UNITED STATES DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
NATIONAL MARINE FISHERIES SERVICE
West Coast Region
7600 Sand Point Way N.E.
Seattle, Washington 98115

May 7, 2014 **Refer to NMFS No: 2000-1484**

Dan Opalski, Director
Office of Water and Watersheds
U.S. Environmental Protection Agency
1200 Sixth Avenue
Seattle, Washington 98101

Re: Final Endangered Species Act Section 7 Formal Consultation and Magnuson-Stevens Fishery Conservation and Management Act Essential Fish Habitat Consultation for Water Quality Toxics Standards for Idaho

Dear Mr. Opalski:

The enclosed document contains a biological opinion (Opinion) and letters of concurrence prepared by the National Marine Fisheries Service (NMFS) pursuant to section 7(a)(2) of the Endangered Species Act (ESA) on the effects of approving the Idaho Water Quality Standards for toxic substances. In this Opinion, NMFS concludes that the action, as proposed, is likely to jeopardize the continued existence of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon, and Snake River Basin steelhead and result in the destruction or adverse modification of designated critical habitat for Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon, and Snake River Basin steelhead.

As required under the ESA for consultations concluding with jeopardy and adverse modification determinations, NMFS discussed with U.S. Environmental Protection Agency (EPA), the availability of reasonable and prudent alternatives that the EPA can take to avoid violation of the EPA's ESA section 7(a)(2) responsibilities (50 CFR 402.14(g)(5)). Reasonable and prudent alternatives refer to alternative actions identified during formal consultation: (1) That can be implemented in a manner consistent with the intended purpose of the action; (2) that can be implemented consistent with the scope of the Federal agency's legal authority and jurisdiction; (3) that 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). The Opinion includes a reasonable and prudent alternative which NMFS believes can be implemented to avoid jeopardy and adverse modification of critical habitat, while meeting each of the other requirements listed above. Accordingly, NMFS prepared an incidental take statement describing and exempting the extent of incidental take reasonably certain to occur under the reasonable and prudent alternative.

This document also includes the results of our analysis of the action's likely effects on essential fish habitat (EFH) pursuant to section 305(b) of the Magnuson-Stevens Fishery Conservation and

Management Act, and includes three Conservation Recommendations to avoid, minimize, or otherwise offset potential adverse effects on EFH. These Conservation Recommendations are a non-identical set of the ESA terms and conditions.

If you have questions regarding this consultation, please contact David Mabe, Snake Basin Office, (208) 378-5698.

Sincerely,

William W. Stelle, Jr.
Regional Administrator

Enclosure

cc: D. Miller – IOSC
R. Holder – USFWS
J. Martin – DOJ
M. Lopez – NPT
C. Colter – SBT

bcc: SBAO - File copy, Read File,
GCNW – L. Beale

Mebane:Final:2014 May 7 EPA WQS Toxics.docx:5/7/14:NMFS No: 2000-1484

cc Addresses:

Dustin Miller, Administrator
Idaho Office of Species Conservation
304 N. 8th Street, Room 149
Boise, Idaho 83702

Russ Holder
U.S. Fish and Wildlife Service
Snake River Basin Office
1387 S. Vinnell Way Room 368
Boise, Idaho 83709

John Martin
Trial Attorney, Wildlife & Marine Resources Section
US Department of Justice
Environment and Natural Resources Division
1961 Stout Street, 8th Floor, Room 812
Denver, Colorado 80294

Mike Lopez, Staff Attorney
Nez Perce Tribe
Office of Legal Counsel
P.O. Box 365
Lapwai, Idaho 83540

Chad Colter
Shoshone-Bannock Tribes
Fort Hall Indian Reservation
Fisheries Department
P.O. Box 306
Fort Hall, Idaho 83203

Endangered Species Act Section 7(a)(2) Biological Opinion and Magnuson-Stevens Fishery Conservation and Management Act Essential Fish Habitat (EFH) Consultation

Idaho Water Quality Standards for Toxic Substances

NMFS Consultation Number: 2000-1484

Action Agency: United States Environmental Protection Agency

Affected Species and Determinations:

ESA-Listed Species	Status	Are Actions Likely to Adversely Affect Species or Critical Habitat?	Are Actions Likely To Jeopardize the Species?	Are Actions Likely To Destroy or Adversely Modify Critical Habitat?
Snake River Basin steelhead	Threatened	Yes	Yes	Yes
Snake River Spring/Summer Chinook Salmon	Threatened	Yes	Yes	Yes
Snake River Fall Run Chinook Salmon	Threatened	Yes	Yes	Yes
Snake River Sockeye Salmon	Endangered	Yes	Yes	Yes

Fishery Management Plan That Describes EFH in the Project Area	Does Action Have an Adverse Effect on EFH?	Are EFH Conservation Recommendations Provided?
Pacific Coast Salmon	Yes	Yes

Consultation Conducted By: National Marine Fisheries Service, West Coast Region

Issued By:

William W. Stelle, Jr.
Regional Administrator

Date: May 7, 2014

Table of Contents

1. INTRODUCTION 1
1.1. Background 1
1.2. Consultation History 1
1.3. Proposed Action 3
1.3.1. Idaho's Water Quality Standards for Toxic Pollutants 4
1.3.2. Application of the IWQS for Metals 8
1.4. Action Area 10
2. ENDANGERED SPECIES ACT: BIOLOGICAL OPINION AND INCIDENTAL TAKE STATEMENT 13
2.1. Introduction to the Biological Opinion 13
2.2. Rangewide Status of the Species and Critical Habitat 15
2.2.1. Status of the Species 15
2.2.1.1. Snake River Sockeye Salmon 16
2.2.1.2. Snake River Spring/Summer Chinook Salmon 19
2.2.1.3. Snake River Fall Chinook Salmon 27
2.2.1.4. Snake River Basin Steelhead 29
2.2.2. Status of Critical Habitat 32
2.2.3. Climate Change 36
2.3. Environmental Baseline 37
2.3.1. Basins in Action Area 38
2.3.1.1. Clearwater River Basin 38
2.3.1.2. Salmon River Basin 41
2.3.1.3. Snake River Basin 42
2.3.2. Baseline for Metals 43
2.3.2.1. Baseline for Arsenic in Action Area 44
2.3.2.2. Baseline for Chromium 48
2.3.2.3. Baseline for Copper 48
2.3.2.4. Baseline for Cyanide 55
2.3.2.5. Baseline for Lead 56
2.3.2.6. Baseline for Mercury 56
2.3.2.7. Baseline for Nickel 56
2.3.2.8. Baseline for Selenium 57
2.3.2.9. Baseline for Silver 57
2.3.2.10. Baseline for Zinc 58
2.3.2.11. Baseline for Organic Pollutants 59
2.4. Effects of the action on the species and its Designated Critical Habitat 60
2.4.1. Evaluation of issues that are common to multiple aquatic life criteria 61
2.4.1.1. The assumption that not harming more than 5% of the species tested in laboratories is sufficient protection of ESA-listed species and critical habitats 64
2.4.1.2. The assumption that effects in laboratory tests are reasonable predictors of effects in field situations 65
2.4.1.3. Susceptibility of Salmonids to Chemicals at Different Life Stages 70

2.4.1.4. Effects of Acclimation on Susceptibility to Chemicals 76
2.4.1.5. Implications of the use of the "chronic value" statistic in setting criteria 77
2.4.1.6. The assumption that dividing a concentration that killed 50% of a test population by two will result in a safe concentration 79
2.4.1.7. Issue of Using Flow Through, Renewal, or Static Exposure Test Designs 82
2.4.1.8. The "Water-Effect Ratio" Provision 83
2.4.1.9. Issue of Basing Criteria on Dissolved or Total-Recoverable Metals 85
2.4.1.10. Mixture Toxicity: criteria were developed as if exposures to chemicals occur one at a time, but chemicals always occur as mixtures in effluents and ambient waters 87
2.4.1.11. Frequency, Duration and Magnitude of Allowable Criteria Concentration Exposure Exceedences. 92
2.4.1.12. Special Consideration for Evaluating the Effects of the Action on Critical Habitat 98
2.4.2. The Effects of Expressing Metals Criteria as a function of Water Hardness 102
2.4.2.1. The Use of a "Hardness Floor" in Calculating Metals Limits. 104
2.4.2.2. Summary of Effects of the Hardness Floor for Calculating Metals Criteria 117
2.4.3. The Effects of EPA Approval of the Arsenic Criteria 117
2.4.3.1. Species Effects of Arsenic Criteria 118
2.4.3.2. Habitat Effects of Arsenic Criteria 123
2.4.3.3. Summary of Effects for Arsenic 124
2.4.4. The Effects of EPA Approval of the Copper Criteria 125
2.4.4.1. Species Effects of Copper Criteria 125
2.4.4.2. Habitat Effects of Copper Criteria 133
2.4.4.3. Summary for Copper 137
2.4.5 The Effects of EPA Approval of the Cyanide Criteria 138
2.4.5.1. Species Effects of Cyanide Criteria 142
2.4.5.2. Habitat Effects of Cyanide Criteria 144
2.4.5.3. Summary for Cyanide 144
2.4.6. The Effects of EPA Approval of the Mercury Criteria 144
2.4.6.1. Species Effects of Mercury Criteria 145
2.4.6.2. Habitat Effects of Mercury Criteria 147
2.4.6.3. Summary for Mercury 162
2.4.7. The Effects of EPA Approval of the Nickel Criteria 162
2.4.7.1. Species Effects of Nickel Criteria 162
2.4.7.2. Habitat Effects of Proposed Nickel Criteria 165
2.4.7.3. Summary for Nickel 166
2.4.8. The Effects of EPA Approval of the Selenium Criteria 166
2.4.8.1. Species Effects of Selenium Criteria 168
2.4.8.2. Habitat Effects of Selenium Criteria 169
2.4.8.3. Summary for Selenium 181
2.4.9. The Effects of EPA Approval of the Silver Criteria 181
2.4.9.1. Species Effects of Silver Criteria 181
2.4.9.2. Habitat Effects of Silver Criteria 185
2.4.9.3. Summary for Silver 186
2.4.10. The Effects of EPA Approval of the Zinc Criteria 186
2.4.10.1. Species Effects of Zinc Criteria 187

2.4.10.2. Habitat Effects of Zinc Criteria 192
2.4.10.3. Summary for Zinc 193
2.4.11. The Effects of EPA Approval of the Chromium III and VI Criteria 193
2.4.11.1. Species Effects of Chromium Criteria 194
2.4.11.2. Habitat Effects of Chromium Criteria 195
2.4.11.3. Summary for Chromium 196
2.4.12. The Effects of EPA Approval of the Lead Criteria 197
2.4.12.1. Species Effects of Lead Criteria 197
2.4.12.2. Habitat Effects of Lead Criteria 201
2.4.12.3. Summary for Lead 202
2.4.13. Organic Pollutants: General Issues 202
2.4.14. The Effects of EPA Approval of Pentachlorophenol (PCP) Criteria 206
2.4.14.1. Species Effects of Pentachlorophenol Criteria 206
2.4.14.2. Habitat Effects of Pentachlorophenol Criteria 209
2.4.14.3. Summary for PCP 209
2.4.15. The Effects of EPA Approval of the Aldrin/Dieldrin Criteria 210
2.4.15.1. Species Effects of Aldrin/Dieldrin Criteria 211
2.4.15.2. Habitat Effects of Aldrin/Dieldrin Criteria 215
2.4.15.3. Summary for Aldrin/Dieldrin 218
2.4.16. The Effects of EPA Approval of the Chlordane Criteria 220
2.4.16.1. Species Effects of Chlordane Criteria 220
2.4.16.2. Habitat Effects of Chlordane Criteria 223
2.4.16.3. Summary for Chlordane 225
2.4.17. The Effects of EPA Approval of the Dichlorodiphenyltrichloroethane Criteria 225
2.4.17.1. Species Effects of DDT Criteria 226
2.4.17.2. Habitat Effects of DDT Criteria 230
2.4.17.3. Summary for DDTs 232
2.4.18. The Effects of EPA Approval of the Endosulfan Criteria 233
2.4.18.1. Species Effects of Endosulfan Criteria 234
2.4.18.2. Habitat effects of Endosulfan Criteria 236
2.4.18.3 Summary for Endoculfan 238
2.4.19. The Effects of EPA Approval of the Endrin Criteria 239
2.4.19.1. Species Effects of Endrin Criteria 239
2.4.19.2. Habitat Effects of Endrin Criteria 241
2.4.19.3. Summary for Endrin 242
2.4.20. The Effects of EPA Approval of the Heptachlor Criteria 243
2.4.20.1. Species Effects of Heptachlor Criteria 244
2.4.20.2. Habitat Effects of Heptachlor Criteria 245
2.4.20.3. Summary for Helptchlor 247
2.4.21. The Effects of EPA Approval of Lindane (gamma-BHC) Criteria 247
2.4.21.1. Species Effects of Proposed Lindane Criteria 248
2.4.21.2. Habitat Effects of Proposed Lindane Criteria 250
2.4.21.3. Summary for Lindane 251
2.4.22. The Effects of EPA Approval of the Polychlorinated Biphenyl Criterion 251
2.4.22.1. Species Effects of PCB Criterion 252
2.4.22.2. Habitat Effects of PCB Criterion 254

2.4.22.3. Summary for PCBs 257
2.4.23. The Effects of EPA Approval of the Toxaphene Criteria 257
2.4.23.1. Species Effects of Toxaphene Criteria 258
2.4.23.2. Habitat Effects of Toxaphene Criteria 259
2.1.23.3. Summary for Toxaphene 260
2.5. Cumulative Effects 260
2.6. Integration and Synthesis 261
2.6.1. Integration and Synthesis Summary for Each Affected Species 273
2.7. Conclusion 274
2.8. Reasonable and Prudent Alternatives (RPAs) and Analysis of Effects of the RPAs 281
2.8.1. The RPA for the Hardness Floor 281
2.8.1.1. New Aquatic Life Criteria 281
2.8.2. The RPAs for Arsenic 281
2.8.2.1. Interim Protection for Listed Species 281
2.8.2.2. New Chronic Aquatic Life Criterion for Arsenic 282
2.8.3. The RPAs for Copper 282
2.8.3.1. Interim Protection for Listed Species 282
2.8.3.2. New Acute and Chronic Aquatic Life Criteria for Copper 283
2.8.4. The RPAs for Mercury 284
2.8.4.1. Interim Protection for Listed Species 284
2.8.4.2. New Chronic Aquatic Life Criteria for Mercury 284
2.8.5. The RPA for Cyanide 284
2.8.6. The RPAs for Selenium 284
2.8.6.1. Interim Protection for Listed Species 284
2.8.6.2. New Chronic Aquatic Life Criterion for Selenium 285
2.8.7. Notification of EPA Final Decision 285
2.8.8. Analysis of the RPAs 285
2.8.8.1. Analysis of the Reasonable and Prudent Alternative for the Hardness Floor 286
2.8.8.2. Analysis of the Reasonable and Prudent Alternative for Arsenic 286
2.8.8.3. Analysis of the Reasonable and Prudent Alternative for Copper 287
2.8.8.4. Analysis of the Reasonable and Prudent Alternative for Cyanide 287
2.8.8.5. Analysis of the Reasonable and Prudent Alternative for Mercury 288
2.8.8.6. Analysis of the Reasonable and Prudent Alternative for Selenium 288
2.9. Incidental Take Statement 288
2.9.1. Amount or Extent of Take 289
2.9.2. Effect of the Take 291
2.9.3. Reasonable and Prudent Measures and Terms and Conditions 291
2.9.3.1. Reasonable and Prudent Measures 291
2.9.3.2. Term and Conditions 292
2.10. Conservation Recommendations 294
2.10.1. Conservation Recommendation for Arsenic 295
2.10.2. Conservation Recommendations for Silver 295
2.10.3. Conservation Recommendation for Cyanide 295
2.10.4. Conservation Recommendation for use of bioassessment data in permitting decisions 295
2.11. Reinitiation of Consultation 296

2.12. Summary of Conclusions 296
3. MAGNUSON-STEVENS FISHERY CONSERVATION AND MANAGEMENT ACT ESSENTIAL FISH HABITAT CONSULTATIONS 303
3.1. Essential Fish Habitat Affected by the Project 303
3.2. Adverse Effects on Essential Fish Habitat 303
3.3. Essential Fish Habitat Conservation Recommendations 304
3.4. Statutory Response Requirement 304
3.5. Supplemental Consultation 305
4. DATA QUALITY ACT DOCUMENTATION AND PRE-DISSEMINATION REVIEW 305
4.1. Utility 305
4.2. Integrity 305
4.3. Objectivity 306
5. References 307

APPENDICES

Appendix A A-1
A Review of Water Hardness Data for Idaho — 1979-2004 ..... A-1
Appendix B B-1
How to measure insignificance? Comparisons between NOECs, EC1s, and EC0s and the lower confidence limit of EC10s to estimate “insignificant effects” B-1
Appendix C C-1
An evaluation of the accuracy and protectiveness of EPA’s 2007 biotic ligand model (BLM)-based copper criteria for copper C-1
Appendix D D-1
Conservative assumptions to be used in implementing criteria through effluent limits D-1
Appendix E E-1
Biomonitoring of Effects E-1
Appendix F F-1
Salmonid Zone of Passage Considerations F-1

TABLES

Table 1.3.1. Ambient Water Quality Criteria for toxic pollutants submitted for consultation in EPA's 2000 Biological Evaluation. Also shown are AWQC that have subsequently been revised by the State of Idaho (Idaho Department of Environmental Quality 2011). In two parts, inorganic and organic substances: 6
Table 1.4.1. Fourth field HUCs containing listed salmon or steelhead. 12
Table 2.2.1. 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. 15
Table 2.2.2. Adult returns passing Lower Granite Dam (LGD) and returning to the area of Redfish Lake (Sawtooth Basin, Idaho) (IDFG 2011; Fish Passage Center 2011a; NMFS 2008). 18
Table 2.2.3. Summary of VSP parameter risks and viability status for Snake River Spring/Summer Chinook Salmon MPGs and independent populations (Ford 2011; NMFS 2011). 26
Table 2.2.4. Summary of VSP parameter risks and viability status for Snake River Basin Steelhead MPGs and independent populations (Ford 2011; NMFS 2011). 32
Table 2.2.5. Types of sites and essential physical and biological features designated as PCEs, and the species life stage each PCE supports. 33
Table 2.2.6. Description of designated critical habitat for ESA-listed species considered in this Opinion. 34
Table 2.3.1. Baseline concentrations of organic pollutants in sediments and fish tissue measured in waters within the action area, or upstream waters that drain into the action area. 60
Table 2.4.1.1. Reasons why the effects of a chemical substance could be more- or less-severe on listed steelhead and salmon in laboratory or in wild settings 67
Table 2.4.1.2. Relative sensitivity of standard 7-day WET tests with *Ceriodaphnia* and fathead minnows to rainbow trout with copper under directly comparable test conditions (ASTM moderately-hard water, hardness 170 mg/L). 91
Table 2.4.1.3. Relative sensitivity of the standard WET *Ceriodaphnia dubia* 7-day test in relation to a surrogate salmonid for listed salmon and steelhead (rainbow trout except where noted), pooled from data compilations 91
Table 2.4.2.1. Ranges of low hardnesses observed in Salmon River basin receiving waters of industrial mine effluents or nonpoint source mine runoff (limited to major facilities discharging to waters either designated as critical habitats for listed salmonids or at least some portions are accessible and presumably used by listed salmonids. 115
Table 2.4.3.1. Relevant concentrations of arsenic in the diet of juvenile fish that were associated with adverse effects 121
Table 2.4.3.2. Relevant concentrations of arsenic in stream water, sediment, and in the tissues of aquatic invertebrates collected from the same streams. Selected undiluted mine effluent concentrations from within the action area are included for comparison. Unless otherwise noted, concentrations are averages, values in parentheses are ranges 122
Table 2.4.4.1. Relevant effects and risk ratios of copper to salmonids or other ecosystem components, emphasizing effects that occurred at lower concentrations than the relevant Idaho criteria. Long-term effects (> 4 days to occur) are compared to the chronic criterion, short-term sublethal effects to the Idaho acute criteria, or for acute LC_{50}s, the Idaho final acute value. Risk ratios greater than 1.0 are considered harmful. 131

Table 2.4.5.1. Contrasting effects of cyanide on salmonids at different temperatures. For lethal effects data, if LC_{50}s are greater than the Final Acute Value of 44 µg/L that is assumed to indicate lack of harm at acute criteria concentrations; for sublethal effects, lowest effects concentrations should be greater than 5.2 µg/L. 142
Table 2.4.6.1. Examples of mercury tissue residues co-occurring with the presence or absence of adverse effects. 151
Table 2.4.6.2. Examples of mercury concentrations in water and or mercury tissue burdens in fish muscle tissue (unless noted otherwise), in relation to the minimal effects threshold of 0.2 mg/kg in tissue and the 12 ng/L criteria in water under consultation 157
Table 2.4.6.3. Ranges of potential tissue concentrations that would result from (A) applying field-based BAFs to the chronic mercury water quality criterion of 12 ng/l, and (B) ranges of water concentrations that would result from applying BAFs to low-risk tissue concentrations. Calculations showing the laboratory water-only bioconcentration factor (BCF) used in EPA (1985g) to derive the 12 ng/L criterion are also included for comparison. 161
Table 2.4.8.1. Effects concentration (EC) estimates for selenium whole-body tissue residues on growth or survival of juvenile salmonids, assuming no maternal pre-exposure. Underlined value indicates EC selected for primary effects analysis 173
Table 2.4.8.2. Median selenium concentrations in coldwater stream webs relevant to the area of interest, data collected between 2001 and 2008 for all study locations. 177
Table 2.4.8.3 Median trophic transfer factors (TTF) and water-biofilm partitioning coefficients of selenium within coldwater stream food webs. 178
Table 2.4.12.1. Comparison of the most sensitive chronic endpoints (in µg/L, except hardness in mg/L) from relevant studies with dissolved lead and salmonids or benthic invertebrates (i.e., potential prey) for related species 199
Table 2.4.14.2. Reported Toxicity Equivalent Factors (TEFs) for the early life stage of fish . 254
Table 2.12.1. Aspects of the action that would or would not likely contribute to "adverse modifications" of critical habitat or "jeopardy". 297
Table 2.12.2. Summary of conclusions on the protectiveness of the Idaho Toxics aquatic life criteria for inorganic chemicals. 298
Table 2.12.3. Summary of conclusions on the protectiveness of Idaho aquatic life criteria for organic chemicals (abbreviations follow table) 301

FIGURES

Figure 1.4.1. Fourth-field hydrologic unit codes (HUCs) containing list salmon or steelhead. Each HUC is labeled with the last 4 digits of the 8-digit HUC code. The first 4 digits are 1706, Lower Snake subregion. 11
Figure 2.2.1. Boundaries of listed anadromous species and the action area (State of Idaho within the range of anadromous species). "Species" are defined as "evolutionarily significant units" or ESUs. The Panther Creek watershed is emphasized because the extirpation of the Panther Creek Chinook salmon population due poor water quality was considered a specific factor for the decline of and ESA listing of the Snake River spring/summer Chinook salmon ESU. Contemporary water quality and biological conditions in Panther Creek are described in the Status of Species section and in the Analyses of Effects sections for arsenic and copper. 22
Figure 2.2.2. Juvenile Chinook salmon abundance in Panther Creek, Idaho, from electrofishing surveys. "Middle" or "Upper" Panther Creek are downstream and upstream of mining influenced Blackbird Creek, respectively. Inset shows trends in aerial redd counts from approximately the same IDFG trend sections. Data from 1992 and earlier were taken from Mebane (1994), 1993 data from LeJeune *et al*. (1995), and subsequent data are from EcoMetrix (2011). 24
Figure 2.2.3. Major population groups and independent populations of Snake River spring/summer Chinook salmon. The populations codes are contracted from the above table, for example SRUMA=upper Salmon River, Salmon River mainstem 27
Figure 2.3.1.1. Arsenic in Panther Creek sediments sampled in similar stream reaches before and after remediation efforts. In both surveys arsenic declined with increasing distance downstream from Blackbird Creek. Arsenic appears to have generally declined over time, although arsenic is still greatly elevated until the diluting flows of Napias Creek, a large tributary, enter. This suggests a reservoir of arsenic may persist in sediments or riparian soils that may be difficult to further control. As of 2011, EPA is evaluating the feasibility of additional remediation to further reduce arsenic releases from Blackbird Creek. Data from Mebane (1994) and Golder (2009), probable effect concentration from MacDonald *et al*. (2000a). 45
Figure 2.3.1.2. Arsenic in periphyton (algae and other organic material collected from stream rocks) in Panther Creek sampled in similar stream reaches before and after remediation efforts. Periphyton is the primary food source for many aquatic insects. Data from Beltman *et al*. (1994) and EcoMetrix (2011)..... 46
Figure 2.3.1.3. Arsenic in macroinvertebrate tissues of Panther Creek sampled in similar stream reaches before and after remediation efforts. In both time periods arsenic declined with increasing distance downstream from Blackbird Creek. At the uppermost mining-affected sites, Panther Creek downstream of Blackbird Creek, arsenic initially declined markedly following remediation, but has not further declined from 2006 through 2010. The 2008 spike in arsenic concentrations apparent in sediment and periphyton graphs was not apparent in macroinvertebrates, suggesting limited bioavailability of arsenic in that event. Data from Beltman *et al*. (1994) and EcoMetrix (2011)..... 47

Figure 2.3.1.4. Copper in Panther Creek foodwebs has greatly declined following Blackbird Mine remediation efforts that have been ongoing from 1995 to date. (Top) copper in sediments in 1992 and 2008 (Mebane, 1994; Golder, 2009); (Middle), copper in periphyton; and (Bottom), copper in aquatic insect tissues (Beltman and others, 1994; EcoMetrix, 2011). 51
Figure 2.3.1.5. Copper concentrations (a) and corresponding diversity of all aquatic insects and abundance of mayflies (b), abundance of rainbow trout/steelhead and shorthead sculpin in (c) in Panther Creek, Idaho, downstream of mining-influenced Blackbird Creek. Aquatic insect diversity and abundance and fish abundance are scaled against concurrent upstream reference collections. Copper concentrations at upstream reference sites have been <3µg/L in measurements from 1993 to date, since the routine use of appropriate low detection levels and "clean" field sampling and laboratory techniques. 52
Figure 2.3.1.6. Copper concentrations (a) and corresponding diversity of all aquatic insects and mayflies (b), and abundance of mayflies (c), in Panther Creek, Idaho, downstream of mining-influenced Big Deer Creek. (See Figure 2.3.1.5 for data sources) 53
Figure 2.4.1.1. Size-developmental stage patterns with coho salmon from 2 to 7 weeks post hatch, data from Chapman (1975). Species and genus mean acute values (SMAVs and GMAV) are from the respective criteria documents (EPA 1984b, 1984a, 1985, 1987b), adjusted to test water hardness. All tests used Willamette River water, TOC 3.4 mg/L, hardness 22 mg/L. 73
Figure 2.4.1.2. Relations between size of swim-up rainbow and cutthroat trout and toxicity to zinc and lead sensitivity in renewal tests conducted in water from the South Fork Coeur d'Alene River, Idaho. Data from (Mebane *et al*. 2012). All test values adjusted to a median test hardness of 35 mg/L $CaCO_3$ using hardness-toxicity regressions from (Mebane *et al*. 2012). SMAVs were adjusted using the hardness-criteria equations from the respective criteria documents. 74
Figure 2.4.1.3. Resistance to cadmium and zinc toxicity decreased with increasing size over a weight range of 0.2 to 1.6g for swim-up rainbow trout. Data from Hansen (2002a) and Stratus (1999) using 96-h probit LC_{50} values. All tests conducted at a hardness of 30 mg/L and pH of 7.5 SMAV values were adjusted using the hardness-criteria equations from the respective criteria documents. 74
Figure 2.4.1.4. Resistance to copper toxicity decreased with increasing size over a weight range of 0.06 to 0.4g for swim-up rainbow trout, but above about 1g weight, resistance to copper toxicity increased with increasing size. Dashed lines indicate hardness-adjusted rainbow trout species mean acute value (SMAV) from EPA (1984). A. Relation between copper toxicity and the size of swim-up rainbow trout (<0.5g), from renewal tests conducted in water from the Clark Fork River, MT (Erickson *et al*. 1999); B. Relation between copper toxicity and the size of larger juvenile rainbow trout (>0.7g, older than swim-up fish), data from Chakoumakos *et al*'s (1979) tests under uniform water conditions (hardness 194 mg/L); C. Rainbow trout of difference sizes tested under uniform conditions at hardness 99 to 102 mg/L, data from Howarth and Sprague (1978). 75
Figure 2.4.1.5. Effect of developmental stage at the onset of continuous copper exposure (13.4 µg/L) on the survival of juvenile steelhead trout (figure from Chapman 1994). 77
Figure 2.4.1.6. Examples of percentages of coho salmon or rainbow trout killed at one-half their LC_{50} concentrations with cadmium, copper, and zinc. 81

Figure 2.4.1.7. Three example allowable scenarios for criteria exceedence magnitudes..... 97
Figure 2.4.2.1. Zinc toxicity versus water hardnesses for swim-up stage rainbow trout pooled across test groups and westslope cutthroat trout (data from Mebane *et al*. (2012). 109
Figure 2.4.2.2. Zinc (Zn) toxicity versus water hardnesses for swim-up stage rainbow trout by concurrent test groups, cutthroat trout, and steelhead tested under similar conditions by the same people (average fry weights are in parentheses) Data from (data from Mebane *et al*. (2012) except for steelhead data which are from Cusimano, Brakke and Chapman (1986)and Chapman (1978b). 110
Figure 2.4.2.3. Lead (Pb) toxicity versus water hardnesses for swim-up stage rainbow trout either pooled across test groups, or separated into synoptic and other tests and pooled westslope cutthroat trout (data from Mebane *et al*. (2012). 111
Figure 2.4.2.4. Cadmium (Cd) toxicity versus water hardnesses for rainbow trout tested under the same conditions on 5/23/99 versus "other" rainbow trout tested by the same people in the same facility, using the same source of fish eggs, same water sources, but using fish that were a few weeks apart in age (data from Mebane *et al*. (2012). 111
Figure 2.4.2.5. Soft-water ecoregions of the USA where most water hardness values are <50 mg/L CaCO3 (Whittier and Aitkin 2008). 112
Figure 2.4.2.6. Minimum hardness values measured at 323 sites in Idaho between 1979-2004 (data from Appendix A). 114
Figure 2.4.2.7. Examples of the effects of the "hardness floor" on cadmium and zinc criteria in very-soft and soft water settings. In the Pine Creek example (top), all hardness observations were less than the 25 mg/L floor. In this very-soft water example, applying a hardness floor would result in the criteria being considerably less protective than intended by EPA Guidelines at all times, with the floor-limited criteria as much as 3X higher. The Yankee Fork example (bottom) is probably more typical of soft water streams in the Salmon River drainage. There the floor has little or no effect during much of the record, at the worst the floor-limited criteria were about 1.25X higher than the hardness-dependent criteria. 116
Figure 2.4.4.1. Comparison of 96-hour LC_{50}s for salmonids with copper and the Idaho criterion final acute values, calculated for hardnesses up to 200 mg/L as CaC03. LC_{50}s limited to species within the genera Oncorhynchus, Salvelinus, and Salmo. If all LC_{50} values fell above the line, that would suggest that for the most part, few mortalities would be likely at criterion concentrations. 126
Figure 2.4.4.2. Comparison of the copper Idahochronic criterion and adverse chronic or sublethal effects and estimates of no-effect concentrations to salmonids. 130
Figure 2.4.4.3. Correlations of relevant macroinvertebrate metrics with mean exceedence factors of the chronic criterion for stations monitored in Panther Creek, Idaho, September 2005 and 2006 (EcoMetrix 2006, 2007). 136
Figure 2.4.5.1. Examples of the occurrence of different salmonid life stages and annual temperature patterns for a coldwater stream: salmonid species with (top) fall-spawning life histories (e.g., Chinook salmon, coho salmon, Atlantic salmon, brown trout, bull trout, brook trout), and (bottom) spring-spawning life histories (e.g., steelhead, rainbow trout, cutthroat trout, most non-salmonid fishes). Temperature data from the Salmon River, Idaho near Sunbeam, Idaho; data from Idaho Department of Environmental Quality, 2002 water year. 141

Figure 2.4.6.1. Modeled concentrations of mercury in fish-tissue for the Snake River at Lewiston, standardized by length and species (modeled and figure generated using the Environmental Mercury Mapping, Modeling, and Analysis (EMMMA) website *http://emmma.usgs.gov/*) 155
Figure 2.4.6.2. Concentrations of mercury in salmonid tissues from waters in southern Idaho and northern Nevada versus length. Data from Maret and MacCoy (2008) 156
Figure 2.4.7.1 Acute LC_{50}s for nickel with rainbow trout, any life stage (no data on other salmonids) vs. the Idaho and Idaho final acute values (FAVs). 164
Figure 2.4.7.2. Chronic effects, no-observed effect concentrations, and avoidance concentrations with rainbow trout vs. the NTR and Idaho chronic values for nickel. .. 165
Figure 2.4.8.1. Estimates of thresholds for no- and low-effect concentrations for selenium in diet and whole-body tissues of juvenile Chinook salmon or rainbow trout. Curve fitting and curve fitting and effects concentration percentiles (ECp) were estimated using threshold sigmoid regression (Erickson 2008). EC10=concentration causing a 10% reduction in growth or survival..... 174
Figure 2.4.8.2. Apparent concentration dependence of selenium water-particulate partitioning coefficients (K_d) from coldwater, salmonid streams. 179
Figure 2.4.9.1. Acute silver criterion in comparison with acute and chronic silver effects data 183
Figure 2.4.10.1. Comparison of reviewed 96-hour LC_{50}s for salmonids with zinc and the Idaho criterion final acute values (FAV), calculated for hardnesses up to 200 mg/L as $CaC0_3$. LC_{50}s limited to species within the genera *Oncorhynchus*, *Salvelinus*, and *Salmo*. If LC_{50} values fell above the FAV line, that would suggest few if any mortalities would be likely at criterion concentrations. 188
Figure 2.4.10.2. Example of a 96-hour toxicity test with rainbow trout in which zinc at its acute criterion concentration (CMC) killed about half of the fish tested. At the CMC, few if any fish are supposed to be killed. In this instance, the final acute value that the criterion was based on (i.e., the LC_{50} for a hypothetical organism more sensitive than 95% of organisms) was twice as high as the rainbow trout value.) Rainbow trout data from Mebane *et al*. (2012), test hardness 35 mg/L, 0.5g fish, wet wt. 189
Figure 2.4.10.3. Comparison of the Idaho chronic criterion and adverse chronic or sublethal effects and estimates of no-effect concentrations to salmonids..... 190
Figure 2.4.12.1. Acute LC_{50}s with salmonids, any life stage vs. the Idaho final acute value for lead..... 200
Figure 2.4.12.2. Chronic effects, no-effects, and avoidance concentrations of lead with salmonids vs. the Idaho chronic criterion concentrations for lead. 201

ACRONYMS

ACR	Acute-to-Chronic Ratio
ADR	Alternative Dispute Resolution
Ag	Silver
Ah	Aryl Hydrocarbon
ALC	Aquatic Life Criterion
As	Arsenic
ASTM	American Society for Testing and Materials
ATPase	A class of enzymes that catalyze the decomposition of adenosine triphosphate and is essential for metabolism in all known forms of life
AWQC	Ambient Water Quality Criteria
BA	Biological Assessment
BAF	Measured Bioaccumulation Factor
BCF	Bioconcentration Factor
BLM	Biotic Ligand Model
BMC	Benchmark Concentrations
BMP	Best Management Practice
BSAF	Biota-sediment Accumulation Factors
Ca	Calcium
CCC	Criterion Continuous Concentration
CCU	Cumulative Criterion Units
Cd	Cadmium
CERLA	Comprehensive Environmental Response, Compensation, and Liability Act (Superfund)
CF	Conversion Factor
cfs	cubic feet per second
Chronic Value	A synonym for MATC in the context of water quality criteria
CMC	Criterion Maximum Concentration
CN	Cyanide
COE	Army Corps of Engineers
Cr	Chromium
CRB	Columbia River Basin
CrIII	Trivalent Chromium
CrIV	Hexavalent Chromium
Cu	Copper
CWA	Clean Water Act
DDD	Dichlorodiphenyldichloroethane
DDE	Dichlorodiphenylethylene
DDT	Dichlorodiphenyltrichloroethane

DOC	Dissolved Organic Carbon
DOM	Dissolved Organic Matter
DPS	Distinct Population Segment
DQA	Data Quality Act
dw	dry weight
EC	Effects Concentration
EC_{50}	Concentration that caused effects to 50% of the test population
ECp	Effect Concentration Percentile
EEC	extreme effect
EFH	Essential Fish Habitat
EFSFSR	East Fork of the South Fork Salmon River
ELS	Early Life Stages
EMMA	Environmental Mercury Mapping, Modeling, and Analysis
EPA	U.S. Environmental Protection Agency
ESA	Endangered Species Act
ESU	Evolutionary Significant Unit
FAV	Final Acute Value
FDA	Food and Drug Administration
FPC	Fish Passage Center
GMAV	Genus Mean Acute Value
Hg	Mercury
HH	Human-Health
HOC	hydrophobic organic compounds
HUC	Hydrologic Unit Codes
ICE	Interspecies Correlation Estimates
ICTRT	Interior Columbia River Basin Technical Recovery Team
IDEQ	Idaho Department of Environmental Quality
IDFG	Idaho Department of Fish and Game
ISAB	Independent Scientific Advisory Board
ITS	Incidental Take Statement
IWQS	Idaho Water Quality Standards
LAA	Likely to Adversely Affect
LC_{50}	Lethal Concentration of 50%
LGD	Lower Granite Dam
LOEC	Lowest Observed Effects Concentration
ln(X)	Natural logarithim of the number “X”
MATC	Maximum Acceptable Toxicant Concentration
MEC	Midrange Effect
Mg	Magnesium
mg/L	milligram per liter, or parts per million

Mixing Zone	an area where an effluent discharge undergoes initial dilution and is extended to cover the secondary mixing in the ambient water body
MLDE	Maximum Likelihood Estimate
MPG	Major Population Groups
MSA	Magnuson-Stevens Fishery Conservation and Management Act
ng/L	nanogram per liter, or parts per trillion
Ni	Nickel
NLAA	Not Likely to Adversely Affect
NMFS	National Marine Fisheries Service
NOEC	No-Observed Effects Concentration
Nominal Concentrations	In toxicity testing, the intended or target concentration is often called the nominal concentration to distinguish it from measured concentrations, i.e., those concentrations that had been analytically verified. For economy, some studies only report nominal concentrations. In this Opinion, study results that were derived solely from "nominal data" were discounted as not being "best available science
NPDES	National Pollution Discharge Elimination System
NTR	National Toxics Rule (EPA, 57 Fed. Reg. 60848 (Dec. 22, 1992))
NTR Criteria	Water quality criteria for certain toxic pollutants promulgated by EPA for several states. (EPA 1992, 57 Fed. Reg. 60848 (Dec, 22, 1992))
Opinion	Biological Opinion
Pb	Lead
PCB	Polychlorinated Biphenyl
PCE	Primary Constituent Elements
PCP	Pentachlorophenol
PFMC	Pacific Fishery Management Council
RER	Rough Endoplasmic Reticulum
RM	River Mile
RPA	Reasonable and Prudent Alternative
RPM	Reasonable and Prudent Measures
RUP	Registered Use Product
Se	Selenium
SEC	Sediment Effect Concentration
SeMe	Selenomethionine
SER	Smooth Endoplasmic Reticulum
SMAV	Species Mean Acute Values
SMI	Stream Macroinvertebrate Index
SQG	Sediment Quality Guidelines
SSD	Species Sensitivity Distribution
TCDD	Tetrachlorodibenzo-p-dioxin
TCM	Thompson Creek Mine
TEC	Threshold Effect

TEF	Toxicity Equivalence Factor
TEQ	Toxicity equivalence Quantity
TIE/TRE	Toxicity Identification Evaluation and Toxicity Reduction Evaluation
TMDL	Total Maximum Daily Loads
TOC	Total Organic Carbon
TOST	Test of Significant Toxicity
TTF	Trophic Transfer Factor
TU	Toxic Unit
USFWS	U.S. Fish and Wildlife Service
USGS	U.S. Geological Survey
VSP	Viable Salmonid Populations
WAD	Weak Acid Dissociable
WER	Water Effects Ratio
WET	Whole- Effluent Toxicity
WQBEL	Water Quality Based Effluent Limitation
WQS	Water Quality Standards
ww	wet weight
YOY	Young-Of-year
Zn	Zinc
µg/L	microgram per liter or parts-per-billion
1Q10	The lowest 1-day average of streamflows occurring in a 10-year period
7Q10	The lowest 7-day running average of streamflows occurring in a 10-year period

1. INTRODUCTION

This introduction section provides information relevant to the other sections of this document and is incorporated by reference into Sections 2 and 3 below.

1.1. Background

The biological opinion (Opinion) and incidental take statement (ITS) portions of this consultation 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.

NMFS also completed an Essential Fish Habitat (EFH) consultation. It was prepared in accordance with section 305(b)(2) of the Magnuson-Stevens Fishery Conservation and Management Act (MSA) (16 U.S.C. 1801, *et seq*.) and implementing regulations at 50 CFR 600.

The Opinion and EFH Conservation Recommendations are both 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

This Opinion is based on information provided originally in U.S. Environmental Protection Agency's (EPA's) July 2000 biological assessment (BA) and modified in a December 2013 letter. In the interim there were many interactions including telephone conversations, meetings and written correspondence and regulatory changes that occurred to arrive at the final action as described in section 1.3 of this Opinion. The following is a summary of those interactions. A complete record of this consultation is on file at the Snake Basin Office in Boise, Idaho.

Section 303 of the Clean Water Act (CWA) mandates that states adopt water quality standards (WQS) to restore and maintain the chemical, physical, and biological integrity of the nation's waters. The WQS consist of beneficial uses to protect both aquatic life communities and recreational and subsistence based uses (i.e. salmonid spawning, cold water biota, primary or secondary contact recreation) designated for specific water bodies and water quality criteria to protect uses. States have primary responsibility for developing appropriate beneficial uses for water bodies in their state. States review and, if appropriate, revise their WQS on a triennial basis in accordance with CWA section 303(c). Also under CWA section 303(c), EPA must review and approve or disapprove any revised or new standards. If EPA disapproves any portion of the state standards the state has 90 days to adopt the changes specified by the EPA, after which time the EPA must propose and promulgate standards for the state.

On June 25, 1996, staff from EPA's Region 10 completed a review of the Idaho Water Quality Standards (IWQS) adopted August 24, 1994. During this review, the EPA disapproved seven elements within the state's WQS. Most of these elements have since been revised by Idaho and

approved by the EPA. These 1996 approvals were included as part of EPA's (2000) BA initiating this consultation. The elements which EPA disapproved and did not subsequently approve were not included in EPA's BA or the proposed action for this consultation.

As a result of several meetings held in 1999 between the U.S. Fish and Wildlife Service (USFWS), NMFS, EPA, and Idaho Department of Environmental Quality (IDEQ), all agencies agreed that two BAs should be developed. The first BA would consist of EPA evaluation of Idaho's numeric water quality criteria for 22 toxic constituents (listed below).

In a letter of March 23, 2000, the IDEQ informed the EPA, NMFS, and the USFWS that it wished to be considered an "applicant" to this action for this consultation as defined by 50 CFR § 402.02.

On August 9, 2000, EPA submitted its final BA to USFWS and NMFS and requested initiation of formal consultation under section 7 of the ESA. The BA concluded that the proposed criteria were not likely to adversely affect (NLAA) Snake River sockeye salmon, Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, and Snake River Basin steelhead for the following parameters:

> Criteria for aldrin/dieldrin, chlordane, Dichlorodiphenyltrichloroethane (DDT), endrin, heptachlor, lindane, polychlorinated biphenyl (PCB), pentachlorophenol (PCP), toxaphene, trivalent chromium (Cr[III]) and hexavalent chromium (Cr[VI]), nickel(Ni), and silver (Ag);
>
> Acute and chronic criteria for arsenic (As), cadmium (Cd), copper (Cu), cyanide (Cn), endosulfan, mercury (Hg) lead (Pb), and zinc (Zn); and
>
> Acute criteria for mercury (Hg) and selenium (Se).

The BA concluded that Idaho's proposed criteria were likely to adversely affect (LAA) Snake River sockeye salmon, Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, and Snake River Basin steelhead, for the following parameters:

> Chronic criteria for selenium (Se).

The BA did not include an analysis of effects for southern resident killer whales, which are listed as endangered and rely on listed salmonids as a food source. NMFS will complete an analysis on southern resident killer whales within 6 months.

On September 4, 2003, NMFS circulated a draft Opinion to EPA, USFWS, and the IDEQ for review. This was followed by a series of conference calls and meetings. No formal comments were received. Instead, EPA representatives proposed that all parties commit to working through the technical and policy issues through a facilitated alternative dispute resolution (ADR) process. The disputes involved effects determinations and the methods used to determine effects.

The EPA, NMFS, USFWS, and IDEQ representatives formed a technical committee and policy committee and participated in a series of facilitated meetings and conference calls, supported by EPA's ADR contractor. The interagency group did not reach final agreement on a set of recommended actions for completing the consultation. A final report was issued by the ADR contractor on September 22, 2005.

In 2005, IDEQ began negotiated rulemaking to revise the criteria values under consultation. On April 11, 2006, Idaho formally amended its water quality criteria. These criteria were subsequently approved by EPA in 2007, subject to ESA consultation.

On September 2, 2010, EPA provided NMFS a revised BA for cadmium criteria only, and asked NMFS to concur with their determination that their approval of Idaho's cadmium criteria was protective of and NLAA Snake River salmon and steelhead. On January 31, 2011, NMFS wrote to EPA concurring with their determination, accompanied by an independent review (NMFS 2011).

On February 6, 2013, NMFS provided a draft Opinion to EPA for comment. The EPA and NMFS met several times in 2013 and worked on modification to the Opinion and changes to the proposed action.

On November 22, 2013, EPA advised NMFS that they were revising their action for several criteria values under consultation to match those updated by IDEQ in 2006 and subsequently approved by EPA, subject to consultation. The revisions consisted of new acute and chronic criteria values for arsenic, chromium, nickel, and zinc. No new technical analyses of effects were included with the revised action letter and EPA's determinations were unchanged for these criteria.

1.3. Proposed Action

"Action" means all activities or programs of any kind authorized, funded or carried out, in whole or in part, by federal agencies. Interrelated actions are those that are part of a larger action and depend on the larger action for their justification. Interdependent actions are those that have no independent utility apart from the action under consideration.

