12M
40	EGG	12M
47.2	SAC FRY, 21.7 MM, 0.075 G	5D
100	EGG, LATE-EYED STAGE	5D
300	EGG, LATE-EYED STAGE	24D
300	EGG, LATE-EYED STAGE	5D
1000	EGG, LATE-EYED STAGE	20D
1000	EGG, LATE-EYED STAGE	5D
1000	EGG, LATE-EYED STAGE	5D
1100	60 MM	16D
2200	NEWLY FERTILIZED EGG, <48 H	5D
3000	EGG, LATE-EYED STAGE	70D
6300	NEWLY FERTILIZED EGG, <48 H	90D
8600	FRY, 0.5 G	24H
10000	EGG, LATE-EYED STAGE	42D
10400	60 MM	16D
13100	ADULT, 1.8 MO, 210.8 MM, 99.6 G	16H
16600	1.6 G, FRY	7.6H
17200	1.6 G, FRY	49H
23800	ADULT, 1.8 MO, 210.8 MM, 99.6 G	120H
36300	ADULT, 1.8 MO, 210.8 MM, 99.6 G	12H
38200	FRY, 0.7 G	70H
39600	1.6 G, FRY	7.6H
43200	FRY, 2.4 G	5H
50100	FRY, 2.4 G	5H
50500	FRY, 0.46 G	20H
63700	ADULT, 1.8 MO, 210.8 MM, 99.6 G	16H
63800	1.6 G, FRY	7.6H
65400	FRY, 2.4 G	5H
74000	FRY, 0.5 G	5H

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Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 68398**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 10953**
Endpoint/Effect Mortality	**pH 6.1-9.6**	**Harmonic Mean 417**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
74200	ADULT, 1.8 MO, 210.8 MM, 99.6 G	90H
79400	FRY, 1.8 G	7.6H
86000	FRY, 0.7 G	5H
94000	FRY, 1.6 G	90H
136000	FRY, 1.6 G	24H
236000	FRY, 1.6 G	90H
360000	FRY, 0.7 G	42H
361000	FRY, 0.5 G	5H
600000	FRY, 1.6 G	30H

Table 2.6.2.2.9.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium.

Criterion Freshwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 619**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-334 mg/L $CaCO_3$**	**Geometric Mean 167**
Endpoint/Effect NOEC/Mortality	**pH 6.1-9.6**	**Harmonic Mean 73**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
40	EGGS	12M
40	EGG-FRY	1Y
2200	NEWLY FERTILIZED EGG, <48 H	90D
47.2	SAC FRY, 21.7 MM, 0.075 G	1D
99.5	SAC FRY, 21.7 MM, 0.075 G	90D
1290	NR	12H

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Table 2.6.2.2.9.4 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium.

Criterion Freshwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 34707**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-340 mg/L $CaCO_3$**	**Geometric Mean 1513**
Endpoint/Effect Growth	**pH 6.1-9.6**	**Harmonic Mean 16**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1	NR	21D
40	EGG	4M
47.2	SAC FRY, 21.7 MM, 0.075 G	30D
50	2.78(2.4-3.0) CM/	42D
50	2.78(2.4-3.0) CM/	120D
99.5	SAC FRY, 21.7 MM, 0.075 G	90D
220	6.57(5.1-10.1) CM/	30D
310	NR	12D
2200	NEWLY FERTILIZED EGG, <48 H	30D
7000	60 MM	30H
7000	JUVENILE, 49.6 MM, 1.04 G	12H
10000	5-10 CM	42H
25000	JUVENILE, 49.6 MM, 1.04 G	21H
35800	FRY, 0.5 G	90H
39300	FRY, 0.5 G	30H
57100	FRY, 0.6 G	30H
66500	FRY, 0.5 G	90H
374000	ALEVIN, 20.8 MM, 0.10 G	4H

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Table 2.6.2.2.9.5 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater selenium.

Criterion Freshwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 190 Micrograms Liter^{-1}	**Temperature 5-30° Celsius**	**Arithmetic Mean 17450**
Criterion Concentration Chronic 5 Micrograms Liter^{-1}	**Hardness 17-334 mg/L $CaCO_3$**	**Geometric Mean 4844**
Endpoint/Effect Cellular	**pH 6.1-9.6**	**Harmonic Mean 392**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
100	EGG, LATE-EYED STAGE	21D
10000	EGG, LATE-EYED STAGE	20D
11400	FRY, 0.7 G	21H
48300	FRY, 0.5 G	20H

Selenium Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50

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percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to selenium, NMFS added an additional step to its analysis for selenium to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 470 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.9.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.9.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 1,000,000 µg/L to a high of an LC_{100} at a concentration of 0.4 µg/L. In other words, the acute criterion of 470 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{1.8}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for selenium, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for selenium, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

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Sublethal Effects. The behavior of selenium in biological systems is complex. Selenium is a metalloid that exists in three oxidation states in water: selenide (-2), selenite (+4) and selenate (+6). The toxicity of selenium varies with its chemical species. Inorganic selenium is the predominant form in aquatic environments. Organic and reduced forms of selenium (*e.g.*, seleno-methionine and selenite) are generally more toxic and will bioaccumulate more readily (Kiffney and Knight 1990, Besser *et al.* 1993). Toxicity also varies with the species exposed. Species at higher trophic levels, such as piscivorous fish and birds, are affected by the lowest concentrations of selenium. Long-term, low-level exposures from water or food appear to have the greatest effect on aquatic organisms (Lemly 1985). Like mercury, selenium bioaccumulates in muscle tissue and is associated with reproductive impairment and reduced hatching success. Toxic effects of selenium range from physical malformations during embryonic development to sterility and death. Other effects include reduced smolting success, reduced red blood cell volumes and cellular blood iron content, and impaired immune responses (Eisler 1985b, Hamilton *et al.* 1986, Lemly and Smith 1987, Felton *et al.* 1990, Sorensen 1991).

Of all the priority and non-priority pollutants, selenium has the narrowest range of what is beneficial for biota and what is detrimental. Aquatic and terrestrial organisms require 0.5 µg/g dry weight (dw) of selenium in their diet to sustain metabolic processes, whereas concentrations of selenium that are only an order of magnitude greater than the required level have been shown to be toxic to fish. Acute effects are observed after short exposure durations of typically 96 hours or less. Acute effects from the inorganic forms of selenium, selenite and selenate, require concentrations exceeding 300 µg/L, concentrations rarely reached in the environment. In contrast, toxic effects from long-term chronic exposure via diet and water can result in reduction of species in aquatic systems with aqueous concentrations less than 20 µg/L (Lemly 1985 as cited in EPA 2008). As a result of the greater sensitivity to selenium from chronic exposures, water quality management practices over the last 10-15 years have focused on the control of chronic effects. Studies have shown that diet is the primary route of exposure that controls chronic toxicity to fish, the group considered to be the most sensitive to chronic selenium exposure (Coyle *et al.* 1993, Hamilton *et al.* 1990, Hermanutz *et al.* 1996 as cited in EPA 2008).

Effects of selenium toxicity to freshwater organisms range from physical malformations during embryonic development to sterility and death (Lemly and Smith 1987) and include reduced hatch, reduced growth, behavioral alterations (avoidance), shifts in species composition of freshwater algal communities, loss of equilibrium, lethargy, muscle spasms, protruding eyes, liver degeneration, reduction in blood hemoglobin, chromosomal aberrations, and reduced survival (Eisler 1985b).

Selenium is an essential nutrient for normal cell functions. Inadequate dietary uptake (food and water) of selenium results in selenium deficiency syndromes such as reproductive impairment, poor body condition, and immune system dysfunction (Oldfield 1990, CAST 1994). However, excessive dietary uptake of selenium also results in toxicity syndromes that are similar to the deficiency syndromes (Koller and Exon 1986). Selenium is a "hormetic" chemical, *i.e.*, a chemical for which levels of safe dietary uptake are bounded on both sides by adverse-effects thresholds. Most essential nutrients are hormetic, but what distinguishes selenium from other nutrients is the very narrow range between the deficiency threshold and the toxicity threshold (Wilber 1980, Sorensen 1991, Skorupa *et al.* 1996, USDI-BOR/FWS/GS/BIA 1998). In other

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words, the difference between useful amounts of selenium and toxic amounts is small.

Water-borne selenium is depurated in fish via a passive excretion pathway, while dietary selenium is excreted more actively. The half-life of selenium is inversely proportional to dietary loading. Inorganic selenium absorbed from water is stored in fish as inorganic selenium. However, inorganic selenium absorbed from the diet is transformed by the liver to an organic form that is more toxic, but can be excreted easily (Hodson *et al.* 1984). Nevertheless, the transformation of selenium to organoselenium is associated with bioconcentration in fish ovaries, resulting in significant pathology and reproductive failure (Baumann and Gillespie 1986, Srivastava and Srivastava 1994). Selenium taken up from water is absorbed across the gills and taken directly to all tissues. Dietary selenium is taken up through the gut, from which the liver receives its blood supply via a portal system. The tissue distribution of selenium within fish is a function of the loading rate, but not the source of selenium (Hodson and Hilton 1983, Sorensen 1991).

Selenium protects some species from the toxicity of other chemicals. For example, selenium antagonizes mercury toxicity in rainbow trout (Eisler 1985b). Selenium criteria are not hardness dependent. The dose-response curves for selenium are relatively steep, indicating a rapid shift to toxic conditions with small increases in metal concentration (Lemly 1998, Skorupa 1998)

Salmonids are sensitive to chronic selenium contamination (Lemly 1996a,b). Depending on the form of selenium and the life-stage of fish considered, water-borne concentrations of selenium less than 5 µg/L can have direct toxic effects on salmonids (Hodson *et al.* 1980, Moore *et al.* 1990). Lemly (1998) concluded that the larval fish life stage is the most sensitive to exposure to selenium, with adverse effects expressed through teratogeny and mortality. Hodson *et al.* (1980) reported that rainbow trout (*O. mykiss*) eggs respond physiologically (reduced median time to hatch) at selenium (as selenite) concentrations above 4.3 µg/L. Studies have also shown that chronic exposure to selenium can reduce fish growth in terms of weight and to a lesser extent length (Eisler 1985b, Hamilton *et al.* 1986, Hamilton *et al.* 1990). Van Derveer and Canton (1997) concluded, based on a sediment-water transfer model, that a 5 µg/L concentration may not always avoid harm to listed salmonids, depending on the organic carbon content in the sediment. Using their model, Mebane (2000) estimated protective selenium levels ranging between 2 µg/L and 8 µg/L for higher gradient mountain streams in the upper Salmon River basin, effectively demonstrating that the chronic criterion is unlikely to avoid adverse effects under the range of environmental conditions.

Skorupa (1998) noted collapse of natural fish populations chronically exposed to 10 µg/L selenium in selenite-dominated waters. Hodson *et al.* (1980) observed significant mortality in rainbow trout eyed eggs exposed to concentrations greater than or equal to 25 µg/L after 44 weeks, and hatchability of eggs was affected at concentrations as low as 16 µg/L. Hamilton *et al.* (1986) determined that exposures to 17 µg/L (selenate:selenite ratio = 6:1) for 30 days caused a significant increase in mortality of Chinook salmon fry.

Kennedy *et al.* (2000) determined, in the case of eggs taken from wild female cutthroat living in a contaminated river with higher exposure concentrations (13.3 µg/L to14.5 µg/L), that there was no significant effect of the resulting elevated selenium concentrations in the eggs on subsequent

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survival to hatch or fry deformities when the eggs and fry were reared in water with concentrations below 1 µg/L. They concluded that their result may reflect an evolved tolerance to higher tissue concentrations of selenium in the test population, although it is possible that the absence of subsequent exposure during development may also have influenced the results.

In the CTR biological opinion (USFWS and NMFS 2010), the NMFS and FWS determined that under most circumstances, a 5 µg/L chronic criterion should be protective of aquatic life with regard to direct contact toxicity. However, based on data collected by the U.S. Department of Interior's National Irrigation Water Quality Program from 26 study areas in 14 western states, the Services determined that a 5 µg/L chronic criterion for selenium is only 50% to 70% protective (Seiler and Skorupa 1999), as opposed to the 95% level of protection that EPA's national water quality criteria are intended to achieve.

The consensus of researchers lately, however, is that water-borne exposure to selenium in any form is much less important than dietary exposure and bioaccumulation in determining the potential for chronic effects (EPA 1998). The Services similarly determined in the CTR biological opinion that the 5 µg/L chronic aquatic life criterion for selenium does not protect listed fish in other respects because of bioaccumulation hazards, which may be a reason for results listed above that reported finding adverse effects at concentrations below the proposed criterion. Determinations of effect using solely studies of water-borne exposure underestimate the danger of selenium exposure to fish through bioaccumulation (Hermanutz *et al.* 1992).

Bioaccumulation. Dietary bioaccumulation of selenium is the most dangerous exposure pathway for salmonids and other fish species (EPA 1998). Bioconcentration of selenium is influenced by exposure concentration, selenium speciation, water temperature, age of receptor organism, organ, tissue specificity, and mode of administration (Eisler 1985b). Lemly and Smith (1987) noted that bioconcentration factors in fish experiencing chronic toxicity have ranged from around 100 to more than 30,000, and that bioconcentration can occur when water-borne selenium concentrations are within the range of 2 µg/L to 5 µg/L. Selenium bioconcentration factors appear to be inversely related to water exposure concentrations (EPA 1998). A concentration as little as 0.1 µg/L of dissolved selenomethionine has been found to be sufficient to cause bioaccumulation of an average concentration of 14.9 mg/kg (dry weight) selenium in zooplankton (Besser *et al.* 1993), a concentration that could cause dietary toxicity to most species of fish (Lemly 1996a). Fish bioconcentrate selenium in higher levels in ovaries than in muscle tissues (Lemly 1985, Hamilton *et al.* 1990) and milt (Hamilton and Waddell 1994).

As for the water-borne case, selenium biomagnification factors similarly appear to be inversely related to dietary exposure concentrations (Hamilton *et al.* 1986). Hamilton *et al.* (1990) determined that Chinook salmon fingerlings fed organic selenium in their study accumulated the metal to whole body concentrations that were not significantly different from that in their artificial diet, suggesting that biomagnification may not be significant in this life stage of listed salmonids. Overall, however, magnitudes of biomagnification appear to range from two to six times between producers and lower consumers including invertebrates and forage fish (Lemly and Smith 1987). Piscivorous fish generally accumulate the highest levels of selenium and are one of the first organisms affected by selenium exposure, followed by planktivores and omnivores (Lemly 1985).

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Studies of dietary uptake indicate that selenium can be bioaccumulated through the diet to tissue levels resulting in adverse effects in fish. In a comprehensive review, Lemly (1996b) determined that rainbow trout were sensitive to selenium contamination and exhibited toxic symptoms when their tissue concentrations exceeded 2 mg/kg dry weight in several experiments, and 1 mg/kg in one experiment (note: Lemly (1996b) estimated dry weight concentrations to be four times wet-weight concentrations). Mortality was associated with tissue concentrations greater than 5 mg/kg dry weight (Lemly 1996b). However, Hamilton *et al.* (1986), noted adverse effects on parr-smolt transformation for fall Chinook salmon fed a selenium-contaminated diet when whole-body tissue concentrations were much higher, at 23 mg/kg dry weight (4.9 mg/kg wet weight; conversion factor = 4.63).

Adverse effects have been demonstrated in fish when dietary concentrations exceed approximately 3 mg/kg dry weight (Hamilton *et al.* 1990, Lemly 1996b). However, selenium is also required in the diet as a nutrient at concentrations of about 0.1 to 0.5 mg/kg dry weight (Lemly 1998), so there is a narrow range between healthy and toxic dietary concentrations. Lemly (1996b) noted food chain concentrations on the order of 10 mg/kg to 60 mg/kg were associated with water-borne selenium concentrations in the 2 µg/L to 16 µg/L range. The NMFS and FWS (NMFS 2000) determined in the CTR biological opinion that, assuming a bioaccumulation factor for dry weight concentrations of selenium in aquatic invertebrates (compared to water) of 1,800, a water-borne concentration of as little as 1.8 µg/L selenium could result in food concentrations averaging more than 3 mg/kg selenium, and therefore may be sufficient to result in adverse effects in salmonids.

Variability in experimental and natural conditions influence conclusions regarding safe fish tissue levels, and controlled dietary studies of selenium uptake are subject to questions regarding whether the method through which selenium was administered in the diet reflects natural feeding patterns and food types. Nonetheless, the results of such studies suggest collectively that adverse effects related to bioaccumulation to are likely to occur when water-borne concentrations are below the proposed chronic criterion of 5 µg/L.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for selenium is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. According to Lemly (1996b), the results of field studies generally indicate that benthic invertebrates can accumulate relatively large quantities of selenium (*e.g.*, 20 mg/kg to 370 mg/kg dry weight) and still maintain stable, reproducing populations. Peterson and Nebeker (1992) estimated a dry weight bioaccumulation factor of 1,800 for aquatic insects and invertebrates in the Kesterson National Wildlife Refuge, and noted that Lemly had summarized wet weight factors in a previous review to range between 371 and 5,200. The most significant concern for food organisms from the perspective of listed species is probably bioaccumulation from eating aquatic invertebrates that themselves have elevated selenium levels, rather than changes in aquatic invertebrate production due to selenium toxicity. Hence, the proposed criteria can result in diminished food source quality for listed species through the effects of bioaccumulation.

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Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for selenium is unlikely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Selenium. The available evidence for selenium indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderate intensity), reduced growth (moderate intensity), cellular trauma (low intensity), and bioaccumulation (moderately-high-intensity).

2.6.2.2.10 Silver

Silver Criteria. The proposed acute and chronic criteria for silver are 3.2 µg/L and 0.10 µg/L, respectively, at a hardness of 100 mg/L $CaCO_3$.

Tables 2.6.2.2.10.1 through 2.6.2.2.10.3 report toxicity data from the ECOTOX database for freshwater silver, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.10.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater silver.

Criterion Freshwater Silver		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 3.2 Micrograms Liter^{-1}	**Temperature 9.7-18.4° Celsius**	**Arithmetic Mean 345**
Criterion Concentration Chronic 0.10 Micrograms Liter^{-1}	**Hardness 5-255 mg/L $CaCO_3$**	**Geometric Mean 63**
Endpoint/Effect LC_{50}/Mortality	**pH 6.2-9**	**Harmonic Mean 21**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.28	167 MM	96H
2.71	JUVENILE, 2.2 G	96H
7.32	20 D	96H
9.98	20 D	96H
10.03	1-4 G, JUVENILE	96H
13.52	0.25-1.0G	96H
16.03	0.25-1.0 G	96H
16.32	20 D	96H
20.37	1.2 G	96H
22.22	1.0-1.5 G	96H
22.85	20 D	96H

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Criterion Freshwater Silver		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **3.2 Micrograms Liter^{-1}**	**Temperature** **9.7-18.4° Celsius**	**Arithmetic Mean** **345**
Criterion Concentration Chronic **0.10 Micrograms Liter^{-1}**	**Hardness** **5-255 mg/L $CaCO_3$**	**Geometric Mean** **63**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.2-9**	**Harmonic Mean** **21**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
25.38	20 D	96H
27.05	1.0-1.5 G	96H
27.72	JUVENILE, 2.2 G	96H
28.88	NR	96H
31.37	0.25-1.0 G	96H
33.77	69 MM	96H
34.30	0.25-1.0 G	96H
34.34	1-3 G	96H
36.66	Juvenile	
37.56	20 D	96H
38.00	2.5-3.5 G	96H
40.77	NR	96H
40.77	NR	96H
43.73	alevin, 0.24 g	
43.96	Juvenile	
45.33	FORK LENGTH, 0.2 G, 32 MM	96H
47.57	NR	96H
49.20	3-10 G	96H
49.24	Juvenile, 0.41 g	
53.58	Juvenile, 0.1 - 0.2 g	
53.58	Juvenile, 0.51 - 1.44 g	
53.68	3-10 G	96H
59.84	1-3 G	96H
61.46	FORK LENGTH, 0.2 G, 28 MM	96H
63.42	alevin, 0.1 g	
63.79	20 D	96H
69.85	173 MM	96H
75.64	Juvenile, 0.6 g	
83.95	146 MM	96H
93.99	FORK LENGTH, 0.2 G, 28 MM	96H
95.52	1-3 G	96H

Criterion Freshwater Silver		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **3.2 Micrograms Liter^{-1}**	**Temperature** **9.7-18.4° Celsius**	**Arithmetic Mean** **345**
Criterion Concentration Chronic **0.10 Micrograms Liter^{-1}**	**Hardness** **5-255 mg/L $CaCO_3$**	**Geometric Mean** **63**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **6.2-9**	**Harmonic Mean** **21**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
115.08	20 D	96H
117.75	1-3 G	96H
132.46	1-3 G	96H
191.60	20 D	96H
299.64	Juvenile	
350.66	2.5-3.5 G	96H
396.69	Juvenile	
1102.18	JUVENILE, 2.2 G	96H
1352.01	JUVENILE, 2.2 G	96H
2704.01	JUVENILE, 2.2 G	96H
2718.71	JUVENILE, 2.2 G	96H
3762.10	JUVENILE, 2.2 G	96H
4070.71	JUVENILE, 2.2 G	96H

Table 2.6.2.2.10.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater silver.

Criterion Freshwater Silver		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 3.2 Micrograms Liter^{-1}	**Temperature 5-18.4° Celsius**	**Arithmetic Mean 136**
Criterion Concentration Chronic 0.10 Micrograms Liter^{-1}	**Hardness 12.7-140 mg/L $CaCO_3$**	**Geometric Mean 31**
Endpoint/Effect Growth	**pH 6.1-8.8**	**Harmonic Mean 3**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.96	20 D	28D
1.3	25 (20-30) G, JUVENILE	28D
77	25 (20-30) G, JUVENILE	18M
98.2	20 D	6W
196	20 D	18M
440	20 D	6W

Table 2.6.2.2.10.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater silver.

Criterion Freshwater Silver		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 3.2 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 1.2**
Criterion Concentration Chronic 0.10 Micrograms Liter^{-1}	**Hardness 28-36 mg/L $CaCO_3$**	**Geometric Mean 1.1**
Endpoint/Effect NOEC/Mortality	**pH NR**	**Harmonic Mean 0.98**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.68	NR	NR
1.77	NR	NR

Silver Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

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The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to selenium, NMFS added an additional step to its analysis for selenium to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS

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calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 3.2 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.10.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.10.1, predicts a magnitude of effect ranging from a low of an LC_{zero} at a concentration of 4,070.71 µg/L to a high of an LC_{100} at a concentration of 1.28 µg/L. In other words, the acute criterion of 3.2 µg/L has an equivalent toxicity potential predicted to kill zero percent to 100 percent, with a median toxicity potential of an $LC_{3.4}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for silver, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for silver, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects, but may not suffer chronic toxic effects.

Sublethal Effects. Silver is one of the most toxic metals to freshwater organisms and is highly toxic to all life stages of salmonids. Ionic silver is the primary form responsible for causing acute toxicity in freshwater fish (EPA 1980o, 1987b, Eisler 1996, Hogstrand and Wood 1998, Bury *et al.* 1999a). Toxicity varies widely depending on the anion present; silver nitrate has a much higher toxicity than silver chloride or silver thiosulfate, by approximately four orders of magnitude (Hogstrand *et al.* 1996). Documented effects of silver toxicity in fish include interruption of ionoregulation at the gills, cell damage in the gills, altered blood chemistry, interference with zinc metabolism, premature hatching, and reduced growth rates (Hogstrand and Wood 1998, Webb and Wood 1998).

Silver is not known to be mutagenic, teratogenic, or carcinogenic (Eisler 1996). It bioconcentrates and may bioaccumulate (Eisler 1996). Toxicity of Ag may be altered by a number of factors including pH, organic carbon, cation exchange capacity, presence of mixtures (Ratte 1999), sulfides, and duration of exposure. Silver, as ionic Ag^+, is one of the most toxic metals known to aquatic organisms in laboratory testing (Nebeker *et al.* 1983). Aquatic insects concentrate silver in relative proportion to environmental levels (Nehring 1976 as cited in EPA 2008), and more efficiently than most fish species (Diamond *et al.* 1990 as cited in EPA 2008). Effects of silver toxicity to freshwater algae and phytoplankton include growth inhibition and altered species composition and species succession (Eisler 1996 as cited in EPA 2008). Effects of silver toxicity to freshwater invertebrates include inhibited feeding and coordination, reduced growth, elevated oxygen consumption, and reduced survival (Eisler 1996 as cited in EPA 2008). Effects of silver toxicity to freshwater fish include inhibited ionic flux across gills, reduced growth, premature hatch, and reduced survival (Eisler 1996 as cited in EPA 2008). Interspecies

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differences in the ability to accumulate, retain, and eliminate silver are large (Baudin *et al.* 1994 as cited in EPA 2008).

In the original aquatic life criteria document for silver (EPA 1980o), variation in the results of a limited number of chronic toxicity tests precluded determining a freshwater chronic criterion, but it was also noted that chronic toxicity may occur to selected aquatic organisms at concentrations as low as 0.12 µg/L.

The work of Davies *et al.* (1978) suggests that the maximum acceptable silver concentration to prevent chronic mortality in rainbow trout embryos, fry, and juveniles, and avoid premature hatching, is less than 0.17 µg/L for a water hardness equal to 26 mg/L. Nebeker *et al.* (1983 as cited in Hogstrand and Wood 1998) determined that the maximum acceptable toxicant concentration of silver to prevent inhibition of growth of steelhead embryos was less than 0.1 µg/L for a water hardness equal to 36 mg/L.

The EPA (1987b) reported the results of Davies and Goettl (1978), where chronic limits for silver were listed as between 0.03 µg/L and 0.06 µg/L for a water hardness equal to 28 mg/L, and between 0.03 µg/L and 0.06 µg/L for a water hardness equal to 29 mg/L. Birge *et al.* (1981) estimated an LC_{10} and LC_1 of 0.9 µg/L and 0.1 µg/L, respectively, for rainbow trout embryos and larvae in static renewal tests lasting until 4 days post-hatching.

Accumulation of silver is predominantly associated with exposure to its ionic forms rather than complexes. Bioaccumulation occurs primarily in the liver (Hogstrand *et al.* 1996, Galvez and Wood 1997, 1999). Significant food chain biomagnification by fish has been reported to be unlikely because of the low silver concentrations typically encountered in the aquatic environment (Eisler 1996, Hogstrand and Wood 1998, Ratte 1999).

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for silver is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. The LC_{50}s that have been reported for cladocera species that are below the acute criterion (EPA 1980o). Other invertebrate taxa serving as potential food for juvenile salmonids die only at concentrations that are above the acute criterion. Other observed adverse effects include reductions in growth and inhibition of molting (EPA 1980o, Eisler 1996, Call *et al.* 1999). Chronic effects appear to be documented only for daphnids when silver concentrations are below the proposed chronic criterion. Aquatic invertebrates have been reported to accumulate silver more efficiently than fish, in concentrations that are proportional to exposure levels (Eisler 1996, Hogstrand and Wood 1998). Studies involving silver sulfide bioaccumulation through sediment interactions from an amphipod and an oligochaete indicated low potential for listed species to accumulate harmful silver concentrations through this exposure pathway (Hirsch 1998a,b). Adverse effects of the silver criterion on the food organisms of listed species may be potentially meaningful when cladoceran species are a primary food source.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for silver is likely to appreciably affect invertebrate productivity and abundance.

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Summary of Effects: Silver. The available evidence for silver indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (moderate intensity), and sublethal effects (moderate intensity).

2.6.2.2.11 Tributyltin

Tributyltin Criteria. At a pH of 7.5 and temperature of 18°C the acute criterion for TBT is 0.46 µg/L, and the chronic criterion is 0.063 µg/L, respectively.

Tables 2.6.2.2.11.1 through 2.6.2.2.11.5 report toxicity data from the ECOTOX database for freshwater tributyltin, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.2.2.11.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		**Data Set ECOTOX**
Criterion Concentration Acute 0.46 Micrograms Liter^{-1}	**Temperature 4-15.5° Celsius**	**Arithmetic Mean 8**
Criterion Concentration Chronic 0.063 Micrograms Liter^{-1}	**Hardness 246-280 mg/L $CaCO_3$**	**Geometric Mean 3**
Endpoint/Effect LC_{50}	**pH 6.4-7.95**	**Harmonic Mean 1**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.21	13.8 G	96H
0.54	8.3-8.8 CM, 5.6-6.4 G	6D
0.6	NR	96D
0.6	NR	96D
0.6	NR	24D
1.02	1.47 G	96H
1.16	1.47 G	96H
1.34	1.47 G	96H
3.5	8.8 CM, 6.4 G	96D
4.6	0.77 g	96H
4.84	5.94 G	96H
5.5	1.4 g	96H
6.2	0.68(0.17-1.2) G, 45(39-53) MM	96H
6.6	0.68(0.17-1.2) G, 45(39-53) MM	48H
7.9	0.68(0.17-1.2) G, 45(39-53) MM	72H
11.2	JUVENILE	96H

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Criterion Freshwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute **0.46 Micrograms Liter^{-1}**	**Temperature** **4-15.5° Celsius**	**Arithmetic Mean** **8**
Criterion Concentration Chronic **0.063 Micrograms Liter^{-1}**	**Hardness** **246-280 mg/L $CaCO_3$**	**Geometric Mean** **3**
Endpoint/Effect **LC_{50}**	**pH** **6.4-7.95**	**Harmonic Mean** **1**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
11.2	JUVENILE	96H
15	0.68(0.17-1.2) G, 45(39-53) MM	48H
21	UNDER-YEARLING	96H
50	NR	96MIN

Table 2.6.2.2.11.2 LC_{100} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute **0.46 Micrograms Liter^{-1}**	**Temperature** **4-15.5° Celsius**	**Arithmetic Mean** **28**
Criterion Concentration Chronic **0.063 Micrograms Liter^{-1}**	**Hardness** **246-280 mg/L $CaCO_3$**	**Geometric Mean** **28**
Endpoint/Effect **LC_{100}**	**pH** **6.4-7.95**	**Harmonic Mean** **28**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
28	UNDER-YEARLING	14H

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Table 2.6.2.2.11.3 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.46 Micrograms Liter^{-1}	**Temperature 4-15.5° Celsius**	**Arithmetic Mean 7.3**
Criterion Concentration Chronic 0.063 Micrograms Liter^{-1}	**Hardness 246-280 mg/L $CaCO_3$**	**Geometric Mean 2.4**
Endpoint/Effect Growth	**pH 6.4-7.95**	**Harmonic Mean 1.1**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.5	3 WK	21D
1.46	24.5 G, 25.1 CM FORK LENGTH	NR
20	24.5 G, 25.1 CM FORK LENGTH	21H

Table 2.6.2.2.11.4 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.46 Micrograms Liter^{-1}	**Temperature 4-15.5° Celsius**	**Arithmetic Mean 1**
Criterion Concentration Chronic 0.063 Micrograms Liter^{-1}	**Hardness 246-280 mg/L $CaCO_3$**	**Geometric Mean 0.95**
Endpoint/Effect Physiological	**pH 6.4-7.95**	**Harmonic Mean 0.86**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.6	4-24 MO, 8.5-20.7 CM, 6.0-94.5 G	65D
1.49	24.5 G, 25.1 CM FORK LENGTH	28H

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Table 2.6.2.2.11.5 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater tributyltin.

Criterion Freshwater Tributyltin		**Data Set ECOTOX**
Criterion Concentration Acute 0.46 Micrograms Liter^{-1}	**Temperature 4-15.5° Celsius**	**Arithmetic Mean 0.77**
Criterion Concentration Chronic 0.063 Micrograms Liter^{-1}	**Hardness 246-280 mg/L $CaCO_3$**	**Geometric Mean 0.69**
Endpoint/Effect Cellular	**pH 6.4-7.95**	**Harmonic Mean 0.63**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.6	4-24 MO, 8.5-20.7 CM, 6.0-94.5 G	28D
0.5	3 WK	28D
0.5	3 WK	28D
1.49	24.5 G, 25.1 CM FORK LENGTH	72H

TributyltinToxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50

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percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to tributyltin, NMFS added an additional step to its analysis for tributyltin to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 0.46 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.11.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.11.1, predicts a magnitude of effect ranging from a low of an $LC_{0.5}$ at a concentration of 50 µg/L to a high of an LC_{100} at a concentration of 0.21 µg/L. In other words, the acute criterion of 0.46 µg/L has an equivalent toxicity potential predicted to kill 0.5 percent to 100 percent, with a median toxicity potential of an $LC_{4.9}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute criterion concentration for tributyltin, which implies that listed species exposed to waters equal to criteria concentrations will suffer acute toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute criterion concentration for tributyltin, which implies that listed species exposed to waters equal to criteria concentrations may not suffer acute toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute criterion concentration will suffer acute toxic effects.

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None of the toxicity studies reported concentrations that are less than the chronic criterion for tributyltin, which implies that listed species exposed to waters equal to the chronic criterion concentration may not suffer chronic toxic effects. Based on the available toxicity data and the considerations of the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the chronic criterion concentration may not suffer chronic toxic effects.

Summary of Effects: TBT. The available evidence for TBT indicates that listed species exposed to waters equal to the acute criterion concentration will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (moderate intensity), physiological trauma (moderate intensity), and cellular trauma (moderate intensity).

2.6.2.2.12 Zinc

Zinc Criteria. At hardness of 100 mg/L, the acute criterion is 120 µg/L, and the chronic criterion is 120 µg/L, respectively.

Tables 2.6.2.2.12.1 through 2.6.2.2.12.7 report toxicity data from the ECOTOX database for freshwater zinc, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters, the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.2.2.12.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 5-18° Celsius**	**Arithmetic Mean 1172**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 5-350 mg/L $CaCO_3$**	**Geometric Mean 1190**
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.3**	**Harmonic Mean 818**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
238	7 MO, 4.95 G, 8.6 CM, JUVENILE	96H
265	LARVAE	96H
268	7 MO, 4.95 G, 8.6 CM, JUVENILE	96H
308	3.9-6.8 CM FORK LENGTH	96H
316	SWIM-UP, 0.17 G	96H
330	SWIM-UP, 0.23 G	96H
330	7 MO, 4.95 G, 8.6 CM, JUVENILE	96H
353	7 MO, 4.95 G, 8.6 CM, JUVENILE	96H
412	FINGERLING, 2-4 G	96H
425	JUVENILE, 5 MO, 3.0 G, 7.0 CM	120H
444	55 MM	96H
453	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
462	PARR, 6.96 G, 8.6 CM	96H
478	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
487	2.36-3.01 G	96H
487	2.36-3.01 G	96H
487	2.36-3.01 G	168H
510	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
530	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
565	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
616	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
620	NR	96H
628	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
678	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
689	JUVENILE, 3.9 G	96H
709	JUVENILE, 3-10 G	96H
716	FY, 2.36-3.01 G	96H
716	FY, 2.36-3.01 G	168H
720	EYED STAGE	96H

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Criterion Freshwater Zinc		Data Set ECOTOX Hardness=100
Criterion Concentration Acute **120 Micrograms Liter^{-1}**	**Temperature** **5-18° Celsius**	**Arithmetic Mean** **1172**
Criterion Concentration Chronic **120 Micrograms Liter^{-1}**	**Hardness** **5-350 mg/L $CaCO_3$**	**Geometric Mean** **1190**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **4.7-8.3**	**Harmonic Mean** **818**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
728	NR	96H
743	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
847	70 MM	96H
861	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
959	JUVENILE, 4.9 G	96H
962	190 MM	96H
1166	30.5 g	
1173	JUVENILE, 5 MO, 3.0 G, 7.0 CM	96H
1193	JUVENILE, 28.4 G	96H
1361	JUVENILE	96H
1471	JUVENILE, 28.4 G	96H
1509	JUVENILE, 3.9 G	96H
1573	PARR, 11.58 G, 9.6 CM	96H
1577	ALEVIN, 1 MO	115H
1686	120 MM	96H
1768	JUVENILE, 4.9 G	96H
1903	140 MM	96H
2010	NR	96H
2191	3-5 G	96H
2197	22.6 g	
2212	SMOLT, 68.19 G, 18.8 CM	96H
2246	ALEVIN, 0.05 G	96H
2251	179 MM	96H
2382	SMOLT, 32.46 G, 14.4 CM	96H
2385	ADULT, 16-18 CM	96H
2564	JUVENILE	96H
2642	PARRI, 9 MO	96H
2674	110 MM	96H
2769	ALEVIN	96H
2865	NR	96H
2885	JUVENILE, 3.0 G	96H

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Criterion Freshwater Zinc		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 5-18° Celsius**	**Arithmetic Mean 1172**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 5-350 mg/L $CaCO_3$**	**Geometric Mean 1190**
Endpoint/Effect LC_{50}/Mortality	**pH 4.7-8.3**	**Harmonic Mean 818**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
2906	ALEVINS, 2-D POSTHATCH	144H
3111	JUVENILE, 19.0 G	96H
3466	Juvenile	
3691	JUVENILE, 3.0 G	96H
3700	FY, 2.36-3.01 G	168H
3829	parr	
4168	JUVENILE, 3.9 G	96H
4699	YEARLING, 10-18 MO	96H
4709	JUVENILE, 19.0 G	96H
4741	YEARLING, 10-18 MO	96H
4955	FY, 2.36-3.01 G	96H
5623	FINGERLING	96H
9784	FINGERLING	96H

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Table 2.6.2.2.12.2 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 5-18° Celsius**	**Arithmetic Mean 1642**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 5-350 mg/L $CaCO_3$**	**Geometric Mean 1020**
Endpoint/Effect Mortality	**pH 4.7-8.3**	**Harmonic Mean 173**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
11	EGG	18M
320	FINGERLING, 2 G	21M
320	NR	27M
680	ADULT, 66.3 G	120H
695	ADULT, 66.3 G	131H
724	4 WK, LARVAE, SWIM-UP	56D
724	4 WK LARVAE, SWIM-UP	56D
724	EGG	84D
1368	4 WK LARVAE, SWIM-UP	56D
1368	4 WK, LARVAE, SWIM-UP	56D
1368	NEWLY HATCHED LARVAE	84D
1368	EGG	84D
2058	NEWLY HATCHED LARVAE	84D
2476	JUVENILE, 0.316 G	114H
2818	JUVENILE, 0.316 G	117H
3004	JUVENILE, 0.316 G	156H
3077	JUVENILE, 0.316 G	141H
3090	JUVENILE, 0.316 G	141H
5000	JUVENILE, 0.316 G	120H

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Table 2.6.2.2.12.3 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 3-20° Celsius**	**Arithmetic Mean 193**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 20-374 mg/L $CaCO_3$**	**Geometric Mean 174**
Endpoint/Effect Growth	**pH 4.7-8.64**	**Harmonic Mean 161**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
104	NR	4D
104	NR	85D
104	NR	85D
104	NR	40W
104	NR	40W
132	NR	180D
132	NR	191D
132	NR	50D
132	NR	40W
172	NR	191D
172	NR	191D
172	NR	180D
172	NR	30D
172	NR	30D
172	NR	40W
172	NR	40W
172	NR	40W
172	NR	21M
172	NR	13W
172	NR	2M
172	NR	13W
358	45 G, YEARLING	13W
384	NR	30D
384	NR	40W
384	NR	1H
384	NR	55D

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Table 2.6.2.2.12.4 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 5-18° Celsius**	**Arithmetic Mean 615**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 20-374 mg/L $CaCO_3$**	**Geometric Mean 436**
Endpoint/Effect NOEC/Mortality/Reproduction	**pH 4.7-8.3**	**Harmonic Mean 277**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
98	NR	1M
108	NR	27M
380	EGG	18M
432	JUVENILE	NR
595	ADULT-SMOLT	NR
862	ADULT-SMOLT	NR
1028	YEARLING, 70 G, 3RD GENERATION	82D
1417	EGG	72D

Table 2.6.2.2.12.5 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 3-20° Celsius**	**Arithmetic Mean 38541**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 45-374 mg/L $CaCO_3$**	**Geometric Mean 3075**
Endpoint/Effect Cellular	**pH 4.7-8.64**	**Harmonic Mean 235**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
91	6-18 MO	3.15H
166	45 G, YEARLING	96H
76954	8-12 G, 9-11 CM	0.5H
76954	8-12 G, 9-11 CM	4H

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Table 2.6.2.2.12.6 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		Data Set ECOTOX Hardness=100
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 3-20° Celsius**	**Arithmetic Mean 2753**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 22-90 mg/L $CaCO_3$**	**Geometric Mean 2427**
Endpoint/Effect Physiological	**pH 4.7-8.64**	**Harmonic Mean 2199**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1360	YEARLING, 70 G, 3RD GENERATION	96H
1370	4 WK, LARVAE, SWIM-UP	4M
1370	EGG	1H
1370	4 WK LARVAE, SWIM-UP	1H
1984	NR	30D
2025	14.4 CM	17H
2074	14.4 CM	16W
2387	13.5 CM	2H
2588	NEWLY HATCHED LARVAE	4H
2588	4 WK, LARVAE, SWIM-UP	4H
2588	EGG	3.15H
2729	13.5 CM	43MIN
3212	14.4 CM	72H
3528	14.4 CM	2H
4857	13.5 CM	6H
8020	NR	30D

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Table 2.6.2.2.12.7 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for freshwater zinc.

Criterion Freshwater Zinc		**Data Set ECOTOX Hardness=100**
Criterion Concentration Acute 120 Micrograms Liter^{-1}	**Temperature 3-20° Celsius**	**Arithmetic Mean 224**
Criterion Concentration Chronic 120 Micrograms Liter^{-1}	**Hardness 30-350 mg/L $CaCO_3$**	**Geometric Mean 147**
Endpoint/Effect Reproductive	**pH 4.7-8.64**	**Harmonic Mean 84**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
30	NR	0.67H
108	NR	0.67H
379	8.3 CM	21M
379	FINGERLING, 2 G	10D

Zinc Toxicity Data Summary. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50

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percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

To assess the potential magnitude of acute toxic effects from exposure to zinc, NMFS added an additional step to its analysis for zinc to look at the relationship of the acute criterion to the LC_{50} data in terms of predicting the magnitude of acute toxic effects. To do this, NMFS calculated an acute toxicity ratio or relative percent mortality. This assessment involved taking the acute criterion of 120 µg/L and dividing it by each LC_{50} concentrations in Table 2.6.2.2.12.1 to calculate a ratio, *i.e.*, a prediction of the relative percent mortality of the criterion to the acute toxicity data. This ratio, relative to the LC_{50} data set in Table 2.6.2.2.12.1, predicts a magnitude of effect ranging from a low of an $LC_{0.6}$ at a concentration of 9,784 µg/L to a high of an $LC_{25.2}$ at a concentration of 238 µg/L. In other words, the acute criterion of 120 µg/L has an equivalent toxicity potential predicted to kill 0.6 percent to 25.2 percent, with a median toxicity potential of an $LC_{5.1}$, of the exposed test population, and therefore by inference, field-exposed individuals.

In summary, a number of toxicity studies reported concentrations that are less than the acute and chronic criteria concentrations for zinc, which implies that listed species exposed to waters equal to criteria concentrations will not be protected from acute or chronic toxic effects. Conversely, a number of toxicity studies reported concentrations that are greater than the acute and chronic criteria concentrations for zinc, which implies that listed species exposed to waters equal to criteria concentrations will be protected from acute or chronic toxic effects. When the available information is equivocal, NMFS gives the benefit of the doubt in its analysis to the listed species. Based on this principle and the considerations of the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the ecological consequences for field-exposed fishes, listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects.

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Sublethal Effects. Zinc is an essential element required for healthy fish, and is present in healthy fish tissues in greater concentrations than other heavy metals. However, increased levels of zinc over natural body concentrations can result in mortality, growth retardation, histopathological alterations, respiratory and cardiac changes, and inhibition of spawning and many other elements critical to fish survival. Exposure to high zinc concentrations can result in damage to the gills, liver, kidney and skeletal muscle and cause a physiological shift to occur, making gas exchange more difficult. Toxicity varies with hardness, pH, alkalinity, dissolved oxygen, water temperature, species and life stage, acclimation, and ambient concentrations of other chemicals in the water (EPA 1987c, Sorensen 1991, Eisler 1993). For example, the toxicity of zinc is influenced by antagonistic interactions with cadmium, copper, iron, and molybdenum (Hammond and Beliles 1980). There is evidence that fish acclimated to elevated temperature are more tolerant of zinc toxicity (Hodson and Sprague 1975).

Behavioral avoidance reactions have been noted in three trout species at zinc concentrations that were below the proposed chronic criterion. Juvenile rainbow trout avoidance was documented at zinc concentrations of 5.6 µg/L at a hardness of 13 mg/L (Sprague 1968) and 47 µg/L at a hardness of 112 mg/L (Birge and Black 1980 as cited in EPA 1987c). Juvenile brown trout avoidance was documented at 25 µg/L at a hardness of 100 mg/L (Woodward *et al.* 1995). Juvenile cutthroat trout avoidance was documented at 28 µg/L at a hardness of 50 mg/L (Woodward *et al.* 1997). Avoidance behavior by adult salmonids has not been studied as extensively. As with copper, there are insufficient data available to identify whether these behavioral effects translate into adverse effects in the field because of the confounding influence of acclimation, complexing organic material in natural waters, uncontrolled variables, presence of other metals, and field observations that found fish in "impacted" streams when "un-impacted" streams were also available.

Zinc bioconcentrates but does not biomagnify (EPA 1999). Zinc may be mutagenic and teratogenic (Eisler 1993). Toxicity of zinc to aquatic organisms is dependent on water hardness, pH, DO, presence of mixtures, and trophic level (Sorensen 1991, Eisler 1993). Zinc interacts with many chemicals to produce altered patterns of accumulation, metabolism, and toxicity; some interactions reduce toxicity and others increase toxicity (Eisler 1993). Most of the zinc introduced into aquatic environments is eventually partitioned into sediments (Eisler 1993). Zinc bioavailability from sediment is increased under conditions of high DO, low salinity, low pH, and high levels of inorganic oxides and humic substances. Effects of zinc toxicity to freshwater organisms include reduced growth, reduced populations, and reduced survival in algae species; reduced growth, activity, larval settlement, and reproduction, osmoregulatory impairment and reduced survival in freshwater invertebrates (including molluscs); and reduced growth, behavioral alteration (avoidance), reproduction impairment, increased respiration, decreased swimming ability, increased jaw and branchial abnormalities, hyperactivity, hyperglycemia, and reduced survival in freshwater fish (Eisler 1993).

In Farag *et al.* (1994), they determined that continuous exposure to zinc at the proposed chronic criterion concentration was associated with bioaccumulation of the metal by juvenile and adult rainbow trout. In Mount *et al.* (1994), they determined that tissue concentrations increased in rainbow trout fry fed a diet containing enriched levels of zinc. However, the issue of zinc bioaccumulation in salmonids is confounded by naturally high tissue concentrations and the

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ability of fish to regulate internal concentrations. In Alsop *et al.* (1999), they determined that tissue concentrations of zinc in fish exposed to approximately one to two times the acute criterion were not a good indicator of non-lethal, chronic zinc exposure. Physiological costs related to zinc acclimation were determined to be few. The work by Mount *et al.* (1994) did not detect significant effects on survival or growth in rainbow trout fry fed quantities of zinc that were 10 times or greater in concentration than other metals. These studies suggest collectively that the ability of salmonids to regulate internal zinc concentrations may minimize adverse effects of bioaccumulation when the fish are exposed to zinc concentrations near the proposed chronic criterion.

Sublethal Effects Summary. The available evidence indicates that the chronic criterion for zinc is likely to result in sublethal effects to listed species considered in this opinion.

Toxicity to Food Organisms. Many freshwater insects and crustaceans appear to be tolerant of zinc concentrations that are similar to the acute criterion (Eisler 1993), although some taxa can be more sensitive to chronic effects than salmonids (Kemble *et al.* 1994). Aquatic invertebrates bioaccumulate zinc to a greater degree than salmonids (EPA 1987c, Eisler 1993). Kiffney and Clements (1994) determined that mayflies were sensitive to zinc, and that the response varied with stream size or location in the stream network. Data in EPA (1987c) indicate that the zinc criteria are usually non-lethal to invertebrates that juvenile listed species feed on, although in two cases in EPA (1987c), cladoceran species exhibited LC_{50}s that were lower than the acute and chronic criteria at a hardness of 45 mg/L. Invertebrate communities in rivers appear to respond to elevated zinc levels in the sediments by changing composition to pollution-tolerant taxa, rather than by reducing overall biomass (Canfield *et al.* 1994, Clements and Kiffney 1994). It is not clear if this adversely affects foraging ability of juvenile salmon.

Zinc contained in bed sediments has been found to be elevated in benthic invertebrates in field studies conducted in metals-contaminated streams (Ingersoll *et al.* 1994; Woodward *et al.* 1994). However, Kiffney and Clements (1996) determined an inverse relation existed between aquatic macroinvertebrate body size and survival at zinc levels in excess of the proposed chronic criterion, which partially counters the effects of bioaccumulation, as organisms die before they are large enough to bioaccumulate high concentrations of zinc. Indirect effects of elevated zinc levels to listed species include reductions in production of larger bodied invertebrate taxa that could influence the availability of food for larger juvenile salmonids, and ingestion of bioconcentrated zinc by fry and juveniles of all sizes.

Summary on Toxicity to Food Organisms. The available evidence indicates that the chronic criterion for zinc is likely to appreciably affect invertebrate productivity and abundance.

Summary of Effects: Zinc. The available evidence for zinc indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (moderately-high-intensity), reduced growth (moderately-high-intensity), cellular trauma (moderate intensity), physiological trauma (moderate intensity), and reproductive failure (moderately-high-intensity).

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2.6.3 Saltwater Criteria Toxicity Analysis

The ESA directs that section 7 consultations use the best available scientific and commercial data. While EPA conducted an extensive data call and has developed a large database of toxicity (ECOTOX), thousands of toxicity studies were rejected by EPA for use in criteria development and formulation of the BE. A majority of these toxicity studies were rejected because the test duration was non-standard; EPA generally does not consider toxicity tests with non-standard durations (*e.g.*, 4-hr LC_{50} or 144-hr LC_{50}). However, these studies mat still meet the standard of the "best available scientific data" as defined by the ESA. For this consultation, NMFS used a much more extensive toxicity data set, including toxicity studies from the ECOTOX database that were excluded by EPA, for its analysis.

The analysis on saltwater criteria starts with a review of the chemical and toxicological concepts, principals, and factors that influence toxicity for each compound, and an assessment of critical exposure-response factors pertinent to the overall analysis. The data analysis in this section has four general components: (1) Available toxicity data presented in table format by endpoint; (2) a summary statistical analysis performed for each endpoint data set consisting of the arithmetic mean, the geometric mean, and the harmonic mean to assess the distribution of the data for each data set, and the statistical analysis is used later in the analysis on chemical mixtures; (3) a sublethal effects analysis on the chronic criteria, and (4) an analysis on food items (when data was available). Due to the paucity of acute saltwater data, NMFS did nor calculate a relative percent mortality for each acute saltwater criterion.

The toxicity data for salmonid fishes includes data for listed and non-listed salmonid fishes, *e.g.*, rainbow trout are used to directly assess toxicity effects on steelhead as the resident form is indistinguishable from the anadromous form in juvenile life stages. Other salmonid fishes, *e.g.*, brook trout (*Salvelinus fontinalis*) and cutthroat trout (*Oncorhynchus clarki*), are used in addition to the species-specific toxicity data and/or as a surrogate for listed species where toxicity data is not available for listed species to analyze effects on additional endpoints. Our analysis of surrogate species toxicity data showed no difference in the range of concentrations when compared to the toxicity data for listed species. Furthermore, toxicity data for green sturgeon and Eulachon was limited or non-existent for most of the compounds in Table 1.1. Therefore, NMFS used the salmonid fishes toxicity data as a surrogate for these two species as these toxicity data sets for salmonid fishes were the closest taxonomic data available. The summary conclusions provided in this section are based on a toxicity exposure-response potential to listed species considered in this opinion for each freshwater compound listed in Table 1.1, based exclusively on an examination of the available toxicity data from exposure to a single compound. The summary conclusions do not take into account effects to listed species considered in this opinion from exposure to multiple compounds. The issue of chemical mixtures, as well as criteria development and implementation issues, direct mortality population modeling, *etc.*, are examined in the *Integration and Synthesis*.

2.6.3.1 Arsenic

Saltwater Arsenic Criteria. The proposed acute and chronic criteria for saltwater arsenic are 69 µg/L and 36 µg/L, respectively.

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Tables 2.6.3.1.1 and 2.6.3.1.2 report toxicity data from the ECOTOX database for saltwater arsenic, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.1.1 Mortality toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater arsenic.

Criterion Saltwater Arsenic		**Data Set BE**
Criterion Concentration Acute 69 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 6658**
Criterion Concentration Chronic 36 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 6658**
Endpoint/Effect Mortality	**pH NR**	**Harmonic Mean 6658**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
6658	NR	NR

Table 2.6.3.1.2. NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater arsenic.

Criterion Saltwater Arsenic		**Data Set BE**
Criterion Concentration Acute 69 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 3974**
Criterion Concentration Chronic 36 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 3974**
Endpoint/Effect NOEC	**pH NR**	**Harmonic Mean 3974**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3974	NR	NR

Summary of Effects: Arsenic. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

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The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater arsenic indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and sublethal effects (moderate intensity).

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2.6.3.2 Cadmium

Cadmium Criteria. The proposed acute and chronic criteria for saltwater cadmium are 40 µg/L and 8.8 µg/L, respectively.

Tables 2.6.3.2.1 through 2.6.3.2.3 report toxicity data from the ECOTOX database for saltwater cadmium, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.2.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater cadmium.

Criterion Saltwater Cadmium		**Data Set ECOTOX**
Criterion Concentration Acute 40 Micrograms Liter^{-1}	**Temperature 11.2° Celsius**	**Arithmetic Mean 1200**
Criterion Concentration Chronic 8.8 Micrograms Liter^{-1}	**Salinity 28.3 ppt**	**Geometric Mean 1200**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 1200**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1200	SMOLTS, 128 MM	96H

Table 2.6.3.2.2 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater cadmium.

Criterion Saltwater Cadmium		**Data Set ECOTOX**
Criterion Concentration Acute 40 Micrograms Liter^{-1}	**Temperature 11.2° Celsius**	**Arithmetic Mean 1200**
Criterion Concentration Chronic 8.8 Micrograms Liter^{-1}	**Salinity 28.3 ppt**	**Geometric Mean 1200**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 1200**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1200	SMOLTS, 128 MM	96H

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Table 2.6.3.2.3 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater cadmium.

Criterion Saltwater Cadmium		**Data Set BE**
Criterion Concentration Acute 40 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 163.7**
Criterion Concentration Chronic 8.8 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 163.7**
Endpoint/Effect NOEC	**pH NR**	**Harmonic Mean 163.7**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
163.7	Smolts	

Summary of Effects: Cadmium. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

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The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for cadmium indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and sublethal effects (moderate intensity).

2.6.3.3 Chromium VI

CR (VI) Criteria. The proposed acute and chronic criteria for chromium (VI) are 1100 µg/L and 50 µg/L, respectively.

Tables 2.6.3.3.1 through 2.6.3.3.4 report toxicity data from the ECOTOX database for saltwater chromium (VI), except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.3.3.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater chromium VI.

Criterion Saltwater Chromium VI		**Data Set ECOTOX**
Criterion Concentration Acute 1100 Micrograms Liter^{-1}	**Temperature 3.5-19° Celsius**	**Arithmetic Mean 98129**
Criterion Concentration Chronic 50 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 68333**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 44884**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
12079	NR	96H
27201	NR	96H
27496	NR	96H
37905	NR	96H
69722	NR	96H
74239	NR	96H
98200	NR	96H
109002	NR	96H
141408	NR	96H
201310	NR	96H
280852	NR	96H

Table 2.6.3.3.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater chromium VI.

Criterion Saltwater Chromium VI		**Data Set ECOTOX**
Criterion Concentration Acute 1100 Micrograms Liter^{-1}	**Temperature 3.5-19° Celsius**	**Arithmetic Mean 91**
Criterion Concentration Chronic 50 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 47**
Endpoint/Effect Growth	**pH NR**	**Harmonic Mean 24**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
10	NR	7M
13	NR	110D
49	NR	
192	NR	
192	NR	

Summary of Effects: Chromium VI. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

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than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater chromium (VI) indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and sublethal effects (moderately-high-intensity).

2.6.3.4 Copper

Copper Criteria. The proposed acute and chronic criteria for saltwater copper are 4.8 µg/L and 3.1 µg/L, respectively.

Tables 2.6.3.4.1 through 2.6.3.4.3 report toxicity data from the ECOTOX database for saltwater copper, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.4.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater copper.

Criterion **Saltwater Copper**		**Data Set** **ECOTOX**
Criterion Concentration Acute **4.8 Micrograms Liter^{-1}**	**Temperature** **13° Celsius**	**Arithmetic Mean** **329**
Criterion Concentration Chronic **3.1 Micrograms Liter^{-1}**	**Salinity** **28.6 ppt**	**Geometric Mean** **329**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **8.1**	**Harmonic Mean** **329**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
329	SMOLTS, 132 MM	96H

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Table 2.6.3.4.2 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater copper.

Criterion Saltwater Copper		Data Set ECOTOX
Criterion Concentration Acute 4.8 Micrograms Liter^{-1}	**Temperature 10.3-13Celsius**	**Arithmetic Mean 329**
Criterion Concentration Chronic 3.1 Micrograms Liter^{-1}	**Salinity 12-35 ppt**	**Geometric Mean 329**
Endpoint/Effect LC_{50}/Mortality	**pH 7.8-8.1**	**Harmonic Mean 329**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
329	SMOLT, 132 MM	96H
329	SMOLTS, 132 MM	96H

Table 2.6.3.4.3 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater copper.

Criterion Saltwater Copper		Data Set ECOTOX
Criterion Concentration Acute 4.8 Micrograms Liter^{-1}	**Temperature 10.3-13Celsius**	**Arithmetic Mean 31**
Criterion Concentration Chronic 3.1 Micrograms Liter^{-1}	**Salinity 12-35 ppt**	**Geometric Mean 31**
Endpoint/Effect Reproductive	**pH 7.8-8.1**	**Harmonic Mean 31**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
31	Gamete	60MIN

Summary of Effects: Copper. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity

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tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater copper indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and reproductive failure (moderate intensity).

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2.6.3.5 Endosulfan (Endosulfan-alpha and Endosulfan-beta)

Endosulfan-a and Endosulfan-b Criteria. The proposed acute and chronic criteria for saltwater endosulfan-a and endosulfan-b are 0.034 µg/L and 0.0087 µg/L, respectively.

Tables 2.6.3.5.1 and 2.6.3.5.2 report toxicity data from the ECOTOX database for saltwater endosulfan, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.5.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater endosulfan-alpha and endosulfan-beta.

Criterion **Saltwater Endosulfan-alpha and Endosulfan-beta**		**Data Set** **ECOTOX**
Criterion Concentration Acute **0.034 Micrograms Liter^{-1}**	**Temperature** **11.4° Celsius**	**Arithmetic Mean** **1.7**
Criterion Concentration Chronic **0.0087 Micrograms Liter^{-1}**	**Salinity** **NR**	**Geometric Mean** **1.7**
Endpoint/Effect **LC_{50}**	**pH** **8.1**	**Harmonic Mean** **1.7**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
1.69	SMOLT, 127 MM	96H

Table 2.6.3.5.2 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater endosulfan-alpha and endosulfan-beta.

Criterion **Saltwater Endosulfan-alpha and Endosulfan-beta**		**Data Set** **ECOTOX**
Criterion Concentration Acute **0.034 Micrograms Liter^{-1}**	**Temperature** **11.4-12° Celsius**	**Arithmetic Mean** **765.5**
Criterion Concentration Chronic **0.0087 Micrograms Liter^{-1}**	**Salinity** **NR**	**Geometric Mean** **765.5**
Endpoint/Effect **Reproductive**	**pH** **7.8-8.2**	**Harmonic Mean** **765.5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
765.5	GAMETE	60MIN

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Summary of Effects: Endosulfan-a and Endosulfan-b. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

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than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater endosulfan-alpha and endosulfan-beta indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity) and reproductive failure (low intensity).

2.6.3.6 Heptachlor Epoxide

Heptachlor Epoxide Criteria. The proposed acute and chronic criteria for saltwater heptachlor epoxide are 0.053 µg/L and 0.0036 µg/L, respectively.

Tables 2.6.3.6.1 and 2.6.3.6.2 report toxicity data from the ECOTOX database for saltwater heptachlor epoxide, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.6.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater heptachlor epoxide.

Criterion **Saltwater Heptachlor**		**Data Set BE**
Criterion Concentration Acute **0.053 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **0.37**
Criterion Concentration Chronic **0.0036 Micrograms Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **0.37**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **NR**	**Harmonic Mean** **0.37**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.367		

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Table 2.6.3.6.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater heptachlor epoxide.

Criterion Saltwater Heptachlor		Data Set BE
Criterion Concentration Acute **0.053 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **0.2**
Criterion Concentration Chronic **0.0036 Micrograms Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **0.2**
Endpoint/Effect **NOEC**	**pH** **NR**	**Harmonic Mean** **0.2**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
0.214		

Summary of Effects: Heptachlor Epoxide. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

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The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater heptachlor epoxide indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity) and sublethal effects (low intensity).

2.6.3.7 Lead

Lead Criteria. The proposed acute and chronic criteria for lead are 210 µg/L and 8.1 µg/L, respectively.

Tables 2.6.3.7.1 through 2.6.3.7.3 report toxicity data from the ECOTOX database for saltwater lead, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.3.7.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater lead.

Criterion Saltwater Lead		Data Set BE
Criterion Concentration Acute **210 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **805**
Criterion Concentration Chronic **8.1 Micrograms Liter^{-1}**	**Hardness** **NR**	**Geometric Mean** **805**
Endpoint/Effect **LC_{50}/Mortality**	**pH** **NR**	**Harmonic Mean** **805**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
805		

Table 2.6.3.7.2 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater lead.