The CWA requires all states to adopt WQS to restore and maintain the physical, chemical, and biological integrity of the Nation's waters. Section 303(c)(2)(E) of the CWA 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 the EPA. Since 1980, the EPA has published numerous criteria development guidelines for states and tribes and recommended national criteria for numerous pollutants. The national criteria include recommended acute and chronic criteria for the protection of aquatic life resources. States and tribes may choose to adopt EPA's recommended criteria, or modify these criteria to account for site-specific or other scientifically defensible factors. The state of Idaho has adopted criteria for toxic pollutants (IDAPA 58.01.02, 250.02 (a)(iv)). As initially adopted, all of the criteria were identical to criteria promulgated by EPA for several in EPA's 1992 National Toxics Rule (NTR)

(57 Fed. Reg. 60848, Dec. 22, 1992) (EPA 2000a). The state of Idaho subsequently revised several criteria, as listed in Table 1.3.1. The EPA has approved Idaho's adoption, subject to consultation for 23 toxic pollutants (Table 1.3.1). The EPA is consulting only on those aquatic life criteria for the chemicals in Table 1.3.1. There are many criteria for additional water quality parameters in the IWQS that are not part of the proposed action. Those primarily affecting fish include temperature, dissolved oxygen and sediment. Any impaired waters are shown in Idaho's 303(d) list are discussed further in the baseline section.

The IWQS for aquatic life contain two expressions of allowable magnitude that are constrained by allowable exposure duration and frequency:

> An acute, or criterion maximum concentration (CMC), to protect against short-term effects, that is not to be exceeded on average for longer than 1 hour and more than once every 3 years.
>
> A chronic, or criterion continuous concentration (CCC), to protect against long-term effects, that is not to be exceeded on average for longer than 4 days and more than once every 3 years.

1.3.1. Idaho's Water Quality Standards for Toxic Pollutants

The EPA has approved, subject to this consultation, Idaho's aquatic life criteria for 11 organic chemicals and replacement of existing aquatic life criteria for 11 metals. The proposed aquatic life criteria would apply to all waters in the state that are protected for aquatic life beneficial uses. The proposed numeric criteria are ambient water quality criteria (AWQC), which are concentrations of each pollutant measured in the water column. Under EPA policy, states may choose to adopt metals criteria measured as either dissolved metal or total recoverable metal. Idaho's aquatic life criteria for metals were based on total recoverable metal (dissolved + suspended). The proposed action would change the aquatic life criteria to concentrations based on dissolved metals only, using a conversion factor (CF) to account for the suspended fraction. With the use of dissolved criteria, water samples are filtered to remove suspended solids before analysis.

The proposed IWQS will apply to actions that require National Pollutant Discharge Elimination System (NPDES) permits, to development of total maximum daily loads (TMDLs) in streams with impaired water quality, and in situations where remedial actions are required to clean up spills or contaminated sites. When a TMDL is needed to regulate discharges into an impaired water body, the dissolved metals criteria must be converted or translated back to a total recoverable 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 a dissolved-to-total ratio based on site-specific total/dissolved metal concentrations in the receiving water.

For some of the pollutants subject to this consultation, Idaho has also adopted criteria to protect human health from risk from exposure to the substances through eating fish or shellfish or

ingestion of water through recreating on water. Although EPA is not consulting on the human health-based criteria, on a practical level, permitted discharges to a given water body would be constrained by the most stringent applicable criteria. In other words, the human health criteria will constrain discharge levels where they are more stringent than the aquatic life criteria. During the pendency of this consultation, Idaho has further revised some of the criteria under consultation. The EPA has updated its action to reflect these revisions and they are being consulted on as shown Table 1.3.1.

The application of AWQC is based on the principle of designated beneficial uses of water. Together, AWQC and use designations are used to meet the primary objective of the CWA – to "restore and maintain the chemical, physical and biological integrity of the Nation's waters." A further goal of the CWA is that wherever attainable, an interim goal of water quality is to provide "for the protection and propagation of fish, shellfish, and wildlife and provides for recreation in and on the water." (Clean Water Act, §101(a)).

Table 1.3.1. Ambient Water Quality Criteria for toxic pollutants submitted for consultation in EPA's 2000 Biological Evaluation. Also shown are AWQC that have subsequently been revised by the State of Idaho (Idaho Department of Environmental Quality 2011). In two parts, inorganic and organic substances:

Part 1. Criteria for metals and other inorganic substances

Substance (except as noted, as 0.45 µm filtered "dissolved" concentrations)	**Criteria evaluated in EPA's 2000 BA (µg/L)**		**Idaho revised criteria included in EPA's updated action (25 November 2013) (µg/L)**		**Relevant IWQS human health based criteria also applicable to waters in the action area (IDEQ 2011)**
	Acute	**Chronic**	**Acute**	**Chronic**	
Arsenic (As)	360	190	340	150	10 µg/L human health criterion also applies
Cadmium (cd) [f]	3.7	1.0	1.3	0.6	
[Note: Cd was included in the BA but was subsequently consulted on separately. See NMFS (2011)]					
Copper (Cu) [b]	17	11	17	11	
Cyanide (CN, weak acid dissociable)	22	5.2	22	5.2	
Lead (Pb) [b, c]	65	2.5	65	2.5	
Mercury (Hg)	2.1	0.012 (unfiltered)	g	g	0.3 mg/kg in fish tissue, fresh weight
Selenium (Se)	20	5.0 (unfiltered)	20	5. (unfiltered	
Zinc (Zn) [b]	114	105	120	120	
Chromium (Cr) (III) [b]	550	180	570	74	
Chromium (Cr) (VI)	15	10	16	11	
Nickel (Ni)[b]	1,400	160	470	52	
Silver (Ag)[b]	3.4	–	3.4	–	

(µg/L: micrograms per liter; Metals criteria are shown for a water hardness of 100 mg/L).

Part 2. Criteria for organic toxic substances

Substance	Aquatic life criteria evaluated in EPA's 2000 BA (µg/L)		Human-health based AWQC that also apply to waters designated to support "cold water biota" or "salmonid spawning" and to critical habitats for listed species in the action area (µg/L)	Idaho criteria that were revised subsequent to EPA's 2000 BA (µg/L) [h]
	Acute	**Chronic**		**Idaho**
Endosulfan (α and β)	0.22	0.056	2	89
Aldrin	3	–	0.00014	0.000050
Chlordane	2.4	0.0043	0.00057	0.00081
4,4'-DDT	1.1	0.001	0.00059	0.00022
Dieldrin	2.5	0.0019	0.00014	0.000054
Endrin	0.18	0.0023	0.81	0.060
Heptachlor	0.52	0.0038	0.00021	0.000079
Lindane (gamma-BHC)	2	0.08	0.063	1.8
Polychlorinated biphenyls (PCBs)	N/A	0.014	0.000045	0.000064
Pentachlorophenol (PCP)	20[e]	13[e]	6.2	3
Toxaphene	0.73	0.0002	0.00075	0.00028

– - no applicable criteria

a. Conversion factors for translating between dissolved and total recoverable criteria.

b. For comparison purposes, the values displayed in this table correspond to a total hardness of 100 mg/l $CaCO_3$ and a Water Effects Ratio (WER) of 1.0. Criteria for these metals are actually expressed as a function of total hardness (mg/L as $CaCO_3$), and the following equation:
Acute Criteria = WER $\exp(m_A[\ln(\text{hardness})]+b_A)$ x Acute Conversion Factor
Chronic Criteria = WER $\exp(m_C[\ln(\text{hardness})]+b_C)$ x Chronic Conversion Factor
where:

Metal	m_A[f]	b_A[f]	m_C[f]	b_C[f]
Chromium (III)	0.8190	3.688	0.8190	1.561
Copper	0.9422	-1.464	0.8545	-1.465
Lead	1.273	-1.460	1.273	-4.705
Nickel	0.8460	3.3612	0.8460	1.1645
Silver	1.72	-6.52	N/A	N/A
Zinc	0.8473	0.8604	0.8473	0.7614

The term "exp" represents the base e exponential function. m_A and m_c are the slopes of the relationship for hardness, while b_A and b_C are the Y-intercepts for these relationships.

c. The conversion factor for lead is hardness dependent. The values shown in the table correspond to a hardness of 100 mg/L $CaCO_3$. Conversion factors for lead: Acute and Chronic- CF=1.46203-[(ln(hardness))x(0.145712)]
d. Criteria expressed as Weak Acid Dissociable
e. Criteria for pentachlorophenol increase as pH increases and are calculated as follows:
Acute Criterion = exp(1.005 (pH) - 4.830)
Chronic Criterion = exp(1.005 (pH) - 5.290) Values shown in the table are for pH 7.8
f. Cadmium aquatic life criteria are listed for descriptive purposes only. Cadmium aquatic life criteria were originally part of EPA's action and the consultation package (EPA 2000a). However in 2006, Idaho substantially revised their aquatic life criteria for cadmium, which EPA (2010a) subsequently proposed separate approval of, and initiated consultation on the revised cadmium criteria. EPA's (2010a) determination was that Idaho's 2006 revised cadmium criteria was NLAA listed salmonids, to which NMFS (2011) concurred.
g. The state of Idaho repealed the water column aquatic life criteria for mercury in 2006, based upon IDEQ's (2005) analysis that concluded the available science no longer supported EPA's (1985g) aquatic life criteria, and that a fish tissue based human-health criteria would be better supported by the science, be adequate to protect aquatic life, and would be more stringent than the 1985 chronic aquatic life criterion of 0.012 µg/L. EPA disapproved Idaho's repeal of its water column acute and chronic mercury criteria on policy grounds that, an exception for California notwithstanding, water column based aquatic criteria were required for Idaho, Idaho's criteria did not include a sufficiently detailed implementation for translating the human health tissue criterion to a protective aquatic life criteria that could be used with effluent limits (Gearheard 2008). The disapproval addressed policy interpretations and was silent on IDEQ's arguments that the EPA (1985g) mercury chronic was outdated and that a 0.3 mg/kg fish tissue criterion was more protective. Gearheard (2008) considered the 0.012 µg/L chronic criterion to be effective for NPDES discharge permits and TMDLs issued by EPA, although the criterion remains repealed under state law and nowhere appears in Idaho administrative rules.
h. Although Idaho's revised human health criteria are considerably more stringent than the previous human health criteria, EPA has not approved these revised criteria and EPA does not consider the more stringent criteria to be effective for Clean Water Act purposed.

1.3.2. Application of the IWQS for Metals

Per EPA's guidance, states, when adopting criteria for metals, may adopt criteria measured as either dissolved or total recoverable metal. The Idaho metals criteria under consultation are expressed as dissolved metals, meaning that water samples are filtered to remove suspended solids before analysis.

Metals and inorganic toxic substances addressed in this consultation include: As, cyanide, chromium (III), chromium (VI), copper, lead, mercury, nickel, selenium, silver, and zinc. For several of these chemicals, the water quality criteria are equation-based, meaning the criteria applicable to a particular site vary based on site-specific conditions. The equation-based metals are chromium (III), chromium (VI), copper, lead, mercury, nickel, silver, and zinc. To determine criteria for these metals for a given water body, site-specific data must be obtained, input to an equation, and numeric criteria computed. There are three types of site-specific data that may be necessary to determine and/or modify the criteria for these metals at a site: water hardness, CF and translators, and water effect ratios. Following is a brief description of these types of data.

The general equation for a hardness-based acute (CMC) or chronic (CCC) criterion with respect to total metal concentration (dissolved plus particulate) is:

CMC or CCC (total recoverable) = $e^{(m[\ln(\text{hardness})]+b)}$

Note that this is algebraically equivalent to the simpler expression:

CMC or CCC (total recoverable) = $K \cdot (\text{hardness})^m$

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 recoverable based criteria. The Idaho AWQC as evaluated in the BA are dissolved. The CFs for the metals are in the footnote to Table 1.3.1. The appropriate equation is:

CMC or CCC (dissolved) = $CF \cdot e^{(m[\ln(\text{hardness})]+b)} = CF \cdot K \cdot (\text{hardness})^m$

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 water hardness. For those metals that are hardness dependent, EPA calculates NPDES permit limits and load allocations for TMDLs using the 5^{th} percentile of the available ambient and or effluent hardness values

If a TMDL is needed to regulate discharges into an impaired water body, the dissolved criterion must be converted or translated back to a total recoverable 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.

Equations for trivalent chromium, copper, lead, nickel, silver, and zinc also include a Water Effects Ratio (WER), a number that acts as a multiplication factor. If no site-specific WER is determined, then the WER is presumed to be 1 and does not modify the equation result. A WER is intended to account for the difference in toxicity of a metal in 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. The EPA has provided procedures and requirements for determining "site-specific" WER values, which include extensive comparative toxicity testing with several test organisms and statistical analysis of results (Stephan *et al*. 1994b) (see Section 2.4.1.8 for additional discussion). The example provided below illustrates the basic principle in defining a WER value.

Example WER calculation:

Suppose the lethal concentration of 50% of test organisms (LC_{50}) of copper in site water is 15 µg/L
Suppose the LC_{50} of copper in laboratory water is 10 µg/L
Assume a site hardness of 100 mg/L
The freshwater CF for copper = 0.96
Acute AWQC for total recoverable copper without the WER = 18 µg/L

A LC_{50}15 µg/L in site water and a laboratory water LC_{50}10 µg/L yields a WER of 1.5. Then:

Cu Site-Specific CMC=WER x CF x $e^{(m[\ln(40)]+b)}$
=1.5 x 0.96 x 18 µg/L
=24 µg/L

In this hypothetical example, this approach yielded a site-specific criterion that is higher than the concentration killing 50% of a sensitive organism in the same site water, which is one of the logical problems with the WER approach to setting metals criteria. Additional discussion of implementation of WERs is provided in section 2.4.1.8. The "Water-Effect Ratio" Provision.

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).

For this project, the action area includes all watersheds within Idaho that contain anadromous species or their habitats (Figure 1.4.1) or upstream areas where discharges occur that may affect listed salmon, steelhead or their habitat. Table 1.4.1 lists all the 4th field hydrologic unit codes (HUCs) that contain listed salmon or steelhead. Each of these HUCs is located within the larger hydrologic unit, Lower Snake subregion (HUC 1706).

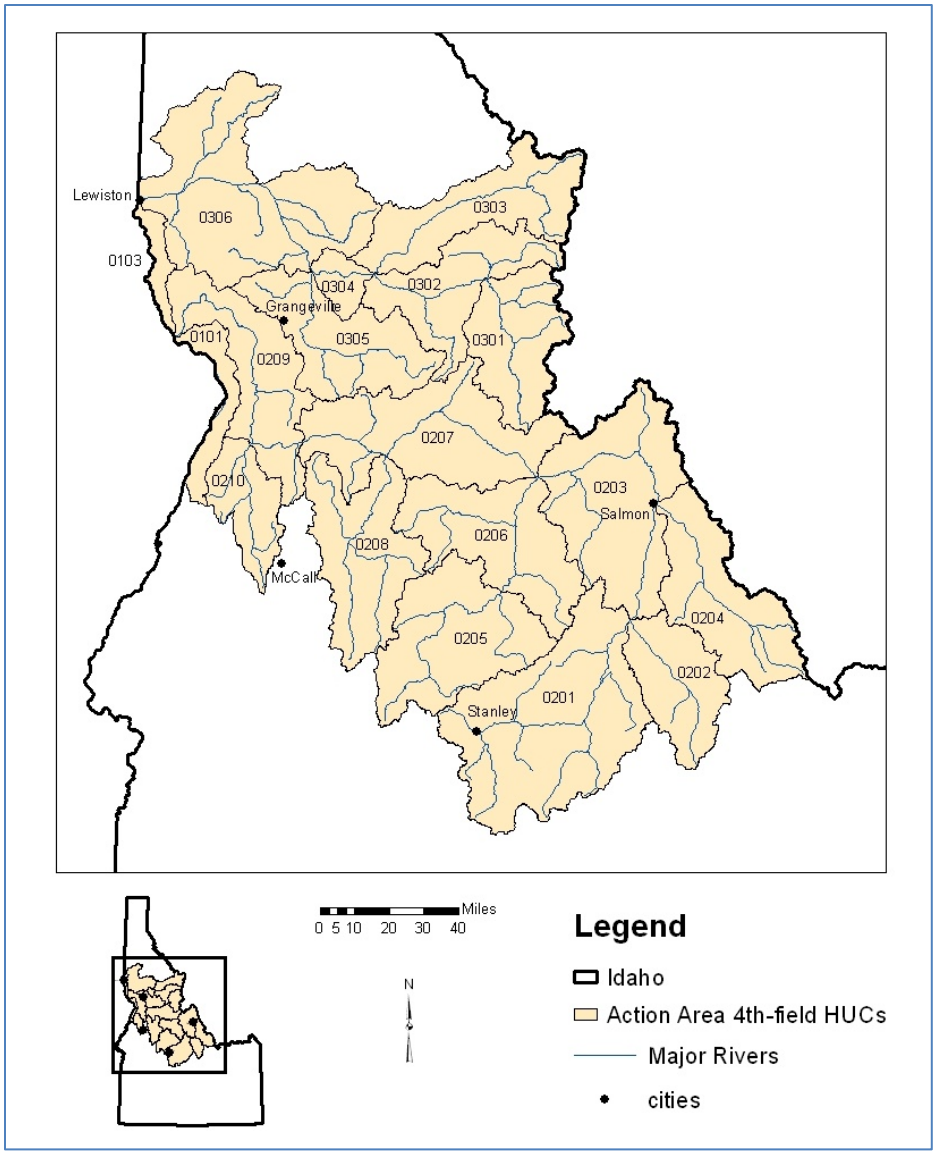


Figure 1.4.1. Fourth-field hydrologic unit codes (HUCs) containing list salmon or steelhead. Each HUC is labeled with the last 4 digits of the 8-digit HUC code. The first 4 digits are 1706, Lower Snake subregion.

Table 1.4.1. Fourth field HUCs containing listed salmon or steelhead.

HUC Number	HUC Name	HUC Number	HUC Name
17060101	Hells Canyon	17060209	Lower Salmon
17060103	Lower Snake-Asotin	17060210	Little Salmon
17060201	Upper Salmon	17060301	Upper Selway
17060202	Pahsimeroi	17060302	Lower Selway
17060203	Middle Salmon-Panther	17060303	Lochsa
17060204	Lemhi	17060304	Middle Fork Clearwater
17060205	Upper Middle Fork Salmon	17060305	South Fork Clearwater
17060206	Lower Middle Fork Salmon	17060306	Clearwater
17060207	Middle Salmon-Chamberlain	17060307	Upper North Fork Clearwater
17060208	South Fork Salmon	17060308	Lower North Fork Clearwater

The action area is used by all the freshwater life history stages (spawning, rearing, and migration) of threatened Snake River spring/summer and fall Chinook salmon, Snake River sockeye salmon, and Snake River Basin steelhead. Designated critical habitat for fall Chinook includes all reaches of the Snake River 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; the North Fork Clearwater River from its confluence with the Clearwater River upstream to Dworshak Dam; and the Salmon River reaches in the lower Salmon hydrologic unit. Designated critical habitat for the Snake River spring/summer Chinook salmon includes all river reaches presently or historically accessible to the species (64 FR 57399; October 25, 1999). Within Idaho, designated critical habitat for sockeye salmon includes the Snake and Salmon Rivers; Alturas Lake Creek; Valley Creek; and Stanley, Redfish, Yellowbelly, Pettit, and Alturas Lakes (including their inlet and outlet creeks). Designated critical habitat for Snake River Basin steelhead includes specific reaches of streams and rivers, as published in the Federal Register (70 FR 52630; September 2, 2005). The action area also contains EFH for Chinook salmon and coho salmon (Pacific Fishery Management Council [PFMC] 1999).

The Snake River below the Idaho border is not considered part of the action area because it is subject to water quality standards in Oregon and Washington and either have been or will be subject to separate consultations. EPA and the state of Idaho are responsible to ensure that downstream standards are attained at the state border (40 CFR 131.10(b)). For example the Potlatch NPDES permit which discharges into the Snake River near the Washington border undergoes a 401 certification review by both states to assure it meets all applicable criteria within both states.

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 United States 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 Services 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 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 C.F.R. 402.02. Instead, we have relied upon the statutory provisions of the ESA to complete the following analysis with respect to critical habitat.[1]

NMFS uses the following approach to determine whether the proposed action described in Section 1.3 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 species' "reproduction, numbers, or distribution" (50 CFR 402.02). In describing 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 Species Act) (November 7, 2005).

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 (MPG), 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 2.2.

- *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 2.3 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. NMFS also evaluates the proposed action's effects on critical habitat features. The effects of the action are described in Section 2.4 of this Opinion.

- *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 2.5 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 2.4) to the environmental baseline (Section 2.3) and the cumulative effects (Section 2.5) 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 2.2). Integration and synthesis occurs in Section 2.6 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 2.7. These conclusions flow from the logic and rationale presented in the Integration and Synthesis (Section 2.6) of this Opinion.

- *If necessary, define a reasonable and prudent alternative to the proposed action.* If, in completing the last step in the analysis, NMFS determines that the action under consultation is likely to jeopardize the continued existence of listed species or destroy or

adversely modify designated critical habitat, NMFS must identify a reasonable and prudent alternative (RPA) to the action in Section 2.8. The RPA must not be likely to jeopardize the continued existence of ESA-listed species nor adversely modify their designated critical habitat and it must meet other regulatory requirements.

2.2. Rangewide Status of the Species and Critical Habitat

This Opinion examines the status of each species that is likely to be affected by the action. The status is the level of risk that the listed species face, based on parameters considered in documents such as recovery plans, status reviews, and listing decisions. The species status section helps to inform the description of the species' current "reproduction, numbers, or distribution" as described in 50 CFR 402.02. The Opinion also examines the condition of critical habitat throughout the designated area, evaluates the conservation value of the various watersheds and coastal and marine environments that make up the designated area, and discusses the current function of the essential physical and biological features that help to form that conservation value. The listed species in the action area and the listing status are shown in Table 2.2.1.

Table 2.2.1. 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.

Species	Listing Status	Critical Habitat	Protective Regulations
Chinook salmon (*Oncorhynchus tshawytscha*)			
Snake River spring/summer run	T 6/28/05; 70 FR 37160	12/28/93; 58 FR 68543 10/25/99; 64 FR 57399	6/28/05; 70 FR 37160
Snake River fall run	12/28/93; 58 FR 68543	12/28/93; 58 FR 68543	12/28/93; 58 FR 68543
Sockeye salmon (*O. nerka*)			
Snake River	E 6/28/05; 70 FR 37160	12/28/93; 58 FR 68543	ESA Section 9 applies
Steelhead (*O. mykiss*)			
Snake River Basin	T 1/05/06; 71 FR 834	9/02/05; 70 FR 52630	6/28/05; 70 FR 37160

2.2.1. Status of the Species

For Pacific salmon and steelhead, NMFS commonly uses four parameters to assess the viability of the populations that, together, constitute the species: spatial structure, diversity, abundance, and productivity (McElhany *et al.* 2000). These VSP criteria therefore encompass the species' "reproduction, numbers, or distribution" as described in 50 CFR 402.02. When these parameters are collectively at appropriate levels, they maintain a population's capacity to adapt to various environmental conditions and allow it to sustain itself in the natural environment. These attributes are influenced by survival, behavior, and experiences throughout a species' entire life cycle, and these characteristics, in turn, are influenced by habitat and other environmental conditions.

"Spatial structure" refers both to the spatial distributions of individuals in the population and the processes that generate that distribution. A population's spatial structure depends fundamentally on habitat quality and spatial configuration and the dynamics and dispersal characteristics of individuals in the population. "Diversity" refers to the distribution of traits within and among populations. These range in scale from DNA sequence variation at single genes to complex life history traits (McElhany *et al.* 2000).

"Abundance" generally refers to the number of naturally-produced adults (i.e., the progeny of naturally-spawning parents) in the natural environment (e.g., on spawning grounds). "Productivity," as applied to viability factors, refers to the entire life cycle; (i.e., the number of naturally-spawning adults produced per parent). When progeny replace or exceed the number of parents, a population is stable or increasing. When progeny fail to replace the number of parents, the population is declining. McElhany *et al.* (2000) use the terms "population growth rate" and "productivity" interchangeably when referring to production over the entire life cycle. They also refer to "trend in abundance," which is the manifestation of long-term population growth rate.

For species with multiple populations, once the biological status of a species' populations has been determined, NMFS assesses the status of the entire species using criteria for groups of populations, as described in recovery plans and guidance documents from technical recovery teams. Considerations for species viability include having multiple populations that are viable, ensuring that populations with unique life histories and phenotypes are viable, and that some viable populations are both widespread to avoid concurrent extinctions from mass catastrophes and spatially close to allow functioning as metapopulations (McElhany *et al.* 2000).

2.2.1.1. Snake River Sockeye Salmon

The Snake River sockeye salmon, listed as endangered on November 20, 1991 (56 FR 58619), includes all populations of sockeye salmon originating from the Snake River basin, Idaho (extant populations occur only in the Salmon River drainage), as well as sockeye salmon from one artificial propagation program, the Redfish Lake Captive Broodstock program. On August 15, 2011, NMFS completed a 5-year review for the Snake River sockeye salmon ESU and concluded that the species should remain listed as endangered (76 FR 50448).

In Idaho, Snake River sockeye salmon historically spawned and reared in several high mountain lakes (Waples *et al*. 1991a). In the Salmon River basin, sockeye salmon occurred in five lakes (i.e., Alturas, Stanley, Redfish, Yellowbelly, and Pettit Lakes), all of which are near the headwaters of the Salmon River. In the Payette River basin, sockeye salmon historically occurred in the Payette Lakes (Evermann 1895; Fulton 1970); however, access to this basin was blocked upon construction of the Hells Canyon Dam. Thus, spawning and juvenile rearing habitat is currently restricted to the upper portions of the Salmon River Basin. Currently, the Snake River sockeye salmon population is highly dependent on a captive brooding program at the Sawtooth Hatchery (Ford *et al*. 2011).

Since the 1941 completion of the Grand Coulee Dam on the Columbia River that cut off the Arrow Lakes population of sockeye salmon in British Columbia, Snake River sockeye salmon

represent the longest inland spawning migration in North America (approximately 930 miles) (Bjornn *et al.* 1968; Behnke and Tomelleri 2002) to the highest elevation (approximately 6,500 feet in elevation) and the most southern destination in the world. Snake River sockeye salmon adults enter the Columbia River primarily during June and July. Arrival at Redfish Lake, which now supports the only remaining run of Snake River sockeye salmon, peaks in August, and spawning occurs primarily in October (Bjornn *et al*. 1968). Eggs hatch in the spring between 80 and 140 days after spawning. Fry remain in the gravel for 3 to 5 weeks, emerge from April through May, and move immediately into the lake. Once there, juveniles feed on plankton for 1 to 3 years before they migrate to the ocean (Bell 1986). Migrants leave Redfish Lake during late April through May (Bjornn *et al*. 1968) and travel to the Pacific Ocean. Smolts reaching the ocean remain inshore or within the influence of the Columbia River plume during the early summer months. Later, they migrate through the northeast Pacific Ocean (Hart 1973; Hartt and Dell 1986). Snake River sockeye salmon usually spend 2 to 3 years in the Pacific Ocean and return in their fourth or fifth year of life.

From 1991 to 1998 a total of 16 natural-origin adult anadromous sockeye salmon returned to Redfish Lake. These natural-origin fish were incorporated into the NMFS/IDFG captive broodstock program that began in 1992. Releases from the NMFS and IDFG captive broodstock programs generated seven returning adults in 1999, 257 adults in 2000, and 1355 adults in 2010 (Table 2.2.2). The 2010 adult return of Snake River sockeye salmon to Redfish Lake reached numbers not seen in decades. For each of the past 3 years for which data is available (2008, 2009, and 2010), the number of returning adults captured in the upper Sawtooth basin was more than the cumulative annual adult return that occurred between the time the fish were listed as endangered in 1991 and 2007.

Table 2.2.2. Adult returns passing Lower Granite Dam (LGD) and returning to the area of Redfish Lake (Sawtooth Basin, Idaho) (IDFG 2011; Fish Passage Center 2011a; NMFS 2008).

Adult Return Year	Number of Adults Passing LGD	#of Adults Returning to Sawtooth Basin	Percent Survival from LGD to Sawtooth Basin
1995	3	0	0
1996	3	1	33
1997	11	0	0
1998	2	1	25
1999	14	7	50
2000	299	257	86
2001	36	26	72
2002	55	22	40
2003	11	3	21
2004	113	27	24
2005	18	6	32
2006	17	3	18
2007	52	4	8
2008	909	650	71
2009	1219	833	68
2010	2201	1355	62

The high return of adult Snake River sockeye salmon is likely due to a combination of factors, including an increased number of fish released from captive broodstock programs, good conditions during downstream and upstream migrations (river flow and temperature, and dam passage conditions), and favorable ocean conditions (Ford 2011). The captive broodstock program has expanded from a starting point of 16 natural-origin adults that returned in the early 1990s to currently releasing hundreds of thousands of juvenile fish each year (Ford *et al*. 2011).

The Snake River sockeye salmon ESU consists of a single MPG. This MPG potentially has five component populations: Redfish Lake (including Little Redfish Lake); Alturas Lake; Pettit Lake; Yellowbelly Lake; and Stanley Lake. Of these, only the Redfish Lake population is currently extant (ICTRT 2007). Assuming there are five populations in this single MPG ESU, three populations would need to achieve viable status for the MPG and ESU to be viable. Since this is a single-MPG ESU, two of the three populations would need to be rated "Highly Viable" based on the four VSP parameters described in McElhany *et al*. (2000), and a third population needs to be rated "Viable." The latest available Interior Columbia River Basin Technical Recovery Team (ICTRT) recommendation (2007) is to achieve viable populations in three different lakes, with at least at least 1,000 naturally produced spawners per year in each of Redfish and Alturas lakes and at least 500 in Pettit Lake.

The viability status of populations in the ESU and the (single-MPG) ESU as a whole were determined by application of the ICTRT (2007) viability criteria. Viability determinations at the population level were based on extinction risk assessments for the four VSP parameters; abundance, productivity, spatial structure and diversity. A quantitative assessment risk for the VSP abundance/productivity metric was not completed for the populations in the ESU and the single-MPG ESU as a whole because of the lack of abundance and productivity data. Ford (2011) has preliminarily made a qualitative determination that abundance/productivity risk is *High*, based on the current status of the ESU (Endangered) and the recent absence of natural-origin anadromous adults returning to the Stanley Basin. The current average productivity likely is substantially less than the productivity required for any population to be at Low (1% to 5%) extinction risk at the minimum abundance threshold. In addition, the overall spatial structure and diversity has been rated *High* risk for the Redfish Lake population. This rating has been applied to this population because it rated high risk of not being able to maintain: (1) The natural patterns of phenotypic and genotypic expression; (2) natural patterns of gene flow; and (3) the integrity of natural systems. Overall, the Snake River sockeye salmon ESU does not meet the ESU-level viability criteria (non-negligible risk of extinction over 100-year time period) based on current abundance and productivity information.

There have been higher returns in recent years, the annual abundances of natural-origin (or, naturally spawned) sockeye salmon returning to the Stanley basin continue to be extremely low. The captive brood program has been successful in providing substantial numbers of hatchery produced sockeye salmon for use in supplementation efforts, which reduces the risk of immediate loss; yet, 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). Current smolt-to-adult survival of sockeye originating from the Stanley basin lakes is rarely greater than 0.3% (Hebdon *et al*. 2004). Although the risk status of the Snake River sockeye salmon ESU appears to be on an improving trend due to the successes of the captive propagation program, the 5-year review concluded that the ESU remains at high risk (Ford 2011).

2.2.1.2. Snake River Spring/Summer Chinook Salmon

The Snake River spring/summer Chinook salmon ESU was listed as threatened on April 22, 1992 (57 FR 14653). This ESU occupies the Snake River basin which drains portions of southeastern Washington, northeastern Oregon, and north/central Idaho. Environmental conditions are generally drier and warmer in these areas than in areas occupied by other Chinook species. This ESU includes all natural-origin populations in the mainstem Snake River (below Hells Canyon Dam) and the Tucannon, Grande Ronde, Imnaha, and Salmon Rivers. The ESU also includes 15 artificial propagation programs: the Tucannon River (conventional and captive broodstock programs), Lostine River, Catherine Creek, Lookingglass Creek, Upper Grande Ronde River, Imnaha River, and Big Sheep Creek programs in Oregon; and the South Fork Salmon River (McCall Hatchery), Johnson Creek, Lemhi River, Pahsimeroi River, East Fork Salmon River, West Fork Yankee Fork Salmon River, and Upper Salmon River (Sawtooth Hatchery) programs in Idaho (70 FR 37160; June 28, 2005). On August 15, 2011, NMFS completed a 5-year review for the Snake River ESU and concluded that the species should remain listed as threatened (76 FR 50448).

Chinook salmon exhibit a variety of complex life history patterns that include variation in age at seaward migration; length of freshwater, estuarine, and oceanic residence; ocean distribution; ocean migratory patterns; and age and season of spawning migration. Two distinct races of Chinook salmon are generally recognized: "stream-type" and "ocean-type" (Healey 1991; Myers *et al.* 1998). Snake River spring/summer Chinook salmon exhibit stream-type life history characteristics. Adult Snake River spring/summer Chinook salmon enter the Columbia River in late February and early March after 2 or 3 years in the ocean. In high elevation areas, mature fish hold in cool, deep pools until late summer and early fall, when they return to their native streams to begin spawning. They typically spawn in moderate to large-sized streams in shallow gravel bars at the downstream end of pools. Eggs incubate over the winter, and emergence begins in late winter and early spring of the following year. Juveniles rear through the summer, overwinter, and migrate to sea in the spring of their second year of life. During freshwater rearing, juvenile Chinook salmon disperse into tributary streams near their natal streams, and are often concentrated near the mouths of stream confluences. Depending on the tributary and the specific habitat conditions, juveniles may migrate extensively from natal reaches into alternative summer-rearing or overwintering areas. Habitats used by juvenile stream-type Chinook salmon and their feeding habits are similar to those for steelhead. In general, Chinook salmon tend to occupy streams with lower gradients than steelhead, but there is considerable overlap between the distributions of the two species.

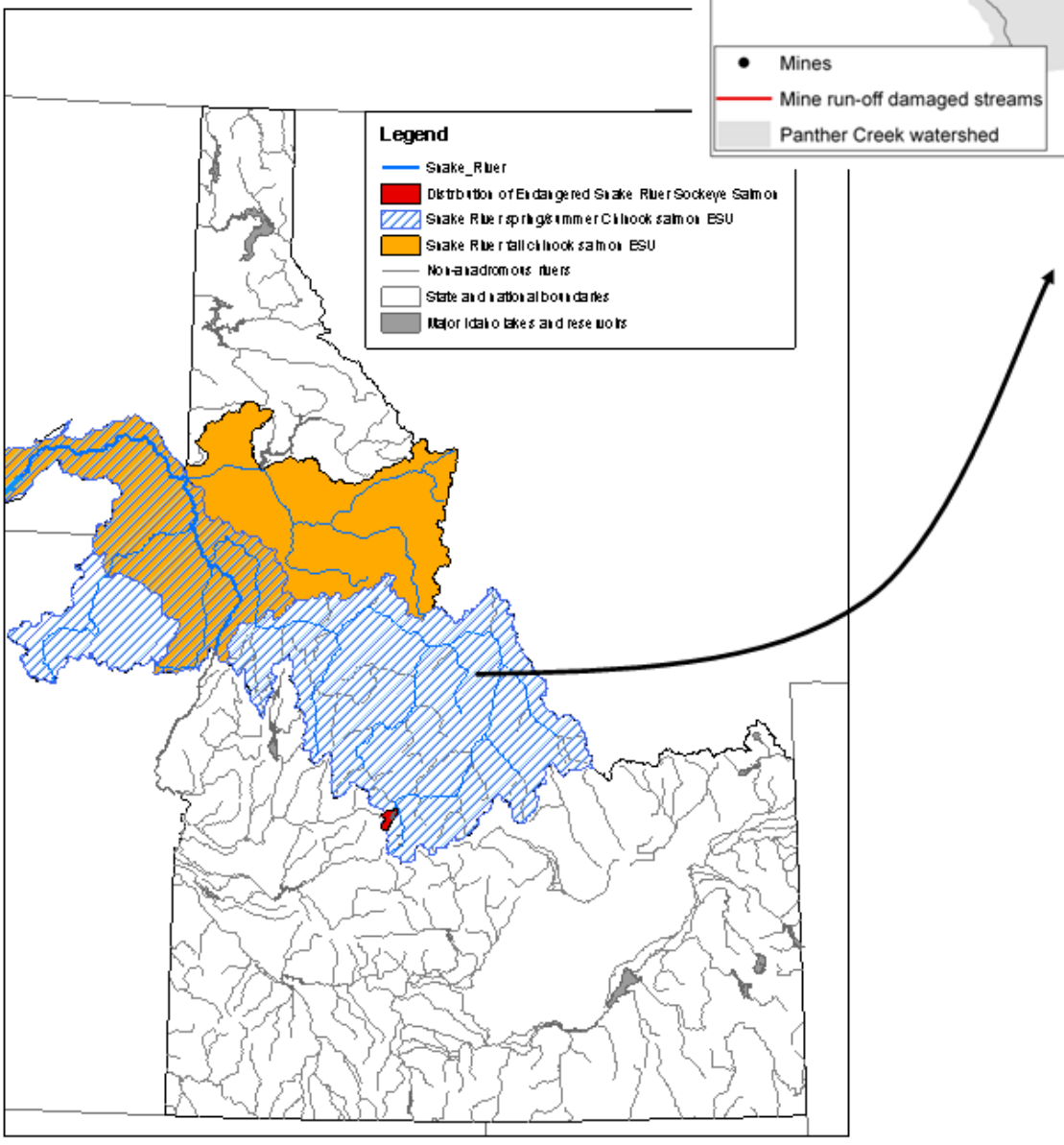
Although direct estimates of historical annual Snake River spring/summer Chinook returns are not available, returns may have declined by as much as 96% between the late 1800s and 2010. According to Matthews and Waples (1991), the Snake River drainage is thought to have produced more than 1.5 million adult spring/ summer Chinook salmon in some years during the late 1800s. By the 1950s the abundance of spring/summer Chinook had declined to an annual average of 125,000 adults and total (natural + hatchery origin) returns fell to roughly 100,000 spawners by the late 1960s (Fulton 1968). Adult returns counted at LGD reached all-time lows in the mid-1990s, although numbers have begun to increase since 1997 (FPC 2011b). The 2001 and 2002 total returns increased to over 185,000 and 97,000 adults, respectively. These large returns are thought to have been a result of favorable ocean conditions (Logerwell *et al.* 2003; Meeings and Lackey 2005) and above average flows in the Columbia River basin (CRB) when the smolts migrated downstream. However, it is important to note that over 80% of the 2001 return and over 60% of the 2002 return originated in hatcheries (Good *et al.* 2005). Furthermore, even these large returns are only a fraction (approximately 5% to 10%) of the estimated returns of the late 1800s. According to the Fish Passage Center (FPC) annual adult passage data (2011b), the 2003 and 2004 runs remained relatively high at 87,031 and 79,509 respectively, and fluctuated over the following years. Adult returns appeared to decline during 2005 to 2007 (average 30,856 total adults), but then increased again from 2008 to 2010. Despite the recent increases in total spring/summer Chinook salmon returns to the basin, natural-origin abundance and productivity are still far below their targets. As such, the Snake River spring/summer Chinook salmon ESU remains likely to become endangered (Good *et al.* 2005; Ford 2011).

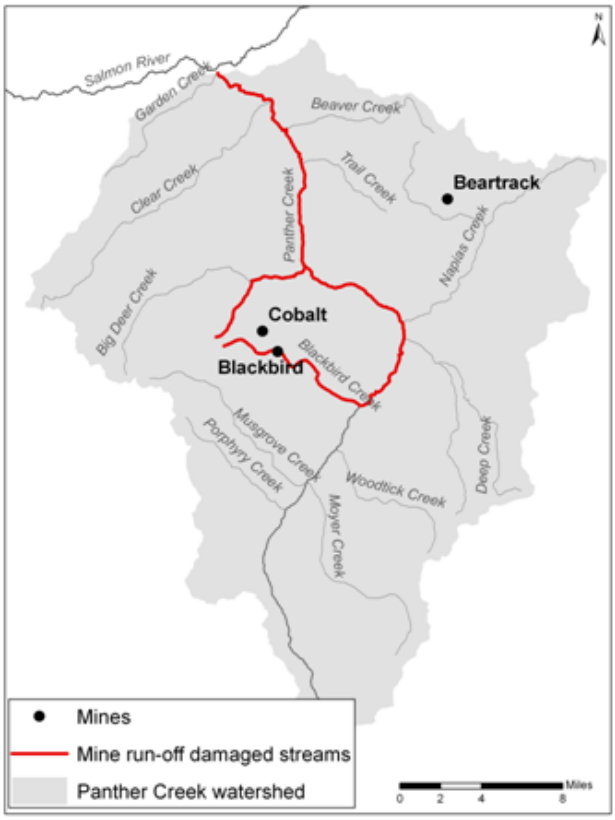
Within the Snake River spring/summer Chinook salmon ESU, independent populations have been grouped into larger aggregates (MPGs) that share similar genetic, geographic, and/or habitat characteristics. This ESU was broken down into five MPGs with 28 extant independent populations and four extirpated or functionally extirpated independent populations (Ford 2011;

ICTRT 2003); McClure *et al*. 2005). Only three of the MPGs (i.e., South Fork Salmon, Middle Fork Salmon, and Upper Salmon) are within the action area. There are 22 independent populations within these three MPGs, one of which (Panther Creek) is considered extirpated by the ICTRT (2003)

In 2005, the ICTRT concluded that the Panther Creek Chinook salmon population was extirpated during the 1960s due to legacy mining and the heavy metal wastes deposited in Lower Panther Creek from the Blackbird Mine operations (ICTRT 2005). The loss of habitat in Panther Creek resulting from water quality degradation from the Blackbird Mine was specifically cited as a contributing factor leading to the decline and subsequent ESA-listing of the Snake River spring/summer Chinook salmon species (NMFS 1991). Once a sizable population, spring/summer Chinook salmon runs declined during the 1940s when extensive mining activity began in the Blackbird Creek Drainage, and was eliminated by the early 1960s. At the time that spring/summer Chinook salmon were being considered for listing under the ESA, the Panther Creek drainage remained largely uninhabitable due to toxic conditions resulting from mine drainage (NMFS 1991).

Figure 2.2.1. Boundaries of listed anadromous species and the action area (State of Idaho within the range of anadromous species). "Species" are defined as "evolutionarily significant units" or ESUs. The Panther Creek watershed is emphasized because the extirpation of the Panther Creek Chinook salmon population due poor water quality was considered a specific factor for the decline of and ESA listing of the Snake River spring/summer Chinook salmon ESU. Contemporary water quality and biological conditions in Panther Creek are described in the Status of Species section and in the Analyses of Effects sections for arsenic and copper.





Recovery has been slow. Poor water quality, primarily copper contamination, precluded recolonization through the 1990s, despite supplementation efforts including the release of about 3,383 adult Chinook salmon in 1986. Two Chinook salmon redds each were observed downstream of the Blackbird Mine again in Panther Creek in 1990,1991, and 1992 (Mebane 1994) although no adult or juvenile Chinook salmon could be found despite extensive surveys in 1993 (LeJeune *et al*. 1995). By the early 2000s, extensive mine remediation efforts began to succeed with greater than 90% reductions in copper concentrations in Panther Creek (described more in the Section 2.4.4, Copper)

In the 2000s, Chinook salmon began returning to Panther Creek following improvements in water quality in Panther Creek. The returns and successful reproduction resulted both from natural recolonization and from reproduction following a large release of adult Chinook salmon in 2001. In 2001, as part of "an effort to increase natural production in areas with depressed populations," 1,053 adult Chinook salmon captured from South Fork Salmon River weir were released into Panther Creek (Leth *et al*. 2004). In the fall of 2001, 42 redds were counted and in 2010, 102 redds were counted, both counts were from ground surveys of Panther Creek conducted by the Shoshone Bannock Tribes (EcoMetrix 2011). Aerial counts of Chinook salmon in Panther Creek conducted by the IDFG, which will be lower than ground surveys (e.g., 15 vs. 42 in 2001), ranged from five to 18 from 2001 to 2009 (Figure 2.2.1).

Juvenile Chinook salmon have been found throughout the middle reaches (i.e., downstream of mining influenced Blackbird Creek) and upper reaches of Panther Creek in annual quantitative electrofishing surveys from 2002 through 2010 (Figure 2.2.1). The highest densities were found in 2002, following the large release of adults the previous summer. Peaks in densities in upper reaches of Panther Creek in 2006 (5 years post spawning) and in the middle reaches of Panther Creek in 2005 (4 years after spawning) are consistent with general patterns with inland Chinook populations as well as specific patterns found in the Salmon River drainage, where higher elevation, headwater populations with longer migrations tended to have greater proportions of fish with a 5-year life cycle compared to lower elevation populations where 4-year life cycles are more common with Chinook salmon (Healey 1991; Mebane and Arthaud 2010). This life history pattern, together with the patterns of declining peak densities of juvenile Chinook salmon in the middle reach of Panther Creek, (fish that presumably have a 4-year life cycle) suggests that the juvenile Chinook salmon abundance may be in decline in the middle sections of Panther Creek downstream of Blackbird Creek (Figure 2.2.2). However, no declines are obvious in the Chinook salmon densities in upper Panther Creek, and fish populations may be extremely variable, and with short periods of record, a trend that is apparent 1 year may be gone when the next year's data are added.

Not all the Chinook salmon recently observed or captured in Panther Creek can be attributed to the 2001 release of fish from the South Fork Salmon River. Adult Chinook salmon that were observed in Panther Creek in 2002 and 2004, and young-of-year (YOY) (subyearling) Chinook salmon captured in Panther Creek in 2003 and 2004 cannot be attributed to the artificial release of adult fish in 2001 (Stantec 2004; EcoMetrix 2005). However by 2010, the great majority of the Chinook salmon that continue to return and naturally reproduce in Panther Creek are likely descendants of the 2001 South Fork Salmon River fish (Smith *et al*. 2011).

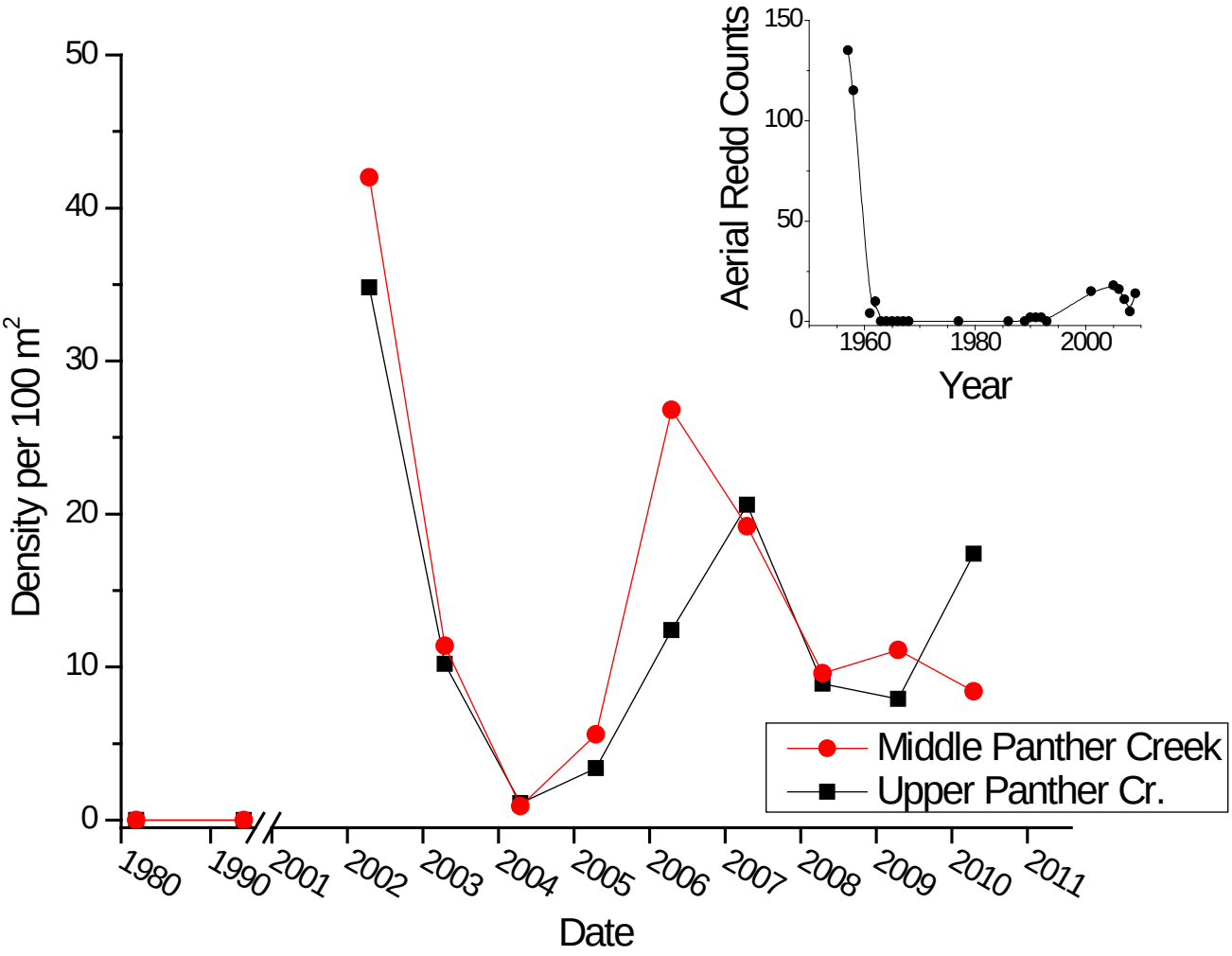


Figure 2.2.2. Juvenile Chinook salmon abundance in Panther Creek, Idaho, from electrofishing surveys. "Middle" or "Upper" Panther Creek are downstream and upstream of mining influenced Blackbird Creek, respectively. Inset shows trends in aerial redd counts from approximately the same IDFG trend sections. Data from 1992 and earlier were taken from Mebane (1994), 1993 data from LeJeune *et al.* (1995), and subsequent data are from EcoMetrix (2011).

Under the approach recommended by the ICTRT, the overall rating for an ESU depends upon population level ratings organized by MPG within that ESU (2007). In order for the Snake River spring/summer Chinook salmon ESU to be considered viable, all five MPGs need to achieve viable status. The overall viability ratings for all of the populations in this ESU remain at High risk after the addition of more recent year abundance and productivity data (Ford 2011). Table 2.2.3 summarizes the viability ratings for each population and the overall viability status for each MPG that occurs within the action area. Currently, all of the populations have an overall viability rating of "high risk," and none of the MPGs meet MPG viability criteria (Ford 2011). As such, this ESU does not meet ESU viability criteria (non-negligible risk of extinction over a 100-year time period).

Relatively low natural production rates and spawning levels below minimum abundance thresholds remain a major concern across this ESU. The ability of populations to be self-sustaining through normal periods of relatively low ocean survival remains uncertain. Factors such as habitat modification/degradation, artificial propagation, disease, or predation (NMFS 2011) remain as concerns or key uncertainties for this ESU.

Detailed information on the range wide status of Snake River spring/summer Chinook salmon under the environmental baseline is described in Chinook salmon status reviews (Myers *et al.* 1998; Good *et al*. 2005; Ford *et al*. 2011).

Table 2.2.3. Summary of VSP parameter risks and viability status for Snake River Spring/Summer Chinook Salmon MPGs and independent populations (Ford 2011; NMFS 2011).

MPG	Population Name	Pop. Size & Complexity	VSP Parameter Risk		Viability Status (Meets Viability Criteria?)	
			A/P	SS/D	Population	MPG
South Fork Salmon River	Little Salmon River	Intermediate	High	High	Does Not Meet	Does Not Meet
	South Fork Salmon River mainstem	Large	High	Moderate	Does Not Meet	
	Secesh River	Intermediate	High	Low	Does Not Meet	
	East Fork South Fork Salmon River	Large	High	Low	Does Not Meet	
Middle Fork Salmon River	Chamberlain Creek	Basic	High	Low	Does Not Meet	Does Not Meet
	Middle Fork Salmon River below Indian Creek	Basic	High	Moderate	Does Not Meet	
	Big Creek	Large	High	Moderate	Does Not Meet	
	Camas Creek	Basic	High	Moderate	Does Not Meet	
	Loon Creek	Basic	High	Moderate	Does Not Meet	
	Middle Fork Salmon River above Indian Creek	Intermediate	High	Moderate	Does Not Meet	
	Sulphur Creek	Basic	High	Moderate	Does Not Meet	
	Bear Valley Creek	Intermediate	High	Low	Does Not Meet	
	Marsh Creek	Basic	High	Low	Does Not Meet	
Upper Salmon River	North Fork Salmon River	Basic	High	Low	Does Not Meet	Does Not Meet
	Lemhi River	Very Large	High	High	Does Not Meet	
	Salmon River Lower Mainstem	Very Large	High	Low	Does Not Meet	
	Pahsimeroi River	Large	High	High	Does Not Meet	
	East Fork Salmon River	Large	High	High	Does Not Meet	
	Yankee Fork Salmon River	Basic	High	High	Does Not Meet	
	Valley Creek	Basic	High	Moderate	Does Not Meet	
	Salmon River Upper Mainstem	Large	High	Moderate	Does Not Meet	
	Panther Creek	Intermediate	N/A	N/A	Extirpated	
Grande Ronde Imnaha	Wenaha River	Intermediate	High	Moderate	Does Not Meet	Does Not Meet
	Minam River	Intermediate	High	Moderate	Does Not Meet	
	Catherine Creek	Large	High	Moderate	Does Not Meet	
	Lostine/Wallowa Rivers	Large	High	Moderate	Does Not Meet	
	Upper Grande Ronde River	Large	High	High	Does Not Meet	
	Imnaha River	Intermediate	High	Moderate	Does Not Meet	
Lower Snake	Tucannon River	Intermediate	High	Moderate	Does Not Meet	Does Not Meet

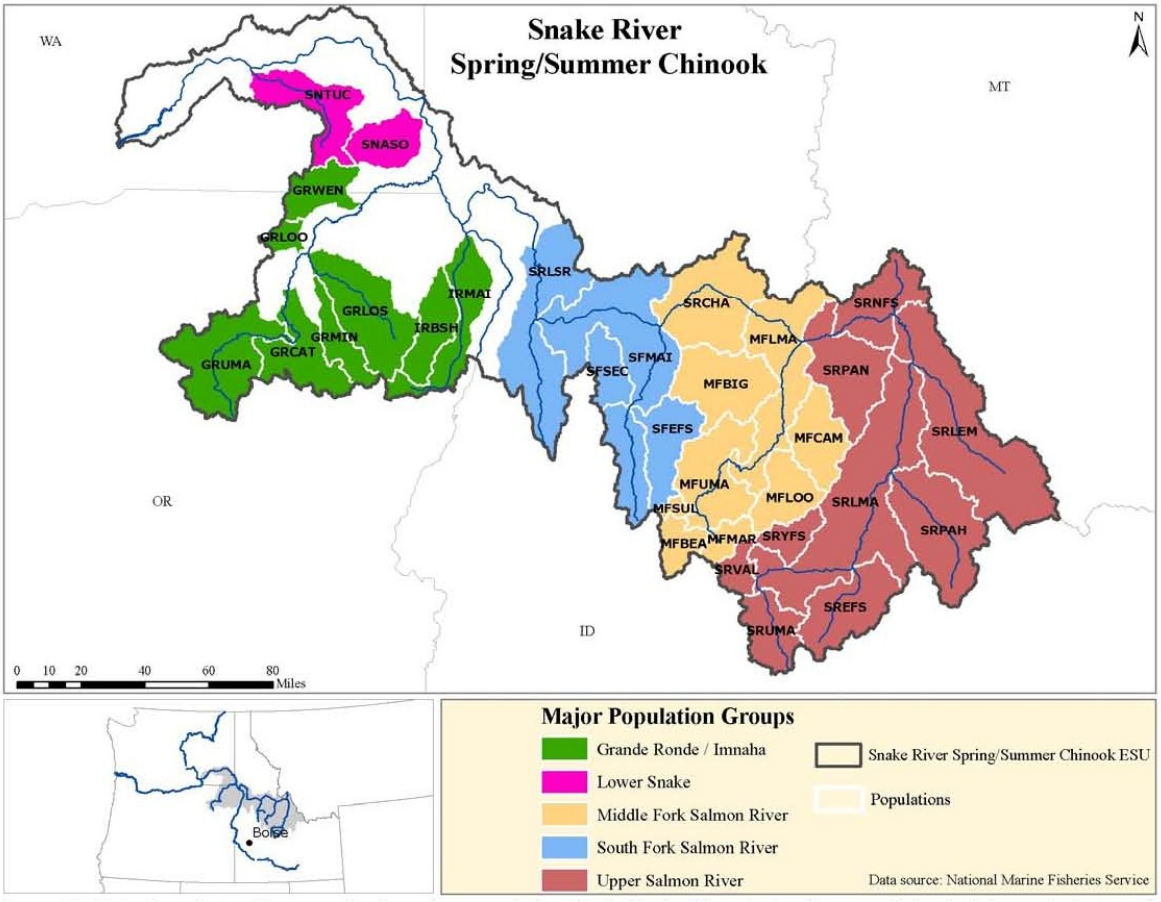


Figure 2.2.3. Major population groups and independent populations of Snake River spring/summer Chinook salmon. The populations codes are contracted from the above table, for example SRUMA=upper Salmon River, Salmon River mainstem

2.2.1.3. Snake River Fall Chinook Salmon

The Snake River fall Chinook salmon ESU was listed as threatened on April 22, 1992 (57 FR 14653). This ESU includes all natural-origin populations in the mainstem Snake River below Hells Canyon Dam, and the Tucannon, Grande Ronde, Imnaha, Salmon, and Clearwater Rivers. The ESU also includes four artificial propagation programs: the Lyons Ferry Hatchery; fall Chinook acclimation ponds program, Nez Perce Tribal Hatchery, and Oxbow Hatchery (70 FR 37160; June 28, 2005). On August 15, 2011, NMFS completed a 5-year review for the Snake River fall Chinook salmon ESU and concluded that the species should remain listed as threatened (76 FR 50448).

Fall Chinook salmon in the Columbia River generally exhibit an ocean-type life history. In general, fall Chinook salmon are larger than stream-type Chinook salmon and spawn in larger, mainstem rivers and the lower sections of larger tributaries. Adult Snake River fall Chinook salmon return when they are between 2 and 5 years of age, with 4 years being the most common. Adults typically return to fresh water beginning in July, migrate past the lower Snake River mainstem dams from August through November, and spawn from October through early

December. Juveniles emerge from the gravels in March and April of the following year. Parr undergo a smolt transformation usually as subyearlings in the spring and summer at which time they migrate to the ocean. However, in recent years, in both the upper Columbia River basin and in the Snake River basin, some ocean-type Chinook salmon have been utilizing the reservoirs upstream of the mainstem dams and migrating as yearlings the following year. Subadults and adults forage in coastal and offshore waters of the North Pacific Ocean prior to returning to spawn in their natal streams.

Historically, fall Chinook salmon were widely distributed throughout the Snake River and many of its major tributaries from its confluence with the Columbia River upstream to Shoshone Falls, Idaho (Fulton 1968). Prior to the 1960s, the Snake River was considered the most important drainage in the Columbia River system for the production of anadromous fishes. The majority of historic Snake River fall Chinook salmon production was centered on the middle and upper mainstem Snake River in island/channel habitats. This portion of the Snake River represented approximately 85% of the historically available habitat for this ESU (NMFS 2010a).

Construction of the Swan Falls Dam in 1901 and the Hells Canyon Dam complex between 1956 and 1968 eliminated access to this habitat, reducing the distribution of fall Chinook salmon to mostly remnant areas in the Snake River basin with lower natural production potential than the habitats available in their former range (Connor *et al*. 2002; Dauble *et al*. 2003). Within Idaho, the current distribution of fall Chinook salmon is located in the Snake River below Hells Canyon Dam; along the lower/middle main Salmon River, from the mouth upstream to approximately its confluence with French Creek; and the lower reaches of the Clearwater River.

Historical abundance of Snake River fall Chinook salmon prior to 1938 is not known. The estimated annual return for the period 1938 to 1949 was 72,000 fish and had declined to an annual average of 29,000 fish by the 1950s (Bjornn and Horner 1980). Numbers of fall Chinook salmon continued to decline during the 1960s and 1970s with the construction of numerous dams in the Snake River. Counts of returning natural-origin fall Chinook salmon at LGD from 1975 through 1980 averaged 610 fish per year (Waples *et al*. 1991b). The first hatchery-reared Snake River fall Chinook salmon returned to the Snake River in 1981 (Busack 1991), and since then, adult counts represent a mixture of hatchery and natural production. Since 1983, about 20% to 80% of the total fall Chinook salmon reaching the LGD each year is estimated to have been of hatchery origin (Waples *et al*. 1991b).

Counts of natural-origin[2] adult Snake River fall Chinook salmon at LGD were 1,000 fish in 1975 and declined to an annual low of 78 adults in 1990 (Good *et al*. 2005). Numbers of natural-origin Snake River fall Chinook salmon began to increase after 1990, with a 5-year geometric mean for 1997 to 2001 of 871 natural-origin fish (Good *et al*. 2005). The total spawning escapement over LGD has remained relatively high since the rapid increase in the late 1990s. The current 5-year geometric mean (2003 to 2008) of natural-origin fish is 2,291, which is substantially more than the previous estimate. When considering hatchery-origin fish, the 5-year geometric mean of total adult returns for that same time period exceeded 11,000 (Ford 2011). Clearly, hatchery supplementation continues to play a significant role in the overall abundance of fish, accounting for approximately 78% of the returns during 2003 to 2008 cycle.

[2] Adult fish produced from naturally spawning parents (regardless of the origin of the parents).

There is only one extant[3] population in the Snake River fall Chinook salmon ESU, the Lower Snake River Mainstem population. This population occupies the Snake River from its confluence with the Columbia River to Hells Canyon Dam, and the lower reaches of the Clearwater, Imnaha, Grande Ronde, Salmon, and Tucannon Rivers. The majority of the fish spawn in the mainstem Snake River between the head of Lower Granite Reservoir (River Mile [RM] 146.8) and Hells Canyon Dam (RM 247.6), with the remaining fish distributed among lower sections of the major tributaries. Fall Chinook salmon in the mainstem Snake River appear to be distributed in a series of aggregates from the mouth of Asotin Creek to RM 219, although smaller numbers have been reported spawning in the tailraces of the lower Snake dams. Due to their proximity and the likelihood that individual tributaries did not support separate populations of sufficient size to be self-sustaining, the ICTRT considered these aggregates and the fish in the lower portions of major tributaries to the Snake River to be a single population (McClure *et al*. 2005).

Because there is only one extant population of Snake River fall Chinook salmon, ICTRT criteria indicate that this population should be "Highly Viable" to achieve recovery of this ESU (ICTRT 2007). To be "Highly Viable" under the VSP guidelines, this population must have: (1) A combination of abundance and productivity that create a *very low* risk of extinction (<1% over a 100-year period); and (2) spatial structure and genetic/phenotypic diversity that have no more than a *low* risk of not maintaining key components of spatial structure and diversity described by the ICTRT.

The single extant population of Snake River fall Chinook salmon, the Lower Snake River Mainstem population, is currently not viable. Based upon productivity and escapement estimates, the abundance/productivity metric risk rating is moderate. Similarly, based upon spawner distribution and hatchery composition data, the spatial structure/diversity risk rating is moderate. As such, the overall viability rating for this population is "maintained." To meet the criteria for Highly Viable, the abundance/productivity levels and spatial structure/diversity risk ratings would need to improve.

Detailed information on the range-wide status of Snake River spring/summer Chinook salmon under the environmental baseline is described in Chinook salmon status reviews (Myers *et al*. 1998; Good *et al*. 2005; Ford 2011).

2.2.1.4. Snake River Basin Steelhead

The Snake River Basin steelhead was listed as a threatened ESU on August 18, 1997 (62 FR 43937), with a revised listing as a distinct population segment (DPS) on January 5, 2006 (71 FR 834). The listed DPS includes all natural-origin populations of anadromous steelhead in the Snake River basin downstream from long-standing barriers in southeast Washington, northeast Oregon, and Idaho. The DPS also includes six artificial production programs: Tucannon River, Dworshak National Fish Hatchery, Lolo Creek, North Fork Clearwater, East Fork Salmon River,

[3] The ICTRT also designated two populations of Snake River fall Chinook salmon that are not extant: the Marsing Reach population and the Salmon Falls population (ICTRT May 11, 2005, memorandum regarding updated population delineation in the Interior Columbia Basin).

and the Little Sheep Creek/Imnaha River Hatchery. The Snake River Basin steelhead listing does not include resident forms of *O. mykiss* (rainbow trout) co-occurring with these steelhead.

Steelhead are anadromous fish that spawn in freshwater streams and mature in the ocean. Adult Snake River Basin steelhead return to the Snake River basin from late summer through fall, where they hold in larger rivers for several months before moving upstream into smaller tributaries. Adult dispersal toward spawning areas varies with elevation, with the majority of adults dispersing into tributaries from March through May; earlier dispersal occurs at lower elevations and later dispersal occurs at higher elevations. Spawning begins shortly after fish reach spawning areas, which is typically during a rising hydrograph and prior to peak flows (Thurow 1987). Steelhead generally select spawning areas at the downstream end of pools, in gravels ranging in size from 0.5 to 4.5 inches in diameter (Pauley *et al.* 1986). Juveniles emerge from the gravels in 4 to 8 weeks, depending on temperature. After emergence, fry have poor swimming ability. Steelhead fry initially move from the gravels into shallow, low-velocity areas in side channels and along channel margins to escape high velocities and predators (Everest and Chapman 1972), and progressively move toward deeper water as they grow in size (Bjornn and Rieser 1991). Juveniles typically reside in fresh water for 2 to 3 years (Behnke and Tomelleri 2002). Smolts migrate downstream during spring runoff, which occurs from March to mid-June depending on elevation.

Anadromous Snake River Basin steelhead exhibit two distinct morphological forms, identified as "A-run" and "B-run" fish, which are distinguished by differences in body size, run timing, and length of ocean residence. B-run fish predominantly reside in the ocean for 2 years, while A-run steelhead typically reside in the ocean for 1 year. As a result of differences in ocean residence time, B-run steelhead are typically larger than A-run fish. The smaller size of A-run adults allows them to spawn in smaller headwater streams and tributaries. The differences in the two fish stocks represent an important component of phenotypic and genetic diversity of the Snake River Basin steelhead DPS through the asynchronous timing of ocean residence, segregation of spawning in larger and smaller streams, and possible differences in the habitats of the fish in the ocean.

Although direct historical estimates of production from the Snake River basin are not available, the basin is believed to have produced more than half of the steelhead in the CRB (Mallet 1974). There are some historical estimates of returns to portions of the drainage. Returns to the Clearwater, Grande Ronde, Imnaha, and Tucannon Rivers may have reached or exceeded 62,000 to 82,000 fish in the mid-1950s to early 1960s (Cichosz *et al.* 2003; ODFW 1991; Thompson *et al.* 1958). The Salmon River basin likely supported substantial production as well (Good *et al.* 2005). The longest, consistent indicator of steelhead abundance in the Snake River basin is derived from counts of natural-origin steelhead at the uppermost dam on the LGD. According to these estimates, the abundance of natural-origin steelhead at the uppermost dam on the Snake River has declined from a 4-year average of 58,300 in 1964 to a 4-year average of 8,300 ending in 1998. In general, steelhead abundance declined sharply in the early 1970s, rebuilt modestly from the mid-1970s through the 1980s, and declined again during the 1990s. The 2001 and 2002 total and natural-origin returns of steelhead over LGD (average 240,643 and 52,503, respectively) were substantially higher relative to the low levels seen in the 1990s. The rolling 5-year average abundance of natural-origin returns has generally increased from 2000 (12,090

fish between 1996 and 2000) to 2010 (48,740 fish between 2006 and 2010). Although steelhead numbers have dramatically increased, natural-origin steelhead comprise only 10% to 30% of the total returns since 1994 (FPC 2011c).

The ICTRT identified 29 independent populations (excluding the historically occupied but currently inaccessible habitats upstream of the Hells Canyon Dam complex) in the Snake River Basin steelhead DPS, grouped into six MPGs (McClure *et al*. 2005). Fish from all of these MPGs are found at one time or another migrating through Idaho waters, but only three of the MPGs (i.e., Clearwater River, Salmon River, and Hells Canyon) are located in Idaho. There are 22 independent populations within these three MPGs, of which three are extirpated, one is functionally extirpated, and one (North Fork Clearwater) is blocked from its historic habitat (Table 2.2.4). The three MPGs outside Idaho are Lower Snake MPG (Tucannon River population and Asotin Creek population), the Grande Ronde MPG (Upper and Lower Grande Populations, Joseph Creek population and Wallowa River population) and the Imnaha River MPG (Imnaha River population).

Under the approach recommended by the ICTRT, the overall rating for an ESU depends upon population level ratings organized by MPG within that ESU (2007). In order for the Snake River Basin steelhead DPS to be considered viable, the Clearwater and Salmon MPGs need to achieve viable status. Table 2.2.4 summarizes the viability ratings for each population and the overall viability status for each MPG that occurs within the action area (Ford 2011; NMFS 2011). Currently, none of the MPGs meet MPG viability criteria. As such, this DPS does not meet DPS-level viability criteria (non-negligible risk of extinction over a 100-year time period).

Although recent increases in fish abundances are encouraging, population-level natural-origin abundance and productivity inferred from aggregate data and juvenile indices indicate that many populations in the ESU are likely below the minimum combinations defined by the ICTRT viability criteria. A great deal of uncertainty remains regarding the relative proportion of hatchery fish in natural spawning areas near hatchery release sites. Furthermore, the natural-origin abundance and productivity are still below their targets (Ford 2011).

Detailed information on the range wide status of Snake River Basin steelhead under the environmental baseline is described in status reviews (Myers *et al*. 1998; Good *et al*. 2005; Ford 2011).

Table 2.2.4. Summary of VSP parameter risks and viability status for Snake River Basin Steelhead MPGs and independent populations (Ford 2011; NMFS 2011).

MPG	Population Name	Population Size & Complexity	VSP Parameter Risk		Status (Meets viability Criteria?)	
			A/P	SS/D	Population	MPG
Clearwater River	Lower Mainstem	Large	Moderate	Low	Does Not Meet	Does Not Meet
	North Fork		Blocked	Blocked	Extirpated	
	Lolo Creek	Basic	High	Moderate	Does Not Meet	
	Lochsa River	Intermediate	High	Low	Does Not Meet	
	Selway River	Intermediate	High	Low	Does Not Meet	
	South Fork	Intermediate	High	Moderate	Does Not Meet	
Salmon River	Little Salmon River	Basic	Moderate	Moderate	Does Not Meet	Does Not Meet
	Secesh River	Basic	High	Low	Does Not Meet	
	South Fork Salmon	Intermediate	High	Low	Does Not Meet	
	Chamberlain Creek	Basic	Moderate	Low	Does Not Meet	
	Lower Middle Fork	Intermediate	High	Low	Does Not Meet	
	Upper Middle Fork	Intermediate	High	Low	Does Not Meet	
	Panther Creek	Basic	Moderate	High	Does Not Meet	
	North Fork Salmon	Basic	Moderate	Moderate	Does Not Meet	
	Lemhi River	Intermediate	Moderate	Moderate	Does Not Meet	
	Pahsimeroi River	Intermediate	Moderate	Moderate	Does Not Meet	
	East Fork Salmon	Intermediate	Moderate	Moderate	Does Not Meet	
	Upper Salmon Mainstem	Intermediate	Moderate	Moderate	Does Not Meet	
Grande Ronde	Upper Grande Ronde	Large	Moderate	Moderate	Does Not Meet	Does Not Meet
	Lower Grande Ronde	Intermediate	N/A	Moderate	Does Not Meet	
	Joseph Creek	Basic	Very Low	Low	Meets	
	Wallowa River	Intermediate	High	Low	Does Not Meet	
Imnaha	Imnaha	Intermediate	Moderate	Moderate	Doe Not Meet	Does Not Meet
Lower Snake River	Tucannon	Intermediate	High	Moderate	Does Not Meet	Does Not Meet
	Asotin	Basic	Moderate	Moderate	Does Not Meet	

2.2.2. Status of Critical Habitat

NMFS reviews the status of designated critical habitat affected by the proposed action by examining the condition and trends of essential features for Chinook salmon or PCEs for steelhead throughout the designated area (hereinafter referred to PCEs). The PCEs consist of the physical and biological features identified as essential to the conservation of the listed species because they support one or more of the of the species' life stages (e.g., sites with conditions that support spawning, rearing, migration and foraging).

The ESA-listed species addressed in this Opinion occupy many of the same geographic areas and have similar life history characteristics. The PCEs or essential physical and biological features are also similar and are referred to jointly as PCEs (Table 2.2.5). In general, these PCEs include sites essential to support one or more life stages of the ESA-listed species (i.e., sites for spawning, rearing, migration, and foraging) and contain physical or biological features essential to the conservation of the listed species (e.g., spawning gravels, water quality and quantity, side channels, or food). The PCEs associated with the freshwater spawning, rearing and migratory

sites potentially affected by this action include water quality, forage/food, and access/safe passage.

Table 2.2.5. Types of sites and essential physical and biological features designated as PCEs, and the species life stage each PCE supports.

Essential Physical and Biological Features		ESA-listed Species Life Stage
Snake River Basin Steelhead[a]		
Freshwater spawning	Water quality, water quantity, and substrate	Spawning, incubation, and larval development
Freshwater rearing	Water quantity & floodplain connectivity to form and maintain physical habitat conditions	Juvenile growth and mobility
	Water quality and forage[b]	Juvenile development
	Natural cover[c]	Juvenile mobility and survival
Freshwater migration	Free of artificial obstructions, water quality and quantity, and natural cover[c]	Juvenile and adult mobility and survival
Snake River Spring/summer and Fall Chinook Salmon		
Spawning and Juvenile Rearing	Spawning gravel, water quality and quantity, cover/shelter, food, riparian vegetation, and space	Juvenile and adult.
Migration	Substrate, water quality and quantity, water temperature, water velocity, cover/shelter, food[d], riparian vegetation, space, safe passage	Juvenile and adult.
Snake River Sockeye Salmon		
Spawning and Juvenile Rearing	Spawning gravel, water quality and quantity, water temperature, food, riparian vegetation, and access	Juvenile and adult.
Migration	Substrate, water quality and quantity, water temperature, water velocity, cover/shelter, food[d], riparian vegetation, space, safe passage	Juvenile and adult.

a. Additional PCEs pertaining to estuarine, nearshore, and offshore marine areas have also been described for Snake River Basin steelhead. These PCEs will not be affected by the proposed action and have therefore not been described in this Opinion.
b. Forage includes aquatic invertebrate and fish prey that support growth and maturation.
c. Natural cover includes shade, large wood, log jams, beaver dams, aquatic vegetation, large rocks and boulders, side channels, and undercut banks.
d. Food applies to juvenile migration only.

Table 2.2.6 provides a brief description of the designated critical habitat for the four ESA-listed species considered in this Opinion. Critical habitat includes the stream channel and water column with the lateral extent defined by the ordinary high-water line, or the bankfull elevation where the ordinary high-water line is not defined. In addition, critical habitat for the three salmon species includes the adjacent riparian zone, which is defined as the area within 300 feet of the line of high water of a stream channel or from the shoreline of standing body of water (58 FR 68543; December 28, 1993). The riparian zone is critical because it provides shade; streambank stability; organic matter input; and sediment, nutrient, and chemical regulation.

Table 2.2.6. Description of designated critical habitat for ESA-listed species considered in this Opinion.

ESU/DPS	Designation	Description of Critical Habitat in Idaho
Snake River sockeye salmon	58 FR 68543; December 28, 1993	Snake and Salmon Rivers; Alturas Lake Creek; Valley Creek, Stanley Lake, Redfish Lake, Yellowbelly Lake, Pettit Lake, Alturas Lake; all inlet/outlet creeks to those lakes
Snake River spring/summer Chinook salmon	58 FR 68543; December 28, 1993 64 FR 57399; October 25, 1999	All river reaches presently or historically accessible, except river reaches above impassable natural falls and Dworshak and Hells Canyon Dams
Snake River fall Chinook salmon	58 FR 68543; December 28, 1993	Snake River from state line to Hells Canyon Dam, Clearwater River from its confluence with the Snake River upstream to Lolo Creek, North Fork Clearwater River from its confluence with the Clearwater River upstream to Dworshak Dam, all other river reaches presently or historically accessible within the Clearwater, Lower Clearwater, Lower Snake Asotin, Hells Canyon and Lower Salmon subbasins
Snake River Basin steelhead	70 FR 52630; September 2, 2005	Specific stream reaches are designated within the Snake, Salmon, and Clearwater basins. Table 21 in the Federal Register details habitat areas within the ESU's geographical range that are excluded from critical habitat designation.

During all life stages, salmon and steelhead require cool water that is relatively free of contaminants. From a water quality perspective, cool, clean water ensures there is adequate passage conditions for these species to access various habitats required to complete their life cycle. It also contributes to the establishment and maintenance of a healthy, properly functioning ecosystem for prey communities upon which salmon can forage. Water quality degradation within the action area can influence survival and productivity of salmon and steelhead (Regetz 2003).

The PCE for necessary water quality in critical habitats is considered to include the following features. Waters in critical habitats need to be free from substances in concentrations that could cause effects that directly or indirectly, could interfere with important life histories of anadromous salmonids. Potential adverse effects of concern from toxic chemicals include biologically important behaviors and physiological effects to chemoreception, homing, orientation and rheotaxis, downstream migrations, predator avoidance, prey capture, avoidance of habitats or loss of avoidance ability, swimming speed or endurance, altered social status (e.g., dominance and competitive interactions), feeding efficiency, food conversion or growth effects, reproductive impairment, or death, whether resulting from direct exposure or secondary to intermediate effects. The "water quality" PCE also implies waters need to be free from other indirect effects such as effects to invertebrate communities that serve as the prey base for

juvenile salmonids, reduced invertebrate diversities, or reduced abundances of preferred prey. Because there are interchanges between the water column and sediments in aquatic habitats, because benthic macroinvertebrate prey are closely linked to sediments, sediments also need to be free from toxic chemicals in concentrations that could cause adverse effects.

Snake River spring/summer Chinook salmon designated critical habitat in the Snake and Columbia Rivers have been altered by: (1) Operation of dams upstream from the migration corridor for water storage and flood control; (2) water diversion for irrigation upstream from the migration corridor; (3) construction of dams, reservoirs, and a navigation channel within the migration corridor; and (4) operation of dams and reservoirs for power generation, flood control, water storage, and navigation within the migration corridor. Use of water, primarily for irrigation, has greatly reduced water quantity available for rearing and migration and construction and operation of storage and flood control reservoirs has further reduced water quantity during spring when juvenile Chinook salmon migrate downstream through the Snake and Columbia Rivers. The eight mainstem dams and their associated reservoirs along the migration route have greatly reduced water velocity and have increased habitat for native and introduced predators, such as pikeminnow, smallmouth bass, and channel catfish. The eight mainstem dams also constitute physical barriers that can substantially decrease migration survival. Impounding water for storage, flood control, and navigation may also increase summer water temperatures, which could adversely affect late migrating juvenile and adult Chinook salmon (NMFS 2014).

Designated critical habitat in the Salmon River drainage has not been affected by mainstem dams and large storage reservoirs, so it is somewhat less altered than habitat in the Snake and Columbia Rivers, but it has been affected by extensive water use, mining, construction and maintenance of water diversion structures, construction and maintenance of roads, conversion of wetlands into agriculture land, and by livestock grazing. Amount of development and condition of habitat varies greatly within the Salmon River drainage. Most of the development, and consequent adverse impacts on habitat, have occurred upstream from the confluence of the Middle Fork Salmon and main Salmon Rivers (RM 199) and within the Little Salmon River drainage. For example: There are approximately 154,000 acres of irrigated agriculture in the Salmon River drainage, the impacts of which deplete flows in the Little Salmon River, North Fork Salmon River, Lemhi River, Pahsimeroi River, portions of the mainstem Salmon River, and numerous smaller Salmon River tributaries; past mining activities have devastated habitat in portions of the Yankee Fork Salmon River drainage and Panther Creek drainages; livestock grazing may also impact riparian habitat throughout this area; and impacts of small cities and towns, which are primarily located on waterways, have cause localized impacts on riparian and instream habitat. In contrast, the Middle Fork Salmon River drainage, large portions of the South Fork Salmon River drainage, and the Chamberlin Creek drainage are largely undeveloped and contain some of the most unimpaired salmonid habitat in the contiguous United States (NMFS 2011).

Spawning and rearing habitat quality in tributary streams in the Snake River varies from excellent in wilderness and roadless areas to poor in areas subject to intensive human land uses (NMFS 2011). Critical habitat throughout much of the Snake River basin has been degraded by intensive agriculture, alteration of stream morphology (*i.e.*, channel modifications and diking),

riparian vegetation disturbance, wetland draining and conversion, livestock grazing, dredging, road construction and maintenance, logging, mining, and urbanization. Reduced summer streamflows, impaired water quality, and reduction of habitat complexity are common problems for critical habitat in non-wilderness areas. Human land use practices throughout the basin have caused streams to become straighter, wider, and shallower, thereby reducing rearing habitat and increasing water temperature fluctuations.

In many stream reaches designated as critical habitat in the Snake River basin, streamflows are substantially reduced by water diversions (NMFS 2011). Withdrawal of water, particularly during low-flow periods that commonly overlap with agricultural withdrawals, often increases summer stream temperatures, blocks fish migration, strands fish, and alters sediment transport (Spence *et al.* 1996). Reduced tributary streamflow has been identified as a major limiting factor for Snake River spring/summer Chinook and Snake River Basin steelhead in particular (NMFS 2011).

Many stream reaches designated as critical habitat are listed on the state of Idaho's CWA section 303(d) list for impaired water quality, such as elevated water temperature (IDEQ 2010). Some areas that were historically suitable rearing and spawning habitat are now unsuitable due to high summer stream temperatures. Removal of riparian vegetation, alteration of natural stream morphology, and withdrawal of water for agricultural or municipal use all contribute to elevated stream temperatures (Poole *et al.* 2001; Arthaud *et al.* 2010). Water quality in spawning and rearing areas has also been impaired by high levels of sedimentation and by heavy metal contamination from mine waste (e.g., Nelson *et al*. 1991).

Migration habitat quality for Snake River salmon and steelhead has also been severely degraded, primarily by the development and operation of dams and reservoirs on the mainstem Columbia and Snake Rivers (Ford 2011). Hydroelectric development has modified natural flow regimes in the migration corridor—causing in higher water temperatures and changes in fish community structure that have led 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.

2.2.3. Climate Change

Climate change is likely to have negative implications for the conservation value of designated critical habitats in the Pacific Northwest (CIG 2004; Scheuerell and Williams 2005; Zabel *et al*. 2006; Independent Scientific Advisory Board [ISAB] 2007). Average annual Northwest air temperatures have increased by approximately 1ºC since 1900, or about 50% more than the global average warming over the same period (ISAB 2007). The latest climate models project a warming of 0.1ºC to 0.6ºC per decade over the next century. According to the ISAB, these effects may have the following physical impacts within the next 40 or so years:

- Warmer air temperatures will result in a shift to more winter/spring rain and runoff, rather than snow that is stored until the spring/summer melt season.

- With a shift to more rain and less snow, the snowpacks will diminish in those areas that typically accumulate and store water until the spring freshet.

- With a smaller snowpack, these watersheds will see their runoff diminished and exhausted earlier in the season, resulting in lower streamflows in the June through September period.

- River flows in general and peak river flows are likely to increase during the winter due to more precipitation falling as rain rather than snow.

- Water temperatures will continue to rise, especially during the summer months when lower streamflow and warmer air temperatures will contribute to the warming regional waters.

These changes will not be spatially homogenous. 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 and contribute little to total streamflow are likely to be more affected. These long-term effects may include, but are not limited to, depletion of cold water habitat, variation in quality and quantity of tributary rearing habitat, alterations to migration patterns, accelerated embryo development, premature emergence of fry, and increased competition among species.

2.3. 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 general, the environment for ESA-listed species has been dramatically affected by the development and operation of the Federal Columbia River Power System (FCRPS). Storage dams have eliminated mainstem spawning and rearing habitat, and have altered the natural flow regime of the Snake and Columbia Rivers, decreasing spring and summer flows, increasing fall and winter flow, and altering natural thermal patterns. Slowed water velocity and increased temperatures in reservoirs delays smolt migration timing and increases predation in the migratory corridor (NMFS 2014; Independent Scientific Group 1996; National Research Council 1996). Formerly complex mainstem habitats have been reduced to predominantly single channels, with reduced floodplains and off-channel habitats eliminated or disconnected from the main channel (Sedell and Froggatt 2000; Coutant 1999). The amount of large woody debris in these rivers has declined, reducing habitat complexity and altering the rivers' food webs (Maser and Sedell 1994).

Other anthropogenic activities that have degraded aquatic habitats or affected native fish populations in the Snake River basin include stream channelization, elimination of wetlands,

construction of flood-control dams and levees, construction of roads (many with impassable culverts), timber harvest, splash dams, mining, water withdrawals, unscreened water diversions, agriculture, livestock grazing, urbanization, outdoor recreation, fire exclusion/suppression, artificial fish propagation, fish harvest, and introduction of non-native species (Henjum *et al.* 1994; Rhodes *et al*. 1994; National Research Council 1996; Spence *et al*. 1996; Lee *et al*. 1997; NMFS 2004). In many watersheds, land management and development activities have:

- Reduced connectivity (i.e., the flow of energy, organisms, and materials) between streams, riparian areas, floodplains, and uplands;

- Elevated fine sediment yields, degrading spawning and rearing habitat;

- Reduced large woody material that traps sediment, stabilizes streambanks, and helps form pools;

- Reduced vegetative canopy that minimizes solar heating of streams;

- Caused streams to become straighter, wider, and shallower, thereby reducing rearing habitat and increasing water temperature fluctuations;

- Altered peak flow volume and timing, leading to channel changes and potentially altering fish migration behavior; and,

- Altered floodplain function, water tables and base flows (Henjum *et al*. 1994; McIntosh *et al*. 1994; Rhodes *et al*. 1994; Wissmar *et al*. 1994; National Research Council 1996; Spence *et al*. 1996; and Lee *et al*. 1997).

2.3.1. Basins in Action Area

The action area encompasses all areas potentially affected directly or indirectly by this consultation. Because of the potential for downstream effects and additive effects within watersheds, the action area encompasses entire subbasins where ESA-listed species and designated critical habitat occur. A general review of the environmental baseline has been divided up into the three major basins within the action area: (1) The Clearwater River basin; (2) the Salmon River basin; and (3) the Snake River basin.

2.3.1.1. Clearwater River Basin

The Clearwater River basin is located in north-central Idaho between the 46th and 47th latitudes in the northwestern portion of the continental United States. It is a region of mountains, plateaus, and deep canyons within the Northern Rocky Mountain geographic province. The basin is bracketed by the Salmon River basin to the south and St. Joe River subbasin to the north.

The Clearwater River drains approximately a 9,645-mi^2 area. The basin extends approximately 100 miles north to south and 120 miles east to west. There are four major tributaries that drain into the mainstem of the Clearwater River: the Lochsa, Selway, South Fork Clearwater, and North Fork Clearwater Rivers. The Idaho–Montana border follows the upper watershed boundaries of the Lochsa and Selway Rivers, and the eastern portion of the North Fork Clearwater River in the Bitterroot Mountains. The North Fork Clearwater River then drains the Clearwater Mountains to the north, while the South Fork Clearwater River drains the divide along the Selway and Salmon Rivers. Dworshak Dam, located 2 miles above the mouth of the North Fork Clearwater River, is the only major water regulating facility in the basin. Dworshak Dam was completed in 1972 and eliminated access to one of the most productive systems for anadromous fish in the basin. The mouth of the Clearwater is located on the Washington–Idaho border at the town of Lewiston, Idaho, where it enters the Snake River 139 river miles upstream of the Columbia River (NPCC 2004).

More than two-thirds of the total acreage of the Clearwater River basin is evergreen forests (over 4 million acres), largely in the mountainous eastern portion of the basin. The western third of the basin is part of the Columbia plateau and is composed almost entirely of crop and pastureland. Most of the forested land within the Clearwater basin is owned by the Federal government and managed by the USFS (over 3.5 million acres), but the State of Idaho and Potlatch Corporation also own extensive forested tracts. The western half of the basin is primarily in the private ownership of small forest landowners and timber companies, as well as farming and ranching families and companies. There are some small private in-holdings within the boundaries of USFS lands in the eastern portion of the basin. Nez Perce Tribe lands are located primarily within or adjacent to Lewis, Nez Perce, and Idaho Counties within the current boundaries of the Nez Perce Indian Reservation. These properties consist of both Fee lands owned and managed by the Nez Perce Tribe, and properties placed in trust status with the Bureau of Indian Affairs. Other agencies managing relatively small land areas in the Clearwater basin include the National Park Service, the BLM, Idaho Transportation Department, and IDFG (Ecovista 2004a).

Water quality limited segments are streams or lakes which are listed under section 303(d) of the CWA for either failing to meet their designated beneficial uses, or for exceeding state water quality criteria. The current list of 303(d) listed segments was compiled by the Idaho Department of Environmental Quality (IDEQ) in 2010, and includes many stream reaches within the Clearwater River basin (IDEQ 2010). Individual stream reaches are listed for parameters such as water temperature, sedimentation/siltation, fecal coliform, ammonia, oil and grease, dissolved oxygen, etc. Please refer to the following website for reach-specific 303(d) listed stream segments: http://www.deq.idaho.gov/water-quality/surface-water/monitoring-assessment/integrated-report.aspx.

Small-scale irrigation, primarily using removable instream pumps, is relatively common for hay and pasture lands scattered throughout the lower elevation portions of the subbasin, but the amounts withdrawn have not been quantified. The only large-scale irrigation/diversion system within the Clearwater basin is operated by the Lewiston Orchards Irrigation District within the Lower Clearwater subbasin.

Seventy dams currently exist within the boundaries of the Clearwater Basin. The vast majority of existing dams exist within the Lower Clearwater (56), although dams also currently exist in the Lower North Fork (3), Lolo/Middle Fork (5), and South Fork (6) watersheds (NPPC 2004).

The seven largest reservoirs in the basin provide recreational and other beneficial uses. Dworshak, Reservoir A, Soldiers Meadows, Winchester, Spring Valley, Elk River, and Moose Creek Reservoirs all provide recreational fishing opportunities. Reservoir A and Soldiers Meadows Reservoir are also part of the Lewiston Orchards Irrigation District irrigation system. Capacity of other reservoirs within the Clearwater basin is limited to 65 acre-feet or less, and in most cases is less than 15 acre-feet, limiting their recreational capacity (NPPC 2004).

Agriculture primarily affects the western third of the basin on lands below 2,500 feet in elevation, primarily on the Camas Prairie both south and north of the mainstem Clearwater and the Palouse. Additional agriculture is found on benches along the main Clearwater and its lower tributaries such as Lapwai, Potlatch, and Big Canyon Creeks. Hay production in the meadow areas of the Red River and Big Elk Creek in the American River watershed accounts for most of the agriculture in the South Fork Clearwater. Total cropland and pasture in the subbasin exceeds 760,000 acres. Agriculture is a particularly large part of the economy in Nez Perce, Latah, Lewis, and Idaho Counties, which all have large areas of gentle terrain west of the Clearwater Mountains. Small grains are the major crop, primarily wheat and barley. Landscape dynamics, hydrology, and erosion in these areas are primarily determined by agricultural practices (NPPC 2004).

Subwatersheds with the highest proportion of grazeable area (less than 50%) within the Clearwater basin are typically associated with USFS grazing allotments in lower-elevation portions of their ownership areas. However, the majority of lands managed by the USFS within the Clearwater basin are not subjected to grazing by cattle or sheep, including all or nearly all of the Upper Selway, Lochsa, and Upper and Lower North Fork watersheds. Subwatersheds outside of the USFS boundaries typically have less than 25% of the land area defined as grazeable, although this is as much as 75% for some. Privately owned property within the basin typically contains a high percentage of agricultural use, with grazeable lands found only in uncultivated areas. In contrast, grazing allotments on USFS lands are typically large, often encompassing multiple HUCs, resulting in higher proportions of grazeable area than those contained in primarily privately owned lands (NPPC 2004).

Mines are distributed throughout all eight watersheds in the Clearwater Basin, with the lowest number of occurrences in the upper and lower Selway. Ecological hazard ratings for mines (delineated by the Interior Columbian Basin Ecosystem Management Project) indicate that the vast majority of mines throughout the subbasin pose a low relative degree of environmental risk. However, clusters of mines with relatively high ecological hazard ratings are located in the South Fork Clearwater River and in the Orofino Creek drainage (Lolo/Middle Fork) (NPPC 2004).