Criterion Saltwater Lead		Data Set ECOTOX
Criterion Concentration Acute **210 Micrograms Liter^{-1}**	**Temperature** **12-13.7° Celsius**	**Arithmetic Mean** **150**
Criterion Concentration Chronic **8.1 Micrograms Liter^{-1}**	**Salinity** **27-30 ppt**	**Geometric Mean** **150**
Endpoint/Effect **Physiological**	**pH** **7.8-8.2**	**Harmonic Mean** **150**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
150	200 G, SALTWATER ADAPTED	2W

Table 2.6.3.7.3 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater lead.

Criterion Saltwater Lead		Data Set ECOTOX
Criterion Concentration Acute **210 Micrograms Liter^{-1}**	**Temperature** **12-13.7° Celsius**	**Arithmetic Mean** **24000**
Criterion Concentration Chronic **8.1 Micrograms Liter^{-1}**	**Salinity** **27-30 ppt**	**Geometric Mean** **24000**
Endpoint/Effect **Reproductive**	**pH** **7.8-8.2**	**Harmonic Mean** **24000**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
24000	GAMETE	2W

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Summary of Effects: Lead. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less

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than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater lead indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (moderate intensity), physiological trauma (moderate intensity), and reproductive failure (low intensity).

2.6.3.8 Nickel

Nickel Criteria. The proposed acute and chronic criteria for saltwater nickel are 74 µg/L and 8.2 µg/L, respectively.

Tables 2.6.3.8.1 and 2.6.3.8.2 report toxicity data from the ECOTOX database for saltwater nickel, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.8.1. LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater nickel.

Criterion **Saltwater Nickel**		**Data Set BE**
Criterion Concentration Acute **74 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **4893**
Criterion Concentration Chronic **8.2 Micrograms Liter^{-1}**	**Salinity** **NR**	**Geometric Mean** **4893**
Endpoint/Effect **LC_{50}**	**pH** **NR**	**Harmonic Mean** **4893**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
4893		

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Table 2.6.3.8.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater nickel.

Criterion Saltwater Nickel		Data Set BE
Criterion Concentration Acute **74 Micrograms Liter^{-1}**	**Temperature** **NR**	**Arithmetic Mean** **1793**
Criterion Concentration Chronic **8.2 Micrograms Liter^{-1}**	**Salinity** **NR**	**Geometric Mean** **1793**
Endpoint/Effect **NOEC**	**pH** **NR**	**Harmonic Mean** **1793**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
1793		

Summary of Effects: Nickel. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

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The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater nickel indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity) and sublethal effects (low intensity).

2.6.3.9 Pentachlorophenol

Pentachlorophenol Criteria. The proposed chronic criterion for saltwater PCP is 7.9 µg/L, respectively.

Table 2.6.3.9.1 reports toxicity data from the ECOTOX database for saltwater pentachlorophenol, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

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Table 2.6.3.9.1 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater pentachlorophenol.

Criterion **Saltwater Pentachlorophenol**		**Data Set BE**
	Temperature **NR**	**Arithmetic Mean** **10.5**
Criterion Concentration Chronic **7.9 Micrograms Liter^{-1}**	**Salinity** **NR**	**Geometric Mean** **10.5**
Endpoint/Effect **NOEC**	**pH** **NR**	**Harmonic Mean** **10.5**
Concentration **Micrograms Liter^{-1}**	**Life-Stage**	**Duration**
10.5		

Summary of Effects: Pentachlorophenol. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater PCP indicates that listed species exposed to waters equal to the chronic criterion concentrations will suffer chronic toxic effects including sublethal effects (moderately-high-intensity).

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2.6.3.10 Selenium

Selenium Criteria. The proposed acute and chronic criteria for saltwater selenium are 290 µg/L and 71 µg/L, respectively.

Tables 2.6.3.10.1 and 2.6.3.10.2 report toxicity data from the ECOTOX database for saltwater selenium, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.10.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater selenium.

Criterion Saltwater Selenium		**Data Set ECOTOX**
Criterion Concentration Acute 290 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 76750**
Criterion Concentration Chronic 71 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 43547**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 30929**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
11600	FRY, 2.6 G	96H
11600	FRY, 2.6 G	96H
16600	1.6 G, FRY	96H
16600	1.6 G, FRY	96H
17200	1.6 G, FRY	96H
17200	1.6 G, FRY	96H
18300	FRY, 2.6 G	96H
18300	FRY, 2.6 G	96H
19600	FRY, 2.6 G	96H
19600	FRY, 2.6 G	96H
23900	FRY, 2.6 G	96H
23900	FRY, 2.6 G	96H
28200	FRY, 2.6 G	96H
28200	FRY, 2.6 G	96H
29000	FRY, 1.7 G	96H
29000	FRY, 1.7 G	96H
36100	FRY, 2.6 G	24H
39600	1.6 G, FRY	24H
43200	FRY, 2.4 G	96H
43200	FRY, 2.4 G	96H

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Criterion Saltwater Selenium		Data Set ECOTOX
Criterion Concentration Acute 290 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 76750**
Criterion Concentration Chronic 71 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 43547**
Endpoint/Effect LC_{50}	**pH NR**	**Harmonic Mean 30929**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
50100	FRY, 2.4 G	96H
50100	FRY, 2.4 G	96H
63800	1.6 G, FRY	24H
65400	FRY, 2.4 G	96H
65400	FRY, 2.4 G	96H
79400	FRY, 1.8 G	96H
79400	FRY, 1.8 G	96H
94000	FRY, 1.6 G	96H
94000	FRY, 1.6 G	96H
136000	FRY, 1.6 G	96H
136000	FRY, 1.6 G	96H
236000	FRY, 1.6 G	24H
369000	FRY, 1.7 G	24H
600000	FRY, 1.6 G	24H

Table 2.6.3.10.2 NOEC toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater selenium.

Criterion Saltwater Selenium		Data Set BE
Criterion Concentration Acute 290 Micrograms Liter^{-1}	**Temperature NR**	**Arithmetic Mean 5551**
Criterion Concentration Chronic 71 Micrograms Liter^{-1}	**Salinity NR**	**Geometric Mean 5048**
Endpoint/Effect NOEC	**pH NR**	**Harmonic Mean 4591**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3243		
7859		

Summary of Effects: Selenium. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species

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exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

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In summary, the available evidence for saltwater selenium indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity) and sublethal effects (low intensity).

2.6.3.11 Silver

Silver Criteria. The proposed acute criterion for saltwater silver is 1.9 µg/L.

Tables 2.6.3.11.1 reports toxicity data from the ECOTOX database for saltwater silver, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.11.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater silver.

Criterion Saltwater Silver		**Data Set ECOTOX**
Criterion Concentration Acute 1.9 Micrograms Liter^{-1}	**Temperature 11.5-14° Celsius**	**Arithmetic Mean 195**
	Salinity 25-28.6 ppt	**Geometric Mean 194**
Endpoint/Effect LC_{50}	**pH 7.8-8.2**	**Harmonic Mean 193**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
176	25 G	96H
214	SMOLT, 131 MM	96H

Summary of Effects: Silver. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some

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compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

In summary, the available evidence for saltwater silver indicates that listed species exposed to waters equal to the acute criterion concentrations will suffer chronic toxic effects including sublethal effects (low intensity).

2.6.3.12 Tributyltin

Tributyltin Criteria. The proposed acute and chronic criteria for saltwater TBT are 0.37 µg/L and 0.01 µg/L, respectively.

Tables 2.6.3.12.1 through 2.6.3.12.4 report toxicity data from the ECOTOX database for saltwater tributyltin, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.12.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater tributyltin.

Criterion Saltwater Tributyltin		**Data Set ECOTOX**
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 12**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 6.7**
Endpoint/Effect LC_{50}/Mortality	**pH 6.4-7.8**	**Harmonic Mean 3.6**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
1.02	1.47 G	96H
1.16	1.47 G	96H
1.34	1.47 G	96H
1.46	24.5 G, 25.1 CM FORK LENGTH	96H

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Criterion Saltwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 12**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 6.7**
Endpoint/Effect LC_{50}/Mortality	**pH 6.4-7.8**	**Harmonic Mean 3.6**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
4.6	0.77 g	96H
4.84	5.94 G	96H
5.5	1.4 g	96H
6.2	0.68(0.17-1.2) G, 45(39-53) MM	96H
6.6	0.68(0.17-1.2) G, 45(39-53) MM	72H
7.9	0.68(0.17-1.2) G, 45(39-53) MM	48H
11	JUVENILE	96H
11	JUVENILE	96H
15	0.68(0.17-1.2) G, 45(39-53) MM	24H
20	24.5 G, 25.1 CM FORK LENGTH	12H
21	UNDER-YEARLING	48H
28	UNDER-YEARLING	24H
54	24.5 G, 25.1 CM FORK LENGTH	6H

Table 2.6.3.12.2 Growth toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater tributyltin.

Criterion Saltwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 0.52**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 0.52**
Endpoint/Effect Growth	**pH 6.4-7.8**	**Harmonic Mean 0.52**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.5	3 WK	21D
0.54	8.3-8.8 CM, 5.6-6.4 G	10D

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Table 2.6.3.12.3 Cellular toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater tributyltin.

Criterion Saltwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 0.58**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 0.58**
Endpoint/Effect Cellular	**pH 6.4-7.8**	**Harmonic Mean 0.58**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
0.5	3 WK	7D
0.6	NR	28D
0.6	NR	28D
0.6	NR	28D
0.6	4-24 MO, 8.5-20.7 CM, 6.0-94.5 G	10D

Table 2.6.3.12.4 Physiological toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater tributyltin.

Criterion Saltwater Tributyltin		Data Set ECOTOX
Criterion Concentration Acute 0.37 Micrograms Liter^{-1}	**Temperature 10-18° Celsius**	**Arithmetic Mean 27**
Criterion Concentration Chronic 0.01 Micrograms Liter^{-1}	**Salinity 28 ppt**	**Geometric Mean 13**
Endpoint/Effect Physiological	**pH 6.4-7.8**	**Harmonic Mean 6.5**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
3.5	8.8 CM, 6.4 G	28D
50	NR	65MIN

Summary of Effects: Tributyltin. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range

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between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

In summary, the available evidence for saltwater tributyltin indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity), sublethal effects (low intensity), physiological trauma (low intensity), and cellular trauma (low intensity).

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2.6.3.13 Zinc

Zinc Criteria. The proposed acute and chronic criteria for saltwater zinc are 90 µg/L and 81 µg/L.

Tables 2.6.3.13.1 through 2.6.3.13.2 report toxicity data from the ECOTOX database for saltwater zinc, except where noted. Each table identifies the respective endpoint for the data set, toxicity test concentration, species life stage, test duration, toxicity-associated water quality parameters (when available), the arithmetic mean, the geometric mean, and the harmonic mean of each data set.

Table 2.6.3.13.1 LC_{50} toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater zinc.

Criterion Saltwater Zinc		**Data Set ECOTOX**
Criterion Concentration Acute 90 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 3000**
Criterion Concentration Chronic 81 Micrograms Liter^{-1}	**Salinity 27 ppt**	**Geometric Mean 2828**
Endpoint/Effect LC_{50}	**pH 7.8-8.2**	**Harmonic Mean 2667**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
2000	2 YR, PARR, 14.8 CM FL	48H
4000	YEARLING, 14.5 CM FL	48H

Table 2.6.3.13.2 Reproductive toxicity data for salmonid fishes, Eulachon, and green sturgeon for saltwater zinc.

Criterion Saltwater Zinc		**Data Set ECOTOX**
Criterion Concentration Acute 90 Micrograms Liter^{-1}	**Temperature 12° Celsius**	**Arithmetic Mean 819**
Criterion Concentration Chronic 81 Micrograms Liter^{-1}	**Salinity 27 ppt**	**Geometric Mean 819**
Endpoint/Effect Reproductive	**pH 7.8-8.2**	**Harmonic Mean 819**
Concentration Micrograms Liter^{-1}	**Life-Stage**	**Duration**
819	GAMETE	60MIN

Summary of Effects: Zinc. In order to understand the context of the toxicity data and its relationship to the criteria, NMFS not only considered the toxicity test concentrations in comparison to the criterion/criteria concentrations in determining whether or not listed species

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exposed to waters equal to criteria concentrations will result in acute or chronic toxic effects, but the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes.

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, which indicate the concentration at which 50 percent of the test population was killed. However, what is often not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). While the range of post-exposure effects identified in these studies is likely to be less than or greater than the 15 to 35 percent range depending on compound and species, these studies highlight the inherent flaws associated with acute toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes. Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic, ammonia (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias (underestimate) the magnitude of acute toxic effects. These factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that minimize acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that LC_{50} data that is above the acute criterion is protective against acute toxic effects based solely on a comparison of concentrations. Therefore, LC_{50} data that is above the acute criterion concentration does not necessarily ensure that there are no acute toxic effects, but that the criterion concentration is likely to result in acute toxic effects to a subset, *i.e.*, less than 50 percent, of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration.

The chronic criterion is based on toxicity tests, *e.g.*, a NOEC (NOECs are summary statistics, *i.e.*, hypothesis tests, and not actual data, Crane and Newman 2000) where the magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be greater than 30 percent on average for some endpoints, and much higher for individual tests (Skalski 1981, Moore and Caux 1997, Crane and Newman 2000, Landis *et al.* 2011). These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that minimize chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

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In summary, he available evidence for saltwater zinc indicates that listed species exposed to waters equal to the acute and chronic criteria concentrations will suffer acute or chronic toxic effects including mortality (low intensity) and reproductive failure (low intensity).

2.6.4 Chemical Mixtures

Where multiple toxic effluents are discharged to receiving water, the resultant ambient toxicity is of interest. Since each effluent is composed of individual toxic substances, a mixture of the effluents in receiving water produces a mixture of these individual pollutants. The overall ambient toxicity could be equal to the sum of each discharge's toxicity (additivity), less than the sum (antagonism), or greater than the sum (synergism). Although the technology does exist to conduct site-specific chemical mixtures analysis, neither the data nor the technical capabilities exist to conduct a chemical mixtures analysis for the compounds listed in Table 1.1 at the scale of this consultation. This is because there are more than 3,000 point source discharges in Oregon, and each discharge represents a unique mixture of pollutants that varies considerably seasonally or more frequently. Once in the receiving water bodies, these discharged pollutants mix with pollutants from non-point sources and natural sources, at rates that are influenced by changes in river discharges. The result is an almost unlimited number of combinations of pollutant types and concentrations that varies nearly continuously and makes a quantitative mixture analysis across the State of Oregon impracticable and unrealistic task. Nonetheless, the issue of chemical mixtures is an important line of evidence to consider when assessing the exposure-response effects and risks to the listed species considered in this opinion.

The concept of independent joint action (also commonly termed response addition) was formalized by Loewe and Muischnek (1926 as cited in EPA 2008) and is used to describe the toxicity of a mixture in which the chemical constituents elicit their effects independently via different mechanisms of action. The other commonly used method to assess mixture toxicity is termed concentration addition (Bliss 1939) and assumes a common mechanism of action. Rider and LeBlanc (2005) and Meyer *et al.* (2007) have integrated these models in a manner that allows assessment of mixture toxicity using both concentration addition and independent joint action in which the toxic response associated with each group of compounds that share a common mechanism of action is first calculated using the concentration addition approach. The combined toxic responses associated with all groups of compounds are then calculated by independent joint action to the yield the predicted effect for the entire mixture.

Norwood *et al.* (2003), in a review of the toxicity of metal mixtures to aquatic species derived from a database of information from 68 literature citations, and mixture effects on 77 species, observed that the commonly used concentration addition approaches accurately predicted metal mixture toxicity 27% of the time. Mixture toxicity was less than additive (*i.e.* the concentration response approach overpredicted mixture toxicity) 43% of the time. The remaining 29% of the mixtures were more than additive (*i.e.* the concentration response approach underestimated mixture toxicity). Norwood *et al.* (2003) attributed the underprediction of mixture toxicity largely to interactions between mixture components. The variability in the studies could be due to different mixtures of metals being used and that some metals may share a common mechanism of action while others may not.

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The available information in EPA's technical support document for water quality-based toxics control (EPA 1991) indicates that the combined effects of individual acutely toxic pollutants are 0.4 to 2.8 times the effects predicted by adding the individual effects. The median combined effect is approximately additive (EPA 1991). For this reason, EPA recommends in the absence of site-specific data that regulatory authorities consider combined acute toxicity to be additive. In relation to chronic toxicity, for the growth of fish, Alabaster and Lloyd (1965 as cited in EPA 1991) conclude the joint effect of toxicants has been consistently less than additive, which suggests that dose addition is not the appropriate model for that endpoint.

Although each method described above has its pros and cons, NMFS used a concentration addition analysis to assess whether or not the criteria exposed to multiple compounds under the proposed criteria pose a greater risk to listed species considered in this opinion than does exposure to individual compounds. Here the purpose was to predict the cumulative toxicity that is expected for the mixture. For example, if the assessment effect is 50 percent mortality (*i.e.* the assessment effect concentration, the denominator, is LC_{50}), a result of 1 predicts that the mixture would produce 50 percent mortality. A result of < 1 predicts that, based on additivity, the mortality would be less than 50 percent. A result of > 1 predicts more that 50 percent mortality. The concentration addition analysis is based on an assumption of a similar mechanism of action for each set of compounds, *e.g.*, metals or organics (includes ammonia even though it does not have a C-H bond). For the freshwater acute analysis NMFS used the LC_{50} data from Table 2.6.5.1.2. For the freshwater chronic, saltwater acute and chronic analysis, NMFS used the geometric mean of the respective data sets (Tables 2.6.2.1.5 through 2.6.3.13.2), or the BE if no chronic toxicity data (*i.e.*, ACR value) were available. The NMFS used the following equation in this analysis:

$$\sum_{i=1}^{n} \frac{C_i}{EC_{xi}}$$

where n = the number of compounds in the mixture, C_i = assessment exposure concentration (criterion) and EC_{xi} = assessment effects concentration (geometric mean of the criterion-specific toxicity data set).

Assumptions

This analysis is specific to the compounds listed in Table 1.1, assumes that the listed species considered in this opinion are exposed to the compounds in combination that follow concentration addition. For freshwater and saltwater metals, this scenario is highly likely based on the information in section 2.5.2.1 on compounds discharged in MS4 and NPDES permits (12 of 12 metals). For freshwater and saltwater organic compounds, this scenario is less likely based on the information in the environmental baseline (Section 2.5.2.1) on compounds discharged in MS4 and NPDES permits (1 of 8 organic compounds in freshwater and 1 of 4 in saltwater). The results of NMFS' concentration addition analysis are provided in Table 2.6.4.1.

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Table 2.6.4.1 Results of the concentration addition analysis.

Metal Compounds	Criteria	Mixture Prediction
Al, As, Cd, Cr (III), Cr (VI), Cu, Pb, Ni, Se, Ag, Tributyltin, Zn	Freshwater acute	1.2
Al, As, Cd, Cr (III), Cr (VI), Cu, Pb, Ni, Se, Ag, Tributyltin, Zn	Freshwater chronic	4.7
As, Cd, Cr (VI), Cu, Pb, Ni, Se, Ag, Tributyltin, Zn	Saltwater acute	0.4
As, Cd, Cr (VI), Cu, Pb, Ni, Se, Tributyltin, Zn	Saltwater chronic	1.4
Organic Compounds	**Criteria**	**Mixture Prediction**
Ammonia, Lindane, Dieldrin, Endosulfan-alpha, Endosulfan-beta, Endrin, Heptachlor expoxide, Pentachlorophenol	Freshwater acute	1.3
Ammonia, Dieldrin, Endosulfan-alpha, Endosulfan-beta, Endrin, Heptachlor expoxide, Pentachlorophenol	Freshwater chronic	0.8
Endosulfan-alpha, Endosulfan-beta, Heptachlor expoxide	Saltwater acute	0.2
Endosulfan-alpha, Endosulfan-beta, Heptachlor expoxide, Pentachlorophenol	Saltwater chronic	0.001

Summary: The results of the concentration addition analysis infer that for acute and chronic freshwater criteria for metal compounds, acute freshwater criteria for organic compounds, and chronic saltwater criteria for metal compounds, fish exposed to multiple compounds, versus a single compound exposure, are likely to suffer toxicity greater than the assessment effects (*e.g.*, 50 percent mortality) such as mortality, reduced growth, impairment of essential behaviors related to successful rearing and migration, cellular trauma, physiological trauma, and reproductive failure. For example, the toxicity of a mixture at the freshwater acute criterion is predicted to be equivalent to an exposure to a single compound at 1.2 times the compounds' LC_{50} (*e.g.*, an exposure to cadmium at 2.4 µg/L compared to the proposed criterion concentration of 2 µg/L). The mixture toxicity will be greater than 50 percent mortality, but quantifying this prediction is dependent upon knowing the concentration-response curve. On the other hand, the results of the concentration addition analysis infer that for chronic freshwater criteria for organic compounds, acute saltwater criteria for metal compounds, and for acute and chronic saltwater criteria for organic compounds, fish exposed to multiple compounds, versus a single compound criterion exposure, are unlikely to suffer toxicity greater than the assessment effect concentrations.

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2.6.5 Direct Mortality Population Modeling

To determine if population productivity would be at risk due to direct mortality resulting from either acute or chronic exposures to the criterion concentrations of the chemicals of concern, a series of modeling applications was undertaken. These assessed whether juvenile salmon during their freshwater residence encountering the established criterion concentrations would be impacted, and if those changes would be sufficient to produce a change in the population growth rate, *i.e.*, lambda (λ). Model Run I examined the potential lethal and sublethal effects of ammonia, cadmium and copper on salmon productivity. These compounds were chosen because they are more data rich for specific life stages of salmonids and could potentially parameterize population models assessing direct mortality and somatic growth. Specific details regarding model design and parameterization are described in detail in Appendix 3. Model Run II assessed direct mortality impacts on population productivity resulting from exposure to the acute criteria for compounds with limited data.

Model Run I uses the direct mortality population model to assess the impact of the acute and chronic freshwater criteria on population productivity using a taxa- and life stage-specific subset of the acute and chronic toxicity data for ammonia, copper, and cadmium, and uses data-specific calculated dose-response slopes for the toxicity model runs (Appendix 3). This included direct mortality from either acute or chronic exposures. The model applied a mortality factor to first-year survival of the respective life-history models to assess changes in λ.

Model Run II uses the direct mortality population model (Appendix 3) to assess the impact of the acute freshwater criteria on population productivity using the acute toxicity data (LC_{50}), and a default dose-response slope. To assess the impact of the acute freshwater criteria on population productivity, we used the direct mortality population models. To do this, the dose-response slope for each LC_{50} toxicity test is needed. The BE does not provide any dose-response information for the data used in the analysis. Many of toxicity studies we reviewed either did not report the slope or did not provide the information required to calculate the dose-response curve. Since the direct mortality population model requires an LC_{50} slope, we used a default slope (probit slope of 4.5 converted to a sigmoid slope of 3.6) as recommended by EPA:

> In the event that dose response information is not available to estimate a slope, a default slope assumption of 4.5 (lower and upper bounds of 2 to 9) (Urban and Cook 1986 as cited in EPA 2007) is used.

In the analysis for Model Run I and Model Run II we assess the potential for effects associated with chemical exposure during subyearling freshwater rearing on Pacific salmon and steelhead populations using quantitative methods; a direct mortality model linked to a life history population model and a somatic growth model linked to the life history population model. Both methods predict changes in the modeled population's intrinsic rate of growth, *i.e.*, λ. General life-history strategies were constructed and analyzed for coho salmon, sockeye salmon and ocean-type and stream-type Chinook salmon. The model assesses direct mortality to subyearling salmon and its impact on population productivity. Data was reviewed in an attempt to paramaterize a somatic growth population model that explicitly links impairments in the somatic growth of individual subyearling salmon to the productivity of salmon populations.

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Available data was insufficent to parameterize the somatic growth model. Both models address impacts on first-year survival, and the results are incorporated into one of four life-history strategies in the model to quantify changes in population productivity (for a detailed description, see Appendix 3).

Primary differences between the four modeled life-history strategies are life span of the female, time to reproductive maturity, the number and relative contribution of the reproductive age classes and general demographic rates (Appendix 3). The models depict general populations representing each life-history strategy and were constructed based upon literature data described in Appendix 3. Specific populations were not modeled due to the difficulty in finding sufficient demographic data for single populations. Due to similarities in life-history strategies, the ocean-type Chinook model was used to estimate impacts on chum salmon and the stream-type Chinook model to estimate impacts on steelhead.

The endpoint used to assess population-level impacts for the direct mortality population model was the percent change in the intrinsic population growth rate (lambda, λ) resulting from chemical exposure. Change in λ is an accepted population parameter often used in evaluating population productivity, status, and viability. The NMFS uses changes in λ when estimating the status of species, conducting risk and viability assessments, developing ESA recovery plans, composing opinions, and communicating with other Federal, state and local agencies (McClure *et al.* 2003 as cited in Appendix 3). While values of $\lambda < 1.0$ indicate a declining population, in cases when an exposure causes the population growth rate to decrease more than natural variability, a loss of productivity will result even if lambda remains above 1.0. Decreases in response to chemical exposures can be a cause for concern since the impact could make a population more susceptible to decline (*i.e.*, λ dropping below 1.0) due to impacts from other stressors.

2.6.5.1 Direct Mortality Population Model Description

A direct mortality population model was constructed that estimated the population-level impacts of first-year mortality resulting from exposure to the criterion concentrations of aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc (Model Run II). For Model Run II, impacts of first-year mortality resulting from exposure to the criterion concentrations of ammonia, copper, and cadmium over various time frames and life stages of data. These models excluded sublethal and indirect effects of the chemical exposures and focused on the population-level outcomes resulting from an annual exposure of young-of-the-year to a chemical at the criterion concentrations. Scenarios were chosen to represent both the acute and chronic criteria. This was done by parameterizing the model with toxicity data (LC_{50}s) derived from short term (<96 hrs) and long term (>28 days, based on the available data, see Table A3 in Appendix 3) experiments. The lethal impact was implemented as a change in first-year survival for each of the salmon life-history strategies. In order to understand the relative impacts of a short-term exposure of a single chemical on exposed vs. unexposed fish, we used parameters for an idealized control population that exhibits an increasing population growth rate. Four life-history strategies were modeled: ocean-type and stream-type Chinook salmon, coho salmon and sockeye salmon. The details for

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each general population model are provided in Appendix 3. Due to similarities in life-history strategies, the ocean-type Chinook model was used to estimate impacts on chum and the stream-type Chinook model to estimate impacts on steelhead.

Population model output consists of the percent change in λ from the unexposed control populations derived from the mean of one thousand calculations each of the unexposed control and the chemical exposed populations. The percent change in lambda (with standard deviation), representing alterations to the population productivity, was selected as the primary model output for reasons outlined previously. The percent change in lambda is considered different from the control when the difference is greater than the percent of one standard deviation of the control λ.

Model Run I: Direct mortality, somatic growth, and population modeling— ammonia, cadmium, and copper.

Model Toxicity Scenario Parameterization

Ammonia (acute criterion = 5.6 mg/L; chronic criterion = 1.7 mg/L): The documents identified by the first round of literature review applying to acute toxicity of ammonia to salmonids were further reviewed for data appropriate to parameterize the direct mortality population model. Data needed to conform to 96-hr LC50 values for subyearling salmonids (free-swimming, 1-4g fish preferred, but did include data on fish of less than 10 g when that was all that was available). The range of values identified for Chinook salmon, coho salmon, rainbow trout and cutthroat trout and are shown below in the units of mg NH_3-N/L, as N (total ammonia-nitrogen). All values were normalized to a pH of 8 using an un-ionized ammonia computer worksheet available from the American Fisheries Society, as cited in Appendix 3. Following the practice in the ammonia Ambient Water Quality Criteria documents (1999, 2009, all as cited in Appendix 1), the fish LC50 values were not normalized for temperature. The normalized species mean values were 26.8, 15.1, 26.2 and 29.4 mg NH_3-N/L for Chinook salmon, coho salmon, rainbow trout and cutthroat trout, respectively (Servizi and Gordon 1990; Buckley 1978; Thurston and Russo 1983; Thurston *et al.,* 1981, Table A3, all as cited in Appendix 3). The genus geometric mean from these data was 23.6 mg NH_3-N/L. A sigmoid dose-response slope was calculated as 6.4 (Broderius and Smith 1979; Buckley 1978, as cited in Appendix 3). Both the genus geometric means and minimum species mean values were used to parameterize the model as discussed above. To assess the chronic criterion, a chronic study was found that exposed cutthroat trout to ammonia for 29 days and reported an LC50 of 21.3 mg NH_3-N/L (Thurston *et al.,* 1978, as cited in Appendix 3). No slope was identified, so the 96-hr slope was used in the model.

Documents investigating the effects of ammonia on growth of fish were reviewed for data appropriate as input to the somatic growth model. No studies were found that could provide the appropriate data. Most studies on exposure of juvenile salmonids to ammonia found that any effects on growth or food intake were temporary and compensation occurred before the end of the exposure period (Lang *et al.*, 1987, Linton *et al.*, 1998, Beamish and Tandler 1990, Larmoyeux and Piper 1973 as cited in Appendix 3). Other studies have shown effects on growth, but exposure occurred over early developmental stages and also produced developmental delays and abnormalities, so differences in size may not have been attributable to direct impacts on

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metabolism or growth (Brinkman *et al.*. 2009 as cited in Appendix 3). From a 90-day exposure (Brinkman *et al.*. 2009 as cited in Appendix 3) calculated an EC_{20} that includes hatch effects, delayed swimup, and sac-fry growth of 5.56 mg NH_3-N/L normalized to pH 8. In addition, Lazorchak and Smith (2007 as cited in Appendix 1) reported decreases in growth of rainbow trout (size range <0.2 g) after a 7 day exposure to ammonium chloride, but at concentrations that overlapped with those inducing mortality in the test population inhibition concentration (IC) IC_{25} ranged from 104-210 mg/L ammonium chloride and LC_{50} ranged from 163-271 mg/L ammonium chloride). Moreover, the study organisms used by Lazorchak and Smith (2007 as cited in Appendix 3) were too young to fit within the life stage criteria established for this modeling exercise. In addition, pH was not reported in this study, so accurate normalization was not possible. Broderius and Smith (1979 as cited in Appendix 3) also exposed small rainbow trout (0.18 g) to ammonia over a 30-day period. Significant reductions in growth were seen at 0.32 mg NH_3-N/L, but survival was 70% of that observed in the controls (60%), so the quality and usefulness of this data is suspect. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects if studies where significant mortality occurred were included. Since data for the appropriate life stages or time frames were unavailable, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium (acute criterion = 2.0 µg/L; chronic criterion = 0.25 µg/L): Studies identified by the first round of literature review as having data on acute and chronic toxicity for the freshwater phase of salmonids were examined to gather data for parameterizing the population models. All data were hardness adjusted to 100 mg $CaCO_3$/L and reported as dissolved cadmium in µg/L using the hardness equations found in Mebane (2006 as cited in Appendix 3). The acute toxicity focused on 96-h mortality data for swimup fry, parr and subyearling smolt. Species mean values (geometric means of LC_{50} values) were calculated for salmonid fishes, and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species means at 4.53 µg/L (Appendix 3, Table A3). Sigmoid slopes were calculated when dose-response data were available. The resulting geometric mean of the slopes was 6.4 and the range was 4.7-7.8 (Besser *et al.* 2007, Finlayson and Verrue 1982, Davies *et al.* 1993 as cited in Appendix 3). Besser *et al.*. (2007 as cited in Appendix 1) estimated a 28-day LC_{50} for rainbow trout of 5.5 µg/L (Appendix 1, Table A3). The normalized LC_{50} value of 5.36 µg/L and the acute slope of 6.4 were used to parameterize the chronic criteria scenario of the mortality model.

Chronic cadmium studies were examined for applicable input data for the somatic growth model. Studies on the effects of cadmium on the growth of subyearling salmonids supported the statement by Mebane (2006 as cited in Appendix 3) that growth is seldom a sensitive endpoint for cadmium. At concentrations that produced changes in somatic growth, increased mortality was also observed in most studies (Mebane *et al.*. 2008, Brinkman and Hansen 2007, Hansen *et al.*, 2002b). In 24- and 30-day exposures of Atlantic salmon (*Salmo salar*), a reduction in size was seen after alevins were exposed to 6.75-21.8 µg Cd/L but these concentrations also produced 80-90% mortality (Rombough and Garside 1982, Peterson *et al.*, 1983). Bull trout (*Salvelinus confluentus*) fry (0.2 g) exposed to 1.57 µg Cd/L for 55 days (hardness adjusted to 100 mg $CaCO_3$/L) showed a 28% reduction in growth at this single time point, along with a 37% reduction in survival (Hansen *et al.* 2002b as cited in Appendix 3). No dose response curve for

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growth was generated by the study, so these data could not be used for extrapolation to other concentrations.

Brinkman and Hansen (2007 as cited in Appendix 3) exposed brown trout fry (*Salmo* trutta) to cadmium for 30 days under different water chemistries and calculated a range of IC_{20}s from 1.7-4.8 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) for reduced growth in the surviving individuals. Mortality chronic values for the same tests ranged from 2.04 to 4.79 µg Cd/L. They also calculated LC_{50} values for the first 96 h of the exposures and these ranged from 3.27 to 6.75 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L). Possible size-selective mortality or growth compensation due to decreased density were not addressed in the study design. Rainbow trout fry exposed to cadmium for 28 days exhibited increased mortality and dry weight at concentrations above a calculated NOEC of 1.3 µg Cd/L (Besser *et al.* 2007 as cited in Appendix 3). This may be attributed to size-selective mortality or an increase in somatic growth. One rainbow trout early-life-stage exposure lasting 62 days determined an EC_{10} for growth of 0.31 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) without the increased mortality (Mebane *et al.* 2008 as cited in Appendix 3). Changes in growth at these life stages (embryos and alevins) are not compatible with the somatic growth model that assesses changes in free-swimming, feeding fry during the linear portion of their growth phase, and could not be used to parameterize the model. Similarly, brook trout (*Salvelinus fontinalis*) exposed to 0.36 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) for 30 days showed reduced prey capture efficiencies and differences in prey selection in artificial stream channels (Riddell *et al.* 2005 as cited in Appendix 3), which may link to changes in somatic growth, but this link could not be translated into appropriate input parameters for the current growth model.

Copper (acute criterion = 13 µg/L; chronic criterion = 9 µg/L): Studies having data on acute and chronic toxicity for the freshwater phase of salmonids were examined to gather data needed to establish values for several parameters of the population models. All data was hardness adjusted to 100 mg $CaCO_3$/L using the acute and chronic hardness equations for copper (EPA 2002 as cited in Appendix 3). For studies with non-laboratory water that reported total instead of dissolved copper, total copper was adjusted by 80% to estimate the dissolved portion of copper in µg/L. The acute toxicity focused on 96-h mortality data for swim-up fry, parr and subyearling fish. Species mean values (geometric means of LC_{50} values) were calculated (Appendix 1, Table A3) and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species. For direct mortality, the genus mean LC_{50} was 86.8 µg/L with species means ranging from 48.3-190.6 µg/L, while for chronic toxicity (exposures of at least 30 days) the genus mean value was 98.9 µg/L with a range of 73.9-132.2 µg/L. Sigmoid slopes were calculated when dose-response data were available (Appendix 3, Table A3). The resulting geometric means (with ranges) of the slopes were 5.2 (4.1-7.6) for the 96-hr exposures and 4.2 (3.1-5.4) for the longer term mortality studies.

Growth studies on fry over 0.2 grams and under 6 grams produced EC_{50} values ranging from 20.33 µg/L to 112.43 µg/L (all values hardness adjusted, Appendix 3, Table A4). Exposures lasted 15 - 98 days. NOEC values ranged from 5.83 - 113.82µg/L. Mortality was often observed in these studies and ranged from none reported to well over 50% at similar concentrations to those that produced growth effects (Appendix 1, Table A4). For example, Besser *et al.*. (2005 as cited in Appendix 3) reported the lowest growth EC_{50} of 20.33µg/L for 0.2 g fry after a 30 day

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exposure, but also reported a 30-day LC_{50} of 16.83µg/L with a slope of 5.4 (Appendix 3, Table A4). Therefore, similar to the results with cadmium exposures occurring to subyearling salmonids between 1 and 6g, growth effects often were confounded by mortality since most of the growth studies reported mortality assessment values (LC_{50}s, chronic values, NOECs) that overlapped with or were less than the growth assessment values (EC_{50}s, NOECs; Appendix 1, Table A4). Hansen *et al.*(2002c as cited in Appendix 3) used the IC_{20} as an endpoint for comparison since concentrations producing over 20% growth inhibition were often accompanied by significant mortality. Many other growth studies found in the literature search were excluded for reasons such as using too few exposure concentrations, using exposures beginning before swim-up (usually just after fertilization), or reporting no effect on growth for the concentrations tested. As mentioned above, in the remaining studies concentrations that produced effects on growth often also showed significant decreases in survival. For example, Mudge *et al.*. (1993 as cited in Appendix 3) reported that, for three of their five tests in coho, mortality was more sensitive than growth (Appendix 3, Table A4). Nonetheless, some limited scenarios were run in the somatic growth model that looked at whether growth alone would be affected by exposures at the chronic criteria value for copper. The time-to-effect and time-to-recovery values used for copper were both 0.5 days.

Model Output

Ammonia: Using the genus geometric mean LC_{50} and dose-response slope, with 100% of the population exposed to the criteria concentrations, the direct mortality population model output showed 0% mortality to subyearlings and a zero percent change in the population growth rate (lambda) for all four life-history models (Table 2.6.5.1.47). The lowest species mean value in the *Oncorhynchus* range was also tested at 15.1 mg NH_3-N/L, and resulted in zero percent mortality and zero percent change in λ. When the chronic criterion was assessed with a 29-d exposure, the direct mortality population model predicted no mortality or change in λ.

Studies on chronic exposures of juvenile salmonids to ammonia reported no or very little effects on somatic growth, but these were accompanied by mortality. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium: Direct mortality population model runs were conducted using exposures to the criteria concentrations and the genus mean value calculated for *Oncorhynchus* (Table 2.6.5.1.1). This value produced 1 percent mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC_{50} and slope values as opposed to the minimum end of the range for species mean values (Table 2.6.5.1.1). Only when the minimum species mean value and the minimum slope were used did mortality rise to a level that produced changes in lambda that were greater than the standard deviation of the control models (Table 2.6.5.1.47). Changes in population growth rates for the stream-type Chinook and coho salmon were larger than one standard deviation from the control models. An estimated 28-day exposure to the chronic criterion produced no mortality or change in lambda.

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Studies on chronic cadmium toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model was not run for cadmium.

Copper: Direct mortality population model runs were conducted using exposures to the criteria concentrations and both the acute and chronic parameters calculated for *Oncorhynchus* (Table 2.6.5.1). The acute LC_{50} and slope produced 0% mortality and no changes in the population growth rate for any of the four life history population models. The chronic LC_{50} and slope produced 0 percent mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC_{50} and slope values as opposed to the minimum end of the range for species mean values, but no mortality was projected (Table 2.6.5.1.1).

Studies on copper toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. In spite of this, some growth model scenarios were run. When the maximum exposure period was used for the chronic criteria value in the growth model (140, 164 or 184 days depending on the life history), with an EC_{50} of 20.33, slope of 2.7 (Besser 2005 as cited in Appendix 3) and the chronic criterion value of 9 µg/L, the percent change in λ ranged from -1 to -4 percent (depending on life history). None of these reductions exceeded the control standard deviations. A 30-day exposure produced no decline in population growth rates. When a 30-day exposure for direct mortality was modeled using the minimum species values with a LC_{50} of 73.9 µg/L and a slope of 4.2, the chronic criterion (9 µg /L) produced no change in λ for the four life history models.

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Table 2.6.5.1.1 Direct mortality population model scenarios for ammonia, cadmium and copper criteria. Standard scenarios used the genus mean values for the criteria. Since no effect resulted, the minimum species mean values were assessed. The numbers in parentheses are the natural variability in λ. Bold indicates a percent change in lambda greater than one standard deviation from the baseline population model. The direct mortality population model scenarios for ammonia, cadmium, and copper do not take into account sublethal responses, indirect effects, mixture toxicity, and baseline stressors.

		Mortality input parameters			**Output**	**Percent change in lambda**			
Chemical	Test length	LC_{50} (mg/L)	Sigmoid slope	Criteria Conc.	Percent mortality	Chinook ocean-type	Chinook stream-type	Sockeye	Coho
Ammonia	96-hr	23.6[1]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	96-hr	15.1[2]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	29-d	21.3	6.4[3]	1.7	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Cadmium	96-hr	4.53[1]	6.4[1]	2.0	1	0(13)	0(4)	0(8)	0(7)
Cadmium	96-hr	4.53[1]	4.7[2]	2.0	2	-1(13)	-1(4)	-1(8)	-1(7)
Cadmium	96-hr	2.67[2]	6.4[1]	2.0	14	-4(12)	-3(4)	-3(8)	-5(7)
Cadmium	96-hr	2.67[2]	4.7[2]	2.0	20	-7(12)	**-5(4)**	-5(8)	-7(7)
Cadmium	28-d	5.36[1]	6.4[3]	0.25	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Copper	96-hr	86.8[1]	5.2[1]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	96-hr	48.3[2]	4.1[2]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	98.9[1]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	73.9[2]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)

[1]Genus geometric mean for *Oncorhynchus* values
[2]Minimum species mean value from the range of *Oncorhynchus* values.
[3]Slope for chronic exposures not identified, used genus mean slope from 96-hr exposures.

Summary: The only scenarios producing direct mortality sufficient to decrease the population growth rates or productivity were those using the lowest species mean values for cadmium. The other scenarios assessing the direct mortality from exposure to the suggested criteria values for ammonia, cadmium and copper did not result in significant changes in population productivity greater than one standard deviation from baseline population model.

Model Run II: Acute toxicity exposure-response analysis and direct mortality population modeling—aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc.

The statistical inputs for the Model Run II are displayed in Table 2.6.5.1.2. Tables 2.6.5.1.3 through 2.6.5.1.243 provide the output of the direct mortality population modeling on the percent mortality and changes in λ for each freshwater compound and for each of the six

salmonid fishes life history strategies. The NMFS only used LC_{50} toxicity data for free-swimming juvenile life stages for the direct mortality population modeling. Each table provides information on the chemical, concentration (criterion), LC_{50}, the geometric mean and the minimum species mean value of the 96-hour LC_{50} for the respective acute toxicity data set; the default dose-response sigmoid slope; species; percent mortality resulting from the LC_{50} and slope; the percent of the population exposed; the percent change in λ and its standard deviation (impacted) measured against the baseline population model; the mean value of lambda and its standard deviation, the first-year survival rate (S1); and the significant change, which is the percent change in lambda that exceeds one standard deviation of the baseline model. The first table is for each life history type and provides the results of the model run based on the geometric mean of the 96-hour LC_{50}. The second table is for each life history type and provides the results of the model run based on the minimum species mean value of the 96-hour LC_{50}. For details regarding the model output information in Tables 2.6.5.3 through 2.6.5.1.243, refer to Appendix 3.

The direct mortality population model scenarios for aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc do not take into account sublethal responses, indirect effects, mixture toxicity, and baseline stressors.

Table 2.6.5.1.2 Freshwater toxicity data statistics used as inputs for the Model Run II.

Compound	Acute Criterion	Acute Data (Geometric Mean)	Acute Data Used in the Direct Mortality Population Model (the geometric mean and the minimum species mean values)
Aluminum	750	2247	2671—445
Ammonia	5.6	32	32—7.3
Arsenic	340	16698	34269—10
Lindane	0.95	22.7	19.7—1
Cadmium	2	9.1	9—1.16
Chromium (III)	570	9825	9825—7762
Chromium (VI)	16	74908	74908—12079
Copper	13	96	96—5.7
Dieldrin	0.24	27	24—0.56
Endosulfan-alpha	0.22	0.66	0.66—0.17
Endosulfan-beta	0.22	0.66	0.66—0.17
Endrin	0.086	1.1	0.6—0.089
Heptachlor Epoxide	0.52	13.6	13.6—6.7
Lead	65	14675	17042—320
Nickel	470	18793	17663—588
Pentachlorophenol	19	86.9	86.1—10
Selenium	190	2850	4268—0.4
Silver	3.2	63	63—1.28
Tributyltin	0.46	3.2	2.6—0.21
Zinc	120	1190	1188—238

Aluminum

Table 2.6.5.1.3 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg 1 std	-	12.9
LC50	2671	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.62e-003	5.56e-003
% Mortality	1	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.4 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-43
Concentration	750	% chg 1 std	-	7.1
LC50	445	lambda mean	1.09	0.62
LC50 slope	3.6	lambda std	0.10	0.05
species	chinook, ot	S1	5.62e-003	7.47e-004
% Mortality	87	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.5 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg 1 std	-	4.3
LC50	2671	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	6.37e-002
% Mortality	1	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.6 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-39
Concentration	750	% chg l std	-	2.6
LC50	445	lambda mean	1.00	0.61
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.44e-002	8.53e-003
% Mortality	87	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.7 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	7.9
LC50	2671	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.55e-002
% Mortality	1	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.8 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-38
Concentration	750	% chg l std	-	4.8
LC50	445	lambda mean	1.01	0.63
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.56e-002	3.41e-003
% Mortality	87	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.9 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	7.5
LC50	2671	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.93e-002
% Mortality	1	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.10 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-49
Concentration	750	% chg l std	-	3.8
LC50	445	lambda mean	1.03	0.52
LC50 slope	3.6	lambda std	0.05	0.03
species	coho	S1	2.97e-002	3.93e-003
% Mortality	87	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.11 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	4.3
LC50	2671	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	6.37e-002
% Mortality	1	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.12 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-39
Concentration	750	% chg l std	-	2.6
LC50	445	lambda mean	1.00	0.61
LC50 slope	3.6	lambda std	0.03	0.02
species	steelhead	S1	6.44e-002	8.53e-003
% Mortality	87	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.13 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	0
Concentration	750	% chg l std	-	12.9
LC50	2671	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.58e-003
% Mortality	1	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.14 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Aluminum	% change lambda	-	-43
Concentration	750	% chg l std	-	7.1
LC50	445	lambda mean	1.09	0.62
LC50 slope	3.6	lambda std	0.10	0.05
species	chum	S1	5.62e-003	7.47e-004
% Mortality	87	Significant change		9.1
Percent Exposed	100	[]		

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Ammonia

Table 2.6.5.1.15 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg 1 std	-	12.9
LC50	32	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.62e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.16 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-9
Concentration	5.6	% chg 1 std	-	11.7
LC50	7.3	lambda mean	1.09	0.99
LC50 slope	3.6	lambda std	0.10	0.09
species	chinook, ot	S1	5.64e-003	4.06e-003
% Mortality	28	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.17 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg 1 std	-	4.4
LC50	32	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	6.42e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.18 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-8
Concentration	5.6	% chg 1 std	-	4.1
LC50	7.3	lambda mean	1.00	0.92
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	4.65e-002
% Mortality	28	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.19 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg 1 std	-	8.0
LC50	32	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.20 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-7
Concentration	5.6	% chg 1 std	-	7.4
LC50	7.3	lambda mean	1.01	0.93
LC50 slope	3.6	lambda std	0.06	0.05
species	sockeye	S1	2.57e-002	1.86e-002
% Mortality	28	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.21 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg l std	-	7.5
LC50	32	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.22 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-10
Concentration	5.6	% chg l std	-	6.7
LC50	7.3	lambda mean	1.03	0.92
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.14e-002
% Mortality	28	Significant change		5.2
Percent Exposed	100	[]		

Table 2.6.5.1.23 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg l std	-	4.4
LC50	32	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	6.42e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.24 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-8
Concentration	5.6	% chg l std	-	4.1
LC50	7.3	lambda mean	1.00	0.92
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	4.65e-002
% Mortality	28	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.25 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	0
Concentration	5.6	% chg l std	-	12.9
LC50	32	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.62e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.26 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Ammonia	% change lambda	-	-9
Concentration	5.6	% chg l std	-	11.7
LC50	7.3	lambda mean	1.09	0.99
LC50 slope	3.6	lambda std	0.10	0.09
species	chum	S1	5.64e-003	4.06e-003
% Mortality	28	Significant change		9.1
Percent Exposed	100	[]		

Arsenic

Table 2.6.5.1.27 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg 1 std	-	12.8
LC50	34269	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.62e-003	5.62e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.28 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-95
Concentration	340	% chg 1 std	-	0.6
LC50	10	lambda mean	1.09	0.05
LC50 slope	3.6	lambda std	0.10	0.00
species	chinook, ot	S1	5.63e-003	1.73e-008
% Mortality	100	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.29 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg 1 std	-	4.4
LC50	34269	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.30 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-95
Concentration	340	% chg l std	-	0.2
LC50	10	lambda mean	1.00	0.05
LC50 slope	3.6	lambda std	0.03	0.00
species	chinook, st	S1	6.43e-002	1.97e-007
% Mortality	100	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.31 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg l std	-	7.9
LC50	34269	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.57e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.32 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-94
Concentration	340	% chg l std	-	0.4
LC50	10	lambda mean	1.01	0.06
LC50 slope	3.6	lambda std	0.06	0.00
species	sockeye	S1	2.57e-002	7.86e-008
% Mortality	100	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.33 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg 1 std	-	7.5
LC50	34269	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.2
Percent Exposed	100	[]		

Table 2.6.5.1.34 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-99
Concentration	340	% chg 1 std	-	0.1
LC50	10	lambda mean	1.03	0.01
LC50 slope	3.6	lambda std	0.05	0.00
species	coho	S1	2.97e-002	9.09e-008
% Mortality	100	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.35 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg 1 std	-	4.4
LC50	34269	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.36 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-95
Concentration	340	% chg l std	-	0.2
LC50	10	lambda mean	1.00	0.05
LC50 slope	3.6	lambda std	0.03	0.00
species	steelhead	S1	6.43e-002	1.97e-007
% Mortality	100	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.37 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	0
Concentration	340	% chg l std	-	13.0
LC50	34269	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.38 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Arsenic	% change lambda	-	-95
Concentration	340	% chg l std	-	0.6
LC50	10	lambda mean	1.09	0.05
LC50 slope	3.6	lambda std	0.10	0.00
species	chum	S1	5.63e-003	1.73e-008
% Mortality	100	Significant change		9.2
Percent Exposed	100	[]		

Lindane

Table 2.6.5.1.39 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg 1 std	-	12.9
LC50	19.7	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.64e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.40 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-16
Concentration	0.95	% chg 1 std	-	10.8
LC50	1	lambda mean	1.09	0.91
LC50 slope	3.6	lambda std	0.10	0.08
species	chinook, ot	S1	5.61e-003	3.07e-003
% Mortality	45	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.41 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg 1 std	-	4.4
LC50	19.7	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.42 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-14
Concentration	0.95	% chg 1 std	-	3.8
LC50	1	lambda mean	1.00	0.86
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	3.51e-002
% Mortality	45	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.43 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg 1 std	-	7.9
LC50	19.7	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.44 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-13
Concentration	0.95	% chg 1 std	-	6.9
LC50	1	lambda mean	1.01	0.87
LC50 slope	3.6	lambda std	0.06	0.05
species	sockeye	S1	2.57e-002	1.41e-002
% Mortality	45	Significant change		5.7
Percent Exposed	100	[]		

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Table 2.6.5.1.45 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg 1 std	-	7.6
LC50	19.7	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.06	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.4
Percent Exposed	100	[]		

Table 2.6.5.1.46 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-18
Concentration	0.95	% chg 1 std	-	6.1
LC50	1	lambda mean	1.03	0.84
LC50 slope	3.6	lambda std	0.05	0.04
species	coho	S1	2.97e-002	1.62e-002
% Mortality	45	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.47 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg 1 std	-	4.4
LC50	19.7	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.48 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-14
Concentration	0.95	% chg l std	-	3.8
LC50	1	lambda mean	1.00	0.86
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	3.51e-002
% Mortality	45	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.49 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	0
Concentration	0.95	% chg l std	-	12.9
LC50	19.7	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.50 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lindane	% change lambda	-	-16
Concentration	0.95	% chg l std	-	10.8
LC50	1	lambda mean	1.09	0.91
LC50 slope	3.6	lambda std	0.10	0.08
species	chum	S1	5.61e-003	3.07e-003
% Mortality	45	Significant change		9.2
Percent Exposed	100	[]		

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Cadmium

Table 2.6.5.1.51 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	12.9
LC50	10.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.52 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-45
Concentration	2	% chg l std	-	7.0
LC50	1.16	lambda mean	1.09	0.60
LC50 slope	3.6	lambda std	0.10	0.05
species	chinook, ot	S1	5.62e-003	6.94e-004
% Mortality	88	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.53 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	4.3
LC50	10.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	6.42e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.54 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-40
Concentration	2	% chg l std	-	2.6
LC50	1.16	lambda mean	1.00	0.60
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	7.94e-003
% Mortality	88	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.55 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	7.9
LC50	10.6	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.56e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.56 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-39
Concentration	2	% chg l std	-	4.8
LC50	1.16	lambda mean	1.01	0.62
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.57e-002	3.17e-003
% Mortality	88	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.57 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	7.5
LC50	10.6	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.58 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-50
Concentration	2	% chg l std	-	3.7
LC50	1.16	lambda mean	1.03	0.51
LC50 slope	3.6	lambda std	0.05	0.03
species	coho	S1	2.97e-002	3.66e-003
% Mortality	88	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.59 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg l std	-	4.4
LC50	10.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.41e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.60 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-40
Concentration	2	% chg 1 std	-	2.5
LC50	1.16	lambda mean	1.00	0.60
LC50 slope	3.6	lambda std	0.03	0.02
species	steelhead	S1	6.43e-002	7.93e-003
% Mortality	88	Significant change		3.0
Percent Exposed	100	[]		

Table 2.6.5.1.61 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	0
Concentration	2	% chg 1 std	-	12.8
LC50	10.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.61e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.62 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Cadmium	% change lambda	-	-45
Concentration	2	% chg 1 std	-	7.0
LC50	1.16	lambda mean	1.09	0.60
LC50 slope	3.6	lambda std	0.10	0.05
species	chum	S1	5.63e-003	6.94e-004
% Mortality	88	Significant change		9.2
Percent Exposed	100	[]		

Chromium (III)

Table 2.6.5.1.63 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	12.8
LC50	9825	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.64 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	12.8
LC50	7762	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.65e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.65 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	4.4
LC50	9825	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.66 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	4.4
LC50	7762	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.67 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	7.9
LC50	9825	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.68 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	8.0
LC50	7762	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

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Table 2.6.5.1.69 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg 1 std	-	7.5
LC50	9825	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.70 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg 1 std	-	7.5
LC50	7762	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.71 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg 1 std	-	4.4
LC50	9825	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.72 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	4.4
LC50	7762	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.73 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	12.8
LC50	9825	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.74 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium III	% change lambda	-	0
Concentration	570	% chg l std	-	12.9
LC50	7762	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.61e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Chromium (VI)

Table 2.6.5.1.75 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	12.8
LC50	74908	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.65e-003	5.64e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.76 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	12.8
LC50	12079	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.62e-003	5.62e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.77 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	4.4
LC50	74908	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.78 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	4.4
LC50	12079	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.79 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	8.0
LC50	74908	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.80 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	8.0
LC50	12079	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.81 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	7.5
LC50	74908	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.82 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	7.6
LC50	12079	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.06	0.06
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.4
Percent Exposed	100	[]		

Table 2.6.5.1.83 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg l std	-	4.4
LC50	74908	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.84 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg 1 std	-	4.4
LC50	12079	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.85 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg 1 std	-	12.8
LC50	74908	lambda mean	1.09	1.09
LC50 slope	4.5	lambda std	0.10	0.10
species	chum	S1	5.65e-003	5.64e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.86 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Chromium VI	% change lambda	-	0
Concentration	16	% chg 1 std	-	12.9
LC50	12079	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.64e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Copper

Table 2.6.5.1.87 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	12.9
LC50	96	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.88 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-57
Concentration	13	% chg l std	-	5.5
LC50	5.7	lambda mean	1.09	0.47
LC50 slope	3.6	lambda std	0.10	0.04
species	chinook, ot	S1	5.64e-003	2.75e-004
% Mortality	95	Significant change		9.3
Percent Exposed	100	[]		

Table 2.6.5.1.89 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	4.4
LC50	96	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.42e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.90 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-52
Concentration	13	% chg l std	-	2.0
LC50	5.7	lambda mean	1.00	0.48
LC50 slope	3.6	lambda std	0.03	0.01
species	chinook, st	S1	6.44e-002	3.14e-003
% Mortality	95	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.91 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	7.8
LC50	96	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.92 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-51
Concentration	13	% chg l std	-	3.7
LC50	5.7	lambda mean	1.01	0.50
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.57e-002	1.26e-003
% Mortality	95	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.93 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	7.5
LC50	96	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.94 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-63
Concentration	13	% chg l std	-	2.7
LC50	5.7	lambda mean	1.03	0.38
LC50 slope	3.6	lambda std	0.05	0.02
species	coho	S1	2.97e-002	1.45e-003
% Mortality	95	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.95 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg l std	-	4.4
LC50	96	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.96 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-52
Concentration	13	% chg 1 std	-	2.0
LC50	5.7	lambda mean	1.00	0.48
LC50 slope	3.6	lambda std	0.03	0.01
species	steelhead	S1	6.43e-002	3.14e-003
% Mortality	95	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.97 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	0
Concentration	13	% chg 1 std	-	13.0
LC50	96	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.98 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Copper	% change lambda	-	-57
Concentration	13	% chg 1 std	-	5.4
LC50	5.7	lambda mean	1.09	0.47
LC50 slope	3.6	lambda std	0.10	0.04
species	chum	S1	5.64e-003	2.75e-004
% Mortality	95	Significant change		9.1
Percent Exposed	100	[]		

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Dieldrin

Table 2.6.5.1.99 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg 1 std	-	13.0
LC50	24	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.63e-003	5.65e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.100 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg 1 std	-	12.6
LC50	0.56	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.37e-003
% Mortality	5	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.101 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg 1 std	-	4.4
LC50	24	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.102 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg l std	-	4.3
LC50	0.56	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.14e-002
% Mortality	5	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.103 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg l std	-	8.0
LC50	24	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.104 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg l std	-	7.9
LC50	0.56	lambda mean	1.01	1.00
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.46e-002
% Mortality	5	Significant change		5.7
Percent Exposed	100	[]		

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Table 2.6.5.1.105 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg 1 std	-	7.5
LC50	24	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.106 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-2
Concentration	0.24	% chg 1 std	-	7.4
LC50	0.56	lambda mean	1.03	1.01
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.83e-002
% Mortality	5	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.107 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg 1 std	-	4.4
LC50	24	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.108 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg l std	-	4.3
LC50	0.56	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.15e-002
% Mortality	5	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.109 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	0
Concentration	0.24	% chg l std	-	13.0
LC50	24	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.65e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.110 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Dieldrin	% change lambda	-	-1
Concentration	0.24	% chg l std	-	12.7
LC50	0.56	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.64e-003	5.38e-003
% Mortality	5	Significant change		9.1
Percent Exposed	100	[]		

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Endosulfan-alpha

Table 2.6.5.1.111 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg 1 std	-	12.7
LC50	0.66	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.1
species	Chinook, ot	S1	5.63e-003	5.53E-03
% Mortality	2	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.112 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-30
Concentration	0.22	% chg 1 std	-	8.8
LC50	0.17	lambda mean	1.09	0.76
LC50 slope	3.6	lambda std	0.10	0.07
species	chinook, ot	S1	5.63e-003	1.60e-003
% Mortality	72	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.113 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg 1 std	-	4.4
LC50	0.66	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.31E-02
% Mortality	2	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.114 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-27
Concentration	0.22	% chg 1 std	-	3.2
LC50	0.17	lambda mean	1.00	0.73
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	1.82e-002
% Mortality	72	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.115 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg 1 std	-	7.9
LC50	0.66	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.58e-002	2.52E-02
% Mortality	2	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.116 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-26
Concentration	0.22	% chg 1 std	-	5.8
LC50	0.17	lambda mean	1.01	0.75
LC50 slope	3.6	lambda std	0.06	0.04
species	sockeye	S1	2.57e-002	7.26e-003
% Mortality	72	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.117 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg 1 std	-	7.4
LC50	0.66	lambda mean	1.03	1.02
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.91E-02
% Mortality	2	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.118 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-34
Concentration	0.22	% chg 1 std	-	4.9
LC50	0.17	lambda mean	1.03	0.68
LC50 slope	3.6	lambda std	0.05	0.04
species	coho	S1	2.97e-002	8.41e-003
% Mortality	72	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.119 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
oncentration	0.22	% chg 1 std	-	4.4
LC50	0.66	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.31E-02
% Mortality	2	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.120 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-27
Concentration	0.22	% chg 1 std	-	3.2
LC50	0.17	lambda mean	1.00	0.73
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	1.82e-002
% Mortality	72	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.121 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-1
Concentration	0.22	% chg 1 std	-	12.7
LC50	0.66	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.1
species	chum	S1	5.63e-003	5.53E-03
% Mortality	1	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.122 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-alpha	% change lambda	-	-30
Concentration	0.22	% chg 1 std	-	8.8
LC50	0.17	lambda mean	1.09	0.76
LC50 slope	3.6	lambda std	0.10	0.07
species	chum	S1	5.65e-003	1.60e-003
% Mortality	72	Significant change		9.1
Percent Exposed	100	[]		