2.3.1.2. Salmon River Basin

The Salmon River flows 410 miles north and west through central Idaho to join the Snake River. The Salmon River is the largest subbasin in the Columbia River drainage, excluding the Snake River, and has the most stream miles of habitat available to anadromous fish. The total subbasin is approximately 14,000 square miles in size. Major tributaries include the Little Salmon River, South Fork Salmon River, Middle Fork Salmon River, Panther Creek, Lemhi River, Pahsimeroi River, and East Fork Salmon River (IDFG 1990).

Public lands account for approximately 91% of the Salmon River Basin, with most of this being in Federal ownership and managed by seven National Forests or the BLM. Public lands within the basin are managed to produce wood products, domestic livestock forage, and mineral commodities; and to provide recreation, wilderness, and terrestrial and aquatic habitats. Approximately 9% of the basin is privately owned. Private lands are primarily in agricultural cultivation, and are concentrated in valley bottom areas within the upper and lower portions of the basin.

Land management practices within the basin vary among landowners. The greatest proportion of National Forest lands are Federally designated wilderness area or areas with low resource commodity suitability. One-third of the National Forest lands in the basin are managed intensively for forest, mineral, or range resource commodity production. The BLM lands in the basin are managed to provide domestic livestock rangeland and habitats for native species. State of Idaho endowment lands within the basin are managed for forest, mineral, or range resource commodity production. Near-stream or in-channel activities of relevance to fish and wildlife conservation include efforts by landowners, private or otherwise, to modify stream channels in order to protect property. Examination of the geographic distribution of permitted channel alterations during the past 30 years suggests that the long-term frequency of these activities was relatively consistent across much of the Salmon River Basin, but less common in the Upper Middle Fork Salmon, Lower Middle Fork Salmon, Middle Salmon-Chamberlain, and Pahsimeroi subbasins. It is unclear to what degree channel-modifying activities completed without permits may have had on the observed pattern. Stream channels in the basin are also altered, albeit on a smaller scale, by recreational dredging activities (NPCC 2004).

Water quality in many areas of the basin is affected to varying degrees by land uses that include livestock grazing, road construction, logging and mining (Ecovista 2004b). Water quality limited segments are streams or lakes which are listed under section 303(d) of the CWA for either failing to meet their designated beneficial uses, or for exceeding state water quality criteria. The current list of 303(d) listed segments was compiled by the IDEQ in 2010, and includes numerous defined stream reaches within the Salmon River Basin. Individual stream reaches are listed for parameters such as water temperature, escherichia coli, sedimentation/siltation, fecal coliform, ammonia, copper, etc. Please refer to the following website for reach-specific 303(d) listed stream segments: http://www.deq.idaho.gov/water-quality/surface-water/monitoring-assessment/integrated-report.aspx.

In the Lemhi, Upper Salmon, Pahsimeroi, and Middle Salmon-Panther subbasins, less than 20% of the larger streams meet all designated uses (i.e., specific uses identified for each water body

through state and tribal cooperation, such as support of salmonid fishes, drinking water supplies, maintenance of aquatic life, consumption of fish, recreational contact with water, and agriculture) (NPCC 2004).

Partial and seasonal barriers have been created on a few of these streams. Partial to complete barriers to anadromous fish exist on the Lemhi, Pahsimeroi and upper Salmon Rivers at water diversions for irrigation. Twenty minor tributaries contain dams that are used for numerous purposes such as irrigation, recreation, and fish propagation (IDFG 1990).

The diversion of water, primarily for agricultural use within the Salmon River Basin, has a major impact on developed areas – particularly the Lemhi, Pahsimeroi, the mainstem Salmon, and several tributaries of the Salmon River. Although many diversions are screened, many need repair and upgrading. A major problem is localized stream dewatering. In addition to water diversions, numerous small pumping operations for private use occur throughout the subbasin. Impacts of water withdrawal on fish production are greatest during the summer months, when streamflows are critically low (IDFG 1990).

The Salmon River Basin encompasses portions of five USFS wilderness areas. The Frank Church River of No Return Wilderness area, one of the five within the subbasin, is the largest wilderness area in the contiguous United States. Specific management guidelines for wilderness areas generally prohibit motorized activities and allow natural processes to function in an undisturbed manner.

Mining, though no longer a major land use as it was historically, it is still very prevalent in parts of the Salmon River Basin. Impacts from mining include severe stream alterations in substrate composition, channel displacement, bank and riparian destruction, and loss of instream cover and pool-forming structures. All of these impacts are typical of large-scale dredging and occur with other types of mining. Natural stream channels within the Yankee Fork, East Fork South Fork, and Bear Valley Creek, have all had documented spawning and rearing habitat destroyed by dredge mining. Furthermore, heavy metal pollution from mine wastes and drainage can eliminate all aquatic life and block access to valuable habitat as seen in Panther Creek (IDFG 1990).

2.3.1.3. Snake River Basin

The Snake River originates at 9,500 feet, along the continental divide in the Wyoming portion of Yellowstone National Park. The Snake River flows 1,038 miles westward toward the Idaho-Oregon border, northwest to its confluence with Henry's Fork near Rexburg, and then to Pasco, Washington, where it flows into the Columbia River. The Snake River is a large river that is one of the most important water resources in the State of Idaho. The Boise, Payette, and Weiser Rivers in Idaho, and the Owyhee, Malheur, Burnt, and Powder Rivers in Oregon, join the Snake River in this Idaho-Oregon border reach. The Snake River passes through Hells Canyon and Idaho Power Company's Hells Canyon Complex. Brownlee Dam, near River Mile 285, is the uppermost facility, with Oxbow and Hells Canyon dams downstream. The basin includes agriculture, and private and Federal irrigation.

The Snake River basin upstream from Brownlee Dam includes 31 dams and reservoirs with at least 20,000 acre-feet of storage each. The Bureau of Reclamation (BOR), Idaho Power Company, and a host of other organizations own and operate various facilities. These facilities have substantial influence on water resources, supplies, and the movement of surface and groundwater through the region. The total storage capacity of these reservoirs is more than 9.7 million acre-feet. In addition, there are numerous smaller state, local, and privately owned and operated dams and reservoirs throughout the upper Snake River Basin.

Within the action area, water quality limited segments are streams or lakes which are listed under section 303(d) of the CWA for either failing to meet their designated beneficial uses, or for exceeding state water quality criteria. The current list of 303(d) listed segments was compiled by the IDEQ in 2010, and includes 7 defined stream reaches within the Hells Canyon and Lower Snake River Asotin 4th-field HUCs. Individual stream reaches are listed for parameters such as water temperature, sedimentation/siltation, escherichia coli, dissolved oxygen, pH, and nutrient/eutrophication biological indicators. Please refer to the following website for reach-specific 303(d) listed stream segments: http://www.deq.idaho.gov/water-quality/surface-water/monitoring-assessment/integrated-report.aspx.

2.3.2. Baseline for Metals

Because of their wide variety of uses, metals enter the environment through many pathways. The most direct routes are through acid mine drainage from active and abandoned mines and point-source discharges from industrial activities such as plating, textile, tanning, and steel industries. Municipal waste water treatment plants and urban runoff are also significant source of metals to the environment. Arsenic, copper, and zinc used as pesticides and wood preservatives enter the environment via drift, erosion, surface runoff, and leaching. Copper is applied directly to the water as an aquatic herbicide. Particulate metals from combustion and dust can be transported through the air.

Metals can enter the aquatic environment in a dissolved form or be attached to organic and inorganic particulate matter. The amount of metal in the dissolved versus particulate form in natural waters can vary greatly, but the particulate form is usually found in greater concentrations. Metals can flux between different states and forms in an aquatic environment due to changes in pH, temperature, oxygen, presence of other compounds, and biological activity. These transformations can occur within and between water, sediment, and biota as the cycles of nature change. Dredging and disposal operations can result in substantial suspension and re-suspension of particulates in the water column, including those contaminated with metals.

Most metals addressed in this Opinion can enter the environment through natural and anthropogenic pathways, and many of these metals naturally occur in the region in low background concentrations. Most elevated concentrations of toxic metals in critical habitat have

been associated with hard rock mining operations, particularly in the Salmon River basin. There has been extensive degradation of critical habitat in many streams, some of which had been associated with complete extirpation of salmon and steelhead populations because of poor water quality (e.g., Panther Creek).

2.3.2.1. Baseline for Arsenic in Action Area

Concentrations of arsenic in river waters are usually low, typically in the range 0.1 to 2.0 µg/L worldwide. However, relatively high concentrations of naturally occurring arsenic in rivers can occur as a result of geothermal activity or the influx of high-arsenic groundwaters. Arsenic in surface water is strongly associated with sediments and is highest in the toxic zones near the surface water interface (Mok and Wai 1989; Nicholas and others 2003).

Arsenic concentrations of 10 to 70 µg/L have been reported in river waters from geothermal areas, including the western USA (Plant *et al*. 2007; McIntyre and Linton 2011; Table 2.4.3.1). In a probabilistic study of arsenic in 55 Idaho rivers, the median total concentration was 2.0 µg/L, ranging from 0.06 to 17 µg/L, from unfiltered samples (Essig 2010). In the Stibnite Mining District located in the East Fork of the South Fork Salmon River (EFSFSR), arsenic is naturally elevated in groundwater (up to 1000 µg/L), which then has been mobilized by mining and milling. Arsenic concentrations up to 96 µg/L in filtered samples and 109 µg/L in unfiltered have been measured in the EFSFSR downstream of the Stibnite Mining District (Woodward-Clyde 2000).

Arsenic is greatly elevated above background levels in the Panther Creek watershed, downstream of the Blackbird Mine. The loss of the Panther Creek population of Chinook salmon from Blackbird Mine contamination was one of the factors leading to the decline and ESA listing of Snake River spring/summer Chinook salmon (NMFS 1991). High arsenic in whole (unfiltered) surface waters (>100 µg/L) has been detected, although dissolved arsenic in filtered samples has been very low (<2 µg/L) in all samples (Table 2.4.3.2). Based on their relative toxicities and ambient concentrations, copper was probably the biggest factor causing the loss of the Panther Creek Chinook population, although arsenic contributes to aquatic risk (Section 2.4.3; NMFS 2007).

Arsenic, cobalt, and copper were greatly elevated in sediments, periphyton, and in the tissues of aquatic insects in Panther Creek at the time of Chinook listing (Figures 2.3.1.1 to 2.3.1.3). Ongoing remedial efforts that began in 1995 have led to some reductions in arsenic concentrations in Panther Creek sediments and in the foodweb, although concentrations in both remain elevated above upstream reference concentrations as of 2010 (Figures 2.3.1.1 and 2.3.1.3). Arsenic in tissues of aquatic insects declined with initial remedial efforts, but from 2006 to 2010, there have been no further decreases in arsenic in insect tissues. In contrast to marked reductions in copper in Panther Creek (Section 2.3.3.), arsenic in periphyton has yet to decline in Panther Creek. This suggests the presence of a persistent reservoir of arsenic in sediments and floodplain soils.

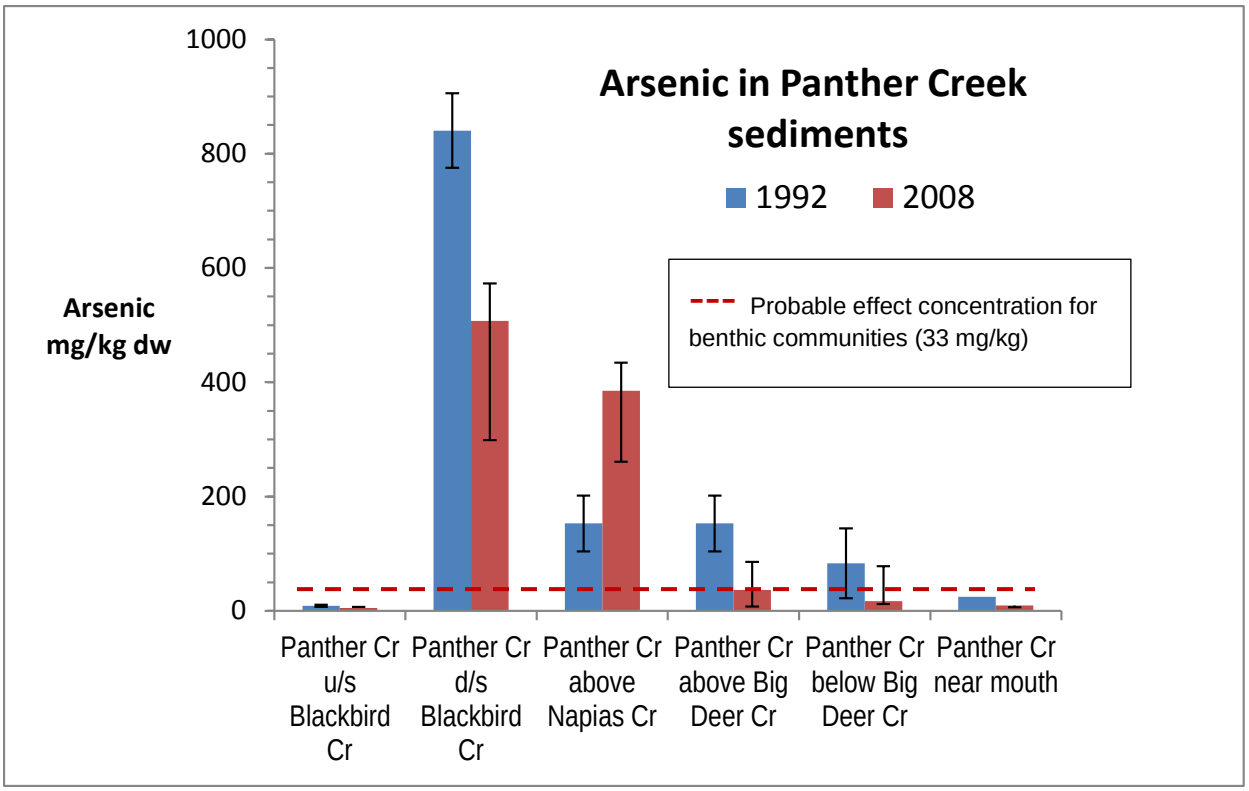


Figure 2.3.1.1. Arsenic in Panther Creek sediments sampled in similar stream reaches before and after remediation efforts. In both surveys arsenic declined with increasing distance downstream from Blackbird Creek. Arsenic appears to have generally declined over time, although arsenic is still greatly elevated until the diluting flows of Napias Creek, a large tributary, enter. This suggests a reservoir of arsenic may persist in sediments or riparian soils that may be difficult to further control. As of 2011, EPA is evaluating the feasibility of additional remediation to further reduce arsenic releases from Blackbird Creek. Data from Mebane (1994) and Golder (2009), probable effect concentration from MacDonald *et al*. (2000a).

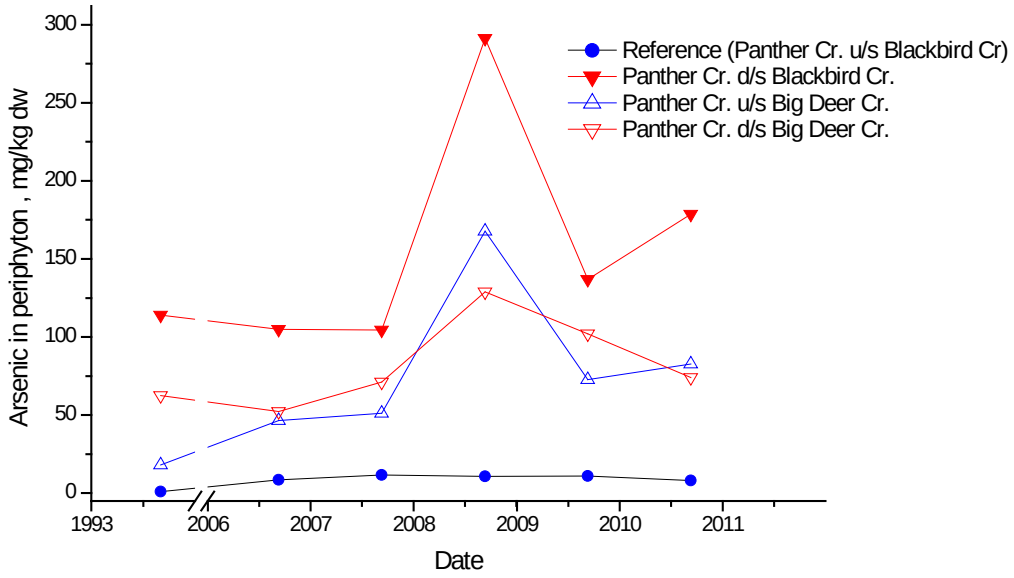


Figure 2.3.1.2. Arsenic in periphyton (algae and other organic material collected from stream rocks) in Panther Creek sampled in similar stream reaches before and after remediation efforts. Periphyton is the primary food source for many aquatic insects. Data from Beltman *et al*. (1994) and EcoMetrix (2011).

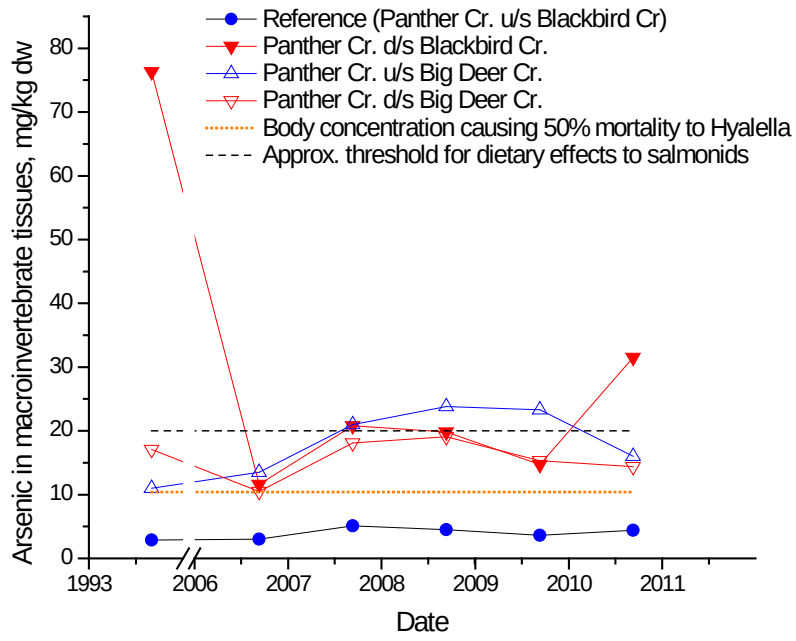


Figure 2.3.1.3. Arsenic in macroinvertebrate tissues of Panther Creek sampled in similar stream reaches before and after remediation efforts. In both time periods arsenic declined with increasing distance downstream from Blackbird Creek. At the uppermost mining-affected sites, Panther Creek downstream of Blackbird Creek, arsenic initially declined markedly following remediation, but has not further declined from 2006 through 2010. The 2008 spike in arsenic concentrations apparent in sediment and periphyton graphs was not apparent in macroinvertebrates, suggesting limited bioavailability of arsenic in that event. Data from Beltman *et al*. (1994) and EcoMetrix (2011).

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; McIntyre and Linton 2011). Juvenile salmonids have been found 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, and inorganic forms of arsenic (including pentavalent) are typically more toxic than organic forms (EPA 1985a; Eisler 1988a; Sorensen 1991). Chronic toxicity in fish appears to be inversely proportional to water temperature under certain experimental conditions (McGeachy and Dixon 1990).

2.3.2.2. Baseline for Chromium

Although weathering processes result in the natural mobilization of chromium, the amounts added by anthropogenic activities are thought to be far greater. Major sources are the industrial production of metal alloys, atmospheric deposition from urban and industrial centers, and large scale wrecking yards and metals recycling and reprocessing centers (Reid 2011). Few, if any, of these major urban or industrial sources are expected in the largely rural action area in Idaho.

Few data on chromium concentrations in Idaho were located. In the Stibnite Mining District in the EFSFSR basin, total chromium concentrations collected under low flow conditions in September 2011 ranged from <0.2 µg/L to 0.24 µg/L (*http://waterdata.usgs.gov/nwis*, HUC 17060208). In the Blackbird Mining District, concentration of chromium in seeps and adits around the Blackbird Mine were not higher than average background filtered surface water concentrations near the Blackbird Site (<2.9 µg/L) (Beltman and others 1993)

2.3.2.3. Baseline for Copper

Copper concentrations of about 0.4 to 4 µg/L have been considered typical of major river waters in the United States, not directly influenced by industrial or urban activities (Stephan and others 1994). Specific data reviewed within the Idaho action area mostly fell within that range. Whenever available, data given here were limited to the data collected in 1993 or later using "clean" sampling and analyses and quality control measures. This is because prior to the implementation of "clean" procedures, contamination of metals samples during collection and analyses was nearly ubiquitous (Shiller and Boyle 1987; Windom and others 1991; Stephan and others 1994).

In the Salmon River basin, reliable copper data are available for several locations. With the exception of the Panther Creek drainage, discussed separately, almost all other locations had low copper concentrations relative to Stephan *et al*.'s (1994) range. In the Salmon River upstream of Panther Creek, dissolved copper ranged from 1.4 to 1.6 µg/L in six samples collected during high and low flows in 1993. Yet, in the Salmon River sampled a few miles downstream of Panther Creek at the same time, copper ranged from 5.3 to 25.9 µg/L (Maest and others 1994). In the Stibnite Mining District in the EFSFSR basin, copper concentrations collected under low flow conditions in September 2011 ranged from <0.5 µg/L to 4 µg/L which is almost the same as the range given by Stephan *et al*. (1994) (*http://waterdata.usgs.gov/nwis*, HUC 17060208). In the mainstem upper Salmon River in the vicinity of the Thompson Creek Mine (TCM), copper concentrations in 1998 to 2000 ranged from <0.2 to 1 µg/L, in 17 of 18 samples, with a single much higher value of 5.5 µg/L during October 1998. That single high value may not have been reliable, since the tributaries to the Salmon River that directly receive Thompson Creek effluent, and thus should have had higher copper concentrations had the high copper value originated from the mine, showed consistently lower copper concentrations, <0.2 to 2 µg/L (Mebane 2000).

Copper has been monitored in the vicinity of the Hecla Grouse Creek Mine, which discharges to the Yankee Fork River via a pipeline and diffuser, and also to Jordan Creek, a smaller stream. Copper in Jordon Creek downstream of mine discharges in 2010 was very low, ranging from <0.5 µg/L to 1 µg/L, and in the Yankee Fork River, downstream of mine effluents similarly ranged from 0.5 to 0.9 µg/L (Hecla Mining Company data).

In wilderness regions of the Middle Fork Salmon River, Idaho, filtered copper concentrations in Loon Creek and Big Creek ranged from 0.6 to 0.93 µg/L (Maest and others 1994). Other locations in the Salmon River, Idaho drainage with copper data included the Pahsimeroi River at Ellis, Lemhi River near Lemhi, Salmon River near Salmon, Salmon River near White Bird, and Johnson Creek, a tributary to the South Fork Salmon River. Copper concentrations at all these sites ranged from <1 to 4 µg/L from 1991 to 1995 (Hardy and others 2005).

In the Clearwater River basin, much less information is available, which is probably because there is less recent mining activity and associated monitoring in the Salmon River basin. The available copper data located had low values. For instance, in Lapwai Creek and in the South Fork Clearwater River at Stites, copper in filtered samples collected between 1991 and 1995 ranged from <1 µg/L to 2 µg/L, n=8 each (Hardy and others 2005).

In the Hells Canyon reach of the lower Snake River, near Anatone, Washington, copper concentrations from the same time period were a little higher than those usually reported from the Clearwater or Salmon River drainages, ranging from 1 to 4 µg/L, n=18 (Hardy and others 2005).

Other than the Panther Creek drainage, the highest copper concentrations from the state of Idaho's statewide monitoring project was from the Clark Fork River, at Cabinet Gorge, Idaho with values up to 38 µg/L from 1992 to 1995 (Hardy and others 2005). The Clark Fork has been subject to large scale mining disturbances and copper contamination, although these disturbances are >200 miles upstream of Cabinet Gorge.

Natural background concentrations of copper and other metals can occur; however, these seem to be rare and limited to very small streams or springs. Areas of Panther Creek, Idaho, that had no evidence of mining disturbances but were near mining prospects did sometimes have much higher copper concentrations than noted above. For example, Mebane (1994) reported 312 µg/L in a spring in the headwaters of Little Deer Creek, and 10.7 µg/L in Little Deer Creek at its mouth.

In summary, other than Panther Creek and the Salmon River shortly downstream, copper concentrations measured throughout the action area are usually in the range of <0.5 to 4 µg/L.

Baseline for Copper Concentrations in the Panther Creek Watershed. Baseline conditions are described separately for the Panther Creek watershed, because copper contamination and the resulting loss of the Panther Creek Chinook salmon population was one specific factor leading to the decline of the species, and listing of spring/summer Chinook salmon under the ESA (NMFS 1991). Because of this, copper concentrations and associated biological conditions in Panther Creek at the time of listing and contemporary conditions are considered here in detail. Concerted

site remediation efforts began in 1995 and have been sustained to date. The objectives of these remedial efforts are specifically intended to restore water quality to restore lost anadromous fish populations. To wit, the remedial action objective for Panther Creek is to "*restore and maintain water quality and aquatic biota conditions capable of supporting all life stages of resident and anadromous salmonids and other fishes in Panther Creek*" (EPA 2003d; 2008). As follows, the effectiveness of these efforts is evaluated through comparisons with upstream reference concentrations over time. The following information and series of figures were prepared by compiling available data that had been collected before and after the onset of remedial efforts.

Copper was greatly elevated above background levels in the Panther Creek watershed, downstream of the Blackbird Mine from the 1950s through 1990s. The loss of the Panther Creek population of Chinook salmon was attributed to Blackbird Mine contamination, rather than copper specifically (NMFS 1991). Blackbird Mine contamination to Panther Creek consisted mostly of copper, cobalt, arsenic, and iron (Maest and others 1994; Mebane 1994). However, based on their relative toxicities and ambient concentrations, copper was probably the biggest factor causing the loss of the Panther Creek Chinook population, although arsenic continues to contribute to aquatic risk (Section 2.4.3; NMFS 2007).

Copper was greatly elevated in the Panther Creek stream food webs, that is, sediments, periphyton, and in the tissues of aquatic insects in Panther Creek at the time of Chinook listing (Figure 2.3.1.4). The magnitude of contamination at that time was extreme, with values in sediment, periphyton, and aquatic insects hundreds of times higher than upstream background concentrations. Following initial remedial efforts, copper concentrations in Panther Creek downstream of Blackbird Mine influences dropped markedly by the mid-2000s. These efforts have led to reductions in copper concentrations in Panther Creek water, sediments and in the foodweb on the order of 90%, and are approaching upstream reference concentrations (Figure 2.3.1.4). Sediment, periphyton, and aquatic insect copper values obtained upstream of mine influences have been very consistent over time, even across different studies. This indicates that the more recent, lower copper values obtained downstream of mine influences are likely real, and cannot be attributed to methods differences.

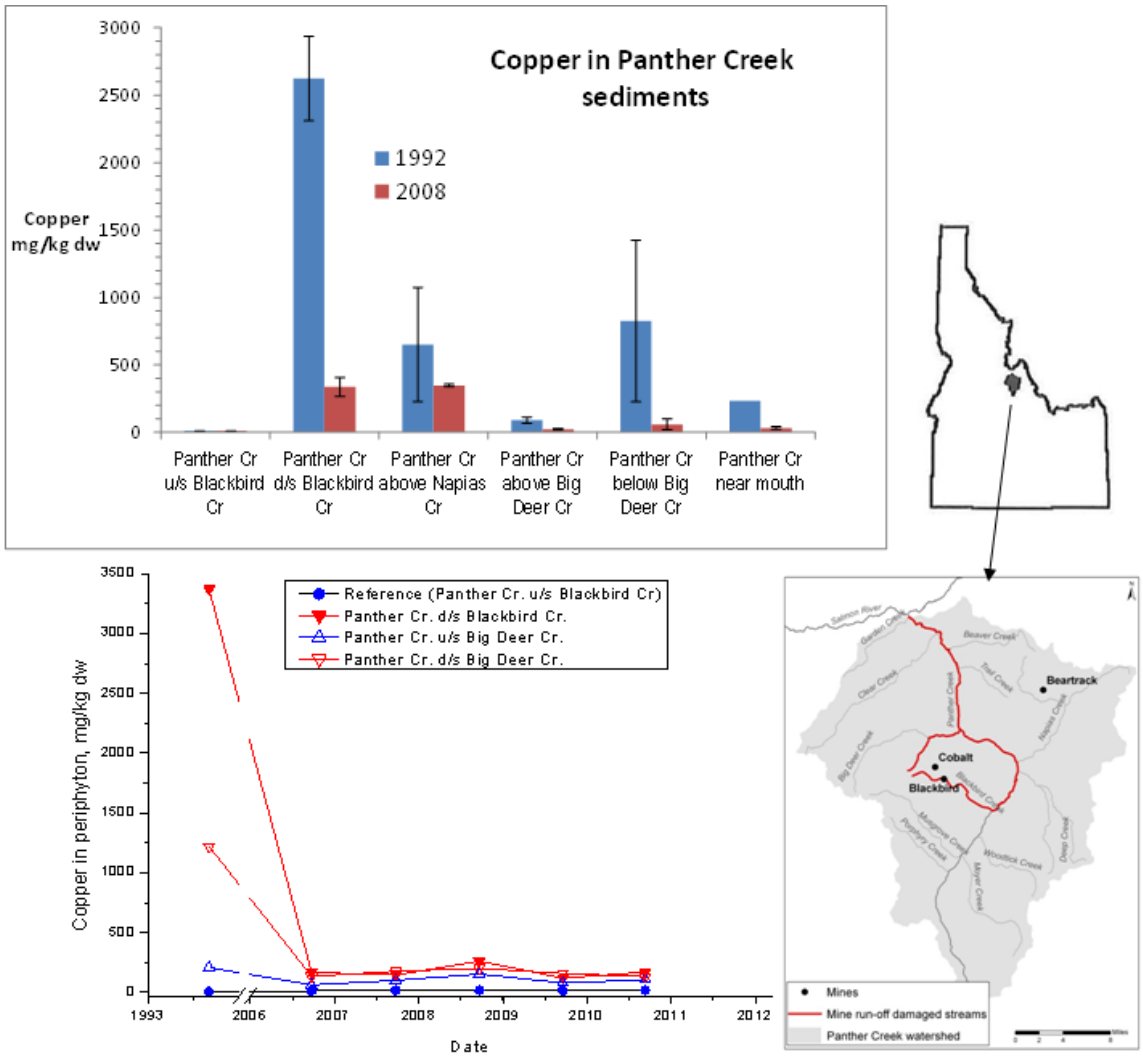
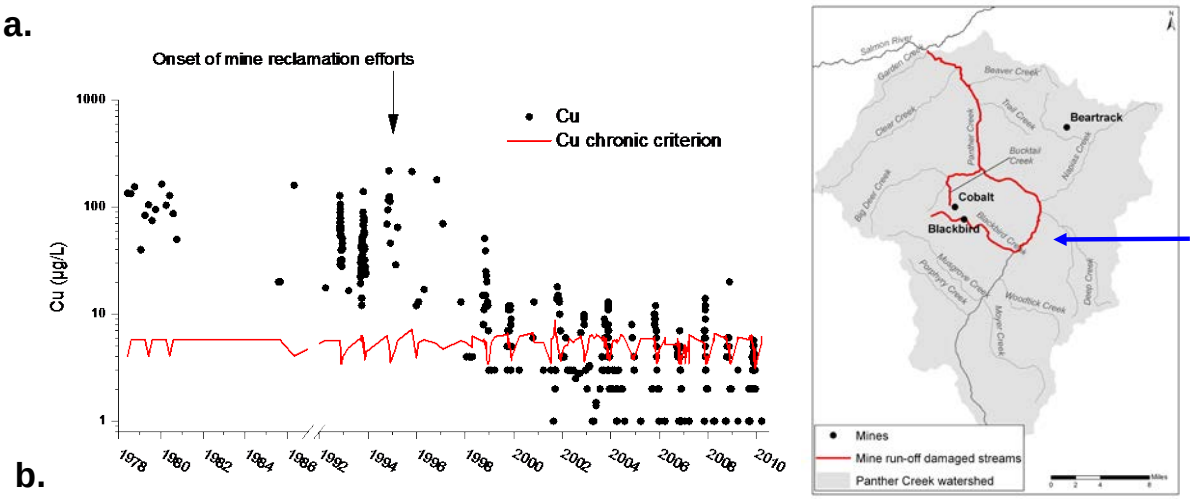
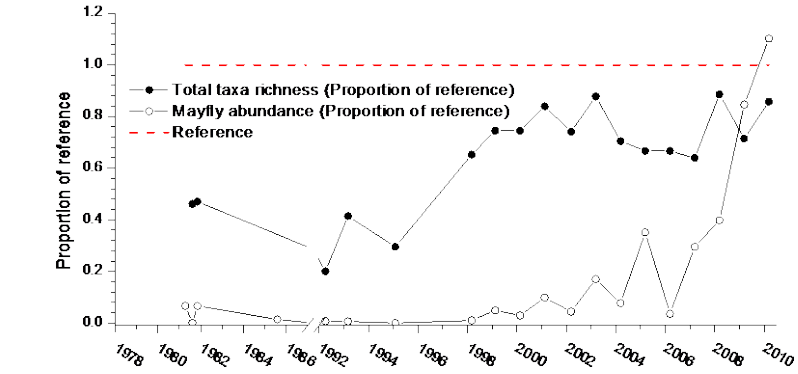
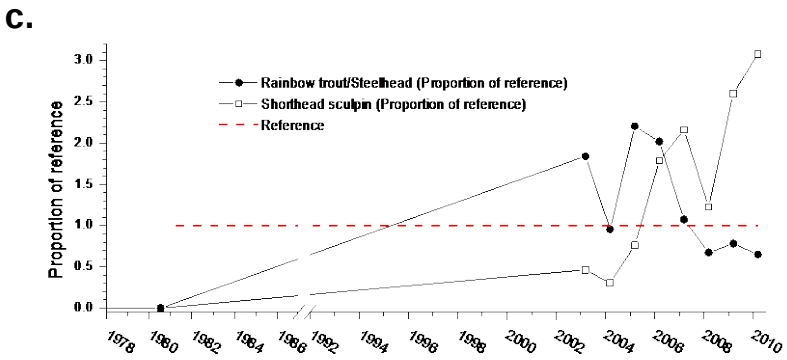


Figure 2.3.1.4. Copper in Panther Creek foodwebs has greatly declined following Blackbird Mine remediation efforts that have been ongoing from 1995 to date. (Top) copper in sediments in 1992 and 2008 (Mebane, 1994; Golder, 2009); (Middle), copper in periphyton; and (Bottom), copper in aquatic insect tissues (Beltman and others, 1994; EcoMetrix, 2011).







Copper data sources: (Davies 1982; Wai and Mok 1986; Beltman *et al*. 1993; Maest *et al*. 1994; Maest *et al*. 1995; Golder 2009); Biological data sources: (Beltman *et al*. 1994; Mebane 1994; Golder 2003; Stantec 2004; EcoMetrix 2005, 2006, 2007, 2008, 2009, 2010, 2011).

Figure 2.3.1.5. Copper concentrations (a) and corresponding diversity of all aquatic insects and abundance of mayflies (b), abundance of rainbow trout/steelhead and shorthead sculpin in (c) in Panther Creek, Idaho, downstream of mining-influenced Blackbird Creek. Aquatic insect diversity and abundance and fish abundance are scaled against concurrent upstream reference collections. Copper concentrations at upstream reference sites have been <3µg/L in measurements from 1993 to date, since the routine use of appropriate low detection levels and "clean" field sampling and laboratory techniques.

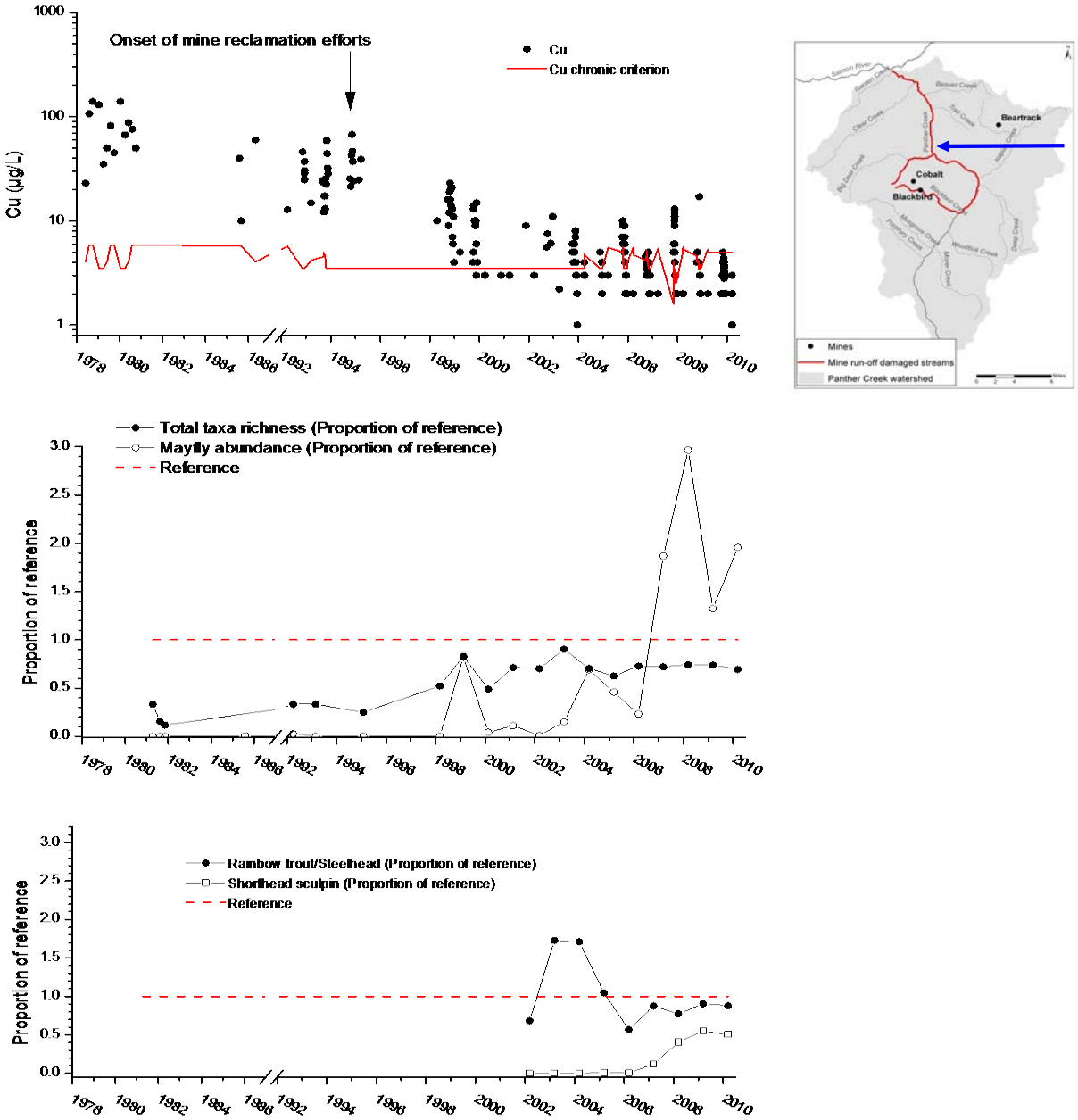


Figure 2.3.1.6. Copper concentrations (a) and corresponding diversity of all aquatic insects and mayflies (b), and abundance of mayflies (c), in Panther Creek, Idaho, downstream of mining-influenced Big Deer Creek. (See Figure 2.3.1.5 for data sources)

Data for copper in water and associated biological data were compiled and evaluated for four key locations: Panther Creek downstream of the upstream mining influenced tributary, Blackbird Creek (Figure 2.3.1.5), and Panther Creek downstream of the downstream mining influenced tributary, Big Deer Creek (Figure 2.3.1.6). These locations are particularly data rich with a remarkable 30-year period of record for copper and stream invertebrates. To make the invertebrate data comparable between years and between different studies, all of the results from the mining-influenced locations are scaled as a proportion of the upstream reference locations that were collected concurrently for each sampling event shown.

The ecology of Panther Creek, as measured by the abundance and diversity of aquatic insects and fish populations began to rebound as copper declined. In Panther Creek downstream of Blackbird Creek, prior to about 1998, aquatic invertebrate communities were extremely impoverished with species richness less than half that of upstream samples. Mayflies were absent or scarce. After 1998, mayflies began to appear in the samples and by 2009 were about as abundant as upstream reference (Figure 2.3.1.5, middle). Insect species richness reached about 80% of upstream reference station counts by about 2002 and seems to have plateaued. Quantitative fish data are fewer than for insects. In electrofishing surveys in 1967 and 1980, no fish of any species were captured from Panther Creek downstream of Blackbird Creek. By 2002, when the recent program of biomonitoring started, rainbow trout were more abundant than at the upstream reference. Sculpin were present but were about half the density of the nearby upstream reference stations. By 2006, the sculpin were more abundant than at upstream reference, and as the sculpins became increasingly abundant, rainbow trout densities declined (Figure 2.3.1.5, bottom).

Sculpin are emphasized in these comparisons because they may be a useful indicator species in biomonitoring of potential pollution effects. Sculpin have been observed to decline or disappear from streams with elevated metals from mining, may be more sensitive or at least as sensitive as listed salmonids, and decline with increasing proportions of fine sediments on the stream bottoms (Mebane, 2001; Maret and MacCoy, 2002; Mebane and others, 2003; Besser and others, 2007).

The insect and fish communities in Panther Creek downstream of Big Deer Creek have shown a similar recovery pattern. Prior to the mine reclamation work, insect diversity was even lower than at Panther Creek downstream of Blackbird Creek, and sculpins were completely absent until about 2006. By 2010, sculpin densities had recovered to the point where they were about half as abundant as upstream of Blackbird Creek (Figure 2.3.1.6). This does not necessarily indicate that copper concentrations are still limiting sculpin densities for two reasons. First, in Idaho, there are natural transitions in fish communities from headwaters downstream. Higher elevation headwater streams tend to be steeper and colder than lower elevation streams. Often, trout are the only fish found in perennial headwater streams. As streams drop in elevation they tend to become less steep, warmer, and larger. These mid-sized streams, such as upper Panther Creek tend to be dominated by sculpins and salmonids. As streams transition into larger rivers, the sculpin become less abundant and minnows and suckers appear (Mebane 2002b; Mebane and others 2003). Therefore, sculpin densities would be expected to decline in lower Panther Creek relative to upstream monitoring sites.

Big Deer Creek had higher copper concentrations than did Panther Creek, and biological impairment was so severe that almost all aquatic life had been extirpated. In 1992, total number of aquatic insects in Big Deer Creek upstream of mine influences ranged from 1,938 to 4,995 insects/m^2 compared to 0 to 68 insects/m^2 downstream of mine influences (Mebane 1994). No fish could be found downstream of the Blackbird Mine influences. Recovery has been slower in Big Deer Creek than Panther Creek, but by 2010 the aquatic insect communities were as diverse as upstream reference, and by 2009 rainbow trout populations had recovered to reference conditions. Sculpins do not occur in Big Deer Creek even upstream of mine pollution.

In summary, in comparison to conditions at the time that Snake River spring/summer Chinook salmon were listed, copper concentrations in Panther Creek have declined and associated biological communities have largely recovered. Aquatic insect diversity is still lower than in reference conditions. Current copper criteria are not consistently met in Panther Creek, particularly during spring runoff. However, whether these spring copper criteria exceedences are likely related to residual effects on aquatic insect communities cannot be determined from the available data. A given relatively low copper concentration such as 3 µg/L would likely be more toxic in Panther Creek during baseflow conditions from late summer to early spring than during high spring flows when more organic carbon is also present (Appendix C).

2.3.2.4. Baseline for Cyanide

NMFS located few cyanide data that were specific to Idaho. The most likely sources of cyanide in waters are probably forest fires, gold mining operations that use cyanide leaching, and perhaps road salting. The most comprehensive monitoring data were associated with the Grouse Creek Mine, located in the Yankee Fork of the Salmon River near Custer, Idaho. The Grouse Creek Mine is an inactive gold mine that operated from about 1995 to 1997, and used a cyanide vat leach process. When operating, up to 110 µg/L weak acid dissociable (WAD) cyanide was present in effluent discharged to either Jordan Creek (a tributary to the Yankee Fork), or was discharged directly to the Yankee Fork. Subsequently, cyanide levels in the effluent declined to mostly undetectable levels. In 2003 maximum effluent WAD cyanide was 3 µg/L; from 2004 through 2010 all ambient values in Jordon Creek or the Yankee Fork River were less than the detection limit of 2 µg/L (D. Landres, Hecla Mining Company, letter of 31 March 2011 to Michael Gearheard, EPA, Seattle, Washington).

While no Idaho specific data were located, the major current risk of cyanide toxicity in the action area is probably from forest fires or other biomass burning (e.g. burning waste biomass for energy conversion, crop burning, prescribed forest fires and wildfires) (Barber and others 2003; Pilliod and others 2003). Barber *et al*. (2003) examined releases of cyanides from biomass burning and their effect on surface runoff water. In laboratory test burns, available cyanide concentrations in leachate from residual ash were much higher than in leachate from partially burned and unburned fuel and were similar to or higher than a 96-h median lethal concentration (LC_{50}) for rainbow trout (45 µg/L). Free cyanide concentrations in stormwater runoff collected after a wildfire in North Carolina averaged 49 µg/L, again similar to the rainbow trout LC_{50} and an order of magnitude higher than in samples from an adjacent unburned area (Barber and others 2003).

In other areas, greatly elevated cyanide had been shown to occur in snow exposed to urban traffic and highway deicing. Deicing salts contain cyanide compounds as anticaking agents. In the Cincinnati area, cyanide in snow around urban highways averaged 154 µg/L compared to 20 µg/L in urban areas that were not close to major highways (Glenn and Sansalone 2002). Similar results could be expected in Idaho if similar deicing compounds are used.

2.3.2.5. Baseline for Lead

In natural waters, lead is usually complexed with particulate matter resulting in much lower dissolved than total concentrations (Mager 2011). For instance, in the pervasively lead contaminated Coeur d'Alene River of northern Idaho, dissolved lead concentrations rarely exceed 20 µg/L whereas total concentrations often exceed 100 µg/L. A maximum dissolved lead concentration of 420 µg/L was reported for this location (Clark 2002; Balistrieri and Blank 2008). The Coeur d'Alene River is north of occupied habitat, as is the Clark Fork River, Idaho, where up to 60 µg/L dissolved lead has been reported (Hardy and others 2005). Within the action area, reliable lead data are sparse but the available data are quite low. The highest lead concentration obtained by the Idaho IDEQ/U.S. Geological Survey (USGS) statewide monitoring program within the action area was from the Hells Canyon reach of Snake River near Anatone, Washington (7 µg/L). All other measurements from within the Clearwater and Salmon River basins and the Snake River downstream of Hells Canyon dam were <1 µg/L (Hardy and others 2005). Mebane (2000) reported lead concentrations in the upper Salmon River near the TCM as high as 2 µg/L, but most values were <0.2 µg/L.

2.3.2.6. Baseline for Mercury

Mercury is distinguished from other contaminants with natural sources (metals[4]) considered in this Opinion for several reasons, one of which is that ambient concentrations in water as well as concentrations of concern are two to four orders of magnitude lower than for other metals. As explained in the "*Species Effects of Mercury Criteria*" (Section 2.4.6.1), there are no species effects of concern, only habitat effects through food chain exposure. Thus the baseline concentrations of mercury are described in the context of the subsection "*Factors influencing mercury tissue concentrations in fish.*" Generally, mercury concentrations measured in salmonids in Idaho streams and lakes ranged from <0.05 to 1.1 mg/kg ww (Table 2.4.6.2*)* Baseline concentrations of mercury in Idaho waters ranged from <0.2 to 6.8 ng/L (Table 2.4.6.2*).*

2.3.2.7. Baseline for Nickel

Nickel is rare in the waters of Idaho, even in areas disturbed by mining. In the Blackbird Mine area, Beltman *et al*. (1993) reported Ni concentrations in mine waters and seeps in excess of 1500 µg/L; however, in the streams that were large enough to support fish populations and that were affected by mining (Blackbird and Big Deer Creeks), nickel ranged from <10 to 60 µg/L.

[4] i.e., naturally occurring elements as opposed to invented, purely synthetic compounds such as PCBs and most pesticides

In the samples with high nickel concentrations, copper concentrations were greater than 900 µg/L which is sufficient to kill all the aquatic life without any contribution from nickel. These two streams are upstream of critical habitats. In designated critical habitats (Panther Creek and lower Big Deer Creeks) nickel was <10 µg/L. Although few other data were located, what was found indicates nickel concentrations may be assumed to be low in the action area. In the mining affected SF Coeur d'Alene River, located in northern Idaho, Mebane *et al*. (2012) reported nickel concentrations ranging from <2 to 8 µg/L.

2.3.2.8. Baseline for Selenium

In Idaho rivers, the median selenium concentration determined from a probabilistic sampling of 55 river sites was 0.13 µg/L in water (range <0.09 to 1.75 µg/L) and in fish, median muscle selenium residues were 1.28 mg/kg dw (range 0.22 to 14.7 mg/kg dw) (Essig 2010). Essig's study used a randomized design, that is, each sampling site was selected from a random draw of feasible sampling sites, rather than targeting areas of interest because of potentially elevated selenium concentrations. Within the range of listed anadromous salmon and steelhead in Idaho, an area of the upper Salmon River basin was identified as having anomalously high selenium in soils, aquatic habitats, and food webs. These are evaluated further in Section 2.4.8 in the subsection "*Bioaccumulation of selenium through stream food web trophic transfer*."

2.3.2.9. Baseline for Silver

Silver is sparingly soluble and rare in aquatic environments. The EPA (1987b) give natural background silver concentrations as being in the 0.1 to 0.5 µg/L. Wood (2011) however, noted that values in this range were obtained before the widespread adoption of clean sampling techniques in the 1990s and considered values in this range to be orders of magnitude too high. Instead better estimates of natural background silver concentrations were in the range of 0.1 to 5 ng/L (0.0001 to 0.005 µg/L). Such concentrations are not detectable with the technology used in non-specialty analytical laboratories. Even in highly contaminated areas, silver concentrations rarely exceed 0.1 to 0.3 µg/L. In nature, silver is unlikely to be found in its ionic form. Given the extremely high affinity of silver for reduced sulfur, most silver in the environment is expected to occur as silver sulfides, even in oxygenated waters (Wood 2011). Even in Idaho's Silver Valley where 100-plus years of silver mining resulted in one of the largest superfund cleanup projects in the nation, silver is not a contaminant of concern (NRC 2005). No data specific to the action area were located.

Although silver sulfides are the form most likely found in the environment, the form of silver usually used in toxicity tests is silver nitrate, which is much more toxic (Wood,2011). Chronic toxicity to freshwater aquatic life from silver nitrate may occur at concentrations as low as 0.12 µg/L (EPA 1980o) and the literature reviewed for silver criterion ranges from 0.3 to 11 µg/L over a hardness range of 25 to 200 mg/L.

2.3.2.10. Baseline for Zinc

Median baseline concentrations of zinc in large rivers, not directly influenced by mining, urban, or industrial activities are usually in the neighborhood of 0.5 to 4 µg/L (Gaillardet and others 2007). In contrast, streams with extensive mining disturbances such as the Coeur d'Alene River basin in north Idaho, sometimes have very high zinc concentrations, in excess of 2000 µg/L. Such ambient Zinc concentrations killed juvenile salmonids in hours to a few days, and fish and aquatic insect populations are depressed. (Maret and MacCoy 2002; Maret and other, 2003; Mebane and others 2012).

In mineralized areas in Idaho with naturally high zinc concentrations in watershed rock and soils, but that have not been highly disturbed, average zinc concentrations may be up to 10X higher than typical large river concentrations. In Jordan Creek, a tributary to the Yankee Fork River in the upper Salmon River subbasin, average zinc concentrations in monthly sampling from 2004 through 2009 were about 12 µg/L, with a maximum measurement of 40 µg/L. This maximum measurement is higher than Idaho's proposed acute criterion of 32 µg/L, calculated assuming the hardness was 25 mg/L, per IDEQ policy (Table 1.3.1). If the criterion were calculated using the actual measured hardness of 15 mg/L, the applicable criterion under Idaho's proposed standard would be about 24 µg/L. This sampling site is located upstream of the Grouse Creek Mine, and presumably mostly natural. Zinc concentrations measured directly in the tailings pond effluent from the Grouse Creek Mine were similar, with a 2010 mean of 11 µg/L and a maximum of 31 µg/L. In the Yankee Fork River, upstream of Jordan Creek and upstream of the tailings pond effluent outfall, the average zinc concentrations were a little lower than they were in Jordan Creek. Average 2004 to 2009 zinc concentrations were 9 µg/L with a maximum of 30 µg/L. If calculated using the sample hardness of 18 mg/L, the zinc acute criterion would be about the same, 28 µg/L (Hecla Mining Company data, sites "S-6" and "S-9," Cindy Gross, Hecla Mining Company, personal communication).

Zinc concentrations measured in the Salmon River near Clayton, in the vicinity of the TCM from 1998 to 2000 ranged from about 2 to 6 µg/L. In Thompson Creek itself, just downstream of a permitted mine effluent discharge, zinc was noticeably higher during that time period, averaging about 7 µg/L, with a maximum concentration of about 30 µg/L. Based on the minimum hardness of Thompson Creek during that period, about 50 mg/L, the acute zinc criteria as calculated under Idaho's proposed standard would be about 65 µg/L, well above measured ambient zinc concentrations downstream of the mining discharges. Upstream background zinc concentrations in Thompson Creek are about 2 µg/L (Mebane 2000).

Zinc has been elevated in a third watershed in the close vicinity of the Yankee Fork and Thompson Creek areas. Kinnikinic Creek, is a small tributary to the upper Salmon River, near Clayton, Idaho, and is the home of the Clayton Silver Mine. In 1999, zinc concentrations ranged from <5 upstream of the Clayton Silver Mine to 224 µg/L downstream of the tailings pile that was encroaching into the stream. Following a 2001 EPA removal action, IDEQ monitoring in Kinnikinic Creek yielded zinc concentrations ranging from 2 µg/L upstream of the mine to 64 µg/L just above the confluence with the Salmon River. In the latter sampling, water hardness was about 100 mg/L, which would yield zinc criteria of about 106 µg/L (IDEQ 2003).

Elsewhere in the Idaho action area, available zinc concentrations were low, with some noticeable exceptions. In USGS monitoring in the mid-1990s in the Lapwai Creek near Lapwai, Pahsimeroi River at Ellis, the Little Salmon River near Riggins, and the Snake River in Hells Canyon near Anatone, Washington, the maximum zinc concentrations were 7 µg/L (n=8). The Lemhi River near Lemhi was a noticeable exception with a maximum zinc concentration of 210 µg/L during this time period, although the median was much lower, 5 µg/L. Other streams that occasionally had anomalously high zinc measurements were Johnson Creek near Yellow Pine, the Salmon River near Salmon, and the Salmon River near White Bird, with maximum zinc measurements of 20, 16, and 24 µg/L respectively (Hardy and others 2005).

2.3.2.11. Baseline for Organic Pollutants

There has not been a comprehensive water quality study conducted of organic pollutant levels in the action area, and little information concerning the occurrence of most organic pollutants is available. There are reports of measurable concentrations of PCBs, DDTs, and organochlorine pesticides (lindane, chlordane, and heptachlor) at specific sites within Idaho (Munn and Gruber 1997; Pinza *et al.* 1992; EPA 1992b; Wegner and Campbell 1991; Apperson and Anders 1990), but contamination does not appear to be extensive. Data collected as part of the National Water Quality Assessment Program in the nearby Central Columbia Plateau suggests that elevated levels of toxic organic pollutants of concern in the action area are most likely to be found in areas influenced by urbanization and agriculture (Williamson *et al.* 1998).

Because of the low usage of these compounds, water column concentrations are expected to be negligible. Water column concentration data from the Snake River, Oregon/Idaho within the Hells Canyon Dam complex are the most relevant environmental concentration data located (Table 2.3.1). The complex is just above the Hells Canyon Dam on the Snake River. Sediment and fish tissue residue data for most of the organic chemicals of concern in this Opinion were available from the lower Snake River downstream of Hells Canyon and the lower Salmon River (Clark and Maret 1998). Clark and Maret (1998) also report data from within Brownlee Reservoir and many sites in the Snake River basin upstream of Brownlee. For the most part, the highest concentrations of organic chemicals of concern within the state of Idaho occurred within Brownlee Reservoir. However, the available concentration data in water, sediment, and fish were generally close to or below the levels of detection (Table 2.3.1). The “true” concentrations from Brownlee Reservoir have some uncertainty because the analytical reporting limits for the available data were sometimes close to, and in the case of PCBs, greater than the most stringent applicable water quality criteria.

Table 2.3.1. Baseline concentrations of organic pollutants in sediments and fish tissue measured in waters within the action area, or upstream waters that drain into the action area.

Substance	Most stringent water criteria from Table 1.3.1	Water - measured values (range)	Sediment (range)	Fish tissue, any species (range)
	µg/L	**µg/L**	**mg/kg dry weight**	**mg/kg wet weight**
Endosulfan (α and β)	0.056	<0.0007	<0.001	No data
Aldrin	0.00014	<0.0005	<0.001	<0.005
Chlordane	0.00057	0.00082	<0.002	0.020
4,4'-DDT (note 1)	0.00059	<0.00066	0.0081	0.072
Any DDE/DDT metabolite	None	0.00015	0.011	3.3
Dieldrin	0.00014	0.00093	0.0007	0.037
Endrin	0.0023	<0.00017	<0.002	<0.005
Heptachlor	0.00021	<0.00097	<0.001	<0.005
Lindane (gamma-BHC)	0.063	No data	<0.001	<0.005
Polychlorinated biphenyls (PCBs)	0.000045	<0.1	<0.05	0.160
Pentachlorphenol (PCP) (note 2)	6.2	0.00047	<0.001	<0.005
Toxaphene	0.0002	No data	<0.2	0.26

Data sources: Water data from Brownlee Reservoir, 2011, Idaho Power Co., unpublished data; Sediment and Fish tissue, various locations in Idaho although highest values tended to be from Brownlee Reservoir (Clark and Maret 1998). Note 1: Sediment and tissue DDT samples are as p,p'-DDT; Note 2: as pentachloroanisole, the principal degradation product of PCP.

2.4. Effects of the action on the species and its Designated Critical Habitat

"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 (50CFR 402.02). Indirect effects are those that are caused by the proposed action and are later in time, but still are reasonably certain to occur.

This analysis identifies potential effects of each of the criteria that would be expected to occur if water concentrations were equal to the proposed criteria.

NMFS' general analytical approach for evaluating effects for the various chemical criteria under consideration was to first consider general issues related to EPA's methodology for deriving the criteria, which affect all or multiple criteria. We then evaluated the individual constituent criteria for potential species or habitat effects on listed salmon and steelhead. Consistent with the two

part structure of EPA's aquatic life criteria, on which the proposed Idaho criteria are based, with CMC to protect against short-term effects of exposures to criteria chemicals, and a CCC to protect against long or indefinite term exposures, the protectiveness of the CMCs were evaluated against data on effects in short-term exposures (≤ 96 hours) and CCCs were evaluated against data on effects in longer-term exposures.

In most instances, direct testing evidence for the listed salmon species was not available, and test data obtained with other fish species was used as surrogate estimates of potential effects to listed salmon. Steelhead were an exception, since they and rainbow trout are different forms of the same species (Behnke and Tomelleri 2002; Quinn 2005). In most cases, rainbow trout data were available since rainbow trout are commonly tested in ecotoxicology. Rainbow trout are often used as a surrogate for all listed *Oncorhynchus*, using geometric means. At least with several metals, rainbow trout are probably similar in sensitivity to Chinook salmon and probably considerably more sensitive than sockeye salmon. Few direct data with sockeye salmon were located, which may be related to Chapman's (1975) recommendation against testing sockeye salmon following his observations that they were much less sensitive to metals than were Chinook or coho salmon or rainbow/steelheads (Chapman 1975).

In addition to Idaho's aquatic life criteria, EPA has also approved Idaho criteria designed to protect human health from recreational, fish consumption, and drinking water uses which are also applicable to the waters in the action area. In practice, when multiple criteria are applicable to the same water body, the most stringent criteria will drive discharge limits and other pollution management efforts (IDEQ 2007a; subsection 70.1, "Applicability of standards, multiple criteria"). For our analysis, if review of the aquatic life CCC indicated that adverse effects to listed species or their habitats were likely, then we reviewed the human health-based ambient water quality criteria concentrations for the same substance to see if the human-health concentrations would be protective of the listed steelhead and salmon.

2.4.1. Evaluation of issues that are common to multiple aquatic life criteria

All criteria being evaluated as part of this action were developed by EPA following EPA's guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses. For short, these are referred as the "Guidelines" (Stephan *et al*. 1985). Thus it is important to consider the structure of the Guidelines in regard to protection of listed salmon, steelhead, and their critical habitats to evaluate whether criteria derived following them would likely be protective.

The EPA's Guidelines for criteria development represent the best judgments of a committee of EPA scientists as of the mid-1980s. As the title states, the objectives of the criteria development was the "protection of aquatic organisms and their uses." Because the Guidelines are quite detailed and have much explicit guidance, their use has tended to make criteria documents (the supporting documents prepared by EPA in deriving national recommended water quality criteria) objective, transparent, and reproducible. However, the Guidelines recognize that ecotoxicology and criteria derivation cannot be reduced to a series of decision rules, and many judgments are required to produce an individual criteria document. Because the Guidelines are fundamental to

criteria, they are fundamental to the evaluation of the protectiveness of criteria for ESA-listed species and habitats. The fundamental assumptions and procedures in the Guidelines are inherent to their degree of protectiveness for listed salmon and steelhead. Thus some of key criteria derivation steps are briefly described here and the underlying assumptions are critically examined.

The Guidelines include some fundamental assumptions:

- Effects which occur on a species in appropriate laboratory tests will generally occur on the same species in comparable field situations.

- For a given substance, if average species sensitivities are rank ordered, the species sensitivity distributes itself in a rather consistent way for most chemicals. Thus, each species tested is not representative of any other species but is one estimate of the general species sensitivity (i.e. a point along the distribution).

- The goal of aquatic life criteria is to protect aquatic communities and socially valued species within those communities. Aquatic organisms may have ecologically redundant functions in communities. The loss of some species might not be important if other species would fill the same ecological function. Thus it is not necessary to protect all of the species all of the time.

- If 95% of the species in acceptable datasets were protected, that would be sufficient to protect aquatic ecosystems in general. In the ecological risk assessment literature, this is often referred to as the 5^{th} percentile of a species sensitivity distribution (SSD) or shortened to the HC5 approach, for the hazardous chemical concentration adversely affecting no more than 5% of the species in a natural community.

- To estimate a criterion protective of 95% of the species, it is acceptable to extrapolate from compilations of severely toxic effects from short-term, "acute" tests to less severe effects in long-term, "chronic" exposures.

- If one or more water quality characteristics such as temperature, pH, or water hardness affect the acute toxicity of a substance in a predictable way, then the acute criterion for that substance should be expressed as a function of that characteristic. It is acceptable to assume that toxicity relationships established with short-term exposure data, such as those between water-hardness and metals toxicity, would be the same in long-term exposures. Thus acute-toxicity and hardness or other relations may be applied equally to chronic criteria (Stephan *et al.* 1985; Stephan 1985; Stephan 2002)

Relying on these assumptions, the EPA Guidelines are derived with the following general steps (Stephan *et al.* 1985):

- First, datasets of acute (short-term) responses of aquatic organisms to the substance of interest are compiled and screened for data sufficiency, relevance and quality.

- If a water quality characteristic is considered to affect the toxicity of the substance, then a relation is developed and the acute data are normalized to a common water condition. For example, with several metals, hardness-toxicity regressions were developed and used to adjust acute toxicity values to a common hardness of 50 mg/L.

- The adjusted acute data are averaged to obtain species mean acute values (SMAVs), and SMAVs are averaged to obtain genus mean acute values (GMAVs). The GMAVs are rank ordered, and value close to the 5^{th} percentile most sensitive genus is calculated, called the final acute value (FAV). The FAV is divided by 2 to extrapolate from a lethal concentration for sensitive taxa to a concentration expected to kill few sensitive taxa. The FAV/2 value becomes the CMC, which is commonly referred to as the acute criterion.

  [In this procedure, if multiple values for a species were available, with differing sensitivities, a geometric mean of all values was taken to calculate the SMAV. If different SMAVs were available, a geometric mean was similarly calculated. For example, with EPA's 1984 copper criteria, the SMAVs for Chinook, Coho and Sockeye salmon were calculated as 42, 70, and 233 µg/L, and a GMAV of 89 µg/L was calculated to represent all *Oncorhynchus*. In that era, steelhead and rainbow trout were considered in a different genus, *Salmo*.]

- Chronic (long-term) data are compiled, and acute-to-chronic ratios (ACRs) are calculated for at least 3 species. These are calculated by matching acceptable acute and chronic tests and dividing the acute LC_{50} by the "Chronic Value" from the chronic test. The chronic value in turn is calculated as the geometric mean of the highest tested concentration in which selected responses were not statistically significantly different from the controls, called the no observed effect concentration (NOEC), and the lowest concentration that was statistically different from the controls, called the lowest observed effect concentration (LOEC). The selected responses considered are survival, growth, and reproduction, data on other sublethal effects such as swimming performance, or altered behaviors are put aside. The available ACRs are then selectively averaged, for a Final ACR for the substance. The continuous criterion concentration (CCC), commonly called the chronic criterion then becomes the FAV divided by the final ACR (Stephan *et al.* 1985).

This synopsis reflects the most common way the Guidelines were used with the criteria evaluated in the Opinion, but obviously doesn't reflect all the details of Stephan *et al*.'s (1985) 98 page document.

These steps and other key judgments and practices from the EPA Guidelines for developing aquatic life criteria are critically evaluated in the following parts of this section.

2.4.1.1. The assumption that not harming more than 5% of the species tested in laboratories is sufficient protection of ESA-listed species and critical habitats

The EPA's fundamental approach to setting criteria involves compiling reports of laboratory tests for species and genus mean values, rank ordering the genus mean values, and basing criteria on the 5th percentile of a distribution of the rank ordered values. This approach has been the subject of much criticism and controversy in the ecotoxicology literature. Many arguments relate to further inherent assumptions required of the approach that may not be met, are untested, or are untestable. Published concerns include:

- Whether haphazard collections of data from single-species laboratory toxicity tests can be considered relevant to natural ecosystems;

- Small datasets can be significantly biased toward more or less sensitive species than would be expected in natural ecosystems;

- Whether any species loss from a community due to a toxin is acceptable. Reducing community integrity to a simple proportion of species could discount keystone or dominant species if they were in the lower 5th percentile of sensitivity;

- Whether the 5th percentile of the SSD as the appropriate level of protection is a scientifically sound number or just a familiar number;

- Because the approach depends on comparable data, it is biased toward mortality data (which are most abundant) and biased against less abundant data on abnormal behavior or other sublethal data that may be as important for maintaining biological integrity and more relevant at low, ambient concentrations;

- The few species for which multiple tests results are available sometimes show high variability in sensitivity, yet this variability is often omitted from SSD presentations, which implies greater precision than is the case. Thus apparent differences between species' ranks on a SSD may not be meaningful, especially for species with only single or few datapoints; and

- Uncertainties in the statistical properties of the distributions and appropriate models.

(Cairns 1986; Forbes and Forbes 1993; Hopkin 1993; Smith and Cairns 1993; Underwood 1995; Power and McCarty 1997; Aldenberg and Jaworska 2000; Newman *et al*. 2000; Forbes and Calow 2002; Suter *et al*. 2002; Duboudin *et al*. 2004; Brix *et al*. 2005; Maltby *et al*. 2005; Forbes *et al*. 2008)

In contrast to these many criticisms, other studies or reviews have found reasonably good agreement between effects in laboratory and field tests (Geckler *et al*. 1976; de Vlaming and Norberg-King 1999), and lack of pronounced adverse effects in ecosystem tests at criteria-like concentrations below the 5th percentiles of SSDs (Versteeg *et al*. 1999; Mebane 2010).

No explicit consideration of protection of exceptionally vulnerable populations of threatened or endangered species was included in the criteria guidelines. However, it is clear from contemporaneous and subsequent writings by the authors that they thought criteria should specifically protect or be adjusted to protect socially valued special status species, including threatened and endangered species. For instance, the introduction to the Guidelines states that "*to be acceptable to the public and useful in field situations, protection of aquatic organisms and their uses should be defined as prevention of unacceptable long-term and short-term effects on (1) commercially, recreationally, and other important species….*" as well as fish and invertebrate assemblages (Stephan *et al*. 1985). Other writings and guidance are more explicit about the need to consider protection of species listed under the ESA; suggesting a review of whether the 95% of protected species included listed species and adequate prey for them (Stephan 1985, 1986; EPA 1994). If not, the criteria should be adjusted to protect these "critical" species. Such reviews and adjustments were recommended to be done on a site-specific basis, where a "site" may be a state, region, watershed, water body, or segment of a water body (EPA 1994). The recommendation to consider listed species at the "site" rather than national level was not stated but presumably related to complexity and the fact that imperiled species often have limited distributions.

2.4.1.2. The assumption that effects in laboratory tests are reasonable predictors of effects in field situations

The preceding discussion concerned whether compilations of laboratory test values were appropriate to treat as surrogates of the diversity of natural systems. A related but even more fundamental question 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 likely a bias in the sensitivity of responses from either the lab or wild settings? That is, are the effects of chemical contamination more likely to be 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.

While by definition, laboratory toxicity testing is conducted in controlled, artificial condition rather than in the wild under uncontrolled conditions, some laboratory tests are designed such that they are of questionable environmental relevance. By "environmentally relevant" in the context of interpreting laboratory toxicity tests we mean whether the test conditions were designed in a way to be relevant to conditions that might occur in the environment. Whether or not test data were environmentally relevant include the questions such as: Were fish or other organisms exposed to chemicals in concentrations ranges and ratios that actually occur in the environment? Or were organisms exposed to conditions contrived to produce effects, such as massive doses over short time periods? Were organisms exposed in a manner similar to that in the wild such as by water across the gills or diet? Or were organisms exposed in a manner designed to produce effects but wouldn't occur outside of laboratories, such as injection or a bollus in feed? In feeding studies, were chemicals in a form similar to that that might be

encountered in ambient conditions? In water studies, was the dilution water a natural water type, rather than a preparation with mineral content unlike that that would occur in nature? "Environmental relevance" cannot be a hard and fast test, because studies would then be limited to field studies, which have the converse problem of being uncontrolled and difficult to unambiguously attribute apparent effects to causes. However, some studies clearly have little direct environmental relevance, and these studies are given less reliance in this opinion than "environmentally relevant" studies. For instance, in vitro tests using excised tissues, or cell lines bathed in a dosed solution are often valuable for investigations comparative biochemistry orphysiology, or on mechanisms of toxicity, but standing alone, have little direct relevance responses of a whole, living organism under conditions experienced in the wild.

There are myriad of 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. A number of reasons why the effects of a chemical could be more- or less-severe on listed steelhead and salmon in laboratory or in wild settings were considered and are summarized in table 2.4.1.1.

Table 2.4.1.1. Reasons why the effects of a chemical substance could be more- or less-severe on listed steelhead and salmon in laboratory or in wild settings

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 skinny 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 starvation of an acute toxicity test, the test may be rejected from inclusion in the criteria dataset. In the wild, not all fish can be assumed to 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 (Hodson and Sprague 1975; Kovacs and Leduc 1982b; Dixon and Hilton 1985; Erickson *et al*. 1987; Lemly 1993b; Hansen *et al*. 2002a).
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 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. Chemical exposures in soft water can be expected to exacerbate effects (Adams 1975; Kovacs and Leduc 1982b; McGeer *et al*. 2000; De Boeck *et al*. 2006).
Disease and parasites	In the wild. Disease and parasite burden are common in wild fish, but toxicity tests that used diseased fish are likely to be considered compromised and results would not be used in criteria compilations. Chemical exposure may weaken immune responses and increase morbidity or deaths (Stevens 1977; Arkoosh *et al*. 1998a,b).
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; Phillips 2003; Scott *et al*. 2003; Labenia *et al*. 2007).

Factor	Are effects likely more severe in typical lab settings or in the wild?
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 four day interval per 3 years), will have an average concentration that is less than the criteria concentration. For some chemicals, such as copper, fish might detect and avoid harmful concentrations if clean-water refugia were readily available.
Metal form and bioavailability	Uncertain. Metals other than Hg and some organics are commonly assumed to be more bioavailable in the lab because dissolved organic carbon (DOC), 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 order to be used in criteria (Stephan *et al*. 1985), but probably more often TOC is <2 mg/L in laboratory studies However, in mountainous streams in Idaho, TOC is often as low (≈1-2 mg/L) during baseflow conditions (Appendix C), so differences in bioavailability between streams and laboratory waters that both have low TOC are not necessarily large. (Organic carbon is more often discussed as DOC in this Opinion. TOC includes particulates, which other than during runoff conditions in streams will tend to be low and thus TOC and DOC would be similar during conditions without runoff).
Chemical equilibria	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*. 2007a). Likewise, in conventional laboratory flow-through test designs chemicals may not have long enough contact time to reach equilibria. 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 potentially 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 (Chapman 1983, 1985; Sprague 1985; Brinkman and Hansen 2007). (further discussion follows in the text).
Life stages exposed	In the wild. Most lab studies are short term; 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 YOY fry, sensitivity can vary substantially (this review). Unless the most sensitive life stages are tested, lab tests could provide misleadingly high toxicity values for listed species (further discussion follows in the text).

Factor	Are effects likely more severe in typical lab settings or in the wild?
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., Norwood *et al*. 2003; Borgert 2004; Playle 2004; Scholz *et al*. 2006; Laetz *et al*. 2009). In general, it seems prudent to assume that if more than one toxicant were jointly 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. However, the magnitude or increased effects at environmentally relevant concentrations is uncertain and for some combinations may be slight or imperceptible.
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 (e.g., dioxins, PCBs) dietary exposures are more important than water exposures as is the case for some inorganics (As, Hg, Se). For some other metals (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 to fish (Hansen *et al*. 2004; Schlekat *et al*. 2005; Erickson *et al*. 2010). However, while dietary exposures of metals have not yet been implicated in adverse effect to fish at or below criteria concentrations, they may in fact be both the primary route of exposure and an important source of toxicity for benthic invertebrates (Irving *et al*. 2003; Poteat and Buchwalter 2014). For instance Besser *et al*. (2005a) found that the effects threshold for Pb to the benthic crustacean *Hyalella* 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* caused a 50% growth reduction at about the NTR chronic criteria.
Population dynamics	
Density effects	In the lab. Salmonid fishes are highly fecund (~500 to 5000 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 YOY 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 YOY fish prior to their entering a resource bottleneck, then assuming no residual contaminant effects, the losses to later life stages and to adult spawners would be buffered.
Meta-population dynamics	In the lab. If habitats are interconnected, as is the case in intact stream networks, then 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).

Considering all the reasons why the effects of a given chemical concentration could have more or less severe effects in laboratory settings or the wild, general conclusions are elusive. It may be that the best overall conclusion is the same as that reached by Chapman (1983) that "*when appropriate test parameters are chosen, the response of laboratory organisms is a reasonable index of the response of naturally occurring organisms.*" His conclusion in turn contributed to one the most fundamental assumptions of EPA Guidelines, that is, "*these National Guidelines have been developed on the theory that effects which occur on a species in appropriate laboratory tests will generally occur on the same species in comparable field situations.*"

Summary: Based on this analysis, the assumption that effects in laboratory tests are reasonable predictors of effects to species in the wild is dependent upon the specific factor being considered. While it is generally reasonable to interpret effects from laboratory tests as being applicable to field situations where criteria are applied, there is some risk that laboratory tests may underpredict effects in the wild.

2.4.1.3. Susceptibility of Salmonids to Chemicals at Different Life Stages

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 at least a factor of two more resistant than other life stages, 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 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 and 5.0g in weight (ASTM 1997).

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 5.0g 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 indicated that in practice, there were sometimes greater influences of life stage on the sensitivity of salmonids to some substances than was apparent to the authors of the individual criteria documents using the datasets available to them at the time. Some of the SMAVs and GMAVs which were used to rank species sensitivity and set criteria were considerably higher than EC_{50}s with salmonids that were tested at the most sensitive life stages (Figures 2.4.1.1 to 2.4.1.4).

For three Pacific salmonid species for which comparable test data were available for different life stages; coho salmon (*Oncorhynchus kisutch*), rainbow trout (*O. mykiss*) and cutthroat trout (*O. clarki*), the data suggest that swim-up fish weighing around 0.5g to about 1g may be the most sensitive life stage. None of the data sets examined in detail or other published studies reviewed had sufficient resolution to truly define at what weight fish became most sensitive to metals, but along with other data they suggest that larger fish may be less sensitive than fish at 0.4 to 0.5g. For instance with zinc, rainbow trout in the size range of about 0.1 to about 1.5 g consistently became more sensitive to zinc in two studies with multiple tests in that size range (Figure 2.4.1.2

and Figure 2.4.1.3). The paucity of data with salmonids in the size range of about 0.5 to 2g prevents definitive statements of a most sensitive size across species or even tests. All data located for early swim-up stage *Oncorhynchus* in the 0.1 to 0.5g range were consistent with increasing sensitivity with size. With Hansen *et al's.* (2002c) rainbow trout studies, this relationship continued with fish up to about 1.5g. However, with cutthroat trout, the few data available suggests that fish larger than about 0.5g become less sensitive with increasing size (Figure 2.1.4.2).

Some studies with older and larger rainbow trout have found that the fish became more resistant to zinc and copper (Chapman 1978b; Chapman and Stevens 1978; Howarth and Sprague 1978; Chakoumakos *et al*. 1979). 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 eight times (Figure 2.4.1.4), but steelhead (*O. mykiss*) that were tested with copper at sizes of 0.2, 7, 70, and 2700g showed little pattern of sensitivity with size (Chapman 1978b; Chapman and Stevens 1978). However, the large differences in sizes may have missed changes at intermediate sizes in the ranges compared at Figures 2.4.1.1 to 2.4.1.4. Similarly, with copper and rainbow trout, Anderson and Spear (1980) found that three sizes of rainbow trout (3.9, 29 and 176g) had similar sensitivities.

NMFS reviewed several data sets that indicated increasing susceptibility of salmonids to at least metals with increasing size and age as fish progressed from the resistant alevin stage. The "U" shaped size-sensitivity response with the most sensitive life stage for salmonids fish around 0.5g in weight seems a reasonable interpretation of the available data, but few data were available in the size range of 0.5 to 2g, so it is possible the most sensitive stage is larger. Hedtke *et al*. (1982) tested coho salmon for the influence of body size and developmental stage with copper, zinc, nickel, and PCP. Fish were exposed as alevins, swim-up fry, and juveniles, and within these developmental stages smaller fish were tested against larger fish. For copper, zinc, and PCP, the swim-up fry stage was most susceptible, and within the swim-up stage, the larger fish were more susceptible to copper and zinc than smaller fish (~0.25g vs. 0.7g fish, wet weight). For PCP, there was no difference for size of fish within the sensitive alevin to swim-up stage, and with Ni all fish were very resistant (Hedtke *et al*. 1982). In three test pairs with rainbow trout exposed to cadmium and zinc under similar hardness, pH, and temperature, the fish tended to become more sensitive with increasing size from 0.4 to 0.9g for rainbow trout and zinc, and 0.26 to 0.66g with Cd. Further growth in juvenile rainbow up to 1.1 and 1.6g for cadmium and zinc had little effect on sensitivity (Figure 2.4.1.3). In parallel tests with bull trout (*Salvelinus confluentus)*, size had little effect on sensitivity over a range of 0.08 to 0.22g for cadmium although with zinc; however, the smallest fish (0.1g) were also least sensitive (Hansen *et al*. 2002c). Similar tests with copper and rainbow and bull trout showed roughly similar patterns. Three tests with rainbow trout at the same hardness and using fish from the same source had the most sensitive results for 0.43g fish (LC_{50}s of 36, 54, and 93 µg/L for rainbows weighing 0.43, 0.3, and 0.68g, respectively). Bull trout tested at constant temperature of 8°C tended to become more sensitive with increasing size up to ~1g (Hansen *et al*. 2002a). Besser *et al*. (2007) similarly found that 0.5g rainbow trout were more sensitive than 0.13g fish to copper and zinc, but not for cadmium.

These patterns do not seem to hold for all species. Contrary to the patterns with the salmonids, newly hatched sculpins were more sensitive to cadmium, copper, and zinc than were older juveniles (Besser *et al*. 2007). Similar to the sculpin results but contrary to all the other salmonid results, Carney *et al*. (2008) found that the brown trout (*Salmo trutta*) became less sensitive to copper with increasing size. Guppies exposed to toxicants with different modes of action tended to become more susceptible with increasing size and age (dieldrin, PCP, cyanide, copper, zinc, and nickel) (Anderson and Weber 1975).

Summary: Salmonids can have profound differences in susceptibility to chemicals at different life stages, and in some instances, species mean acute values used in criteria may be skewed high because insensitive life stages were included. A "U" shaped pattern of sensitivity with life stage was suggested for several datasets with Pacific salmon or trout species (i.e., *Oncorhynchus)* and some metals. Across several good datasets, the most vulnerable life stage and size appeared to be swim-up fry weighing between about 0.5 to 1.5g. However, no consistent pattern was obvious across other species of fish, chemicals, and life stages.

Caution is needed when using SMAVs or GMAVs as summary statistics for ranking species sensitivity or setting criteria. Reviews of the protectiveness of chemical concentrations or criteria that rely in large part upon published mean acute values for species of special concern such threatened species, or their surrogates, may be subject to considerable error if the underlying data points are not examined. This may include analyses such as SSD, interspecies correlation estimates (ICE, Asfaw *et al*. (2004), or any other relative sensitivity comparisons that uses mean acute values at the family, genus, or species level.

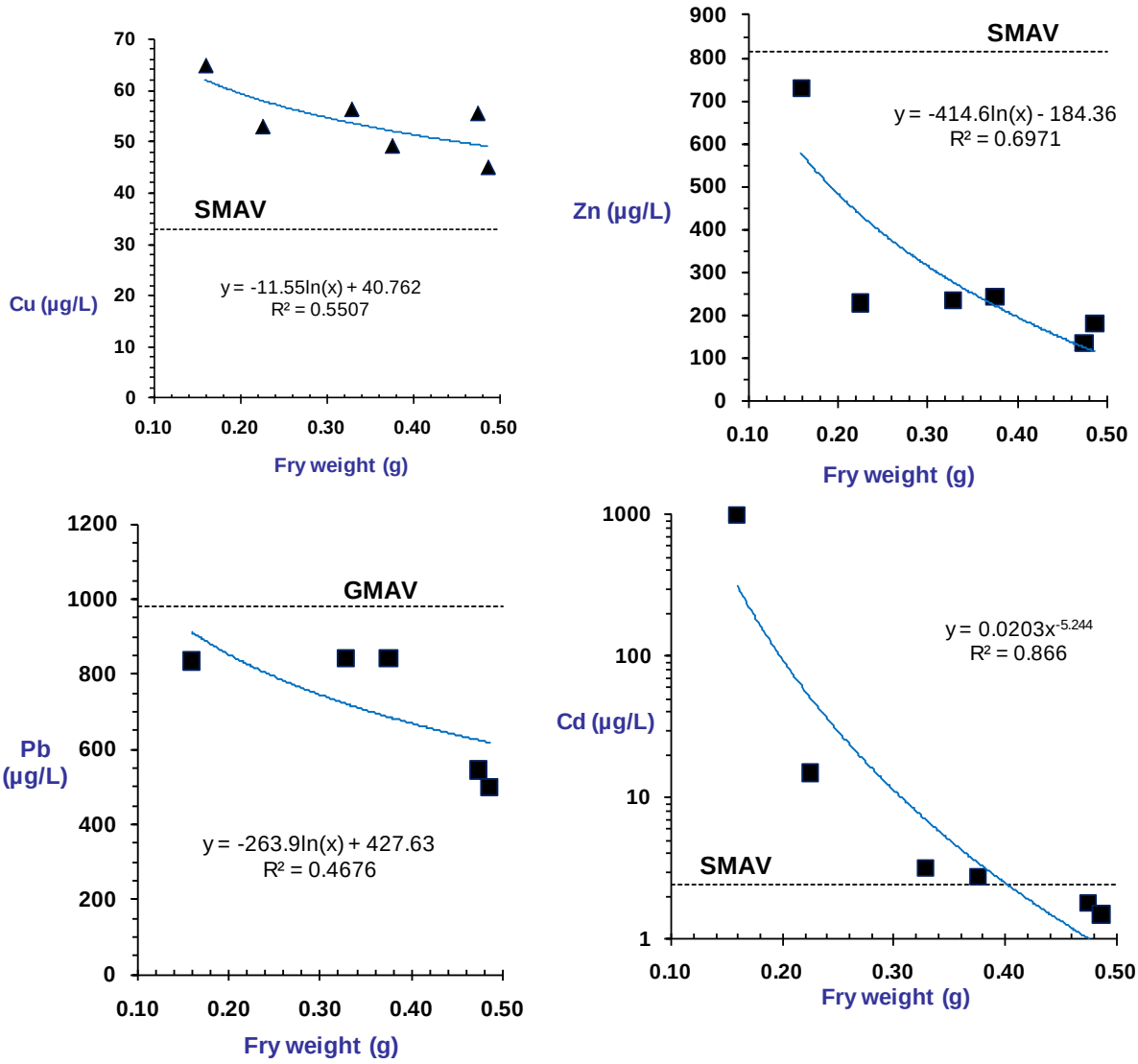


Figure 2.4.1.1. Size-developmental stage patterns with coho salmon from 2 to 7 weeks post hatch, data from Chapman (1975). Species and genus mean acute values (SMAVs and GMAV) are from the respective criteria documents (EPA 1984b, 1984a, 1985, 1987b), adjusted to test water hardness. All tests used Willamette River water, TOC 3.4 mg/L, hardness 22 mg/L.

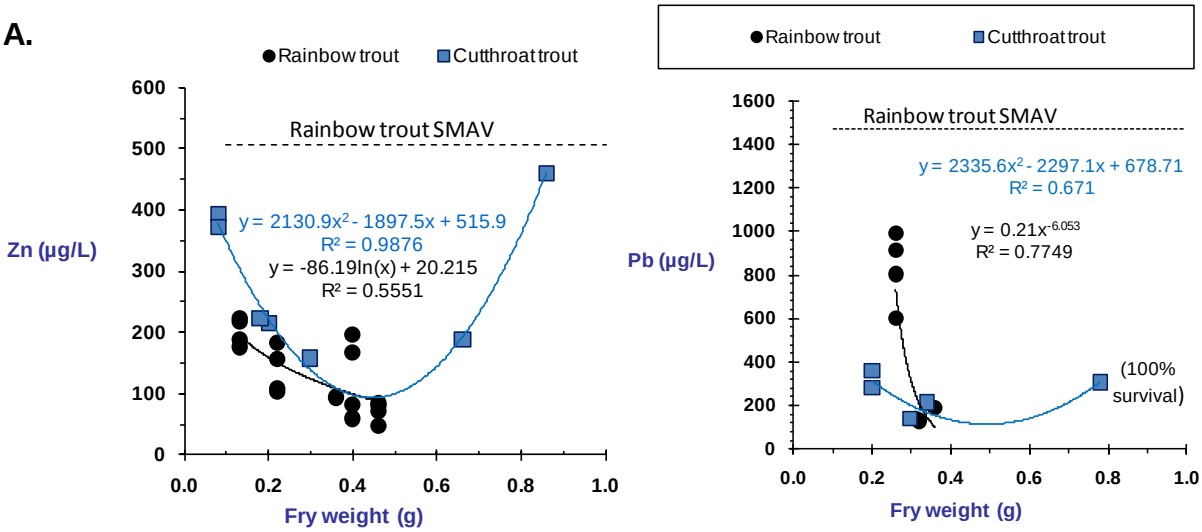


Figure 2.4.1.2. Relations between size of swim-up rainbow and cutthroat trout and toxicity to zinc and lead sensitivity in renewal tests conducted in water from the South Fork Coeur d'Alene River, Idaho. Data from (Mebane *et al*. 2012). All test values adjusted to a median test hardness of 35 mg/L $CaCO_3$ using hardness-toxicity regressions from (Mebane *et al*. 2012). SMAVs were adjusted using the hardness-criteria equations from the respective criteria documents.

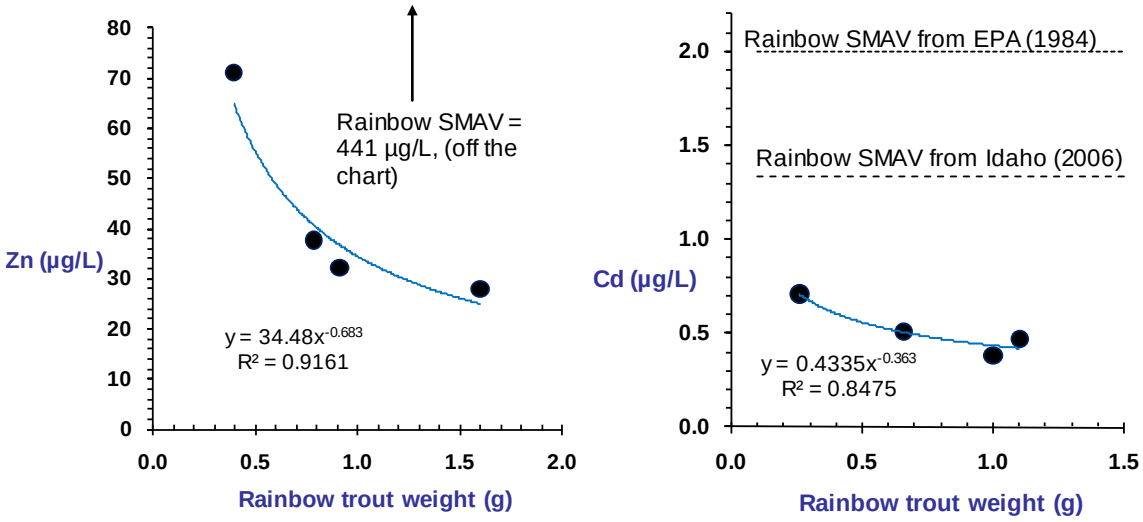


Figure 2.4.1.3. Resistance to cadmium and zinc toxicity decreased with increasing size over a weight range of 0.2 to 1.6g for swim-up rainbow trout. Data from Hansen (2002a) and Stratus (1999) using 96-h probit LC_{50} values. All tests conducted at a hardness of 30 mg/L and pH of 7.5 SMAV values were adjusted using the hardness-criteria equations from the respective criteria documents.

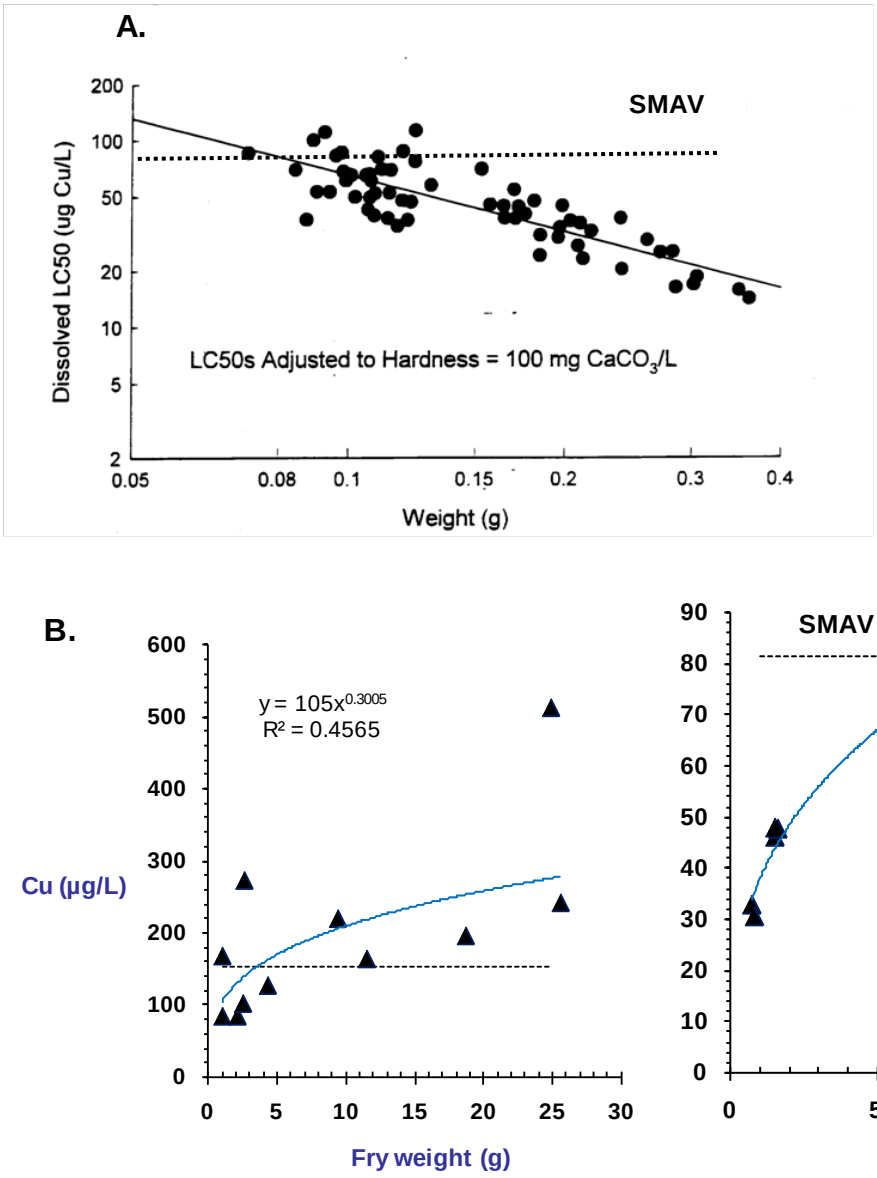


Figure 2.4.1.4. Resistance to copper toxicity decreased with increasing size over a weight range of 0.06 to 0.4g for swim-up rainbow trout, but above about 1g weight, resistance to copper toxicity increased with increasing size. Dashed lines indicate hardness-adjusted rainbow trout species mean acute value (SMAV) from EPA (1984). A. Relation between copper toxicity and the size of swim-up rainbow trout (<0.5g), from renewal tests conducted in water from the Clark Fork River, MT (Erickson *et al*. 1999); B. Relation between copper toxicity and the size of larger juvenile rainbow trout (>0.7g, older than swim-up fish), data from Chakoumakos *et al*'s (1979) tests under uniform water conditions (hardness 194 mg/L); C. Rainbow trout of difference sizes tested under uniform conditions at hardness 99 to 102 mg/L, data from Howarth and Sprague (1978).