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Endosulfan-beta

Table 2.6.5.1.123 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg 1 std	-	12.7
LC50	0.66	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.1
species	Chinook, ot	S1	5.63e-003	5.53E-03
% Mortality	2	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.124 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-30
Concentration	0.22	% chg 1 std	-	8.8
LC50	0.17	lambda mean	1.09	0.76
LC50 slope	3.6	lambda std	0.10	0.07
species	chinook, ot	S1	5.63e-003	1.60e-003
% Mortality	72	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.125 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg 1 std	-	4.4
LC50	0.66	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.31E-02
% Mortality	2	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.126 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-27
Concentration	0.22	% chg 1 std	-	3.2
LC50	0.17	lambda mean	1.00	0.73
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	1.82e-002
% Mortality	72	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.127 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg 1 std	-	7.9
LC50	0.66	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.58e-002	2.52E-02
% Mortality	2	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.128 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-26
Concentration	0.22	% chg 1 std	-	5.8
LC50	0.17	lambda mean	1.01	0.75
LC50 slope	3.6	lambda std	0.06	0.04
species	sockeye	S1	2.57e-002	7.26e-003
% Mortality	72	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.129 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg l std	-	7.4
LC50	0.66	lambda mean	1.03	1.02
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.91E-02
% Mortality	2	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.130 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-34
Concentration	0.22	% chg l std	-	4.9
LC50	0.17	lambda mean	1.03	0.68
LC50 slope	3.6	lambda std	0.05	0.04
species	coho	S1	2.97e-002	8.41e-003
% Mortality	72	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.131 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
oncentration	0.22	% chg l std	-	4.4
LC50	0.66	lambda mean	1.00	0.99
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.31E-02
% Mortality	2	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.132 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-27
Concentration	0.22	% chg 1 std	-	3.2
LC50	0.17	lambda mean	1.00	0.73
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.43e-002	1.82e-002
% Mortality	72	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.133 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-1
Concentration	0.22	% chg 1 std	-	12.7
LC50	0.66	lambda mean	1.09	1.08
LC50 slope	3.6	lambda std	0.10	0.1
species	chum	S1	5.63e-003	5.53E-03
% Mortality	1	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.134 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endosulfan-beta	% change lambda	-	-30
Concentration	0.22	% chg 1 std	-	8.8
LC50	0.17	lambda mean	1.09	0.76
LC50 slope	3.6	lambda std	0.10	0.07
species	chum	S1	5.65e-003	1.60e-003
% Mortality	72	Significant change		9.1
Percent Exposed	100	[]		

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Endrin

Table 2.6.5.1.135 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	12.9
LC50	0.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.64e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.136 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-17
Concentration	0.086	% chg l std	-	10.7
LC50	0.089	lambda mean	1.09	0.91
LC50 slope	3.6	lambda std	0.10	0.08
species	chinook, ot	S1	5.64e-003	2.99e-003
% Mortality	47	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.137 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	4.4
LC50	0.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.138 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	4.4
LC50	0.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.139 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-14
Concentration	0.086	% chg l std	-	3.7
LC50	0.089	lambda mean	1.00	0.85
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	3.41e-002
% Mortality	47	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.140 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	8.0
LC50	0.6	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.58e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.141 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-14
Concentration	0.086	% chg l std	-	6.7
LC50	0.089	lambda mean	1.01	0.87
LC50 slope	3.6	lambda std	0.06	0.05
species	sockeye	S1	2.57e-002	1.36e-002
% Mortality	47	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.142 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg l std	-	7.5
LC50	0.6	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.143 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-19
Concentration	0.086	% chg l std	-	6.1
LC50	0.089	lambda mean	1.03	0.83
LC50 slope	3.6	lambda std	0.05	0.04
species	coho	S1	2.96e-002	1.57e-002
% Mortality	47	Significant change		5.3
Percent Exposed	100	[]		

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Table 2.6.5.1.144 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg 1 std	-	4.4
LC50	0.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.145 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-14
Concentration	0.086	% chg 1 std	-	3.8
LC50	0.089	lambda mean	1.00	0.85
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	3.42e-002
% Mortality	47	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.146 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	0
Concentration	0.086	% chg 1 std	-	12.9
LC50	0.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.64e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.147 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Endrin	% change lambda	-	-17
Concentration	0.086	% chg l std	-	10.7
LC50	0.089	lambda mean	1.09	0.91
LC50 slope	3.6	lambda std	0.10	0.08
species	chum	S1	5.63e-003	2.99e-003
% Mortality	47	Significant change		9.1
Percent Exposed	100	[]		

Heptachlor Epoxide

Table 2.6.5.1.148 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	13.0
LC50	13.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.65e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.149 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	12.8
LC50	6.7	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.150 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	4.4
LC50	13.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.44e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.151 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	4.4
LC50	6.7	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.44e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.152 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	7.9
LC50	13.6	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.153 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	7.9
LC50	6.7	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.58e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.154 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	7.4
LC50	13.6	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.2
Percent Exposed	100	[]		

Table 2.6.5.1.155 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	7.5
LC50	6.7	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.2
Percent Exposed	100	[]		

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Table 2.6.5.1.156 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg 1 std	-	4.4
LC50	13.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.157 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg 1 std	-	4.4
LC50	6.7	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.158 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg 1 std	-	13.0
LC50	13.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.65e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

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Table 2.6.5.1.159 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Heptachlor Epoxide	% change lambda	-	0
Concentration	0.52	% chg l std	-	12.9
LC50	6.7	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Lead

Table 2.6.5.1.160 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	12.7
LC50	17042	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.161 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	12.9
LC50	320	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

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2.6.5.1.162 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	4.4
LC50	17042	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

2.6.5.1.163 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	4.4
LC50	320	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	6.41e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.164 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	8.0
LC50	17042	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.56e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.165 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	8.0
LC50	320	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.55e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.166 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	7.5
LC50	17042	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.167 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	7.5
LC50	320	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

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Table 2.6.5.1.168 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg 1 std	-	4.4
LC50	17042	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.169 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg 1 std	-	4.4
LC50	320	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.41e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.170 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg 1 std	-	12.7
LC50	17042	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.0
Percent Exposed	100	[]		

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Table 2.6.5.1.171 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Lead	% change lambda	-	0
Concentration	65	% chg l std	-	12.9
LC50	320	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.61e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Nickel

Table 2.6.5.1.172 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg l std	-	12.9
LC50	17663	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.62e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.173 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-10
Concentration	470	% chg l std	-	11.5
LC50	588	lambda mean	1.09	0.98
LC50 slope	3.6	lambda std	0.10	0.09
species	chinook, ot	S1	5.62e-003	3.92e-003
% Mortality	31	Significant change		9.1
Percent Exposed	100	[]		

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Table 2.6.5.1.174 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg 1 std	-	4.4
LC50	17663	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.175 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-9
Concentration	470	% chg 1 std	-	4.0
LC50	588	lambda mean	1.00	0.91
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	4.45e-002
% Mortality	31	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.176 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg 1 std	-	8.0
LC50	17663	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.58e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.177 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-8
Concentration	470	% chg l std	-	7.2
LC50	588	lambda mean	1.01	0.92
LC50 slope	3.6	lambda std	0.06	0.05
species	sockeye	S1	2.57e-002	1.78e-002
% Mortality	31	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.178 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg l std	-	7.4
LC50	17663	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.179 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-12
Concentration	470	% chg l std	-	6.6
LC50	588	lambda mean	1.03	0.91
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.05e-002
% Mortality	31	Significant change		5.3
Percent Exposed	100	[]		

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Table 2.6.5.1.180 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg 1 std	-	4.4
LC50	17663	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.181 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-9
Concentration	470	% chg 1 std	-	4.0
LC50	588	lambda mean	1.00	0.91
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	4.45e-002
% Mortality	31	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.182 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	0
Concentration	470	% chg 1 std	-	12.9
LC50	17663	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.62e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

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Table 2.6.5.1.183 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Nickel	% change lambda	-	-10
Concentration	470	% chg 1 std	-	11.6
LC50	588	lambda mean	1.09	0.98
LC50 slope	3.6	lambda std	0.10	0.09
species	chum	S1	5.64e-003	3.87e-003
% Mortality	31	Significant change		9.3
Percent Exposed	100	[]		

Pentachlorophenol

Table 2.6.5.1.184 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg 1 std	-	12.8
LC50	86.1	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.1
species	Chinook, ot	S1	5.63e-003	5.57E-03
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.185 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-49
Concentration	19	% chg 1 std	-	6.4
LC50	10	lambda mean	1.09	0.55
LC50 slope	3.6	lambda std	0.10	0.05
species	chinook, ot	S1	5.62e-003	5.09e-004
% Mortality	91	Significant change		9.1
Percent Exposed	100	[]		

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Table 2.6.5.1.186 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg l std	-	4.4
LC50	86.1	lambda mean	1.00	1
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.37E-02
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.187 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-45
Concentration	19	% chg l std	-	2.4
LC50	10	lambda mean	1.00	0.55
LC50 slope	3.6	lambda std	0.03	0.02
species	chinook, st	S1	6.44e-002	5.81e-003
% Mortality	91	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.188 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg l std	-	7.9
LC50	86.1	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.58e-002	2.55E-02
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.189 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-43
Concentration	19	% chg l std	-	4.4
LC50	10	lambda mean	1.01	0.57
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.56e-002	2.32e-003
% Mortality	91	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.190 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg l std	-	7.5
LC50	86.1	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.94E-02
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.191 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-55
Concentration	19	% chg l std	-	3.4
LC50	10	lambda mean	1.03	0.46
LC50 slope	3.6	lambda std	0.05	0.02
species	coho	S1	2.97e-002	2.68e-003
% Mortality	91	Significant change		5.3
Percent Exposed	100	[]		

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Table 2.6.5.1.192 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg 1 std	-	4.4
LC50	86.1	lambda mean	1.00	1
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.37E-02
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.193 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-45
Concentration	19	% chg 1 std	-	2.4
LC50	10	lambda mean	1.00	0.55
LC50 slope	3.6	lambda std	0.03	0.02
species	steelhead	S1	6.43e-002	5.80e-003
% Mortality	91	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.194 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	0
Concentration	19	% chg 1 std	-	12.8
LC50	86.1	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.1
species	chum	S1	5.63e-003	5.57E-03
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

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Table 2.6.5.1.195 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Pentachlorophenol	% change lambda	-	-49
Concentration	19	% chg 1 std	-	6.4
LC50	10	lambda mean	1.09	0.55
LC50 slope	3.6	lambda std	0.10	0.05
species	chum	S1	5.64e-003	5.07e-004
% Mortality	91	Significant change		9.1
Percent Exposed	100	[]		

Selenium

Table 2.6.5.1.196 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg 1 std	-	12.9
LC50	4268	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.197 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg 1 std	-	0.1
LC50	0.4	lambda mean	1.09	0.01
LC50 slope	3.6	lambda std	0.10	0.00
species	chinook, ot	S1	5.65e-003	1.30e-012
% Mortality	100	Significant change		9.0
Percent Exposed	100	[]		

Table 2.6.5.1.198 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	4.4
LC50	4268	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.199 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg l std	-	0.0
LC50	0.4	lambda mean	1.00	0.01
LC50 slope	3.6	lambda std	0.03	0.00
species	chinook, st	S1	6.44e-002	1.49e-011
% Mortality	100	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.200 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	8.0
LC50	4268	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.57e-002
% Mortality	0	Significant change		5.7
Percent Exposed	100	[]		

Table 2.6.5.1.201 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg l std	-	0.1
LC50	0.4	lambda mean	1.01	0.01
LC50 slope	3.6	lambda std	0.06	0.00
species	sockeye	S1	2.57e-002	5.94e-012
% Mortality	100	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.202 Model output data for coho.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	7.5
LC50	4268	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.203 Model output data for coho.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-100
Concentration	190	% chg l std	-	0.0
LC50	0.4	lambda mean	1.03	0.00
LC50 slope	3.6	lambda std	0.05	0.00
species	coho	S1	2.96e-002	6.85e-012
% Mortality	100	Significant change		5.3
Percent Exposed	100	[]		

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Table 2.6.5.1.204 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	4.4
LC50	4268	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.44e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.205 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg l std	-	0.0
LC50	0.4	lambda mean	1.00	0.01
LC50 slope	3.6	lambda std	0.03	0.00
species	steelhead	S1	6.43e-002	1.49e-011
% Mortality	100	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.206 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	0
Concentration	190	% chg l std	-	12.9
LC50	4268	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

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Table 2.6.5.1.207 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Selenium	% change lambda	-	-99
Concentration	190	% chg 1 std	-	0.1
LC50	0.4	lambda mean	1.09	0.01
LC50 slope	3.6	lambda std	0.10	0.00
species	chum	S1	5.64e-003	1.30e-012
% Mortality	100	Significant change		9.0
Percent Exposed	100	[]		

Silver

Table 2.6.5.1.208 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg 1 std	-	12.9
LC50	63	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.209 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-60
Concentration	3.2	% chg 1 std	-	5.0
LC50	1.28	lambda mean	1.09	0.43
LC50 slope	3.6	lambda std	0.10	0.04
species	chinook, ot	S1	5.63e-003	2.00e-004
% Mortality	96	Significant change		9.2
Percent Exposed	100	[]		

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Table 2.6.5.1.210 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg l std	-	4.4
LC50	63	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.211 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-56
Concentration	3.2	% chg l std	-	1.9
LC50	1.28	lambda mean	1.00	0.44
LC50 slope	3.6	lambda std	0.03	0.01
species	chinook, st	S1	6.43e-002	2.29e-003
% Mortality	96	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.212 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg l std	-	7.9
LC50	63	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.56e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.213 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-54
Concentration	3.2	% chg l std	-	3.5
LC50	1.28	lambda mean	1.01	0.46
LC50 slope	3.6	lambda std	0.06	0.02
species	sockeye	S1	2.58e-002	9.17e-004
% Mortality	96	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.214 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg l std	-	7.5
LC50	63	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.215 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-67
Concentration	3.2	% chg l std	-	2.4
LC50	1.28	lambda mean	1.03	0.34
LC50 slope	3.6	lambda std	0.05	0.02
species	coho	S1	2.97e-002	1.06e-003
% Mortality	96	Significant change		5.2
Percent Exposed	100	[]		

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Table 2.6.5.1.216 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg 1 std	-	4.4
LC50	63	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.217 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-56
Concentration	3.2	% chg 1 std	-	1.9
LC50	1.28	lambda mean	1.00	0.44
LC50 slope	3.6	lambda std	0.03	0.01
species	steelhead	S1	6.43e-002	2.29e-003
% Mortality	96	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.218 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	0
Concentration	3.2	% chg 1 std	-	12.9
LC50	63	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.63e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

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Table 2.6.5.1.219 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Silver	% change lambda	-	-60
Concentration	3.2	% chg l std	-	5.0
LC50	1.28	lambda mean	1.09	0.43
LC50 slope	3.6	lambda std	0.10	0.04
species	chum	S1	5.62e-003	2.00e-004
% Mortality	96	Significant change		9.0
Percent Exposed	100	[]		

Tributyltin

Table 2.6.5.1.220 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg l std	-	13.0
LC50	2.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.65e-003	5.64e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

Table 2.6.5.1.221 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-55
Concentration	0.46	% chg l std	-	5.6
LC50	0.21	lambda mean	1.09	0.49
LC50 slope	3.6	lambda std	0.10	0.04
species	chinook, ot	S1	5.64e-003	3.16e-004
% Mortality	94	Significant change		9.1
Percent Exposed	100	[]		

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Table 2.6.5.1.222 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg l std	-	4.4
LC50	2.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.223 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-51
Concentration	0.46	% chg l std	-	2.1
LC50	0.21	lambda mean	1.00	0.49
LC50 slope	3.6	lambda std	0.03	0.01
species	chinook, st	S1	6.44e-002	3.61e-003
% Mortality	94	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.224 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg l std	-	7.9
LC50	2.6	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.56e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.225 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-49
Concentration	0.46	% chg 1 std	-	3.9
LC50	0.21	lambda mean	1.01	0.51
LC50 slope	3.6	lambda std	0.06	0.03
species	sockeye	S1	2.57e-002	1.44e-003
% Mortality	94	Significant change		5.6
Percent Exposed	100	[]		

Table 2.6.5.1.226 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg 1 std	-	7.4
LC50	2.6	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.96e-002	2.96e-002
% Mortality	0	Significant change		5.3
Percent Exposed	100	[]		

Table 2.6.5.1.227 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-62
Concentration	0.46	% chg 1 std	-	2.9
LC50	0.21	lambda mean	1.03	0.39
LC50 slope	3.6	lambda std	0.05	0.02
species	coho	S1	2.97e-002	1.66e-003
% Mortality	94	Significant change		5.3
Percent Exposed	100	[]		

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Table 2.6.5.1.228 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg 1 std	-	4.4
LC50	2.6	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.229 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-51
Concentration	0.46	% chg 1 std	-	2.1
LC50	0.21	lambda mean	1.00	0.49
LC50 slope	3.6	lambda std	0.03	0.01
species	steelhead	S1	6.44e-002	3.61e-003
% Mortality	94	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.230 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	0
Concentration	0.46	% chg 1 std	-	13.0
LC50	2.6	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.65e-003	5.64e-003
% Mortality	0	Significant change		9.2
Percent Exposed	100	[]		

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Table 2.6.5.1.231 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Tributyltin	% change lambda	-	-55
Concentration	0.46	% chg 1 std	-	5.6
LC50	0.21	lambda mean	1.09	0.49
LC50 slope	3.6	lambda std	0.10	0.04
species	chum	S1	5.64e-003	3.16e-004
% Mortality	94	Significant change		9.1
Percent Exposed	100	[]		

Zinc

Table 2.6.5.1.232 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg 1 std	-	12.9
LC50	1188	lambda mean	1.09	1.09
LC50 slope	3.6	lambda std	0.10	0.10
species	Chinook, ot	S1	5.62e-003	5.63e-003
% Mortality	0	Significant change		9.1
Percent Exposed	100	[]		

Table 2.6.5.1.233 Model output data for ocean-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-3
Concentration	120	% chg 1 std	-	12.5
LC50	238	lambda mean	1.09	1.06
LC50 slope	3.6	lambda std	0.10	0.10
species	chinook, ot	S1	5.64e-003	5.19e-003
% Mortality	8	Significant change		9.1
Percent Exposed	100	[]		

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Table 2.6.5.1.234 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	4.4
LC50	1188	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	Chinook, st	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.235 Model output data for stream-type Chinook salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-2
Concentration	120	% chg l std	-	4.3
LC50	238	lambda mean	1.00	0.98
LC50 slope	3.6	lambda std	0.03	0.03
species	chinook, st	S1	6.43e-002	5.93e-002
% Mortality	8	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.236 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	7.9
LC50	1188	lambda mean	1.01	1.01
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.55e-002	2.57e-002
% Mortality	0	Significant change		5.6
Percent Exposed	100	[]		

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Table 2.6.5.1.237 Model output data for sockeye salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-2
Concentration	120	% chg 1 std	-	7.7
LC50	238	lambda mean	1.01	0.99
LC50 slope	3.6	lambda std	0.06	0.06
species	sockeye	S1	2.57e-002	2.37e-002
% Mortality	8	Significant change		5.5
Percent Exposed	100	[]		

Table 2.6.5.1.238 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg 1 std	-	7.6
LC50	1188	lambda mean	1.03	1.03
LC50 slope	3.6	lambda std	0.06	0.05
species	coho	S1	2.96e-002	2.97e-002
% Mortality	0	Significant change		5.4
Percent Exposed	100	[]		

Table 2.6.5.1.239 Model output data for coho salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-3
Concentration	120	% chg 1 std	-	7.3
LC50	238	lambda mean	1.03	1.00
LC50 slope	3.6	lambda std	0.05	0.05
species	coho	S1	2.97e-002	2.73e-002
% Mortality	8	Significant change		5.3
Percent Exposed	100	[]		

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Table 2.6.5.1.240 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	4.4
LC50	1188	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.241 Model output data for steelhead.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-2
Concentration	120	% chg l std	-	4.3
LC50	238	lambda mean	1.00	0.98
LC50 slope	3.6	lambda std	0.03	0.03
species	steelhead	S1	6.44e-002	5.93e-002
% Mortality	8	Significant change		3.1
Percent Exposed	100	[]		

Table 2.6.5.1.242 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	0
Concentration	120	% chg l std	-	4.4
LC50	1188	lambda mean	1.00	1.00
LC50 slope	3.6	lambda std	0.03	0.03
species	chum	S1	6.43e-002	6.43e-002
% Mortality	0	Significant change		3.1
Percent Exposed	100	[]		

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Table 2.6.5.1.243 Model output data for chum salmon.

Parameters	Value	Output	Control	Impacted
Chemical	Zinc	% change lambda	-	-3
Concentration	120	% chg l std	-	12.6
LC50	238	lambda mean	1.09	1.06
LC50 slope	3.6	lambda std	0.10	0.10
species	chum	S1	5.63e-003	5.20e-003
% Mortality	8	Significant change		9.2
Percent Exposed	100	[]		

Summary. Based on the direct mortality population modeling results, juvenile salmon and steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, chromium (III), chromium (VI), copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, lead, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc is predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) ranging from zero percent to -100 percent based on the exposure scenario. Direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for both modeling scenarios.

2.6.6. Case Study on Extrapolating Growth Reductions in Fish to Changes in Population Extinction Risks: Copper and Chinook Salmon

This section examines the potential consequences of reduced growth on the survival of juvenile Chinook salmon from exposure to low levels of copper that commence prior to hatching. Toxicological assays generally do not consider or attempt to link effects on growth to changes in population and to long-term extinction risks. However, Mebane and Arthaud (2010) suggested that size reductions from early-life stage chronic sublethal copper exposure could potentially reduce juvenile salmon survival and population recovery trajectories. This study is different from the direct mortality, somatic growth, and population modeling in section 2.6.5 in which the literature found that growth of fry, on the whole, was not a sensitive endpoint for the effect of copper on juvenile salmonids relative to mortality. In the case study by Mebane and Arthaud (2010) they conclude that growth resulting from early life stage exposure is usually a more sensitive endpoint than mortality to copper. This case study modeled responses of juvenile Chinook salmon exposed to sustained exposures of low levels of copper starting during early development and extrapolated growth reductions and changes in survival related to individual size. Most of the literature on copper and juvenile salmonid fry that examines reduced growth shows little mortality in laboratory toxicity tests, which tend to be short in exposure duration and do not look at relationships between reduced growth and size-dependant survival. Chapman (1994 as cited in Mebane and Arthaud 2010) exposed different life stages of steelhead (*Oncorhynchus mykiss*) for the same duration (3 months) to the same concentration of copper (13.4 µg/L at a hardness of 24 mg/L as $CaCO_3$). The survival of steelhead that were initially

exposed as embryos was no different than that of the unexposed control fish, even though the embryos developed into the usually-sensitive swim-up fry stage during the exposure. In contrast, steelhead that were initially exposed as swim-up fry, without the opportunity for acclimation during the embryo state, suffered complete mortality.

At low-level, sustained exposures, copper is one substance that commonly causes reduced growth but little direct mortality in laboratory toxicity tests with early life stage fish. To explore the relevance of growth reductions under laboratory conditions to wild populations, they 1) estimated growth effects of low-level copper exposures to juvenile Chinook salmon, 2) related growth effects to reduced survival in downriver Chinook salmon migrations, 3) estimated population demographics, 4) constructed a demographically structured matrix population model, and 5) projected the influence of copper-reduced growth on population size, extinction risks, and recovery chances. Reduced juvenile growth from copper in the range of 11 µg/L (the proposed chronic criteria for copper in Oregon is 9 µg/L) was projected to cause disproportionate reductions in survival of migrating juveniles, with a 7.5 percent length reduction predicting about a 23 percent to 52 percent reduction in survival from a headwaters trap to the next census point located 640 km downstream. Projecting reduced juvenile growth out through six generations (~30 years) resulted in little increased extinction risk; however, population recovery times were delayed under scenarios where copper-reduced growth was imposed.

Reduced growth is a common stress response in fish. A variety of causes can lead to stress responses and reduced growth in fish, including suboptimal nutrition or temperatures, low ion content of water (soft water), crowding, subordinate social status, and either the direct effects of chemical exposures or the energy costs of detoxifying chemicals (Wendelaar Bonga 1997 as cited in Mebane and Arthaud 2010). In ecotoxicological bioassays that run long enough, growth effects are a readily and routinely measured endpoint. In water-quality criteria derivation in the United States, the only sublethal effects that *a priori* are considered biologically important are growth or reproductive impairment, although on a case-by-case basis, data on a variety of other sublethal effects of chemicals to fish could also be important, such as swimming performance, disease resistance, or behaviors related to chemoreception (Stephan *et al.* 1985, Stephan 1986 as cited in Mebane and Arthaud 2010). However, laboratory bioassays seldom are a means unto themselves, but probably are at least indirectly conducted because societal values such as protecting the abundance and persistence of populations, biodiversity, conservation of threatened species, and recreational aesthetics (Stephan 1986, Barnthouse *et al.* 1989 as cited in Mebane and Arthaud 2010).

This motivation implies some consideration of population-level effects when interpreting toxicity bioassays. Yet, from a population biology perspective, the only endpoints that matter for a closed population are birth and death rates. Growth and any other sublethal endpoints are irrelevant unless they can be related to birth or death rates. The reproductive consequences of profound growth effects are selfevident; an organism that fails to grow is unlikely to reproduce.

However, the consequences of transitory or subtle growth reductions are less obvious. For instance, in lifecycle testing with brook trout (*Salvelinus fontinalis*) and copper, McKim and Benoit (1971 as cited in Mebane and Arthaud 2010) reported that, for their first several months of life, fish that were exposed to low, sublethal copper concentrations lagged behind control fish in their growth. However, after about six months of copper exposure, fish experienced

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compensatory growth rates and largely caught up with control fish by the end of the tests (McKim and Benoit 1971 as cited in Mebane and Arthaud 2010). Because the differences were no longer statistically different at the end of their tests, the growth delays were discounted as adverse effects. Similar instances of transitory or subtle growth reductions have been noted for rainbow trout (*Oncorhynchus mykiss*) exposed to copper (Marr *et al.* 1996, Hansen *et al.* 2002 as cited in Mebane and Arthaud 2010). However, delayed growth may not necessarily be a discountable effect in the wild because, if juvenile fish encounter a size-dependent bottleneck in early life, smaller fish may not survive long enough to benefit from compensatory growth. Traits and costs that have been associated with reduced growth in juvenile fish include acquisition of feeding territory or shelter, predation risk, body size at key times, energy reserves at key times, increased thermoregulatory costs, and mortality (Sogard 1997, Metcalfe and Monaghan 2001, Harwood *et al.* 2002, Coleman and Fausch 2007 as cited in Mebane and Arthaud 2010). The magnitudes of size differences that have been important in outcomes of challenges with juvenile fish can be small. For example, torrent sculpin (*Cottus rhotheus*) are a predator of juvenile salmon in streams. Torrent sculpin that were about 60 mm long were no threat to coho salmon (*O. kisutch*) that were also about 60 mm long. However, the 60 mm sculpin can successfully ambush, subdue, and eat 50 mm coho salmon (Patten 1977 as cited in Mebane and Arthaud 2010). Abbott *et al.* (1985 as cited in Mebane and Arthaud 2010) found that bigger fish tend to dominate smaller fish in contests for territory, and a size disparity of only 5 percent in body weight confers significant advantage. However, subtle growth reductions may be discounted as effects in toxicity tests if they are not statistically different from controls in null hypothesis significance testing with less than a 5 percent likelihood of making a Type I error. These purely statistical definitions of significant effects are at best incomplete and at worst misleading, in part because the probability that a given reduction is statistically significant is inversely related to the quality and quantity of the data (*e.g.*, Barnthouse *et al.* 1989 as cited in Mebane and Arthaud 2010).

The case study of growth effects from copper and a Chinook salmon population explored how subtle growth reductions in juvenile fish might affect the abundance and persistence of natural populations of migratory fish. The study objectives included:

1. Estimating the magnitude of growth reductions likely for Chinook salmon resulting from prolonged laboratory test exposure to copper at 11 μ/gL that had been estimated to be safe for most aquatic ecosystems. The chronic criterion for copper in Oregon is 13 μg/L.
2. Estimating potential consequences of reduced growth for the survival of juvenile Chinook salmon during rearing and migration.
3. Quantifying the potential consequences of reduced survival in migrating juvenile salmon as changes in the long-term extinction risk and recovery potential of the salmon populations.

For this exercise, Mebane and Arthaud selected the Marsh Creek Chinook salmon population, located at the headwaters of the Middle Fork of the Salmon River, Idaho, USA (44◦ 27_N, 115◦14_W at its mouth). Marsh Creek is an oligotrophic, forested watershed, with few pollution or human attributable disturbances other than potentially decreased freshwater productivity and correspondingly diminished carrying capacities from the decline of marine derived nutrients (Kohler *et al.* 2008 as cited in Mebane and Arthaud 2010). The lack of pollution sources greatly

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simplifies predicting the potential effects of a chemical stressor. Furthermore, by using a headwaters population for this modeling exercise, the baseline model accounts for a myriad of other factors affecting Chinook salmon populations besides the potential stress of copper pollution considered here.

The projections of potential population-level effects of reduced growth from copper were made in five steps:

- Evaluating the effects of chronic copper toxicity on salmon in laboratory tests
- Extrapolating reduced growth in toxicity test results to survival of juvenile migrants
- Analyzing population demographics
- Developing a baseline population model, and
- Linking changed population vital rates from copper-influenced scenarios to population size and extinction risks.

Nonlinear regression was used to interpolate between effects at the control concentration and the lowest effect concentration to estimate effects at the 1992 NTR criteria concentration of 12 µg/L, total recoverable. Because of this uncertainty, we also examined a chronic test of rainbow trout in soft water that tested lower copper concentrations and required less interpolation (Marr *et al.* 1996 as cited in Mebane and Arthaud 2010). Chinook salmon and rainbow/steelhead trout are closely related, and other tests have shown similar sensitivity to copper and other metals (Chapman 1978 as cited in Mebane and Arthaud 2010).

Logistic regression described the relation between length and copper concentrations well, and it provided an estimated length reduction from controls of 7.5 percent and a weight reduction of 20 percent at 3.6 µg/L, the hardness-adjusted 1992 CCC. The estimated length reductions at 3.6 µg/L ranged from 4 percent to 18 percent, obtained using different statistical distributions and curve fits (*e.g.*, linear, piecewise linear, logistic). For weight reductions, the corresponding reductions were greater, 12 to 20 percent, depending on the model used. The rainbow trout growth reductions were very similar to those estimated at similar concentrations with Chinook salmon using the same statistical models, suggesting that the needed interpolations of the Chinook toxicity data were reasonable.

The selection of a regression model to fit these Chinook salmon data involves fundamental, implicit assumptions of the ecotoxicology of chronic copper and fish. The logistic regression curves slope smoothly downward to interpolate from the control concentration to the first treatment. Thus, an implicit assumption of the model shape is that slight increases in copper result in corresponding slight growth reductions, with no threshold of response. In contrast, the piecewise linear regressions implicitly assume a threshold of response, below which copper concentrations have no effect on growth. It may be unrealistic to assume that no threshold exists for copper exposure and the onset of growth effects. Likewise, the abrupt bend in the corners of the piecewise linear regression that indicate the threshold concentration may also be arbitrary and unrealistic. Because neither model had an obviously better theoretical basis and because both models fit the data well, the effects estimates with each are carried forward through the population modeling using both 7.5 percent and 4 percent length reductions at 3.6 µg/L copper

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from the logistic and piecewise models, respectively. This provided a range of estimates of growth effects of copper to Chinook salmon at the 1992 CCC of 3.6 μg/l.

The Mebane and Arthaud analysis focuses on EPA's (NTR 1992) copper criteria of 18 μg/L (CMC) and 12 μg/L (CCC) (updates have been published, EPA 2006 and 2007, although at the time of writing, the 1992 values remained effective in some states, including Oregon). The EPA's 2006 recommended criteria were based on the same approach as the 1992 version with minor dataset revisions. In contrast, the 2007 values were derived from a fundamentally different approach that predicted copper bioavailability through geochemical modeling to estimate copper accumulation on gills and subsequent toxicity. For the water chemistry conditions of Chapman's (1982 as cited in Mebane and Arthaud 2010) test, the 2006 and 2007 chronic copper criteria values would be about 2.7 and 2.1 μg/L, respectively. The interpolated length reductions with Chapman's (1982 as cited in Mebane and Arthaud 2010) Chinook salmon test at the 2006 criterion value of 2.7μg/L ranged from about 6 percent to zero using logistic regression and piecewise regression models, respectively. For the 2007 criterion value of about 2.1 μg/L, the corresponding length reduction estimates ranged from about 4.5 percent to zero. Thus the modeled scenarios are also relevant to the more recent copper chronic criteria updates. For the 2006 version, the upper effects estimate (6% length reduction) would be intermediate to the 7.5 percent and 4 percent length reduction scenarios modeled. For the 2007 version, the upper effects estimate (4.5 percent length reduction) is close to the lower effects scenario modeled here (4 percent length reduction).

Risk probability statistics may provide more relevant assessments of thepopulation's relative risks of declines or extinction than do the population trajectory projections (Ferson *et al.* 1989 as cited in Mebane and Arthaud 2010). Rather than plotting abundance predictions over time, as was done with adult salmon in abundance, projections can be expressed as the risk that the population will be less than a given number or that it will decline by more than a given amount from the initial conditions.

If the risks are instead expressed as the probabilities that the projected numbers would drop below a given number of fish (quasi-extinction), then the risk curves have a similar, but mirrored shape. The probabilities of five consecutive severe declines are much lower than the risk of a single, very low spawning run. For example, under the baseline scenario ($\lambda = 1.31$) with density dependence, there is about a 50 percent risk that the population drops below its initial numbers (145 adults) and stays below that value for five years, and there is about a 32 percent risk that the population similarly drops and stays below our assumed quasi-extinction threshold of 25 adults. In contrast to population trajectory projections wherein by the third generation, the density independent or dependent projections differed markedly, when the baseline versus copper-growth reduction scenarios are compared as relative risks of decline or quasi-extinction, the risk values were mostly similar but slightly higher under the density dependent than independent model either assumptions of density independence or dependence.

Mebane and Arthaud (2010) interpreted the population recovery chances in three ways. First, the most lenient and optimistic statistic was the probability that the population would exceed the simulation model recovery threshold of 500 adults at any one time interval during the simulations. When these probabilities are plotted as a cumulative probability distribution, the

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cumulative distribution of recovery times increases monotonically. Each point on this cumulative curve can be interpreted as there is a Y percent probability that the population abundance will exceed the 500 adult threshold in or before the year 30. Focusing on the medians of the distributions, the relative times to reaching the recovery abundance threshold can be compared between the scenarios. When the population growth was unconstrained by carrying capacity limitations,median times for the population to reach 500 adults were about 12, 17, and 27 years for the baseline, 4 percent length reduction from copper, and 7.5 percent length reduction from copper scenarios, respectively. When the population was constrained below a carrying capacity ceiling of 518 adults in the density dependent model, this nearly precluded the population from reaching a recovery target that was only slightly lower; median times projected for the population to reach 500 adults ranged from 22 years for the baseline to >30 years for the copper-lower and higher effects scenarios.

Second, when considering recovery as a more persistent increase in adult abundances over for five consecutive years, under the density independent scenarios, there were 50 percent probabilities that at least for one period of five-consecutive years at some time during the 30-year simulations, the adult abundances would reach about 420, 260, and 175 for the baseline, copper-lower effects (4 percent length reduction), and copper-higher effect (7.5 percent length reduction) scenarios, respectively. Under the ceiling density dependent scenarios, the adult abundances were similarly projected, with 50 percent probabilities, to reach about 290, 225, and 150 for the baseline and copper-lower or higher effects scenarios, respectively (Figure 2.6.6.1). When the threshold for recovery was defined as exceeding 500 adults for any one five-year period, attaining this recovery threshold within 30-years was unlikely for any modeled scenario, with chances of reaching that threshold ranging from 41 percent to nearly 0 percent across the scenarios (Figure 2.6.6.1).

Summary. The Chinook salmon length reductions estimated for the 1992 copper criterion concentration of about 4 to 7.5 percent were projected to result in 2 to 10 percent additional risk of quasi-extinction sometime in the next 6-generations, depending on the model. The corresponding estimated length reductions for the 2007 updated-EPA copper criterion concentration would range from about zero to 4 percent and would be projected to result in zero to 5 percent additional risk of quasi-extinction sometime in the next 6-generations. Chances of recovery differed more between the baseline and copper exposed scenarios in the density independent model than in the ceiling density dependence model. For instance, there were about 40 to 60 percent reductions, attributable to length reductions of 4 to 7.5 percent, respectively, of the highest population adult abundances projected with 50 percent likelihood of being reached and maintained for 5-years running in the next 6-generations. With the ceiling density dependent model, the reductions were projected to be about 20 to 50 percent from baseline population model, which indicates that the chronic criterion for copper is not likely to be protective of chronic toxic effects.

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Scenario	Density independent projections			Density dependent projections		
	Baseline	4% length reduction scenario	7.5% length reduction scenario	Baseline	4% length reduction scenario	7.5% length reduction scenario
Expected minimum adult abundances (individuals)	46	34	22	36	28	19
Risk of a single severe (90%) decline of adult spawners (CI)	78% (76–81%)	80% (77–83%)	82% (79–85)	79% (76–82%)	80% (77–82%)	82% (79–85%)
Risk of quasi-extinction (<25 adults per year for 5 consecutive years)	30% (25–36%)	35% (30–42%)	40% (34–48%)	33% (27–39)	35% (29–42%)	42% (35–49%)
Probability of recovery to >500 adults for 5 consecutive years	41% (35–49)	14% (11–17%)	4% (3–5%)	6% (5–9%)	3% (2–4%)	0.2% (0.1–0.3%)
Probability of ending abundances >500 adults (CI)[1]	44% (41–46%)	30% (27–33)	21% (18–24%)	21% (18–24%)	15% (12–18%)	8% (5–11%)
Ending abundance, 50% probability of exceeding (CI)[2]	274 (179–357)	112 (74–167)	62 (43–88)	146 (103–190)	83 (59–133)	41 (23–64)

Table notes: Results of 1000 Monte Carlo simulations, simulations were run through 6 generations. CI- 95% Kolmogorov-Smirnov confidence intervals; [1]Probability that the adult abundance will end up greater than the recovery threshold of 500 after six generations; [2]After 6-generations, there are 50% probabilities that the adult abundances will end up greater than these numbers.

Figure 2.6.6.1 Risks of severe population decline or quasi-extinction, probabilities of recovery greater than a given threshold for different copper effects scenarios, using both density dependent and density independent simulation models (Mebane and Arthaud 2010).

2.6.7 Effects on Critical Habitat

The EPA's approval of the proposed criteria has the potential to adversely affect designated critical habitats through direct water-borne toxicity and bioaccumulation, as described below.

Pacific Salmon and Steelhead

1. ***Freshwater Spawning Sites***
   a. Substrate — Sediment contamination by toxic pollutants is likely to adversely affect critical habitat because the particulate forms of toxicants are either immediately bioavailable via discharge, through re-suspension, are a delayed source of toxicity through bioaccumulation, or are available when water quality conditions favor dissolution at a later date. Specifically, contaminated sediments are expected to influence intra-gravel life stages, food sources, and fish through direct ingestion or deposition on the gill surfaces of particulate forms of toxicants.

      Sediments as a source of contaminant exposure were not considered by EPA in the development of the national criteria, which are the same as the criteria proposed by the State of Oregon. The NMFS recognizes that considerable technical and practical problems exist in defining water quality criteria on a sediment basis, and that this is presently the subject of considerable research and debate. Nevertheless, most organic and metal contaminants adsorb to organic particulates and settle out in sediments, so at sites where there have been past discharges, or where there are continuing discharges of contaminants into the water column, they form a long-term repository and a continuing source of exposure that must be addressed if the water quality component of critical habitat is to be protected. Further, although these substances may not readily be transferred into the water column, they may still be available to fish through food chain transfer from their benthic prey, or through ingestion of sediment while feeding. Not having water quality criteria that consider uptake through these routes leaves a route of exposure to fish that the proposed criteria do not address. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *substrate* be adversely affected, and will be degraded at the watershed and designation scales.
   b. Water Quality — Freshwater spawning sites require water quality conditions that support spawning, incubation, and larval development. Based on the distribution and density, the distribution, fate and transport of the compounds listed in Table 1.1, and the distribution of spawning of UWR Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, LCR Chinook salmon, LCR coho salmon, SR SS Chinook salmon, SR fall-run Chinook salmon, SRB steelhead, CR chum salmon, OC coho

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salmon, and SONCC coho salmon, we expect degraded water quality to coincide in time and space with spawning events.

The most severe effects to water quality within spawning sites will be those sites that are located in areas in close proximity to multiple point-source dischargers. Although spawning sites for UWR Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, LCR Chinook salmon, LCR coho salmon, SR SS Chinook salmon, SR fall-run Chinook salmon, SRB steelhead, CR chum salmon, OC coho salmon, and SONCC coho salmon are generally above high density point-source discharges, the downstream effects of low-density pollutant discharges upstream of spawning areas can reduce spawning success. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, and will be degraded at the watershed or designation scales.

c. Water Quantity — No effects are likely to occur.

2. ***Freshwater Rearing***

a. Floodplain Connectivity — No effects are likely to occur.

b. Forage — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *forage* will be adversely affected, but will not be degraded at the watershed or designation scales.

c. Natural Cover — No effects are likely to occur.

d. Water Quality — Freshwater rearing sites need to provide good water quality and abundant forage to support juvenile development. Reductions in either, can limit the existing and potential carrying capacity of rearing sites and subsequently reduce their conservation value.

Recovery of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR

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chum salmon, OC coho salmon, SONCC coho salmon populations is tied closely to the success of juveniles to fully develop, mature, and grow during freshwater residency periods. Collectively, the toxicity data indicate that concentrations of the compounds listed in Table 1.1 are sufficient to adversely affect water quality in affected watersheds, as they do not support the associated life history events, such as fry/parr growth and development, for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, and will be degraded at the watershed and designation scales.

e. Water Quantity — No effects are likely to occur.

3. ***Freshwater Migration Corridors***

a. Forage — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *forage* will be adversely affected, but will not be degraded at the watershed or designation scales.

b. Free of Artificial Obstruction — No effects are likely to occur.

c. Natural Cover — No effects are likely to occur.

d. Water Quality — Freshwater migration corridors need to provide good water quality and abundant forage to support juvenile development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

Collectively, the toxicity data indicate that concentrations of the compounds listed in Table 1.1 are sufficient to adversely affect water quality in affected watersheds, as they do not support the associated life history events, such as smolt growth and development, for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run

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Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, and will be degraded at the watershed and designation scales.

e. Water Quantity — No effects are likely to occur.

4. ***Estuarine Areas***

a. Forage – Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *forage* will be adversely affected, but will not be degraded at the watershed or designation scales.

b. Free of obstruction – No effects are likely to occur.

c. Natural cover –No effects are likely to occur.

d. Water quality — Estuarine areas require good water quality to support juvenile and adult physiological transitions between fresh water and salt water as well as areas to support growth and maturation.

Collectively, the toxicity data indicate that concentrations of the compounds listed in Table 1.1 are sufficient to adversely affect water quality in affected estuarine areas, as they do not support the associated life history events, such as smolt growth and development, for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon. For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water*

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quality will be adversely affected, but will not be degraded at the watershed and designation scales.

5. ***Nearshore Marine Areas***
   a. None designated.
6. ***Offshore Marine Areas***
   a. None designated.

Based on the above assessment, the effects of the proposed action, in particular on the freshwater PCEs *water quality and substrate*, will appreciably diminish the conservation value of critical habitat at the designation scale for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon.

Green Sturgeon

1. ***Freshwater Riverine Systems***
   a. Food resources — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile, sub-adult and adult green sturgeon. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

      Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *food resources* will be adversely affected, but will not be degraded at the designation scale.
   b. Migratory corridor — Freshwater migration corridors need to provide good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

      For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *migratory corridor* will be adversely affected, and will be degraded at the designation scale.

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c. Sediments as a source of contaminant exposure were not considered by EPA in the development of the national criteria, which are the same as the criteria proposed by the State of Oregon. The NMFS recognizes that considerable technical and practical problems exist in defining water quality criteria on a sediment basis, and that this is presently the subject of considerable research and debate. Nevertheless, most organic and metal contaminants adsorb to organic particulates and settle out in sediments, so at sites where there have been past discharges, or where there are continuing discharges of contaminants into the water column, they form a long-term repository and a continuing source of exposure that must be addressed if the water quality component of critical habitat is to be protected. Further, although these substances may not readily be transferred into the water column, they may still be available to fish through food chain transfer from their benthic prey, or through ingestion of sediment while feeding. Not having water quality criteria that consider uptake through these routes leaves a route of exposure to fish that the proposed criteria do not address. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *substrate* be adversely affected, and will be degraded at the designation scale.

d. Substrate type or size — No effects are likely to occur.

e. Water depth — No effects are likely to occur.

f. Water flow — No effects are likely to occur.

g. Water quality — Freshwater riverine systems need to provide good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, and will be degraded at the designation scale.

2. ***Estuarine Systems***

a. Food resources — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which can be expected to reduce fitness, in estuaries where food is a limiting factor.

Changes in species composition can have the same results in fitness and survival. Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to

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juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *food resources* will be adversely affected, but will not be degraded at the designation scale.

b. Migratory corridor — Estuarine migration corridors need to provide good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

   For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *migratory corridor* will be adversely affected, but will not be degraded at the designation scale.

c. Sediments as a source of contaminant exposure were not considered by EPA in the development of the national criteria, which are the same as the criteria proposed by the State of Oregon. The NMFS recognizes that considerable technical and practical problems exist in defining water quality criteria on a sediment basis, and that this is presently the subject of considerable research and debate. Nevertheless, most organic and metal contaminants adsorb to organic particulates and settle out in sediments, so at sites where there have been past discharges, or where there are continuing discharges of contaminants into the water column, they form a long-term repository and a continuing source of exposure that must be addressed if the water quality component of critical habitat is to be protected. Further, although these substances may not readily be transferred into the water column, they may still be available to fish through food chain transfer from their benthic prey, or through ingestion of sediment while feeding. Not having water quality criteria that consider uptake through these routes leaves a route of exposure to fish that the proposed criteria do not address. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *substrate* be adversely affected, and will be degraded at the designation scale.

d. Water flow — No effects are likely to occur.

e. Water depth — No effects are likely to occur.

f. Water quality — Estuarine areas need to provide good water quality and abundant forage to support growth and development.

   For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds

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listed in Table 1.1, the PCE *water quality* will be adversely affected, but will not be degraded at the designation scale.

3. ***Coastal Marine Areas***

a. Food Resources — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which can be expected to reduce fitness, in coastal marine areas where food is a limiting factor.

Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *food resources* will be adversely affected, but will not be degraded at the designation scale.

b. Migratory Corridor — Coastal marine migration corridors need to provide good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

For these reasons, and the available toxicity data, the limited distribution and density of point-source discharges in saltwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *migratory corridor* will be adversely affected, but will not be degraded at the designation scale.

c. Water Quality — Coastal marine areas require good water quality and abundant forage to support growth and development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

Based on the available toxicity data, the distribution and density of point-source discharges in salt water, the limited area of saltwater habitat for green sturgeon within the action area, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PCE *water quality* will be adversely affected, but will not be degraded at the designation scale.

Based on the above assessment, the effects of the proposed action, in particular on the freshwater PCEs *water quality, migratory corridors, and sediment quality* will appreciably diminish the conservation value of critical habitat at the designation scale for green sturgeon.

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Eulachon

1. ***Freshwater Spawning***
   a. Water Flow — No effects are expected to occur.
   b. Water Quality — Freshwater spawning sites require water quality conditions that support spawning, incubation, and larval development. The degradation of water quality by exposure to the stressors of the action is indicated via the toxic responses in a variety of aquatic organisms including listed species. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *water quality* will be adversely affected, and will be degraded at the designation scale.
   c. Water Temperature — No effects are expected to occur.
   d. Substrate — Sediment contamination by toxic pollutants is likely to adversely affect critical habitat because the particulate forms of toxicants are either immediately bioavailable via discharge, through re-suspension, are a delayed source of toxicity through bioaccumulation, or are available when water quality conditions favor dissolution at a later date. Specifically, contaminated sediments are expected to influence intragravel life stages, food sources, and fish through direct ingestion or deposition on the gill surfaces of particulate forms of toxicants.

      Sediments as a source of contaminant exposure were not considered by EPA in the development of the national criteria, which are the same as the criteria proposed by the State of Oregon. The NMFS recognizes that considerable technical and practical problems exist in defining water quality criteria on a sediment basis, and that this is presently the subject of considerable research and debate. Nevertheless, most organic and metal contaminants adsorb to organic particulates and settle out in sediments, so at sites where there have been past discharges, or where there are continuing discharges of contaminants into the water column, they form a long-term repository and a continuing source of exposure that must be addressed if the water quality component of critical habitat is to be protected. Further, although these substances may not readily be transferred into the water column, they may still be available to fish through food chain transfer from their benthic prey, or through ingestion of sediment while feeding. Not having water quality criteria that consider uptake through these routes leaves a route of exposure to fish that the proposed criteria do not address. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *substrate* be adversely affected, and will be degraded at the designation scale.

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2. ***Freshwater Migration***

a. Migratory Corridor — Freshwater migration corridors need to provide good water quality to support larval development. Reductions in either, can limit the existing and potential carrying capacity of migration corridors and subsequently reduce their conservation value.

For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *migratory corridor* will be adversely affected, and will be degraded at the designation scale.

b. Water Flow — No effects are expected.

c. Water Quality — For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *water quality* will be adversely affected, and will be degraded at the designation scale.

d. Water Temperature — No effects are expected.

e. Forage — Based on the data provided in the BE on fish and aquatic invertebrates, the stressors of the action will adversely affect food items for juvenile fishes. Reductions in food quantity can result in reduced calories for rearing and migrating fish, which is likely to reduce fitness, in watersheds where food is a limiting factor.

Biomass quantity is not a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile fishes. Pollution tolerant prey, which could be favored under the proposed action, may also be less palatable to juvenile fishes and therefore reduce actual food availability. For these reasons, and the available toxicity data, the distribution and density of point-source discharges in freshwater, the fate, transport, chemical transformation, and chemical interactions of the compounds listed in Table 1.1, the PBF *forage* will be adversely affected, but will not be degraded at the designation scale.

Based on the above assessment, the effects of the proposed action, in particular on the freshwater PBFs *water quality, substrate, and migratory corridor* will appreciably diminish the conservation value of critical habitat at the designation scale for eulachon.

2.6.8 Cumulative Effects

"Cumulative effects" are those effects of future State or private activities, not involving Federal activities, that are reasonably certain to occur within the action area of the Federal action subject to consultation (50 CFR 402.02). Future Federal actions that are unrelated to the proposed action are not considered in this section because they require separate consultation pursuant to section 7 of the ESA.

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Some types of human activities that contribute to cumulative effects are likely to have adverse effects on listed species and critical habitat PCEs. Many of which are activities occurred in the recent past and had an effect on the environmental baseline. These can be considered reasonably certain to occur in the future because they occurred frequently in the recent past. Within the freshwater portion of the action area, non-Federal actions are likely to include human population growth, water withdrawals (*i.e.*, those pursuant to senior state water rights) and land use practices. In the action area, state, tribal, and local government actions are likely to be in the form of legislation, administrative rules, or policy initiatives, shoreline growth management and resource permitting.

The states of the west coast region, which contribute water to major river systems, are projected to have the most rapid growth of any area in the U.S. within the next few decades. California, Idaho, Oregon, and Washington are forecasted to have double digit increases in population for each decade from 2000 to 2030 (USCB 2005). Overall, the west coast region had a projected population of 72.2 million people in 2010. The U.S. Census Bureau predicts this figure will grow to 76.8 million in 2015 and 81.6 million in 2020.

Although general population growth stems from development of metropolitan areas, growth in the western states is projected from the enlargement of smaller cities rather than from major metropolitan areas. Of the 46 western state metropolitan areas that experienced a 10% growth or greater between 2000 and 2008, only the Portland-Vancouver-Beaverton, OR (1.81% per year) metropolitan area occurs in the action area (USCB 2009).

As these cities border riverine systems, diffuse and extensive growth will increase overall volume of contaminant loading from wastewater treatment plants and sediments from sprawling urban and suburban development into riverine, estuarine, and marine habitats. Urban runoff from impervious surfaces and roadways may also contain oil, heavy metals, PAHs, and other chemical pollutants and flow into state surface waters. Inputs of these point and non-point pollution sources into numerous rivers and their tributaries will affect water quality in available spawning and rearing habitat for salmon. Based on the increase in human population growth, NMFS expects an associated increase in the number of NPDES permits issued and a concomitant increase of pollutant loading.

Mining has historically been a major component of western state economies. With national output for metals projected to increase by 4.3% annually, output of western mines should increase markedly (Figueroa and Woods 2007). Increases in mining activity will add to existing significant levels of mining contaminants entering river basins. Given this trend, we expect existing water degradation in Oregon streams that feed into or provide spawning habitat for threatened and endangered species to be exacerbated.

As the western states have large tracts of irrigated agriculture, a 2.2% rise in agricultural output is anticipated (Figueroa and Woods 2007). Impacts from heightened agricultural production will likely result in two negative impacts on listed species. The first impact is the greater use and application of pesticide, fertilizers, and herbicides and their increased concentrations and entry into freshwater systems. insecticides, and other pollutants from agricultural runoff may further degrade existing fish habitats. Second, increased output and water diversions for agriculture may

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also place greater demands upon limited water resources. Water diversions will reduce flow rates and alter habitat throughout freshwater systems. As water is drawn off, contaminants will become more concentrated in these systems, exacerbating contamination issues in habitats for protected species.

The above non-federal actions are likely to pose continuous unquantifiable negative effects on listed species addressed in this opinion. These effects include increases in sedimentation, increased point and non-point pollution discharges, decreased infiltration of rainwater (leading to decreases in shallow groundwater recharge, decreases in hyporheic flow, and decreases in summer low flows).

Non-federal actions likely to occur in or near surface waters in the action area may also have beneficial effects on listed species addressed in this opinion. They include implementation of riparian improvement measures and fish habitat restoration projects, for example. Coupled with EPA's approval of the proposed water quality standards for aquatic life, the effects from anthropogenic growth on the natural environment will continue to allow toxic discharges to affect and influence the overall distribution, survival, and recovery of listed species in the Columbia River basin and Oregon.

NMFS also expects the natural phenomena in the action area (*e.g.*, oceanographic features, ongoing and future climate change, storms, natural mortality) will continue to influence listed species. Climate change effects are expected to be evident as alterations of water yield, peak flows, and stream temperature. Other effects, such as increased vulnerability to catastrophic wildfires, may occur as climate change alters the structure and distribution of forest and aquatic systems.

Although these factors are ongoing to some extent and likely to continue in the future, past occurrence is not a guarantee of a continuing level of activity. That will depend on whether there are economic, administrative, and legal impediments or safeguards in place. Therefore, although NMFS finds it likely that the cumulative effects of these activities will have adverse effects commensurate with or greater than those of similar past activities; it is not possible to quantify these effects.

2.7 Integration and Synthesis

The Integration and Synthesis section is the final step of NMFS' assessment of the risk posed to species and critical habitat as a result of implementing the proposed action. In this section, we add the effects of the action (section 2.6) to the environmental baseline (section 2.5) and the cumulative effects (section 2.6.8) to formulate the agency's biological opinion as to whether the proposed action is likely to: (1) Result in appreciable reductions in the likelihood of both survival and recovery of the species in the wild by reducing its numbers, reproduction, or distribution; or (2) reduce the value of designated or proposed critical habitat for the conservation of the species. These assessments are made in full consideration of the status of the species and critical habitat (section 2.4).

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This section is comprised of the following: (1) a description of the multiple lines of evidence and effects decision criteria used by NMFS to assess toxicity and fitness consequences, (2) a synthesis of information regarding likely toxicity and environmental effect pathways, species and critical habitat status, cumulative effects and fitness consequences associated with exposure to Oregon's freshwater and saltwater criteria, and (3) ESU/DPS-specific evaluations. These components are described in detail below.

The analysis on multiple lines of evidence and effects decision criteria provides a breakdown of the significance of the likely effects of each criterion based on the analysis of the freshwater and saltwater toxicity data, an overview of how the toxicity data factor into our effect determinations, and a description of how NMFS applied the results of the direct mortality population modeling. The synthesis of information on acute and chronic endpoints, environmental stressors, species and critical habitat status, cumulative effects, and fitness consequences is a qualitative risk assessment for each criterion that considers endpoint-effects on listed species, risks associated with exposure to chemical mixtures, results of the direct mortality population modeling, and threats associated with interactions of the criteria with environmental baseline stressors. The ESU/DPS-specific evaluations analyze how the proposed action affects population attributes, species viability, and the conservation value of critical habitat.

Legacy Compounds.

In 1987 the EPA banned all uses of dieldrin. In 2010 EPA took action to eliminate all uses of endosulfan in the U.S., with a complete phase-out scheduled by 2016. In 1986 the EPA banned production of endrin in the U.S. In 1988 EPA banned the use of heptachlor epoxide except for limited use for fire ant control in underground transformers. In 2006 EPA issued final orders cancelling pesticide products containing lindane. However, the Food and Drug administration permits the use of lindane in pharmaceutical products to control lice and scabies. The NMFS does not expect population-level adverse effects to listed species considered in this opinion from exposure to any of the six legacy criteria (*i.e.*, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, heptachlor epoxide, and lindane,) as their use is either prohibited by law or highly restricted.

(1) Multiple Lines of Evidence and Effects Decision Criteria.

The foremost line of evidence applied in NMFS' effects decision is the criterion-specific toxicity data. The NMFS coupled this toxicity data analysis with the summary analysis, the chemical mixtures analysis, the direct mortality population modeling, and exposure to baseline chemical stressors. The NMFS then used this information used to assess the risk associated with exposure to the compounds in Table 1.1 on each of the affected species considered in this opinion.

To examine the significance of the effects of all freshwater criteria, NMFS ran the acute criteria (for all chemicals) and chronic criteria (for ammonia, cadmium, and copper only) through a direct mortality population model (see section 2.6.5 and Appendix 3) to evaluate the magnitude of the effects of juvenile mortality on productivity for the salmonid fish species considered in this opinion. The NMFS also examined the available toxicity data on ammonia, cadmium, and copper for inclusion in a somatic growth model to assess changes in fry growth that would affect

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population growth rates, but the available data for these compounds could not be translated into appropriate input parameters for this model (see Appendix 3). Therefore, NMFS relied on the chronic toxicity data analysis for determining the risks of growth impairment and other sublethal effects associated with the chronic criteria and the significance of those risks to the listed species considered in this opinion.

The NMFS applied the results of the direct mortality population model as secondary line of evidence to assess the potential impact that EPA's approval of the numeric criteria would have on species' productivity. The NMFS applied the modeling results to the effects analysis in the following manner:

1. For compounds where all four modeling scenarios (described above in section 2.6.5.1) predicted a measurable level of mortality with a resulting change in λ (except for the legacy compounds), then NMFS considered these compounds to have a very high probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

2. For compounds where three of the four modeling scenarios predicted a level of mortality with a resulting change in λ (except for the legacy compounds), NMFS considered these compounds to have a high probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

3. For compounds where two of the four modeling scenarios predicted a level of mortality with a resulting change in λ (except for the legacy compounds), NMFS considered these compounds to have a moderate-to-high probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

4. For compounds where one of the four modeling scenarios predicted a level of mortality with a resulting change in λ (except for the legacy compounds), NMFS considered these compounds to have a moderate probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

5. For compounds where none of the four modeling scenarios predict a level of mortality, NMFS considered these compounds to have a low probability to appreciably reduce productivity such that the species' survival and recovery would be at increased risk.

These results of the direct mortality population model were then integrated into the primary lines of evidence in the opinion—the acute toxicity data, chronic toxicity data, the analysis on the shortcomings and implications of laboratory-derived toxicity tests, the relative percent mortality analysis, and the mixtures analysis—to determine which compounds result in the highest-intensity of acute and/or chronic toxic effects on the listed species considered in this opinion. As part of this integration, NMFS also considered the exposure scenario and the magnitude of the change in λ when assessing which compounds were associated with significant adverse toxicological and biological effects.

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Depending upon the modeling scenario for the legacy compounds, the direct mortality modeling predicted a negative percent change in λ. However, since the legacy compounds are either prohibited by law or highly restricted, NMFS considered that these compounds would be unlikely to appreciably reduce productivity and abundance such that the listed species' survival and recovery would not be at increased risk as water surface concentrations of these compounds will continue to decrease in the long term.

NMFS used the salmonid fishes toxicity data as a surrogate for green sturgeon and eulachon, as toxicity data for these two species was limited or non-existent, and because the salmonid fishes toxicity data sets were the best taxonomic data available (green sturgeon, eulachon, and salmonid fishes are in the same superorder: *Protacanthopterygii*). However, differences in the life history strategies and the certainty of similar toxic effects among species for all mechanisms and modes of action is not evident in the literature, so the results of the direct mortality population analysis for the salmonid fishes do not necessarily apply to green sturgeon and eulachon. Nonetheless, NMFS gives the benefit of the doubt to the listed species, and, based on the evidence considered in this opinion, NMFS expects that the stressors of the action to result in mortality (albeit an unquantifiable amount) of green sturgeon and eulachon. We further expect, based on the toxicity data, that the fitness of green sturgeon and eulachon will be reduced via sub-lethal effects (*i.e.*, interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis).

(2) Summary analysis on acute and chronic endpoints, chemical mixtures, population modeling, interactions with baseline environmental stressors, and fitness consequences associated with exposure to the proposed freshwater and saltwater criteria.