2.4.1.4. 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 increased resistance may be on the order of two to four times for acute exposures, but may be much higher for some organic contaminants (Chapman 1985). However, the increased resistance can be temporary and can be lost in as little as 7 days after return to unpolluted waters (Bradley *et al*. 1985; Sprague 1985; Hollis *et al*. 1999; Stubblefield *et al*. 1999). For this reason, EPA's Guidelines specify that test results from organisms that were pre-exposed to toxicants should not be used in criteria derivation (Stephan *et al*. 1985).

However, there is a less obvious source of acclimation that is not precluded by the Guidelines and influences chronic values and thus chronic criteria. Several tests have shown that life stages typically sensitive to toxins (e.g., fry stage) become more resistant when toxicity tests were initiated during resistant early life stages (ELS, e.g., embryo stage). This suggests that acclimation to toxin(s) during ELS exposure may lead to greater resistance in later life stages in comparison to the same life stages of naïve fish (fish which had no previous exposure) (Chapman 1978a; Spehar *et al*. 1978; Chapman 1994; Brinkman and Hansen 2004, 2007). The Guidelines could actually be interpreted to exclude chronic exposures that did not pre-expose, and acclimate fish to metals as eggs (Stephan *et al*. 1985), which was probably unintended.

Chapman (1994) 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 which were initially exposed as embryos was no different from 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 which were initially exposed as swim-up fry without the opportunity for acclimation during the embryo state, suffered complete mortality *(*Figure 2.4.1.4*)*. 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., ELS 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 which 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, their life histories often involve spawning migrations to headwater reaches of streams, followed downstream movements of fry shortly after emerging from the substrates, and followed by further seasonal movements to larger, downstream waters to overwinter (Willson 1997; Baxter 2002; Quinn 2005). These life history patterns often correspond to human development and metals pollution patterns such that headwater reaches likely have the lowest metals concentrations, and downstream increases could occur due to point source discharges or urbanization.

From the discussion in the Guidelines 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, at p. 44).

Summary: In chronic tests with salmonids and metals, the Guidelines inadvertently favor a test method (ELS tests) that may be inherently biased toward insensitivity because acclimation can occur during the insensitive egg stage of exposure. Thus, Species Mean Chronic Values listed in criteria documents may be also be biased high.

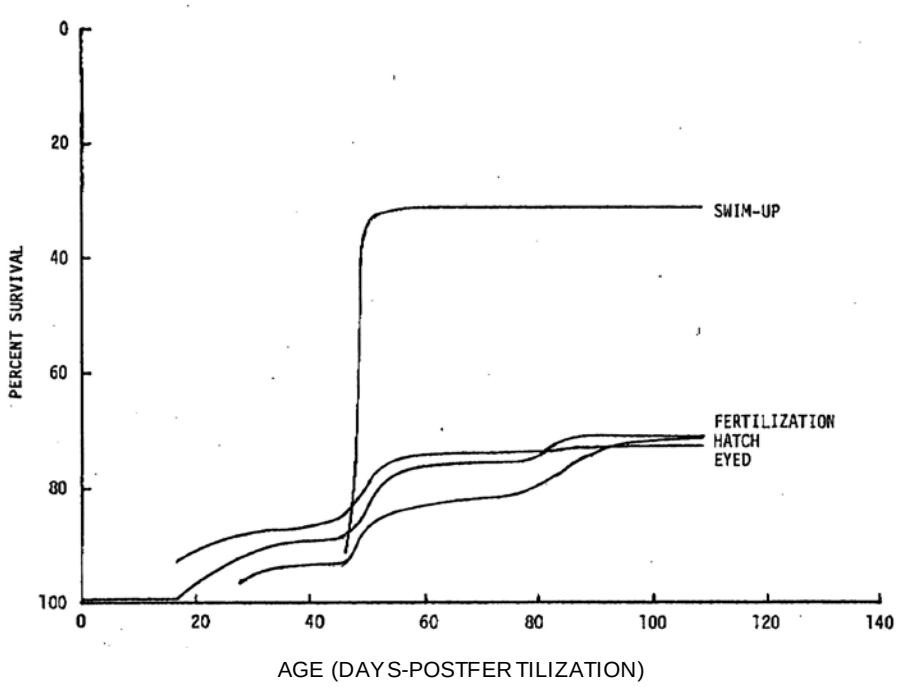


Figure 2.4.1.5. Effect of developmental stage at the onset of continuous copper exposure (13.4 µg/L) on the survival of juvenile steelhead trout (figure from Chapman 1994).

2.4.1.5. Implications of the use of the "chronic value" statistic in setting criteria

A related issue with the derivation of 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 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 criteria

documents reviewed for this consultation. In practice for most of the criteria documents 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% confidence level (lowest observed effects concentration [LOEC]) and the next lower tested concentration (no observed effects concentration [NOEC]). The "chronic value" as used in individual criteria documents is effectively the same thing as the maximum acceptable toxicant concentration (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% accuracy (i.e., Type I error or false positive, the risk of declaring an effect was present when in fact the apparent effects only occurred by chance). 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 but because of the limited sample size or variability, were not significant with 95% confidence).

The magnitude of effect that can go undetected with 95% confidence in a NOEC statistic can be large, greater than 30% on average for some endpoints, and much higher for individual tests (Crane and Newman 2000). This problem is compounded with the "chronic value" or MATC when calculated in its most common form as the geometric mean of a NOEC and LOEC. For instance, 100% 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 1985d) is that it represents a concentration that can be expected to kill somewhere between all or no brook trout in the test population. Similarly, Grosell *et al*. (2006a) showed that the NOECs and LOECs for reduced growth in snails exposed to lead corresponded with about a 57% and 90% growth reduction, and over 70% reduced growth for the MATC. Animals suffering such severe stunted growth may not even reproduce, so the MATC would not seem to be a very acceptable maximum toxicant concentration. Suter *et al*. (1987) evaluated published chronic tests with fish for a variety of chemicals and found that on the average the MATC represented about a 20% 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%.*" 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 criteria documents under review may represent substantial adverse effects for that test species, the criteria on the whole will be less protective than the intended goal of protecting 95% 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, the MATC-based chronic criteria would protect about 92% 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 Cd, the true level of protection is closer to about 90% than the 95% intended by the Guidelines.

A specific question for interpreting ecotoxicological data to evaluate the protectiveness of species listed under the ESA is, what level of effect is "insignificant?" "Insignificant effects" have been defined in this context to "*relate to the size of the impact and should never reach the scale where take occurs*" and "*based on best judgment, a person would not be able to meaningfully measure, detect, or evaluate insignificant effects*" (USFWS and NMFS 1998). To evaluate what test statistic best approximated a "true" no-effect concentration for evaluating risks to ESA-listed species, we made a limited comparison of NOECs versus regression or distribution-based methods for estimating no- or very low effects concentrations. The alternative statistics evaluated were the lower 95th percentile confidence limit of the concentration affecting 10% of the test population (LCL- EC10), or estimates of the EC1 or EC0 (1% or 0% effects). NMFS concluded that the EC0 was the preferred, best estimate of no-effect value from a toxicity test. However, if data were insufficient to calculate an EC0 or other regression based approaches, the NOEC may be the best available statistic for estimating "insignificant" effects (Appendix B).

Summary: The Chronic Value statistic is calculated by splitting the difference between an adverse effects concentration (the LOEC) and a concentration expected to have low adverse effects (the NOEC). However, in practice the NOEC can have more adverse effects than implied by the term "NOEC", and splitting the difference between two adverse effects concentrations produces another adverse effect concentration. Thus the Chronic Value statistic used to set chronic criteria through ACRs, etc., in practice produces an uncertain level of effect and may result in less protection than intended by the EPA Guidelines. This has been estimated to result in a level of protection was closer to about 90% of the species represented in an SSD than the 95% intended by the Guidelines.

2.4.1.6. The assumption that dividing a concentration that killed 50% of a test population by two will result in a safe concentration

One challenge for deriving aquatic life criteria for short-term (acute) exposures is that the great majority of available data is for mortality, which is a concentration that kills 50% of a test population. A fundamental assumption of EPA's criteria derivation methodology is that the FAV, the LC_{50} for a hypothetical species with a sensitivity equal to the 5th percentile of the SSD, may be divided by two in order to extrapolate from a concentration that would likely be extremely harmful to sensitive species in short-term exposures (kill 50% of the population) to a concentration expected to kill few, if any, individuals. This assumption, which must be met for acute criteria to be protective of sensitive species, is difficult to evaluate from published literature 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 usually a series of four to six tests run in parallel in order to test effects at different chemical concentrations. An

LC_{50} is estimated by a statistical distribution or regression model which generates an LC_{50} estimate, usually a confidence interval, and then all other information is thrown away. Thus, while the original test data included valuable information on what concentrations resulted in no, low, or severe effects, that information is lost to reviewers unless the unpublished raw lab data are available to them.

The assumption that dividing an LC_{50} by two will result in a no- or very low effects concentration 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 2.4.1.6. We selected these examples from data sets that were relevant to salmonid species in Idaho and for which the necessary data to evaluate the range of responses could be located (Chapman 1975, 1978b; Marr *et al*. 1995b; Marr *et al*. 1999; Mebane *et al*. 2010; Mebane *et al*. 2012).

The citations are to reports with detailed enough original data to examine the mortality at the LC_{50} concentration divided by two. The vast majority of published data was inadequate for this comparison, because usually only the LC_{50}s are reported, not the actual responses by concentration. We examined around 100 tests for this comparison. The examples shown in Figure 2.4.1.6 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% mortality. However, a more common pattern with the metals data was that an $LC_{50}/2$ concentration would probably result in about a 5% death rate (panels B and F), and in many instances, no deaths at all would be expected (panels C and D).

In one of the few additional published sources that gave relevant information, Spehar and Fiandt (1986) included effect-by-concentration information on the acute toxicity of chemical mixtures. Rainbow trout and *Ceriodaphnia dubia* were exposed for 96 and 48 hours, respectively, to a mixture of six metals, each at their presumptively "safe" acute CMC. In combination, the CMC concentrations killed 100% of rainbow trout and *Ceriodaphnia*, but 50% of the CMC concentrations killed none (Spehar and Fiandt 1986). This gives support to the assumption that dividing a lethal concentration by two would usually kill few if fish, although it does not bode well for arguments of the overall protectiveness of criteria concentrations in mixtures.

Other 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 cadmium, lead, and zinc singly and in mixtures, Dillon and Mebane (2002) found that the $LC_{50}/2$ concentration corresponded with death rates of 0% to 15%.

Summary: The assumption that one-half of an LC_{50} concentration for a sensitive test, i.e., a concentration near the 5^{th} percentile of the ranked species sensitivities, will result in little or no deaths was supported by several data sets plus two published articles. While up to 20% mortality was calculated, in most cases the expected morality associated with a $LC_{50}/2$ was less than 10% and often zero.

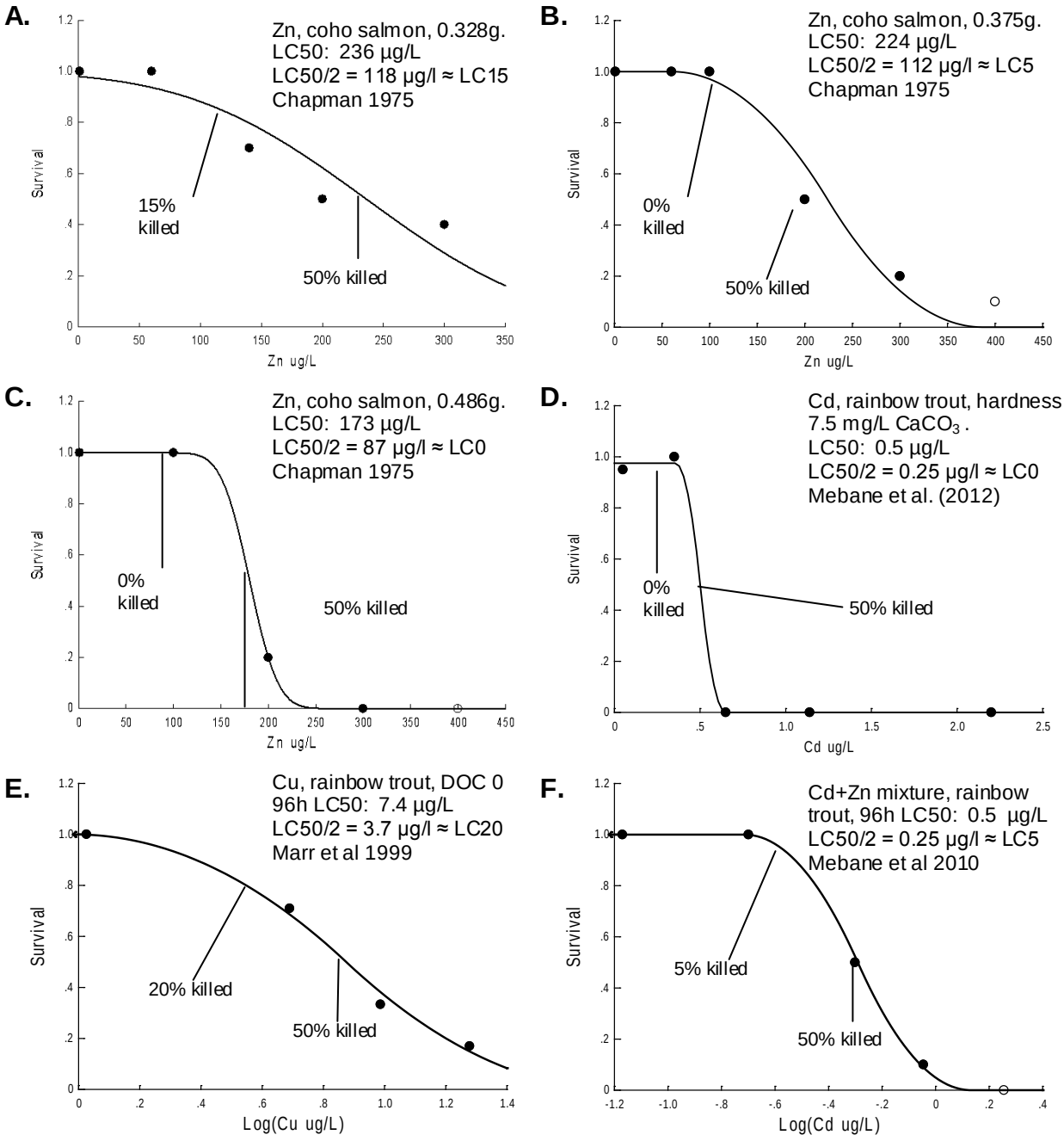


Figure 2.4.1.6. Examples of percentages of coho salmon or rainbow trout killed at one-half their LC_{50} concentrations with cadmium, copper, and zinc.

2.4.1.7. Issue 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 by 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 or 48 hours, by replacing nearly all the test solution. In the "flow-through" technique, test solution flows through the test chamber on a once-through basis throughout the test, usually with at least five volume replacements/day (ASTM 1997).

The term "flow-through test" is commonly mistaken for a test with flowing water, i.e., to mimic a lotic environment in an artificial stream channel or flume. This is not the case; rather the term refers to the once-through, continuous delivery of test solutions (or frequent delivery in designs using a metering system that cycles every few minutes). Flows on the order of about 5-volume replacements per 24 hours are insufficient to cause discernible flow velocities. In contrast, even very slow moving streams have velocities of around 0.04 ft/sec (a half 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 3,600 volume replacements per day. At more typical stream velocities of about 0.5 ft/sec would produce over 20,000 volume replacements/day.

Historically, flow-through toxicity tests were believed 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 EPA 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 1985 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 because flow-through tests are assumed have more stable exposure chemistries and will result in more precise LC_{50} estimates.

With metals, renewal tests have been shown to 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 American Society for Testing and Materials (ASTM) recommendations favoring flow-through testing, Santore and others (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 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*. 2007a), 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 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 versus ~30,000 µg/L for static tests). The fish used in the static tests were described as "immature" weighing about 2g (2000 mg). The size of the fish used in the Pickering and Gast (1972) 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 (EPA 2002c). Using newly hatched fry weighing about $1/1000^{th}$ of the fish used by Pickering and Gast (1972) in the 1960s, 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).

Summary: When all other factors are equal, it appears that renewal tests may indicate chemicals are somewhat less toxic (e.g., higher LC_{50}s), but there is no clear consensus whether this indicates that renewal tests are biased toward lower toxicity than is "accurate" or whether conventional flow-through tests are biased toward higher toxicity. Comparisons with data across studies suggest that factors such as the life stage of exposures, can dwarf the influence of flow-through or renewal methods for the acute toxicity of at least metals.

2.4.1.8. The "Water-Effect Ratio" Provision

The water-quality criteria for metals proposed in this action include a Water Effects Ratio (WER) in their equations. 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. Following EPA's (EPA 1992) precedent, the default WER value for the proposed criteria is 1.0 "*except where the Department assigns a different value*" (Idaho Department of Environmental Quality 2011, at 210.03.c.iii.).

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 (Carlson *et al*. 1984; EPA 1994; Bergman and Dorward-King 1997). 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 a standard reconstituted laboratory water. The results in the laboratory water are presumed to represent the types of waters used in tests used in EPA criteria documents. 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 DOC to ameliorate toxicity, which is not correlated with hardness.

The main problem with the 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 groundwaters (well waters), natural surface waters 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*. 1994b) specified using recipes from EPA or ASTM for making standardized water that results in a water hardness with unusually low calcium relative to magnesium concentrations compared to that of most natural waters ("hardness" is the sum of equivalent concentrations of calcium (Ca) and magnesium (Mg) and is discussed more in Section 2.4.2, "The Influence of Hardness on Metals Toxicity"). This has the effect of making metals in the reconstituted laboratory waters made by standard recipe more toxic than would be expected in waters with more natural proportions of calcium and magnesium. This is because at least for fish and some invertebrates and copper, calcium reduces toxicity somewhat but magnesium affords little or no protection (Welsh *et al*. 2000a; Naddy *et al*. 2002; Borgmann *et al*. 2005b).

The effect of this issue is that unrepresentative lab waters can generate low EC_{50} values which when used as a denominator with higher EC_{50}s from site waters can produce extremely high-biased values. For instance, in WER testing on the Boise River, Idaho, a stream receiving treated municipal wastewater effluent, testing with *Ceriodaphnia* and copper resulted in mean site:lab WER of 18.4, which when multiplied by the copper CMC at a hardness of 40 mg/L would result in a WER adjusted CMC of 132 µg/L. Yet the *Ceriodaphnia* EC_{50}s in that same site water ranged from 18.6 to 60 µg/L (CH2M Hill 2002). Thus, the published WER procedure would generate a site-specific acute copper criterion that was three to seven times higher than concentration that killed 50% of a sensitive species in that same site water. Such a grossly unprotective site-specific criteria was argued for on the grounds that it was procedurally in accordance with the Idaho metals criteria under consultation, because it follows from the WER equation and definition in the NTR and derivative Idaho criteria. Because it arguably followed EPA's 1994 Interim Guidelines for developing Water Effect Ratios (Stephan *et al*. 1994b), whatever the outcome was, was therefore procedurally acceptable.

Both EPA and IDEQ have made steps to reduce the bias that could be introduced by low EC_{50} values in laboratory waters compared with site waters. The EPA (2001a) effectively eliminated the issue by setting the WER as the lesser of the site water EC_{50}/ lab water EC_{50} ratios or the ratio of site water EC_{50} divided by the SMAV from an updated criteria dataset. When this latter

calculation was applied to the Boise River dataset, it produced an average copper WER of 2.6 instead of 18.4 and produced a site-specific acute copper criterion of 18.5 µg/L for a hardness of 40 mg/L (CH2M Hill 2002). Given the *Ceriodaphnia* EC_{50}s of 18.6 to 60 µg/L in site water, this approach may not fully protect species as sensitive as *Ceriodaphnia* but it's an improvement. The IDEQ (2007a) regulations at subsection 210.03.c.iii specify that calcium and magnesium ratios should be similar to those in EPA's criteria laboratory waters or the water body for which WERs are to be applied. However, such an approach was used in the Boise River project and exorbitantly high WERs still resulted so it is not clear that the WER approach can be corrected in this way. Further, IDEQ's implementation procedures for NPDES permits call specifically for the use of EPA's 1994 interim procedures (IDEQ 2007a, at subsection 210.04) although IDEQ has the discretion to use "other scientifically defensible methods" as they see fit.

Other approaches by EPA that might be used as an interim, operational substitute include establishing criteria on a more mechanistic basis that can directly account for the factors that affect toxicity. One example is the biotic ligand model (BLM) which is supposed to capture the major interactions between metals concentrations, competition, and complexation that control bioavailability and thus toxicity (Di Toro *et al*. 2001; Niyogi and Wood 2004). For copper, BLM was used as the basis of EPA's (2007a) updated aquatic life criterion, which for copper at least, should negate much of the need for empirical WER testing. The predictiveness of the copper BLM over a wide range of environmental conditions makes the BLM a more versatile and effective tool for deriving site-specific water quality criteria compared to the WER method (EPA 2000c; Di Toro *et al*. 2001).

This provision has rarely been used in Idaho, but NMFS is recommending a term and condition to help reduce future risk if WERs are developed in critical habitat for listed salmon and steelhead.

Summary: While seldom used to date, the WER is a fundamental part of the formula-based water quality criteria for metals. In guidance and practice, the manner in which WERs are developed has a substantial risk of undermining the protectiveness of criteria. Procedures that are consistent with the action evaluated in this opinion could result in criteria concentrations that were higher than concentrations that were acutely toxic to sensitive organisms when tested in the same site water. Two alternate procedures could achieve the intent of the WER provision (to adjust criteria based on site-specific conditions). First, the WER could be calculated by using the lower ratio from either (a) the site water EC_{50}/ lab water EC_{50} ratios or (b) the ratio of site water EC_{50} divided by the species mean acute value (SMAV) for that test organism (e.g., Ceriodaphnia dubia, fathead minnow, or rainbow trout) from a criterion dataset as described by EPA (2001a). Second, with copper the EPA (2007) BLM-based criteria is intended to adjust for site-specific water quality differences (EPA 2007a; DiToro *et al.* 2001).

2.4.1.9. Issue of Basing Criteria on Dissolved or Total-Recoverable Metals

One difference between the proposed action and the NTR as first published by EPA (1992) is that the proposed metals criteria are defined on the basis of "dissolved" metals rather than for "total recoverable" metals. "Dissolved" metals are those that pass through a 0.45 µm filter, and

"total recoverable" metals are determined from unfiltered samples, and thus consist of both dissolved and particulate or colloidal phases. Metals sorbed to particulates are subject to gravity and will eventually settle from undisturbed water whereas dissolved metals are truly in solution and will not settle from gravity.

This criteria change was based on a 1993 EPA policy statement that "*it is now the policy of the Office of Water that the use of dissolved metal to set and measure compliance with WQSis 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).

To implement Prothro's (1993) policy change, metals criteria had to be recalculated on a dissolved basis. Because the tests in the acute and chronic datasets used to derive metals criteria were mostly reported total recoverable rather than dissolved metals, in order express metals criteria on a dissolved metals basis, a conversion was needed. To do so, Stephan (1995) evaluated what data were available on the proportions of dissolved versus total recoverable metals in different laboratories that contributed data used in the EPA metals criteria. The resulting conversion factors ranged from 0.32 with chromium (III) to 0.99 with chronic zinc. With lead, because its solubility usually decreases as hardness increases, the conversion factor for lead varies with hardness, ranging from 1 at hardness 25 mg/L to 0.69 at hardness 200 mg/L. For most metals, the conversion factors were close to 1 indicating that for the laboratory conditions under which the toxicity tests in the datasets were conducted, almost all metals were present in dissolved form (Stephan 1995)

Because no supporting documentation was given by Prothro (1993) in support of their conclusions, they are 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, no compelling evidence was found that particulate bound metals can be assumed to be non-toxic. Only two studies were located that examined the toxicity of particulate metals in controlled experimental studies. Both found toxicity associated with particulate bound copper (Brown *et al*. 1974; Erickson *et al*. 1996).

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 adsorbed metals could become desorbed, becoming more bioavailable, as the pH of water moving across fish gills decreases. If the pH of water where a fish is living is 6 or greater, then the pH will be lowered as water crosses the gill (Playle and Wood 1989). Most ambient waters in the Snake River basin action area have pH greater than 6.

A further manner in which particulate bound metals could become biologically active is through sediment or food exposure. For instance, in Panther Creek, a tributary to the Salmon River,

Idaho, total copper concentrations were measured at greater than twice that of dissolved concentrations (Maest *et al*. 1995). Copper was also greatly elevated in biofilms (algae and detritus) and sediment, and correlations between copper concentrations in benthic invertebrates and biofilms were stronger than were correlations between invertebrates and water or sediment (Beltman *et al*. 1999). Copper sorbed to sediments was also bioavailable and toxic to benthic invertebrates when exposed to Panther Creek sediments after the sediments were transferred to clean overlying water (Mebane 2002a). In this stream at the time of those studies, dissolved copper consistently exceeded dissolved criteria values, so these studies do not directly help with the question of whether streams with low contamination that largely comply with dissolved criteria could result in sediment contamination at hazardous concentrations. Others have reported toxicity from metals contaminated freshwater sediments even when overlying waters mostly are at dissolved criteria (Canfield *et al*. 1994; Besser *et al*. 2008).

Attempting to define, evaluate, and manage risks associated with 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 assumption 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.

Summary: The component of the action to define metals criteria as applying only to the dissolved fraction of metals rests on the rationale that metal particulates are less toxic than dissolved metals. Criteria are adjusted from total to dissolved metals fraction through conversion factors. The total to dissolved conversion factors for metals criteria were set in a generally conservative manner and are close to 1 for most metals. While the conversion factors per se are not a conservation problem, the concept of basing criteria solely on the dissolved fraction may not always be protective. While we concur that for divalent metals (e.g., cadmium, copper, lead, nickel, zinc), the particulate fraction is less toxic, the particulate fraction is not necessarily non-toxic. Conceptually, the particulate fractions of metals and inorganics could contribute to foodweb exposure pathways from sediments or biofilms to macroinvertebrates to fish. This is of particular concern for substances with primarily dietary routes of exposure (e.g., arsenic, mercury, and selenium).

2.4.1.10. Mixture Toxicity: criteria were developed as if exposures to chemicals occur one at a time, but chemicals always occur as mixtures in effluents and ambient waters

In point or nonpoint pollution, chemicals occur together in mixtures, but criteria for those chemicals are developed in isolation, without regard to additive toxicity or other chemical or biological interactions (Table 2.4.1.1). Whether the toxicity of chemicals in mixtures is likely greater or less than that expected of the same concentrations of the same chemicals singly is a complex and difficult problem. While long recognized, the "mixture toxicity" problem is far from being resolved. Even the terminology for describing mixture toxicity is dense and has been inconsistently used (e.g., Sprague 1970; Marking 1985; Borgert 2004; Vijver *et al*. 2010). One scheme for describing the toxicity of chemicals in mixtures is whether the substances show additive, less than additive, or more than additive toxicity. The latter terms are roughly similar

to the terms “antagonism” and “synergism” that are commonly, but inconsistently used in the technical literature.

For both metals and organic contaminants that have similar mechanisms of toxicity (e.g., different metals, different chlorinated phenols), assuming chemical mixtures to have additive toxicity has been considered a reasonable and usually protective (Norwood *et al*. 2003; Meador 2006). This conclusion is in conflict with the way effluent limits are calculated for discharge of toxic chemicals into receiving water. Each projected effluent chemical concentration occurring during design flow is divided by its respective criterion, along with adjustments for variability and mixing zone allowances (EPA 1991). Thus, each substance would be allowed to reach one “concentration unit” and any given discharge or cleanup scenario would likely have several concentration units allowed, which is sometime referred to as cumulative criterion units.

Experimental approaches in the literature usually report “toxic units” (TUs) based on observed toxicity in single substance tests, rather than criterion units. In this “concentration addition” scheme, toxicity of different chemicals is additive if the concentrations and responses can be summed on the basis of “TUs.” For instance, assume for simplicity that cadmium is more toxic than copper to a species, with the an EC_{50} of 4 µg/L for cadmium, and an EC_{50} of 8 µg/L for copper. We will also call each single metal EC_{50} a TU. The toxicity of mixtures could be estimated as follows:

4 µg/L Cd + 0 µg/L Cu = $\frac{4\ \mu g/L}{4\mu g/L/TU} + \frac{0\ \mu g/L}{8\mu g/L/TU}$ =1 TU, (obviously, for a single substance), *or*

2 µg/L Cd + 4 µg/L Cu = $\frac{2\mu g/L}{4\mu g/L/TU} + \frac{4\mu g/L}{8\mu g/L/TU}$ =0.5+0.5=1 TU.

Using this approach, some studies have shown significant additive toxicity. For instance, Spehar and Fiandt (1986) exposed rainbow trout and *Ceriodaphnia dubia* simultaneously to a mixture of five metals and arsenic, each at their acute CMC, which by definition were intended to be protective. There were no survivors. In chronic tests, adverse effects were observed at mixture concentrations of one-half to one-third the approximate chronic toxicity threshold of fathead minnows and daphnids, respectively, suggesting that components of mixtures at or below no effect concentrations may contribute significantly to the toxicity of a mixture on a chronic basis (Spehar and Fiandt 1986).

A common outcome in metals mixture testing has been that metals combinations have been less toxic than the sum of their single-metal toxicities, i.e., show less than additive toxicity or are antagonistic (Finlayson and Verrue 1982; Hansen *et al*. 2002c; Norwood *et al*. 2003; Vijver *et al*. 2011; Mebane *et al*. 2012). The other possibility, more than additive toxicity (also called synergistic effects) are rare with metals although it has been shown with pesticides (Norwood *et al*. 2003; Laetz *et al*. 2009).

Summary: The water criteria evaluated in this opinion were all developed as if only one chemical was present at a time. However, in the real world chemicals always occur in mixtures. As result, criteria and discharge permits based upon them may afford less protection than intended. Measures to address this potential underprotection need to be included in discharge permits.

The efficacy of whole-effluent toxicity tests to evaluate mixture toxicity. The EPA's approach to the mixture toxicity problem in effluents, including effects of substances without numeric criteria or unmeasured substances, has been to recommend an integrated approach to toxics control (EPA 1991, 1994). The EPA has long recognized that numerical water quality criteria are an incomplete approach to protecting or restoring the integrity of water. A major part of EPA's strategy for measuring and controlling such potential issues has been through the concept of an integrated approach to toxics control, where meeting numerical criteria is but one of three elements. The other two elements are: (1) The concept of regulating whole effluents through whole- effluent toxicity (WET) testing; and (2) through biological monitoring of ambient waters that receive point or nonpoint discharges (EPA 1991, 1994). Because of assumptions that: (1) Chemicals will inevitably occur in ambient waters in mixtures rather than occurring chemical by chemical in the fashion that criteria are developed; and (2) it's not possible to know all the potential contaminants of concern in effluents and receiving waters, let alone measure them, it is not feasible to predict effects by chemical concentrations alone. Thus, the EPA developed procedures for testing the whole-toxicity of effluents and receiving waters, including procedures for identifying and reducing toxicity (e.g., Mount and Norberg-King 1983; Norberg-King 1989; Mount and Hockett 2000). In practice, some consideration of the potential for aggregate toxicity through WET testing is made by EPA for major permits that they administer in Idaho.

Test procedures for WET testing are intended to be practical for permitted dischargers or test laboratories to carry out as a routine monitoring tool. Thus, to simplify testing, improve test repeatability, and to facilitate interpretation of test results by dischargers and permit compliance staff, the EPA has limited WET testing requirements to select standard test species and test conditions (EPA 2002a, 2002c). Most commonly, EPA has required monitoring for chronic WET through testing of two species, fathead minnows and the cladoceran ("water flea") *Ceriodaphnia dubia*. Both tests are administered as 7-day tests. *Ceriodaphnia* have a short life-cycle, so even though the test is only 7 days, it spans three broods, and so can be considered a "true" chronic test that includes all or most of an organism's life cycle. In contrast, the 7-day fathead minnow "chronic" test only spans about 1% of the 2-year or so life span of a fathead minnow and is more properly called a short-term method for predicting chronic toxicity.

The rationale and performance of WET testing for predicting or protecting against impairment have been complicated and controversial and have been debated in conferences and articles, among them a special issue of the journal *Environmental Toxicology and Chemistry* (v19, 1, January 2000) and an entire book (Grothe *et al*. 1996). Issues with WET testing include whether the tests are sensitive, and whether any single species toxicity test can meaningfully predict in stream effects or lack thereof. For instance, Clements and Kiffney (1996) noted that *Ceriodaphnia* effluent tests were correlated with effects detected from stream microcosms or field surveys, but the latter two tended to be more sensitive than the Ceriodaphnia effluent tests. Conversely, Diamond and Daley (2000) and de Vlaming *et al*. (2000) found that the chronic WET methods were useful for predicting ambient impairment.

The best comparison of the sensitivity of WET tests in relation to listed salmon, steelhead and their prey is probably a series of tests conducted at the same laboratory with the same dilution water with copper and different species (Table 2.4.1.2). Neither the *Ceriodaphnia* or 7-day fathead minnow test were as sensitive as 30- or 6-day chronic tests with rainbow trout; the

Ceriodaphnia were about twice as resistant as the rainbow trout, and the 7-day fathead minnow test was almost five times as resistant as the longer rainbow trout test. Dwyer *et al*. (2005a) also found that the *Ceriodaphnia* test was considerably more sensitive than the 7-day fathead test to a complex "effluent" comprised of a mixture of pesticides, chlorinated organic compounds, ammonia, and metals. The low sensitivity of the 7-day fathead minnow test might be because the species is inherently less sensitive to some substances than salmonids or because a 7-day exposure is too short to be an accurate "short-term" chronic measurement (Suter 1990; Lazorchak and Smith 2007).

Comparisons with other metals were less reliable because they required comparing tests across studies and regression-based hardness normalizations (Table 2.4.1.3). Focusing on the more sensitive *Ceriodaphnia* test, sensitivity comparisons were made for four metals with rainbow trout (treating rainbow trout as a surrogate for listed salmon and steelhead). The comparisons used the most convenient, readily available statistics that were comparable across tests, even though those statistics do not reflect protective concentrations in of themselves (e.g. EC20, MATC, see "*Implications of the use of the "chronic value" statistic*"). A sensitivity ratio of 1.0 or less suggests that *Ceriodaphnia* are at least as sensitive as the salmonid surrogate and that the WET testing should be protective for aggregate, direct toxicity of waste mixtures in effluents (Table 2.4.1.2). The comparisons suggest that for cadmium and zinc the *Ceriodaphnia* test would be almost as sensitive or more sensitive as the average rainbow trout test; however, for copper and lead. Chinook salmon or rainbow trout could be much more sensitive than the *Ceriodaphnia*.

A further consideration beyond these simple comparisons of whether reduced survival or reproduction in Ceriodaphnia test results occurred at higher or lower concentrations than mortality to listed salmonids, is whether WET tests such as Ceriodaphnia can be used as a proxy indicator of sublethal effects of chemicals to salmonids, such as olfactory impairment. The limited information available suggests that they can be used in this way, at least for copper. Toxicity of copper to aquatic organisms can often be predicted using a "biotic ligand model" or BLM. The BLM uses geochemical speciation modeling to model bioaccumulation of copper on the organisms' gills or their other biological tissues in contact with water (i.e., their "biotic ligands"), and then uses an empirical species-specific toxicity adjustment to predict effects (Appendix C). This empirical species-specific toxicity adjustment was initially done to predict killing organisms with different sensitivities following short-term exposures (EPA 2007a). However, it has been successfully expanded to predict olfactory impairment (or lack thereof) in coho salmon or behavioral avoidance in rainbow trout or Chinook salmon (Appendix C; Meyer and Adams 2010). These analyses suggest that on the average, adverse effects predicted for Ceriodaphnia dubia would occur at lower copper concentrations than would olfactory impairment or avoidance behavior in rainbow trout, based upon lower modeled critical accumulation values for Ceriodaphnia dubia (0.06 vs. 0.19 nmol/g wet weight (Appendix C; Meyer and Adams 2010).

In contrast, the Ceriodaphnia WET test has been shown to be able to predict adverse effects in benthic macroinvertebrate communities in streams, but that the Ceriodaphnia WET test appeared less sensitive than the more complex stream communities (Clements and Kiffney 1996). This suggests that with a sensitivity adjustment, the Ceriodaphnia WET test could be used to predict

whether effluents were likely to adversely modify critical habitats by reducing the benthic macroinvertebrate forage base for rearing salmonids.

Table 2.4.1.2. Relative sensitivity of standard 7-day WET tests with *Ceriodaphnia* and fathead minnows to rainbow trout with copper under directly comparable test conditions (ASTM moderately-hard water, hardness 170 mg/L).

Organism	Test duration	EC25 for the most sensitive endpoints (µg/L)	Source
Rainbow trout	30-days (starting with fry)	21	(Besser *et al*. 2005b)
Rainbow trout	60-days (starting with eggs)	25	(Besser *et al*. 2005b)
Fathead minnow	30-days	12-24 (range of 3 replicate tests)	(Besser *et al*. 2005b)
Fathead minnow	7-days	103	(Dwyer *et al*. 2005a)
Ceriodaphnia dubia	7-days	51	(Dwyer *et al*. 2005a)

Table 2.4.1.3. Relative sensitivity of the standard WET *Ceriodaphnia dubia* 7-day test in relation to a surrogate salmonid for listed salmon and steelhead (rainbow trout except where noted), pooled from data compilations

Metal	*Ceriodaphnia dubia* SMCV (µg/L)	Surrogate salmonid SMCV (µg/L)	Sensitivity Ratio (*C.dubia* ÷ Salmonid)	Notes (source)
Cd	2.04	1.7	1.2	MATC, (Mebane 2006)
Cu	19	23.8	0.8	EC20s, (EPA 2007a);
Cu	19	5.9	3.2	Chinook salmon biomass EC20 (EPA 2007a); Rainbow trout, geometric mean of 5 tests, normalized to hardness 50; (Mebane *et al*. 2008); C. dubia is from a single test at hardness
Pb	46	28	1.6	52 mg/L, pH 7.56 (Mager *et al*. 2011a) (note)
Zn	33	113	0.3	NOECs; (Van Sprang *et al*. 2004)

Note: Much new data with C. dubia and chronic toxicity of Pb has been recently generated (Parametrix 2010; Mager *et al*. 2011a). While this was too much to synthesize and estimate whether C. dubia are usually more or less than salmonids, recent toxicity values with C. dubia indicate the sensitivities overlap those of rainbow trout and the species may be much more sensitive than previously indicated (Jop *et al*. 1995; Mebane *et al*. 2008)

Summary: Our review generally supports EPA's concept of assessing mixture toxicity of criteria substances under consultation through WET testing and instream bioassessment. However, the more sensitive of the two commonly used chronic WET tests, the three-brood *Ceriodaphnia dubia* test was sometimes less sensitive than chronic tests with salmonids. The 7-day fathead minnow test was consistently less sensitive than chronic salmonid tests in the data reviewed. This suggests that to be protective of listed salmonids, the assessment triggers for the

Ceriodaphnia test might have to be scaled to account for sensitivity and or differences in tolerable risk for a threatened species versus a zooplankton.

In much of EPA's (2000a) biological evaluation of the action, and elsewhere in the present opinion, the effects of criteria provisions or substances are evaluated linearly, one-by-one. Despite this simplification, in the environment chemicals in water never occur in isolation, but rather always occur as mixtures. The toxicity of mixtures is probably dependent upon many factors, such as which chemicals are most abundant, their concentration ratios, differing factors affecting bioavailability, and organism differences. Because of this complexity, accurate predictions of the combined effects of chemicals in mixtures appear to be beyond the present state of the ecotoxicology practice.

Here, despite the complexities and many exceptions, we make a general assumption that, at their criteria concentrations, the effects of chemicals in mixtures would likely be more severe than would be the same concentration of the mixture components singly.

Addressing mixture toxicity through the use of WET testing and instream bioassessment are practical and reasonable approaches for addressing the expected increased toxicity of a given concentration of a chemical in the presence of other chemicals. However, the assessment triggers on WET tests may not be sensitive enough to protect listed salmonids with reasonable certainty, and biomonitoring has not always been well defined. Measures for implementing biomonitoring are provided in Section 2.9 and Appendix E

2.4.1.11. Frequency, Duration and Magnitude of Allowable Criteria Concentration Exposure Exceedences.

For simplicity, much of the discussion of the water quality criteria that are the subject of this consultation treats the criteria as though they were defined solely as a concentration in water. However, the action actually defines aquatic life criteria in three parts: a concentration(s), a duration of exposure, and an allowable exceedence frequency. All of EPA's criteria recommendations define criteria using a statement similar to the following:

> "The procedures described in the 'Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and their uses' indicate that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected unacceptably if the 4-day average concentrations of [the chemical] do not exceed [the 'chronic' criterion continuous concentration] more than once every 3 years on the average and if the 1-hour average concentration does not exceed [the 'acute' criterion maximum concentration] more than once every 3 years on the average."

The 4-day and 1-hour duration 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. The EPA's (1985) Guidelines considered an averaging period of 1 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 1 to 3 hours. Also, even when organisms do not die within the first few hours, few toxicity tests attempt to monitor for latent mortality by transferring the test organism into clean water for observation after the

chemical exposure period is over. Thus, it was not considered appropriate to allow concentrations above the CMC for more than 1 hour (Stephan *et al*. 1985). Recent criteria documents (e.g., EPA 2007a) have used an averaging period of 24 hours for their CMC, although no explanation could be found for the deviation from the 1985 Guidelines and thus, the issue of latent toxicity might not have been considered.

A review of more recent information supported EPA's judgments from the 1980s that if an averaging period is used with acute criteria for metals, it should be short. 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 in the amount of metal bound to the gill occurs above background levels. This rapid increase occurs during exposures on the order of minutes to hours, and these brief exposures have been sufficient to predict toxicity at 96 to 120 hours. The half saturation times for cadmium and copper to bind to the gills of rainbow trout may be on the order of 150 to 200 seconds (Reid and McDonald 1991). Several other studies have shown that exposures well under 24 hours are sufficient for accumulation to develop that is sufficient to cause later toxicity (Playle *et al*. 1992; Playle *et al*. 1993; Zia and McDonald 1994; Playle 1998; MacRae *et al*. 1999; Di Toro *et al*. 2001). 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. The few known studies that tested for latent toxicity following short-term exposures have demonstrated delayed mortality following exposures on the order of 3 to 6 hours (Marr *et al*. 1995a; Zhao and Newman 2004, 2005; Diamond *et al*. 2006; Meyer *et al*. 2007a). Observations or predictions of appreciable mortality resulting from metals exposures on the order of only 3 to 6 hours supports the earlier recommendations by Stephan and others (1985) that the appropriate averaging periods for the CMC is on the order of 1 hour.

The 4-day averaging period for chronic criteria was selected for use by EPA 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 (e.g., Chapman 1978a; Barata and Baird 2000; 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. Whether fluctuating concentrations would result in increased or decreased adverse effects from those expected in constant exposures seems to defy generalization. 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). Similarly, *Daphnia magna* exposed to daily pulses of copper for 6 hours at close to their 48-hour LC_{50} concentrations had more severe effects after 70 days than did comparisons that were exposed to constant copper concentrations that were similar to the average of the daily fluctuations (Ingersoll and Winner 1982). In contrast, cutthroat trout exposed instream to naturally fluctuating zinc concentrations survived better than fish tested under the same average, but constant zinc concentrations (Nimick *et al*. 2007; Balistrieri *et al*. 2012). Thus, literature reviewed either supports or at least do not contradict EPA's position on averaging periods.

The third component of criteria, EPA's once-per-3-years allowable exceedence policy was based on a review of 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 3 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 3-year period seemed reasonably supported or at least was not contradicted by the information we reviewed.

A more difficult evaluation is the exceedence magnitude, which is undefined and thus not limited by the letter of the criteria. Thus, by the definition, a once-per-3-year exceedence that has no defined limits to its magnitude, could be very large, and have large adverse effects on listed species. However, within the 4-day and 1-hour duration constraints of the criteria definitions, some estimates of the potential magnitude of exceedences that could occur without "tripping" the duration constraints can be calculated. 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 can be made. Commonly with water chemical data and other environmental data, the statistical distributions do not follow the common bell-curve 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; Limpert *et al*. 2001; Helsel and Hirsch 2002; Delos 2008).

The following three hypothetical scenarios are intended to illustrate contaminant concentrations that could occur without violating the exceedence frequency and duration limitations of the proposed criteria (Figure 2.4.1.7). The scenarios use randomly generated values from a log-normal distribution with different variabilities and serial correlations. Serial correlation refers to the pattern in environmental data where values at time one are often highly correlated with values at time two and so on. For example, a hot day in summer is much more likely to be followed by another hot day than a bitterly cold day, a low chemical concentration during stable low flows on a day in September will most likely be followed by low chemical concentration the next day, a high chemical concentration in a stream during runoff on a day in April will more likely to be repeated by another high concentration, and so on (Helsel and Hirsch 2002; Delos 2008). Under Scenario 1, effects could be appreciable since the mean concentrations are close to the criteria, and organisms would have little relaxation of exposure for recovery. Under Scenario 2, effects to a population of sensitive organisms would presumably be slight, since the mean concentrations were well below the criterion, and the exceedence magnitude was slight followed by a recovery opportunity. Scenario 3 might be more likely in runoff of nonpoint pollutants from snowmelt or stormwater. In these scenarios, sensitive populations could experience effects ranging from appreciable reductions if the contaminant pulse hit during a sensitive part of their life history, to no effect if it hit during a resistant phase or if the listed species was less sensitive than the species that drove the criteria calculations.

An actual event that was very similar to Scenario 3 occurred when an upset at a large, industrial mining operation caused elevated cadmium concentrations in Thompson Creek, a tributary to the upper Salmon River in Idaho. In April 1999, a pulse of cadmium about 30X higher than background, 2.6 times higher the chronic criterion, and equal to the acute criterion was detected. The duration of exceedence was probably greater than a day and less than a week. By August 1999, when a biological survey was conducted, few if any adverse effects could be detected in the benthic community structure. Whether subtle differences between unaffected upstream survey sites were lingering effects of the disturbance or just differences in naturally patchy stream invertebrate communities was unclear. However, it does suggest that benthic communities in similar mountain streams would be either resilient to, or recover quickly from criteria exceedences of this magnitude (Mebane 2006, pp. 47,62).

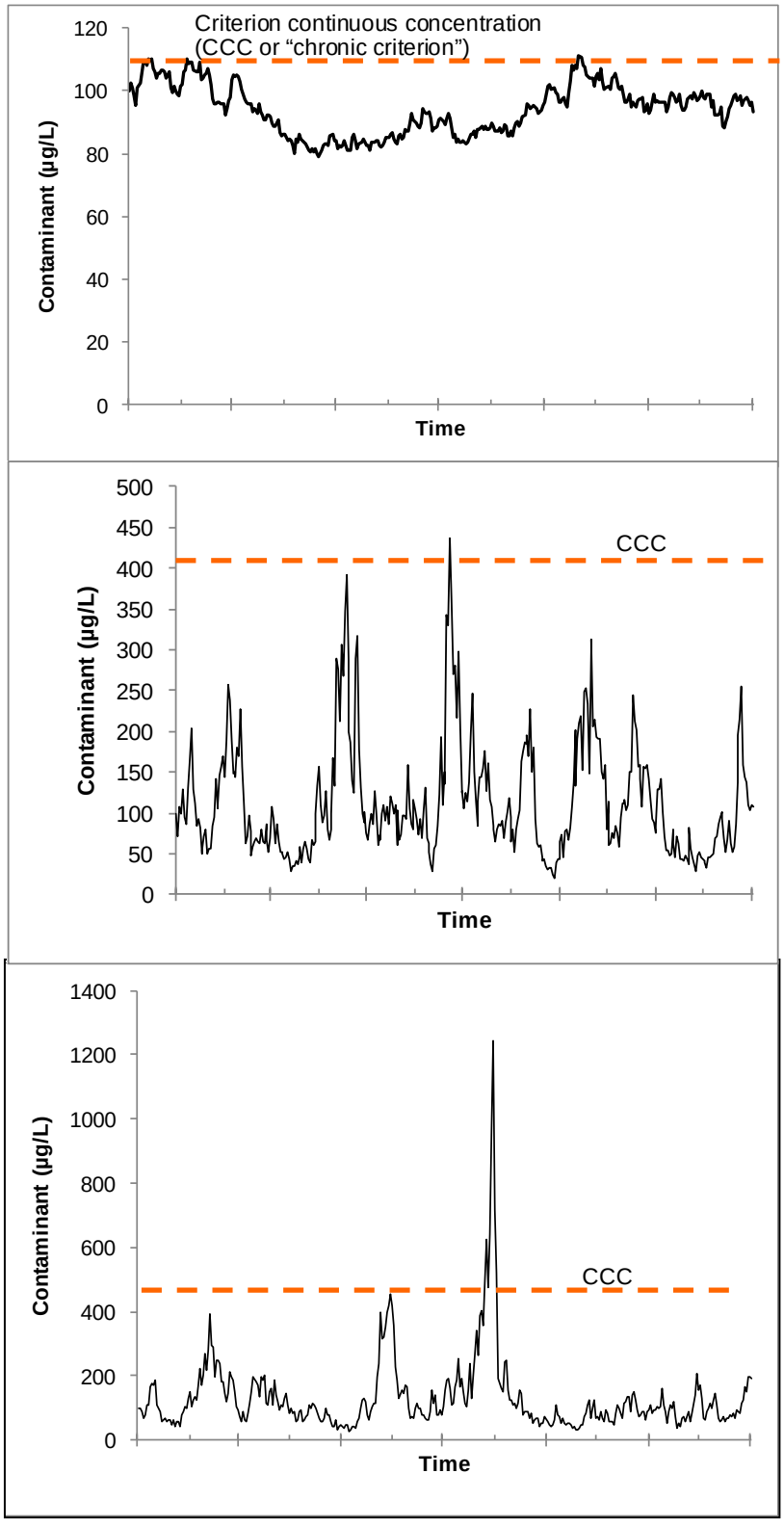
These hypothetical scenarios used a simplified, fixed criterion, whereas in actuality, some of EPA's criteria vary and may be positively correlated with the concentrations of metals in water. If the criteria accurately reflect risks from varying environmental conditions, and if ambient conditions co-vary with and are positively correlated with criteria, this will tend to lessen risks resulting from ambient increases in concentration. In cases where the criteria were positively correlated with the contaminants, such as in the following Section 2.4.4 example for Pine Creek with cadmium or the BLM-copper example for Panther Creek, the frequency and magnitude of exceedences is expected to be less than if the criteria and contaminant concentrations did not rise and fall together. This is because the contaminant and another water quality parameter that mitigates toxicity have common sources and rise and fall together, such as cadmium and calcium in Pine Creek where the source for both is probably weathering of gangue rock and spring snowmelt and runoff appears to dilute both.

In the Panther Creek example, copper and DOC tended to rise and fall together with snowmelt and runoff, similarly mitigating exceedence frequency and magnitude. This was the case in all examples examined. In the Panther Creek example, the hardness-based criterion is negatively correlated with copper concentrations, which gives the impression of risks of copper being exacerbated due to lower hardness corresponding with higher copper. However, this impression is probably misleading because copper risks indicated from the hardness-based criteria are often the opposite from risks indicated by BLM-based criteria, which is considered to more accurately represent the copper risks (Section 2.4.4; Appendix C).

While NMFS did not locate any plausible examples of negative correlations between contaminants and important factors modifying toxicity, it is likely that such scenarios do occur somewhere because if the event that releases the contaminant, such as a runoff pulse from a storm or snowmelt, caused a contaminant spike from washing accumulations into a stream and at the same time lowered the pH and hardness, then the magnitude of exceedences could be more severe. Such a circumstance could be plausible for metals such as cadmium, lead, or zinc in which hardness is a major modifier of toxicity.

Further, the actual possibility that an extreme exceedence would occur and be "allowed" under the exceedence policy seems unlikely. This is because in natural waters seasonal and hydrologic factors tend to cause concentrations to be serially correlated, that is low concentrations follow low concentrations and high concentrations follow high concentrations (Helsel and Hirsch 2002;

Delos 2008). Thus for an extreme exceedence to be allowable under the chronic criteria 4-day average concentration definition, it would also have to not exceed the 1-hour acute criteria definition. A very large exceedence of the sort illustrated in Figure 2.4.1.7, Scenario 3, would likely span across more than one, 1-hour averaging period for acute criteria and "violate" the one exceedence per 3-year recurrence interval term. While there are no regulatory limits on the upper concentration of an exceedence of the 1-hour acute criteria, the idea that a chemical concentration in a natural water could rapidly rise to acutely toxic concentrations and then drop back down to below criteria seems like a remote possibility. In urban watersheds with high proportions of impervious surface, runoff is flashier than in forested watersheds, and short-term pulse exposures could occur in those settings Booth *et al.* (2002). In the predominately forested areas of the action areas, such scenarios seem less likely.



Scenario 1: Contaminant concentrations have low variability, and while the CCC is only briefly exceeded, the average exposure concentration is only slightly lower than the criterion. Such a scenario might result from a stable effluent discharged into a flow regulated receiving water .

Scenario 2: Contaminant concentrations are more variable, and while the frequency and magnitude of criterion exceedences are similar to scenario 1, average concentrations are well below the CCC in this scenario. Such a scenario might result from nonpoint pollutants resulting from snowmelt or precipitation into an unregulated stream, such as stormwater from a mining operation.

Scenario 3: Contaminant concentrations have the same variability as scenario 2, but by chance a high magnitude criterion exceedence of 12X above the average concentrations occurred. Unless the acute criterion for this substance was at least 12X higher than the CCC, such an exceedence would not be allowable because the 1-hour acute criterion averaging period would also be exceeded.

Figure 2.4.1.7. Three example allowable scenarios for criteria exceedence magnitudes

Summary: The 1-hour and 4-day exceedence durations for acute and chronic criteria respectively are supported by the science as reasonable and adequately protective. Whether the allowable 1 in 3 years exceedence frequency is sufficiently protective was difficult to evaluate, in part because the magnitude of allowable exceedences is undefined. However, the likelihood that a runoff pulse could both rise and fall so high within an hour that it could cause acute effects without exceeding the acute criteria seems unlikely. This does remain an aspect of uncertainty regarding the protectiveness of criteria.

2.4.1.12. Special Consideration for Evaluating the Effects of the Action on Critical Habitat

Fundamentally, the analyses of water quality criteria for toxic substances included in this Opinion are most directly analyses of the "water quality" features of the PCE's of critical habitat. The WQS directly characterize and define the conditions and quality of surface waters that listed salmon and steelhead experience, either as incubating embryos in the interstices of spawning gravels, or as juveniles and adults in the water column. Analyzing whether the action would represent an "adverse modification" of water quality is at least conceptually more straightforward than whether these modifications would jeopardize the continued existence of listed species. This is because quantitative causal predictions relating habitat change to species population changes and long-term viability are uncertain. Many simplifying assumptions are required, including things like specifics of species life histories, other interacting physical and biological factors, the nature and magnitude of assumed exposures such as whether the exposures are joint or separate, continuous or intermittent, magnitude of exceedences, and so on. Quantitative models relating water quality changes to extinction risks may provide value in a relative sense for evaluating relative risks of different "what if" scenarios (e.g., McCarthy *et al*. 2004; Baldwin *et al*. 2009; Mebane and Arthaud 2010). However, except for cases of extreme-risk with very high extinction probabilities (perhaps for example, Spromberg and Scholz 2011), the absolute projections from quantitative models of habitat and population changes may be thought of as mathematical speculation. Further, all mathematical population models will project some extinction risk, and policy definitions or scientific consensus are elusive on how much habitat modification or extinction risk is too much under narrative Endangered Species Act definitions (DeMaster *et al*. 2004; McGowan and Ryan 2009; McGowan and Ryan 2010; Owen 2012).

The types of adverse effects reported in the scientific literature that we consider to directly or indirectly reduce survival or reproduction included such things as reductions in survival, growth, swimming performance, ability to detect or evade predators (e.g., chemoreception), ability to detect or capture prey, ability to detect and avoid harmful concentrations of chemicals, homing ability, disease resistance, certain fish health indicators that have been related to survival or growth such as gill or liver tissue damage, spawning success, or fecundity. For evaluating what severity of effects to invertebrates would be considered an appreciable enough reduction in forage to reduce the conservation value of habitats for freshwater rearing, if a general reduction in diversity or abundance of invertebrates was expected at criteria conditions, we would consider that to be "appreciable." Because salmonids are opportunistic feeders, effects to a single invertebrate species for example, might not be important. This assumption must be tempered by the availability of data. Often data were available for very few invertebrate species, so if few

data were available, but they indicated adverse effects, that could be considered a diminishment in water quality and habitat value.

Examples of types of effects that we do not consider to be sufficiently severe to represent an "appreciable diminishment" of water quality and thus the value of critical habitat include simple bioaccumulation of chemical in tissues, enzyme changes, gene expression or transcription, molecular changes, or other markers of exposure that may be considered sub-organismal, without known correlation to other changes such as reduced growth or survival. A human-health analogy of the latter types of effects would be those considered asymptomatic or sub-clinical, that is, not rising to the level that caused negative symptoms.

Because multiple criteria (acute and chronic aquatic life criteria, human health based water quality criteria) for the same substances would apply to any given area of critical habitat, we compared adverse effects indicated from short-term experiments of 4 days or less duration to the acute criteria that are intended to protect against short-term effects, and compared adverse effects shown in longer-term studies to the proposed chronic criteria. Human health-based criteria were only evaluated if they were both more stringent than chronic criteria and if the chronic criteria failed to be fully protective. In Idaho, water quality criteria for the protection of "fishable" beneficial uses based on avoiding health risks from consuming tainted fish, were clearly intended to be some sort of backstop to the aquatic life criteria because the human-health based criteria explicitly apply to waters designated for "cold water biota" and "salmonid spawning" aquatic life uses (Table 1.3.1).

For most of the substances, there were at least some conflicts in the scientific literature where for the same species and similar types of experiments, one study might find no ill effects from a given concentration and another might find severe effects. Thus, we considered the overall strength of the evidence for or against the protectiveness of criteria.

Sediments. If sufficiently elevated, toxic pollutants in ambient water may adversely modify critical habitat through contamination of stream and lake bed sediments. In general, sediment contamination by toxic pollutants adversely modifies critical habitat because the particulate forms of toxicants are either immediately bioavailable through re-suspension, or are a delayed source of toxicity through bioaccumulation or when water quality conditions favor dissolution at a later date. Specifically, contaminated sediments are expected to influence: (1) The intra-gravel life stages of listed salmon and steelhead; (2) the food source of listed salmonids; and (3) the fish through direct ingestion or deposition on the gill surfaces of particulate forms of toxicants. However, other than for mercury, it is not clear whether moderately-elevated concentrations in water (i.e, up to criteria concentrations), would be likely to result in concentrations in bed sediments that are elevated to a degree that would pose appreciable risks to listed salmonids or their prey.

The proposed criteria do not explicitly account for exposure to contaminants via sediments. 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. Thus, at sites where there have been past discharges, or where there

are continuing discharges of contaminants into the water column, sediments 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 salmonids through food chain transfer from their benthic prey, or through ingestion of sediment while feeding, as has been described in preceding sections. Not having water quality criteria that consider uptake through direct ingestion or food chain transfer leaves potential routes for harm to listed species that the proposed criteria do not directly address.

Salmonid Prey Items. An important type of indirect adverse effect of toxic substances to listed salmon and steelhead is the potential reduction of their invertebrate prey base. This is because for many substances, invertebrates tend to be among the most sensitive taxonomic groups and because juvenile salmonids depend on aquatic invertebrates during freshwater rearing. Known effects of specific substances to invertebrates are discussed specifically in those sections; however, some general considerations and assumptions applicable to all substances follow.

First, in instances of a pulse of chemical disturbance such as insecticide spraying of forests or crops, effects to aquatic invertebrate communities ranging from increased drift to catastrophic reductions can result (Ide 1957; Gibson and Chapman 1972; Wallace and Hynes 1975; Wallace *et al*. 1986). In such cases, even if the fish are not directly harmed by the chemical, the temporary reduction in food from the reduction in invertebrate prey can lead to reduced growth, and reduced growth in juvenile salmonids can in turn be extrapolated to reduced survival and increased risk of population extinction (Kingsbury and Kreutzweiser 1987; Davies and Cooke 1993; Baldwin *et al*. 2009; Mebane and Arthaud 2010). However, such severe effects would not be expected in waters with chemical concentrations similar to the maximum allowed by aquatic life criteria. The criteria are intended to only allow adverse effects to a small minority of the species in aquatic communities, and for most substances, the analyses of individual criteria that follow in Sections 2.4 are consistent with this expectation (*although copper has exceptions*).

This begs the question, whether the loss of a minority of invertebrate prey species could lead to a reduction in forage for juvenile salmonids that in turn could affect growth and survival? To address that question, NMFS reviewed a large number of studies on food habits of salmonids in streams, lakes, and reservoirs. [5] The body of evidence indicates that juvenile salmonids are opportunistic predators on invertebrates, and so long as suitable, invertebrate prey items are abundant and diverse, the loss of a few "menu items" probably would not result in obvious, adverse effects. Suitable invertebrate prey items for juvenile salmonids are those that are small enough to be readily captured and swallowed, and vulnerable to capture (i.e., not taxa that are burrowers or are armored (Keeley and Grant 2001; Suttle *et al*. 2004; Quinn 2005)). Some otherwise apparently suitable taxa such as water mites (Hydracarina) appear to taste bad to salmonids and others, like copepods, are too small to provide much energy for the effort it takes to eat them (Keeley and Grant 1997). Freshwater aquatic invertebrates have such great diversity (over 1200 species in Idaho alone, Mebane 2006), that they have some ecological overlap and redundancy, so that the loss of a few species would be unlikely to disrupt the stream or lake ecology greatly (Covich *et al*. 1999). However, this apparent ecological redundancy is compromised in streams that have already lost substantial diversity to pollution. For instance, in

[5] Over 90 were reviewed, although only a handful are listed here.

copper-polluted Panther Creek, Idaho, during springtime in the early 1990s, the total count of invertebrates was just as abundant as in reference sites, although the abundance was composed of fewer species. Yet in October, the abundance in the polluted reaches was less than 10% of reference (Mebane 1994). With reduced diversity, after a single species hatches and leaves the streams, a large drop in remaining abundance can occur. Because all species don't hatch at the same time, with greater diversity, the swings in abundance would be less severe. Further, in copper-polluted tributaries to Panther Creek, the usually abundant mayflies were scarce and had been replaced by unpalatable mites and low-calorie copepods (Todd 2008).

One consistent theme in the literature on the feeding of salmonids in streams is the persistent importance of mayflies and chironomid midges (Chapman and Quistorff 1938; Chapman and Bjornn 1969; Sagar and Glova 1987, 1988; Mullan *et al*. 1992; Clements and Rees 1997; Rader 1997; White and Harvey 2007; Iwasaki *et al*. 2009; Syrjänen *et al*. 2011). In lakes zooplankton are disproportionally important, and as stream size increases and gradients drop, amphipods become popular food items with migrating and rearing juvenile salmon and steelhead (Tippets and Moyle 1978; Rondorf *et al*. 1990; Muir and Coley 1996; Budy *et al*. 1998; Karchesky and Bennett 1999; Steinhart and Wurtsbaugh 2003; Teuscher 2004). However, salmonids are opportunistic and will shift their feeding to whatever is abundant, accessible, and palatable, and have sometimes have been reported with their stomachs full of unexpected prey such as snails or hornets (Jenkins *et al*. 1970; NCASI 1989; Mullan *et al*. 1992).

In general, the body of the evidence suggests that there is some ecological redundancy among aquatic stream and lake invertebrates, and if a small minority of invertebrate taxa were eliminated by chemicals at criteria concentrations, but overall remain diverse and abundant, then aquatic invertebrate overall community structure and functions, and forage value of critical habitats would likely persist. However, case-by-case consideration of the data is required because the previous assumption is tempered by the fact that aquatic insects are typically underrepresented in criteria datasets and toxicity testing in general (Mebane 2010; Brix *et al*. 2011).

Some of the anticipated effects will be to food items for juvenile salmonids, a vital component of juvenile rearing and migration habitat. Reductions in food quantity would result in limited resources to rearing and migrating fish, which can be expected to reduce population viability through increased mortality. Under-nourishment can alter juvenile salmon ability to avoid predators and select habitat within rearing drainages. Mortality can also be expected during migration, as under-nourished juveniles will not be able to withstand the rigors of migration.

Changes in species composition could have the same results. Biomass quantity is not necessarily a substitute for prey suitability, as differing prey behavior patterns and micro-habitat needs can reduce the foraging efficiency of juvenile salmonids. However, juvenile salmonids are opportunistic predators, and the loss of a minority of taxa might not be a severe indirect effect if other prey were still diverse and abundant as described above.

Effects to Other Elements of Critical Habitat. Approval of the proposed criteria may also indirectly affect safe passage conditions and access. Safe passage conditions and access to other habitats may be prevented or modified if a passage barrier exists in a section of stream because

of insufficient mixing at an effluent outfall, or dilution capacity is insufficient to provide a passage corridor. To avoid these forms of adverse modification of critical habitat, the application of criteria must be protective of listed species. To determine this we evaluated if the action as proposed would provide safe passage in the manners described in Appendix F Salmonid Zone of Passage Considerations.

There appears to be little to no relation between adverse changes in water quality caused by adoption of the proposed criteria and effects to the remaining essential features of critical habitat, including: (1) Water quantity; (2) riparian vegetation; (3) instream cover/shelter; (4) water velocity; (5) floodplain connectivity; (6) water temperature; and (7) space.

2.4.2. The Effects of Expressing Metals Criteria as a function of Water Hardness

Some of the metals criteria under review in this consultation are hardness-dependent, meaning that rather than establishing a criterion as a concentration value, the criteria are defined as a mathematical equation using the hardness of the water as the independent variable. Thus, in order to evaluate the protectiveness of the hardness-dependent criteria, it was first necessary to evaluate the hardness-toxicity relations. The criteria that vary based on site-specific hardness are Cd, Cu, Cr (III), Pb, Ni, Ag, and Zn. Hardness measurements for calculating these criteria are expressed in terms of the concentration of $CaCO_3$, expressed in mg/L, required to contribute that amount of calcium plus magnesium. In the criteria equations, hardness and toxicity values and expressed as natural logarithms to simplify the math. In a general sense, these are referred to by the shorthand "ln(hardness) vs. ln(toxicity)" relations.

In the 1980s, hardness was considered a reasonable surrogate for the factors that affected toxicities of several metals. It was generally recognized that pH, alkalinity and hardness were involved in moderating the acute toxicity of metals. While it wasn't clear which of these factors was more important, because pH, alkalinity, and hardness were usually correlated in ambient waters, it seemed reasonable to use hardness as a surrogate for other factors that might influence toxicity (Stephan *et al*. 1985). In the case of copper, dissolved organic matter or carbon (DOM or DOC) was also recognized as being important. It was assumed that DOC would be low in laboratory waters and might be high or low in ambient waters, and that hardness-based copper criteria would be sufficiently protective in waters with low DOC and conservative in waters with high DOC (EPA 1985). Most of these relations were established in acute testing, and they were assumed to hold for long-term exposures (chronic criteria). Whether that assumption is reliable was and continues to be unclear. For instance, in at least two major sets of chronic studies with metals conducted in waters with low and uniform DOC concentrations, water hardness did not appear to have a significant effect on the observed toxicity in most cases (Sauter *et al*. 1976; Chapman *et al*. 1980).

In the two decades since the NTR metals criteria were developed, a much better understanding has been developed of the mechanisms of acute toxicity in fish and factors affecting bioavailability and toxicity of metals in water. Generally, acute toxicity of metals is thought to be moderated by complexation of metals, competition for binding sites on the surface of the fish's gill, and binding capacity of the gill before a lethal accumulation (LA_{50}) results (Wood *et al*. 1997; Playle 1998). The interplay of these factors has been modeled through biogeochemical "gill surface models" or "biotic ligand models" (BLMs) (Di Toro *et al*. 2001; Niyogi and Wood 2004). For brevity, "BLM" as used here refers to both.

While BLMs are conceptually applicable for developing water quality guidelines for many metals, the BLM approach is most advanced for copper. The EPA's (2007a) recommended national criteria for copper are based on the BLM. Santore *et al*. (2001) validated acute toxicity predictions of the copper BLM by demonstrating that it could predict the acute toxicity of copper to fathead minnows and *Daphnia* within a factor of 2 under a wide variety of water quality conditions. The predictive capability of the BLM with taxonomically distinct organisms is evaluated in detail in Appendix C. With fathead minnows, rainbow trout, Chinook salmon, planktonic invertebrates (various daphnids), benthic invertebrates (freshwater mussels and the amphipod *Hyalella* sp.) tested in a variety of natural and synthetic waters, predictions were always strongly correlated with measured acute toxicity. In several field studies, adverse effects to macroinvertebrate communities appear likely to have occurred at concentrations lower those allowed by EPA's (2007) chronic copper criterion. Still, the 2007 BLM-based copper criterion was a least as or more protective for macroinvertebrate communities than were EPA's 1985 and 1995 hardness-based criteria for copper.

For copper, the research leading to development of the BLM generally refutes the general relevance of the hardness-toxicity relation in ambient waters (e.g., Meador 1991; Welsh *et al*. 1993; Erickson *et al*. 1996; Markich *et al*. 2005). This is because the important factors that influence copper bioavailability are, in rough order of importance, DOC $\geq\approx$ pH >> >Ca > Na $\approx$ alkalinity $\approx$ Mg. Hardness is likely correlated with pH, calcium, Na, and alkalinity in natural waters, but DOC and hardness are not expected to rise and fall together.

For lead, the situation is probably similar with hardness being less important than DOC in many waters where DOC is abundant, although the BLM for lead is less advanced. With lead, calcium hardness was an important modifier of toxicity in laboratory waters with low DOC concentrations. However, at DOC concentrations reflective of many ambient waters ($\geq\approx$ 2.5 mg/L DOC), DOC was more important (Grosell *et al*. 2006b; Meyer *et al*. 2007b; Mager *et al*. 2011b).

In contrast, for cadmium, nickel, and zinc, the BLM and experimental data generally support the hardness-toxicity assumption in that acute toxicity to fish is influenced by water chemistry variables that are usually correlated with hardness (e.g., calcium, pH, Na, alkalinity, magnesium, in rough order of importance). The DOC is less important (Niyogi and Wood 2004). For silver, the protective effects of hardness are modest for acute or chronic silver toxicity in early life stages, juvenile, and adult rainbow trout and similar to the protection afforded to acute silver toxicity in juvenile and adult rainbow trout (Morgan *et al*. 2005).

For cadmium and zinc, or copper under conditions of low organic carbon, the ratios of calcium to magnesium influences the protective influence of hardness. Under the NTR and Idaho criteria, hardness is determined for a site, expressed as mg/L of $CaCO_3$, and input to the criteria equations 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 than magnesuim. For example, in the case of cadmium and zinc, 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; Davies *et al*. 1993; Alsop *et al*. 1999). Welsh *et al*. (2000b) and Naddy *et al*. (2002) determined that calcium also afforded significantly greater protection to fish against copper toxicity than magnesium.

The calcium:magnesium ratio in natural waters of Idaho vary by about two orders of magnitude (Appendix A). Median molar ratios of calcium:magnesium across a USGS/IDEQ network of 56 sites across Idaho monitored from 1989 to 2002 range from 0.56 to 9.73, and median ratios at all sites except one exceeded 1.3 (Hardy *et al*. 2005). In several important salmon and steelhead streams, calcium to magnesium ratio ranges are on the order of 8:1 in Valley Creek, between 4:1 and 7:1 in the upper Salmon River basin above the Pahsimeroi River, between 0.8:1 and 4:1 in Pahsimeroi River tributaries, 2:1 in the Pahsimeroi River, 1.5:1 in the Lemhi River, and 3:1 in the Salmon River at Salmon (Clark and Dutton 1996). In the review included as Appendix A, some of the lowest ratios were found outside the action area in the Coeur d'Alene region and in south-central and southeastern Idaho. Generally, these analyses indicate that the issue of hardness-toxicity relations failing and not being protective because of low calcium:magnesium ratios is not a big concern within the range of anadromous fish in Idaho.

2.4.2.1. The Use of a "Hardness Floor" in Calculating Metals Limits.

The Idaho hardness-dependent criteria, like the NTR criteria restrict the hardness values used in calculating the criteria to the range of 25 mg/L to 400 mg/L (EPA 1992). For high hardness values this is probably generally protective because the usual pattern of decreasing toxicity with increasing hardness breaks down at high hardness values. Heijerick and others (2002) found that at hardness values greater than 325 mg/L as calcium carbonate, no linearity, and even a decrease in 48-hour EC_{50}s, was observed with *Daphnia magna* and zinc. With copper and fathead minnows, above hardnesses of 150 mg/L, LC_{50}s apparently approached an asymptote (Erickson *et al*. 1996), and with copper and *Daphnia* at hardness of 400 mg/L and above, no relation was observed between hardness and toxicity (Gensemer *et al*. 2002). Thus, while an upper hardness ceiling of 400 mg/L might be too high, the concept of an upper ceiling is logical.

In contrast, at low hardness values this hardness floor is logically underprotective. What follows is a review of the history relating to the hardness floor issue, scientific investigations relevant to the hardness-toxicity relationship at low-hardnesses, and ambient hardness in Idaho.

History of the Hardness Floor. The EPA's 1992 NTR low-end hardness floor appears to have been an administrative invention associated with the promulgation of the NTR (EPA 1992); we found no support for it in any of EPA's scientific literature policy analyses that was available to

date. The EPA's Guidelines (Stephan *et al*. 1985) defines a general scheme for developing criteria with increased conservatism (more protective) when data are sparse and uncertainties high. Their Guidelines specifically describes adjusting criteria based on factors that affecttoxicity, including the general ln(hardness) vs. ln(toxicity) relationship. NMFS did not find the suggestion of imposing a low-end floor on hardness-toxicity relations in the Guidelines or any of the individual criteria documents from the 1980's was any suggestion of imposing a low end floor on hardness-toxicity relations found (e.g., EPA 1984b; 1985c; 1987b). Thus the notion of making unprotective assumptions about water quality criteria in the absence of supporting data or theory is generally counter to the EPA's science approach in the criteria process. Further, the low-end hardness floor notion is contrary to results of EPA research that specifically investigated metals toxicity at very low hardness. For example, Cusimano *et al*. (1986) tested the toxicities of cadmium, copper, and zinc to rainbow trout at low hardness (9 mg/L).

It appears that EPA tacitly recognized the error of the 1992 low-end hardness floor shortly thereafter. No hardness floor appeared with the metals criteria contained in the 1995 Great Lakes Initiative (40 CFR 132.6) nor in EPA's 1997 California Toxic Rule (40 CFR 131.37), and EPA's 1999 national recommended water quality compilation was silent on hardness floor (EPA 1999b). In 2002, EPA directly repudiated the 1992 hardness floor policy, asserting that while data below hardness of about 20 mg/L are limited, "*capping hardness at 25 mg/L without additional data or justification may result in criteria that provide less protection than that intended by EPA's Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses (EPA 822/R-85-100) or 'the Guidelines.' Therefore, EPA now recommends that hardness not be capped at 25 mg/L, or any other hardness on the low end*" (EPA 2002b). The EPA further recommended that "*if there is a state or tribal regulatory requirement that hardness be capped at 25 mg/L, or if there are any situation-specific questions about the applicability of the hardness-toxicity relationship, a Water Effect Ratio (WER) procedure should be used to provide the level of protection intended by the Guidelines*" (2002b).

Beyond the preceding quoted sentence, NMFS located no further details on how to use the WER procedure to remedy the hardness floor issue.

Hardness-toxicity patterns in soft water. Fish maintain their internal mineral balance through osmoregulation, and the greater the difference between their internal plasma mineral balance, and the mineral content of the water they live in, the greater the energy required to maintain homeostasis. In waters with very dilute mineral content (soft water), the energy requirements to maintain their mineral balance, or ionic balance, can be high. Compared to hard water, costs of these energy requirements to maintain ionic balance in soft water include reduced growth, reduced swimming ability, and reduced ability to recover from severe exercise (McFadden and Cooper 1962; Wood *et al*. 1983; Wood 1991; Kieffer *et al*. 2002; Dussault *et al*. 2008; Wendelaar Bonga and Lock 2008). In very soft water, fish may be on the verge of ionoregulatory problems, and because metals also disrupt ionic balance, any increase in metals may result in plasma ion loss (Playle *et al*. 1992; Van Genderen *et al*. 2008). The similarity in responses of fish to soft water acclimation and metals exposure suggest that simple extrapolation of hardness-toxicity relations that were developed at high hardnesses to soft waters may underestimate the additive responses and thus underestimate metals toxicity in very soft waters.

Empirical evidence and theoretical considerations both argue against the assumption that the general pattern of increasing toxicity of metals with decreasing hardness stops at 25 mg/L. However, the slope might be expected to be different than that at higher hardnesses and there are both rationales and data suggesting that the slope would be shallower or steeper at low hardnesses. Based on calculations of cation competition and aqueous complexation, Meyer (1999) predicted that for divalent transition metals such as cadmium, copper, and zinc, the slope of hardness-toxicity relations, as ln(hardness) vs. ln(LC_{50}) was likely to start shallowing below a hardness of about 20 mg/L and would reach a slope of zero at a hardness of about 3 mg/L. The 3 mg/L hardness floor theorized by Meyer (1999) was a data-free prediction because tests of hardness-toxicity relations at low hardnesses seem limited to minimum hardnesses of about 5 to 10 mg/L for cadmium, copper, lead, nickel, and zinc (Miller and Mackay 1980; Cusimano *et al*. 1986; Long *et al*. 2004; Sciera *et al*. 2004; Mebane 2006; Deleebeeck *et al*. 2007; Mebane *et al*. 2012). Morgan *et al*. (2005) did test the comparative effects of silver to rainbow trout at hardnesses of 2, 150, and 400 mg/L, but because the soft water exposure caused adverse effects without any metals addition, and the wide range of hardnesses tested, the data were insufficient to directly evaluate Meyer's theoretical 3 mg/L hardness-toxicity floor. However, because exposure to 2 mg/L hardness water by itself caused a doubling in mortality rates and increased time to hatch for rainbow trout embryos, the notion of 3 mg/L hardness-toxicity floor may be moot. One of the more comprehensive studies of metals toxicity was by Van Genderen *et al*. (2005). They found that over a hardness range of 6 to 40 mg/L in laboratory waters with low organic matter, there was a linear trend between copper toxicity to fathead minnows and hardness. They observed a species-specific slope between ln (hardness) and ln (LC_{50}) of (0.795 for hardness ranging from 6 to 40 mg/L as $CaCO_3$) was less than the pooled value for all species developed for EPA's (1985) copper dataset (0.9422 for hardness ranging from 13 to 400 mg/L as CaCO3). Van Genderen *et al*. (2005) suggested that the lower slope indicated that the influence of cation competition changes in low-hardness waters. The implications of these differing slopes are that Van Genderen *et al*.'s (2005) results showed that as hardness declined, copper becomes more toxic but because of the shallower slope, the increases in toxicity were not as great as predicted by EPA's (1985) steeper slope.

However, a safer interpretation of the general relationship between water hardness and metals toxicity is that aquatic organisms are likely more sensitive to metal exposure than would be expected by hardness-toxicity relations determined at higher ambient hardnesses. This is because fish have higher energy requirements to maintain homeostasis in soft water, and may be more sensitive to metals that inhibit ionoregulation (Greco *et al*. 1995; Taylor *et al*. 2000; Taylor *et al*. 2003; Van Genderen *et al*. 2005; Van Genderen *et al*. 2008; Wendelaar Bonga and Lock 2008). The increased sensitivity of fish to metals in very soft water may persist after fish that were acclimated or incubated in very soft water move into higher hardness water. Mebane *et al*. (2010) incubated rainbow trout in waters above the confluences of two streams, one with very-soft water (average hardness around 11 mg/L) and one with harder water with an average hardness of about 21 mg/L. Then the fish were exposed to cadmium and zinc in the harder of the two waters. The fish that had reared in the stream with softer water were about twice as sensitive as were trout that had been incubated in the higher hardness water (Mebane *et al*. 2010). This has implications for salmonid life histories and habitats. Water hardness tends to be lowest near the headwaters of streams and increase downstream, and some salmonids tend to ascend streams

to spawn in the upper reaches of watersheds and after emerging, their fry move downstream into higher hardness waters.

In Section 2.4.2 of this analysis, NMFS show plots of metals toxicity vs. hardness for various salmonid species at various life stages for cadmium, copper, lead, nickel, and zinc. For at least cadmium, copper, and zinc, those plots show a general relationship of decreasing resistance by the fish with decreasing hardness, a pattern that did not stop at a hardness limit of 25 mg/L $CaCO_3$. However, meta-analyses in this manner have limitations for analyzing specific relations between variables such as hardness-toxicity relations. This is because toxicity to salmonids and other fishes can vary by other factors which can obscure the patterns of interest. The influence of different sizes or developmental state is well known to be important, but other factors could influence the results. These include the strain or stock of fish; incubation or acclimation history conditions; water characteristics other than hardness such as pH, ionic composition, organic matter or particulates; and water renewal rates and frequencies. Data pooling such as was done for the summaries of effects for individual metals later in Section 2.4 is sometimes a beneficial and necessary means of generalizing study findings because this broader view may sometimes reveal patterns that may not be apparent in smaller, individual studies. However, important patterns can be lost.

The following data sets illustrate how pooling data that are only influenced by a few such factors can greatly confound hardness-toxicity relations. In an effort to develop site-specific water quality criteria for a soft-water river, the South Fork Coeur d'Alene River, Idaho, toxicity tests were conducted with cutthroat trout and rainbow across a range of water hardnesses (Mebane *et al*. 2012). Rainbow trout were used to develop hardness-toxicity relations. All the rainbow trout were obtained as eggs from a single supplier (Mt Lassen Trout Farms, Red Bluff, California) and incubated on site; all tests were done in the same test facility, and were directed by the same people. However, because it is seldom feasible to always test fish, at say, 30-days post hatch, some tests were run with fish of slightly different ages. In contrast, some tests were run side-by-side to specifically examine hardness variability using the same batch of fish at the same time, using waters collected from different waters with different hardnesses (Mebane *et al*. 2012).

With zinc, Figures 2.4.2.1 and 2.4.2.2 illustrate how hardness-toxicity patterns were always stronger when hardness was varied within a test series using the same batch of fish at the same time, than were patterns from meta-analyses that pooled data from across tests. The most complete data are with zinc. A simple comparison of hardness-toxicity relations with zinc from cutthroat trout fry over a hardness range of 11 to 63 mg/L shows that hardness can explain nearly 100% of the variability in toxicity. In contrast, when Mount Lassen rainbow trout are pooled across different years and batches, hardness explains less than half of the variability. Yet when the Mount Lassen rainbow trout results are grouped by concurrent test groups, the subgroup hardness toxicity relations explain from around 85% to 98% of the variation in toxicity compared to about 38% when pooled across groups. The reasons for the differences between groups are unclear, although differences in the sizes of fish might be a factor since the largest fry (average 0.46g wet weight) were most sensitive. Other testing has found that in the range of 0.2 to 1.0g, smaller fry tended to be more resistant to zinc toxicity (Hansen *et al*. 2002c).

With zinc, at a hardness of 10 mg/L, the Idaho acute and chronic criteria would both be about 17 µg/L (Table 2.4.2.1), which is similar to an estimated EC_{50} of about 21 µg/L for rainbow trout in waters with hardness of about 7 mg/L (Mebane *et al*. 2012), which was the lowest hardness test found. A concentration killing 50% of the test organisms can hardly be considered protective. If instead, the criteria were calculated with the ambient hardness of 7 mg/L, the criteria would be 12 µg/L, and if calculated with the proposed hardness floor of 25 mg/L the criteria would be 36 µg/L. At 36 µg/L, the lowest concentration actually tested, 80% of the rainbow trout were killed in this test.

With nickel, the most sensitive organisms appear to be zooplankton with approximate thresholds of adverse effects (EC_{10}s) of about 3 to 7 µg/L in very-soft water with hardness of 6 mg/L (Deleebeeck *et al*. 2007) compared to threshold of adverse effects for rainbow trout of <35 µg/L at hardness 27 to 39 mg/L (Nebeker *et al*. 1985). The NTR chronic nickel criterion is well above these values at 49 µg/L. However, Idaho's revised criteria, proposed for approval by EPA (Table 2.4.2.1) are 16, 7, and 5 µg/L at hardnesses of 25, 10, and 6 mg/L.

For lead, a different shortcoming of these types of hardness-toxicity comparisons becomes apparent in Figure 2.4.2.3. As with zinc, cutthroat trout sensitivity to lead is strongly influenced by hardness, with a reasonable spread of hardnesses of a range of 11 to 56 mg/L explaining about 80% of the variability in cutthroat EC_{50}s for lead. For rainbow trout, the range of hardnesses for six tests was only 20 to 32 mg/L, and when all rainbow trout tests were pooled and regressed against hardness, the results had no explanatory value ($r^2 = 0.05$). The only tests conducted as a series (the three points with the highest EC_{50}s) only varied from 23 to 32 mg/L, still only resulted in a regression explaining 48% of the variability (not shown).

Cadmium from the South Fork Coeur d'Alene testing shows a similar pattern with an inadequate spread of the hardness data *(*Figure 2.4.2.4*).* If all tests were pooled, the resulting relation is weak with a best fit regression only explaining only about 36% of the variability; when the regression is limited to the four concurrent tests, hardness can explain about 68% of the variability.

This problem of an inadequate spread in the hardness as the independent variable in regressions or pooling disparate data is a common limitation in hardness-toxicity meta-analyses of found data. For example, Meyer *et al*. (2007b) includes a comprehensive review of metals toxicity versus hardness. Their plots often show clumps of poorly distributed hardness values. Two unpublished reviews focusing on soft-water metals toxicity hardness relations showed similar patterns (CEC 2004a; Lipton *et al*. 2004). Mebane (2006, p.20) pooled hardness-toxicity data for rainbow trout and cadmium from across a variety of studies for a total of 37 studies. The plot shows a fair amount of scatter and hardness explained about half the variability in the cadmium acute toxicity data with rainbow trout ($r^2 = 0.56$). In contrast, hardness-toxicity data for brown trout where most data were from a single study that explicitly tested cadmium toxicity across a wide range of hardness showed a much tighter relation between hardness and acute toxicity ($r^2 = 0.97$).

These comparisons show that pooling datasets may also wash out patterns that are only apparent in the smaller, synoptic datasets.

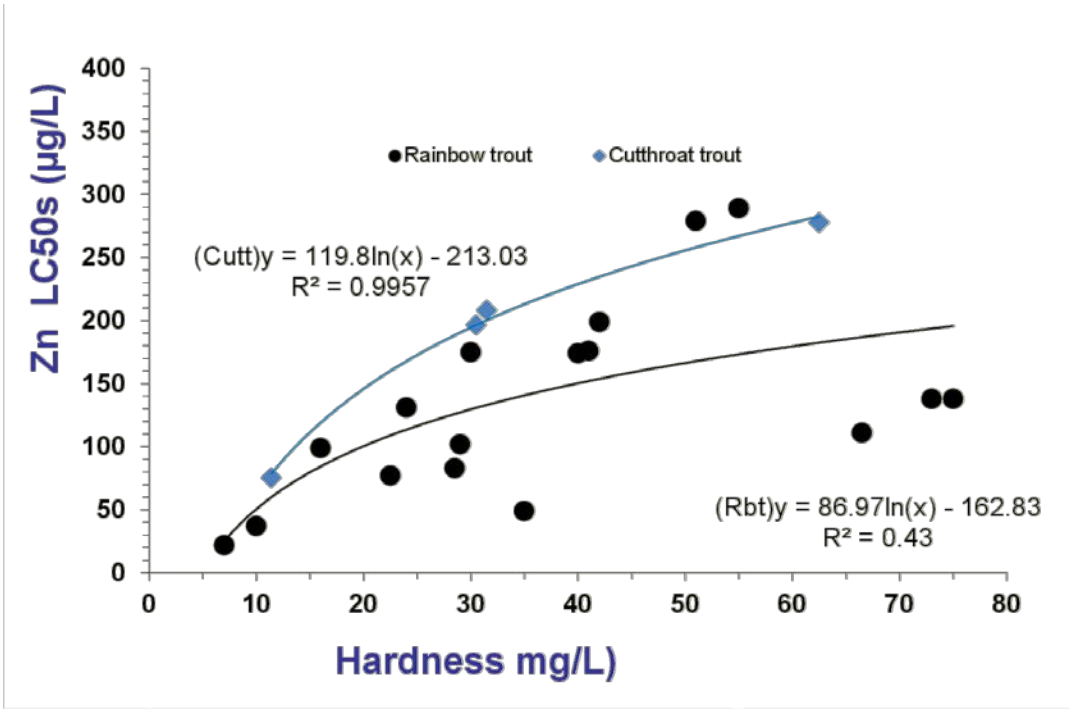


Figure 2.4.2.1. Zinc toxicity versus water hardnesses for swim-up stage rainbow trout pooled across test groups and westslope cutthroat trout (data from Mebane *et al.* (2012).

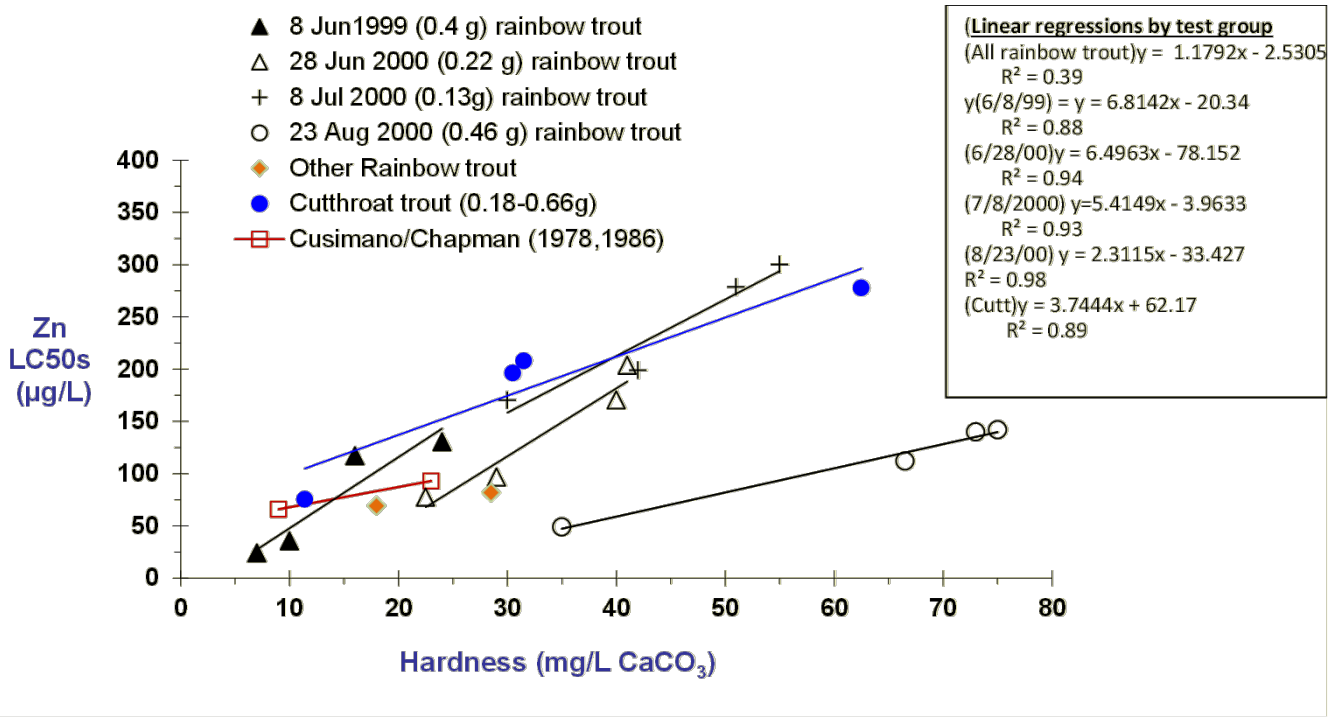


Figure 2.4.2.2. Zinc (Zn) toxicity versus water hardnesses for swim-up stage rainbow trout by concurrent test groups, cutthroat trout, and steelhead tested under similar conditions by the same people (average fry weights are in parentheses) Data from (data from Mebane *et al*. (2012) except for steelhead data which are from Cusimano, Brakke and Chapman (1986)and Chapman (1978b).