The summary analysis is a qualitative assessment of likely fitness consequences due to approval and implementation of each proposed criterion that considers:

- Acute and chronic toxicity data for the criteria compounds to listed species.
- The likelihood that listed species will encounter mixtures of multiple criteria chemicals in mixing zones due to the typical presence of these mixtures in wastewater and stormwater discharges under NPDES permits.
- The likelihood that listed species will encounter chemicals at concentrations greater than criteria concentrations due to overlapping mixing zones in some areas, and to environmental baseline stressors that add to the exposures.
- Results of the direct mortality population model
- The likely effects of interactions of the criteria compounds with other environmental baseline stressors (*e.g.*, high water temperature, other toxic substances)

The results of the summary analysis are given in Tables 2.7.1 and 2.7.2.

The summary analysis assesses the overall effects of approving the compounds listed in Table 1.1, individually and in combination with each other and with environmental baseline stressors, on the listed species considered in this opinion. In the summary analysis, we did not add up or otherwise mathematically combine its components. Rather, we applied best professional

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judgment to characterize the intensity of adverse effects on individuals and populations of the listed species. We took this approach in large part because the available toxicity data for each compound varies significantly by quantity, test method, water source, life stage, *etc*. Therefore, we were not able to generate a mathematical expression or hazard quotient in the summary analysis, but did apply the qualitative results in the *Integration and Synthesis*.

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Table 2.7.1. Results of the summary analysis on acute and chronic endpoints, chemical mixtures, environmental stressors, and fitness consequences associated with exposure to Oregon's freshwater criteria (empty cells = no data).

Stressor	Mortality	Growth	Behavioral	Cellular	Physiological	Biochemical	Reproductive	Sublethal	Bioaccumulation	Chemical Mixtures	Criteria Interactions with Environmental Baseline Stressors
Compound											
Aluminum	++++	+++	+++	++	+++					+++	+++
Ammonia	++++	++++		++	++++	++++				+++	+++
Arsenic	++	++	+		+					+++	+++
Lindane	++									+	+
Cadmium	++++	+++	++		++		++			+++	+++
Chromium (III)	++	+++								+++	+++
Chromium (VI)	+	+++								+++	+++
Copper	++++	++++	++++	++	+++		++++	++++		+++	+++
Dieldrin	++	++			++		+	+		++	++
Endosulfan-alpha	+++							++		+	+
Endosulfan-beta	+++							++		+	+
Endrin	+++			+	+		+			+	+
Heptachlor Epoxide	++							+		+	+
Lead	++	++	+++	+++	++	++	+			+++	+++
Nickel	++	+++								+++	+++
Pentachlorophenol	++	++								+++	+++
Selenium	++	++		+					+++	+++	+++
Silver	+++	++						++		+++	+++
Tributyltin	+++	++		++	++					+++	+++
Zinc	+++	+++		++	++		+++			+++	+++

+ Low intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals
++ Moderate intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals
+++ Moderately-high-intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals, but not at the scale of any population
++++ High-intensity increase in toxicity effects on listed species that affects one or more population attributes

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Table 2.7.2. Results of the summary analysis on acute and chronic endpoints, chemical mixtures, environmental stressors, and fitness consequences associated with exposure to Oregon's saltwater criteria (empty cells = no data).

Stressor	Mortality	Growth	Behavioral	Cellular	Physiological	Biochemical	Reproductive	Sublethal	Chemical Mixtures	Criteria Interactions with Environmental Baseline Stressors
Compound										
Arsenic	++							++	++	+++
Cadmium	++							++	++	+++
Chromium (VI)	++	+++							++	+++
Copper	++						++		++	+++
Endosulfan-alpha	++						+		+	+
Endosulfan-beta	++						+		+	+
Heptachlor Epoxide	+++							+	+	+
Lead	+++				+++		+		++	+++
Nickel	++							++	++	+++
Pentachlorophenol								+++	+	+++
Selenium	++							++	++	+++
Silver	++								++	+++
Tributyltin	++			++	+			++	++	+++
Zinc	++						++		++	+++

+ Low intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals
++ Moderate intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals
+++ Moderately-high-intensity increase in toxicity effects on listed species at the scale of individuals or groups of individuals, but not at the scale of any population
++++ High-intensity increase in toxicity effects on listed species that affects one or more population attributes

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(3) ESU/DPS-Specific Evaluations

The ESU/DPS-specific evaluations are an integration of the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model (when applicable), and the summary analysis. For each ESU or DPS, the evaluations are partitioned into six parts: (1) a summary of the acute and chronic toxicity data analysis on each species considered in this opinion, (2) a summary of the results of the direct mortality population model (when applicable), (3) an explanation of how effects of the proposed action are likely to affect productivity and abundance from multiple stressors, (4) a summary of how reductions in productivity and abundance are likely to affect the population attributes spatial structure and genetic diversity (when applicable), (5) a summary of effects associated with the freshwater and saltwater criteria that are likely to adversely affect critical habitat (when applicable) within the action area, and (6) conclusions on the listed species and critical habitat.

Furthermore, based on the summary analysis that we described earlier, certain compounds proposed by EPA are likely to have significant (high-intensity toxicological effects), long-term negative effects on one or more population attributes for the listed species considered in this opinion (Tables 2.7.1 and 2.7.2).

LCR Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; LCR Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 32 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all four modeling scenarios for each of the 32 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to

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baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect LCR Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for LCR Chinook salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity for LCR Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for LCR Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of LCR Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (40.2 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of LCR Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of LCR Chinook salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UWR Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests

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(uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; UWR Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 7 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 7 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect UWR Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for UWR Chinook salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UWR Chinook salmon through multiple mechanisms, including including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of UWR Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for UWR Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances,

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and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of UWR Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of UWR Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of UWR Chinook salmon critical habitat such that it will not retain the current ability for the PCE water quality to serve the intended conservation role for the species for either survival or recovery.

UCR Spring-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; UCR spring-run Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 4 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 4 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect UCR spring-run Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for UCR spring-run Chinook salmon.

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(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UCR spring-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of UCR spring-run Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for UCR spring-run Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of UCR spring-run Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (30.8 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of UCR spring-run Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of UCR spring-run Chinook salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Spring/Summer-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SR SS-run Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

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(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 27 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 27 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SS-run Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for SS-run Chinook salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SS-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SS-run Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR SS-run Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SR SS-run Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE

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water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (25.3 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery for SR SS-run Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SR SS-run Chinook salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Fall-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SR fall-run Chinook salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for the single SR fall-run Chinook salmon ESU (which consists of eight spawning populations). The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for the single SR fall-run Chinook salmon ESU (which consists of eight spawning populations).

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SR fall-run Chinook salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for SR fall-run Chinook salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat

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quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SR fall-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SR fall-run Chinook salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR fall-run Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SR fall-run Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (25.3 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of SR fall-run Chinook salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SR fall-run Chinook salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

CR Chum Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; CR chum salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to

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the freshwater acute criteria (one compound at a time). The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 17 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 17 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect CR chum salmon, and is likely to appreciably affect the VSP parameters productivity and abundance diversity of CR chum salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of CR chum salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of CR chum salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for CR chum salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of CR chum salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This

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is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (26 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of CR chum salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of CR chum salmon critical habitat such that it will not retain the current ability for the PCE water quality to serve the intended conservation role for the species for either survival or recovery.

LCR Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; LCR coho salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 27 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 27 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect LCR coho salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for LCR coho salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in

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abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of LCR coho salmon.

(5) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of LCR coho salmon.

SONCC Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SONCC coho salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 42 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 42 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SONCC coho salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for SONCC coho salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat

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quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SONCC coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SONCC coho salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SONCC coho salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SONCC coho salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (37.8 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of SONCC coho salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SONCC coho salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

OC Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; OC coho salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to

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the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 56 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 56 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect OC coho salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for OC coho salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of OC coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of OC coho salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for OC coho salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of OC coho salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation).

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(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of OC coho salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of OC coho salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Sockeye Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SR sockeye salmon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile salmon exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for the single SR sockeye salmon population. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for the single SR sockeye salmon population.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SR sockeye salmon, and is likely to appreciably affect the VSP parameters productivity and abundance for SR sockeye salmon.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SR sockeye salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of

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environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SR sockeye salmon.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR sockeye salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SR sockeye salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (34.5 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of SR sockeye salmon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SR sockeye salmon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

LCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; LCR steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 26 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 26 populations.

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(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect LCR steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for LCR steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of LCR steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for LCR steelhead. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of LCR steelhead. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (33 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of LCR steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of LCR steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

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UWR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; UWR steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 5 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 5 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect UWR steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for UWR steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UWR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of UWR steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR fall-run Chinook salmon.

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Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of UWR steelhead. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of UWR steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of UWR steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

MCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; MCR steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 17 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 17 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream

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temperatures), the proposed action is likely to adversely affect MCR steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for MCR steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of MCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of MCR steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SR fall-run Chinook salmon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SR fall-run Chinook salmon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (75.7 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of MCR steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of MCR steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; UCR steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the

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concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 4 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 4 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect UCR steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for UCR steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of UCR steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for UCR steelhead. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of UCR steelhead. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in

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particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (30.8 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of UCR steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of UCR steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SRB Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; SRB steelhead will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria (one compound at a time). Based on the direct mortality population modeling results, juvenile steelhead exposed to aluminum, ammonia, arsenic, lindane, cadmium, copper, dieldrin, endosulfan-alpha, endosulfan-beta, endrin, nickel, pentachlorophenol, selenium, silver, tributyltin, and zinc are predicted to result in mortality at the population level—relative to the baseline population model. The level of mortality will result in negative changes in the median population growth rate (λ) for each of the 24 populations. The direct mortality population modeling on chromium (III), chromium (VI), heptachlor epoxide, and lead predicted zero percent mortality for all modeling scenarios for each of the 24 populations.

(3) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, the direct mortality population model, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect SRB steelhead, and is likely to appreciably affect the VSP parameters productivity and abundance for SRB steelhead.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in

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abundance can negatively affect the spatial distribution and/or the genetic diversity of SRB steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, these effects, combined with changes in productivity and abundance, are likely to adversely affect the VSP parameters spatial distribution and genetic diversity of SRB steelhead.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for SRB steelhead. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of SRB steelhead. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events, in particular fry/parr/smolt growth and development) at the watershed and designation levels. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (34.5 percent of the total designation).

(6) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of SRB steelhead. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of SRB steelhead critical habitat such that it will not retain the current ability to serve the intended conservation role for the species' for either survival or recovery.

Green Sturgeon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, and the summary analysis; green sturgeon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect green sturgeon, and is likely to appreciably affect the productivity and abundance for green sturgeon.

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(3) The NMFS expects the stressors of the action to result in unquantifiable mortality of green sturgeon, and affect green sturgeon fitness via sub-lethal effects (*i.e.*, interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis).

(4) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for green sturgeon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of green sturgeon. In particular, the PCE water quality is unlikely to remain functional (*i.e.*, support associated life history events at the designation level. This is based on the magnitude of likely effects on the PCE water quality (high-intensity increase in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (10.4 percent of the total designation).

(5) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of green sturgeon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of green sturgeon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

Eulachon.

(1) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, and the summary analysis; eulachon will suffer acute and chronic toxic effects from exposure to the compounds listed in Table 1.1 at the concentrations that EPA proposes to approve. We summarize the evidence for these effects and describe their significance below.

(2) Based on the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis), the relative percent mortality analysis, the chemical mixtures analysis, and the summary analysis; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures), the proposed action is likely to adversely affect eulachon, and is likely to appreciably affect the productivity and abundance for Eulachon.

(3) The NMFS expects the stressors of the action to result in unquantifiable mortality of Eulachon, and affect eulachon fitness via sub-lethal effects (*i.e.*, interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis).

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(4) In the long term, approval and implementation of the numeric criteria listed in Table 1.1 will incrementally improve water quality conditions for eulachon. Nonetheless, based on the analysis of the acute and chronic toxicity data, the critical habitat analysis, the chemical mixtures analysis, and the summary analysis, the proposed action would allow continued toxic discharges to alter water chemistry, increase mass loading of toxic substances, and reduce habitat quality. Based on our analysis of the acute and chronic toxicity data, and the fate, transport and chemical interactions of the criteria compounds, the proposed action is likely to appreciably reduce the conservation value of the critical habitat of eulachon. In particular the PBF water quality, is unlikely to remain functional, *i.e.*, support associated life history events, at the designation level. This is based on the magnitude of likely effects on the PBF water quality (high-intensity increase in toxicity that affects one or more PBFs) and the overall percentage of critical habitat for this species that would be adversely affected (53.9 percent of the total designation).

(5) After considering all the information in this opinion, NMFS concludes that the proposed action is likely to reduce appreciably the likelihood of both the survival and recovery of eulachon. Furthermore, NMFS concludes that the proposed action is likely to reduce appreciably the conservation value of Eulachon critical habitat such that it will not retain the current ability to serve the intended conservation role for the species for either survival or recovery.

Synthesis

Even though our predicted outcomes regarding the survival and recovery of the listed species considered in this opinion, as well the conservation value of their critical habitats, is based on the effects of the proposed action as a whole, our analysis is structured such that the proposed numeric criteria with the highest-intensity adverse toxicological and adverse biological effects on the listed species can be separated and identified. The multiple lines of evidence used in our analysis to identify the numeric criteria with the highest-intensity adverse toxicological and adverse biological effects include: the compound-specific acute and chronic toxicity data analyses; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis); the relative percent mortality analysis; the chemical mixtures analysis; the direct mortality population model; and the summary analysis. Table 2.7.3 provides a summary of the relative percent mortality analysis in section 2.6. Table 2.7.4 then provides a list of the proposed criteria that are likely to cause the highest-intensity adverse toxicological and adverse biological effects. Table 2.7.4 also shows which compounds, individually and in combination with other compounds and environmental stressors, are likely to reduce appreciably the likelihood of both the survival and recovery of the listed species, or reduce appreciably the conservation value of their critical habitat.

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Table 2.7.3. Relative percent mortality analysis summary for freshwater acute criteria.

Compound	Median LC_{50}
Chromium VI	0.01
Pentachlorophenol	0.09
Lead	0.5
*Dieldrin	0.7
Arsenic	0.7
Nickel	1
*Lindane	1.5
*Heptachlor Epoxide	1.6
Selenium	1.8
Chromium III	3
Silver	3.4
Tributyltin	4.9
Zinc	5.1
*Endrin	5.4
Copper	7
Ammonia	8.6
Cadmium	12.7
*Endosulfan-alpha	13.9
*Endosulfan-beta	13.9
Aluminum	15

*Legacy compounds.

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Table 2.7.4. Findings as to whether compounds associated with significant adverse toxicological and biological effects on the listed species considered in this opinion that, individually and in combination with exposure to multiple compounds and stressors, are likely to reduce appreciably the likelihood of both the survival and recovery (S/R), and are likely to reduce appreciably the conservation value (CV) of their critical habitat.

Stock	Cadmium (Acute)	Aluminum (Acute and Chronic)	Ammonia (Acute and Chronic)	Copper (Acute and Chronic)
LCR Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
UWR Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
UCR spring-run Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SR spring/summer-run Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SR fall-run Chinook Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
CR Chum Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
LCR Coho Salmon	S/R	S/R	S/R	S/R
SONCC Coho Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
OC Coho Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SR Sockeye Salmon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
LCR Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
UWR Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
MCR Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
UCR Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SRB Steelhead	S/R and CV	S/R and CV	S/R and CV	S/R and CV
Green Sturgeon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
Eulachon	S/R and CV	S/R and CV	S/R and CV	S/R and CV
SR Killer Whales	*S/R determination is based on a long-term, permanent reduction in primary prey—Chinook salmon			

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2.8 Southern Resident Killer Whales—Effects Analysis

The best available information indicates that salmon are the primary prey of Southern Residents year round (Section 2.4), including in coastal waters, and that the whales predominantly consume Chinook salmon, likely including Oregon salmon stocks. Based on coded wire tag recoveries, Oregon salmon stocks are available to Southern Residents across their coastal range (Weitkamp 2010). The proposed action has the potential to affect Southern Residents indirectly by reducing prey quality, increasing toxic chemicals in the whales, and reducing availability of Chinook salmon. A decrease in the quality and availability of salmon, and Chinook salmon in particular, and an increase of toxic chemicals in individual whales, may adversely affect the entire DPS of Southern Resident killer whales.

In this analysis, NMFS considers effects of the proposed action on the Southern Residents by qualitatively evaluating the reduction of prey quality caused by the action as well as the potential accumulation of toxic chemicals in the whales, and the reduction of prey availability.

Effects of Reduced Prey Quality and Toxic Chemical Accumulation in the Southern Resident Killer Whales

The NMFS anticipates increased contaminant loading in Chinook salmon, as described above, and therefore also anticipates reduced prey quality and subsequent toxic chemical accumulation in the Southern Residents. First, we briefly review the mechanisms for reduced prey quality and then discuss the anticipated resulting accumulation of toxic chemicals in the whales.

Reduced Prey Quality

The quality of Chinook salmon is likely influenced by a variety of factors including size of the fish and the contaminant load. In addition to the anticipated fish mortality (as described in section 2.6.5), some toxic chemicals can cause sub-lethal effects such as a reduction in growth, a common stress response observed in fish (review in section 2.6.7). Because Southern Residents consume mostly large Chinook salmon (review Status of the Species), a reduction in fish growth could affect the foraging efficiency of Southern Resident killer whales. However, the degree to which reduced fish growth could affect Southern Resident foraging is unknown. When compared to current conditions, approval of the proposed criteria will result in reduced pollutant loading and reduced body burden of contaminants in fishes. Nonetheless, the proposed water quality standards will continue to increase mass loading of toxic substances in the Southern Residents' primary prey with implications for toxic chemical accumulation in the whales, as discussed below.

Toxic Chemical Accumulation in the Southern Residents

The NMFS evaluated the effects of toxic chemical accumulation qualitatively. We reviewed the best available information about the bioaccumulation, biomagnification, concentration levels in the whales, and toxicity of the compounds in Table 1.1 (as introduced earlier), which are: aluminum, ammonia, arsenic, cadmium, chromium (III and VI), copper, dieldrin, endosulfan

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(alpha and beta), endrin, heptachlor epoxide, lead, lindane, nickel, PCP, selenium, silver, TBT, and zinc.

In many cases the best available information was limited. For example, there is limited information about the levels of these compounds in the environment or in the whales, and no information about chemical toxicity specifically in Southern Residents. Where there was no data on chemical levels in Southern Residents, we considered levels in other marine mammals to estimate the potential extent of bioaccumulation in the Southern Residents. This literature review helped us put in context the potential killer whale health effects from the proposed water quality criteria. First, we identified the compounds in Table 1.1 that were not anticipated to cause adverse health effects in the Southern Residents. Second, we identified the compounds in Table 1.1 that may cause adverse health effects in the Southern Residents.

Compounds with No Anticipated Health Effects. The available data indicate that Southern Residents are not at risk of health effects from aluminum, ammonia, nickel, selenium, silver, zinc, and PCP. Some of these compounds are essential elements to the nutrition of marine mammals (*e.g.*, aluminum, nickel, selenium, and zinc; Das *et al.* 2003) and are generally found in low levels in marine mammals distributed throughout the world's oceans (see Appendices 10-5 to 10-8 in O'Shea 1999 for summaries of selected surveys of metals and trace element concentrations in tissues of seals, sea lions, toothed whales, baleen whales, sea otters, dugongs, manatees, and polar bears). Therefore, these essential elements found in low concentrations in marine mammals distributed globally are not anticipated to cause adverse health effects for Southern Resident killer whales. Although silver is not considered an essential element for mammals, its toxicity is generally not a concern and it has not been measured often in marine mammals (O'Hara *et al.* 2003). Ammonia does not build up in the food chain, but serves as a nutrient for plants and bacteria (EPA 2003) and is not anticipated to accumulate in the whales. PCP is an organochlorine pesticide that does not readily bioaccumulate. When found in marine mammals, its presence is likely the result of biotransformation of other chemicals and not bioaccumulation (*e.g.*, as observed in bowhead whales, Hoekstra *et al.* 2003). Furthermore, PCP readily degrades in the environment and by all available evidence does not appear to biomagnify (Garrett and Ross 2010). The NMFS does not anticipate that the proposed action will affect accumulation of PCPs in Southern Residents. For these reasons, NMFS does not anticipate that the proposed action will result in any health effects from these compounds and we do not discuss these compounds further.

Compounds that May Cause Adverse Health Effects. In order to evaluate effects of these remaining compounds, we first review the current levels measured in the blubber of Southern Residents (or in surrogate marine mammals if data are unavailable for Southern Residents), and compare levels to health effect thresholds found for surrogate species. We then consider the effects the proposed criteria will have on the whales' levels over time.

Long-lived, upper trophic-level predators, such as the Southern Residents, are susceptible to compounds that biomagnify because even low concentrations in the prey can accumulate and magnify to high concentration levels in the predators. Bioaccumulative compounds that have the potential to biomagnify are likely to pose the greatest health risks to the Southern Residents. Therefore, we evaluate the effects of compounds that may bioaccumulate but are not anticipated

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to biomagnify separate from the compounds that may bioaccumulate and biomagnify. These steps are described in more detail below: (1) identify the compounds that may bioaccumulate (or increase in concentration in an individual) but are not anticipated to biomagnify (or not anticipated to increase in concentration up the food chain), (2) identify the compounds that may bioaccumulate and biomagnify, and compare the concentrations of these compounds in the Southern Residents or in surrogate species to known health effects levels in surrogate species, and (3) put the effects of the proposed action in context by comparing the existing numeric criteria with the proposed numeric criteria, and evaluating the anticipated trend in the Southern Residents' long-term bioaccumulation.

Compounds that may bioaccumulate but are not anticipated to biomagnify. Metals can bioaccumulate in the aquatic environment (EPA 2007). However, most metals (with the exception of methylmercury), do not appear to biomagnify and are regulated and excreted (Gray 2002, EPA 2007). As discussed in section 2.6.1., arsenic, cadmium, chromium, copper, and lead do not appear to biomagnify. Therefore, NMFS anticipates that these metals will not biomagnify in the Southern Residents.

Upper trophic-level predators can still accumulate metals even in the absence of biomagnification (Reinfelder *et al.* 1998). However, low levels of arsenic, chromium, copper, and lead have been measured in marine mammal tissues (O'Shea 1999, Grant and Ross 2002, Das *et al.* 2003). Although high cadmium levels are measured in some marine mammals, cadmium is known to combine with metallothionein (a protein molecule) to mitigate the toxic effects (Dietz *et al.* 1998, Klaassen *et al.* 2009). Further, no toxic effects of cadmium have been observed in marine mammals. Although threshold levels at which adverse health effects occur are currently unknown for these metals, the available data indicate that the low levels measured in their tissues do not pose a health risk to marine mammals (O'Shea 1999).

Compounds that may bioaccumulate and biomagnify. The remaining compounds with proposed criteria are the organic pollutants that have the ability to biomagnify up the food chain. These compounds are dieldrin, endosulfan, endrin, heptachlor epoxide, lindane, and TBT. The best available data indicate that Southern Residents (or surrogate species) have relatively low concentration levels of these compounds (see the Status of the Species). In contrast, the Southern Residents have higher levels of the legacy organochlorines, PCBs and DDTs, and the emerging PBDEs[9].

At certain concentrations, dieldrin, endosulfan, endrin, heptachlor epoxide, lindane, and TBT can have a wide variety of toxic effects on organisms including neurotoxicity, reproductive defects, tremors and convulsions, organ tissue damage (*e.g.*, liver or kidney tissue damage), cancer, endocrine disruption, and reduced immune response (see the Status of the Species). Here we compare the concentrations of these compounds in the Southern Residents or in surrogate species to known threat levels found in surrogate species. There are currently no known killer whale-specific health effects thresholds, thereby requiring the use of surrogate species to estimate risks. There are several different types of threat levels or measures of toxicity used in laboratory studies. A median lethal dose, LD_{50}, is the dose required to kill half the tested population in 2 weeks and generally indicates a substance's acute toxicity. In contrast, a Lowest Observable

[9] PCBs, DDTs, and PBDEs are not among the proposed criteria in the current action.

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Adverse Effect Level (LOAEL) is the smallest dose that causes a detectable adverse effect typically measured when assessing chronic toxicity. Additionally, a No Observable Adverse Effect Level (NOAEL) is the highest dose at which no adverse effects occur. Dieldrin, endosulfan, endrin, heptachlor epoxide, lindane, and TBT levels in Southern Residents and surrogate marine mammals are below the threat levels (*e.g.*, LD_{50}, NOAEL, LOAEL) in laboratory species from different studies identified in Table 2.8.1. For example, alpha endosulfan levels determined in the blubber of Southern Residents were below the limits of quantification (< 2.2 - < 14 ng/g wet weight). This average level is substantially below the NOAEL found for rats and grey partridge at 2,400 to 40,000 ng/g wet weight, respectively (see Table 2.6.9.1). Therefore, we anticipate that the Southern Residents' current levels of these compounds do not pose a health threat to the whales.

Table 2.8.1 Measured concentration levels in marine mammals compared to threat levels found in laboratory species.

	Current Levels		**Threat Levels**		
Compound	Measured Concentration/Species (ng/g wet weight)	Reference	Concentration (ng/g wet weight)	Species	Reference
Dieldrin	9.2 – 440 / Southern Residents	1	25,000 - 168,000	2 week-old rats	7
Endosulfan	< 2.2 - < 14 / Southern Residents	1	40,000	grey partridge	8; 9
			2,400	rat	10; 9
Endrin	ND - 12.7 (µg/g lipid) / blue and humpback whales	2	25	dog	11
Heptachlor epoxide	5.3 – 660 / Southern Residents	1	195,000-250,000 (ng/g bw)	rat	12
Lindane	< 1.9 – 17 / Southern Residents	1	0.3 ng/g/day	rat	13
TBT	100/killer whales	3	>10,000	Dall's porpoise	14
	180/ killer whales	4	> 120	rat* and rabbit**	15*; 16**
PCB	1,306 -39,420 / Southern Residents	5, 6	100-200 (dietary NOAEL & LOAEL)	seals and dolphins	17
DDT	426 - 35,040 / Southern Residents	5, 6	50,000 ng/g/day	mallard	18
PBDE	199 -2,745/ Southern Residents	5, 6	170-460 ng/g lw in blubber	grey seal	19

ND = non detect, lw = lipid wet References: (1) G. Ylitalo NWFSC, pers. comm.; (2) Metcalfe *et al.* 2004; (3) Kannan *et al.* 1997; (4) Tanabe *et al.* 1998; (5) Krahn *et al.* 2007a; (6) Krahn *et al.* 2009; (7) EPA 2003; (8) Sample *et al.* 1996; (9) Small and Solomon 2005; (10) USEPA 2005, as cited in Small and Solomon 2005; (11) FAO/WHO 1971; (12) Heptachlor epoxide fact sheet CAS Number: 1024-57-3; (13) USEPA 1999; (14) Kim *et al.* 1998; (15) Snoeij *et al.* 1986; (16) Elferink *et al.* 1986; (17) Kannan *et al.* 2000; (18) Tucker and Crabtree 1970) ; (19) Hall *et al.* 2003.

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Comparison Between Existing Criteria and Proposed Criteria and the Resulting Trend in Long Term Accumulation in Southern Residents

In this section, we put the effects of the proposed action in context by comparing the existing numeric criteria with the proposed numeric criteria (see Table 2.8.1), and evaluating the resulting trend in long term bioaccumulation in the Southern Residents. As discussed above, several compounds (*i.e.*, arsenic, cadmium, chromium, copper, and lead) are not anticipated to biomagnify, are likely to be low in concentration in the Southern Residents, and are not currently toxic. The proposed numeric criteria for arsenic, cadmium, and chromium (III) are likely to result in less accumulation in the Southern Residents than with the existing numeric criteria (see Table 2.8.2). The proposed numeric criteria for chromium (VI) will not change from the existing criteria, and therefore we assume the accumulation of chromium (VI) in the whales will remain the same. Lastly, the proposed criteria for copper and lead are more strict for freshwater and less strict for saltwater. Given that copper and lead are not likely to biomagnify, we do not anticipate that a small increase of these compounds in saltwater will cause a measurable increase in concentration in the whales. Therefore, we anticipate that approval of the proposed criteria for these compounds will not increase the potential for adverse health effects in the whales.

The proposed numeric criteria for the bioaccumulative compounds that biomagnify (*e.g.*dieldrin, endosulfan, endrin, heptachlor epoxide, lindane, and TBT) are likely to result in less accumulation than with the existing numeric criteria (see Table 2.8.2). For example, several of these compounds (*e.g.*, endosulfan, heptachlor epoxide, and TBT) were previously unregulated. Although dieldrin and endrin have both more strict and less strict proposed criteria, the exposure of dieldrin and endrin will be from past usage since they have been banned for 20 to 30 years. Dieldrin and endrin could theoretically be in surface waters, however, occurrence will be very minimal as these compounds strongly adhere to sediment (as previously discussed). Overall, accumulation of these compounds will be either reduced, or the same, and is not a health concern. Therefore, we anticipate that approval of the proposed criteria for these compounds will either not change accumulation or potential health effects or, in some cases may reduce accumulation and the risk of health effects in the whales.

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Table 2.8.2. Resulting accumulation in the Southern Resident killer whales from the proposed changes in the numeric criteria.

Compound	Change in Criteria				Accumulation in Whales
	Freshwater		Salwater		
	Acute	Chronic	Acute	Chronic	
Arsenic	decrease	decrease	same	same	decrease
Cadmium	decrease	decrease	decrease	decrease	decrease
Chromium (III)	decrease	decrease			decrease
Chromium (VI)	same	same	same	same	same
Copper	decrease	decrease	increase	increase	same
Dieldrin	decrease	increase			decrease
Endosulfan (-a,-b)	prev. unreg.	prev. unreg.	prev. unreg.	prev. unreg.	decrease
Endrin	decrease	increase			decrease
Heptachlor epoxide	prev. unreg.	prev. unreg.	prev. unreg.	prev. unreg.	decrease
Lead	decrease	decrease	increase	increase	same
Lindane	decrease				decrease
TBT	prev. unreg.	prev. unreg.	prev. unreg.	prev. unreg.	decrease

In summary, when compared to current conditions, the proposed criteria will result in reduced bioaccumulation and biomagnification in the Southern Residents. Based on the best available information, we anticipate that the currently low concentrations of bioaccumulative compounds in the whales will remain low, and that these levels are substantially lower than threat levels found in surrogate species and are not anticipated to pose a risk to the Southern Residents.

Effects of Reduced Prey Availability

We rely on the salmon determinations to ensure that the proposed action does not appreciably reduce the likelihood of survival and recovery of the Southern Residents in the long term. Later in this opinion, NMFS concludes that the proposed action is likely to appreciably reduce the likelihood of survival and recovery of the UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, LCR Chinook salmon, LCR coho salmon, SR fall-run Chinook salmon, SR sockeye salmon, CR chum salmon, OC coho salmon, SONCC coho salmon, green sturgeon, and Eulachon. In other words, the proposed action appreciably increases the risk of extinction of these listed species.

Our analysis focused on the short- and long-term reductions in Chinook salmon available to the whales as a result of the proposed action. Below we discuss the effects from (1) the short-term or annual reduction in Chinook salmon stocks, and (2) the long-term appreciable reduction in the likelihood of survival and recovery of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon.

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Short-term or annual reduction in Chinook stocks

Mortality of Chinook could affect the annual prey availability to the whales where the marine ranges of the affected Chinook stocks and the whales overlap. Mortality of adult Chinook salmon could affect the quantity of prey available to the whales in a given year, whereas mortality of juvenile Chinook salmon could affect prey availability in future years. Juvenile mortality from exposure to the compounds in Table 1.1 translates to the effective loss of only a few adult-equivalent Chinook salmon from a variety of runs three to five years after the juvenile mortality occurred (*i.e.*, by the time these juveniles would have grown to be adults and available prey of killer whales). This reduction would occur each year that the proposed criteria remain in place.

Given the total quantity of prey available to Southern Resident killer whales throughout their range, this annual reduction in prey is extremely small, and although measurable, the percent reduction in prey abundance is not anticipated to be different from zero by multiple decimal places (based on NMFS' previous analyses of the effects of salmon harvest on Southern Residents; *e.g.*, NMFS 2008e, NMFS 2011). Because the annual reduction is so small, there is also a low probability that any of the juvenile Chinook salmon killed from implementation of the proposed action would be intercepted by the killer whales across their vast range in the absence of the proposed action. Therefore, NMFS anticipates that the short-term reduction of Chinook salmon would have an insignificant effect on Southern Resident killer whales.

Long-term appreciable reduction in the likelihood of survival and recovery of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon

NMFS qualitatively evaluated long-term effects on the Southern Residents from the anticipated appreciable reduction in the likelihood of survival and recovery of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon. We assessed the likelihood for localized depletions, and long-term implications for Southern Residents' survival and recovery, resulting from the increased risk of extinction of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon. In this way, NMFS can determine whether the increased likelihood of extinction of prey species is also likely to appreciably reduce the likelihood of survival and recovery of Southern Residents.

A reduction in prey would occur over time as abundance declined for UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon. Hatchery programs, which account for a portion of the production of these ESUs, may provide a short-term buffer, but it is uncertain whether hatchery-only stocks could be sustained indefinitely. The total 5-year geometric mean abundance for the 5 ESUs (UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and the SR fall-run Chinook salmon) is 128,534 total spawners. The loss of these ESUs would also preclude the potential for their future recovery to healthy, more substantial numbers. Fewer populations contributing to Southern Residents' prey base will reduce the representation of diversity in life histories, resiliency in withstanding

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stochastic events, and redundancy to ensure there is a margin of safety for the salmon and Southern Residents to withstand catastrophic events.

The long-term reduction of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon can lead to nutritional stress in the whales. Nutritional stress can lead to reduced body size and condition of individuals and can also lower reproductive and survival rates. Prey sharing would distribute more evenly the effects of prey limitation across individuals of the population that would otherwise be the case. Therefore, poor nutrition from the reduction of prey could contribute to additional mortality in this population. Food scarcity could also cause whales to draw on fat stores, mobilizing contaminants stored in their fat and affecting reproduction and immune function.

Differences in adult salmon life histories and locations of their natal streams likely affect the distribution of salmon across the Southern Residents' coastal range. The continued decline and potential extinction of the UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon, and consequent interruption in the geographic continuity of salmon-bearing watersheds in the Southern Residents' coastal range, is likely to alter the distribution of migrating salmon and increase the likelihood of localized depletions in prey, with adverse effects on the Southern Residents' ability to meet their energy needs. A fundamental change in the prey base originating from Oregon is likely to result in Southern Residents abandoning areas in search of more abundant prey or expending substantial effort to find depleted prey resources. This potential increase in energy demands should have the same effect on an animal's energy budget as reductions in available energy, such as one would expect from reductions in prey.

In summary, approval of the numeric criteria listed in Table 1.1 in the long term will increase the likelihood of extinction of the Chinook salmon stocks which will appreciably reduce the likelihood of survival and recovery of the Southern Resident killer whales.

2.8.1. Integration and Synthesis: Southern Resident Killer Whales.

Based on the analysis of the acute and chronic toxicity data, the results of the summary analysis, and the predicted long-term effects on UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, and LCR Chinook salmon, the proposed action is likely to affect the productivity and abundance, spatial distribution, and affect the long-term viability of Southern Resident killer whales.

Several factors identified in the final recovery plan for Southern Resident killer whales may be limiting recovery. These are quantity and quality of prey, toxic chemicals that accumulate in top predators, and disturbance from sound and vessels. Oil spills are also a risk factor. It is likely that multiple threats are acting together. For example, reduction in prey availability makes it harder for the whales to locate and capture prey, which can cause them to expend more energy and catch less food. Although it is not clear which threat or threats are most significant to the survival and recovery of Southern Residents, all of the threats are important to address.

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The Southern Resident killer whale DPS is composed of one small population (88 whales) which is currently at most half of its likely previous size (140 to as many as 400 whales). The effective population size (based on the number of breeders under ideal genetic conditions) of 26 whales is very small, and this in combination with the absence of gene flow from other populations may elevate the risk from inbreeding and other issues associated with genetic deterioration. This population has a variable growth rate (28-year mean=0.3% ± 3.2% s.d), and risk of quasi extinction that ranges from 1% to as high as 66% over a 100-year horizon, depending on the population's survival rate and the probability and magnitude of catastrophic events. Because of this population's small size, it is susceptible to demographic stochasticity and genetic deterioration, as described in the Status of the Species. The influences of demographic stochasticity and potential genetic issues in combination with other sources of random variation combine to amplify the probability of extinction, known as the extinction vortex.

The larger the population size, the greater the buffer against stochastic events. It also follows that the longer the population stays at a small size, the greater its exposure to demographic stochastic risks and genetic risks. In addition, as described in the Status of the Species section, small populations are inherently at risk because of the unequal reproductive success of individuals within the population. The more individuals added to a population in any generation, the more chances of adding a reproductively successful individual. Random chance can also affect the sex ratio and genetic diversity of a small population, leading to lowered reproductive success of the population as a whole. For these reasons, the failure to add even a few individuals to a small population in the near term can have long-term consequences for that population's ability to survive and recover into the future. A delisting criterion for the Southern Resident killer whale DPS is an average growth rate of 2.3% for 28 years (NMFS 2008a). In light of the current average growth rate of 0.3%, this recovery criterion and the risk of stochastic events and genetic issues described above underscore the importance for the population to grow quickly.

The effects of the proposed action include bioaccumulation, biomagnification, and reduced prey quality and quantity. As explained in the section [*Toxic Chemical Accumulation in the Southern Residents*], compared to current conditions, the proposed criteria will result in the same levels for some compounds and reduced bioaccumulation and reduced biomagnification in the Southern Residents for some compounds. The NMFS anticipates that the relatively low concentrations of the bioaccumulative compounds in the whales will remain low and below health effects thresholds found in surrogate species. For these reasons, NMFS anticipates that the effects of the proposed action on the accumulation of the toxic chemicals in Southern Residents will be insignificant.

As explained in the section *Effects of Prey Reduction*, the anticipated short-term reduction of Chinook salmon associated with the proposed action would result in an insignificant annual reduction in adult equivalent prey resources for Southern Resident killer whales.

Over the long-term, however, the proposed action will increase the risk of extinction of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, and LCR Chinook salmon stocks and could result in a greater reduction in prey quantity and affect availability of prey in other ways (*i.e.*, spatially or temporally). Fewer populations contributing to Southern Residents' prey base will reduce the representation of

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diversity in life histories, resiliency in withstanding stochastic events, and redundancy to ensure there is a margin of safety for the salmon and Southern Residents to withstand catastrophic events. These reductions increase the extinction risk of Southern Residents.

The extinction of UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, and LCR Chinook salmon would reduce prey availability and increase the likelihood for local depletions of prey in particular locations and times. In response, the Southern Residents would increase foraging effort or abandon areas in search of more abundant prey. Reductions in prey or a resulting requirement of increased foraging efficiency increase the likelihood of physiological effects. The Southern Residents would likely experience nutritional, reproductive, or other health effects (*e.g.*, reduced immune function from drawing on fat stores and mobilizing contaminants in the blubber) from this reduced prey availability. These effects would lead to reduced body size and condition of individuals and can also lower reproductive and survival rates and thereby diminish the potential for Southern Residents to recover.

In summary: (1) The toxic chemicals discussed in this opinion have the ability to accumulate in the Southern Residents, however, bioaccumulation and biomagnification is expected to be relatively low, and levels in the whales are not anticipated to cause health effects. Furthermore, the proposed criteria will result in reduced bioaccumulation and biomagnifications of some compounds and levels will remain low and below health effects thresholds in the Southern Residents. (2) Short-term (or annual) reduction in prey availability associated with the proposed action would result in an insignificant annual reduction in adult equivalent prey resources for Southern Resident killer whales. (3) Increased risk of extinction of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, as a long-term consequence of the proposed action increases the risk of a permanent reduction in prey available to Southern Residents, and increases the likelihood for local depletions of prey in particular locations and times. (4) Losing the potential for future recovery of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, appreciabley diminishes the potential for Southern Residents to recover.

2.9 Conclusion

After reviewing the best available scientific and commercial information regarding the biological requirements and the status of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, Eulachon and Southern Resident killer whales considered in this opinion (section 2.4), the environmental baseline (section 2.5) for the action area, the effects of the proposed action (section 2.6), and the cumulative effects (section 2.6.8), NMFS concludes that the proposed action is likely to jeopardize the continued existence of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon,

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SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, Eulachon, and Southern Resident killer whales.

Furthermore, NMFS has determined NMFS has determined that the proposed action will result in the destruction or adverse modification of critical habitat as a result of degraded water quality in Oregon for LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, and Eulachon.

2.10. Reasonable and Prudent Alternative

This opinion has concluded that the proposed action will jeopardize the continued existence of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, eulachon, and Southern Resident killer whales.

This opinion also concluded that the proposed action will destroy or adversely modify critical habitat for LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, and eulachon.

Therefore, NMFS must discuss with EPA the availability of reasonable and prudent alternatives (RPAs) that EPA can take to avoid violation of EPA's ESA section 7(a)(2) responsibilities (50 CFR 402.14(g)(5)). RPAs are alternative actions identified during formal consultation that: (1) can be implemented in a manner consistent with the intended purpose of the action, (2) can be implemented consistent with the scope of the Federal agency's legal authority and jurisdiction, (3) are economically and technologically feasible, and (4) that NMFS believes would avoid the likelihood of jeopardizing the continued existence of listed species or resulting in the destruction or adverse modification of critical habitat (50 CFR 402.02).

This section presents EPA with an RPA that will avoid jeopardy and destruction or adverse modification of critical habitat, while meeting the requirements listed above. Because this opinion has found jeopardy and destruction or adverse modification of critical habitat, the EPA is required to notify NMFS of its final decision on the implementation of the reasonable and prudent alternative.

2.10.1 Proposed RPA

The NMFS identified seven criteria (*i.e.*, copper [acute and chronic], ammonia [acute and chronic], cadmium [acute], and aluminum [acute and chronic])—that would cause significant adverse toxicological and biological effects on the listed species considered in this opinion. Individually and in combination with exposure to multiple compounds and stressors, these

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criteria are likely to reduce appreciably the likelihood of both the survival and recovery of the listed species, and are likely to reduce appreciably the conservation value of their critical habitats.

The NMFS and the EPA considered a variety of alternatives to avoid jeopardy and destruction or adverse modification of critical habitat to the listed species considered in this opinion. Based on the best available information, NMFS and EPA were able to identify alternative numeric criteria for three of the seven criteria (acute and chronic copper, chronic ammonia). The alternative criteria are supported by both the best available information considered in this opinion as well as recent reanalysis conducted by EPA under the CWA.[10] These criteria will avoid jeopardy/adverse modification and are also within EPA's authority to implement.

For the remaining four criteria found to result in jeopardy/adverse modification, discussions between NMFS and EPA about the availability of an RPA that meets the regulatory criteria did not result in revised numeric criteria. Instead, the RPA specifies biological requirements to satisfy the conservation needs of the affected species and specific parameters EPA must work within to derive criteria that meet those requirements and avoid jeopardy and adverse modification of critical habitat.

Copper

Acute. The EPA shall disapprove the State of Oregon's acute criterion of 13 µg/L at 100 mg/L $CaCO_3$ for freshwater copper.

The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, a new acute criterion of 2.3 µg/L for freshwater copper using EPA's 2007 BLM-based aquatic life criteria. The EPA will ensure that the new acute copper criterion will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Chronic. The EPA shall disapprove the State of Oregon's chronic criterion of 9 µg/L at 100 mg/L $CaCO_3$ for freshwater copper.

The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, a new chronic criterion of 1.45 µg/L for freshwater copper using EPA's 2007 BLM-based aquatic life criteria. The EPA will ensure that the new chronic copper criterion will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Ammonia

Acute. The EPA shall use the Process for Deriving Criteria, specified below, to derive an acute criterion for freshwater ammonia at pH 8 and 20°C (total ammonia-N). The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, the derived

[10] http://water.epa.gov/scitech/swguidance/standards/criteria/aqlife/pollutants/copper/upload/2009_04_27_criteria_copper_2007_criteria-full.pdf

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acute ammonia criteria. The EPA will ensure that the derived acute ammonia criteria will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Chronic. The EPA shall disapprove the State of Oregon's chronic criterion of 1.7 mg/L at pH 8 and 20°C for freshwater ammonia (total ammonia-N).

The EPA shall recommend that the State of Oregon maintain the current chronic criterion of 0.76 mg/L at pH 8 and 20°Cfor freshwater ammonia (total ammonia-N).

Cadmium

Acute. The EPA shall disapprove the State of Oregon's acute criterion of 2.0 µg/L at 100 mg/L $CaCO_3$ for freshwater cadmium.

The EPA shall use the Process for Deriving Criteria, specified below, to derive an acute criterion for the State of Oregon for freshwater cadmium. The EPA shall recommend that the State adopt, and EPA will promulgate if necessary, the derived acute cadmium criteria. The EPA will ensure that the derived acute ammonia criteria will be effective within 24 months after EPA's final to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Aluminum[11]

Acute. The EPA shall disapprove the State of Oregon's acute criterion of 750 µg/L at pH 6.5-9.0for freshwater aluminum.

The EPA shall use the Process for Deriving Criteria, specified below, to derive an acute criterion for the State of Oregon for freshwater aluminum at pH 6.5-9.0. The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, the derived acute aluminum criteria. The EPA will ensure that the derived acute aluminum criteria will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Chronic. The EPA shall disapprove the State of Oregon's chronic criterion of 87 µg/L at pH 6.5-9.0for freshwater aluminum.

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[11] On August 9, 2012, EPA sent NMFS a letter withdrawing their request for consultation on Oregon's acute and chronic aluminum criteria as "EPA has determined that the BE submitted to NMFS in January 2008 incorrectly described the proposed federal action under consultation for aluminum (*i.e.*, CW A § 303(c)(3) approval of Oregon's submission of aluminum criteria). Specifically, Oregon's submitted description of the pollutant refers to aluminum in waters with a pH of 6.5- 9.0, but a footnote in the criterion itself indicates that the criterion is meant to apply to waters with pH less than 6.6 and hardness less than 12 mg/L (as $CaCO_3$)." Due to the court-ordered deadline of August 14, 2012, NMFS did not have time to modify its opinion to exclude acute and chronic aluminum from the document. The NMFS acknowledges EPA's revision to the proposed action, however, and notes it does not anticipate EPA will carry out the RPA for aluminum in light of this change. The NMFS will await a further request from EPA relating to EPA's potential future actions regarding Oregon's aluminum criteria.

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The EPA shall use the Process for Deriving Criteria, specified below, to derive a chronic criterion for the State of Oregon for freshwater aluminum at pH 6.5-9.0. The EPA shall recommend that the State of Oregon adopt, and EPA will promulgate if necessary, the derived chronic aluminum criteria. The EPA will ensure that the derived chronic aluminum criteria will be effective within 24 months after EPA's final action to approve or disapprove Oregon's proposed water quality criteria under the CWA.

Process for Deriving Criteria

The EPA shall utilize analytical methods that meet specified requirements to derive numeric criteria for aquatic life, taking into account the same factors that NMFS did in completing its analysis for the other criteria in this opinion. The EPA will then evaluate the analytical results with a population model that meets the requirements set out below, and thus is equivalent to that used by NMFS in this opinion, to confirm that the derived criteria will not jeopardize listed fish or adversely modify their critical habitat.

In particular, the EPA shall derive criteria for acute ammonia, acute cadmium, and acute and chronic aluminum in compliance with the following five requirements:

1) Only use toxicity data for ammonia, cadmium, and aluminum that is specific to salmonid fishes (if new information becomes available for these compounds for green sturgeon and eulachon, then EPA shall include this data in its analysis);
2) All toxicity data used to derive the numeric criteria must be curve-fitted, where the literature provides the necessary data to perform this step;
3) When available, the curve-fitted toxicity data must be used to extrapolate threshold acute and chronic toxic effect concentrations;
4) Derived criteria must be model-adjusted to account for chemical mixtures; and,
5) An appropriate population model must be applied to the derived criteria, and must predict no negative change in the intrinsic population growth rate (*e.g.*, lambda, λ).

More specifically, EPA shall ensure that the derived criteria are developed in compliance with the following mandatory sideboards:

- The EPA shall use toxicity data specific to salmonid fishes. The EPA shall use the acute and chronic toxicity data in this opinion as a minimum data set. For green sturgeon and eulachon, EPA shall use the salmonid fishes toxicity data for this analysis, as described in section 2.6.2 in this opinion, in addition to any new data that becomes available for green sturgeon and eulachon.
- The EPA shall use toxicity data based on exposure-response curves and fixed durations toxicity tests to estimate acute and chronic toxic effect thresholds to assess effects on multiple life stages and multiple endpoints, to include at a minimum: mortality, latent mortality, reproduction, growth, physiological, cellular, behavioral, and biochemical effects, where the data exists. The EPA may use existing toxicity data for ammonia, cadmium, and aluminum or generate new data, but the data shall be curve-fitted (see Figure 2.6.1.1) to determine the minimum effect thresholds (*e.g.*, 5%) at which acute and chronic toxic effects are predicted. The minimum effects thresholds shall be used to

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derive the criteria instead of using the EPA acute adjustment factor or the acute-to-chronic ratio to derive criteria.

- The EPA shall ensure that each derived criterion for ammonia, cadmium, and aluminum is adjusted to account for chemical mixtures using a concentration–addition model or response-addition model to determine whether or not exposure to multiple compounds will result in additive effects to the listed species considered in this opinion. The concentration–addition model or response-addition model shall include all compounds listed in Table 1.1. If the mixture effects prediction is greater than one, EPA shall adjust the concentrations for ammonia, cadmium, and aluminum until the mixture effects prediction is less than one.
- The EPA shall ensure that the derived criteria for ammonia, cadmium, and aluminum do not result in a negative change in the intrinsic population growth rate based on the geometric mean abundance data for each life history type, *i.e.*, coho salmon (*O. kisutch*), sockeye salmon (*O. nerka*) and ocean-type and stream-type Chinook salmon (*O. tshawytscha*), of salmonid fish considered in this opinion, at the population scale. The EPA shall use stream-type Chinook salmon as a surrogate for steelhead, and ocean-type Chinook salmon as a surrogate for chum salmon in the population model, as described in section 2.6.5.1 of this opinion. Pacific salmon and steelhead abundance data is available from the Northwest Fisheries Science Center Salmon Population Summary Database[12] or from the Columbia Basin Fish and Wildlife Authority Status of the Fish and Wildlife Resources Database[13]. The abundance data used for the population growth rate analysis shall include data from all years with available abundance data. For green sturgeon and eulachon, EPA shall use the salmonid fishes toxicity data and modeling results as surrogate data and outputs for this analysis.
- To ensure that the derived numeric criteria for ammonia, cadmium, and aluminum meet the population growth rate condition of the RPA, EPA shall run the criteria for ammonia, cadmium, and aluminum through a population model (*e.g.*, Leslie Matrix), parameterized for Pacific salmonid fishes. Model requirements include: (1) scenarios based on change in first year survival; (2) an assumption that the populations are density-independent, to reduce the probability of Type II errors; (3) sigmoid slopes are generated from the data used to derive the numeric criteria, and if a slope cannot be generated from the data, EPA shall use the default sigmoid slope of 3.6 used in this opinion; and (4) exposure-response scenarios using the geometric mean of the curve-fitted data, and the minimum species mean value of the curve-fitted data, from the toxicity data used to derive the numeric criteria.

2.10.2 Compliance with RPA Criteria

A reasonable and prudent alternative to the proposed action is one that avoids jeopardy by ensuring that the action's effects do not appreciably increase the risks to the species' potential for survival or to the species' potential for recovery. It also must avoid destruction or adverse modification of designated critical habitat. A detailed analysis of how the RPA avoids jeopardy

[12]https://www.webapps.nwfsc.noaa.gov/sps
[13]http://sotr.cbfwa.org

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and destruction or adverse modification of critical habitat is set out in section 2.10.3, below. In summary:

Implementation of the RPA avoids jeopardy to the listed species of fish because:

- We find that, based on the acute and chronic data in this opinion, effects of the revised action will not manifest at the population scale.
- We considered factors such as latent mortality and hypothesis tests in our effects analysis to assess the uncertainty of the revised action.
- The revised action will not result in appreciable population-level effects, (*i.e.*, lethal and sublethal effects do not result in a negative change in the intrinsic population growth rate, *e.g.*, lambda, λ).
- The available evidence indicates that the revised action is unlikely to appreciably affect invertebrate productivity and abundance.
- The requirement to adjust the criteria using a concentration–addition model or response-addition model will ensure that the revised action has a low probability of causing additive effects to the listed species.
- It can reasonably be concluded that the time needed to fully implement the revised action will not measurably impact the listed ESUs/DPSs or their critical habitat affected by this action.

For similar reasons, implementation of the RPA avoids adverse modification of the critical habitats for the listed species fish because:

- The revised action will not adversely modify critical habitats for the listed species considered in this opinion as the data suggests that the criteria concentrations are likely to have low-intensity adverse effects on the PCEs substrate, forage, or water quality at the watershed and designation scales. The available evidence indicates that the revised action is unlikely to appreciably affect invertebrate productivity and abundance.
- The revised action will minimize loading of copper, ammonia, cadmium, and aluminum in the affected watersheds so that habitat functions are maintained consistent with the conservation needs of the species.
- It can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their critical habitat affected by this action.

Implementation of the RPA avoids jeopardy to Southern Resident killer whales because, for those listed fish species that are prey for Southern Resident killer whales and the subject of this opinion, the RPA will ensure the impact on productivity and abundance is at a level where it does not pose an appreciable risk to the listed fish species and their designated critical habitats. Implementation of the RPA will also decrease the accumulation of toxic chemicals in the whales by reducing the bioaccumulation and toxic burdens in their prey to levels consistent with recovery of the listed species. For these reasons, NMFS expects that implementation of the RPA will avoid jeopardy for Southern Resident killer whales.

The reasonable and prudent alternative must also be: (1) consistent with the intended purpose of the action; (2) within the scope of the Federal agency's legal authority and jurisdiction; and

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(3) economically and technologically feasible. This RPA is consistent with the purpose of EPA's action, as it will ensure that Oregon's water quality criteria for toxic pollutants will be protective of aquatic species. The EPA has authority, under the Clean Water Act, to ensure that state water quality standards are consistent with the requirements of the Clean Water Act requirements, which include ensuring that aquatic life is adequately protected.

Implementation of the RPA may impose some additional costs on the State of Oregon by requiring the state to meet more stringent numeric criteria than proposed, but neither the State of Oregon nor EPA conducted an economics analysis for the proposed action. With respect to chronic ammonia and acute and chronic copper, the RPA has been demonstrated to be economically and technologically feasible, because the freshwater chronic criterion of 0.76 mg/L for freshwater ammonia (total ammonia-N) at pH 8 and 20°C is currently being implemented in Oregon, and the acute and chronic criteria for copper are EPA's nationally recommended aquatic life criteria. For acute ammonia, acute cadmium, and acute and chronic aluminum, the RPA is economically and technologically feasible for EPA since it requires the agency to conduct an analysis and ensure the derived criteria are implemented in the State of Oregon, both functions that can be readily accommodated within the agency's normal course of business.

2.10.3 RPA Effects Analysis

The RPA Effects analysis is provided with reference to the effects of the action detailed above (section 2.6), which analyses effects of all criteria. This section provides particularized discussion of the seven criteria for which an RPA is provided.

2.10.3.1 Copper – Acute and Chronic

The revised criteria for copper are 1.45 µg/L (chronic) and 2.3 µg/L (acute), using EPA's 2007 BLM-based aquatic life criteria.[14]

The NMFS has determined that these revised criteria satisfy the conservation needs of the species and function of critical habitat PCEs because when we apply the same analysis that we used in the Effects Analysis, as described in section 2.6 of this opinion to the revised copper criteria, we find that the revised acute and chronic criteria for copper are unlikely to cause acute or chronic toxic effects to the listed fishes considered in this opinion that would manifest at the population scale.

More specifically:

- The NMFS compared the acute and chronic toxicity data in section 2.6.2.2.6 of this opinion to the revised criteria. For the acute criterion, none of the LC_{50} data was identified as being

[14]With regard to BLM-derived freshwater criteria, to develop a site-specific criterion for a stream reach, one is faced with determining what single criterion is appropriate even though a BLM criterion calculated for the event corresponding to the input water chemistry conditions will be time-variable. This is not a new problem unique to the BLM—hardness-dependent metals criteria are also time-variable values. Although the variability of hardness over time can be characterized, EPA has not provided guidance on how to calculate site-specific criteria considering this variability.

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less than the revised acute criterion, the relative percent mortality analysis predicts a median toxicity potential of an $LC_{1.2}$, and only 11 of the 150 chronic data points were identified as being less than the revised chronic criterion.

- To take into account the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes, we considered factors such as latent mortality and hypothesis tests in our effects analysis to assess the uncertainty of the revised criteria
- The NMFS ran the revised acute criterion for freshwater copper of 2.3 µg/L through the direct mortality populat$_{io}$n model (Appendix 1) using the geometric mean and the minimum species mean values of the LC_{50} data for copper to assess effects on mortality and lambda. The exposure-response scenario using the minimum species mean value with the revised criterion concentration of 2.3 µg/L predicted 1% mortality for all life history types with a 0% change in λ for all life history types. The exposure-response scenario using the geometric mean value predicted 0% mortality with 0% change in λ for all life history types. The NMFS considers the results of the direct mortality population model using the minimum species mean value to be a very conservative exposure-response scenario. The fact that this conservative exposure-response scenario predicts no change in λ for any of the life history types provides a level of assurance that the revised acute criterion for freshwater copper of 2.3 µg/L is unlikely to cause population-level adverse effects.
- Our analysis of the revised chronic criterion suggests that the revised criterion concentration is likely to avoid adverse chemosensory and behavioral effects to juvenile salmonid fishes (Hecht *et al.* 2007).
- The available evidence indicates that the chronic criterion for copper is unlikely to appreciably affect invertebrate productivity and abundance.
- For similar reasons, the revised criteria for copper will not adversely modify critical habitats for the listed species considered in this opinion as the data suggests that the criteria concentrations are likely to have low-intensity adverse effects on the PCEs substrate, forage, or water quality at the watershed and designation scales.

2.10.3.2 Ammonia – Chronic

The revised chronic criterion for ammonia is 0.76 mg/L as N (NH_3-nitrogen) at pH of 8.0 and 20°C.

The NMFS has determined that these revised criteria satisfy the conservation needs of the species and function of critical habitat PCEs because when we apply the same analysis that we used in the Effects Analysis, as described in section 2.6 of this opinion to the revised ammonia criterion, we find that, the revised chronic criterion for ammonia is unlikely to cause chronic toxic effects to the listed fishes considered in this opinion that would manifest at the population scale.

More specifically:

- The NMFS compared the chronic toxicity data in section 2.6.2.1.7 of this opinion to the revised criterion. For the chronic criterion only 9 of the 19 chronic data points were identified as being less than the revised chronic criterion. As described in the opinion, NMFS only

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selected toxicity data in the core data file with a reported concentration type of total ammonia. For these toxicity studies, temperature and pH were not reported in the core data files; therefore verification regarding normalization was not possible and creates uncertainty. Therefore, as an additional step to address this uncertainty and to assess the potential for chronic toxic effects of ammonia to the listed species considered in this opinion using an additional line of evidence, NMFS used four ACRs described in section 2.6.2.1.7 of this opinion to estimate a NOEC for ammonia. These produced no concentrations less than the chronic criterion concentration, which indicates that listed species exposed to waters equal to chronic criterion concentrations may not suffer chronic toxic effects. To take into account the shortcomings and implications of laboratory-derived toxicity tests and the ecological consequences for field-exposed fishes, we considered factors such as hypothesis tests in our effects analysis to assess the uncertainty of the revised criteria.

- The NMFS also considered non-lethal effects based on best available information and determined that they would be suffered at low-intensity.
- The revised criterion for ammonia will not adversely modify critical habitats for the listed species considered in this opinion as the data suggests that the criteria concentrations are likely to have low-intensity adverse effects on the PCEs substrate, forage. Ammonia does not bioaccumulate or bind to sediments—therefore effects on the PCEs substrate and forage are unlikely to be affected in a manner inconsistent with the recovery requirements of the listed fishes considered in this opinion. Furthermore, based on the ACR analyses, the revised criterion is likely to have low-intensity adverse effects on the PCEs substrate, forage, or water quality at the watershed and designation scales.

2.10.3.3 Derived Criteria

The EPA will derive criteria for acute ammonia, acute cadmium, and acute and chronic aluminum in accordance with the Process for Deriving Criteria set out above to ensure an adequately protective criterion is established.

The NMFS has determined that the derived criteria will satisfy the conservation needs of the species and function of critical habitat PCEs because the RPA relies on a conservative, well-defined methodology and requires EPA to ensure that the acute criterion for ammonia, the acute criterion for cadmium, and the acute and chronic criteria for aluminum do not cause a change in the intrinsic population growth rate (*e.g.*, λ). More specifically, NMFS developed the following requirements to address the uncertainties associated with the toxicity data, sublethal effects, multiple environmental stressors, and biological requirements consistent with the principles of conservation biology.

Toxicity Data

Because EPA is required to use toxicity data specific to salmonid fishes (and green sturgeon and eulachon, if it becomes available), this will minimize the uncertainties regarding the use of surrogate species and methodologies, *e.g.*, interspecies correlation analyses, to derive criteria that are consistent with the biological requirement of the species considered in this opinion.

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Curve-fitted Data to Extrapolate Threshold Concentrations

The EPA is required to use toxicity data based on exposure-response curves and fixed durations toxicity tests to estimate acute and chronic toxic effect thresholds to assess effects on multiple life stages and multiple endpoints, to include at a minimum: mortality, latent mortality, reproduction, growth, physiological, cellular, behavioral, and biochemical effects, where the data exists. This requirement operates to ensure the derived criteria account for effects beyond the standard mortality, growth, and reproduction endpoints, but considers effects on a species life cycle and on sublethal endpoint that can affect the fitness and survival of affected species.

Adjust for Chemical Mixtures

The EPA is required to adjust each derived criterion for chemical mixtures using a concentration–addition model or response-addition model to determine whether or not exposure to multiple compounds will result in additive effects to the listed species. This requirement operates to ensure that environmental exposure conditions are considered in the development of the derived criteria. Fish exposed to multiple compounds, versus a single compound exposure, are likely to suffer toxicity greater than the assessment effects such as mortality, reduced growth, impairment of essential behaviors related to successful rearing and migration, cellular trauma, physiological trauma, and reproductive failure. The requirement to adjust the criteria using a concentration–addition model or response-addition model will ensure that the derived criteria have a low probability of causing additive effects to the listed species.

No Negative Change in Intrinsic Population Growth

Important assurances are provided by the requirement that the derived criteria do not result in a negative change in the intrinsic population growth rate based on the geometric mean abundance data for each life history type (as determined by a population model parameterized for Pacific salmonid fishes and otherwise meeting the RPA requirements). The requirement that the derived criteria are run through a population model is a method to assess population-level effects. A change in the intrinsic population growth rate, *e.g.*, λ, is an accepted population parameter often used in evaluating population productivity, status, and viability. The NMFS uses changes in λ when estimating the status of species, conducting risk and viability assessments, developing recovery plans, ESA consultations, and communicating with other federal, state and local agencies (McClure *et al.*, 2003). While values of $\lambda<1.0$ indicate a declining population, in cases when an exposure causes the population growth rate to decrease more than natural variability, a loss of productivity will result even if lambda remains above 1.0. Decreases in response to chemical exposures can be a cause for concern since the impact could make a population more susceptible to declining (lambda dropping below 1.0) due to impacts from other stressors. Therefore, the no change in the intrinsic population growth rate ensures that effects from the derived criteria will not manifest at the population scale, and are consistent with the recovery of the species considered in this opinion.

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2.10.3.4. Mixtures Analysis

Since EPA has not derived specific numeric criteria for acute ammonia, acute cadmium, and acute and chronic aluminum, NMFS cannot run the revised numbers through the concentration-addition model used in this section 2.6.4 of this opinion to generate a revised mixtures effects prediction. Nonetheless, the requirement to adjust the criteria using a concentration–addition model or response-addition model will ensure that the revised criteria have considered environmental exposure conditions of multiple compounds.

2.10.3.5 Implementation Period

The NMFS evaluated the impact of the time lag between completion of the opinion and implementation of the revised action. In the proposed action, EPA assumed that the numeric criteria would be met outside the State's applicable mixing zone boundaries, *i.e.*, that the criteria represent ambient water quality conditions. The NMFS carried the assumption that the criteria concentrations represent the ambient water quality conditions through its analysis of the proposed action and of the RPA. Yet, based on Oregon DEQ's water quality assessment program data,[15] it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before ambient water quality conditions reach criteria concentrations.

To explore this in more detail, NMFS compared the current water quality baseline against the ambient criteria identified in the RPA to determine the likelihood that concentrations of these toxics would exceed the criteria identified in the RPA during the implementation period. The NMFS focused its analysis on the chronic criteria for ammonia. The NMFS determined that ammonia is a reasonable proxy for the remaining criteria because the RPA criteria for chronic ammonia is the same criterion currently in place;[16] thus, ammonia provides a natural reflection of the current distribution of the proposed new criterion, which is conducive to assessing the likelihood that the new criterion will be exceeded in a significant manner across the State during the implementation period. In addition, the other criteria do not so readily lend themselves to analysis.[17]

The data that we used was derived from Oregon Department of Environmental Quality's Water Quality Assessment Database. We extracted all available records associated with lakes and streams that had data for ammonia. The data included 273 records from river reaches in 64 subbasins across Oregon. Only four reaches in four subbasins were identified as sufficiently water quality limited as a result of ammonia to warrant listing on the State's CWA section 303(d) list. Three of these subbasins are above the range of anadromous fish. The remainder of the subbasins had no reaches that had high enough concentrations of ammonia to warrant listing on the 303(d) list. Even in the more densely populated area of the Willamette, approximately 68%

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[15] http://www.deq.state.or.us/lab/wqm/watershed.htm

[16] The RPA states that EPA shall approve a new chronic criterion for the State of Oregon by maintaining the current chronic criterion of 0.76 mg/L at pH 8 and 20°C for freshwater ammonia (total ammonia-N).

[17] The derived criteria are not yet available for this type of analysis and because the copper criteria will be developed using the BLM approach it cannot be evaluated independent of other parameters necessary to determine site specific values.

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of the reported reaches were fully attaining for ammonia. Extrapolating generally from the ammonia data, which demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations, it can reasonably be concluded that the time needed to implement the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

2.10.4 RPA Integration and Synthesis

For the RPA Effects Analysis, NMFS integrated the effects of the revised and derived criteria for copper, ammonia, cadmium, and aluminum into an overall effects analysis, taking into account the effects of the other criteria proposed by EPA. Similar to the RPA Effects Analysis, the RPA Integration and Synthesis considers the effects of the action as a whole, with additional focus on the seven compounds that NMFS identified with the highest-intensity adverse toxicological and adverse biological effects on the listed species considered in this opinion.

The RPA Integration and Synthesis section fully considers the effects of the action (section 2.6) to the environmental baseline (section 2.5), the cumulative effects (section 2.6.8), and the Integration and Synthesis (section 2.7) to formulate the agency's biological opinion as to whether the revised action is likely to: (1) Result in appreciable reductions in the likelihood of both survival and recovery of the species in the wild by reducing its numbers, reproduction, or distribution; or (2) reduce the value of designated or proposed critical habitat for the conservation of the species. These assessments are made in full consideration of the status of the species and critical habitat (section 2.4).

ESU/DPS-Specific Evaluations

LCR Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; LCR Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for LCR Chinook salmon or the broader watershed scale for their critical habitat.

(2) NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population

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model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 32 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect LCR Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for LCR Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for LCR Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of LCR Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of LCR Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of LCR Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely

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affected (40.2 percent of the total designation), but will not appreciably reduce the conservation value.

(6) NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSsor their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of LCR Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of LCR Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UWR Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; UWR Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for UWR Chinook salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 7 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the

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relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect UWR Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for UWR Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UWR Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UWR Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for UWR Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UWR Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will incrementally improve water quality conditions for UWR Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances, and improve habitat quality that adequately provides for the conservation needs of UWR Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of UWR Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it

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may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of UWR Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of UWR Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UCR Spring-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; UCR spring-run Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for UCR spring-run Chinook salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 4 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect UCR spring-run Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for UCR

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spring-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UCR spring-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UCR spring-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for UCR spring-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UCR spring-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of UCR spring-run Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of UCR spring-run Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of UCR spring-run Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (30.8 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will

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not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of UCR spring-run Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of UCR spring-run Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Spring/Summer-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SS-run Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for SS-run Chinook salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 27 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SS-run Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for SS-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SS-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

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(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SS-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SS-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SS-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SS-run Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SS-run Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SS-run Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (25.3 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery for SR SS-run Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to

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reduce appreciably the conservation value of SR SS-run Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Fall-run Chinook Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SR fall-run Chinook salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for SR fall-run Chinook salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for the single SR fall-run Chinook salmon ESU (which consists of eight spawning populations).

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SR fall-run Chinook salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for SR fall-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SR fall-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial

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arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SR fall-run Chinook salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SR fall-run Chinook salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SR fall-run Chinook salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SR fall-run Chinook salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SR fall-run Chinook salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SR fall-run Chinook salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (25.3 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of SR fall-run Chinook salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of SR fall-run Chinook salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

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CR Chum Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; CR chum salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for CR chum salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 17 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect CR chum salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for CR chum salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of CR chum salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of CR chum salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced

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fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for CR chum salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of CR chum salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of CR chum salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of CR chum salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of CR chum salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (26 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(6) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of CR chum salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of CR chum salmon critical habitat such that it will retain the current ability for the PCE water quality to serve the intended conservation role for the species for either survival or recovery.

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LCR Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; LCR coho salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for LCR coho salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 27 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect LCR coho salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for LCR coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced

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fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for LCR coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(6) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of LCR coho salmon.

SONCC Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SONCC coho salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for SONCC coho salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 42 populations.

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(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SONCC coho salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for SONCC coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SONCC coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SONCC coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SONCC coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SONCC coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SONCC coho salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SONCC coho salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SONCC coho salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (37.8 percent of the total designation), but will not appreciably reduce the conservation value.

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(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of SONCC coho salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of SONCC coho salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

OC Coho Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; OC coho salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for OC coho salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 56 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum;

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and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect OC coho salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for OC coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of OC coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of OC coho salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for OC coho salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of OC coho salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of OC salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of OC coho salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of OC coho salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally

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from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of OC coho salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of OC coho salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SR Sockeye Salmon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SR sockeye salmon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the ESU and its PCEs, but will not rise to the population level for SR sockeye salmon or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for the single SR sockeye salmon population.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SR sockeye salmon, but is not likely to appreciably affect the VSP parameters productivity and abundance for SR sockeye salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SR sockeye salmon such

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that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SR sockeye salmon through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SR sockeye salmon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SR sockeye salmon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SR sockeye salmon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SR sockeye salmon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SR sockeye salmon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (34.5 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

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(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of SR sockeye salmon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of SR sockeye salmon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

LCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; LCR steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for LCR steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 26 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect LCR steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for LCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat

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quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of LCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for LCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of LCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of LCR steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of LCR steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of LCR steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (33 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of LCR steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of LCR steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

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UWR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; UWR steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for UWR steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 5 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect UWR steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for UWR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UWR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UWR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental

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variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for UWR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UWR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of UWR steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of UWR steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of UWR steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (100 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of UWR steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of UWR steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

MCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with

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consideration of the other proposed numeric criteria; MCR steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for MCR steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 17 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect MCR steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for MCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of MCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of MCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for MCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of MCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

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(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of MCR steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of MCR steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of MCR steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (75.7 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of MCR steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of MCR steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

UCR Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; UCR steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for UCR steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to

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the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 4 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect UCR steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for UCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of UCR steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for UCR steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of UCR steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of UCR steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of UCR steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of UCR steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and

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the overall percentage of critical habitat for this species that would be adversely affected (30.8 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of UCR steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of UCR steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

SRB Steelhead.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; SRB steelhead and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for SRB steelhead or the broader watershed scale for their critical habitat.

(2) The NMFS used the direct mortality population model as a quantitative method to assess the significance of acute toxic effects on long-term productivity and abundance from exposure to the freshwater acute criteria for copper. The direct mortality population modeling predicted 1% mortality for all life history types at the population level—relative to the baseline population model. This level of mortality will not result in negative changes in the median population growth rate (λ) for each of the 24 populations.

(3) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the

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relative percent mortality analysis for copper, the direct mortality population model for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect SRB steelhead, but is not likely to appreciably affect the VSP parameters productivity and abundance for SRB steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SRB steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(4) The analysis in this opinion primarily evaluates effects on productivity and abundance. However, population abundance is directly related to the quantity (capacity) of habitat available, productivity (intrinsic smolts per spawner or adults per spawner) is most strongly tied to habitat quality, and spatial distribution and genetic diversity are both strongly influenced by the spatial arrangement and variety of suitable habitats within a population's range (Wainwright *et al.* 2008). The link between long-term changes in productivity and long-term reductions in abundance can negatively affect the spatial distribution and/or the genetic diversity of SRB steelhead through multiple mechanisms, including sustained declines in spawner:spawner ratios; decreased smolt-to-adult returns; changes in traits, such as size and age of spawners; reduced fecundity; greater pre-reproductive spawning risk; increased influences of environmental variation and demographic stochasticity; changes in source-sink dynamics; and changes in patterns of gene flow. In the long term, the revised action will not produce changes in productivity and abundance. Therefore, the effects described in this section, with consideration of the VSP parameters productivity and abundance, are unlikely to appreciably diminish the VSP parameters spatial distribution and genetic diversity for SRB steelhead. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of SRB steelhead such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(5) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of SRB steelhead. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of SRB steelhead. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of SRB steelhead. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (34.5 percent of the total designation), but will not appreciably reduce the conservation value.

(6) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an

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example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(7) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of SRB steelhead. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of SRB steelhead critical habitat such that it will retain the current ability to serve the intended conservation role for the species' for either survival or recovery.

Green Sturgeon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; green sturgeon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for green sturgeon or the broader watershed scale for their critical habitat.

(2) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely affect green sturgeon, but is not likely to appreciably affect the VSP parameters productivity and abundance for green sturgeon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of green sturgeon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(3) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of green sturgeon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of green sturgeon.

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Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of green sturgeon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (10.4 percent of the total designation), but will not appreciably reduce the conservation value.

(4) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(5) After considering all the information in this opinion, NMFS concludes that the revised action is likely not to reduce appreciably the likelihood of both the survival and recovery of green sturgeon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of green sturgeon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

Eulachon.

(1) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum along with consideration of the other proposed numeric criteria; eulachon and the PCEs of their designated critical habitat will experience some acute and chronic toxic effects from exposure to the 65 numeric criteria under the revised action. For the reasons discussed in the prior RPA sections above and summarized below, these effects will manifest at an individual level (or group of individuals) for the DPS and its PCEs, but will not rise to the population level for eulachon or the broader watershed scale for their critical habitat.

(2) Based on the compound-specific acute and chronic toxicity data analysis for copper and chronic ammonia; the analysis on considerations of the shortcomings and implications of laboratory-derived toxicity tests (uncertainty analysis) for copper and chronic ammonia, the relative percent mortality analysis for copper, the summary analysis for copper and chronic ammonia, and the RPA-specific minimum biological requirements for acute ammonia, acute cadmium, and acute and chronic aluminum; and the likelihood of exposure to baseline chemical and physical stressors (*e.g.*, cyanide, PCBs, aldrin, DDT, and high stream temperatures) along with consideration of the other proposed numeric criteria, the revised action is likely to adversely

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affect eulachon, but is not likely to appreciably affect the VSP parameters productivity and abundance for eulachon. Based on the aforementioned analysis in this opinion and in the RPA Effects Analysis, the revised action adequately provides for the conservation needs of eulachon such that effects will manifest at an individual level (or group of individuals), but not at the population level.

(3) In the long term, approval and implementation of the numeric criteria listed in Table 1.1, and the revised and derived criteria for copper, ammonia, cadmium, and aluminum, will ensure water quality conditions necessary to support the conservation needs of eulachon. Specifically, the revised action would minimize mass loading of toxic substances to levels that adequately provides for the function of the PCEs and the conservation needs of eulachon. Therefore, EPA's approval is not likely to appreciably reduce the conservation value of the critical habitat of eulachon. This conclusion is based on the magnitude of likely effects on the PCE water quality (low-to-moderate decrease in toxicity that affects one or more PCEs) and the overall percentage of critical habitat for this species that would be adversely affected (53.9 percent of the total designation), but will not appreciably reduce the conservation value.

(4) The NMFS evaluated the impact of the implementation period, i.e., the time between completion of the opinion and implementation of the revised action. Based on Oregon DEQ's water quality assessment program data, it appears there is variability in the current concentrations of toxics and their distribution throughout the subbasins in Oregon. Therefore, it may be some time before criteria concentrations reach ambient water quality conditions. As an example, ammonia data demonstrates that actual current concentrations are distributed in very irregular patterns almost entirely below the RPA criteria concentrations. Extrapolating generally from this, it can reasonably be concluded that the delay in implementing the revised action will not measurably impact the listed ESUs/DPSs or their designated critical habitat affected by this action.

(5) After considering all the information in this opinion, NMFS concludes that the revised action is not likely to reduce appreciably the likelihood of both the survival and recovery of eulachon. Furthermore, NMFS concludes that the revised action is not likely to reduce appreciably the conservation value of Eulachon critical habitat such that it will retain the current ability to serve the intended conservation role for the species for either survival or recovery.

Southern Resident Killer Whales.

As explained in section 2.8, we previously concluded that in the short-term, annual reductions in salmon prey caused by the proposed action would not have significant effects on Southern Resident killer whales. However, we determined that in the long-term, the continued decline and potential extinction of the UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, LCR Chinook salmon, and SR fall-run Chinook salmon, and consequent interruption in the geographic continuity of salmon-bearing watersheds in the Southern Residents' coastal range was likely to alter the distribution of migrating salmon and increase the likelihood of localized depletions in prey, with adverse effects on the Southern Residents' ability to meet their energy needs. We concluded that the proposed action would

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appreciably reduce the likelihood of survival and recovery of the Southern Resident killer whales.

Under the RPA, there will remain a reduction in prey in the short-term. However, as discussed in section 2.8, the annual prey reduction will be extremely small, and the probability is low that any of the juvenile Chinook salmon killed from implementation of the RPA would be intercepted by the killer whales across their vast range. Therefore, NMFS anticipates that the short-term reduction of Chinook salmon from the implementation of the RPA will have an insignificant effect on Southern Resident killer whales. The RPA will remove the long-term threat to killer whales by avoiding population-level and ESU/DPS-level effects to salmonids. Because the RPA will avoid ESU/DPS-level effects on abundance and productivity, and because we expect any short-term prey reductions to be insignificant, we also expect long-term effects from the RPA to be insignificant for Southern Resident killer whales. Also as discussed in Section 2.8, the available data indicate that Southern Residents are not at risk of health effects from the toxic criteria considered in this opinion. Because the RPA will further reduce levels of copper, ammonia, cadmium, and aluminum, we expect that any effects from the revised criteria will be insignificant and/or discountable.

In summary, implementation of the RPA avoids jeopardy to Southern Resident killer whales because it will reduce the impact on salmonids productivity and abundance to a level where it will not cause a discernable reduction in prey for Southern Resident killer whales and will also avoid adverse health effects to the whales.

Conclusion

Based on these considerations and the foregoing description of the RPA, NMFS finds that the RPA meets each of the criteria stated at 50 CFR 402.02.

After reviewing the best available scientific and commercial information regarding the biological requirements and the status of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, eulachon and Southern Resident killer whales considered in this opinion (section 2.4), the environmental baseline (section 2.5) for the action area, the effects of the proposed action (section 2.6), the cumulative effects (section 2.6.8), and the RPA (section 2.10), NMFS concludes that the revised action is not likely to jeopardize the continued existence of LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon, SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, LCR coho salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, eulachon, and Southern Resident killer whales.

Furthermore, NMFS has determined NMFS has determined that the revised action will not result in the destruction or adverse modification of critical habitat as a result of degraded water quality in Oregon for LCR Chinook salmon, UWR Chinook salmon, UCR spring-run Chinook salmon,

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SR spring/summer-run Chinook salmon, SR fall-run Chinook salmon, CR chum salmon, SONCC coho salmon, OC coho salmon, SR sockeye salmon, LCR steelhead, UWR steelhead, MCR steelhead, UCR steelhead, SRB steelhead, green sturgeon, and eulachon.

2.11 Incidental Take Statement

Section 9 of the ESA and Federal regulations pursuant to section 4(d) of the ESA prohibit the take of endangered and threatened species, respectively, without a special exemption. Take is defined as to harass, harm, pursue, hunt, shoot, wound, kill, trap, capture or collect, or to attempt to engage in any such conduct. Harm is further defined by regulation to include significant habitat modification or degradation that results in death or injury to listed species by significantly impairing essential behavioral patterns, including breeding, feeding, or sheltering. Incidental take is defined as take that is incidental to, and not the purpose of, the carrying out of an otherwise lawful activity. For this consultation, we interpret "harass" to mean an intentional or negligent action that has the potential to injure an animal or disrupt its normal behaviors to a point where such behaviors are abandoned or significantly altered.[18] Section 7(b)(4) and section 7(o)(2) provide that taking that is incidental to an otherwise lawful agency action is not considered to be prohibited taking under the ESA if that action is performed in compliance with the terms and conditions of this incidental take statement.

The NMFS has not yet promulgated an ESA section 4(d) rule prohibiting take of threatened eulachon. Anticipating that such a rule may be issued in the future, we have included a prospective incidental take exemption for eulachon. The elements of this ITS that relate to eulachon would take effect on the effective date of any future 4(d) rule prohibiting take of eulachon.

2.11.1 Amount or Extent of Take

All of the species of ESA-listed salmon, steelhead, green sturgeon, and eulachon analyzed in this opinion will be exposed to concentrations of criteria chemicals in the action area that are directly related to the action under the RPA. These concentrations of chemicals are likely to cause deaths and injuries of the listed species. These concentrations are also likely to cause habitat degradation that will result in the death or injury of listed species by reducing the availability of suitable prey organisms and thereby significantly impairing the essential behavioral pattern of feeding. All life stages are likely to be affected due to direct exposure of adults and/or juveniles to the chemicals and to latent effects on gametes following exposure of gravid adults. For the reasons set forth in the RPA section (section 2.10), incidental take of Southern Resident killer whales is not likely and therefore killer whales are not included within this ITS.

[18] NMFS has not adopted a regulatory definition of harassment under the ESA. The World English Dictionary defines harass as "to trouble, torment, or confuse by continual persistent attacks, questions, etc." The U.S. Fish and Wildlife Service defines "harass" in its regulations as "an intentional or negligent act or omission which creates the likelihood of injury to wildlife by annoying it to such an extent as to significantly disrupt normal behavioral patterns which include, but are not limited to, breeding, feeding, or sheltering (50 CFR 17.3). The interpretation we adopt in this consultation is consistent with our understanding of the dictionary definition of harass and is consistent with the Service's interpretation of the term.

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Incidental take caused by the habitat-related effects of this action cannot be accurately quantified as a number of fish to be taken, because the number of fish at a given location at a given time are affected by myriad abiotic and biotic factors such as habitat quality and availability, competition, and predation, as well as interactions among these factors. These factors interact in ways that may be random or directional, and may operate across broader temporal and spatial scales that are affected by the proposed action. Thus, the distribution and abundance of fish within the action area cannot be attributed entirely to habitat conditions, nor can NMFS precisely predict the number of fish that are reasonably certain to be injured or killed due to habitat degradation related to the proposed action. Also, there is no feasible way to count, observe, or determine the number of fish that would be injured or killed by exposure to compounds listed in Table 1.1. This is because (1) the effects of the action would take place over a large geographic area (the action area for this consultation covers approximately 90,000 square miles, including the nearshore environment of the Pacific Ocean along the Oregon coast), and most injuries or deaths are likely to occur in areas where fish cannot be observed (*e.g.*, deep water or remote areas); (2) even if injured or dead fish were observed, it would be difficult or impossible in many cases to determine an exact cause of injury or death; and (3) sublethal effects of the proposed action could manifest later in time at locations where they could not readily be observed (*e.g.*, the Pacific Ocean).

In this case, NMFS will use quantitative measurements of ambient concentrations of ammonia and copper as surrogates for the amount of incidental take due to the action under the RPA. Ammonia and copper are suitable surrogates for the amount of incidental take for several reasons. Both chemicals are commonly discharged throughout the action area. These were among the most toxic chemicals analyzed by NMFS, and therefore they are likely to contribute significantly to incidental take. As described in the effects analysis, exposure to these chemicals is likely to cause chronic toxic effects at criterion concentrations that are reasonably certain to result in eventual death or injury of some individuals of the listed species considered in this opinion. There is abundant data about how both chemicals affect fish and invertebrate species that may be prey items. Although many of the criteria chemicals under the RPA action may be discharged at or below levels that can be accurately measured with current analytical methods, ammonia and copper concentrations that are likely to cause sublethal, adverse effects on the ESA-listed species are readily measurable. Because of similar fate and transport pathways (particularly with respect to copper and other metals), concentrations of ammonia and copper are likely to correlate reasonably well with concentrations of other criteria compounds and can thereby serve as surrogates for the overall extent of take indicator.

The NMFS selected the chronic criterion concentrations for ammonia and copper because, as compared to the acute concentrations, they provide a more continuous environmental concentration that could be monitored over the long term at the scale of the stream/river reach or watershed. Acute concentrations are more likely to be exceeded in highly localized areas for short periods of time, and therefore would be difficult to detect by monitoring designed to determine trends at larger scales of time and space that are needed to assess the overall extent of take. Also, exceedences of chronic concentrations in many cases likely will result in exceedences of acute concentrations.

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The NMFS proposes to use the ambient water quality monitoring network program of the DEQ to determine whether the extent of take is exceeded. The DEQ monitors a fixed station network of 131 sites on more than 50 large rivers and streams across the state in its ambient program.[19] These sites, shown in Figure 2.11.1.1., cover 4th order and larger rivers in 16 basins delineated by the DEQ. Some of these basins are inhabited by only one ESA-listed species considered in this opinion, some are inhabited by more than one ESA-listed species, and some are not inhabited by ESA-listed species (*e.g.*, the Powder and Malheur basins). The DEQ selected these sites to represent all major rivers in the state and provide statewide geographical representation. The sites are primarily "integrator" sites, meaning they reflect the integrated water quality effects from point and nonpoint source activities as well as the natural geological and hydrological factors for the watershed. Larger river basins have multiple sites, which may be based on tributaries, land use changes, topographical changes, ecoregions, point sources, and nonpoint sources.

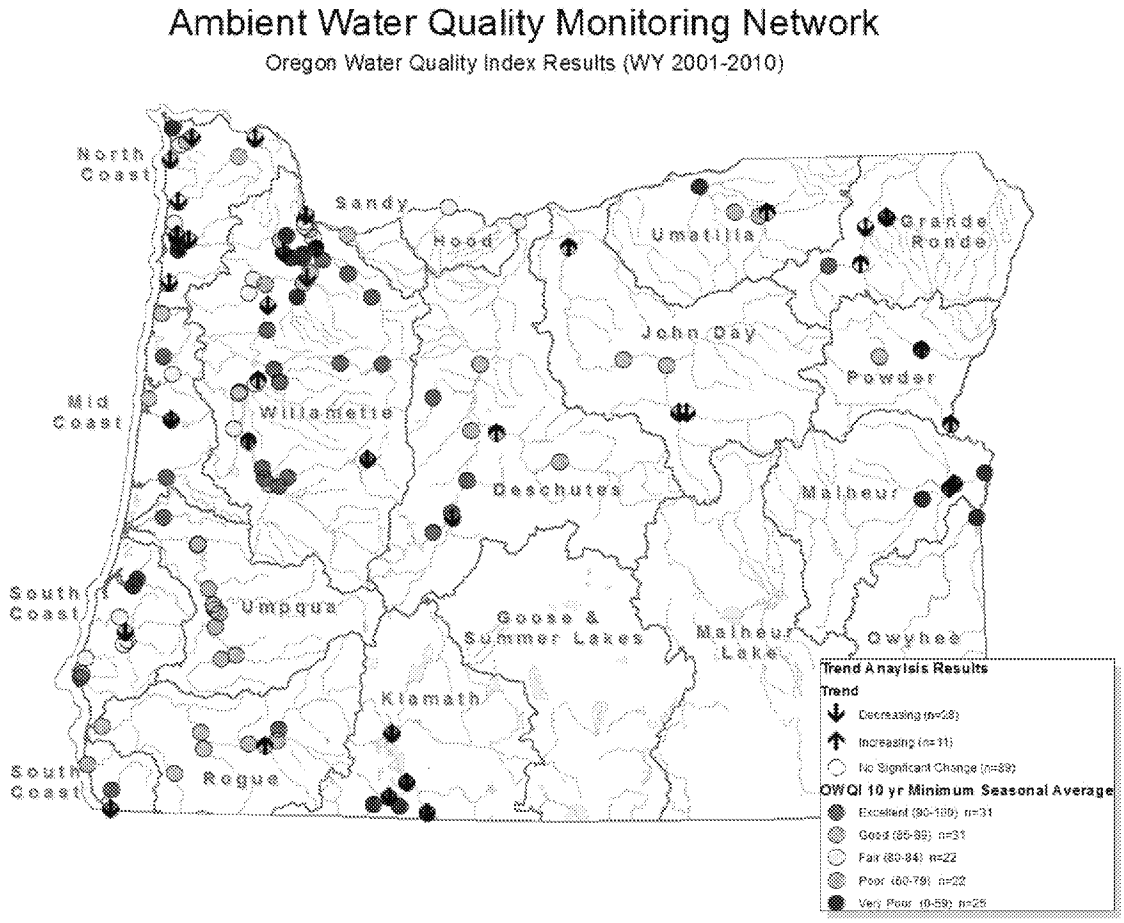

Figure 2.11.1.1. Fixed stations in the ambient water quality monitoring network of the DEQ. Text box in lower right is not relevant to the incidental take statement.

[19]Telephone discussion between Jeff Lockwood, NMFS, and Aaron Borisenko, DEQ, August 7, 2012.

The DEQ uses its ambient monitoring program to understand trends in Oregon's water quality over time, determine whether there is too much pollution in a water body, and set limits of how much pollution a water body can safely receive. The DEQ regularly samples sites within the action area for this consultation. At its ambient monitoring sites, DEQ monitors ammonia concentrations, but it does not currently monitor concentrations of any metals.

In order to comply with this incidental take statement, EPA will need to ensure that monitoring for ambient concentrations of ammonia and copper occurs at DEQ sample sites consistent with the final monitoring plan that will be developed within 12 months of the signing of this opinion. The EPA shall ensure that implementation of the monitoring plan (which will incorporate both the ammonia and the copper criteria) within 6 months of when EPA approves the new criteria for ammonia and copper.

The extent of take for a given ESA-listed species will be exceeded if, in any given DEQ fourth-field or larger USGS hydrologic unit code watershed (as delineated and labeled in Figure 2.11.1.1) that is inhabited by that species, the median value of the valid results for freshwater samples taken in that watershed for ammonia or copper are higher than the threshold values of 0.76 mg/L at pH 8 and 20°C for ammonia, or 1.45 µg/L for copper, respectively, for two consecutive sampling periods. As recognized in the biological opinion, there will be a time lag between establishment of the criteria and incorporation within the terms of all NPDES permits in the state.

To account for this lag period in the event of an exceedence in a given watershed, the extent of take indicator will be triggered only when at least 75 percent of the watershed NPDES permits have been issued under the new criteria. This approach is necessary because it would be unreasonable to assume that all NPDES permits will incorporate the new criteria until existing permits written under the old criteria are renewed over the 5-year permit cycle.

Although the extent of take indicators are the same as the revised criteria for freshwater chronic ammonia and copper, they nevertheless will function as an independent trigger for reinitiation of consultation, because establishing the criteria does not ensure that the criteria always will be met. As the State of Oregon's current CWA section 303(d) list illustrates, waters within the state for various reasons can exceed established water quality standards. The chosen surrogates of chronic copper and ammonia measured as specified above will function to establish predetermined instances where monitored watersheds exceed established levels of toxic compounds and therefore the effects analysis of this biological opinion.

2.11.2 Effect of the Take

In section 2.10, NMFS determined that the anticipated level of incidental take, coupled with other effects of the proposed action, is not likely to result in jeopardy to the species or destruction or adverse modification of critical habitat when the RPA is implemented.

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2.11.3 Reasonable and Prudent Measures

"Reasonable and prudent measures" are nondiscretionary measures to minimize the amount or extent of incidental take (50 CFR 402.02). The following measures are necessary and appropriate to minimize the impact of incidental take of listed species from the proposed action.

1. The EPA shall monitor and report to NMFS on the implementation of the RPA.
2. The EPA shall ensure completion of the monitoring and reporting program to ensure that the extent of take is not exceeded, and to confirm that the terms and conditions in this incidental take statement are effective in avoiding and minimizing incidental take.

2.11.4 Terms and Conditions

The terms and conditions described below are non-discretionary, and the EPA must comply with them in order to implement the reasonable and prudent measures (50 CFR 402.14). The EPA has a continuing duty to monitor the impacts of incidental take and must report the progress of the action and its impact on the species as specified in this incidental take statement (50 CFR 402.14). If the EPA does not comply with the following terms and conditions, the protective coverage of section 7(o)(2) likely will lapse.

1. To implement reasonable and prudent measure # 1 (monitoring the implementation of the RPA) the EPA shall:
    a. Implement oversight of the State of Oregon's NPDES program to ensure that the NPDES permit protocols are implemented in a manner consistent with the EPA technical support document (EPA 1991) and that (a) the State of Oregon is renewing NPDES permits in a manner consistent with the Clean Water Act and its implementing regulations; and (b) the numeric criteria proposed for approval by EPA, as well as any numeric criteria that change when derived by EPA or adopted by the State of Oregon consistent with the RPA, are being implemented in all new and renewed NPDES permits.
    b. Provide NMFS with annual reports on the monitoring requirements by October 31 of each year, for a minimum of 10 years from the date of EPA's final action under the Clean Water Act on Oregon's proposed criteria. Each of these reports shall include:
        i. An assessment of whether or not the State of Oregon is renewing all NPDES permits within the normal 5-year renewal period.
        ii. An assessment of the extent to which the State of Oregon is implementing the numeric criteria proposed for approval by EPA, as well as any numeric criteria that change when derived by EPA or adopted by the State of Oregon consistent with the RPA, in new and renewed NPDES permits.

2. To implement reasonable and prudent measure #2 (monitoring and reporting program) the EPA shall:
    a. Work with NMFS and the DEQ to develop a plan to collect, analyze and summarize the data on ambient concentrations of ammonia and copper in all freshwater monitoring sites in the DEQ's ambient monitoring network that are in

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streams or rivers inhabited by ESA-listed species. The monitoring plan shall be finalized no later than 12 months from the date of this opinion.

b. Ensure that sampling, analysis and reporting the monitoring for ambient concentrations of ammonia and copper at the DEQ sample sites begins within 6 months of when EPA approves the new criteria for ammonia and copper.
c. After monitoring and reporting begin, notify NMFS if any of the incidental take thresholds described in this incidental take statement are exceeded within 1 month of receiving the information from the DEQ.
d. Provide NMFS with annual reports on the monitoring requirements by October 31 of each year, for a minimum of 10 years from the date of EPA's final action under the Clean Water Act on Oregon's proposed criteria. Each of these reports shall include a summary of the results of the monitoring of ambient concentrations of ammonia and copper (as described in term/condition 1.b. above).

2.12 Conservation Recommendations

Section 7(a)(1) of the ESA directs Federal agencies to use their authorities to further the purposes of the ESA by carrying out conservation programs for the benefit of the threatened and endangered species. Conservation recommendations are discretionary measures suggested to minimize or avoid adverse effects of a proposed action on listed species or critical habitats, or regarding development of additional information. The following conservation recommendations are discretionary measures that are suggestions regarding discretionary measures to minimize or avoid adverse effects of a proposed action on listed species or critical habitat or regarding the development of information (50 CFR 402.02) consistent with these obligations, and therefore should be carried out by the EPA for the proposed action:

1. To improve the potential for recovery of listed species in the State of Oregon, the EPA should carry out management actions to reverse threats to survival as identified in the Columbia River Basin recovery plans for salmon and steelhead, the SONCC coho salmon recovery plan, and futire recovery plans for green sturgeon and eulachon.

2. The EPA should replace the fixed duration LC_{50} acute toxicity tests used for criteria development with acute toxicity tests based on exposure-response curves to describe the relationship between exposure and toxicological effects, and EPA should replace the current chronic tests, *i.e.*, hypothesis testing, used for criteria development with chronic toxicity tests based on exposure-response curves to describe the relationship between exposure and toxicological effects.

3. The EPA should work with the State of Oregon to develop a monitoring protocol for toxic pollutants that establishes a consistent monitoring program across the state, and is designed to measure, in real-time, whether or not a particular point-source discharger is in compliance with the aquatic life criteria.

4. The EPA should work with the State of Oregon to minimize effects from chemical mixtures and decrease mixing zone dimensions such that no mixing zones overlap in space and time, or impact more than 5 percent of the cross-sectional area of the affected

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waterbody , and are calculated using the "one-day, once in ten year low flow" (1Q10) statistic or its equivalent.

2.13 Reinitiation of Consultation

As provided in 50 CFR 402.16, reinitiation of formal consultation is required where discretionary Federal action agency involvement or control over the action has been retained, or is authorized by law, and if: (1) the amount or extent of incidental take is exceeded, (2) new information reveals effects of the agency action on listed species or designated critical habitat in a manner or to an extent not considered in this opinion, (3) the agency action is subsequently modified in a manner that causes an effect on the listed species or critical habitat not considered in this opinion, or (4) a new species is listed or critical habitat designated that may be affected by the action.

To reinitiate consultation, contact the Oregon State Office Habitat Office of NMFS and refer to NMFS Number **2008/00148**.

2.14 Not Likely to Adversely Affect Determinations

In this opinion NMFS concludes that the proposed action is not likely to adversely affect (NLAA) Steller sea lions, humpback whales, blue whales, fin whales, Sei whales, sperm whales, North Pacific Right whales, loggerhead sea turtles, green sea turtles, leatherback sea turtles, or Olive Ridley sea turtles.

The above identified marine mammal and sea turtle species are distributed in coastal areas and may be exposed to effects related to the proposed numeric criteria. Similar to Southern Resident killer whales, effects would be indirect and would include reduced prey availability, reduced prey quality, and potential accumulation in the individuals exposed. However, the occurrence of the subject ESA-listed sea turtles and large whales would be rare, infrequent, and transitory in the action area. For example, the blue whale and Sei whale are likely to have limited exposure to contaminant sources as their migratory patterns are circumglobal with definite seasonal movements to offshore areas outside the likely extent of effects. In the event that the turtles and large whales are present, they would be unlikely to accumulate a significant amount of persistent pollutants because they primarily consume lower trophic-level prey. Thus, sea turtles and large whales are unlikely to accumulate significant levels of contaminants in the action area that would be a cause for concern.

Steller sea lions of the eastern DPS occur in Oregon waters throughout the year, with breeding rookeries on offshore rocks and islands and haulout locations on and offshore along the coast and in the Columbia River (Table 2.14.1). Steller sea lions are not known to predictably occur along coastal reaches, in coastal bays or in river systems of Oregon aside from areas proximate to their haulout and rookery locations and their seasonal occurrence in the lower Columbia River and Rogue River. Steller sea lions are generalist predators that eat a variety of fishes and cephalopods, including salmon (NMFS 2008k). It is likely that Steller sea lions will be exposed to pollutants from the proposed numeric criteria through ingestion of prey; however, the extent of likely exposure is difficult to determine. Unlike Southern Resident killer whales that consume

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primarily salmonids (which are highly contaminated. upper-trophic level prey), Steller sea lions have a large foraging base and consume prey at a relatively lower trophic level (*i.e.*, Steller sea lions are likely exposed to less-contaminated prey than the Southern Resident killer whales are). There is limited information on the contaminant levels in Steller sea lions. Heavy metal concentrations in Steller sea lions are generally lower than northern fur seals (Noda *et al.* 1995, Beckmen *et al.* 2002). Overall, studies suggest a decline in contaminant concentrations over time, which is consistent with that reported for other wildlife species (NMFS 2008k). Additionally, comparable levels of zinc, copper, and metallothionein were measured in pups from both the eastern and western Steller sea lion DPSs (Castellini and Cherian 1999). Although these studies are not comprehensive, they indicate that heavy metals were not likely a significant factor in the decline of the Steller sea lions (NMFS 2008k). However, the population has grown steadily for the past 20 to 30 years, with no indication that contaminant-induced health effects are limiting recovery. For these reasons, the potential for exposure to contaminants from ingesting contaminated prey and for any subsequent chance of bioaccumulation of contaminants in Steller sea lions are likely to be insignificant.

The proposed action may reduce the quantity of prey available, due to the incidental take of salmon, green sturgeon, and eulachon. The NMFS anticipates similar effects on non-listed species that may be prey items for the subject listed species. Any salmonid take up to the aforementioned maximum extent and amount would result in an insignificant reduction in prey resources for marine mammals that may intercept these species within their range.

The NMFS finds that all effects of the action are likely to be discountable or insignificant, and therefore concludes that the proposed action is not likely to adversely affect Steller sea lions, humpback whales, blue whales, fin whales, Sei whales, sperm whales, North Pacific Right whales, loggerhead sea turtles, green sea turtles, leatherback sea turtles, or Olive Ridley sea turtles.

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Table. 2.14.1 Steller Sea Lion Haulout and Rookery Locations in Oregon Waters (ODFW 2010).

Marine Location	Haulout Site	Count/Use	Latitude/Longitude[1]	Rookery?
Columbia River	Tip of the South Jetty	>500 Common	46.2338 / -124.0702	
	East Mooring Basin	<10 Rare	46.1963 / -123.8006	
	Phoca Rock	10-<100 Occasional	45.5720 / -122.1820	
	Bonneville Dam, Tailrace	10-<100 Occasional	45.6450 / -121.9480	
Tillamook Head	Tillamook Rock, Offshore from Tillamook Head	500-1,000 Common	45.9368 / -124.0185	
Ecola Point	Ecola Point	<10 Rare	45.9185 / -123.9805	
Three Arch Rocks	Three Arch Rocks	10-<100 Common	45.4637 / -123.9833	Yes
Cascade Head	Sea Lion Cove_2	10-<100 Common	45.0692 / -124.0085	
	Sea Lion Cove_3	100-500 Common	45.0670 / -124.0123	
Seal Rock	North Offshore	10-<100 Occasional	44.5022 / -124.0943	
Cape Arago	Simpsons Reef	10-<100 Common	43.3137 / -124.4082	
	Shell Island Area	100-500 Common	43.3133 / -124.4013	
Blanco Reef	Blanco Reef	100-500 Common	42.8239 / -124.5836	
Orford Reef	Large Brown Rock	10-<100 Common	42.7922 / -124.6008	
	Long Brown Rock	>500 Common	42.79136 / -124.6060	Yes
	Best Rock	100-500 Common	42.7906 / -124.5955	
	Square White Rock	10-<100 Occasional	42.7882 / -124.6048	
	Seal Rock (Orford Reef)	100-500 Common	42.7870 / -124.5946	Yes
	Miscellaneous (Orford Reef)	10-<100 Occasional	42.7825 / -124.6047	
	Arch Rock	100-500 Common	42.7784 / -124.5974	Yes
	West Conical Rock	100-500 Common	42.7774 / -124.6010	Yes
	Steamboat Rocks	10-<100 Common	42.7760 / -124.6041	
Rogue Reef	Double Rock	10-<100 Common	42.4494 / -124.4901	
	Needle Rock	100-500 Common	42.4484 / -124.4837	Yes
	Pyramid Rock- Miscellaneous	10-<100 Common	42.4467 / -124.4695	
	Miscellaneous (Rogue Reef)	10-<100 Common	42.4455 / -124.4793	
	Pyramid Rock	>500 Common	42.4441 / -124.4693	Yes
	Southern Seal Rock (Rogue)	10-<100 Common	42.4365 / -124.4652	
Crook Point	Crook Point	10-<100 Occasional	42.2453 / -124.4141	

[1]Latitude and longitude reported in decimal degrees.
Source: ODFW.

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Critical Habitat

Steller Sea Lion and Leatherback Turtle. The NMFS designated critical habitat for the Steller sea lion in certain areas and waters of Alaska, Oregon and California on August 27, 1993 (NMFS 1993). Certain rookeries, haulouts, and associated areas with essential prey resources for at least lactating adult females, young-of-the-year, and juveniles were designated as critical habitat. In Oregon, these areas include Long Brown Rock and Seal Rock at Orford Reef and Pyramid Rock at Rogue Reef. There are no "special aquatic foraging areas" identified as critical habitat in Oregon. Critical habitat includes air zones extending 3,000 feet above the terrestrial and aquatic zones, and aquatic zones extending 3,000 feet seaward from the major rookeries and haul-outs.

Designated critical habitat for leatherback sea turtles in the action area includes one 24,500 square-mile marine area stretching from Cape Flattery, Washington, to the Umpua River, Oregon. The PCEs that NMFS identified as essential for the conservation of leatherback sea turtles when it proposed to revise critical habitat to include marine waters off the U.S. West Coast include: (1) A sufficient quantity and quality of their jellyfish prey; and (2) migratory pathway conditions that allow for safe and timely passage to, from, and within high-use forage areas.

Based on the best scientific and commercial data available, as discussed previously, NMFS does not expect that the proposed action would adversely affect the quantity, quality, or availability of any of the constituent elements of critical habitat, or the physical, chemical, or biotic phenomena that give the designated area value for the conservation of the species when no constituent elements were identified in the designation. Although NMFS would expect critical habitat for Eastern Steller sea lions and proposed critical habitat for the leatherback sea turtle to be exposed to toxic chemicals due to the proposed action, the concentrations would be sufficiently low that the effects would be insignificant. Critical habitat for green sea turtles does not occur in the action area.

The NMFS finds that all effects of the action are likely to be insignificant, and therefore concludes that the proposed action is not likely to adversely affect Steller sea lion and leatherback turtle critical habitat.

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3. DATA QUALITY ACT DOCUMENTATION AND PRE-DISSEMINATION REVIEW

Section 515 of the Treasury and General Government Appropriations Act of 2001 (Public Law 106-554) (Data Quality Act) specifies three components contributing to the quality of a document. They are utility, integrity, and objectivity. This section of the opinion addresses these Data Quality Act (DQA) components, documents compliance with the DQA, and certifies that this opinion has undergone pre-dissemination review.

3.1 Utility: Utility principally refers to ensuring that the information contained in this consultation is helpful, serviceable, and beneficial to the intended users. The intended users are EPA and the State of Oregon.

An individual copy was provided to EPA. This consultation will be posted on the NMFS Northwest Region website (http://www.nwr.noaa.gov). The format and naming adheres to conventional standards for style.

3.2 Integrity: This consultation was completed on a computer system managed by NMFS in accordance with relevant information technology security policies and standards set out in Appendix III, 'Security of Automated Information Resources,' Office of Management and Budget Circular A-130; the Computer Security Act; and the Government Information Security Reform Act.

3.3 Objectivity:

Information Product Category: Natural Resource Plan.

Standards: This consultation and supporting documents are clear, concise, complete, and unbiased; and were developed using commonly accepted scientific research methods. They adhere to published standards including the NMFS ESA Consultation Handbook, and the ESA Regulations, 50 CFR 402.01, *et seq*.

Best Available Information: This consultation and supporting documents use the best available information, as referenced in the Literature Cited section. The analysis in this opinion contains more background on information sources and quality.

Referencing: All supporting materials, information, data and analyses are properly referenced, consistent with standard scientific referencing style.

Review Process: This consultation was drafted by NMFS staff with training in ESA implementation, and reviewed in accordance with Northwest Region ESA quality control and assurance processes.

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APPENDIX 1: EPA's Guidelines for Deriving Numerical National Water Quality Criteria and Issues Common to All Criteria

The following discussion and analysis examines the shortcomings of EPA's methodology for deriving the national criteria and is critical to understanding the relationship between the numeric criteria and the exposure-response analysis in this opinion. The discussion and analysis in this Section is separated into two main categories: (1) EPA's methodology for deriving the national aquatic life criteria, and (2) overview of the effects assessment methodology in EPA's BE for the Oregon criteria.

Derivation of EPA Aquatic Life Criteria

The foremost problem with EPA's national aquatic life criteria lies with the derivation methodology, which is set out in EPA's *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (Stephan *et al.* 1985) (Guidelines). The extent of technical issues delineated in this section regarding the Guidelines produces far more uncertainty than predictability regarding the reliability of the criteria to protect aquatic life, and in particular, listed species. This analysis highlights the risks associated with use of the Guidelines and assesses how they are likely to influence the chemical and environmental stressors affecting the listed species evaluated in this opinion.

First, we look at EPA's general approach as described in the Guidelines. Second, we look at the risks or conservatisms associated with EPA's approach. Third, we provide a summary that qualitatively assesses the degree of uncertainty and likely influences on the effects associated with exposure-response risks to the listed species considered in this opinion.

The derivation methodology for EPA's water quality criteria, the basis of Oregon's proposed water quality criteria, is detailed in the Guidelines (Stephan *et al.* 1985). An overview of the Guidelines, as described in EPA's BE, is presented below.

> The first stage in deriving water quality criteria is to compile the available data on the chemical of interest regarding its toxicity to and bioaccumulation by aquatic animals and plants. These data then go through a review process to identify studies that should not be used to derive national criteria. Although there are a number of reasons why data are not included in the data sets used to develop national criteria, some of the more common ones are that one or more pieces of information regarding study methodology or calculation of results needed to assess the reliability of the study is missing; data quality of the study is less than acceptable (*e.g.* unacceptably high control mortality); the tested species does not have a reproducing population in North America; the test species was exposed to a chemical mixture or was previously exposed to the test chemical; the study reported effects on an endpoint other than survival, reproduction of growth; or the test duration was a non-standard test duration (*e.g.* fish toxicity test reporting a 24-hr LC_{50} instead of the more standard 96-hr LC_{50}).

Once the available data have been reviewed and unacceptable or inappropriate study results have been removed from the data set, the data are reviewed to ensure that certain types of data are available. Specifically, for freshwater aquatic biota, the following eight types of toxicity data should be available:

- Data for a fish species in the family *Salmonidae* of the class *Osteichthys*
- Data for a fish species from a second family in the class *Osteichthys*
- Data for a third family in the phylum *Chordata* (may be a third fish species or an amphibian species)
- Data for a planktonic crustacean species
- Data for a benthic crustacean species
- Data for an aquatic insect species
- Data for a species in a phylum other than *Arthropoda* or *Chordata* (*e.g. Rotifera*, *Annelida*, *Mollusca*, etc.)

Data for a species in any family in any order of insect or any aquatic phylum not already represented.

Additionally, the following three other pieces of information are needed before a national water quality criterion can be developed for a given chemical (required to derive both freshwater and saltwater criteria). Unlike toxicity data, which must be from exposures of species to chemicals in freshwater in order to derive freshwater criteria, the following information can be either for freshwater data only or a specified mixture (Stephan *et al.* 1985) of freshwater and saltwater data.
Acute-chronic ratios (ACRs) for at least three different families of aquatic species. Toxicity data for at least one freshwater plant (can be either algal or a vascular plant)
At least one bioconcentration factor (BCF).

The eight taxa for which saltwater toxicity data are required prior to derivation of a saltwater criterion obviously differ from those for freshwater, and must be from the taxonomic groupings listed below:

- Data from two families in the phylum *Chordata*
- Data from a family in a phylum other than *Arthropoda* or *Chordata*
- Data from a species in either the *Mysidae* or *Penaeidae* family
- Data from three other families not in the phylum *Chordata* (may include data for a species from a phylum or family listed in taxa groups 1 - 3 above but which was not used)
- Data from any other saltwater family

Ideally, the above freshwater and marine species toxicity data have both LC_{50} data of appropriate duration and chronic NOEC data available. In practice, most chemicals with water quality criteria have sufficient LC_{50} data to permit derivation of an acute water quality criterion from measured LC_{50} data, but do not have sufficient measured chronic NOEC to use the above procedure to directly calculate a chronic criterion. Instead, most chronic criterion are calculated by dividing the calculated acute criterion by the available ACR value.

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If toxicity data are available from multiple studies (*e.g.* three LC_{50} results are available for rainbow trout), a species mean acute value (SMAV) (or species mean chronic value if one is deriving a chronic criterion, although the rest of this discussion will assume that only measured acute toxicity data are available) is calculated as the geometric mean of the three available LC_{50} values in this example. Similarly, if two or more LC_{50} results are available for different species of the same genus (*e.g.* LC_{50} data are available for rainbow trout and Chinook salmon, both members of the genus *Oncorhynchus*), a genus mean acute value (GMAV) is calculated from the geometric mean of all toxicity data for members of that genus. If only one LC_{50} value is available for a species from a given genus, that single value becomes both the SMAV and GMAV for subsequent criteria calculations.

Geometric means are used to calculate central tendency species mean, genus mean, ACR and BCF values throughout the development of water quality criteria. This is because toxicity data and ratio data (ACRs and BCFs are ratios) tend to be lognormally distributed instead of normally distributed.

Acute water quality criteria are calculated by rank ordering the GMAV values from the lowest LC_{50} to the highest LC_{50}, and using a formula given in Stephan *et al.* (1985) to estimate the 5th percentile of the resulting species sensitivity distribution (SSD). This 5th percentile of measured GMAVs is termed the final acute value (FAV) in the EPA criteria development documents. As a criterion based on a concentration causing mortality to 50 percent of a test species would not be a protective criterion, the FAV is divided by two to convert LC_{50} values to concentrations expected to cause little or no mortality to test species. The FAV divided by two value becomes the EPA acute water quality criterion unless a commercially or recreationally important species, or an ESA listed species has a GMAV lower than the calculated water quality criterion. In these cases, the results of one or more individual species GMAVs is used to directly calculate an acute criterion.

If sufficient chronic NOEC data are available for the freshwater and/or saltwater taxa described earlier, the same approach described above is used with the measured NOEC data to calculate a final chronic value (FCV) from the 5th percentile of the NOEC data. Final chronic values are not divided by two to obtain the chronic criterion, as unlike LC_{50} data, NOEC values are already assumed to be concentrations that have no adverse effects on survival, reproduction and growth of the tested species. Much more common is the situation where the calculated acute criterion is divided by an acute-chronic ratio (ACR) to obtain the chronic criterion.

Additional details of the Guidelines to develop national water quality criteria and the assumptions that go into their derivation are provided in Stephan *et al.* (1985). Of all the assumptions that are made during the derivation of EPA water quality criteria, perhaps the most critical is that the species sensitivity distribution of

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measured toxicity data used during the calculation of criteria values is representative of the range of toxicity of a chemical to all aquatic species. There are over 700 species of freshwater fish alone in North America, making it impractical to perform toxicity tests on all species with all chemicals for which criteria exist.

Water quality criteria calculated from the methodology described above have several levels of conservatism built into them, including:

- protection of 95 percent of all aquatic genera
- division of the 5th percentile of all genus mean acute values by two during the derivation of acute criteria
- use of no effect concentrations to derive chronic criteria
- short exposure durations at criteria concentrations relative to the lifespan of many aquatic species

However, water quality criteria are not designed to protect all aquatic species from exposure to chemical concentrations that may adversely affect some of the more sensitive species to a given chemical. Nor are criteria designed to protect all individuals of a given species, whether or not that species is a listed species. Despite these design aspects of the national water quality criteria, many of them are protective of more than 95 percent of aquatic genera from adverse effects, and are protective of all ESA listed species known to occur within many discrete geographical areas. ESA listed aquatic species as a group are generally not believed to be more sensitive to chemicals than aquatic species as a whole (Dwyer *et al.* 2005, Sappington *et al.* 2001, Dwyer *et al.* 1999).

The toxic criteria proposed by the State of Oregon for EPA approval are identical to the corresponding national toxic criteria developed by EPA as guidance for the states.

The following section provides NMFS' analysis on the Guidelines.

Risks from Using Acute Criteria Based on LC_{50} Concentrations and the EPA Acute Adjustment Factor. The acute criteria for aquatic life have been primarily based on compilations of toxicity study results reported in terms of the concentration resulting in 50 percent mortality over a fixed time period [usually 96 hours: *e.g.*, LC_{50}, effects concentration $(EC)_{50}$, EPA 1986a]. Although there are a number of reasons why data are not included in the data sets used to develop criteria, some of the more common ones are that one or more pieces of information regarding study methodology or calculation of results needed to assess the reliability of the study is missing; data quality of the study is less than acceptable (*e.g.* unacceptably high control mortality); the test species was exposed to a chemical mixture or was previously exposed to the test chemical; the study reported effects on an endpoint other than survival, reproduction or growth; or the test duration was a non-standard test duration (*e.g.*, fish toxicity test reporting a 24-hr LC_{50} instead of the more standard 96-hr LC_{50}).

The acute criterion is based on acute toxicity tests, *i.e.*, 96-hour LC_{50} toxicity tests, that indicate the concentration at which 50 percent of the test population was killed. However, what is often

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not considered are exposure-related effects such as latent mortality, which can range between 15 and 35 percent greater than the LC_{50} predictions compared to the control (Zhao and Newman 2004, Lee and Lee 2005). Furthermore, because 4- to 8-hour LC_{50}s are about the same as the 96-hour LC_{50} for some compounds, *e.g.*, selenium, lead, arsenic (EPA 1991), criteria concentrations that do not take fast-acting compounds into consideration are likely to bias the magnitude of acute toxic effects. Theses factors create significant uncertainty regarding the reliability and predictability of the acute criterion to represent concentrations that are protective against acute toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on fixed durations instead of an exposure-response curve, and challenge the notion that LC_{50} data that is above the acute criterion is protective against acute toxic effects based soley on a comparison of concentrations.

Acute water quality criteria are calculated by rank ordering the GMAV values from the lowest LC_{50} to the highest LC_{50}, and using a formula given in Stephan *et al.* (1985) to estimate the 5th percentile of the resulting SSD. This 5th percentile of measured GMAVs is termed the FAV in the EPA criteria development documents. As a criterion based on a concentration causing mortality to 50 percent of a test species would not be a protective criterion, EPA divides the FAV by a safety factor of 2.27 (referred to as a factor of 2 in the below analysis) to convert LC_{50} values into concentrations that EPA projects to be near or below lethality.

The database from which the safety factor was derived (actually the safety factor is 2.27) was published in the Federal Register in 1978. Table 10 from the Federal Register notice (43 FR 21506-21518) lumps data for freshwater and marine fish and invertebrates. The data are broken out by the chemicals tested. There are 219 data points, but a large proportion of them aren't for a specific chemical, but rather for whole effluents of various sources—115 of the 219 data points used to derive the acute adjustment factor are based on effluent studies where individual pollutants are not measured. Interestingly, effluent studies are one of EPA's "not pertinent" or "reject" categories identified in EPA (2005).

The assumption that dividing an LC_{50} by 2 will result in effect concentrations near or below leathility rests on further assumptions of the steepness of the concentration-response slope. Several examples of tests with metals which had a range of response slopes are shown in Figure A1. These examples were selected from data sets that were relevant to salmonid species in Oregon and for which the necessary data to evaluate the range of responses could be located (Chapman 1975, 1978b, Marr *et al.* 1995, Marr *et al.* 1999, Mebane *et al.* 2010, Windward 2002). The citations given include both reports with detailed original data as well as the summarized, published forms of the same tests. The examples range from tests with some of the shallowest concentration-response slopes located to very steep response slopes. In the shallowest tests (panels A and E), an $LC_{50/2}$ concentration would still result in 15 to 20 percent mortality.

One challenge for deriving acute criteria for short-term exposures is that the great majority of available data is for mortality; that is, a concentration that kills 50 percent of a test population. A fundamental assumption of EPA's criteria derivation is that the FAV, which is the LC_{50} for a hypothetical species with a sensitivity equal to the 5th percentile of the SSD, may be divided by 2 in order to extrapolates from a concentration that would likely be extremely harmful to sensitive species in short-term exposures (*i.e.*, kill 50 percent of the population) to a concentration

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expected to kill few, if any, individuals. This assumption must be met for acute criteria to be protective of sensitive species. It is difficult to evaluate from published literature if this assumption is met because so few studies report the data behind an LC_{50} test statistic. While LC_{50}s are almost universally used in reporting short-term toxicity testing, they are not something that can be "measured," but are statistical model fits. An acute toxicity test is actually a series of 4 to 6 tests runs in parallel in order to test effects at these (usually) four to six different chemical concentrations. An LC_{50} is estimated by some statistical distribution or regression model, which generates an LC_{50} estimate, and some confidence interval, and then all other information is thrown away. Thus, while the original test data included valuable information on what were no, low and severe effects concentrations, that information is lost to reviewers unless the unpublished, raw, lab data are available. However, a more common pattern with the metals data was that an $LC_{50/2}$ concentration would probably result in about a 5 percent death rate (panels B and F), and in many instances, no deaths at all would be expected (panels C and D).

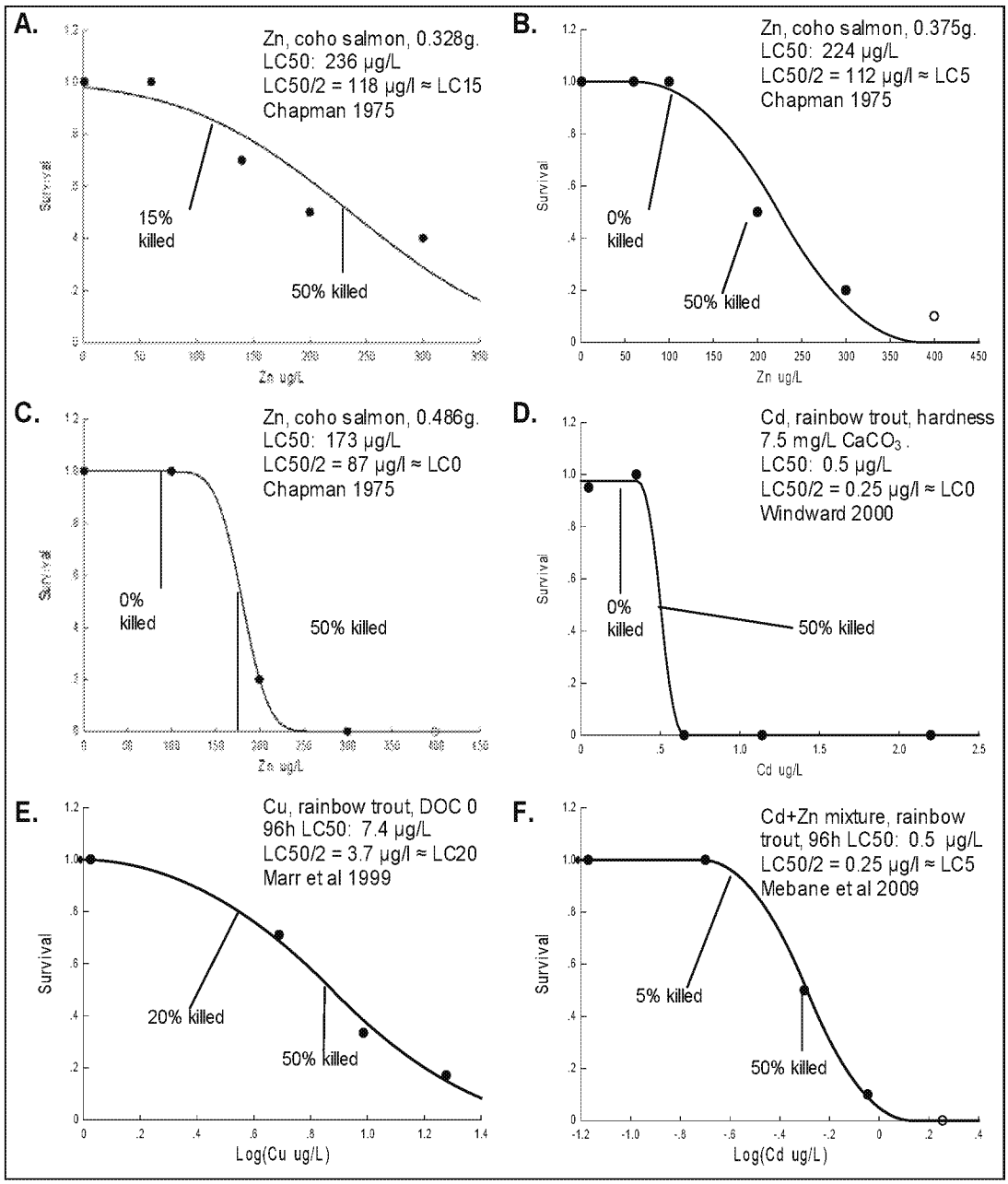

Figure A1. Examples of percentages of coho salmon or rainbow trout killed at one-half their LC_{50} concentrations and at LC_{50} concentrations with cadmium, copper, and zinc.

In one of the few additional published sources that gave relevant information, researchers happened to include effect-by-concentration information on the acute toxicity of chemical mixtures. Rainbow trout and the invertebrate zooplankton *Ceriodaphnia dubia* were exposed for 96 and 48 hours respectively to mixture of six metals, each at their presumptively "safe" acute CMC concentrations. In combination, the CMC concentrations killed 100% of rainbow trout and C. dubia, but 50% of the CMC concentrations killed none (Spehar and Fiandt 1986). This gives some support to the assumption that one-half the FAV divided by 2 is likely to kill a low

percentage of fish, although it raises questions about the overall protectiveness of criteria concentrations in mixtures.

Other relevant reviews include Dwyer *et al.* (2005b), who evaluated the $LC_{50/2}$ assumption with the results of the acute toxicity testing of 20 species with five chemicals representing a broad range of toxic modes of action. In those data, multiplying the LC_{50} by a factor of 0.56 resulted in a low (10%) or no-acute effect concentration. Testing with cutthroat trout and Cd, Pb, and Zn singly and in mixtures, Dillon and Mebane (2002) found that the $LC_{50/2}$ concentration corresponded with death rates of 0 to 15 percent.

Summary: Based on this analysis, there are increased risks to listed species considered in this opinion from using acute criteria based on LC_{50} concentrations and the acute adjustment factor, as acute criteria based on a hazard quotient—the acute adjustment factor, instead of acute toxicity tests that predict in $LC_{near\text{-}zero}$ concentrations, and are based on fixed duration toxicity tests instead of an exposure-response curve, are likely to underestimate the magnitude of effects for field-exposed fishes. Therefore, the risks identified in the above analysis are likely to result in mortality greater than the LC_{50} test predictions and the presumed protection from the acute adjustment factor in deriving acute criteria.

Risks from Using the Chronic Value Statistic in Setting Criteria. An issue of concern with the derivation of the chronic criteria is the test statistic used to summarize chronic test data for species and genus sensitivity rankings. Literature on chronic effects of chemicals often contains a variety of measurement endpoints, different terms, and judgments by the authors of what constitutes an acceptable or negligible effect. While the Guidelines give a great deal of advice on considerations for evaluating chronic or sublethal data (Stephan *et al.* 1985, at p. 39), those considerations were not usually reflected in the individual national EPA-recommended ambient water quality criteria documents NMFS reviewed. In practice, for most of the criteria documents we reviewed, "chronic values" were simply calculated as the geometric mean of the lowest tested concentration that had a statistically significant adverse effect at the 95 percent confidence level (LOEC), and the next lower tested concentration (NOEC). The "chronic value" as used in individual criteria documents is effectively the same thing as the maximum acceptable toxicant concentration[20] (MATC) used in much environmental toxicology literature, even though the MATC term is never used in the Guidelines. This MATC approach has the potential to seriously underestimate effects because the statistical power in typical toxicity tests is fairly low. A bias in many ecotoxicology papers is to focus on avoiding "false accusations" of a chemical with 95 percent accuracy (*i.e.*, Type I error or false positive, the risk of declaring an effect was present when in fact there was no effect). Often no consideration whatsoever is given to the companion problem, known as Type II error, or false negatives (*i.e.*, declaring no adverse effects occurred when in fact they did occur, but because of the limited sample size or variability, they were not significant with 95 percent confidence).

The magnitude of effect that can go undetected with 95 percent confidence in a NOEC statistic can be large (greater than 30 percent on average for some endpoints), and much higher for individual tests (Crane and Newman 2000). This problem is compounded when the "chronic value" or MATC is calculated in its most common form as the geometric mean of a NOEC and

[20] The MATC is the range between the NOEC and LOEC.

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LOEC. For instance, in one study, 100 percent of juvenile brook died after being exposed to 17 µg/L copper for 8 months; this was considered the LOEC for the test. The next lowest concentration tested (9.5 µg/L) had no reduced survival relative to controls. (McKim and Benoit 1971). Therefore, the only thing that can be said about the geometric mean of these two effect concentrations (*i.e.*, the chronic value of 12.8 µg/L that was used in the chronic copper criteria, EPA 1985) is that it represents a concentration that can be expected to kill somewhere between all and no brook trout in the test population. These factors create significant uncertainty regarding the reliability and predictability of the chronic criterion to represent concentrations that are protective against chronic toxic effects. Furthermore, these factors highlight the risks of toxicity data that are based on statistical hypothesis tests instead of an exposure-response curve (because the exposure-response curve describes the relationship between exposure and effect), and challenges the supposition that NOEC data that is above the chronic criterion is protective against chronic toxic effects based solely on a comparison of concentrations. Therefore, NOEC data that is above the chronic criterion does not necessarily ensure that there are no chronic toxic effects, but that the criterion concentration may result in chronic toxic effects to a subset of the test population, and therefore by inference, field-exposed individuals, relative to the criterion concentration in the range of 10 to 34 percent (Crane and Newman 2000). While the range of chronic effects predicted in these studies is likely to be less than or greater than the 10 to 34 percent range depending on compound and species, these studies highlight the inherent flaws associated with chronic toxicity tests, and provide evidence for long-term survival implications for field-exposed fishes.

Suter *et al.* (1987) evaluated published chronic tests with fish for a variety of chemicals and found that, on average, the MATC represented about a 20 percent death rate and a 40% reduction in fecundity. They noted that "although the MATC is often considered to be the threshold for effects on fish populations, it does not constitute a threshold or even a negligible level of effect in most of the published chronic tests. It corresponds to a highly variable level of effect that can only be said to fall between 0 and 90 percent." Barnthouse *et al.* (1989) further extrapolated MATC-level effects to population-level effects using fisheries sustainability models and found that the MATC systematically undervalued test responses such as fecundity, which are both highly sensitive and highly variable.

One implication of this issue is that because the MATC chronic values typically used in the EPA water quality criteria documents for aquatic life criteria may cause a substantial adverse effect for that test species, the criteria on the whole will be less protective than the Guidelines' intended goal of protecting 95 percent of the species. How much less protective is unclear and probably varies among the criteria datasets. One dataset from which a hypothetical NOEC-based chronic criterion could readily be recalculated and compared with the usual MATC criteria was a 2006 cadmium criteria update (Mebane 2006). In this comparison, Mebane determined that the MATC-based chronic criteria would protect about 92 percent of the aquatic species in the dataset at the NOEC level. Because the NOEC statistic also can reflect a fairly sizable effect (Crane and Newman 2000) it may be that at least with cadmium, the true level of protection is closer to about 90 percent than the 95 percent intended by the guidelines.

Summary: Based on this analysis, there are increased risks from using the chronic value statistic in setting criteria is high, as it is likely to result in sublethal effects, such as interference

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in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Risks from the CMC and CCC Duration and Frequency of Exposure. The CMC and the CCC are just two of six parts of an aquatic life criterion; the other four parts are the acute averaging period, the chronic averaging period, acute frequency of allowed exceedence, and chronic frequency of allowed exceedence (EPA 2006), refered to as the concentration-duration-frequency format (EPA 1991).

Concentration (magnitude) refers to how much of a pollutant, expressed as a concentration, is allowable. Duration refers to the period of time (averaging period) over which the instream concentration is averaged for comparison with criteria concentrations. This specification limits the duration of concentrations above the criteria. And, frequency refers to how often criteria can be exceeded (EPA 1991).

The 1-hour CMC averaging period means that the 1-hour average concentration of the compound does not exceed the CMC more than once every three years on the average. In other words, an organism should not be exposed to a pollutant concentration *greater* than the CMC for more than 1 hour, and an exceedence, *i.e.*, a concentration *greater* than the respective CMC, of the CMC 1-hour average concentration should not occur more than once every three years on the average. The 4-day CCC averaging period means the 4-day average concentration of the compound does not exceed the CCC more than once every three years on the average. In other words, an organism should not be exposed to a pollutant concentration *greater* than the CCC for more than 4 days, and an exceedence, *i.e.*, a concentration *greater* than the respective CCC, of the CCC 4-day average concentrations should not occur more than once every three years on the average.

This means that the averaging periods are average concentrations that are measured against the respective numeric parts of the criterion with the purpose being to minimize the duration of exposure above the CMC and CCC criteria concentrations. Figures A2 and A3 provide conceptual examples of the 1-hour and the 4-day chemical averaging periods for acute and chronic criteria, respectively. These figures show that excursions (short term concentrations above the CMC or CCC) can produce concentration "spikes" that, when compared to the available toxicity data, can result in exposure with lethal and sub-lethal responses in listed species, but that the average concentration is below the respective criterion and thus in compliance.

Figures A2 and A3 conceptually represent respective averaging concentrations for acute and chronic criteria. For example, the 1-hour averaging concentration must be evaluated for each hour of the day. That is, the average concentration in the acute example of 55.2 μg/L is a series of continuous (persistent) receiving water concentrations that occurs each hour on a continuum. The same holds true for the chronic average concentration, where the 4-day average concentration in the chronic example of 23.7 μg/L is a series of continuous (persistent) receiving water concentrations that occurs on a continuum. In these examples, the chronic criterion concentration will eventually determine the concentration outside the regulated mixing zone [defined as an area where an effluent discharge undergoes initial dilution and is…an allocated impact zone where water quality criteria can be exceeded as long as acutely toxic conditions are

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prevented (EPA 1991)] boundary, and is a more accurate representation of ambient concentrations outside of regulated mixing zones. Inside regulated mixing zones, water quality criteria are permitted to be higher than criterion concentrations. While a particular toxic criterion must be met at the acute and chronic mixing zone boundaries, mixing zone boundaries vary with flow and discharge. For example, based on publically-available information from ODEQ analyzed by NMFS in this consultation, in the Willamette River mixing zone size varies greatly from a low of 1,089 square feet to a high of 1,000,000 square feet (n=19). So, meeting the aquatic life criteria at the edge of the mixing zone is a misleading protective assumption.

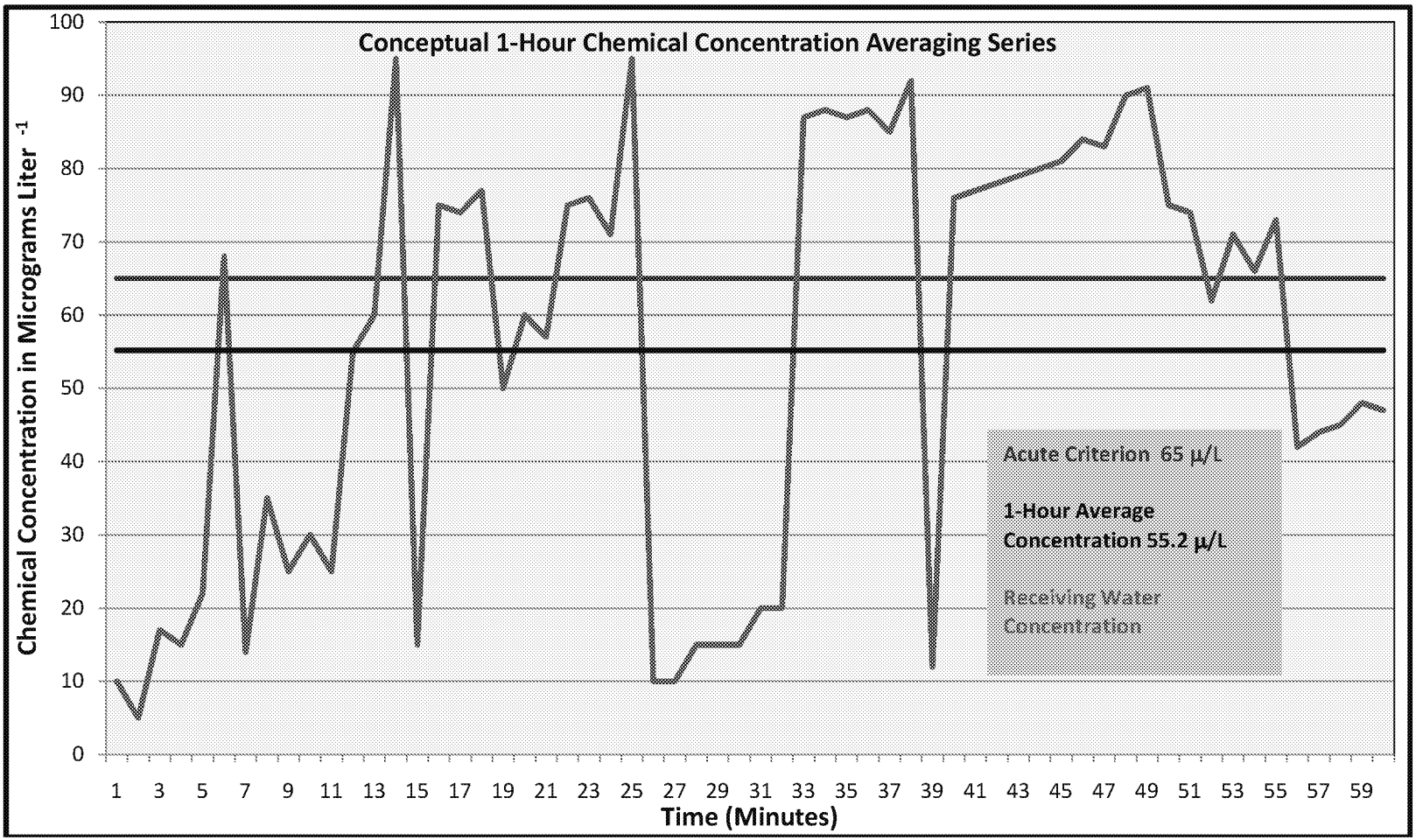

Figure A2. Conceptual concentration averaging series for acute criteria.

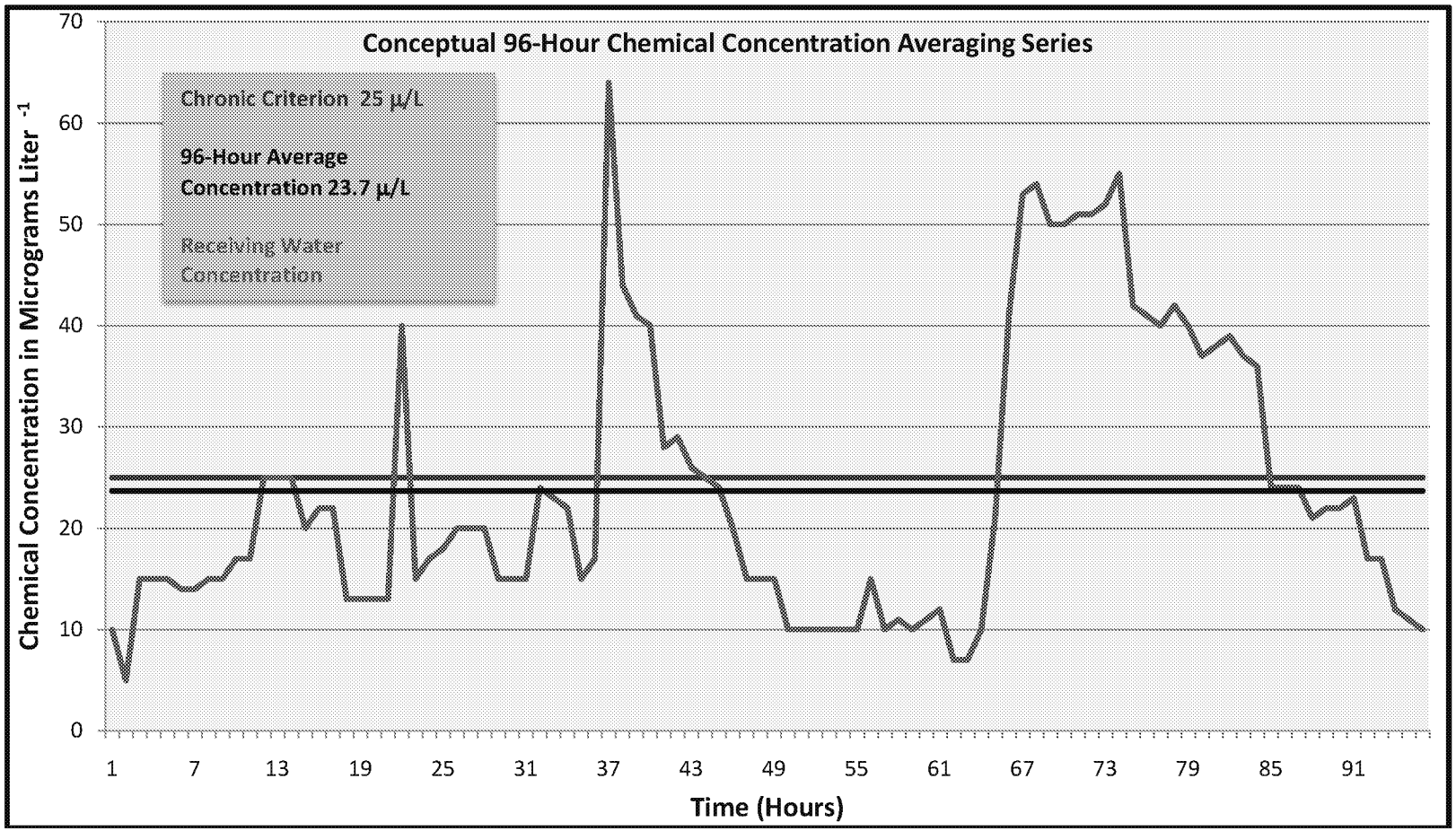

Figure A3. Conceptual concentration averaging series for chronic criteria.

Outside regulated mixing zones, chemical concentrations are theoretically lower than the proposed criteria, especially the acute criteria. However, waters that are 303(d)-listed for toxics do not meet water quality standards for toxics. So the assumption of lower concentrations at the edge of mixing zones is not met. That is, there is no assimulative capacity outside mixing zones.

The 1-hour and 4-day durations and averaging periods for criteria were based upon judgments by EPA authors that included considerations of the relative toxicity of chemicals in fluctuating or constant exposures. EPA's (1985) Guidelines considered an averaging period of one hour most appropriate to use with the criterion maximum concentration or (CMC or acute criterion) because high concentrations of some materials could cause death in one to three hours. Also, even when organisms do not die within the first few hours, few toxicity tests continue to monitor for delayed mortality after the exposure period is over. Thus it was not considered appropriate to allow concentrations *above* the CMC for more than one hour (Stephan *et al.* 1985). Recent criteria documents (*e.g.*, USEPA 2007) have used an averaging period of 24 hours for their CMC, although no explanation could be found for the deviation from the 1985 Guidelines.

A review of more recent information did not contradict these judgments. Some of the more relevant research relates the rapid accumulation of metals on the gill surfaces of fish to their later dying. When fish are exposed to metals such as cadmium, copper, or zinc, a relatively rapid increase occurs above background levels of metal bound to the gill. This rapid increase occurs on the order of <3 to 24 hours, and this brief exposure has been sufficient to predict toxicity at 120 hours (Di Toro *et al.* 2001, MacRae *et al.* 1999, Playle 1998, Playle *et al.* 1993). Acute exposures of 24-hours might not result in immediate toxicity, but deaths could result over the next few days. Simple examination of the time-to-death in 48 or 96 hour exposures would not detect latent toxicity from early in the exposures. Observations or predictions of appreciable mortality resulting from metals exposures on the order of only three to six hours supports the earlier recommendations by Stephan *et al.* (1985) that the appropriate averaging periods for the CMC is on the order of one hour.

The 4-day averaging period for chronic criteria was selected for use with the CCC for two reasons (Stephan *et al.* 1985): First, "chronic" responses with some substances and species may not really be due to long-term stress or accumulation, but rather the test was simply long enough that a briefly occurring sensitive stage of development was included in the exposure (Barata and Baird 2000, Chapman 1978a, De Schamphelaere and Janssen 2004, Grosell *et al.* 2006b, Mebane *et al.* 2008). Second, a much longer averaging period, such as 1 month would allow for substantial fluctuations above the CCC. Substantial fluctuations may result in increased adverse effects from those expected in constant exposures. A comparison of the effects of the same average concentrations of copper on developing steelhead, *Oncorhynchus mykiss*, that were exposed either through constant or fluctuating concentrations found that steelhead were about twice as resistant to the constant exposures as they were to the fluctuating exposures (Seim *et al.* 1984). The literature reviewed by NMFS either supports or at least does not contradict the Guidelines' recommendations on averaging periods.

In addition to the averaging periods, the Guidelines recommend for exceedence of the CMCs and the CCCs once every three years, on average. This recommendation was based on a review case studies of recovery times of aquatic populations and communities from locally severe

disturbances such as spills, fish eradication attempts, or habitat disturbances (Yount and Niemi 1990, Detenbeck *et al.* 1992). In most cases, once the cause of the disturbance was lifted, recovery of populations and communities occurred on a time frame of less than three years. The EPA has subsequently further evaluated the issue of allowable frequency of exceedences through extensive mathematical simulations of chemical exposures and population recovery. Unlike the case studies, these simulations addressed mostly less severe disturbances that were considered more likely to occur without violating criteria (Delos 2008). Unless the magnitude of disturbance was extreme or persistent, this three-year period seemed reasonably supported or at least was not contradicted by the information reviewed by NMFS.

A more difficult evaluation is the allowable exceedence magnitude, which is undefined and unlimited by the proposed criteria. Thus, theoretically, a once-per three year exceedence with no defined limits to its magnitude could be infinitely large, and have adverse effects on listed species. This is because environmental data such as chemical concentrations in water are not unpredictable, but can be described with statistical distributions and statements of exceedence probabilities. Commonly with water chemical data and other environmental data, the statistical distributions do not follow the common bellcurve or normal distribution, but have a skewed distribution with more low than high values. This pattern may be approximated with a log-normal statistical distribution (Blackwood 1992, Delos 2008, Helsel and Hirsch 2002, Limpert *et al.* 2001).

An important consideration that is often not addressed in water quality monitoring is the issue of sampling frequency. In order to accurately compare water quality samples with regulatory criteria, samples need to be collected at least at the same frequency as the criteria (*i.e.*, every hour for CMC and every four days for CCC). Otherwise, an exceedence could occur without detection. Samples, however, are not often taken at the specified frequency, and instead exceedence is detected indirectly through observed fish kills.

Summary: Based on this analysis, the duration and frequency parts of an aquatic life criterion seem like reasonable measures to keep the numeric criteria from exceeding criteria concentrations over long periods. However, the issue of excursions, exceedences with no defined limits on magnitude, and water quality monitoring and sampling sufficient to detect exceedences poses adverse risks likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Metals Toxicity and Risks from Using Formula-based Metal Criteria. Pursuant to EPA policy, states may adopt criteria for metals measured as either the amount of metal dissolved in water or the total recoverable amount of metal. For dissolved criteria, water samples are filtered to remove any suspended solids before analysis, and a conversion factor (CF) is applied to add back a fraction of the suspended metal based on assumptions regarding bioavailability. Total recoverable metals criteria are a measurement of the suspended and dissolved amounts added together. In its National Toxics Rule (NTR) (58 FR 31177), EPA originally promulgated criteria for metals as total recoverable metals. Subsequently, EPA issued a new policy for setting water quality criteria for metals measured as dissolved metals and promulgated revised national metals criteria expressed in terms of dissolved metals (60 FR 22228, May 4, 1995). At the same time,

EPA promulgated recommended conversion factors for converting between dissolved and total recoverable criteria. The metals criteria in Oregon are expressed as dissolved metals, meaning that water samples are filtered to remove suspended solids before analysis.

Metals addressed in this consultation include: As, Cd, Cr(III), Cr(VI), Cu, Pb, Ni, Se, Ag, and Zn. The proposed ambient water quality criteria are formula-based, meaning that the criteria vary based on site-specific conditions, for the following metals: As, Cd, Cr(III), Cr(VI), Cu, Pb, Ni, Ag, and Zn. To determine criteria for these metals that are applicable to a given water body, site-specific hardness data must be obtained, input to a formula, and numeric criteria computed. There are three types of site-specific data that may be necessary to determine and/or modify the criterion for a metal at a site: water hardness, conversion factors (CF) and translators, and water effect ratios (WER). The following is a brief description of these types of data.

The general formula for a hardness-based acute (CMC) or chronic (CCC) criterion with respect to total metal concentration (dissolved and particulate) is:

$$\text{CMC or CCC (total recoverable)} = e^{(m[\ln(\text{hardness})]+b)}$$

Note that this is algebraically equivalent to the simpler expression:

$$\text{CMC or CCC (total recoverable)} = K\ (\text{hardness})^m$$

where $K = e^b$. When the m-exponent is close to 1.0, the relationship is approximately linear. Dissolved concentrations are evaluated using a total-to-dissolved CF that is based on the fraction of the metal that was in a dissolved form during the laboratory toxicity tests and that was used to develop the original total based criteria. The appropriate formula is:

$$\text{CMC or CCC (dissolved)} = CF \times e^{(m[\ln(\text{hardness})]+b)} = CF \times K \times (\text{hardness})^m$$

There is an added level of complexity in the computations of criteria for cadmium and lead because the CFs for these metals also vary with hardness.

If a total maximum daily load (TMDL) is needed to regulate discharges into an impaired water body, the dissolved criterion must be converted or translated back to a total value so that the TMDL calculations can be performed. The translator can simply be the CF (*i.e.*, divide the dissolved criterion by the CF to get back to the total criterion), or site-specific data on total and dissolved metal concentrations in the receiving water are collected and a dissolved-to-total ratio is used as the translator.

Formulae for all the metals listed above also include a WER, a number that acts as a multiplication factor. A WER is intended to account for the difference in toxicity of a metal in a site water relative to the toxicity of the same metal in reconstituted laboratory water. The reason is that natural waters commonly contain constituents which "synthetic" or "reconstituted" laboratory waters lack, such as dissolved organic compounds, that may act to bind metals and reduce their bioavailability. Where such constituents act to modify the toxicity of a metal in a site water compared to the toxicity of the same metal in laboratory water, a "water effect" is

observed. If no site-specific WER is determined, then the WER is presumed to be 1 and would not modify a formula result.

The EPA has provided specifications and guidance regarding procedures and requirements for determining "site-specific" WER values that include extensive comparative toxicity testing with several test organisms and statistical analysis of results. The example provided below only illustrates the basic principle in defining a WER value.

Example WER calculation:

Suppose the LC_{50} of copper in site water is 30 µg/L
Suppose the LC_{50} of copper in laboratory water is 20 µg/L
Assume a site hardness of 100 mg/L
The freshwater CF for copper = 0.96
Acute criteria (CMC) for total recoverable copper without the WER = 18 µg/L

$$\text{WER} = \frac{\text{Site } LC_{50}}{\text{Lab } LC_{50}} = \frac{30\ \mu g/L}{20\ \mu g/L} = 1.5$$

$$\begin{aligned} \text{Copper Site-Specific CMC} &= \text{WER} \times \text{CF} \times e^{(m[\ln(40)]+b)} \\ &= 1.5 \times 0.96 \times 18 \\ &= 24\ \mu g/L \end{aligned}$$

In the NTR, the EPA described and required minimum and maximum hardness values (25 mg/L and 400 mg/L as $CaCO_3$, respectively) to be used when calculating hardness-dependent freshwater metals criteria. Most of the data that the EPA used to develop the hardness formulae were in the hardness range of 25 to 400 mg/L. Therefore, the EPA stated that the formulae were most accurate in that range.

Formula-based metals criteria are discussed as a group here because the key issues of how dissolved metal criteria are derived and the implications of using the present formulae are similar for each of them. Issues include the influence of hardness, site-specific water quality characteristics, and the speciation of metal considered. The present formula-based metal method in the Guidelines does not consider the environmental fate, transport, and transformations of metals in natural environments (specifically for As, Cd, Cr (III), Cr (VI), Cu, Pb, Ni, Ag, and Zn), nor the influence of other water quality constituents on toxicity, and therefore affords incomplete protection for listed species.

A direct pathway for dissolved metals into aquatic organisms is through the gills. Dissolved forms of metals can adsorb to particulate matter in the water column and enter organisms through various routes. Metals adsorbed to particulates can also be transferred across the gill membranes (Lin and Randall 1990, Playle and Wood 1989, Sorensen 1991, Wright *et al.* 1986). Planktonic and benthic invertebrates can ingest particulate metals from the water column and sediments and then be eaten by other organisms. Thus, dietary exposure may be a significant source of metals to aquatic and aquatic dependent organisms.

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Although metals bound to sediments are generally less bioavailable to organisms, they are still present, and changes in the environment (*e.g.*, dredging, storm events, temperature, lower water levels, biotic activity) can significantly alter the bioavailability of these metals. The feeding habits of fish can determine the amount of uptake of certain metals. Piscivorous fish are exposed to different levels of metals than omnivorous and herbivorous fish. For example, cadmium is more commonly found in omnivorous fish tissues than in carnivorous fish tissues from the same location (Enk and Mathis 1977).

Listed species are exposed to metals not only through the dissolved fraction in ambient waters, but they are also exposed to toxic effects of particulate metals through the mechanism of respiratory uptake in fish and by ingestion of contaminated particulate material. In addition, Finlayson *et al.* (2000) determined that metal-laden sediments in Keswick Reservoir, California were toxic to rainbow trout when re-suspended in moderately alkaline (pH 7.8) and soft (38 mg/L) water and elutriated. As fish respire, a nearly continuous flow of water passes across their gills (Moyle and Cech 1988) and particulate metals suspended in the water column may become entrapped. At the lowered pHs occurring near gill surfaces associated with gas exchange (Lin and Randall 1990, Playle and Wood 1989, Wright *et al.* 1986), entrapped particulate metals may release soluble metal ions, the form that is most bioavailable and efficiently taken up by aquatic organisms (EPA 1993a, 1997a). Although most research has been done on particulate exposures to gills of fish including salmonids, it is possible that other gill-breathing organisms (*e.g.*, aquatic macroinvertebrates) can be affected in the same way.

Current guidance for waste load allocation calculations (EPA 1996a) consists of simple dilution formulations using effluent metal loads, receiving water flows, and dissolved-to-total metals ratios in the receiving waters. Formula-based metal criteria are not protective of threatened or endangered aquatic species with respect to loading because the criteria development methods do not adequately consider the environmental fate, transport, and transformation of metals in natural environments. This concern is based in part on analyses conducted during the California Toxics Rule (CTR) consultation (USFWS and NMFS 2000), in which NMFS determined that substantial increases in total metals would be permitted in hypothetical discharges under the proposed criteria. The CTR analysis determined that as the fraction of particulate metal in the receiving water increases, the allowable discharge of particulate metals also increases rather than decreases. Such increases would be expected to occur through allowable TMDLs under the proposed ODEQ criteria because a TMDL is is based on the instream total metal concentration (EPA 1996a). Under Oregon's proposed water quality standards, total metal discharges may increase as long as the dissolved criteria are not exceeded.

Further, discharges from agricultural or urban non-point sources are largely uncontrolled through the discharge-permitting process. Metals criteria based only on dissolved concentrations provide little incentive for reducing non-point sources, which involve largely the particulate form. Thus, metals criteria based on dissolved concentrations in the absence of sediment criteria linked to total metals will not effectively prevent sediment contamination by metals and may lead to increased allowable loads of metals to sediments.

Formulae used to compute toxicity criteria for Cd, Cu, Cr(III), Pb, Ni, Ag, and Zn are presently functions of water hardness. By convention, hardness measurements are expressed in terms of

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the equivalent concentration of $CaCO_3$ (expressed in mg/L) required to contribute that amount of calcium + magnesium hardness. Under the proposed criteria, hardness is determined for a site (expressed as mg/L of $CaCO_3$), and input to the criteria formulae for each metal. In natural waters considerable variation can occur in the calcium:magnesium ratio, contributing to site-specific water hardness. Studies show significant differences in toxicity for some metals depending on this ratio. In general, calcium provides greater reductions in toxicity. Site-specific hardness values with contributions from other multivalent cations (*e.g.*, iron, aluminum, manganese) that are evaluated using criteria based only on calcium + magnesium hardness result in site criteria that may not be protective. For example, in the case of cadmium, the presence of calcium is protective against toxicity whereas, magnesium, sodium, sulfate ions and the carbonate system appear to give little to no protection (Carroll *et al.* 1979). Welsh *et al.* (2000b) determined that calcium also afforded significantly greater protection against copper toxicity than magnesium.

The calcium:magnesium ratio in natural waters of Oregon varies substantially (Table A1).

Table A1. Total hardness for selected watersheds in Oregon in mg/L CaCO3. Data from USGS (1977).

Watershed	Mean	Standard Deviation	Range
Snake River ID-OR Border	141.3	33.7	97-190
Rogue River (RM 25)	37.5	5.1	30-45
John Day River	88.4	32.8	46-140
Deschutes River	41.5	2.7	37-45
Columbia River (RM 140)	69	11.8	45-94
Tualatin River	38.1	14.2	25-80
Willamette River (RM 10)	24	3.4	19-32
Nehalem River	18.9	6.5	12-32
Umpqua River	28.3	4.3	19-34

The majority of hardness data used to develop the EPA hardness-dependent criteria formulae were in the range of 25 mg/L to 400 mg/L (40 CFR Part 131). Consequently, EPA's regulations (40 CFR 131.36) specify that the minimum hardness that can be used in criteria equations is 25 mg/L. This requirement reflects that toxicity effects at hardness concentrations less than 25 mg/L are not known with a reasonable degree of certainty. Existing criteria formulae can result in toxic concentrations in water with hardness below the 25 mg/L lower threshold. There are some streams in Oregon where hardness concentrations average less than 25 mg/L, for which concentrations of contaminants with hardness ameliorated toxicity should be calculated on actual site conditions.

Comparable toxicity test data for hardness values greater than 400 mg/L appear to exist only for zinc, which precludes direct evaluation of the effects of extrapolating the criteria equations upwards. However, the ameliorating effect of increasing concentration of calcium ions means that the use of a default limiting value of 400 mg/L is protective for listed species in harder water in the case of metals for which toxicities are influenced by hardness.
The value of the site-specific hardness value will depend on where samples are collected. The calculated criteria may be less protective when samples are collected downstream of effluent

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sources that may increase hardness locally (it is highly unlikely that discharges decrease downstream hardness). In otherwords, the use of hardness values measured downstream of the effluent source could lead to greater-than-intended site criteria. In some cases, certain effluents may alter ambient hardness, but not other important water quality constituents that influence metal toxicity (*e.g.*, pH, alkalinity, dissolved organic carbon, calcium, sodium, chloride, *etc.*). Alterations in receiving water chemistry by a discharge (*e.g.*, abrupt elevation of hardness, changes in pH, exhaustion of alkalinity, abrupt increases in organic matter *etc.*) could result, depending on the hardness value applied in the criteria formulae, in increased allowable discharges of toxic metals.

Water hardness and the hardness acclimation status of a fish will affect toxicity and toxic response. However the use of hardness alone as a universal surrogate for all water quality parameters that can modify metal toxicity will not always correlate well with the predicted toxic effect on listed species. The importance of water quality parameters other than hardness on metals toxicity has been understood for some time (Howarth and Sprague 1978). Numerous studies have been performed on the toxicity of metals in test waters of various compositions, and the results do not confer a singular role to hardness in ameliorating metals toxicity. Test water characteristics in most studies, including pH, calcium, alkalinity, dissolved organic carbon, chloride, sodium, suspended solids, and other chemical properties, are varied in a controlled manner while observing the responses of test organisms. It is likely that understanding metal toxicity in waters of various chemical makeups is not possible without the use of a geochemical model, and that a univariate regression formula will not suffice. It is also possible that simple toxicity tests (using mortality, growth, or reproductive endpoints) are not capable of discriminating the role of hardness relative to other water chemistry characteristics in modulating metals toxicity (Erickson *et al.* 1996).

Summary**:** Based on this analysis, using formula-based criteria for aquatic life criteria derived following the Guidelines are likely to be underprotective of listed species considered in this opinion. Formula-based metal criteria are discussed as a group here because the key issues of how dissolved metal criteria are derived and the implications of using the present formulae are similar for each of them. Issues include the influence of hardness, site-specific water quality characteristics, and the speciation of metal considered. The present formula-based metal method does not consider the environmental fate, transport, and transformations of metals in natural environments (specifically for arsenic, cadmium, chromium (III), chromium (VI), copper, lead, nickel, silver, and zinc), nor the influence of other water quality constituents on toxicity, and therefore affords incomplete protection for listed species and is likely to result in sublethal effects, such as central nervous system disruption, altered liver and kidney function, impaired reproduction, decreased olfactory response, delayed smoltification, impaired ability to avoid predation and capture prey, growth inhibition, growth stimulation, changes in prey species community composition (which will increase foraging budgets), and death of listed species considered in this opinion.

Additive and Synergistic Toxicity. When two or more toxic pollutants are present, their combined effect may be either additive, synergistic (where the net effect exceeds the sum of effects), or antagonistic. The proposed water quality standards do not take these effects into account. Relatively few toxicity studies have addressed this issue, and some studies have

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indicated conflicting results due to complex interactions that vary with the combination(s) and concentrations involved (Sorenson 1991). However, a number of studies have determined conclusively that adverse effects due to additive or synergistic toxicity mechanisms occur when several criteria are near or equal to acute criteria concentrations (*e.g.*, Alabaster and Lloyd 1982, Spehar and Fiandt 1986, EIFAC 1987, Enserink *et al.* 1991, Sorenson 1991). Spehar and Fiandt (1986) determined that rainbow trout embryo survival and growth were not reduced when exposed to combinations of arsenic, cadmium, chromium, copper, and lead at chronic concentrations, but production and growth of *Daphnid sp.* were reduced for the same test mixtures. Combinations of organic pollutants also have been shown to result in different toxic responses, as have combinations of organic and metals contaminants.

Alabaster and Lloyd (1982) observed from their data that the combined acutely lethal toxicity to fish and other aquatic organisms is approximately the simple addition of the proportional contribution from each toxicant. The median value of the effect on fish is 0.95 of that predicted; the collective value for sewage effluents, river waters and a few industrial wastes is 0.85. The range for effluents, river wastes, and industrial wastes is 0.4 to 2.8, which indicates that the combined effects of individual acutely toxic pollutants are from 0.4 to 2.8 times the effects predicted by adding the individual effects. The median combined effect is approximately additive (EPA 1991).

Summary: Based on this analysis, the aquatic life criteria derivied following the Guidelines do not take into account additive or synergistic effects, thus increasing the likelihood of acute toxic effects and sublethals effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Assumption that Effects in Laboratory Tests are Reasonable Predictors of Effects in Field Situations. The preceding discussion concerned whether compilations of laboratorytest values were appropriate to treat as surrogates of the diversity of natural systems. A fundamental question in evaluating the Guidelines and the national criteria is whether tests of chemicals in laboratory aquaria with "domesticated" cultures of test animals are likely to produce similar effects as would exposure to the same substance on the same or closely related species in the wild. If the responses between animals in laboratory aquaria or the wild are different, is there a bias in the sensitivity of responses from either the lab or wild settings? That is, are the effects of chemical contamination likely more or less severe in the laboratory or wild settings? This question is important because water quality criteria are designed to apply to and protect ambient waters (that is, streams, rivers, and lakes), yet the data used to develop them are invariably compiled from laboratory testing under tightly controlled and thus quite artificial environments. There are myriad factors that may influence the effects of a chemical stressor on aquatic organisms, and this complexity makes the question of bias in sensitivity difficult or even impossible to answer with any certainty. The conclusion by Chapman (1983) regarding comparability of laboratory exposure-response effects and field exposure-response effects contributed to one the most fundamental assumptions in the Guidelines, that is, "the Guidelines have been developed on the theory that effects which occur on species in appropriate laboratory tests will generally occur on the same species in comparable field situations." A number of reasons why the effects of a criteria chemical could be more or less severe on listed species in laboratory or in wild settings are summarized in Table A2.

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Table A2. Factors influencing the effects of a chemical stressor in a laboratory setting or in the wild.

FACTOR	ARE EFFECTS LIKELY MORE SEVERE IN TYPICAL LAB SETTINGS OR IN THE WILD?
Environmental Conditions	
Nutritional state - acute test exposures	**In the wild:** In acute toxicity tests with fish fry, fish are selected for uniform size, and unusually underweight fish that might be weakened from being in poor nutritional state are culled from tests. For instance, if <90% of control fish survive the 4 days of starvation in an acute toxicity test, the test may be rejected from inclusion in the criteria dataset. In the wild, not all fish will be in optimal nutritional state. While perhaps counterintuitive, starvation can protect fish against waterborne copper exposure (Kunwar *et al.* 2009). Fish are routinely starved during acute laboratory tests of the type used in criteria development.
Nutritional state – chronic test exposures	**In the wild:** Fish in the wild must compete for prey, and if chemicals impair fish's ability to detect and capture prey because of subtle neurological impairment, this could cause feeding shifts and reduce their competitive fitness (Riddell *et al.* 2005). Fish in chronic lab tests with waterborne chemical exposures are often fed to satiation, and food pellets don't actively evade capture like live prey. Perhaps these factors dampen responses in lab settings.
Temperature	**In the wild:** In lab test protocols, nearly optimal test temperatures are recommended (*e.g.*, 12°C for rainbow trout, the most commonly tested salmonid). Fish may be most resistant to chemical insults when at optimal temperatures. At temperatures well above optimal ranges, increased toxicity from chemicals often results from increased metabolic rates (Sprague 1985); Under colder temperatures, fish have been shown to be more susceptible to at least Cu, Zn, Se and cyanide, although the mechanisms of toxicity are unclear (Dixon and Hilton 1985, Erickson *et al.* 1987, Hansen *et al.* 2002a, Hodson and Sprague 1975, Kovacs and Leduc 1982, Lemly 1993).
Flow	**In the wild:** Fish expend energy to hold their position in streams and to compete for and defend preferred positions that provide optimal feeding opportunity from the drift for the energy expended. Subordinate fish in the wild are forced to less profitable positions and become disadvantaged. Subordinate fish in lab settings still get adequate nutrition from feeding. Chemical exposure can reduce swimming stamina or speeds, as can exposure to soft water. (Adams 1975, De Boeck *et al.* 2006, Kovacs and Leduc 1982, McGeer *et al.* 2000).
Disease and parasites	**In the wild:** Disease and parasite burden are common in wild fish, but toxicity tests that used diseased fish likely were considered compromised and results likely were not used in criteria development. Chemical exposure may weaken immune responses and increase morbidity or deaths (Arkoosh *et al.* 1998, Stevens 1977).
Predation	**In the wild:** Fish use chemical cues to detect and evade predators; these can be compromised by some chemical exposures (Berejikian *et al.* 1999, Labenia *et al.* 2007, Phillips 2003, Scott *et al.* 2003)
Exposure	
Variable exposures	**In the lab:** Most toxicity tests used to develop criteria are conducted at nearly constant exposures. Criteria are expressed not just as a concentration but also with an allowed frequency and duration of allowed exceedences. In field settings, most point or non-point pollution scenarios that rarely if ever exceed the criteria concentration (*i.e.*, no more than for one 4-day interval per 3 yrs), will have an average concentration that is less than the criterion concentration. For some chemicals, such as copper, fish might detect and avoid harmful

FACTOR	ARE EFFECTS LIKELY MORE SEVERE IN TYPICAL LAB SETTINGS OR IN THE WILD?
	concentrations if clean-water refugia were readily available.
Metal form and bioavailability	**Uncertain:** Metals other than Hg and some organics are commonly more bioavailable in the lab because dissolved organic carbon, which reduces the bioavailability and toxicity of several metals, is low in laboratory tests that are eligible for use in criteria. The Guidelines call for <5 mg/L TOC (total organic carbon) in studies to be used in criteria (Stephan *et al.* 1985), but probably more often TOC is <2 mg/L in laboratory studies.
Chemical equilibrium	**Uncertain:** While results conflict, metals are usually considered less toxic when in equilibrium with other constituents in water, such as organic carbon, calcium, carbonates and other minerals. In the wild, daily pH cycles prevent full equilibria from being reached (Meyer *et al.* 2007). Likewise, in conventional laboratory flow-through tests, designs chemicals may not have long enough contact time to reach equilibrium. Static-renewal tests are probably nearly in chemical equilibria, although organic carbon accretion can lessen toxicity which may not reflect natural settings (Santore *et al.* 2001, Welsh *et al.* 2008).
Prior exposure	**Uncertain:** If fish are exposed to sublethal concentration of a chemical they could either become weakened or become more tolerant of future exposures. With some metals, normally sensitive life stages of fish may become acclimated and less sensitive during the course of a chronic test if the exposure was started during the resistant egg stage (Brinkman and Hansen 2007, Chapman 1983, 1985, Sprague 1985).
Life stages exposed	**In the wild:** Most lab studies are short term and realistically testing all life stages of anadromous fish is probably infeasible. Reproduction is often the most sensitive life stage with fish but most "chronic" studies are much shorter and just test early life stage survival and growth (Suter *et al.* 1987). At different life stages and sizes, salmonids can have very different susceptibility to some chemicals; even when limited to a narrow window of young-of-year fry, sensitivity can vary substantially. Unless the most sensitive life stages are tested, lab tests could provide misleadingly high toxicity values for listed species.
Chemical mixtures	**In the wild:** In field conditions, organisms never experience exposure to a single pollutant; rather, ambient waters typically have low concentrations of numerous chemicals. The toxic effects of chemicals in mixture can be less than those of the same chemicals singly, greater than, or have no appreciable difference. The best known case of one toxicant reducing the effects of another is probably Se and Hg (*e.g.*, Belzile *et al.* 2006). However, strongly antagonistic responses are probably uncommon, and much more common are situations where chemical mixtures have greater toxicity than each singly or little obvious interaction (*e.g.*, Borgert 2004, Laetz *et al.* 2009, Norwood *et al.* 2003, Playle 2004, Scholz *et al.* 2006). In general, it seems prudent to assume that if more than one toxicant were elevated, it is likely that lower concentrations of chemicals would be required to produce a given magnitude of effect than would be predicted from their actions separately.
Dietary exposures	**In the wild:** Toxicity test data used in criteria development have been mostly based solely on waterborne exposures, yet in the wild, organisms would be exposed to contaminants both through dietary and water exposures. With at least some organics and metals (*i.e.*, As, Se) dietary exposures are more important than water exposures. For some other metals (*i.e.*, Cd, Cu, Ni, Pb, Zn), at environmentally relevant concentrations that would be expected when waterborne concentrations are close to criteria, dietary exposures have not been shown to directly result in appreciable adverse effects on fish (Hansen *et al.* 2004, Schlekat *et al.* 2005). However, while dietary exposures of some metals have not yet been implicated in adverse effects on fish at or below criteria

FACTOR	ARE EFFECTS LIKELY MORE SEVERE IN TYPICAL LAB SETTINGS OR IN THE WILD?
	concentrations, they may in fact be both the primary route of exposure and an important source of toxicity for benthic invertebrates rather than fish (Buchwalter *et al.* 2008, Irving *et al.* 2003). For instance Besser *et al.* (2005a) found that the effects threshold for Pb to the benthic crustacean *Hyalella sp.* was well above the chronic criterion in water exposures, but when Pb was added to the diet, effects threshold dropped to near criteria concentrations. Ball *et al.* (2006) found that feeding Cd-contaminated green algae to the benthic crustacean *Hyalella* sp. caused a 50% growth reduction at about the NTR chronic criterion.
Population Dynamics	
Density effects	**In the lab:** Salmonid fishes are highly fecund (~500 to 17,000 eggs per spawning female). When abundant, overcrowding, and competition for food and shelter may result in relatively high death rates for some life stages, particularly young-of-year during their first winter. After many fish die in a density-dependent bottleneck, the survivors have greater resources and improved growth and survival. Conceptually, if an acute contamination episode killed off a significant portion of young-of-year fish prior to their entering a resource bottleneck, then assuming no residual contaminant effects, the losses to later life stages and to adult spawners could be buffered.
Meta-population dynamics	**In the lab:** If habitats are interconnected, as is the case in intact stream networks, and if pervasive contamination from discharges to a stream were to impair only some endpoints or life-stages, such as reproductive failure or YOY mortalities, immigration from source populations may make detection of population reductions in the affected sink population difficult (Ball *et al.* 2006, Palace *et al.* 2007). If an episodic contamination pulse were to kill a large proportion of fish in a stream, the proximity of refugia and donors from source populations affect recovery rates (Detenbeck *et al.* 1992).

Summary: Based on this analysis, the assumption that effects in laboratory tests as reasonable predictors of effects to species in the wild is dependent upon the specific factor being considered. Overall NMFS finds that laboratory tests are likely to underpredict effects, as adverse effects are generally likely to be more severe in the wild than under laboratory conditions. Thus aquatic life criteria derivied following the Guidelines are likely to result in sublethals effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Risks of Using Flow-Through, Renewal, or Static Exposure Test Designs. One area of controversy in evaluating toxicity test data or risk assessments, or criteria derived from them, has to do with potential bias in how test organisms are exposed to test solutions. Exposures of test organisms to test solutions are usually conducted using variations on three techniques. In "static" exposures, test solutions and organisms are placed in chambers and kept there for the duration of the test. The "renewal" technique is like the static technique except that test organisms are periodically exposed to fresh test solution of the same composition, usually once every 24 hours or 48 hours, by replacing nearly all the test solution. In the "flow-through" technique, test

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solution flows through the test chamber on a once-through basis throughout the test, usually with at least five volume replacements/day (ASTM 1997).

The term "flow-through test" is commonly mistaken for a test with flowing water, *i.e.*, to mimic a lotic environment in an artificial stream channel or flume. This is not the case; rather the term refers to the once-through, continuous delivery of test solutions (or frequent delivery in designs using a metering system that cycles every few minutes). Flows on the order of about five volume replacements per 24 hours are insufficient to cause discernable flow velocities. In contrast, even very slow moving streams have velocities of around 0.04 ft/sec (an inch per second) or more. At that rate, a parcel of water would pass the length of a standard test aquarium (~2 ft) in about 48 seconds, resulting in about 9,000 volume replacements per day. A more typical stream velocity of about 0.5 ft/sec would produce over 100,000 volume replacements per day.

Historically, flow-through toxicity tests were thought to provide a better estimate of toxicity than static or renewal toxicity tests because they provide a greater control of toxicant concentrations, minimize changes in water quality, and reduce accumulation of waste products in test exposure waters (Rand *et al.* 1995). Flow-through exposures have been preferred in the development of standard testing protocols and water quality criteria. The Guidelines first advise that for some highly volatile, hydrolysable, or degradable materials, it is probably appropriate to use only results of flow-through tests. However, this advice is followed by specific instructions that if toxicity test results for a species were available from both flow-through and renewal or static methods, then results from renewal or static tests are to be discounted (Stephan *et al.* 1985). Thus, depending upon data availability, toxicity results in the criteria databases may be a mixture of data from flow-through, renewal or static tests, raising the question of whether this could result in bias. In the Guidelines, the rationale for the general preference for flow-through exposures was not detailed, but it was probably based upon assumptions that static exposures will result in LC_{50}s that are biased high (apparently less toxic) than comparable flow-through tests, or that flow-through tests have more stable exposure chemistries and will result in more precise LC_{50} estimates.

With metals, renewal tests produce higher EC_{50}s (*i.e.*, metals were less toxic), probably because of accretion of dissolved organic carbon (DOC) (Erickson *et al.* 1996, Erickson *et al.* 1998, Welsh *et al.* 2008). However, in contrast to earlier EPA and ASTM recommendations favoring flow-through testing, Santore *et al.* (2001) suggested that flow-through tests were biased low because copper complexation with organic carbon, which reduces acute toxicity, is not instantaneous, and typical flow-through exposure systems allowed insufficient hydraulic residence time for complete copper-organic carbon complexation to occur. Davies and Brinkman (1994) similarly found that cadmium and carbonate complexation was incomplete in typical flow-through designs, although in their study incomplete complete complexation had the opposite effect of the copper studies, with cadmium in the aged, equilibrium waters being more toxic. A further complication is that it is not at all clear that natural flowing waters should be assumed to be in chemical equilibria because of tributary inputs, hyporheic exchanges and daily pH, inorganic carbon, and temperature cycles. Predicting or even evaluating risk of toxicity through these cycles is complex and seldom attempted (Meyer *et al.* 2007), in part because pulse exposures cause latent mortality (*i.e.*, fish die after exposure to the contaminant is removed), a phenomenon that is often overlooked or not even recognized in standard acute toxicity testing.

When comparing data across different tests, it appears that other factors such as testing the most sensitive sized organisms or organism loading may be much more important than if the test was conducted by flow through or renewal techniques. For instance, Pickering's and Gast's (1972) study with fathead minnows and cadmium produced flow-through LC_{50}s that were lower than comparable static LC_{50}s (~ 4,500 to 11,000 µg/L for flow-through tests vs. ~30,000 µg/L for static tests). The fish used in the static tests were described as "immature," weighing about 2 g (2000 mg). The size of the fish used in their flow-through acute tests were not given, but is assumed to have been similar. In contrast, 8 to 9 day old fathead minnow fry usually weigh about 1 mg or less (USEPA 2002b). Using newly hatched fry weighing about 1/1000th of the fish used by Pickering and Gast (1972) in the 1960s, and modern protocols, cadmium LC_{50}s for fathead minnows at similar hardnesses tend to be around 50 µg/L, with no obvious bias for test exposure. Similar results have been reported with brook trout. One each flow-through and static acute tests with brook trout were located, both conducted in waters of similar hardness (41 to 47 mg/L). The LC_{50} of the static test which used fry was < 1.5 µg/L whereas the LC_{50} of the flow-through test using yearlings was > 5,000 µg/L (Carroll *et al.* 1979, Holcombe *et al.* 1983).

Many studies on which the proposed criteria are based involve laboratory-based LC_{50} bioassays using static exposure systems and nominal contaminant concentrations. Such studies often yield LC_{50} values substantially higher than values obtained with flow-through tests or tests in which actual concentrations of contaminants in the system during the experiment are measured, with differences in some cases of an order of magnitude lower. For example, LC_{50} values for static tests have been determined to be approximately 20 times higher than those from flow-through tests for DDT (Earnest and Benville 1971). Mercury toxicity testing of trout embryos has indicated that concentration-based endpoints (*e.g.*, EC_{50}) could be as much as one to two orders of magnitude lower in flow-through than static tests (Birge *et al.* 1979, 1981). Static assays were also found to underestimate the toxicity of endosulfan in comparisons with flow-through systems (Naqvi and Vaishnavi 1993). Several additional studies with a variety of compounds report increased toxicity in flow-through compared to static systems (*e.g.*, Erickson *et al.* 1998, Hedtke and Puglisi 1982, Vernberg *et al.* 1977, Randall *et al.* 1983, Burke and Ferguson 1969). Static conditions may underestimate the true exposure concentration because the fish will deplete the concentration in solution over time, causing a lack of steady-state exposure. There may also be important differences in energy expenditure and metabolism of test fish between static and flow-through tests, depending on the experimental setup. In the case of listed salmonids in Oregon, this may be an important source of variation because they typically live in flowing waters. Acute LC_{50}s for salmonids that are based on static tests could therefore underestimate toxicity, and water quality standards based on such tests may consequently not be sufficiently protective against conditions reasonably expected to occur in Oregon waters.

Summary: Based on this analysis, using flow-through, renewal, or static exposure test designs may result in greater than predicted effects.

Effects of Acclimation on Susceptibility to Chemicals. Exposure to sublethal concentrations of organic chemicals and other metals may result in pronounced increases in resistance to later exposures of the organisms. With metals the resistance may be on the order of two to four times greater for acute challenges, but for some organic contaminants may be much higher (Chapman 1985). However, the increased resistance can be temporary and can be lost in

as little as seven days after return to unpolluted waters (Bradley *et al.* 1985, Hollis *et al.* 1999, Sprague 1985, Stubblefield *et al.* 1999). For this reason, the Guidelines specify that test results from organisms that were pre-exposed to toxicants should not be used in criteria derivation (Stephan *et al.* 1985).

Effects from acclimation, however, are not precluded by the Guidelines and influence chronic values and thus chronic criteria. Several tests have shown that at least with fish and metals, if the toxicity tests were initiated during more resistant early life stages (ELS, *e.g.*, embryo stage), acclimation may occur, and later in the test when the more sensitive life stages become exposed (*e.g.*, fry stage), the usually sensitive life stages may be more resistant than the same life stages of fish which had no pre-exposure (Brinkman and Hansen 2004; 2007, Chapman 1978a; 1994, Spehar *et al.* 1978).

Chapman (1994) exposed different life stages of steelhead (*Oncorhynchus mykiss*) for the same duration (three months) to the same concentration of copper (13.4 µg/L at a hardness of 24 mg/L as $CaCO_3$). The survival of steelhead that were initially exposed as embryos was no different than that of the unexposed control fish, even though the embryos developed into the usually-sensitive swim-up fry stage during the exposure. In contrast, steelhead that were initially exposed as swim-up fry, without the opportunity for acclimation during the embryo state, suffered complete mortality. Brinkman and Hansen (2007) compared the responses of brown trout (*Salmo trutta*) to long-term cadmium exposures that were initiated either at the embryo stage (*i.e.*, early-life stage tests) or the swim-up fry stage (*i.e.*, chronic growth and survival tests). In three comparative tests, fish that were initially exposed at the swim-up fry stage were consistently two to three times less resistant than were the fish initially exposed at the embryo stage.

These studies support the counterintuitive conclusion that because of acclimation, longer-term tests or tests that expose fish over their full life cycle are not necessarily more sensitive than shorter-term tests that are initiated at the sensitive fry stage. Conceptually, whether this phenomenon is important depends on the assumed exposure scenario. If it were assumed that spawning habitats would be exposed, then the less-sensitive ELS tests would be relevant. However, for migratory fishes such as listed salmon and steelhead, life histories often involve spawning migrations to headwater reaches of streams, followed by downstream movements of fry shortly after emerging from the substrates, and followed by further seasonal movements to larger, downstream waters to overwinter (Baxter 2002, Quinn 2005, Willson 1997). These life history patterns often correspond to common human development and metals pollution patterns where headwater reaches likely have the lowest metals concentrations, and downstream increases occur due to point source discharges or urbanization.

From the discussion of the types of chronic data with fish that are acceptable for use in criteria development, it is clear that the intent was to capture information on the most sensitive life stage of a fish species. Unfortunately, the wording of the Guidelines could be interpreted to preclude the use of the more-sensitive chronic growth and survival tests that were initiated with salmonid fry stage, and specify the use of the less-sensitive ELS tests (Stephan *et al.* 1985, p. 44).

Summary: Based on this analysis, the risks of acclimation on susceptibility to chemicals are likely to result in sublethal effects, such as interference in physiochemical processes,

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interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Toxic Responses of Different Species and Life Stages. The chemical concentrations causing toxic effects differ between taxa, with some species being more sensitive than others. The EPA's national water quality criteria, on which the proposed criteria are based, were developed from toxicity data compiled for a wide range of species and life stages and were determined on the basis of protecting roughly 95% of the species considered. However, because the criteria were not developed specifically to protect the most sensitive species or life stage present, it is possible that the proposed criteria may not be protective when that species and life stage is a listed species, *i.e.*, a species at risk of extinction. This is recognized in the Guidelines which indicate that it is possible to revise the criteria if it is determined that there is a more sensitive species and life stage present (EPA 1994a).

The EPA identified SMAVs in their criteria documents for most of the pollutants subject to this consultation that differ between species of salmon and trout. SMAV's for marine mammals, sea turtles, green sturgeon, and eulachon have not been developed. However, the SMAVs were in most cases based on limited toxicity testing data collected under varying conditions, and therefore may not be indicative of actual species differences. Moreover, SMAVs are not completely protective of listed species because they represent an average condition, where lower concentrations may be toxic to those species under certain test conditions. There is evidence that under similar testing conditions, some trout species have similar toxic responses (*e.g.*, rainbow and brown trout, Cohen *et al.* 1993). There is also evidence of differences in toxicity response between species when exposed to specific metals or organic compounds under similar conditions (*e.g.*, Chinook and coho salmon, Hamilton and Buhl 1990; Chinook salmon, Chapman 1978b; rainbow and brook trout, Holcombe and Andrew 1978; brown trout, Chinook and coho salmon, Macek and Allister 1970, Katz 1961; rainbow trout, and Chinook and coho salmon, Macek *et al.* 1969, Katz 1961), so species differences cannot be completely discounted. Overall, however, experimental evidence (including data presented in the various EPA water quality criteria documents) suggests that there is greater variation in toxic response between life stages than between species within the family Salmonidae.

Since a species can only be considered protected from acute toxicity if all life stages are protected, EPA's Guidelines recommend that if the available data indicate that some life stages are more resistant than other life stages by at least a factor of two, the data for the more resistant life stages should not be used to calculate species mean acute values (Stephan *et al.* 1985). Smaller, juvenile life stages of fish are commonly expected to be more vulnerable to metals toxicity than larger, older life stages of the same species. For instance, a standard guide for testing the acute toxicity of fish (ASTM 1997) recommends that tests should be conducted with juvenile fish (that is, post-larval or older and actively feeding), usually in the size range from 0.1 to 5.0 g in weight.

A review of several data sets in which salmonids of different sizes were similarly tested shows that even among juvenile fish in the 0.1 to 5.0-g size range, differences in sensitivity can approach a factor of 10. This emphasizes the importance of EPA's Guidance not to use the more resistant life stages. However, the data sets analyzed by NMFS indicated that in practice, there

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were sometimes greater influences of life stage on the sensitivity of salmonids to some substances than was apparent to the authors of the individual criteria documents using the datasets available to them at the time. Some of the SMAVs and GMAVs which were used to rank species sensitivity and set criteria were considerably higher than EC_{50}s for salmonids that were tested at the most sensitive life stages (Figure A4).

For three Pacific salmonid species for which comparable test data were available for different life stages (coho salmon (*O. kisutch*), rainbow trout (*O. mykiss*) and cutthroat trout (*O. clarki*), the data suggest that swim-up fish weighing around 0.5 g to about 1 g may be the most sensitive life stage. None of the data sets or published studies NMFS examined in detail had sufficient resolution to truly define what weight fish was most sensitive to metals, but along with other data they suggest that larger fish are less sensitive than fish at 0.4 to 0.5 g. For instance, with zinc, rainbow trout in the size range of about 0.1 to about 1.5 g were consistently more sensitive to zinc in two studies with multiple tests in that size range. The paucity of data with salmonids in the size range of about 0.5 to 2 g prevents definitive identification of a most sensitive size across species or even tests. All data located for early swim-up stage *Oncorhynchus* in the 0.1 to 0.5 g range were consistent with increasing sensitivity with size. With Hansen *et al.* (1999b) rainbow trout studies, this relationship continued with fish up to about 1.5 g. However, with cutthroat trout, the few data available suggests that fish larger than about 0.5 g are less sensitive with increasing size.

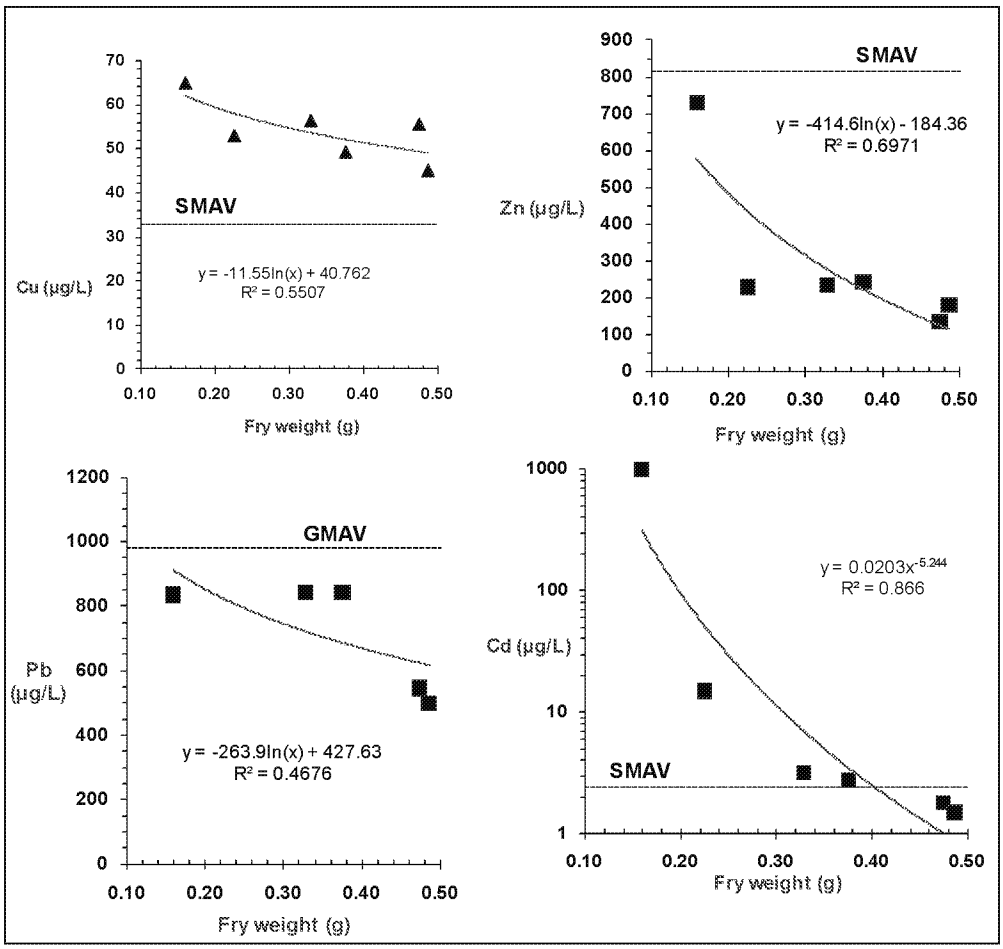

Figure A4. Size-developmental stage patterns SMAVs and GMAVs with coho salmon from 2 to 7 weeks posthatch, with data from Chapman (1975), and EPA (1984a, 1984b, 1985, 1987), adjusted to test water hardness. All tests used Willamette River water, TOC 3.4 mg/L, hardness 22 mg/L.

Some studies with older and larger rainbow trout have found that the fish became more resistant to zinc and copper (Chakoumakos *et al.* 1979, Chapman 1978b, Chapman and Stevens 1978, Howarth and Sprague 1978). Studies with copper all showed this trend, but the strength of size-sensitivity relations varied across studies. Chakoumakos *et al.* (1979) found that fish between about 1 and 25g in weight varied in their sensitivity to copper by about 8 times, but steelhead (*O. mykiss*) that were tested with copper at sizes of 0.2, 7, 70, and 2700 g showed little pattern of sensitivity with size (Chapman and Stevens 1978, Chapman 1978b). However, the large differences in sizes may have missed changes at intermediate sizes in the ranges compared (Figure A4). Similarly, with copper and rainbow trout, Anderson and Spear (1980) found that rainbow trout at sizes of 3.9, 29 to 176 g had similar sensitivities.

The NMFS reviewed several data sets indicated increasing susceptibility of salmonids to at least metals with increasing size and age as fish progressing from the resistant alevin stage. These

patterns indicate caution is needed when using SMAVs or GMAVs as a summary statistics for ranking species sensitivity or setting criteria.

Salmonids can have profound difference in susceptibility to chemicals at different life stages and in some instances SMAVs used in criteria may be skewed high because insensitive life stages were included. Across several good datasets, the most vulnerable life stage and size appeared to be swim-up fry weighing between 0.5 and 1.5g.

Summary: Based on this analysis, the risks from relying on toxicity data from species and life stages that are less sensitive than the most sensitive salmonid life stage is moderate to high, as aquatic life criteria derivied following the Guidelines is likely to result in sublethal effects, such as interference in physiochemical processes, interption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Bioconcentration and Bioaccumulation Factors Used in Determining and Evaluating Proposed Criteria Associated with High Variability and Uncertainty. An important problem with many of EPA's chronic criteria for organic pollutants is that the bioconcentration or bioaccumulation factors used in their determination may not be accurate. The BCFs determined in the laboratory based on water-borne exposure are typically much lower than field-derived values, and so may significantly underestimate uptake in the natural environment. Even among field-derived bioconcentration factors, estimates can vary by several orders of magnitude. Consequently, it is difficult to determine if BCF-based comparisons of water-borne and tissues concentrations are accurate when evaluating the chronic criteria proposed in this action.

The Guidelines include a component designed to assure that the water quality criterion for a substance is sufficiently low that residue accumulations will not impair the use of a waterbody by aquatic organisms, and specify that data from residue studies are to be considered alongside acute and chronic toxicity data in the criteria development process (EPA 1985a). However, metals criteria are presently based solely on results of aquatic toxicity tests (62 FR 42159), where metal exposures occur directly across gills or other respiratory surfaces.

Metals and organic contaminants can bioaccumulate, through either bioconcentration (an increase in concentration of a substance in relation to the concentration in ambient water) or biomagnification (a progressive increase in concentration from one trophic level to the next higher level in the aquatic food chain (Moore and Ramamoorthy 1984, Sorensen 1991).

All of the organic pollutants of concern in this action bioaccumulate. All biomagnify to some extent in the food chain, although this is more of a serious concern for some contaminants than others. The Guidelines include a component designed to address the risks of elevated fish tissue residues of organic compounds to humans and avian and mammalian predators, but not the risk of that residue to fish (EPA 1985a). In fact, this process drives nearly all of the numeric criteria established for organic contaminants. What is not considered in these evaluations, however, is whether these tissue residues would directly affect the health of the aquatic organisms. Similar to metals, the consumption of aquatic invertebrates by fish is never formally considered in the development of the criteria for organic compounds. It is well established that invertebrates may accumulate organic contaminants in aquatic systems, and that these contaminants are passed on

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to fish through the diet (*e.g.*, Streit 1998). Consequently, if the water quality criteria do not protect invertebrate prey species from organic residue accumulations, they may not protect listed species from adverse effects associated with dietary exposure.

In particular, measuring compliance with the criteria through ambient water concentrations alone leaves exposure pathways to several organic pollutants un-regulated. For example, dieldrin, lindane, and heptachlor epoxide are not highly water soluble, and are persistent in both food and sediments. A number of the organic compounds reviewed here (*e.g.*, dieldrin, lindane, heptachlor epoxide), have considerable potential to biomagnify in aquatic systems (Suedal *et al.* 1994). The Guidelines for such compounds do not consider food web transfer and bioaccumulation with respect to the target species. Consequently, they may greatly underestimate the toxicity of these chemicals in the environment. This is particularly important for the juvenile life stage of anadromous salmonids while they reside in rearing habitat, if such exposure later influences their downstream migration and subsequent ability to osmoregulate as they enter saltwater. This is an especially significant concern for organic contaminants such as organochlorine pesticides (*e.g.*, dieldrin, lindane, heptachlor epoxide), for which exposure is primarily via sediments and tissues of prey organisms.

A biologically significant pathway for exposures of aquatic organisms to contaminants is through consumption of contaminated aquatic detritus, plants, invertebrates, and other food items (bioaccumulation). Invertebrates that can accumulate metals in aquatic systems are often prey consumed by salmonids and other fish species (*e.g.*, Moore *et al.* 1991, Luoma and Carter 1991, Cain *et al.* 1992, Kiffney and Clements 1993, Rainbow and Dallinger 1993, Timmermans 1993, Ingersoll *et al.* 1994, Dallinger 1994, Cain *et al.* 1995, Gerhardt and Westermann 1995).

In an experiment that shows how readily contaminated food items lead to elevated fish tissue concentrations, Woodward *et al.* (1994) held paired groups of age 0 rainbow trout in clean and contaminated over a range of metal-concentrations. They fed one group a diet of reconstituted, metals contaminated invertebrates, and the other group a comparable diet based on uncontaminated invertebrates. After 91 days, they observed that only fish fed the contaminated diet exhibited reduced survival and growth. These results demonstrate that exposure to a dissolved metal can be a secondary hazard pathway in cases where food is contaminated and fish can bioaccumulate the substance of concern. In cases where fish can bioaccumulate a metal, these results and similar results from other studies of diet-borne metal exposures to salmonids collectively indicate that toxic effects can occur through dietary pathways (*e.g.*, Dallinger and Kautzky 1985, Dallinger *et al.* 1987, Spry *et al.* 1988, Giles 1988, Harrison and Klaverkamp 1989, Harrison and Curtis 1992, Miller *et al.* 1993, Mount *et al.* 1994, Farag *et al.* 1994).

In general, the metals considered in this opinion do not appear to biomagnify in the food chain, with the exception of selenium. The Guidelines include a component designed to assure that the water quality criterion for a substance is sufficiently low that residue accumulations will not impair the use of a waterbody by aquatic organisms, and that data from residue studies are to be considered alongside acute and chronic toxicity data in the criteria development process (EPA 1985a). However, metals criteria are presently based solely on results of aquatic toxicity tests (62 FR 42159), where metal exposures occur directly across gills or other respiratory surfaces.

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Risk management via water concentration-based water quality criteria is not protective of listed salmonids for toxic pollutants that strongly bioaccumulate (*e.g.*, selenium, and organic pollutants: Pease *et al.* 1992; Taylor *et al.* 1992, 1993; Canton 1997; EPA 2001). This is because the true potential for toxic hazards to fish and wildlife through bioaccumulation is determined not only by an immediate water-borne exposure and direct toxicity effects, but also by the rate of mass loading into an aquatic ecosystem, the corresponding environmental partitioning of mass loads between the water column, sediments, and biota (food chain), and how the toxic pollutant is assimilated and acts on the organism. A water column concentration of a toxic pollutant may not reflect mass loading or be reflected in food chain bioaccumulation. Therefore, water quality criteria are useful guides for risk management only to the extent that they protect aquatic food chains from bioaccumulation.

This is an especially significant concern for organic contaminants such as organochlorine pesticides, for which exposure is primarily via sediments and tissues of prey organisms. Indeed, environmental agencies in some other countries, including Canada, no longer recommend water quality guidelines for these substances, but regulate them through other media such as sediment, soil, or tissue (CCREM 2001a).

Because hydrophobic compounds are expected to show a similar or proportional affinity for the lipid of an organism as that for octanol (which is used to calculate the partition coefficient[21]), the degree of partitioning exhibited between water and octanol, as characterized by the partition coefficient K_{ow}, can be a useful means for evaluating and predicting bioaccumulation (Mackay 1982, Di Toro *et al.* 1991). For organic compounds that are not metabolized, the relationship between the bioconcentration factor (BCF) and K_{ow} is strong (Mackay 1982). The expected wet-weight BCF for a non-metabolized hydrophobic compound is a function of the lipid content of an organism and the value of K_{ow} for the compound. The standard equation for determining the expected BCF is:

$$BCF = 0.046 \times K_{ow}$$

which is derived from fish studies and is based on an average lipid content of 4.6% wet weight (McCarty 1986). This relationship is used in this opinion for evaluating effects related to exposure and bioconcentration of the toxic organic pollutants addressed by the ODEQ.

Sediment concentrations that would result in organic toxic pollutant concentrations in the water column can be calculated using the equation (Di Toro *et al.* 1991):

$$SQC_{oc} = K_{oc} \times F_{CV}$$

where:

SQC_{oc} = sediment contaminant concentration in mg/kg organic carbon
K_{oc} = partitioning coefficient for sediment organic carbon

[21] A coefficient representing the ratio of the solubility of a compound in octanol (a non-polar solvent) to its solubility in water (a polar solvent). The higher to K_{OW}, the more non-polar the compound. Log K_{OW} is generally used as a relative indicator of the tendency of an organic compound to adsorb to soil. Log K_{OW} values are generally inversely related to aqueous solubility and directly proportional to molecular weight.

F_{cv} = the chronic water quality criterion in µg/L

K_{oc} can be calculated from the octanol/water partitioning coefficient, Kow, using the formula:

$$\text{Log}_{10}(K_{oc}) = 0.00028 + 0.983 \text{ X } \text{Log}_{10}(K_{ow})$$

This equation is used in the analysis of effects later in this opinion, provided that the data necessary to conduct the analysis were available, to evaluate the potential for water-borne exposure concentrations of organic pollutants at or below criteria concentrations.

Summary: Based on this analysis, the risks of bioconcentration and biooaccumulation factors are likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Insufficient Information on Behavioral and Other Sublethal Endpoints. In the case of chronic criteria, data are available for a range of sublethal effects such as growth and fecundity or sperm production. However, some important effects reported in mammals, such as immunosuppression and endocrine disruption, are inadequately studied in salmonids therefore were not considered in the development of the national criteria. These sublethal effects cannot be considered trivial, because they are associated with the potential for increased mortality (Arkoosh *et al.* 1998). Sublethal effects involving alterations in behavior can occur during relatively low concentration, short-term exposure, and can have profound biological implications (*e.g.*, chemical migration barrier, interference with spawning behavior). The NMFS recognizes that relevant data may not be available for all toxic substances, and that determination of a repeatable, detectable endpoint may involve a degree of subjectivity. Relatively little data are available to help elucidate these concerns; however, the research that does exist indicates that sublethal effects can be very serious for at least some toxicants.

Summary: Based on this analysis, the risks of sublethal effects will exacerbate adverse effects, and are likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Influence of Temperature, pH, and other Water Quality Stressors on Fish Response to Toxicity. In addition to direct influences on toxic pollutant speciation and chemical toxicity mechanisms, several water quality parameters influence general fish health, and susceptibility and ability to acclimate to and depurate after short-term increases in toxic parameter concentrations. This is generally addressed indirectly (with respect to toxicity) through conventional water quality criteria (*e.g.*, water temperature, pH, dissolved oxygen, dissolved gases, ammonia, *etc.*). However, it is possible for fish to be stressed or become stressed more rapidly when conventional water quality parameters are near or exceed criteria limits. This effect pathway is not addressed by most existing toxic pollutant criteria, and represents a shortcoming of the proposed criteria.

Summary: Based on this analysis, the risk that temperature, pH, and other water quality stressors will exacerbate the effects of the proposed criteria is high, as aquatic life criteria derived following the Guidelines do not take these additional stressors into account and are therefore likely to result in sublethals effects, such as interference in physiochemical processes, interption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Toxicity of Total Recoverable vs. Dissolved Metal Concentrations and the Use of Conversion Factors and Translators. Acute and chronic criteria for metals may be interpreted using either total recoverable or dissolved metal concentrations, depending on the objective of the study. The term "total recoverable" metal refers specifically to metal concentrations determined in unfiltered samples that have been acidified (pH < 2) before analysis. The term "dissolved" metal refers specifically to metal concentrations determined in samples that have been filtered (generally a 0.45 micron pore size) prior to acidification and analysis. Total recoverable metal concentration includes both the dissolved form and the portion either attached to particles in the water or present in suspended insoluble form. Particulate metals can be single atoms or metal complexes adsorbed to or incorporated into silt, clay, algae, detritus, plankton, *etc.*, which can be removed from the test water by filtration through a 0.45 micron filter.

Only dissolved metals are immediately bioavailable and thus immediately toxic to freshwater organisms (however, the particulate form may still affect listed species, as discussed below). The non-dissolved form is generally not directly hazardous to listed salmonids except under certain circumstances were (1) changes in water chemistry conditions lead to increased solubility from particulate forms within the water column, or (2) metal contaminated particulates are ingested or encounter gill surfaces. Factors in addition to hardness that influence solubility, and thus bioavailability and toxicity, include suspended sediment concentration, pH, organic carbon content, and chemical speciation of the metal. Further, some metal compounds are less soluble than others for a given set of water quality conditions.

Studies indicate that particulate metals contribute to organism exposure to metals. Particulates may act as a sink for metals, but they may also act as a source. Through chemical, physical, and biological activity, particulate metals can become bioavailable (Moore and Ramamoorthy 1984). Particulate and dissolved metals that end up in sediments are not rendered entirely nontoxic nor completely immobile, and may still contribute to the toxicity of the metal in natural waters. Of special concern are situations where waters contain both high particulate metal concentrations and dissolved concentrations near the proposed criteria. Additionally, those metals that can bioaccumulate through food-chain organisms and can cause indirect effects through particulate metal contamination.

Particulate metals are removed from the proposed regulatory "equation" through at least two methods: the use of CFs to determine the dissolved metal criteria from total recoverable criteria, and the use of a translator to convert back to a total metal concentration for use in waste load limit calculations. When waste discharge limits are to be developed and TMDLs are determined for a receiving waterbody, the dissolved criterion must be "translated" back to a total concentration because TMDLs are based on total metals.

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EPA originally used total metal concentrations to establish national criteria, as provided in the National Toxics Rule published in 1992. The EPA subsequently changed to use of dissolved metal criteria, as explained in a 1993 policy statement:

> [I]t is now the policy of the Office of Water that the use of dissolved metal to is now the policy of the Office of Water that the use of dissolved metal to set and measure compliance with water quality standards is the recommended approach, because dissolved metal more closely approximates the bioavailable fraction of metal in the water column than does total recoverable metal. This conclusion regarding metals bioavailability is supported by a majority of the scientific community within and outside the Agency. One reason is that a primary mechanism for water column toxicity is adsorption at the gill surface which requires metals to be in the dissolved form (Prothro 1993).

Because no supporting references were given in support of the policy, it is hard to evaluate. There is theoretical support for the assumption that metals need to be in dissolved form to adsorb to the gill surface (Wood *et al.* 1997), and it does seem logical to assume that metals bound to particulates would be less toxic. However, two studies that examined the toxicity of particulate metals in controlled experimental studies (Brown *et al.* 1974, Erickson *et al.* 1996) found toxicity associated with particulate bound copper.

Erickson *et al.* (1996) estimated that the adsorbed copper has a relative toxicity of almost half that of dissolved copper, and noted that the assumption that toxicity can be simply related to dissolved copper was questionable, and a contribution of adsorbed copper to toxicity cannot be generally dismissed (Erickson *et al.* 1996). One possible reason for the observed toxicity from particulate-bound copper is that the pH of water changes as it crosses the gills of fish, and at pH of 6 or greater in the water where a fish is living, the pH of water will be lowered as it crosses the gill (Playle and Wood 1989).

Attempting to define, evaluate and manage risks associated with dietary exposures of metals or contaminated sediments by basing criteria on total recoverable metals would likely be so indirect as to be ineffective. However, in the absence of such efforts, the stance that metals sorbed to particles are in effect biologically inert and can safely be ignored is questionable. The effect of this stance is to give up some conservatism in aquatic life criteria for metals.

Conversion Factors. The EPA derived ambient dissolved metals criteria from aquatic toxicity tests that produced dose-response relationships in test organisms under controlled (laboratory) conditions. In most of these studies, organism responses were plotted against nominal test concentrations of metals or concentrations determined by analyzing unfiltered samples to which soluble metal compounds had been added. Thus, until recently, metals criteria have been expressed in terms of total metal concentrations. Current EPA metals policy (EPA 1993a) and the ODEQ stipulate that criteria be expressed on a dissolved basis. The CF used in the EPA formulae for computing criteria represents a corresponding adjustment so that criteria based on total metal concentrations used in laboratory testing can be "converted" to a dissolved basis actually present in the toxicity test solutions. Metals for which a CF has been applied include arsenic, cadmium, chromium, copper, lead, nickel, silver, and zinc.

CF values for the proposed metals criteria are near 1.0 for most metals, because they were determined using laboratory toxicity-test solutions prepared with purified, soluble metal compounds, rather than using natural waters where relative contributions of water-borne particulate metals are much greater. To develop the coversion factors, EPA reviewed test data that reported both total and dissolved concentrations in their test waters and also conducted simulations of earlier experiments to determine the dissolved to total ratios (60 FR 1536, 62 FR 42159). In this way, the historical toxicity database could be utilized and a large number of new toxicity tests would not have to be performed. However, the CFs in many cases (*e.g.*, As, Ni, Cr, Pb) developed based upon a small number of studies and samples compared to the historical database of toxicity tests. Although additional confirmatory studies were performed to develop the CFs, the database available appears to be limited and calls into question the protectiveness of the CFs determined for these metals in cases when site-specific water quality approaches toxic conditions.

Translators. The EPA provides three methods to translate criteria based on dissolved metals to permit-specific criteria based on total recoverable metals. These three methods may result in greatly different outcomes relative to particulate metal loading. These methods are::

1. Determination of a site-specific translator by measuring site specific ratios of dissolved metal to total metal and then dividing the dissolved criterion by this translator. As an example, a site specific ratio of 0.4 (40 percent of the metal in the site water is dissolved) would result in a 2.5-fold allowable increase in the discharge of total metals. The higher the fraction of particulate metal in the site water the greater the allowable discharge of total metal. This is EPA's preferred method.

2. Theoretical partitioning relationship. This method is based on a partitioning coefficient determined empirically for each metal, and (when available), the concentration of total suspended solids in the site-specific receiving water.

3. The translator for a metal is assumed to be equivalent to the Guidance conversion factor for that metal (*i.e.,* use the same value to convert from total to dissolved and back again).

Since translators are needed to calculate discharge limits they become important in determining the total metals allowed to be discharged. In California, economic analyses performed by the EPA and evaluated by the State Water Resources Control Board (SWRCB 1997) indicated that translators based on site-specific data would decrease dischargers costs of implementing the new CTR criteria by an estimated 50%. This cost savings is "directly related to the less stringent effluent limitations that result from the use of site-specific translators," and implies a strong economic incentive for dischargers to reduce costs by developing site-specific translators and ultimately being allowed to discharge more total metals. This conclusion regarding the impact of site specific translators is supported by documents received by the NMFS in the CTR consultation from EPA (*i.e.*, EPA 1997c).

The EPA performed a sensitivity analysis on the effect of the site specific translator, which relies on determining the ratio of metal in water after filtration to metal in water before filtration in downstream waters. The EPA's analysis indicated that use of a site-specific translators to

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calculate criteria would result in greater releases of toxic-weighted metals loads above the option where the CFs are used as the translators. The potential difference was estimated to be between 0.4 million and 2.24 million "toxic weighted" pounds of metals discharged to California waterways (USFWS and NMFS 2000). Lastly, the current use of conversion factors and site specific translators in formula-based metal criteria is not sufficiently protective of threatened and endangered aquatic species because:

- Particulate metals are not regulated, yet chemical, physical, and biological activity can subsequently cause these particulate metals to become bioavailable and cause adverse effects.
- Particulate metal concentrations are not always negligible in critical habitat in Oregon.
- The national criteria were developed using toxicity tests that expose test organisms to metal concentrations with very low contributions from particulate metals.
- Toxicity tests do not assess whether the toxic contributions of particulate metals are negligible when particulate concentrations are great and dissolved concentrations are at or near criteria levels.
- This method has the potential to allow point sources to significantly increase the discharge of total metal loads into the environment, even though dissolved metal criteria are being met by a discharger.
- Metal loading occurs from the water column to streambed sediments.

Summary: Based on this analysis, the risks of using conversion factors and translators is likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

The Water-Effect Ratio Provision. The water-quality criteria for metals all include a WER in their formulas. The WER is the ratio of the test LC_{50} in site water divided by the LC_{50} in laboratory water; the ratio is then multiplied by the aquatic life criteria to obtain a WER-adjusted site-specific criteria. The approach has probably been most used with copper because of the profound effect of organic carbon (DOC) to ameliorate toxicity, which is not correlated with hardness. The purpose of WERs is to empirically account for characteristics other than hardness that might affect the bioavailability and thus toxicity of metals on a site-specific basis. Because the WERs are directly incorporated into the criteria equations, no separate action is needed to change the criteria values using a WER. The default WER value is 1.0 unless DEQ determines that a different value should apply.

The concept of adjusting metals criteria to account for differences in their bioavailability in site waters has long been a precept of water quality criteria (Bergman and Dorward-King 1997, Carlson *et al.* 1984, USEPA 1994). The WER approach uses one or more standard-test species (usually *Ceriodaphnia* and/or fathead minnows), which are tested in tandem in dilution waters collected from the site of interest and in standard reconstituted laboratory water. The results in the laboratory water are presumed to represent the types of waters used in tests relied on by EPA in criteria documents.

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The main problem with this concept and approach is trying to define a single "typical" laboratory dilution water that reflects that used in criteria documents. Testing laboratories may generate valid results using all sorts of different dilution waters including dechlorinated tap water, natural groundwater (well water), natural surface water such as Lake Superior or Lake Erie, and reconstituted waters made from deionized water with added salts. The widely used "Interim Guidance on Determination and Use of Water-Effect Ratios for Metals" (Stephan *et al.* 1994) specified using recipes from EPA or American Society for Testing and Materials (ASTM) for making standardized test water that results in a water hardness with unusually low calcium relative to magnesium concentrations compared to that of most natural waters. This has the effect of making metals in the reconstituted laboratory water made by standard recipe more toxic than would be expected in water with more natural proportions of Ca and Mg. This is because, at least for fish and some invertebrates and copper, Ca reduces toxicity but Mg affords little or no protection (Borgmann *et al.* 2005, Naddy *et al.* 2002, Welsh *et al.* 2000). Lastly, the water-effect ratio seems to have always been recognized by EPA as an interim, operational substitute to establishing criteria on a more mechanistic basis that could directly account for a lot of the factors that affect toxicity. A major development toward this is the biotic ligand model (BLM) which is supposed to capture the major interactions between metals concentrations, competition, and complexation, which control bioavailability and thus toxicity (Di Toro *et al.* 2001, Niyogi and Wood 2004). For copper, the BLM was used as the basis of EPA's (2007) updated aquatic life criterion, which for copper at least, should negate much of the need for empirical WER testing.

Summary: Based on this analysis, the risks of using water-effect ratios is likely to result in sublethal effects, such as interference in physiochemical processes, interruption of ecological interactions, changes in pathological stress, and toxicosis of listed species considered in this opinion.

Summary of the Derivation of the EPA Aquatic Life Criteria. Based on the analysis on the derivation of the EPA aquatic life criteria, NMFS concludes that predicted effects associated with the aquatic life criteria are likely to be significantly greater than asserted and are likely to have significant consequences for field-exposed species.

APPENDIX 2: ECOTOX References Sources

Freshwater Criteria

Freshwater dieldrin:

Author	Year	Reference Source
Brooke, L.T.	1993	U.S.EPA Contract No.68-C1-0034, Work Assignment No.5, to R.L.Spehar, U.S.EPA, Duluth, MN :18 p.
Chadwick and Shumway	1969	Ambient Water Quality Criteria for Aldrin/Dieldrin, USEPA, October,1980
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
Douglas, M.T., D.O. Chanter, I.B. Pell, and G.M. Burney	1986	Aquat.Toxicol. 8(4):243-249
Gilroy, D.J., H.M. Carpenter, L.K. Siddens, and L.R. Curtis	1993	Fundam.Appl.Toxicol. 20(3):295-301
Hendricks, J.D., T.P. Putnam, and R.O. Sinnhuber	1979	J.Environ.Pathol.Toxicol. 2(3):719-728
Holden, A.V.	1966	J.Appl.Ecol. 3:45-53
Katz, M.	1961	Trans.Am.Fish.Soc. 90(3):264-268
Katz, M.	1961	Ambient Water Quality Criteria for Aldrin/Dieldrin, USEPA, October,1980
Lunn, C.R., D.P. Toews, and D.J. Pree	1976	Can.J.Zool. 54(2):214-219
Macek, *et al.*	1969	Ambient Water Quality Criteria for Aldrin/Dieldrin, USEPA, October,1980
MacPhee, C., and R. Ruelle	1969	Univ.of Idaho Forest, Wildl.Range Exp.Station Bull.No.3, Moscow, ID :112 p.
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)
Mayhew, J.	1955	Proc.Iowa J.Acad.Sci. 62:599-606
Mehrle, P.M., F.L. Mayer, and W.W. Johnson	1977	In: F.L.Mayer and J.L.Hamelink (Eds.), Aquatic Toxicology and Hazard Evaluation, 1st Symposium, ASTM STP 634, Philadelphia, PA :269-280 (Publ in Part As 6797)
Reinert, R.E., L.J. Stone, and H.L. Bergman	1974	Proc.17th Conf.Great Lakes Res .:52-58
Schoettger, R.A.	1970	U.S.Dep.Interior, Bur.Sport Fish.Wildl.Res., Publ. 106:2-40 (Publ in Part As 6797)
Shubat, P.J., and L.R. Curtis	1986	Environ.Toxicol.Chem. 5(1):69-77
Statham, C.N., and J.J. Lech	1975	Toxicol.Appl.Pharmacol. 34(1):83-87
Swedburg, D.	1969	Prog.Sport Fish Res., Div.Fish.Res., Bureau Sport Fish Wildl. 88:8-9
Van Leeuwen, C.J., P.S. Griffioen, W.H.A. Vergouw, and J.L. Maas-Diepeveen	1985	Aquat.Toxicol. 7(1-2):59-78

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Freshwater endosulfan-alpha and endosulfan-beta:

Author	Year	Reference Source
Lemke, A. E.	1980	Ambient Water Quality Criteria for Endosulfan, USEPA , October, 1980.
Macek, K. J., et al	1969	Ambient Water Quality Criteria for Endosulfan, USEPA , October, 1980.
Schoettger, R.A.	1970	Ambient Water Quality Criteria for Endosulfan, USEPA , October, 1980.

Freshwater endrin:

Author	Year	Reference Source
Bennett, R.O., and R.E. Wolke	1987	J.Fish Biol. 31(3):375-385
Bennett, R.O., and R.E. Wolke	1987	J.Fish Biol. 31(3):387-394
Bennett, R.O., and R.E. Wolke	1988	Mar.Environ.Res.24(1-4):351 (ABS)
Dinnel, P.A., J.M. Link, Q.J. Stober, M.W. Letourneau, and W.E. Roberts	1989	Arch.Environ.Contam.Toxicol. 18(5):748-755
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
Eller, L.L.	1971	Am.J.Pathol. 64(2):321-336
Grant, B.F., and P.M. Mehrle	1970	In: Resour.Publ.No.88, Prog.Sport Fish.Res.1969, Div.Fish.Res., Bur.Sport Fish.Wildl., U.S.D.I., Washington, D.C. :13-15
Katz	1961	Ambient Water Quality Criteria for Endrin. USEPA, Oct. 1980
Katz and Chadwick	1961	Ambient Water Quality Criteria for Endrin. USEPA, Oct. 1980
Katz, M.	1961	Trans.Am.Fish.Soc. 90(3):264-268
Katz, M., and G.G. Chadwick	1961	Trans.Am.Fish.Soc. 90(4):394-397
Macek, *et al.*	1969	Ambient Water Quality Criteria for Endrin. USEPA, Oct. 1980
Macek, K.J., C. Hutchinson, and O.B. Cope	1969	Bull.Environ.Contam.Toxicol. 4(3):174-183 (Publ in Part As 6797)
MacPhee, C., and R. Ruelle	1969	Univ.of Idaho Forest, Wildl.Range Exp.Station Bull.No.3, Moscow, ID :112 p.
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)
McKim, J.M., and H.M. Goeden	1982	Comp.Biochem.Physiol.C 72(1):65-74
Post and Schroeder	1971	Ambient Water Quality Criteria for Endrin. USEPA, Oct. 1980
Post, G., and T.R. Schroeder	1971	Bull.Environ.Contam.Toxicol. 6(2):144-155
Thurston, R.V., T.A. Gilfoil, E.L. Meyn, R.K. Zajdel, T.L. Aoki, and G.D. Veith	1985	Water Res. 19(9):1145-1155
Wohlgemuth, E.	1977	Prirodoved.Pr.Ustavu Cesk.Akad.Ved Brne 11(6):1-38 (Author Communication Used); Vertebratologicke Zpravy 1:20-21

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Freshwater heptachlor epoxide:

Author	Year	Reference Source
Johnson, W. W. and M. T. Finley	1980	Human health and aquatic life literature search and data base evaluation for Heptachlor Epoxide. USEPA, Office of Water Regulations and Standards, Sept. 30, 1985
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)

Freshwater lindane:

Author	Year	Reference Source
	1960	Wash.Dep.Fish.Res.Bull. 5:1-161
Biagianti-Risbourg, S., C. Pairault, G. Vernet, and H. Boulekbache	1996	Chemosphere 33(10):2065-2079
Boulekbache, H., and C. Spiess	1974	Bull.Soc.Zool.Fr. 99(1):79-85 (FRE) (ENG ABS)
Katz, M.	1961	Trans.Am.Fish.Soc. 90(3):264-268
Macek, K.J., and W.A. McAllister	1970	Trans.Am.Fish.Soc. 99(1):20-27 (Publ in Part As 6797)
Macek, K.J., K.S. Buxton, S.K. Derr, J.W. Dean, and S. Sauter	1976	EPA-600/3-76-046, U.S.EPA, Duluth, MN :50 p.
MacPhee, C., and R. Ruelle	1969	Univ.of Idaho Forest, Wildl.Range Exp.Station Bull.No.3, Moscow, ID :112 p.
Matsuo, K., and T. Tamura	1970	Sci.Pest Control/Botyu-Kagaku 35(4):125-130
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)
McLeay, D.J.	1976	J.Fish.Res.Board Can. 33(6):1303-1311
Oliver, B.G., and A.J. Niimi	1985	Environ.Sci.Technol. 19(9):842-849
Peterson, R.H.	1976	J.Fish.Res.Board Can. 33(8):1722-1730
Rozados, M.V., M.D. Andres, and M.A. Aldegunde	1991	Aquat.Toxicol. 19(1):33-40
Tooby, T.E., and F.J. Durbin	1975	Environ.Pollut. 8(2):79-89
Tooby, T.E., P.A. Hursey, and J.S. Alabaster	1975	Chem.Ind.(Lond.) 21:523-526

Freshwater pentachlorophenol:

Author	Year	Reference Source
Alabaster, J.S.	1969	Int.Pest Control 11(2):29-35 (Author Communication Used)
Alexander, D.G., and R.M.V. Clarke	1978	Water Res. 12(12):1085-1090
Bentley, R.E., T. Heitmuller, B.H. Sleight III, and P.R. Parrish	1975	U.S.EPA, Criteria Branch, WA-6-99-1414-B, Washington, D.C .:13
Burridge, L.E., and K. Haya	1990	Bull.Environ.Contam.Toxicol. 45(6):888-892
Cardwell, R.D., D.G. Foreman, T.R. Payne, and D.J. Wilbur	1976	EPA-600/3-76-008, U.S.EPA, Duluth, MN :125 p.(Publ in Part As 2149)
Castren, M., and A. Oikari	1987	Comp.Biochem.Physiol.C 86(2):357-360
Chapman, G.A.	1969	Ph.D.Thesis, Oregon State University, Corvallis, OR :87 p.
Chapman, G.A., and D.L. Shumway	1978	In: K.R.Rao (Ed.), Pentachlorophenol: Chemistry,

Author	Year	Reference Source
		Pharmacology, and Environmental Toxicology, Plenum Press, New York, NY :285-299
Davis, J.C., and R.A.W. Hoos	1975	J.Fish.Res.Board Can. 32(3):411-416
Dominguez, S.E., and G.A. Chapman	1984	Arch.Environ.Contam.Toxicol. 13:739-743
Douglas, M.T., D.O. Chanter, I.B. Pell, and G.M. Burney	1986	Aquat.Toxicol. 8(4):243-249
Fogels, A., and J.B. Sprague	1977	Water Res. 11(9):811-817
Glickman, A.H., C.N. Statham, A. Wu, and J.J. Lech	1977	Toxicol.Appl.Pharmacol. 41(3):649-658
Hattula, M.L., V.M. Wasenius, H. Reunanen, and A.U. Arstila	1981	Bull.Environ.Contam.Toxicol. 26(3):295-298
Hickie, B.E., and D.G. Dixon	1987	Aquat.Toxicol. 9(6):343-353
Hickie, B.E., D.G. Dixon, and J.F. Leatherland	1989	Fish Physiol.Biochem. 6(3):175-185
Hodson, P.V., and B.R. Blunt	1981	Aquat.Toxicol. 1(2):113-127
Hodson, P.V., D.G. Dixon, and K.L.E. Kaiser	1984	Environ.Toxicol.Chem. 3(2):243-254
Iwama, G.K., and G.L. Greer	1980	Trans.Am.Fish.Soc. 109(2):290-292
Iwama, G.K., and G.L. Greer	1982	Can.Tech.Rep.Fish.Aquat.Sci.No.1100, Dep.of Fisheries and Oceans, West Vancouver, B.C :9p.
Iwama, G.K., and G.L. Greer	1979	Bull.Environ.Contam.Toxicol. 23(4/5):711-716
Johnson and Finley	1980	Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates, Resource Publication 137. U.S. Department of Interior, Fish and Wildlife Service, Washington, DC, 1980.6-56
Kennedy, C.J.	1990	Ph.D.Thesis, Simon Fraser University, Canada:188 p.; Diss.Abstr.Int.B Sci.Eng.53(1):18 (1992)
MacPhee, C., and R. Ruelle	1969	Univ.of Idaho Forest, Wildl.Range Exp.Station Bull.No.3, Moscow, ID :112 p.
Matida, Y., S. Kimura, M. Yokote, H. Kumada, and H. Tanaka	1971	Bull.Freshwater Fish.Res.Lab.(Tokyo) 20(2):127-146
Mayer, F.L.J., and M.R. Ellersieck	1986	Resour.Publ.No.160, U.S.Dep.Interior, Fish Wildl.Serv., Washington, DC :505 p. (USGS Data File)
McKim, J., P. Schmieder, and G. Veith	1985	Toxicol.Appl.Pharmacol. 77:1-10
McKim, J.M., P.K. Schmieder, and R.J. Erickson	1986	Aquat.Toxicol. 9(1):59-80
McKim, J.M., P.K. Schmieder, R.W. Carlson, E.P. Hunt, and G.J. Niemi	1987	Environ.Toxicol.Chem. 6:295-312
Negilski, D.S.	1973	M.S.Thesis, Oregon State Univ., Corvallis, OR:80 p.(Author Communication Used)
Niimi, A.J., and C.A. McFadden	1982	Bull.Environ.Contam.Toxicol. 28(1):11-19
Office of Pesticide Programs	2000	Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.
Oikari, A.O.J.	1987	Bull.Environ.Contam.Toxicol. 39(1):23-28
Peterson, R.H.	1976	J.Fish.Res.Board Can. 33(8):1722-1730
Sappington, L.C., F.L. Mayer, F.J. Dwyer, D.R. Buckler, J.R. Jones, and M.R. Ellersieck	2001	Environ.Toxicol.Chem. 20(12):2869-2876

Author	Year	Reference Source
Shumway, D.L., and J.R. Palensky	1973	EPA-R3-73-010, U.S.EPA, Washington, D.C. :80 p.
Slooff, W.	1978	In: O.Hutzinger, I.H.Van Lelyveld and B.C.Zoeteman (Eds.), Aquatic Pollutants: Transformation and Biological Effects, Pergamon Press, NY :501-506
Statham, C.N., and J.J. Lech	1975	Toxicol.Appl.Pharmacol. 34(1):83-87
Stehly, G.R., and W.L. Hayton	1989	Aquat.Toxicol. 14(2):131-148
Thurston, R.V., T.A. Gilfoil, E.L. Meyn, R.K. Zajdel, T.L. Aoki, and G.D. Veith	1985	Water Res. 19(9):1145-1155
Van den Heuvel, M.R., L.S. McCarty, R.P. Lanno, B.E. Hickie, and D.G. Dixon	1991	Aquat.Toxicol. 20(4):235-252
Vigers, G.A., and A.W. Maynard	1977	Water Res. 11(4):343-346
Webb, P.W., and J.R. Brett	1973	J.Fish.Res.Board Can. 30(4):499-507

Freshwater ammonia:

Author	Year	Reference Source
Allan, I.R.H.	1955	Int.Assoc.Theor.Appl.Limnol.Proc./Int.Ver.Theor.Angew.Limnol.Verh. 12:804-810
Arillo, A., C. Margiocco, and F. Melodia	1979	J.Fish Biol. 15(4):405-410
Arillo, A., C. Margiocco, and F. Melodia	1979	Boll.Mus.Ist.Biol.Univ.Genova 47:83-91
Arillo, A., C. Margiocco, F. Melodia, P. Mensi, and G. Schenone	1981	Environ.Technol.Lett. 2:285-292
Arillo, A., N. Maniscalco, C. Margiocco, F. Melodia, and P. Mensi	1979	Comp.Biochem.Physiol.C 63(2):325-331
Arillo, A., R. Mantovani, C. Margiocco, F. Melodia, and P. Mensi	1979	Mem.Ist.Ital.Idrobiol.Dott Marco Marchi 37:51-61
Arthur *et al.*	1987	Bull. Environ. Contam. Toxicol. 38:324-331
Belding, D.L.	1927	Trans.Am.Fish.Soc. 57:100-119
Buhl and Hamilton	2000	Trans. Am. Fish. Soc., 129:2, 408-418.
Burrows, R.E.	1964	U.S.Fish Wildl.Serv., Res.Rep.No.66, Washington, DC :12 p.

Author	Year	Reference Source
Calamari *et al.*	1997	Nuovi Ann. Ig. Microbiol. 28:333-345.
Calamari *et al.*	1981	Rapp. P.-v. Reun. Cons. int. Explor. Mer. 178:81-86.
Calamari, D., and R. Marchetti	1975	Prog.Water Technol. 7(3/4):569-577
Corti, U.A.	1951	Int.Assoc.Theor.Appl.Limnol.Proc./Int.Ver.Theor.Angew.Limnol.Verh. 11:84-87
Danecker, E.	1964	Osterreichs Fischerei.3/4:55-68 (ENG TRANSL)
Department of Scientific and Industrial Research	1955	Dep.Sci.Ind.Res., Water Pollut.Res.Bd., London :81 p.
Environment Canada	2004	Guideline for the release of ammonia dissolved in water found in wasterwater effluents.
Environment Canada	2004	Ammoniaproject: summary of pure ammonia rainbow trout toxicity testing.
Fedorov, K.Y., and Z.V. Smirnova	1978	Vopr.Ikhtiol. 19(2):320-328
Fisher, C.J., and C.D. Ziebell	1980	Eisenhower Consortium Bull. 7:1-11
Fitzsimons, J.D.	1989	Proc.32nd Conf.Great Lakes Res.:48 (ABS)
Guerra, M., and N. Comodo	1972	Boll.Soc.Ital.Biol.Sper. 48(22):898-901 (ITA)
Herbert, D.W.M.	1956	Bull.Cent.Belge Etud.Documentation Des Eaux 32:115-120
Holland, G.A., J.E. Lasater, E.D. Neumann, and W.E. Eldridge	1960	Res.Bull.No.5, State of Washington Dept.Fish., Seattle, WA :263 p.
Knoph	1992	Parr. 101C:275-282.
Kreutzmann, H.L., and H. Sordyl	1985	Zool.Jahrb.Abt.Allg.Zool.Physiol.Tiere 89(4):427-439 (GER) (ENG ABS)
MacPhee, C., and R. Ruelle	1969	Bull.No.3, Forest, Wildl.and Range Exp.Stn., Univ.of Idaho, Moscow, ID :112 p.

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Phillips, A.M.	1950	N.Y.State Conservation Dep.Fish.Res.Bull.14, Cortland Hatchery Rep.No.19, Cortland, NY :14-16
Rushton, W.	1921	Salmon Trout Mag. 25:101-117
Servizi and Gordon	1990	Bull. Environ. Contam. Toxicol. 1990; 44(4):650-6.
Servizi, J.A., and R.W. Gordon	1990	Bull.Environ.Contam.Toxicol. 44(4):650-656
Smith, C.E.	1972	Am.Fish.Trout News 17:7-8
Smith, C.E., and R.G. Piper	1975	In: W.E.Ribelin and G.Migaki (Eds.), The Pathology of Fishes, University of Wisconsin Press, Madison, WI :497-514
Soderberg and Meade	1992	J. Appl. Aquaculture 1:83-92
Soderberg, R.W.	1985	J.Fish Dis. 8(1):57-64
Soderberg, R.W., J.B. Flynn, and H.R. Schmittou	1983	Trans.Am.Fish.Soc. 112(3):448-451
Speare, D., and S. Backman	1988	Can.Vet.J. 29:666
Taylor, E.W., and R.W. Wilson	1994	In: D.J.Randall, H.Xiang and R.V.Thurston (Eds.), EPA-600-R-94-138, Fish Physiology, Toxicology and Water Quality Management, U.S.EPA, Athens, GA :36-46
Taylor, J.E.	1973	Trans.Nebr.Acad.Sci. 2:176-181
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Water Pollution, Research Board	1959	In: Water Pollution Research 1959, Water Pollution Research Board, Dep.of Scientific and Industrial Research, H.M.Stationery Office, London, England :74-80
Wicks and Randall	2002	Aquat. Toxicol. 59[1/2], 71-82.
Wicks *et al.*	2002	Aquat. Toxicol. 59[1/2], 55-69.

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Freshwater aluminum:

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Becker, A.J.Jr., and Menendez	1974	
Birge, W.J.	1978	In: J.H.Thorp and J.W.Gibbons (Eds.), Dep.Energy Symp.Ser., Energy and Environmental Stress in Aquatic Systems, Augusta, GA 48:219-240
Birge, W.J., J.A. Black, A.G. Westerman, and J.E. Hudson	1980	In: C.Gale (Ed.), EPA-600/9-80-022, Oil Shale Symposium: Sampling, Analysis and Quality Assurance, March 1979, U.S.EPA, Cincinnati, OH :519-534 (U.S.NTIS PB80-221435)
Birge, W.J., J.A. Black, and A.G. Westerman	1979	In: S.W.Nielsen, G.Migaki, and D.G.Scarpelli (Eds.), Symp.Animals Monitors Environ.Pollut., 1977, Storrs, CT 12:108-118
Birge, W.J., J.E. Hudson, J.A. Black, and A.G. Westerman	1978	In: Symp.U.S.Fish Wildl.Serv., Surface Mining Fish Wildl.Needs in Eastern U.S., W.VA :97-104
Birge, W.J., R.D. Hoyt, J.A. Black, M.D. Kercher, and W.A. Robison	1993	Am.Fish.Soc.Symp. 14:55-65
Brodeur, J.C., T. Ytrestoyl, B. Finstad, and R.S. McKinley	1999	Can.J.Fish.Aquat.Sci. 56(2):184-190
Buckler, D.R., L. Cleveland, E.E. Little, and W.G. Brumbaugh	1995	Aquat.Toxicol. 31(3):203-216
Call, D.J., L.T. Brooke, C.A. Lindberg, T.P. Markee, D.J. McCauley, and S.H. Poirier	1984	Tech.Rep.Project No.549-238-RT-WRD, Center for Lake Superior Environmental Studies, University of Wisconsin, Superior, WI./November 27, 1984 Memo to C.Stephan, U.S.EPA, Duluth, MN :46 p. (Author Communication Used)
Cleveland, L., D.R. Buckler, and W.G. Brumbaugh	1991	Environ.Toxicol.Chem. 10(2):243-248
Cleveland, L., E.E. Little, R.H. Wiedmeyer, and D.R. Buckler	1989	In: T.E.Lewis (Ed.), Environmental Chemistry and Toxicology of Aluminum, Chapter 13, Lewis Publ., Chelsea, MI :229-246
Cleveland, L., E.E. Little, S.J. Hamilton, D.R. Buckler, and J.B. Hunn	1986	Trans.Am.Fish.Soc. 115:610-620
DeLonay, A.J.	1991	M.S.Thesis, University of Missouri-Columbia, Columbia, MO :78 p.
DeLonay, A.J., E.E. Little, D.F. Woodward, W.G. Brumbaugh, A.M. Farag, and C.F. Rabeni	1993	Environ.Toxicol.Chem. 12:1223-1232
Driscoll, C.T.J., J.P. Baker, J.J. Bisogni Jr., and C.L. Schofield	1980	Nature 284(5752):161-164
Everhart, W.H., and R.A. Freeman	1973	EPA/R3-73-011B, U.S.EPA, Washington, D.C :46 p.
Freeman, R.A., and W.H. Everhart	1971	Trans.Am.Fish.Soc. 100(4):644-658
Goss, G.G., and C.M. Wood	1988	J.Fish Biol. 32(1):63-76
Gundersen, D.T., S. Bustaman, W.K. Seim, and L.R. Curtis	1994	Can.J.Fish.Aquat.Sci. 51:1345-1355
Hamilton, S.J., and T.A. Haines	1995	Can.J.Fish.Aquat.Sci. 52(11):2432-2444
Handy, R.D., and F.B. Eddy	1989	J.Fish.Biol. 34(6):865-874
Heming, T.A., and K.A. Blumhagen	1988	Aquat.Toxicol. 12(2):125-140

Author	Year	Reference Source
Hickie, B.E., N.J. Hutchinson, D.G. Dixon, and P.V. Hodson	1993	Can.J.Fish.Aquat.Sci. 50:1348-1355
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Birge, W.J., J.A. Black, A.G. Westerman, and J.E. Hudson	1980	In: C.Gale (Ed.), EPA-600/9-80-022, Oil Shale Symposium: Sampling, Analysis and Quality Assurance, March 1979, U.S.EPA, Cincinnati, OH :519-534 (U.S.NTIS PB80-221435)
Birge, W.J., J.A. Black, and A.G. Westerman	1979	In: S.W.Nielsen, G.Migaki, and D.G.Scarpelli (Eds.), Symp.Animals Monitors Environ.Pollut., 1977, Storrs, CT 12:108-118
Birge, W.J., J.E. Hudson, J.A. Black, and A.G. Westerman	1978	In: Symp.U.S.Fish Wildl.Serv., Surface Mining Fish Wildl.Needs in Eastern U.S., W.VA :97-104
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Buhl, K.J., and S.J. Hamilton	1990	Ecotoxicol.Environ.Saf. 20(3):325-342
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Hale, J.G.	1977	Bull.Environ.Contam.Toxicol. 17(1):66-73
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Birge, W.J.	1978	In: J.H.Thorp and J.W.Gibbons (Eds.), Dep.Energy Symp.Ser., Energy and Environmental Stress in Aquatic Systems, Augusta, GA 48:219-240
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Birge, W.J., J.A. Black, A.G. Westerman, and B.A. Ramey	1983	Fundam.Appl.Toxicol. 3:237-242
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Hamilton, S.J., and K.J. Buhl	1990	Ecotoxicol.Environ.Saf. 20(3):307-324
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Birge, W.J., J.A. Black, A.G. Westerman, and J.E. Hudson	1980	In: C.Gale (Ed.), EPA-600/9-80-022, Oil Shale Symposium: Sampling, Analysis and Quality Assurance, March 1979, U.S.EPA, Cincinnati, OH :519-534 (U.S.NTIS PB80-221435)
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Birge, W.J., J.E. Hudson, J.A. Black, and A.G. Westerman	1978	In: Symp., U.S.Fish Wildl.Serv., Dec.3-6, 1978, Surface Mining Fish Wildl.needs in Eastern U.S., WV :97-104
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Hogstrand, C., F. Galvez, and C.M. Wood	1996	Environ.Toxicol.Chem. 15(7):1102-1108
Holcombe, G.W., G.L. Phipps, A.H. Sulaiman, and A.D. Hoffman	1987	Arch.Environ.Contam.Toxicol. 16:697-710 (OECDG Data File)
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Grande, M.	1966	Adv.Water Pollut.Res. 1:97-111
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Sprague, J.B.	1964	J.Fish.Res.Board Can. 21(1):17-26
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Author	Year	Reference Source
Dinnel, P.A., J.M. Link, Q.J. Stober, M.W. Letourneau, and W.E. Roberts	1989	Arch.Environ.Contam.Toxicol. 18(5):748-755

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Author	Year	Reference Source
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Buhl, K.J., and S.J. Hamilton	1991	Ecotoxicol.Environ.Saf. 22:184-197
Hamilton, S.J., and K.J. Buhl	1990	Ecotoxicol.Environ.Saf. 20(3):307-324
Kazlauskiene, N., A. Burba, and G. Svecevicius	1994	Ekologija 1:33-36
Office of Pesticide Programs	2000	Environmental Fate and Effects Division, U.S.EPA, Washington, D.C.
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Author	Year	Reference Source
Dinnel, P.A., J.M. Link, Q.J. Stober, M.W. Letourneau, and W.E. Roberts	1989	Arch.Environ.Contam.Toxicol. 18(5):748-755
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208

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Author	Year	Reference Source
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
Dinnel, P.A., J.M. Link, Q.J. Stober, M.W. Letourneau, and W.E. Roberts	1989	Arch.Environ.Contam.Toxicol. 18(5):748-755

Saltwater lead:

Author	Year	Reference Source
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Author	Year	Reference Source
Hamilton, S.J., and K.J. Buhl	1990	Arch.Environ.Contam.Toxicol. 19(3):366-373

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Author	Year	Reference Source
Dinnel, P.A., Q.J. Stober, J.M. Link, M.W. Letourneau, W.E. Roberts, S.P. Felton, and R.E. Nakatani	1983	Final Report, FRI-UW-8306, Fisheries Research Inst., School of Fisheries, University of Washington, Seattle, WA :208
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Saltwater tributyltin:

Author	Year	Reference Source
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Short, J.W., and F.P. Thrower	1987	Aquaculture 61(3-4):193-200
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Author	Year	Reference Source
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APPENDIX 3: Direct Mortality Population Modeling

Introduction

To assess the potential for adverse impacts of chemical exposures during subyearling freshwater post-swimup rearing on Pacific salmon populations, two models were developed. One model assesses direct mortality and its impact on population productivity and another model explicitly links impairments in the somatic growth of individual subyearling salmon to the productivity of salmon populations. Both models address impacts on first-year survival, and the results are incorporated into one of four life-history models to quantify changes in population productivity. General life-history models were constructed and analyzed for coho salmon (*Oncorhynchus kisutch*), sockeye salmon (*O. nerka*) and ocean-type and stream-type Chinook salmon (*O. tshawytscha*). For this exercise a population is defined following Ricker's (1972) definition of a "stock" as "a group of fish of the same species that spawns in a particular lake or stream (or portion thereof) at a particular season and which, to a substantial degree, does not interbreed with fish from any other group spawning in a different place or in the same place at a different season." The investigation of population-level responses to chemical exposures uses life-history transition matrix models. Individuals within a population exhibit various growth, reproduction, and survivorship rates depending on their developmental or life-history stage or age. The life-history strategy and demographic rates defining the survival and reproductive contribution of the various age classes determine the population productivity and determine the model transition matrix. Alterations of the demographic rates can impact a population's intrinsic growth rate which is calculated directly from the transition matrix as described below.

The basic salmonid life history consists of hatching and rearing in freshwater, smoltification in estuaries, migration to the ocean, growth to maturation at sea, and returning to the natal freshwater stream for spawning followed shortly by death. Differences between the four modeled life-history strategies are lifespan of the female, time to reproductive maturity, and the number and relative contribution of the reproductive age classes (Figure A1). The coho females modeled reach reproductive maturity at age 3 and provide all of the reproductive contribution at this time. Sockeye females in the modeled life history reach maturity at age 4 or 5, but the majority of reproductive contributions are provided by age 4 females. Chinook females can mature at age 3, 4 or 5, with the majority of the reproductive contribution from ages 4 and 5. The primary difference between the ocean-type and stream-type Chinook is juvenile freshwater residence time, with ocean-type juveniles migrating to the ocean as subyearlings and stream-type Juveniles overwintering in freshwater and migrating to the ocean as yearling smolts. The models depicted general populations representing each life-history strategy and were constructed based upon literature data described below. Specific populations were not modeled due to the difficulty in finding sufficient demographic and reproductive data for single populations.

The endpoint used to assess population-level impacts for both the somatic growth model and the direct mortality population model was the percent change in the intrinsic population growth rate (lambda, λ) resulting from the chemical exposure. Change in λ is an accepted population parameter often used in evaluating population productivity, status, and viability. The National Marine Fisheries Service uses changes in λ when estimating the status of species, conducting risk and viability assessments, developing Endangered Species Recovery Plans, composing

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Biological Opinions, and communicating with other federal, state and local agencies (McClure *et al.*, 2003). While values of $\lambda<1.0$ indicate a declining population, in cases when an exposure causes the population growth rate to decrease more than natural variability, a loss of productivity will result even if lambda remains above 1.0. Decreases in response to chemical exposures can be a cause for concern since the impact could make a population more susceptible to declining (lambda dropping below 1.0) due to impacts from other stressors.

To determine if population productivity would be at risk due to direct mortality resulting from either acute or chronic exposures to the criterion concentrations of the chemicals of concern, a direct mortality population model was constructed. This model assessed whether juvenile salmon during their freshwater residence encountering the established criterion concentrations would experience individual mortality, and if that mortality would be sufficient to produce a change in the population growth rate. This included direct mortality from either acute or chronic exposures. The model applied a mortality factor to first-year survival of the respective life-history models to assess changes in lambda.

In the freshwater portion of their life, Pacific salmon are exposed to chemicals that also may act in a sublethal manner by inhibiting somatic growth. Juvenile growth is a critical determinant of freshwater and marine survival for Chinook salmon (Higgs *et al.* 1995). Reductions in the somatic growth rate of salmon fry and smolts are believed to result in increased size-dependent mortality (Healey 1982, West and Larkin 1987, Zabel and Achord 2004). Zabel and Achord (2004) and Mebane and Arthaud (2010) observed size-dependent survival for Juvenile salmon during the freshwater phase of their outmigration. Mortality is also higher among smaller and slower growing salmon because they are more susceptible to predation during their first winter (Healey 1982, Holtby *et al.* 1990, Beamish and Mahnken 2001). These studies suggest that factors affecting the organism and reducing somatic growth could result in decreased first-year survival and, thus, reduce population productivity. Using a modeling approach, Mebane and Arthaud (2010) suggested that size reductions from early-life stage chronic sublethal copper exposure could potentially reduce Juvenile salmon survival and population recovery trajectories.

Changes in juvenile salmon size due to exposure to the chemicals of concern were linked to size-dependent survival of Juveniles during their first year. Exposures and somatic growth were determined from the free-swimming and feeding fry stage (1.0g fish) to either outmigration, for ocean-type stocks, or to the fall when parr prepare for overwintering, in the case of stream-type stocks. Somatic growth models were constructed for coho, sockeye, ocean-type and stream-type Chinook. A steelhead (*O. mykiss*) life-history model was not constructed due to the lack of demographic information relating to the proportions of resident and anadromous individuals, the freshwater residence time of steelhead, and rates of repeated spawning. Models for chum (*O. keta*) and pink salmon (*O. gorbuscha*) were not constructed due to their short freshwater residence which would not allow sufficient rearing time to alter somatic growth rate and size to the point of altering survival rates. The somatic growth model used here is an extension of one developed for investigating the effects of pesticides on the biochemistry, behavior and growth of ocean-type Chinook salmon (Baldwin *et al.*, 2009).

The following descriptions detail how the direct mortality and somatic growth models were developed to serve as a means to assess the potential effects on ESA-listed salmon populations

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from exposure to chemicals that cause direct mortality and reductions in somatic growth. Comparing the results from different chemical exposure scenarios to a control (*i.e.* unexposed) scenario can indicate the potential for chemical exposures to lead to changes in either mortality or somatic growth and size-dependent survival of individual subyearling salmon. Subsequent changes in salmon population dynamics as indicated by percent change in a population's intrinsic rate of increase assist us in estimating the potential population-level impacts to listed populations.

Methods

Model Life-history Strategies

Both models investigated the population-level responses to chemical exposures using life-history projection matrix models. Individuals within a population exhibit various growth, reproduction, and survivorship rates depending on their developmental or life-history stage or age. These age specific characteristics are depicted in the life-history graph (Figure A1A-C) in which transitions are depicted as arrows. The nonzero matrix elements represent transitions corresponding to reproductive contribution or survival, located in the top row and the subdiagonal of the matrix, respectively (Figure A1C). The survival transitions in the life-history graph are incorporated into the n x n square matrix (A) by assigning each age a number (1 through n) and each transition from age i to age j becomes the element a_{ij} of matrix A (i = row, j = column) and represents the proportion of the individuals in each age passing to the next age as a result of survival. The reproductive element (a_{1j}) gives the number of offspring that hatch per individual in the contributing age, j. The reproductive element value incorporates the proportion of females in each age, the proportion of females in the age that are sexually mature, fecundity, fertilization success, and hatch success.

A prospective analysis of the transition matrix, A, (Caswell 2001) explored the intrinsic population growth rate as a function of the vital rates (survival and reproduction). The intrinsic population growth rate, λ, equals the dominant eigenvalue of A and was calculated using matrix analysis software (MATLAB version 2010b by The Math Works Inc., Natick, MA). Therefore λ is calculated directly from the matrix. Variability was integrated by repeating the calculation of λ 2000 times selecting the values in the transition matrix from their normal distribution defined by their mean and standard deviation. The mean value of λ for control and exposed scenarios were determined. From these values the percent change in λ (and standard deviation) was calculated. The influence of each matrix element, a_{ij}, on λ was assessed by calculating the sensitivity values for A. The sensitivity of matrix element a_{ij} equals the rate of change in λ with respect to a_{ij}, defined by $\delta\lambda/\delta a_{ij}$. Higher sensitivity values indicate greater influence on λ. The elasticity of matrix element a_{ij} is defined as the proportional change in λ relative to the proportional change in a_{ij}, and equals (a_{ij}/λ) times the sensitivity of a_{ij}. One characteristic of elasticity analysis is that the elasticity values for a transition matrix sum to unity (one). The unity characteristic also allows comparison of the influence of transition elements and comparison across matrices.

Due to differences in the life-history strategies, specifically lifespan, age at reproduction and first year residence and migration habits, four separate life-history models were constructed representing coho, sockeye, ocean-type Chinook and stream-type Chinook. This was done to

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encompass the different responses of these species to freshwater chemical exposures and assess potentially different population-level responses. In all cases, transition values were determined from literature data on survival and reproductive characteristics of each species. All characteristics exhibit density independent dynamics. The models assume closed systems, allowing no migration impact on population size. No stochastic impacts are included beyond natural variability as represented by selecting parameter values from a normal distribution about a mean value for each model iteration (year). Ocean conditions, freshwater habitat, fishing pressure, and marine resource availability were assumed constant and density independent.

A life-history model was constructed for coho salmon (*O. kisutch*) with a maximum age of 3 years. Spawning occurs in late fall and early winter with emergence from March to May. Fry spend 14-18 months in freshwater, smolt and spend 16-20 months in the saltwater before returning to spawn (Pess *et al.* 2002). Survival numbers were summarized in Knudsen *et al.* (2002) as follows. The average fecundity of each female is 4500 with a standard deviation of 500. The observed number of males:females was 1:1. Mean survival rate (standard deviation) from spawning to emergence is 0.3 (0.07). Survival from emergence to smolt is 0.0296 (0.00029) and marine survival is 0.05 (0.01). All parameters followed a normal distribution (Knudson *et al.* 2002). The calculated values used in the matrix are listed in Table A1. The growth period for first year coho was set at 184 days to represent the time from mid-spring to mid-fall when the temperatures and resources drop and somatic growth slows (Knudson *et al.* 2002, Table A2).

The life-history model for sockeye salmon (*O. nerka*) was based upon the lake wintering populations of Lake Washington, Washington, USA. These female sockeye salmon spend one winter in freshwater, then migrate to the ocean to spend three to four winters before returning to spawn at ages 4 or 5. Jacks return at age 2 after only one winter in the ocean. The age proportion of returning adults is 0.03, 0.82, and 0.15 for ages 3, 4 and 5, respectively (Gustafson *et al.* 1997). All age 3 returning adults are males. Hatch rate and first year survival were calculated from brood year data on escapement, resulting presmolts and returning adults (Pauley *et al.* 1989) and fecundity (McGurk 2000). Fecundity values for age 4 females were 3374 (473) and for age 5 females were 4058 (557) (McGurk 2000). First year survival rates were 0.737/month (Gustafson *et al.* 1997). Ocean survival rates were calculated based upon brood data and the findings that approximately 90% of ocean mortality occurs during the first 4 months of ocean residence (Pauley *et al.* 1989). Matrix values used in the sockeye baseline model are listed in Table A1. The 168 day growth period represents the time from lake entry in mid-spring to early fall when the temperature drops and somatic growth slows (Gustafson *et al.* 1997, Table A2).

A life-history model was constructed for ocean-type Chinook salmon (*O. tshawytscha*) with a maximum female age of 5 and reproductive maturity at ages 3, 4 or 5. Ocean-type Chinook migrate from their natal stream within a couple months of hatching and spend several months rearing in estuary and nearshore habitats before continuing on to the open ocean. Transition values were determined from literature data on survival and reproductive characteristics from several ocean-type Chinook populations in the Columbia River system (Healey and Heard 1984, Howell *et al.* 1985, Roni and Quinn 1995, Ratner *et al.* 1997, PSCCTC 2002, Green and Beechie 2004). The sex ratio of spawners was approximately 1:1. Estimated size-based fecundity of 4511 (65), 5184 (89), and 5812 (102) was calculated based on data from Howell *et al.*, 1985, using length-fecundity relationships from Healy and Heard (1984). Control matrix values are listed in

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Table A1. The growth period of 140 days encompasses the time the fish rear in freshwater prior to entering the estuary and open ocean (Table A2). The first three months of estuary/ocean survival are the size-dependent stage. Size data for determining subyearling Chinook condition indices came from data collected in the lower Columbia River and estuary (Johnson *et al.* 2007).

An age-structured life-history matrix model for stream-type Chinook salmon with a maximum age of 5 was defined based upon literature data on Yakima River spring Chinook from Knudsen *et al.* (2006) and Fast *et al.* (1988), with sex ratios of 0.035, 0.62 and 0.62 for females spawning at ages 3, 4, and 5, respectively. Length data from Fast *et al.* (1988) was used to calculate fecundity from the length-fecundity relationships in Healy and Heard (1984). The 184-day growth period produces control fish with a mean size of 96mm, within the observed range documented in the fall prior to the first winter (Beckman *et al.* 2000). The size-dependent survival encompasses the 4 early winter months, up until the fish are 12 months old.

Direct Mortality Population Model

A direct mortality population model was constructed that estimated the population-level impacts of first-year mortality resulting from exposure to the criterion concentrations of ammonia, copper and cadmium. These models excluded sublethal and indirect effects of the chemical exposures and focused on the population-level outcomes resulting from an annual exposure of young-of-the-year to a chemical at the criterion concentrations. Scenarios were chosen to represent both the acute and chronic criteria. This was done by parameterizing the model with toxicity data (LC_{50}s) derived from short term (<96hrs) and long term (>28day, based on the available data, see Table A3) experiments. The lethal impact was implemented as a change in first year survival for each of the salmon life-history strategies. In order to understand the relative impacts of a short-term exposure of a single chemical on exposed vs. unexposed fish, we used parameters for an idealized control population that exhibits an increasing population growth rate. Four life-history strategies were modeled, ocean-type and stream-type Chinook salmon, coho salmon and sockeye salmon. The details for each general population model are provided above in the *Model life-History Strategies* section.

The mortality responses are modeled as direct reduction in the first-year survival rate (S1 in Table A1 and Figure A1D). Exposures are assumed to result in a cumulative reduction in survival as defined by the concentration and the dose-response curve as defined by the LC_{50} and slope for each chemical. A sigmoid dose-response relationship is used to model the mortality dose-response to be consistent with other dose-response relationships. The model inputs for each scenario are the exposure concentration and fish LC_{50}, as well as the sigmoid slope for the LC_{50}. For a given concentration a chemical survival rate is calculated and is multiplied by the control first-year survival rate, producing an exposed scenario first-year survival for the life-history matrix. Variability is incorporated using means and standard deviations to select from normally distributed survival and reproductive rates and repeating the calculation of lambda 2000 times as described above.

Population model output consists of the percent change in lambda from the unexposed control populations derived from the mean of one thousand calculations each of the unexposed control and the chemical exposed populations. The percent change in lambda (with standard deviation),

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representing alterations to the population productivity, was selected as the primary model output for reasons outlined previously. The percent change in lambda is considered different from control when the difference is greater than the percent of one standard deviation of the control lambda.

Somatic Growth Model

Toxic impacts on somatic growth to individual juvenile salmon were modeled as a change in daily growth rate resulting from an exposure concentration occurring during the growth phase of first year freshwater residence. Toxicity parameters relied on experiments producing EC50 values (effect concentration producing 50% change in growth) and slopes for chronic exposures. Sigmoidal dose-response relationships, at steady-state, between each exposure and somatic daily growth rate were modeled using growth EC50s and slopes. The timecourse for each exposure was built into the model as a pulse with a defined start and end during which the exposure remained constant (Figure A2B). The timecourse for daily growth rate was modeled using two single-order exponential functions, one for the time required for the exposure to reach full effect and the other for time required for complete recovery following the end of the exposure (time-to-effect and time-to-recovery, respectively). For all compounds, both timecourses were assumed to be within a day, so a value of 0.5 was used for the half-lives of effect and recovery. Incorporating dynamic effects and recovery variables does allow the model to simulate differences in the pharmacokinetics (*e.g.* the rates of uptake from the environment and of detoxification) of various chemicals, but this requires additional, compound-specific, data.

The growth models were replicated for 1000 individual fish to capture the variability of possible output. The initial weight of each replicate was selected from a normal distribution with a mean of 1.0 g and standard deviation of 0.1 g. The size of 1.0 g was chosen to represent subyearling size in the mid-spring at the onset of the stable growth trajectory (*i.e.* the growth rate is not changing). For each iteration (day) of the model, the somatic growth rate is calculated for each fish by selecting the parameter values from normal distributions with specified means and standard deviations (Table A2). The weight for each fish is then adjusted based on the calculated daily growth rate to generate a new weight for the next iteration. The length (days) to run the growth portion of the model was selected to represent the time from when the fish enter the linear portion of their growth trajectory in the mid to late spring until they change their growth pattern in the fall due to reductions in temperature and resources or until they migrate out of the system. The mean weights (with standard deviations) after the species-appropriate growth period (Table A2) were used to calculate the size-dependent survival as described below. A sensitivity analysis was run to determine the influence of the parameter values on the size distribution output of the somatic growth model.

The species-specific parameter values defining control conditions, such as the length of the growth period and control daily growth rate are listed in Table A2. Each exposure scenario was defined by a concentration and exposure time for each chemical.

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Below are the mathematical equations used to derive Figure A2.

Figure A2A uses a sigmoid function:

y= bottom + (top – bottom)/(1 + (exposure concentration/EC50)^slope).

Figure A2B uses a step function:

time < start; exposure = 0

start ≤ time ≤ end; exposure = exposure concentration(s)

time > end; exposure = 0.

Figure A2C uses a series of exponential functions:

time < start; y = c

start ≤ time ≤ end; y = c – (c – i)*(1 – exp(-ke*(time – start)))

time > end; ye = c – (c – i)*(1 – exp(-ke*(end – start)))

y = ye + (c – ye)*(1 – exp(-kr*(time – end))).

For Figure A2A, y = Daily Growth Rate, top = Gc, bottom = 0. For Figure A2C, c = Gc, i = Gi, ke = ln(2)/Growth effect half-life, kr = ln(2)/Growth recovery half-life. For Figure A2C the value of ye is calculated to determine the amount of inhibition that is reached during the exposure time, which may not be long enough to reach the maximum level of inhibition.

Linking to Survival in Population Model

The weight distributions from the somatic growth portion of the model are used to calculate size-dependent first-year survival for a life-history matrix population model for each species and life-history type. This incorporates the impact that reductions in size could have on population growth rate and abundance. The first-year survival element of the transition matrix incorporates a size-dependent survival rate for a three- or four-month interval (depending upon the species) which takes the Juveniles up to 12 months of age. This time represents the 4-month early winter survival in freshwater for stream-type Chinook, coho, and sockeye models. For ocean-type Chinook, it is the 3-month period the subyearling smolt spend in the estuary and nearshore habitats (*i.e.* estuary survival). The weight distributions from the organismal model are converted to length distributions by applying condition factors from data for each modeled species (cf; 0.0095 for sockeye and 0.0115 for all others) as shown in Equation L.

Equation L: length(mm) = ((fish weight(g)/cf)^(1/3))*10

The relationship between length and early winter or estuary survival rate was adapted from Zabel and Achord (2004) to match the survival rate for each control model population (Howell *et al.* 1985, Kostow 1995, Myers *et al.* 2006, Figure A3). The relationship is based on the length of a subyearling salmon relative to the mean length of other competing subyearling salmon of the same species in the system, Equation D, and relates that relative difference to size-dependent survival based upon Equation S. The values for α and resulting size-dependent survival (survival ϕ) for control runs for each species are listed in Table A2. The constant α is a species-specific parameter defined such that it produces the correct control survival ϕ value when Δlength equals zero.

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Equation D: Δlength = fish length(mm) – mean length(mm)

Equation S: Survival $\phi = (e^{(\alpha+(0.0329*\Delta length))}) / (1 + e^{(\alpha+(0.0329*\Delta length))})$

Randomly selecting length values from the normal distribution calculated from the organismal model output size and applying equations 1 and 2 generates a size-dependent survival probability for each fish. This process was replicated 1000 times for each exposure scenario and simultaneously 1000 times for the paired control scenario and results in a distribution with a mean size-dependent survival rate for each population. The resulting size-dependent survival rates are inserted in the calculation of first-year survival in the respective control and chemical-exposed transition matrices of the life-history population models described above.

In the population model an individual fish experiences an exposure once as a subyearling (during its first spring) and never again. The chemical exposure is assumed to occur each year to the subyearling age class. All subyearlings within a given population are assumed to be exposed to the chemical. No other age classes experience the exposure. The model integrates this as every brood class being exposed as subyearlings and thus the vital demographic rates of the transition matrix are continually impacted in the same manner. Regardless of other effects due to the direct exposure, only growth effects are incorporated in the model.

The population model recalculates first-year survival for each run using a size-dependent survival value selected from a normal distribution with the mean and standard deviation produced by Equation S. Population model output consists of the percent change in lambda from the unexposed control populations derived from the mean of two thousand calculations of both the unexposed control population and the chemical exposed population. Change in lambda (with standard deviation), representing alterations to the population productivity, was selected as the primary model output for reasons outlined previously.

Model Toxicity Scenario Parameterization

Literature Review. Data for parameterizing the toxicity scenarios for the direct mortality and somatic growth models were identified by conducting extensive literature searches. The first round of searches broadly gathered papers and reports that had toxicological information on the effects of ammonia, cadmium, and copper on mortality and growth in Juvenile salmonids. Several different online databases and print sources were used in the literature search that was conducted to identify appropriate data:

1. The Thomson Reuters online academic citation index, Web of Science, was used. Search terms included the name of the contaminant: (ammonia), (copper OR cu), (cadmium OR cd); types of effects: (LC50 OR acute OR lethal* OR growth*); and order, family, genus, main species names, and main common names of salmonids: (acantholingua OR amago OR arctic char OR arctic cisco OR baikal omul OR bloater OR brachymystax OR char OR Chinook OR chum OR cisco OR coho OR coregoninae OR coregonus OR dolly varden OR grayling OR hucho OR inconnu OR keta OR kisutch OR kiyi OR lake herring OR nerka OR oncorhynchus OR parahucho OR prosopium OR salmo OR salmon OR salmonid* OR salmonidae OR

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salmoniformes OR salmoninae OR salvelinus OR salvethymus OR sockeye OR steelhead OR stenodus OR taimen OR thymallinae OR thymallus OR trout OR tshawytscha OR whitefish).

2. The U.S. The EPA online ECOTOX database was used. This database includes single chemical toxicity information and citations for aquatic life. The query included genus and species names, common names, chemical names, and growth or mortality as effects endpoints (similar to above).

3. The online database Aquatic Sciences and Fisheries Abstracts (AFSA), a component of the international Aquatic Sciences and Fisheries Information System (ASFIS), was used. Input search terms were ammonia and salmon or salmonids.

4. The bibliography of the EPA Draft 2009 Update Aquatic Life Ambient Water Quality Criteria for Ammonia - Freshwater; the EPA Aquatic Life Ambient Freshwater Quality Criteria – Copper 2007 Revision; and the Draft Idaho Water Quality Standards Biological Opinion (section on copper).

5. Citations from relevant research articles and reports that were obtained as part of the above searches, and citations from published literature reviews, were also used.

Toxicity Value selection for Exposure Scenarios

The publications identified by the broad literature search were reviewed for appropriate methodologies, replication, measurement endpoints, and life stages exposed. Those studies with insufficient replication or single exposure concentrations were omitted. The review of studies focused on those conducted with Juvenile salmonids exposed during the life stages between swim-up to parr or subyearling smolt to match with the exposure regimes of the models. When multiple toxicity values or slopes were found, the genus geometric mean was used as the initial model input value. In addition, the minimum species mean values were used to parameterize the model to examine the range of potential impacts and avoid overlooking impacts to sensitive listed species. Direct mortality endpoints were collected from 96-h continuous exposure studies for modeling acute exposures and >28 day exposure studies to model chronic exposures.

Studies critically assessed for growth reported endpoints including changes in weight (wet or dry), length, or biomass resulting from water exposures lasting at least 28 days. The assumptions regarding initial fish size in the somatic growth model are very sensitive to the study data used for parameterization. The model simulates the stable portion of the growth phase during which the growth rate is relatively constant that occurs in Juvenile salmonids from about 1g to the their first fall or until outmigration to ocean habitats (Weatherley and Gill 1995). Younger fry (*e.g.* 0.2g) have very different rates and efficiencies of food conversion than 1g and larger fry and parr (Weatherly and Gill 1995). Fry that still are absorbing their yolk sac may have this reabsorption affected by contaminants. In addition, somatic growth rate responses across temperatures for younger fry differ from those of larger fish (Weatherly and Gill 1995). Therefore, smaller fry commonly found in these studies could respond very differently to contaminant exposures than those at greater than 1g, and studies on these sizes were excluded from consideration. Similarly,

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data from studies initiated with Juveniles greater than 10g were not considered since this is past the majority of growth during the first summer (*e.g.* Thedinga *et al.* 1998, Johnson *et al.* 2007). The specific review and value selection procedures used for ammonia, cadmium and copper are discussed below.

Ammonia: The documents identified by the first round of literature review applying to acute toxicity of ammonia to salmonids were further reviewed for data appropriate to parameterize the direct mortality population model. Data needed to conform to 96-hr LC50 values for subyearling salmonids (free-swimming, 1-4g fish preferred, but did include data on fish of less than 10 g when that was all that was available). The range of values identified for Chinook salmon, coho salmon, rainbow trout and cutthroat trout and are shown below in the units of mg NH_3-N/L, as N. All values were normalized to a pH of 8 using an un-ionized ammonia computer worksheet available from the American Fisheries Society (http://www.fisheries.org/afs/hatchery.html, Table 9 Ammonia Calculator (Freshwater) Excel spread sheet from the web site). Following the practice in the ammonia Ambient Water Quality Criteria documents (1999, 2009), the fish LC50 values were not normalized for temperature. The normalized species mean values were 26.8, 15.1, 26.2 and 29.4 mg NH_3-N/L for Chinook salmon, coho salmon, rainbow trout and cutthroat trout, respectively (Servizi and Gordon 1990; Buckley 1978; Thurston and Russo 1983; Thurston *et al..,* 1981, Table A3). The genus geometric mean from these data was 23.6 mg NH_3-N/L. A sigmoid dose-response slope was calculated as 6.4 (Broderius and Smith 1979; Buckley 1978). Both the genus geometric means and minimum species mean values were used to parameterize the model as discussed above. To assess the chronic criterion, a chronic study was found that exposed cutthroat trout to ammonia for 29 days and reported an LC50 of 21.3 mg NH_3-N/L (Thurston *et al.,* 1978). No slope was identified, so the 96-hr slope was used in the model.

Documents investigating the effects of ammonia on growth of fish were reviewed for data appropriate as input to the somatic growth model. No studies were found that could provide the appropriate data. Most studies on exposure of Juvenile salmonids to ammonia found that any effects on growth or food intake were temporary and compensation occurred before the end of the exposure period (Lang *et al.,* 1987; Linton *et al.,* 1998; Beamish and Tandler 1990; Larmoyeux and Piper 1973). Other studies have shown effects on growth, but exposure occurred over early developmental stages and also produced developmental delays and abnormalities, so differences in size may not have been attributable to direct impacts on metabolism or growth (Brinkman *et al.,* 2009). From a 90-day exposure Brinkman *et al.,* (2009) calculated an EC20 that includes hatch effects, delayed swimup, and sac-fry growth of 5.56 mg NH_3-N/L normalized to pH 8. In addition, Lazorchak and Smith (2007) reported decreases in growth of rainbow trout (size range <0.2g) after a 7 day exposure to ammonium chloride, but at concentrations that overlapped with those inducing mortality in the test population (IC25 ranged from 104-210 mg/L ammonium chloride and LC50 ranged from 163-271 mg/L ammonium chloride). Moreover, the study organisms used by Lazorchak and Smith (2007) were too young to fit within the life stage criteria established for this modeling exercise. In addition, pH was not reported in this study, so accurate normalization was not possible. Broderius and Smith (1979) also exposed small rainbow trout (0.18g) to ammonia over a 30 day period. Significant reductions in growth were seen at 0.32mg NH_3-N/L, but survival was 70% of that observed in the controls (60%), so the quality and usefulness of this data is suspect. The somatic growth model does not incorporate

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direct mortality and would greatly underestimate population-level effects if studies where significant mortality occurred were included. Since data for the appropriate life stages or time frames were unavailable, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium: Studies identified by the first round of literature review as having data on acute and chronic toxicity for the freshwater phase of salmonids was examined to gather data for parameterizing the population models. All data were hardness adjusted to 100 mg $CaCO_3$/L and reported as dissolved cadmium in µg/L using the hardness equations found in Mebane (2006). The acute toxicity focused on 96-h mortality data for swimup fry, parr and subyearling smolt. Species mean values (geometric means of LC50 values) were calculated for *Oncorhynchus tshawytscha, O. kisutch, O. mykiss, and O. clarki lewisi* and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species means at 4.53 µg/L (Table A3). Sigmoid slopes were calculated when dose-response data were available. The resulting geometric mean of the slopes was 6.4 and the range was 4.7-7.8 (Besser *et al.,* 2007, Finlayson and Verrue 1982, Davies *et al.,* 1993). Besser *et al.,* 2007 estimated a 28-day LC50 for rainbow trout of 5.5 µg/L (Table A3). The normalized LC50 value of 5.36 µg/L, and the acute slope of 6.4 were used to parameterize the chronic criteria scenario of the mortality model.

Chronic cadmium studies were examined for applicable input data for the somatic growth model. Studies on the effects of cadmium on the growth of subyearling salmonids supported the statement by Mebane (2006) that growth is seldom a sensitive endpoint for cadmium. At concentrations that produced changes in somatic growth, increased mortality was also observed in most studies (Mebane *et al.,* 2008, Brinkman and Hansen 2007, Hansen *et al.,* 2002b). In 24 and 30 day exposures of Atlantic salmon (*Salmo salar*) a reduction in size was seen after alevins were exposed to 6.75-21.8 µg Cd/L but these concentrations also produced 80-90% mortality (Rombough and Garside 1982, Peterson *et al.,* 1983). Bull trout (*Salvelinus confluentus*) fry (0.2g) exposed to 1.57 µg Cd/L for 55 days (hardness adjusted to 100 mg $CaCO_3$/L) showed a 28% reduction in growth at this single time point, along with a 37% reduction in survival (Hansen *et al.* 2002b). No dose response curve for growth was generated by the study, so these data could not be used for extrapolation to other concentrations. Brinkman and Hansen (2007) exposed brown trout fry (*Salmo* trutta) to cadmium for 30 days under different water chemistries and calculated a range of IC20s from 1.7-4.8 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) for reduced growth in the surviving individuals. Mortality chronic values for the same tests ranged from 2.04 to 4.79 µg Cd/L. They also calculated LC50 values for the first 96h of the exposures and these ranged from 3.27 to 6.75 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L). Possible size-selective mortality or growth compensation due to decreased density were not addressed in the study design. Rainbow trout fry exposed to cadmium for 28 days exhibited increased mortality and dry weight at concentrations above a calculated NOEC of 1.3 µg Cd/L (Besser *et al.*, 2007). This may be attributed to size-selective mortality or an increase in somatic growth. One rainbow trout early-life-stage exposure lasting 62 days determined an EC10 for growth of 0.31 µg Cd/L (hardness adjusted to 100 mg $CaCO_3$/L) without the increased mortality (Mebane *et al.,* 2008). Changes in growth at these life stages (Embryos and alevins) are not compatible with the somatic growth model that assesses changes in free-swimming, feeding fry during the linear portion of their growth phase, and could not be used to parameterize the model. Similarly, brook trout (*Salvelinus fontinalis*) exposed to 0.36 µg Cd/L (hardness adjusted to 100

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mg $CaCO_3$/L) for 30 days showed reduced prey capture efficiencies and differences in prey selection in artificial stream channels (Riddell *et al.*, 2005a, b), which may link to changes in somatic growth, but this link could not be translated into appropriate input parameters for the current growth model.

Copper: Studies identified by the first round of literature review as having data on acute and chronic toxicity for the freshwater phase of salmonids were examined to gather data needed to establish values for several parameters of the population models. All data was hardness adjusted to 100 mg $CaCO_3$/L using the acute and chronic hardness equations for copper (EPA 2002). For studies with non-laboratory water that reported total instead of dissolved copper, total copper was adjusted by 80% to estimate the dissolved portion of copper in µg/L. The acute toxicity focused on 96-h mortality data for swim-up fry, parr and subyearling fish. Species mean values (geometric means of LC50 values) were calculated (Table A3) and the genus mean for *Oncorhynchus* was calculated as the geometric mean of the species. For direct mortality, the genus mean LC50 was 86.8 µg/L with species means ranging from 48.3-190.6 µg/L, while for chronic toxicity (exposures of at least 30 days) the genus mean value was 98.9 µg/L with a range of 73.9-132.2 µg/L. Sigmoid slopes were calculated when dose-response data were available (Table A3). The resulting geometric means (with ranges) of the slopes were 5.2 (4.1-7.6) for the 96-hr exposures and 4.2 (3.1-5.4) for the longer term mortality studies.

Growth studies on fry over 0.2 grams and under 6 grams produced EC50 values ranging from 20.33 µg/L to 112.43 µg/L (all values hardness adjusted, see Table A4 below). Exposures lasted from 15 to 98 days. NOEC values ranged from 5.83 to 113.82µg/L. Mortality was often observed in these studies and ranged from none reported to well over 50% at similar concentrations to those that produced growth effects (Table A4). For example, Besser *et al.* (2005) reported the lowest growth EC50 of 20.33µg/L for 0.2g fry after a 30 day exposure, but also reported a 30 day LC50 of 16.83µg/L with a slope of 5.4 (Table A4). Therefore, similar to the results with cadmium, an analysis of the available literature found that for exposures occurring to subyearling salmonids between 1 and 6g, growth effects often were confounded by mortality since most of the growth studies reported mortality assessment values (LC50s, chronic values, NOECs) that overlapped with or were less than the growth assessment values (EC50s, NOECs; Table A4). Hansen *et al.* 2002c used the IC20 as an endpoint for comparison since concentrations producing over 20% growth inhibition were often accompanied by significant mortality. Many other growth studies found in the literature search were excluded for reasons such as using too few exposure concentrations, using exposures beginning before swim-up (usually just after fertilization), or reporting no effect on growth for the concentrations tested. As mentioned above, in the remaining studies concentrations that produced effects on growth often also showed significant decreases in survival. For example, Mudge *et al.* (1993) reported that, for 3 of their 5 tests in coho, mortality was more sensitive than growth (Table A4). Nonetheless, some limited scenarios were run in the somatic growth model that looked at whether growth alone would be impacted by exposures at the chronic criteria value for copper. The time-to-effect and time-to-recovery values used for copper were both 0.5 days.

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Results

Sensitivity Analysis

The sensitivity analysis of all four of the control population matrices predicted the greatest changes in population growth rate (λ) result from changes in first-year survival. Parameter values and their corresponding sensitivity values are listed in Table A1. The elasticity values for the transition matrices also corresponded to the driving influence of first-year survival, with contributions to lambda of 0.33 for coho, 0.29 for ocean-type Chinook, 0.25 for stream-type Chinook and 0.24 for sockeye.

Model Output

Ammonia: Using the genus geometric mean LC_{50} and dose-response slope, with 100% of the population exposed to the criteria concentrations, the direct mortality model output showed 0% mortality to subyearlings and a zero percent change in the population growth rate (lambda) for all four life-history models (Table 2.6.5.47). The lowest species mean value in the *Oncorhynchus* range was also tested at 15.1 mg NH_3-N/L, and resulted in 0% mortality and 0% change in λ. When the chronic criterion was assessed with a 29-d exposure, the direct mortality model predicted no mortality or change in λ.

Studies on chronic exposures of juvenile salmonids to ammonia reported no or very little impacts on somatic growth, but these were accompanied by mortality. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model could not be run for ammonia.

Cadmium: Direct mortality population model runs were conducted using exposures to the criteria concentrations and the genus mean value calculated for *Oncorhynchus* (Table A5). This value produced 1% mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC50 and slope values as opposed to the minimum end of the range for species mean values (Table A5). Only when the minimum species mean value and the minimum slope were used, did mortality rise to a level that produced changes in lambda that were greater than the standard deviation of the control models (Table A5). Changes in population growth rates for the stream-type Chinook and coho were larger than one standard deviation from the control models. An estimated 28-day exposure to the chronic criterion produced no mortality or change in lambda.

Studies on chronic cadmium toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. For these reasons, appropriate input data were not identified and the somatic growth model was not run for cadmium.

Copper: Direct mortality population model runs were conducted using exposures to the criteria concentrations and both the acute and chronic parameters calculated for *Oncorhynchus* (Table A5). The acute LC50 and slope produced 0% mortality and no changes in the population growth rate for any of the four life history population models. The chronic LC50 and slope produced 0% mortality and no changes in the population growth rate for any of the four life history population models. Further model runs were conducted to examine the differences due to use of the genus geometric means for the LC50 and slope values as opposed to the minimum end of the range for species mean values but no mortality was projected (Table A5).

Studies on copper toxicity to juvenile salmonids did not show consistent impacts on somatic growth that could be separated from the associated mortality observed at the same exposure concentrations. The somatic growth model does not incorporate direct mortality and would greatly underestimate population-level effects. In spite of this, some growth model scenarios were run. When the maximum exposure period was used for the chronic criteria value in the growth model (140, 164 or 184 days depending on the life history), with an EC50 of 20.33, slope of 2.7 (Besser 2005) and the chronic criteria value of 9 µg/L, the percent change in lambda ranged from -1 to -4% (depending on life history). None of these reductions exceeded the control standard deviations. A 30-day exposure produced no decline in population growth rates. When a 30 day exposure for direct mortality was modeled using the minimum species values with a LC50 of 73.9µg/L and a slope of 4.2, the chronic criteria (9 µg /L) produced no change in lambda for the four life history models.

Summary

The only scenarios producing direct mortality sufficient to decrease the population growth rates were those using the lowest species mean values for cadmium. The other scenarios assessing the direct mortality from exposure to the suggested criteria values did not result in any changes in the population productivity.

Somatic growth during the freshwater subyearling stage of salmon has been shown to directly influence first year survival, so it was the focus of a literature review and modeling exercise to examine population-level impacts that may result from chemical exposures. In studies assessing growth endpoints of subyearling salmonids greater than 1g exposed to ammonia, cadmium or copper, mortality often confounded any growth effects identified since most studies that reported significant impacts on growth also reported significant simultaneous mortality. The somatic growth models do not include other stressors, such as direct mortality and could underestimate impacts for compounds which have overlapping dose response curves for mortality and somatic growth. In addition, the direct mortality population model inherently requires fewer assumptions regarding exposure and physiology than does the somatic growth population model. Overall, for the two impacts modeled here (direct mortality and somatic growth) we feel it is more appropriate when assessing potential risk to populations from exposures to these compounds during the free-swimming to rearing period of Juvenile salmonids to focus on the direct mortality population model output.

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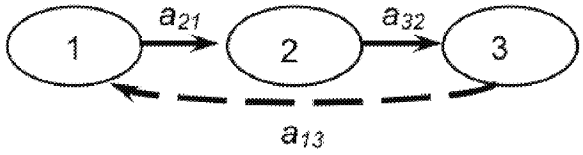
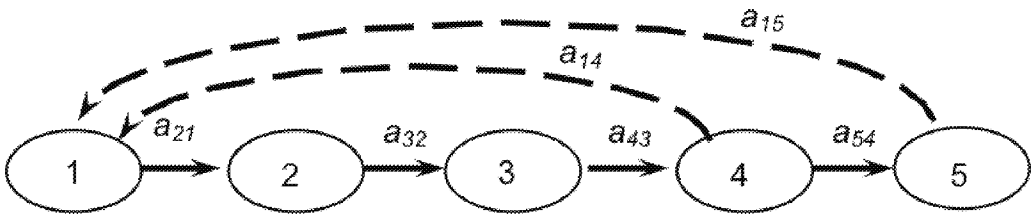
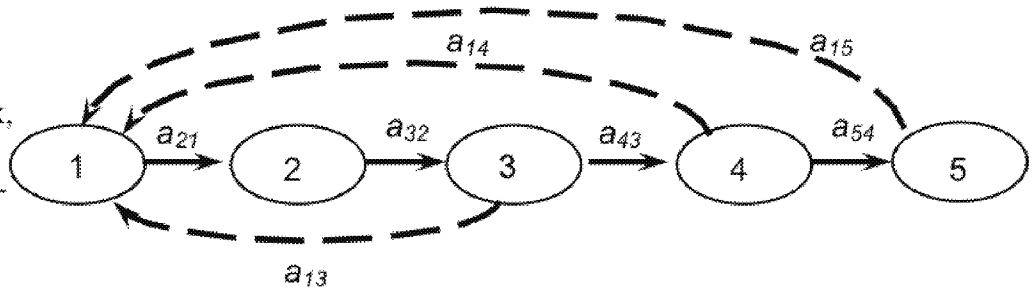
A. Coho



B. Sockeye



C. Chinook, ocean-type and stream-type



D. Transition Matrix

$$A=\begin{bmatrix} 0 & 0 & R3=a_{13} & R4=a_{14} & R5=a_{15} \\ S1=a_{21} & 0 & 0 & 0 & 0 \\ 0 & S2=a_{32} & 0 & 0 & 0 \\ 0 & 0 & S3=a_{43} & 0 & 0 \\ 0 & 0 & 0 & S4=a_{54} & 0 \end{bmatrix}$$

Figure A1. Life-History Graphs and Transition Matrix for coho (A), sockeye (B) and Chinook (C) salmon. The life-history graph for a population labeled by age, with each transition element labeled according to the matrix position, a_{ij}, i row and j column. Dashed lines represent reproductive contribution and solid lines represent survival transitions. D) The transition matrix for the life-history graph depicted in C.

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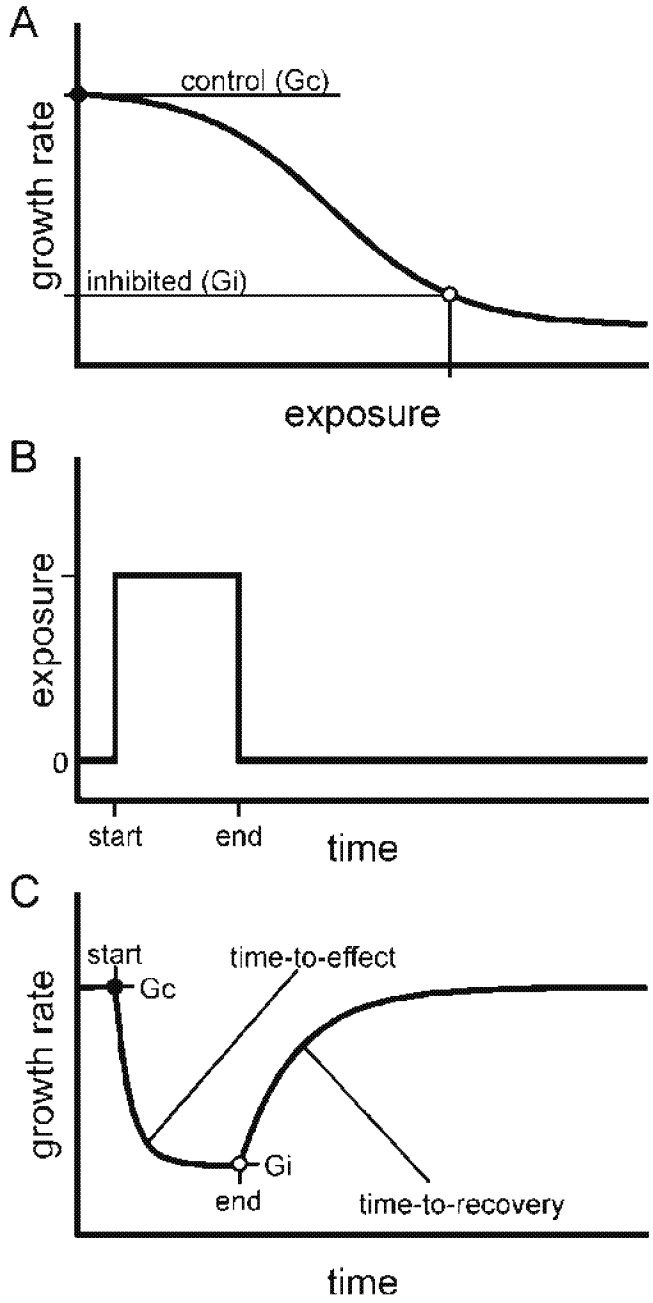

Figure A2.

ED_002991_00017299-00774

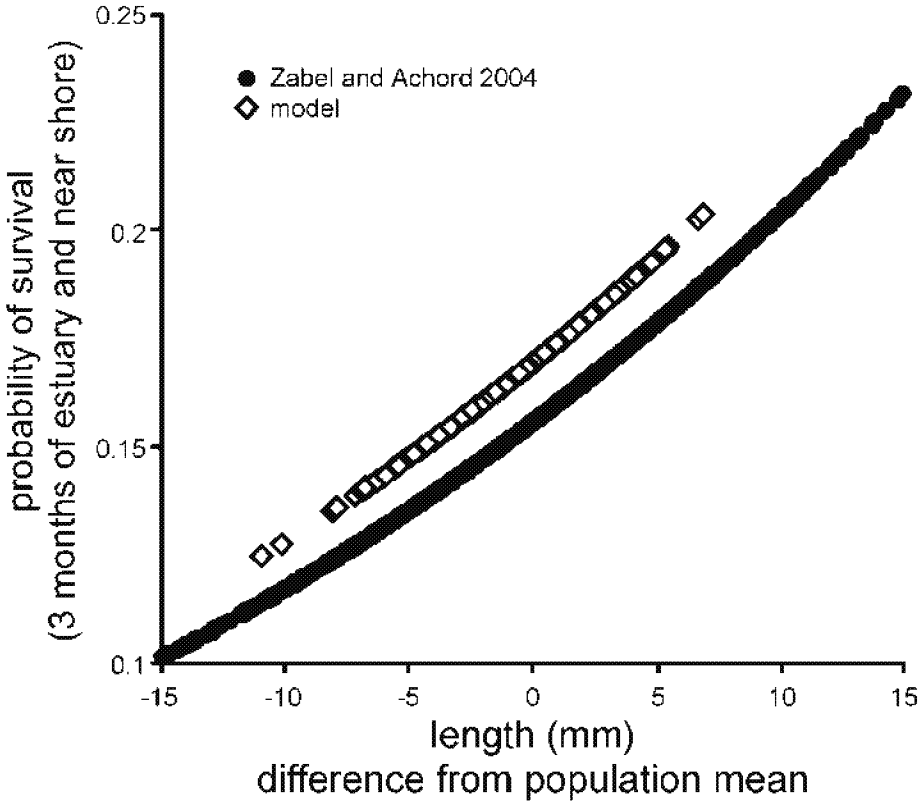

Figure A3. Relationships between difference in length from population mean and probability of survival for three-month period. Values shown are output based upon the original size and survival equations derived by Zabel and Achord (2004) and equations adapted for the model population used in the ocean-type Chinook model. Figure from Baldwin *et al.*, 2009.

ED_002991_00017299-00775

Table A1. Matrix transition element and sensitivity (S) and elasticity (E) values for each model species. These control values are listed by the transition element taken from the life-history graphs as depicted in Figure A1 and the literature data described in the method text. Blank cells indicate elements that are not in the transition matrix for a particular species. The influence of each matrix element on λ was assessed by calculating the sensitivity (S) and elasticity (E) values for A. The sensitivity of matrix element a_{ij} equals the rate of change in λ with respect to the transition element, defined by $\delta\lambda/\delta a$. The elasticity of transition element a_{ij} is defined as the proportional change in λ relative to the proportional change in a_{ij}, and equals (a_{ij}/λ) times the sensitivity of a_{ij}. Elasticity values allow comparison of the influence of individual transition elements and comparison across matrices.

Transition Element	Chinook Stream-type			Chinook Ocean-type			Coho			Sockeye		
	Value[1]	S	E	Value[2]	S	E	Value[3]	S	E	Value[4]	S	E
S1	0.0643	3.844	0.247	0.0056	57.13	0.292	0.0296	11.59	0.333	0.0257	9.441	0.239
S2	0.1160	2.132	0.247	0.48	0.670	0.292	0.0505	6.809	0.333	0.183	1.326	0.239
S3	0.17005	1.448	0.246	0.246	0.476	0.106				0.499	0.486	0.239
S4	0.04	0.319	0.0127	0.136	0.136	0.0168				0.1377	0.322	0.0437
R3	0.5807	0.00184	0.0011	313.8	0.0006	0.186	732.8	0.000469	0.333			
R4	746.73	0.000313	0.233	677.1	0.000146	0.0896				379.57	0.000537	0.195
R5	1020.36	1.25E-05	0.0127	1028	1.80E-05	0.0168				608.7	7.28E-05	0.0437

[1] Value calculated from data in Healy and Heard 1984, Fast *et al.* 1988, Beckman *et al.* 2000, Knudsen *et al.* 2006
[2] Value calculated from data in Healey and Heard 1984, Howell *et al.* 1985, Roni and Quinn 1995, Ratner *et al.* 1997, PSCCTC 2002, Green and Beechie 2004, Johnson *et al.* 2007
[3] Value calculated from data in Pess *et al.* 2002, Knudsen *et al.* 2002
[4] Value calculated from data in Pauley *et al.* 1989, Gustafson *et al.* 1997, McGurk 2000

Table A2. Species specific control parameters to model organismal growth and survival rates. Growth period and survival rate are determined from the literature data listed for each species. Gc and α were calculated to make the basic model produce the appropriate size and survival values from the literature.

	Chinook Stream-type[1]	Chinook Ocean-type[2]	Coho[3]	Sockeye[4]
days to run organismal growth model	184	140	184	168
growth rate % body wt/day (Gc)	1.28	1.30	0.90	1.183
α from equation S	-0.33	-1.99	-0.802	-0.871
Control Survival ϕ	0.418	0.169	0.310	0.295

[1] Values from data in Healy and Heard 1984, Fast *et al.* 1988, Beckman *et al.* 2000, Knudsen *et al.* 2006
[2] Values from data in Healey and Heard 1984, Howell *et al.* 1985, Roni and Quinn 1995, Ratner *et al.* 1997, PSCCTC 2002, Green and Beechie 2004, Johnson *et al.* 2007
[3] Values from data in Pess *et al.* 2002, Knudsen *et al.* 2002
[4] Values from data in Pauley *et al.* 1989, Gustafson *et al.* 1997, McGurk 2000

ED_002991_00017299-00777

Table A3. Acute and chronic exposure studies providing LC_{50} data used in the direct population mortality model. When multiple experiments were summarized in one paper, the geometric mean is reported here (*). All values were incorporated individually in calculating the species and genus geometric means.

		Exposure Information			LC50			Slope		Geometric Mean	
Species	**Age**	**Days**	**pH**	**Temp (C)**	**reported**	**pH adj**			**Reference**	**LC50**	**slope**
Acute and Chronic Exposure Ammonia											
Chinook	fingerling	4	7.8	7	29.3	26.8			Servizi and Gordon 1990	26.8	
coho	fingerling	4	8.1	17.2	12.1	15.1			Buckley 1978	15.1	
rainbow trout	fingerling	4	7.4	14.5	70.1	18.0			Calamari *et al.* 1981		
rainbow trout	fry	4	7.86*	12.9*	35.8*	26.7			Thurston and Russo 1983 (8 tests,1-4g fry)		
rainbow trout	fry	4	7.95	10	36.6	32.7		6.4	Broderius and Smith 1979	26.2	6.40
cutthroat trout	fry	4	7.7	10	29.1	27.0			Thurston *et al.* 1981		
cutthroat trout		fry	4	7.8*	12.6*	47.7*	30.1		Thurston *et al.* 1978 (4 tests)		29.4
Genus mean - acute										**23.6**	**6.40**
cutthroat trout		fry	29	7.8*	12.6*	33.6*	21.3		Thurston *et al.* 1978 (4 tests)		
Genus mean - chronic											**21.3**
Acute and Chronic Exposure Cadmium			**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Chinook	swimup	4	24	total	1.8	5.94	5.61		Chapman 1978		
Chinook	fingerling	4	25	total	1.41	4.50	4.25		Chapman 1978		
Chinook	fingerling	4	21	total	1.1	4.06	3.83		Finlayson and Verrue 1982		
Chinook	parr	4	24	total	3.5	11.55	10.91		Chapman 1978	5.62	6.90
coho	fry	4	22	total	3.66	12.99	12.27		Chapman 1975		
coho	fry	4	22	total	2.76	9.80	9.25		Chapman 1975		
coho	fry	4	22	total	1.73	6.14	5.80		Chapman 1975		
coho	fry	4	22	total	1.4	4.97	4.69		Chapman 1975		

coho	fry	4	22	total	2.7	9.59	9.05		Chapman 1975		
rainbow trout	swimup	4	23	total	1.3	4.45	4.20		Chapman 1978	7.75	
rainbow trout	swimup	4	7.5	dissolved	0.48	4.19	3.96		Windward 2002		
rainbow trout	swimup	4	14	dissolved	0.97	5.03	5.03		Windward 2002		
		Exposure Information			**LC50**			**Slope**		**Geometric Mean**	
Species	**Age**	**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Acute and Chronic Exposure Cadmium – cont.											
rainbow trout	swimup	4	21	dissolved	0.84	3.10	3.10		Windward 2002		
rainbow trout	swimup	4	24	dissolved	1.3	4.29	4.29		Windward 2002		
rainbow trout	swimup	4	26	dissolved	1.58	4.88	4.88		Windward 2002		
rainbow trout	swimup	4	26	dissolved	1.61	4.97	4.97		Windward 2002		
rainbow trout	swimup	4	29	dissolved	0.83	2.34	2.34		Windward 2002		
rainbow trout	swimup	4	30	dissolved	0.99	2.71	2.71		Windward 2002		
rainbow trout	swimup	4	32	dissolved	0.89	2.31	2.31		Windward 2002		
rainbow trout	fry	4	103	total	3.7	3.61	3.61		Besser et al 2007		6.57
rainbow trout	fry	4	103	total	5.2	5.07	5.07		Besser et al 2007		7.78
rainbow trout	fry	4	103	total	5.4	5.27	5.27		Besser et al 2007		
rainbow trout	fry	4	400	total	5.92	1.86	1.75		Davies et al 1993		
rainbow trout	fry	4	200	total	6.57	3.68	3.47		Davies et al 1993		
rainbow trout	fry	4	50	total	3.08	5.50	5.19		Davies et al 1993		4.70
rainbow trout	fry	4	140	total	22	16.60	15.67		Hollis et al 1999		
rainbow trout	fry	4	9.2	total	0.5	3.68	3.48		Cusimano et al 1986		
rainbow trout	fry	4	28	total	0.47	1.36	1.29		Hansen et al 2002a		
rainbow trout	fry	4	30	total	0.51	1.40	1.32		Hansen et al 2002a		
rainbow trout	fingerling	4	44	total	3	5.96	5.63		Phipps and Holcombe 1985		
rainbow trout	parr	4	23	total	1	3.42	3.23		Chapman 1978	3.63	
West Slope cutthroat trout	fry	4	21	dissolved	0.35	1.29	1.29		EVS 1996		
West Slope cutthroat trout	fry	4	21	dissolved	0.93	3.43	3.43		Windward 2002		
West Slope cutthroat trout	fry	4	32	dissolved	1.41	3.66	3.66		Windward 2002		

West Slope cutthroat trout	fry	4	31	dissolved	1.18	3.14	3.14		Windward 2002	2.67	
Genus mean -acute										**4.53**	**6.38**
rainbow trout	swimup	28	103	dissolved	5.50	5.36	5.36		Besser et al 2007	5.36	
Genus mean - chronic										**5.36**	
		Exposure Information			**LC50**			**Slope**		**Geometric Mean**	
Species	**Age**	**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Acute Exposure Copper											
Chinook	alevin	4	23	dissolved	26	103.84	103.84		Chapman 1978		
Chinook	fry	4	23	dissolved	19	75.88	75.88		Chapman 1978		
Chinook	fry	4	21	total	32	139.24	111.39	4.2	Finlayson 1982		
Chinook	fry	4	35	dissolved	12.5	33.61	33.61	2.7	Welsh 2000		
Chinook	fry	4	38	dissolved	14.3	35.58	35.58	4.2	Welsh 2000		
Chinook	fry	4	36	dissolved	18.3	47.92	47.92	3.8	Welsh 2000		
Chinook	fry	4	36	dissolved	7.4	19.38	19.38	9	Welsh 2000		
Chinook	fry	4	25	dissolved	33.1	122.20	122.20		Chapman 1982		
Chinook	fry	4	211	dissolved	54	26.72	26.72		Hamilton 1990		
Chinook	fry	4	211	dissolved	58	28.70	28.70		Hamilton 1990		
Chinook	juvenile	4	100	dissolved	50	50.00	50.00		Chapman 1977		
Chinook	juvenile	4		total	180			4.6	Holland 1960		
Chinook	parr	4	23	dissolved	38	151.76	151.76		Chapman 1978		
Chinook	smolt	4	23	dissolved	26	103.84	103.84		Chapman 1978	57.31	4.42
coho	alevin	1	41	dissolved	67	155.21	155.21		Buhl 1990		
coho	alevin	4	41		20	46.33	46.33		Buhl 1990		
coho	fry	4	31	total	44	132.65	106.12		Mudge 1993		
coho	juvenile	1	41	dissolved	23.4	54.21	54.21		Buhl 1990		
coho	juvenile	1	41	dissolved	42.2	97.76	97.76		Buhl 1990		
coho	juvenile	1	41	dissolved	62.3	144.32	144.32		Buhl 1990		
coho	juvenile	4	33	dissolved	17	48.32	48.32		Buckley 1983		
coho	juvenile	4	41	dissolved	15.1	34.98	34.98		Buhl 1990		
coho	juvenile	4	41	dissolved	23.9	55.36	55.36		Buhl 1990		
coho	juvenile	4	41	dissolved	31.9	73.90	73.90		Buhl 1990		
coho	juvenile	4	128	total	60	47.55	38.04		Hedtke 1982		

coho	juvenile	4	128	total	81	64.19	51.35		Hedtke 1982		
coho	juvenile	4	128	total	150	118.87	95.10		Hedtke 1982		
coho	juvenile	4	128	total	166	131.55	105.24		Hedtke 1982		
coho	juvenile	4	128	total	212	168.01	134.40		Hedtke 1982		
coho	juvenile	4	128	total	192	152.16	121.72		Hedtke 1982		
		Exposure Information			**LC50**			**Slope**		**Geometric Mean**	
Species	**Age**	**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Acute Exposure Copper – Cont.											
coho	juvenile	4	95	total	60	62.97	50.38		Lorz 1976		
coho	juvenile	4	95	total	72	75.57	60.45		Lorz 1976		
coho	juvenile	4	94	total	61	64.66	51.73	5.3	Lorz 1977		
coho	juvenile	4	94	total	71	75.26	60.21	9.6	Lorz 1977		
coho	juvenile	4	94	total	73	77.38	61.91	9.7	Lorz 1977		
coho	juvenile	4	94	total	55	58.30	46.64	6.7	Lorz 1977		
coho	parr	4	31	total	67	201.98	161.59		Mudge 1993		
coho	smolt	4	31	total	44	132.65	106.12		Mudge 1993	73.44	7.58
pink	alevin	4	83	total	143	170.44	136.35		Servizi 1978		
pink	alevin	4	83	total	83	98.93	79.14		Servizi 1978		
pink	fry	4	83	total	199	237.19	189.75		Servizi 1978	126.99	
sockeye	alevin	4	83	total	190	226.46	181.17		Servizi 1978		
sockeye	alevin	4	83	total	120	143.03	114.42		Servizi 1978		
sockeye	fry	4	83	total	150	178.79	143.03		Servizi 1978		
sockeye	parr	4	41	total	240	555.96	444.77		Davis 1978		
sockeye	smolt	4	83	total	200	238.38	190.71		Servizi 1978	190.59	
rainbow trout	alevin	1	41	dissolved	46.4	107.49	107.49		Buhl 1990		
rainbow trout	alevin	4	41	dissolved	36	83.39	83.39		Buhl 1990		
rainbow trout	fry	4	103	dissolved	48	46.68	46.68	4.8	Besser 2007		
rainbow trout	fry	4	90	dissolved	17.2	19.00	19.00	4.4	Welsh 2000		
rainbow trout	fry	4	42	dissolved	3.4	7.70	7.70	3	Welsh 2000		
rainbow trout	fry	4	90	dissolved	32	35.34	35.34	6.7	Welsh 2000		
rainbow trout	fry	4	39	dissolved	8.1	19.67	19.67	2.8	Welsh 2000		
rainbow trout	juvenile	1	41	dissolved	18.9	43.78	43.78		Buhl 1990		

rainbow trout	juvenile	4	100	dissolved	22	22.00	22.00		Gish 1971		
rainbow trout	juvenile	4	100	dissolved	30	30.00	30.00		Taylor 2000		
steelhead	alevin	4	23	dissolved	28	111.82	111.82		Chapman 1978		
steelhead	fry	4	23	dissolved	17	67.89	67.89		Chapman 1978		
steelhead	juvenile	4	22	dissolved	20	83.29	83.29		Chapman 1973		
		Exposure Information			**LC50**			**Slope**		**Geometric Mean**	
Species	**Age**	**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
Acute Exposure Copper – Cont.											
steelhead	parr	4	23	dissolved	18	71.89	71.89		Chapman 1978		
steelhead	parr	4	31	total	57	171.84	137.47		Mudge 1993		
steelhead	smolt	4	23	dissolved	29	115.82	115.82		Chapman 1978	48.34	4.12
Genus mean - acute										**86.79**	**5.17**
Chronic exposure Copper		**Days**	**Hardness**	**Measurement**	**reported**	**Hardness adj**	**Dissolved adj**		**Reference**	**LC50**	**slope**
coho	fry	120	31	total	60	163.22	130.58		Mudge 1993		
coho	fry	120	31	total	80	217.63	174.11		Mudge 1993		
coho	fry	120	31	total	39	106.10	84.88		Mudge 1993		
coho	parr	120	31	total	69	187.71	150.17		Mudge 1993		
coho	parr	120	31	total	52	141.46	113.17		Mudge 1993		
coho	parr	120	31	total	70	190.43	152.34		Mudge 1993		
coho	parr	120	31	total	65	176.83	141.46		Mudge 1993	132.23	
rainbow trout	fry	30	170	total	33.1	21.03	16.83	5.4	Besser 2005		
rainbow trout	fry	56	100	dissolved	55.1	55.10	55.10	4.7	Hansen 2002c		
rainbow trout	fry	28	103	dissolved	56	54.60	54.60	3.1	Besser 2007		
steelhead	parr	120	31	total	84	228.51	182.81		Mudge 1993		
steelhead	parr	120	31	total	70	190.43	152.34		Mudge 1993		
steelhead	parr	120	31	total	53	144.18	115.34		Mudge 1993	73.88	4.29
Genus mean - chronic										**98.84**	**4.29**

Table A4. Copper studies identified that investigated the impacts of copper exposure on juvenile growth.

		Exposure Information									
Species	**Age (size)**	**Days**	**Hardness**	**Measurement**	**Uncorrected Value µg/L**	**hardness adj**	**dissolved adj**	**Notes**	**slope**	**Reference**	**Mortality reported with correction**
rt	fry (swim-up)	30	170	total	40	25.42	**20.33**	EC50, size not specified, fed ad libitum	2.7	Besser 2005	16.78 µg/l LC50, 5.4 slope
rt	fry (0.2 g)	28	103	dissolved	59	57.53	57.53	28% dec in biomass		Besser 2007	50% at 57.53 µg/l
coho	juv (6 g)	98	280	dissolved	271	112.43	**112.43**	EC50	1.28	Buckley 1982	
rt	parr (1.7 - 3.3 g)	21	374	total	194	62.85	50.28	~50% dec in growth, ration based on init biomass		Dixon 1981	
rt	fry (0.2 g)	56	105	dissolved	54	51.79	**51.79**	EC50, fed fixed ration (3.5%)	1.4	Hansen 2002c	52.75 µg/l LC50, 4.7 slope
rt	juv (20 g)	28	120	total	52	44.50	35.60	56% dec in growth, fixed ration		Kamunde 2005	26% at 35.60 µg/l
rt	fry (0.12 g)	60	25	total	13	42.50	**34.00**	EC50, fed fixed ration (4.5%)	1.5	Marr 1996	
rt	juv (3.2 g)	35	140	total	75	56.26	45.01	no effect reported, only conc tested		McGeer 2000	
coho	fry (na)	60	26	total	21	66.39	53.11	NOEC		Mudge 1993	45.53 µg/l NOEC
steelhead	parr (na)	60	26	total	45	142.27	113.82	NOEC		Mudge 1993	60.70 µg/l NOEC
rt	fry (0.1 g)	15	135	total	5	3.87	**3.10**	EC50, fed excess of satiation	1.8	Neville 1995	3.40 µg/l LC50, 2.6 slope
rt	juv (18-20 g)	28	120	total	52	44.50	**35.60**	49% dec in wt, only conc tested, consumption meas		Nyogi 2006	
rt	juv (1-2 g)	30	120	total	62	53.06	42.44	NOEC (highest tested), fed fixed ration		Taylor 2000	
rt	juv (1-2 g)	30	20	total	1.7	6.73	5.38	NOEC (highest tested), fed fixed ration		Taylor 2000	
rt	juv (5-6 g)	20	100	total	77	77.00	**61.60**	EC50 from eq 1 (@pH 7.5, 5.5 g)		Waiwood 1978	

rt = rainbow trout

Table A5. Direct mortality population model scenarios for ammonia, cadmium and copper criteria. Standard scenarios used the genus mean values for the criteria. Since no effect resulted, the minimum species mean values were assessed. * indicates a percent change in lambda of greater than one standard deviation from the baseline population model (Chinook ocean-type 9, Chinook stream-type 3, Sockeye 6, Coho 5).

		Mortality input parameters			Output	% change in lambda			
Chemical	Test length	LC_{50}	Sigmoid slope	Criteria Conc.	Percent mortality	Chinook ocean-type	Chinook stream-type	Sockeye	Coho
		(mg/L)							
Ammonia	96-hr	23.6[1]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	96-hr	15.1[2]	6.4[1]	5.6	0	0(13)	0(4)	0(8)	0(7)
Ammonia	29-d	21.3	6.4[3]	1.7	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Cadmium	96-hr	4.53[1]	6.4[1]	2.0	1	0(13)	0(4)	0(8)	0(7)
Cadmium	96-hr	4.53[1]	4.7[2]	2.0	2	-1(13)	-1(4)	-1(8)	-1(7)
Cadmium	96-hr	2.67[2]	6.4[1]	2.0	14	-4(12)	-3(4)	-3(8)	-5(7)
Cadmium	96-hr	2.67[2]	4.7[2]	2.0	20	-7(12)	**-5***(4)	-5(8)	-7(7)
Cadmium	28-d	5.36[1]	6.4[3]	0.25	0	0(13)	0(4)	0(8)	0(7)
		(ug/L)							
Copper	96-hr	86.8[1]	5.2[1]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	96-hr	48.3[2]	4.1[2]	13.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	98.9[1]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)
Copper	30+d	73.9[2]	4.2[1]	9.0	0	0(13)	0(4)	0(8)	0(7)

[1]Genus Geometric Mean for *Oncorhynchus* values
[2]Minimum Species Mean value from the range of *Oncorhynchus* values
[3]Slope for chronic exposures not identified, used Genus Mean slope from 96-hr exposures

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