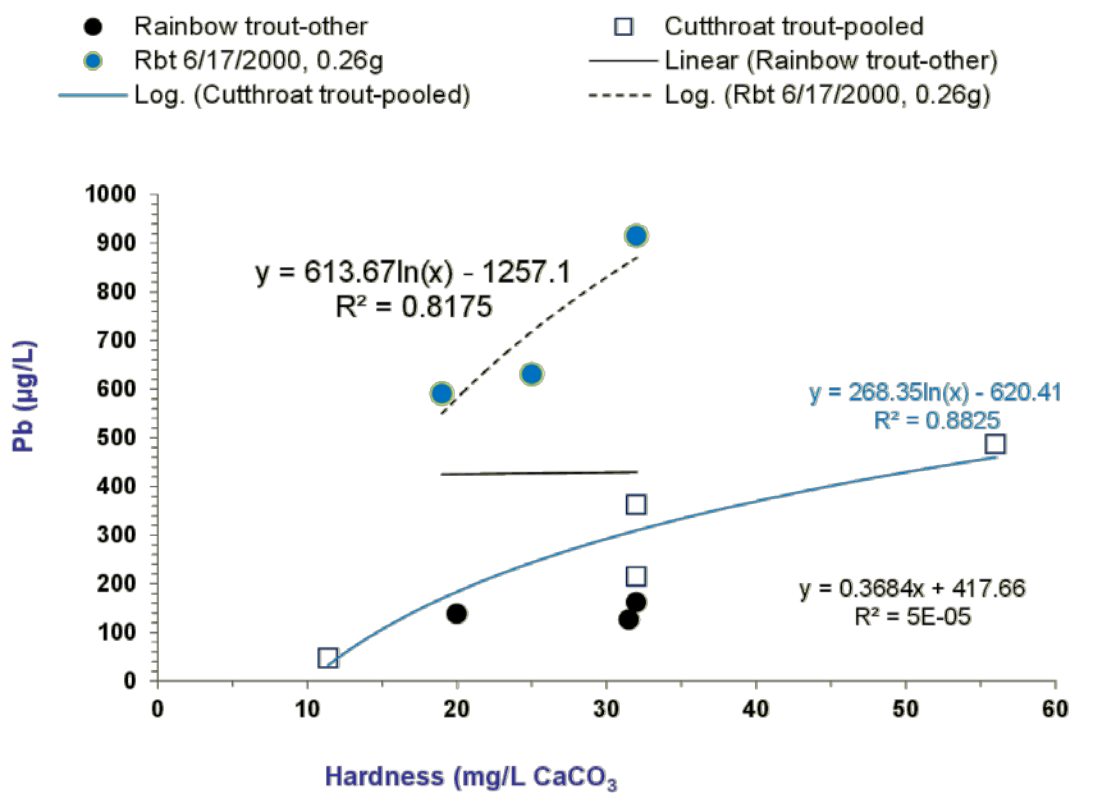


Figure 2.4.2.3. Lead (Pb) toxicity versus water hardnesses for swim-up stage rainbow trout either pooled across test groups, or separated into synoptic and other tests and pooled westslope cutthroat trout (data from Mebane *et al.* (2012).

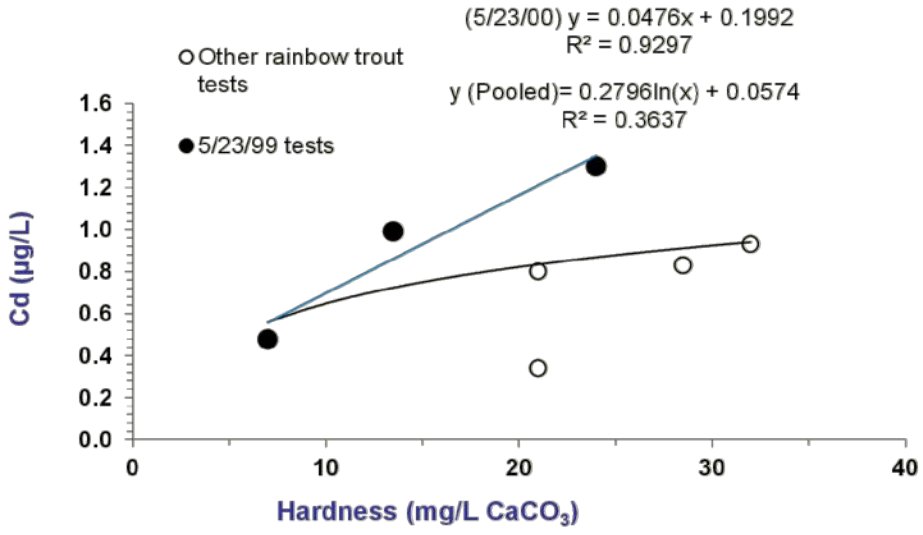


Figure 2.4.2.4. Cadmium (Cd) toxicity versus water hardnesses for rainbow trout tested under the same conditions on 5/23/99 versus "other" rainbow trout tested by the same people in the same facility, using the same source of fish eggs, same water sources, but using fish that were a few weeks apart in age (data from Mebane *et al.* (2012).

Relevance of the hardness floor issue in the action area. Nationally, about 20% of the freshwaters can be considered "softwater" (Figure 2.4.2.5). Within the range of listed salmon or steelhead "salmon country" in Idaho, water hardness tends to decrease from south to north (Figure 2.4.2.6). In the Salmon River drainage in the southernmost portion of the range of anadromous fish in Idaho ("salmon country"), water hardnesses are highly variable, apparently depending on the bedrock geology. Hardnesses are relatively high in drainages with carbonate rock (e.g., Lemhi and Pahsimeroi river drainages), intermediate in watersheds with volcanic rock, and very low in the granitic drainages of the Idaho Batholith. The Idaho Batholith is the dominant geologic feature of much of central Idaho (Appendix A, Thomas *et al.* 2003; Hardy *et al.* 2005). Hardnesses as low as 4 mg/L have been measured in softwater areas of Idaho (Figure 2.4.2.6); however, the true minimum hardnesses in streams in granitic watersheds are probably close to that of snowmelt, which is in the range of 0.5 to 1 mg/L total hardness (Clayton 1998).

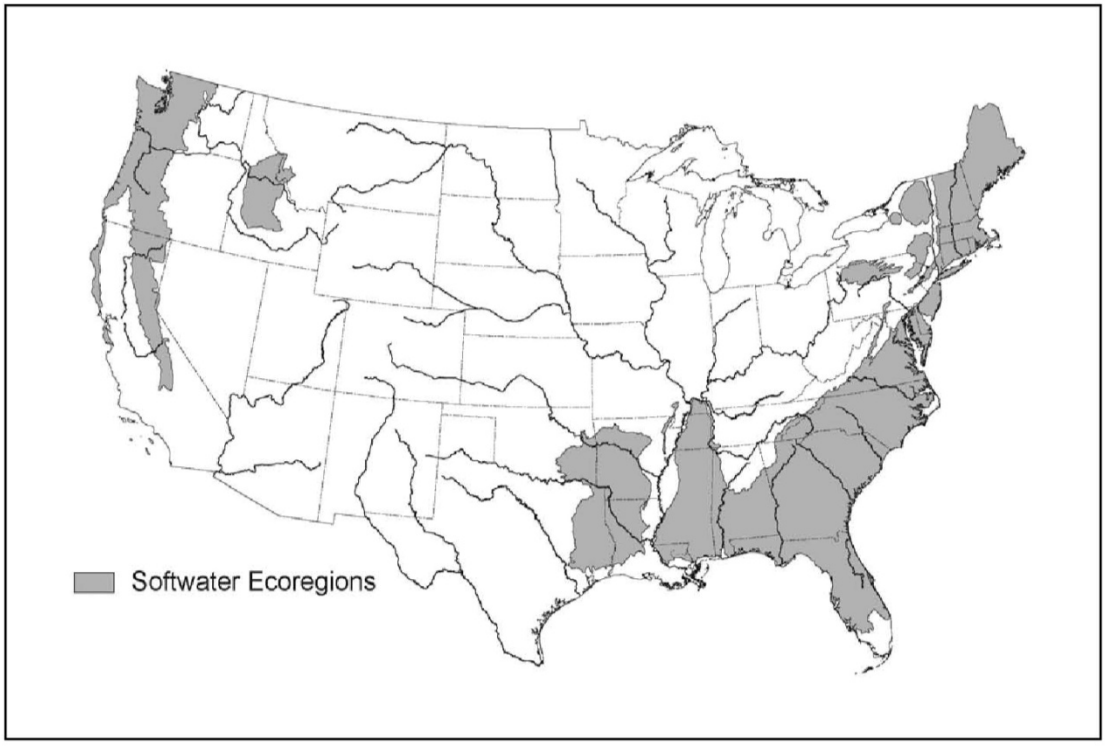


Figure 2.4.2.5. Soft-water ecoregions of the USA where most water hardness values are <50 mg/L CaCO3 (Whittier and Aitkin 2008).

The magnitude of likely effects of the hardness floor on criteria values is probably substantial in waters with the lowest hardnesses within the range of anadromous salmonids in Idaho. The best data sets are from monitoring of waters into which effluents from hard rock mines are discharged. Several major active and inactive mining operations are present in the Salmon River drainage. The inactive operations still discharge effluents and some are regulated by EPA under the NPDES program.

Historically, mining also occurred in the Clearwater River and drainages in the Hells Canyon reach of the Snake River such as around the old mining towns of Cuprum and Florence. However, these mining districts played out and there has been no large scale mining activity in these areas in at least the last 50 years or so. The hardness floor issue in Idaho's salmon country is only relevant to industrial mining. Within salmon country, NPDES effluent limits have been imposed by EPA on one major urban wastewater treatment plant (city of Lewiston), many minor wastewater discharges from small towns and consolidated sewage treatment districts, and two major forest products facilities. NMFS reviewed the fact sheets detailing known or suspected pollutants and calculations of the reasonable potential to exceed metals criteria for these current discharges. Other than the mines, none of the facilities had measured or projected metals concentrations that approached having reasonable potential to exceed any metals criteria. In the case of the city of Lewiston, the maximum concentrations measured in the undiluted effluent exceeded criteria by nine times for copper and about three times for cadmium and zinc. However, the EPA "reasonable potential to exceed" determination assumes that dilution with river water will be allowed using 25% of the receiving water flows, and it is only necessary for facilities to comply with WQS after mixing and dilution. The city of Lewiston discharges into a large river (the Clearwater River) with a minimum dilution ratio of 37 to 1, which would dilute these metals to well below criteria. (*http://yosemite.epa.gov/r10/water.nsf* accessed February 2008). See also Appendix D on issues with mixing zones and dilution assumptions.

Within the Salmon River drainage, the mining operations tend to be located high in watersheds where the waters may have quite low hardness values. In EPA Region 10's effluent limits calculations, EPA tends to use the 5^{th} percentile of measured hardness values, which is a conservative approach. Estimated ranges of water hardnesses for major mining discharges within the ranges of listed salmonids are summarized in Table 2.4.2.1. The hardness floor is a substantive concern in about 75% of the receiving waters.

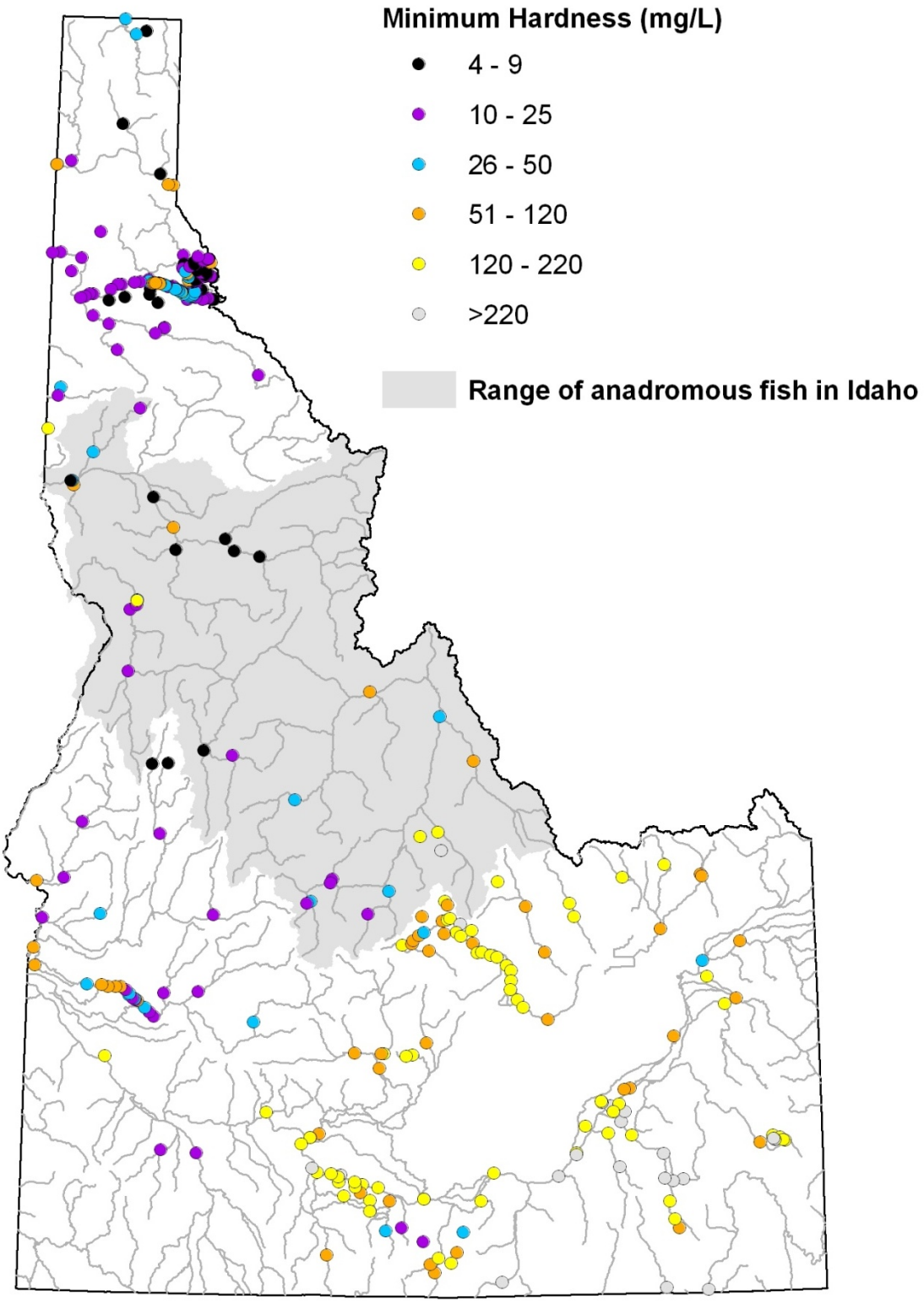


Figure 2.4.2.6. Minimum hardness values measured at 323 sites in Idaho between 1979-2004 (data from Appendix A)

Table 2.4.2.1. Ranges of low hardnesses observed in Salmon River basin receiving waters of industrial mine effluents or nonpoint source mine runoff (limited to major facilities discharging to waters either designated as critical habitats for listed salmonids or at least some portions are accessible and presumably used by listed salmonids.

Stream	Location	Hardness "range" (mg/L as $CaCO_3$)	Statistics for "range"	Source
Napias Creek	Downstream of mine effluent	4 – 6	5th percentiles during high and low-flow tiers respectively	EPA
Big Deer Creek	Downstream of mining-affected tributary (Bucktail Cr)	9 – 36	Range of 6 observations	(Maest *et al*. 1994)
Salmon River	Upstream of permitted but inactive outfall from Thompson Cr. Mine	15 – 54	Range of 14 observations	USGS
Jordan Creek	Downstream of mine effluent	16 – 39	5th percentiles during high and low-flow tiers respectively	EPA
Panther Creek	Downstream of mining-affected tributary (Blackbird Cr)	17 – 48	Range of 68 observations	(Maest *et al*. 1994)
Yankee Fork	Upstream of mine effluent	19 – 54	Range of 47 observations	Note
Yankee Fork	Downstream of mine effluent	24 – 149	Range of 47 observations	Note
Squaw Creek	Downstream of mine effluent	45 – 110	5th percentiles during high and low-flow tiers respectively	EPA
Thompson Creek	Downstream of mine effluent	55 – 85	5th percentiles during high and low-flow tiers respectively	EPA

Notes: "EPA" data from factsheets accessed January 2008 from *http://yosemite.epa.gov/r10/water.nsf*. USGS data from site 13296500, Salmon River below the Yankee Fork, *http://waterdata.usgs.gov/id/nwis/qw*; Yankee Fork data courtesy of B. Tridle, Hecla Mining Co.

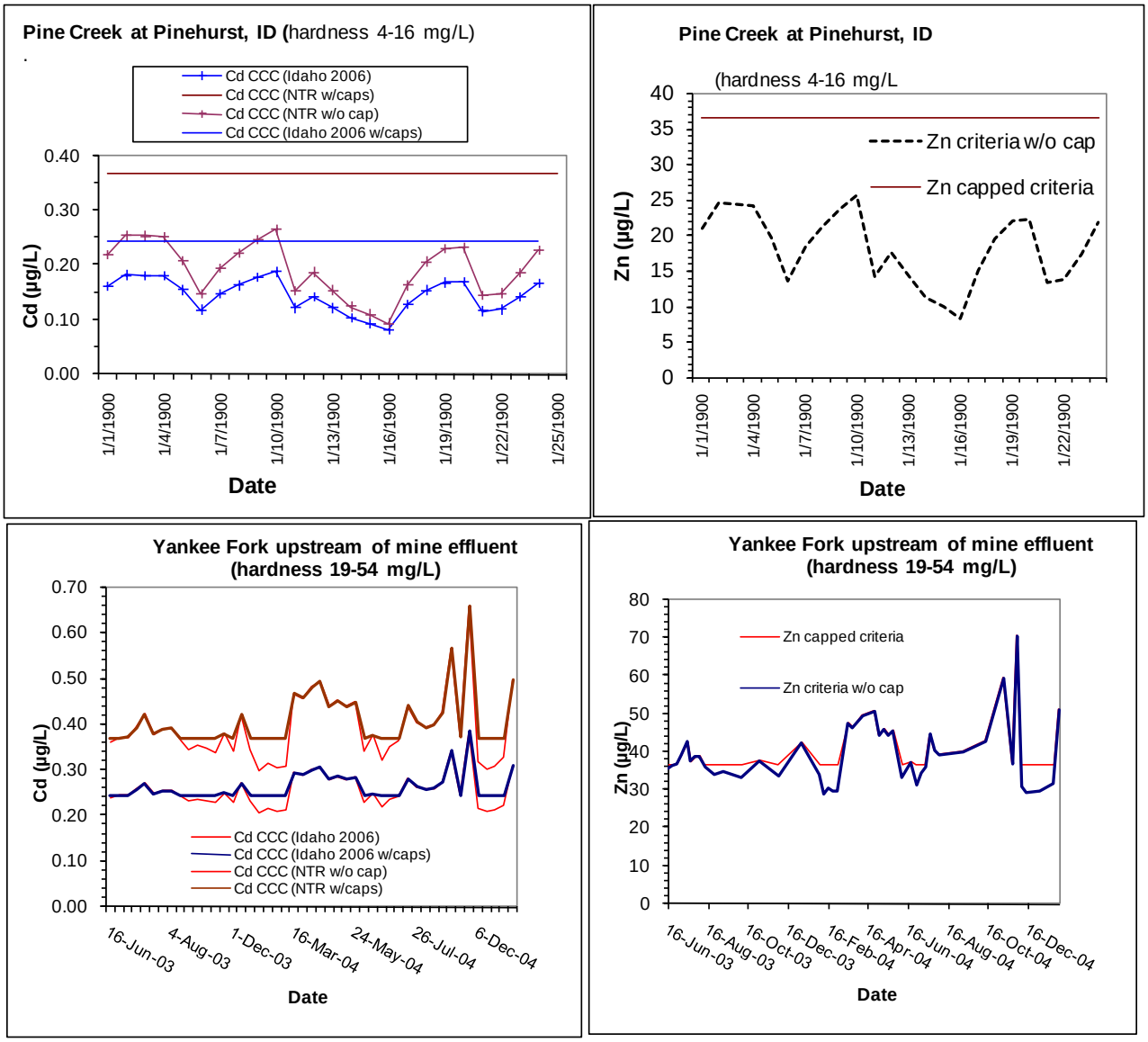


Figure 2.4.2.7. Examples of the effects of the "hardness floor" on cadmium and zinc criteria in very-soft and soft water settings. In the Pine Creek example (top), all hardness observations were less than the 25 mg/L floor. In this very-soft water example, applying a hardness floor would result in the criteria being considerably less protective than intended by EPA Guidelines at all times, with the floor-limited criteria as much as 3X higher. The Yankee Fork example (bottom) is probably more typical of soft water streams in the Salmon River drainage. There the floor has little or no effect during much of the record, at the worst the floor-limited criteria were about 1.25X higher than the hardness-dependent criteria.

Thus, there are many streams in the Salmon River and Clearwater River drainages in Idaho 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, and which have active metals discharges.

The magnitude of likely effects of the hardness floor on criteria values is compared graphically in Figure 2.4.2.6. The first illustration, from Pine Creek, a tributary to the South Fork Coeur d'Alene River, Idaho, is located outside the salmon country area of interest, but is shown because it is probably similar to the streams with very low hardnesses and because it had a robust data set. In this example, the "floor-limited" criteria values are up to three times higher than criteria calculated on relevant site hardness values. In this stream, because the hardness never rises above 25 mg/L, the hardness-floor-limited criteria plot as horizontal lines. While the hardness of Pine Creek is very low, ranging from only 4 to 16 mg/L in Figure 2.4.2.1, it is not uniquely low. In the North Fork Payette River at McCall, Idaho, measured hardnesses only ranged from 6 to 7 mg/L, n=9 (Hardy *et al*. 2005). Since the North Fork Payette River upstream of McCall shares similar geology as much of the adjacent South Fork Salmon River drainage, similarly low hardness values are presumed to occur in the South Fork Salmon River drainage.

In the more intermediate example of the Yankee Fork upstream of mine effluent, the floor-limited criteria are only biased high (unprotective) compared to the uncapped criteria by 1.2 times or less.

2.4.2.2. Summary of Effects of the Hardness Floor for Calculating Metals Criteria

Exposure of listed Snake River salmon and steelhead to levels of metals in discharges at proposed criteria levels will result in adverse effects. Many of the streams in the Salmon River and Clearwater River drainages of Idaho also have hardness concentrations that average less than 25 mg/L which is the current floor in the hardness equation. For copper and lead, hardness is less important than DOC, but if DOC is low, toxicity does increase below the hardness floor. For nickel, and zinc, acute toxicity to fish rises as hardness declines below the 25 mg/L. For silver, acute toxicity increases modestly in early life stages, below the hardness floor.

The use of a hardness floor of 25 mg/l in calculating metals discharge limits will allow for increased exposures of listed fish to levels of metals that result in adverse effects. These effects range from a direct increase in mortality to decreases in growth and survival of juvenile Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River Sockeye salmon and Snake River Basin steelhead.

2.4.3. The Effects of EPA Approval of the Arsenic Criteria

Arsenic is been well known for its high dietary toxicity to humans for hundreds of years, and arsenic poisoning was a popular method of political assassination and murder starting at least in

the Middle Ages. To mammals, arsenic is carcinogenic, mutagenic, and teratogenic, and at high enough dietary exposures can be directly lethal. Compared to mammalian toxicology, relatively little work has been done with fish at environmentally relevant exposures (Sorenson 1991).

At environmentally relevant concentrations, adverse effects in fish from arsenic are most likely from dietary rather than waterborne exposures as discussed below. Arsenic and selenium interact with each other in various metabolic functions and each element can substitute for the other to some extent, which could partly explain the reported protective effect of selenium against some arsenic-linked diseases (Plant *et al*. 2007).

The water quality criteria concentrations that are evaluated as part of this action are: acute criterion, not to exceed 360 µg/L; and chronic criterion, not to exceed 190 µg/L. Also applicable to all waters in the action area is a recreational use criterion of 10 µg/L. Whereas all of Idaho's aquatic life criteria are expressed as dissolved metals, the IWQS are ambiguous whether the human health based 10 µg/L is expressed as dissolved or total recoverable arsenic. The rules only state that the criteria addresses "inorganic arsenic only" (IDEQ 2007a). The latter provision is unexplained and is curious because organic arsenic species probably have different bioavailablity and toxicity than inorganic species. Plant *et al*. (2007) stated that organic arsenic forms are likely more bioavailable and toxic than inorganic forms, although as discussed later in the section, organic arsenic may be less toxic than inorganic arsenic in the diet of fish. Presumably the human-health recreational use standard was intended as total arsenic since those "fishable and swimmable" criteria address exposures from incidental consumption of water while swimming or eating fish. Neither swimmers nor fish can be expected to filter their water prior to ingestion.

The human health based criteria apply to all waters in Idaho unless there are specific exclusions. The IWQS have one such exclusion, Bucktail Creek, a small stream contaminated by mine waste. Bucktail Creek is a tributary to Big Deer Creek, which is a tributary to Panther Creek, which in turn a tributary to the Salmon River, in the Middle Salmon-Panther hydrologic unit (Figure 1.4.3.1). The Middle Salmon-Panther hydrologic unit is designated as critical habitat for Snake River spring/summer Chinook salmon. This critical habitat designation is defined to include river reaches presently or historically accessible (except reaches above impassable natural falls (NMFS 2004). Most of the Big Deer Creek watershed, including Bucktail Creek is located above an impassable natural fall. Within a mile upstream from the mouth of Big Deer Creek with Panther Creek, a series of natural cascades and waterfalls block upstream passage by anadromous fish (Reiser 1986). Therefore Bucktail Creek is not considered to be within the critical habitat designations for either Snake River spring/summer Chinook salmon or steelhead. Designated critical habitat for Snake River Basin steelhead is defined specifically by water body; only the lowest reach of Big Deer Creek, not including Bucktail Creek, is designated critical habitat for Snake River basin steelhead.

2.4.3.1. Species Effects of Arsenic Criteria

Arsenic toxicity does not vary significantly with hardness (Borgmann *et al*. 2005a). Because IDEQ has inclusive rules for designated aquatic life and recreational uses, the human-health

related criteria also apply in all designated critical habitats and waters inhabited by listed salmon and steelhead in Idaho (IDEQ 2007a).

Acute Arsenic Criterion. No studies were found that reported acute toxicity to juvenile or adult salmonids at arsenic concentrations close to the acute criterion. All studies NMFS reviewed indicate that acute toxicity, including to alevins, occurs at concentrations that are significantly higher than the acute criterion (e.g., Buhl and Hamilton 1990). Ambient arsenic concentrations in surface water are never known to approach the acute criterion.

Chronic Arsenic Criterion. A conclusion that can be drawn from a recent comprehensive review of arsenic toxicology in fishes by McIntyre and Linton (2011) is that arsenic is not very toxic in classic toxicity tests with exposures through water. The results of Birge *et al*. (1978, 1981) suggests that chronic arsenic toxicity from waterborne exposures occurs to developing embryos of listed salmonids at concentrations below the chronic criterion. Rainbow trout embryos were exposed to arsenic for 28 days (4-days post-hatching) at 12°C to13°C and a hardness of 93 mg/L to 105 mg/L $CaCO_3$ in static tests. Concentrations of 42 to 134 µg/L were estimated to be associated with the onset of mortality, as LC1 and LC10 respectively (Birge *et al*. 1980). No detail of the results of this test were reported beyond these statistical effects estimates, making these results impossible to critically review. Acclimation appears to enhance resistance to chronic arsenic toxicity (Dixon and Sprague 1981; EPA 1985a). Studies reviewed in Eisler (1988a) and EPA (1985a) indicate that chronic effects do not occur in other lifestages 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. The reported concentrations associated with chronic embryo and fry mortality were much lower than the chronic criterion.

Dietary toxicity of arsenic. Cockell *et al*. (1991) fed rainbow trout arsenic contaminated food under standard laboratory conditions for 12 to 24 weeks and correlated signs of toxicity with diet and tissue arsenic concentrations. They found that the threshold for the onset of organ damage (gall bladder inflammation and lesions) was between 13 and 33 mg/kg arsenic in food. Woodward *et al*. (1994, 1995) fed rainbow trout a diet made from invertebrates collected from the metals contaminated Clark Fork River, Montana, which resulted in lower growth and survival of the fish fed the metals contaminated wild diet. However, because these wild metals-contaminated invertebrates were contaminated with several metals including arsenic, and the effects were equally correlated both with arsenic and copper, effects could not be attributed to either. Subsequently Hansen *et al*. (2004) collected metals-contaminated sediments from the Clark Fork River, reared aquatic earthworms (*Lumbriculus*) in them, and fed the *Lumbriculus* to rainbow trout. Fish fed the *Lumbriculus* diet had reduced growth and physiological effects, and the presence of effects was strongly correlated with arsenic but not to other elevated metals. Bull trout collected from mining-influenced Gold Creek in northern Idaho, showed similar liver damage with inflammation, necrosis and cellular damage. Arsenic was elevated in the sediments, periphyton, and macroinvertebrates, and fish tissues, and was correlated with the liver damage (Kiser *et al*. 2010). Erickson *et al*. (2010) further implicated arsenic as the causative agent by experimentally mixing arsenic into clean sediments, rearing *Lumbriculus* in them, and feeding the *Lumbriculus* to rainbow trout. The rainbow trout fed the worms that had been raised in arsenic dosed sediments again had reduced growth and disrupted digestion. Erickson *et al*. (2010) is difficult to directly compare to feeding studies with field collected invertebrates

because Erickson *et al.* (2010) did not report what tissue concentrations bioaccumulated in fish following 30 days on a diet of arsenic enriched invertebrates. Still, the Erickson *et al.* (2010) study produced similar effects to those from field-collected diets with controlled exposures to contaminated field sediments and strongly implicated arsenic as an important stressor.

Together these studies have shown that inorganic arsenic in the diet of rainbow trout are associated with reduced growth, organ damage and other physiological effects at concentrations in the diet of about 20 mg/kg dry weight (dw) and above (Cockell 1991; Hansen *et al*. 2004; Erickson *et al*. 2010). Ranges of reported effects in other species are wider. Damage to livers and gall bladders occurred in lake whitefish (*Coregonus clupeaformis*) fed arsenic contaminated diets as low as 1 mg/kg food dw (Pedlar *et al*. 2002). Adverse effects of dietary arsenic to salmonids are summarized in Table 2.4.3.1. Bioaccumulation of arsenic in prey organisms to concentrations higher than 30 mg/kg dw has been documented from the Clark Fork River, Montana; Boulder River, Montana; the Coeur d'Alene River, Idaho; and Panther Creek, Idaho. Concentrations of arsenic in these streams have been measured at higher than background (<~ 5µg/L) but were never documented at concentrations even approaching the chronic water quality criterion of 190 µg/L dissolved arsenic (Table 2.4.3.2). Review of waterborne arsenic concentrations collected from the same waters suggests that bioaccumulation of arsenic in invertebrate prey organisms to concentrations harmful to salmonids appears to be able to occur in streams with dissolved arsenic concentrations on the order of 10 µg/L or less. These studies focused mostly on the effects of arsenic on organs and growth; however at least one study has shown that arsenic in fish diets can affect reproduction, although the single dietary exposure tested was higher (135 mg/kg dw) than in the studies mentioned with salmonids (Boyle *et al*. 2008).

Field studies of resident trout populations in streams influenced by natural geothermal drainage in Yellowstone National Park give indirect evidence of tolerance to elevated arsenic or perhaps density-dependent compensation to low-level toxicity. Goldstein *et al*. (2001) found that naturalized rainbow and brown trout were at least present in some streams with arsenic concentrations in water that were greatly above typical background concentrations. Arsenic was elevated both in water and invertebrates collected from the Snake River at the southern boundary of Yellowstone National Park (Table 2.4.3.2). Trout and sculpin densities at that location appeared robust in comparison to surveys at other least-disturbed rivers in Idaho and the Pacific Northwest (Maret *et al*. 1997; Mebane *et al*. 2003), so arsenic concentrations on the order of 30 µg/L in water and 11 mg/kg in insect tissues were causing no obvious harm to resident fish populations.

Most of the fish feeding and field studies reported total arsenic, without speciation analyses of whether the arsenic was in inorganic or organic forms. Recent evidence suggests that organic arsenic in the diet of salmonids is less toxic than inorganic arsenic (Table 2.4.3.1). Whether the arsenic that occurs in salmonid prey items in streams occurs predominately in inorganic or organic forms is unknown, but is assumed here to be primarily inorganic. Whether dissolved or particulate arsenic contributes more to arsenic risk is also debatable, but the present evidence suggests particulate arsenic may be more of a concern. The Idaho water quality criteria are based on dissolved arsenic, the rationale for which is unstated in EPA's criteria documents. Arsenic is a metalloid rather than a metal, but apparently for regulatory purposes, arsenic was simply considered another metal like cadmium or zinc without any known analysis. While the

information is sparse, field data suggests that dissolved arsenic may be far less important as a source to aquatic food webs than particulate and sediment sorbed arsenic. This suggests that the dissolved arsenic criterion may be less relevant than a sediment, dietary, or tissue residue based criterion.

Table 2.4.3.1. Relevant concentrations of arsenic in the diet of juvenile fish that were associated with adverse effects

Fish Species	Diet source	Effect	Arsenic in diet (mg/kg dw)	Reference
Cutthroat trout	Metals-contaminated invertebrates collected from the Coeur d'Alene R, ID	Reduced growth, liver damage	14-51	(Farag *et al*. 1999)
Cutthroat trout	" " "	None apparent	2.6-3.5	Farag *et al*. (1999)
Rainbow trout	Metals-contaminated invertebrates collected from the Clark Fork River, MT	Reduced growth, impaired digestion	19 – 42	Woodward *et al*. (1994,1995)
Rainbow trout	" " "	None apparent	2.8-6.5	Woodward *et al*. (1994,1995)
Rainbow trout	*Lumbriculus* (aquatic earthworms) contaminated using Clark Fork River sediments	Reduced growth, impaired digestion, liver and gall bladder degeneration	21	(Hansen *et al*. 2004)
Rainbow trout	Diet of *Lumbriculus* exposed to arsenic	Reduced growth	34	(Erickson *et al*. 2010)
Rainbow trout	Diet (pellets) amended with arsenate	Reduced growth, impaired digestion, gall bladder inflammation	33	(Cockell *et al*. 1991)
Rainbow trout, subadult	Diet (pellets) amended with arsenite	Reduced growth	≥ 51	(Hoff *et al*. 2011)
Rainbow trout	Diet (live or pellets) amended with inorganic arsenic (arsenite or arsenate)	Reduced growth	>≈ 20 mg/kg	(Erickson *et al*. 2011)
Rainbow trout	Diet (live or pellets) amended with organic arsenic	Reduced growth	>≈ 100 mg/kg	(Erickson *et al*. 2011)
Rainbow trout	" " "	None apparent	13	Cockell *et al*. (1991)
Lake Whitefish	Diet (pellets) amended with As	Liver and gall bladder damage, no effects on growth	≥1	(Pedlar *et al*. 2002)

Table 2.4.3.2. Relevant concentrations of arsenic in stream water, sediment, and in the tissues of aquatic invertebrates collected from the same streams. Selected undiluted mine effluent concentrations from within the action area are included for comparison. Unless otherwise noted, concentrations are averages, values in parentheses are ranges

Location and notes	Arsenic in water (filtered, µg/L)	Arsenic in water (unfiltered, µg/L)	Arsenic in sediment (mg/kg dw)	Arsenic in invertebrate tissues, average (mg/kg dw)
Effects thresholds (j)			7-33	~ 20
"Typical" USA river waters, not in enriched areas		0.1 – 2 (l)		
Idaho rivers– statewide assessment (h)		2.3 (0.06 – 17)		
Stream sediments, USGS national median			6.3 (l)	
Panther Cr, ID, 1992-93, mining influenced reaches (a, f, i)	<1	102 (max)	27-888	76 (f)
Blackbird Creek, ID (a)	1.1	158 (max)	939	
South Fork Coeur d'Alene (b, c)	0.4 – 4	13 (max)	180	42 (d)
Clark Fork River at Galen, MT (b,d)	15 (3-53)	20 (4-80)	170 (3)	21(e)
Snake River leaving Yellowstone NP, WY (b,e)	34 (8-55)	Nm	38	11 (f)
Snake River at King Hill, ID (b,e)	3 (0.5 – 7)	4 (2-9)	5 (4-7)	1 (0.5 – 2) (f)
Hecla Grouse Creek gold mine, near Custer, Idaho (k)	2.4 (<1-5)	7 (<5 – 55)		
Thompson Creek molybdenum mine, nr Clayton, Idaho (l)	2 – 4 (projected max for new discharge was 30)			

nm- not measured. (a) (Beltman *et al*. 1994; Maest *et al*. 1994; Beltman *et al*. 1999); (b) USGS Water-Quality Data for the Nation, *http://nwis.waterdata.usgs.gov/nwis/qw* ;(c) (Farag *et al*. 1998); (d) (Hansen *et al*. 2004); (e) (Ott 1997); (f) Community sample, (g) caddisfly *Hydropsyche* sp. (h) (Essig 2010) (i) (Mebane 2002a); (j) Effects thresholds for invertebrate residues are from this review; values for sediment are MacDonald *et al*.'s (2000a) threshold and probable effect concentrations. (k) R. Tridle, Hecla Mining Company, unpublished data, Jan 2008 (l) Thompson Creek mine "NPDES" wastewater permit factsheets, accessed January 2008 from *http://yosemite.epa.gov/r10/water.nsf* . (l) (Plant *et al*. 2007)

Tissue concentrations of arsenic associated with chronic responses in fish. McIntyre and Linton (2011) report that regardless of exposure route or form, bioaccumulated fish tissue concentrations associated with chronic effects were remarkably similar among fish. Adverse effects appear likely to occur when whole-body tissue concentrations reach about 2 to 5 mg/kg wet weight (ww). The critical tissue residue concentrations in liver associated with reduced growth may be somewhat lower, around 0.7 to 1.0 mg/kg ww. This range of critical liver

concentrations was supported by recent research reported by Hoff *et al*. (2011) who showed a change point in growth of rainbow trout when arsenic in liver reached about 6 mg/kg dw, which would be equivalent to about 1 to 1.5 mg/kg ww.

In a similar study in the Coeur d'Alene River basin, Idaho, Farag *et al*. (1999) fed fish invertebrates collected from mining influenced reaches and reported reduced growth, liver degeneration, and fish tissue concentrations ranging from about 0.5 to 1.2 mg/kg ww. In contrast, arsenic in fish fed a reference diet collected from a minimally polluted reach of the North Fork Coeur d'Alene River ranged from about 0.2 to 0.3 mg/kg ww (Farag *et al*. 1999). Other metals were also elevated in the fish, particularly lead, although results from Erickson *et al*. (2010) and Hansen *et al*. (2004) argue that most of the toxicity in Farag's study was probably attributable to arsenic.

Whole-body arsenic residues associated with reduced growth in fish following feeding studies (>≈ 0.6 mg/kg ww) are difficult to compare to surveys that only sampled edible fillets (muscle). In a probabilistic study of fish captured from 55 randomly selected river sites throughout Idaho, Essig (2010) obtained a median arsenic concentration of 0.06 mg/kg ww, ranging from <0.13 to 0.31 mg/kg ww in muscle fillets. The highest value in Essig's (2010) report was from a brown trout collected from a geothermally influenced reach of the Portneuf River. In targeted collections of trout in the Stibnite Mine area, arsenic concentration in fillets were up to 0.96 mg/kg, fresh weight), considerably higher than the maximum value from Essig's (2010) randomized survey. In the Stibnite study, arsenic in muscle fillets was considerably lower than in the remaining trout carcasses (e.g., organs, bone, viscera, skin) after the fillets had been removed. Arsenic in fillets ranged from <0.25 to 0.96 mg/kg fresh weight versus 0.32 to 6.3 mg/kg fresh weight in the remainders (Woodward-Clyde 2000).

Behavioral and neurotoxic effects. Despite profound neurotoxic effects of arsenic in mammals, there appears to have been minimal research with behavioral and neurotoxic effects of arsenic in fish. However, the available information reviewed suggests that behavioral effects could be important at very low exposure concentrations. Arsenic impaired long-term memory in zebrafish exposed for 96 hours to arsenic concentrations as low as 1 µg/L before avoidance trials. Measurement of elevated levels of oxidized proteins in brain tissue of fish exposed to 10 µg/L arsenic suggested that the observed effects may have been related to oxidative stress in brain tissue (McIntyre and Linton 2011).

The information reviewed indicates that at environmentally relevant concentrations, arsenic in the diets of salmonids poses significant risks for reduced growth. Reduced growth in turn, may lead to reduced survival or reproduction.

2.4.3.2. Habitat Effects of Arsenic Criteria

Toxicity to Food Organisms. The limited data available suggests that the risk of toxicity to salmonid food organisms is lower than the risk of toxicity to salmonids from eating arsenic exposed organisms. However, we did not locate any studies that tested invertebrates using

environmentally relevant exposures through arsenic enriched periphyton or sediments, and conducted through full life exposures or obviously sensitive life stages.

Norwood *et al*. (2007) related bioaccumulation of arsenic in *Hyalella azteca*, a benthic invertebrate common in slow moving rivers and lakes, to mortality in 4-week exposures. Lethal body concentrations associated with 25% and 50% mortality were about 9 and 10 mg/kg dw respectively. *Hyalella* exposed to Panther Creek, Idaho sediments for 10 days had a trend of decreasing growth and survival with increasing arsenic concentrations (Mebane 1994, 2002a). However, arsenic in Panther Creek sediments was also correlated with cobalt and copper, and correlations between decreased *Hyalella* survival and cobalt and copper concentrations in sediments were stronger than for arsenic, and thus adverse effects were attributed to copper and or cobalt (Mebane 1994, 2002a). However, arsenic bioaccumulation in *Hyalella* probably takes more than 10 days to reach saturation (Norwood *et al*. 2006) and in general, 10-day Hyalella tests can be considerably less sensitive than 4 to 7 week tests (Ingersoll *et al*. 1998). Thus, the Panther Creek study may not have had the necessary duration for detecting effects of arsenic-contaminated sediments.

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 dw) 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. Crayfish collected from Australian streams disturbed by mining activities had up to 100 mg/kg dw arsenic in their tissues. Levels of arsenic in the tissues of the crayfish were similar to those found in the sediment, thus it is highly likely that the primary exposure to arsenic for the crayfish came from the sediment (Williams *et al*. 2008).

Other data we reviewed on arsenic toxicity to aquatic macroinvertebrates were from water only exposures that are unlikely to have much relevance to toxicity under environmental conditions (EPA 1985a; Eisler 1988a; Canivet *et al*. 2001). 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 found increased mortality of gammarid amphipods and heptagennid mayflyies at about 100 µg/L which is lower than the chronic criterion of 190 µg/L.

2.4.3.3. Summary of Effects for Arsenic

If only direct water exposures were considered, arsenic would be of minimal concern to listed salmonids at typical ambient concentrations or at the criteria concentrations under review. The risk of harm from short-term water-only exposures to arsenic concentrations at the acute criterion is unlikely enough to be considered a minor risk for short-term exposures.

The chronic criterion appears to avoid chronic adverse effects to the adult and juvenile salmonid life stages from water-only exposures; however, arsenic concentrations below the chronic

criterion have been reported to cause mortality in salmonid embryos. The chronic arsenic criterion is far higher than concentrations of arsenic sufficient to bioaccumulate in invertebrates to concentrations that cause harm to the salmonids that feed on them. Bioaccumulation of arsenic in prey organisms to concentrations that could be harmful to salmonids has occurred in streams at exposures less than 10 µg/L. As such, adverse effects can occur at the chronic criterion, through reduced growth of juveniles via food web transfer.

2.4.4. The Effects of EPA Approval of the Copper Criteria

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. Adverse effects of copper to salmonids that have been documented at environmentally relevant concentrations include reduced growth and reproductive impairment. A host of initially sublethal physiological and behavioral effects to salmonids have been documented following copper exposures including interference with immune response and reduced disease resistance, reduced swimming stamina, damage to olfactory cellular tissue, impaired olfactory function, which in turn impairs ability of fish to avoid predators, find prey, and migrate from and to their natal streams. Benthic macroinvertebrate communities that form the food base of salmonids in freshwater streams appear particularly sensitive to copper, compared to other metals. The Idaho copper criteria under review in this Opinion are hardness dependent. At a hardness of 100 mg/L the acute criteria for copper is 17 µg/L and the chronic criteria for copper is 11 µg/L .

2.4.4.1. Species Effects of Copper Criteria

Acute toxicity. Available toxicity test data indicate that, under certain conditions, juvenile salmonids can be killed by copper concentrations equal to the final acute value (FAV) used to define the acute criterion. Because acute toxicity data are commonly reported only as the concentrations lethal to 50% of the test population (LC_{50}s), and because 50% test population is a severe effect, the protectiveness of acute criterion is not evaluated by comparing it directly to LC_{50} data. Rather, LC_{50} data are compared to the FAV, which is equal to 2X the acute criterion. The assumption in the criteria derivation and in this opinion is that dividing an LC_{50} value by 2 will result in a concentration that kills few if any organisms. This assumption was critically reviewed in Section 2.4.1.6 and in Appendix B. In this manner, the acute criterion, which is intended to protect against short-term exposures in the environment is compared to short-term LC_{50} toxicity data. Because the chronic criterion only comes into play for exposure scenarios longer than 96-hours, the acute criterion regulates allowable concentrations from >1-to-96 hours.

The studies reviewed indicate that LC_{50}s for adult listed salmon and steelhead are slightly higher than the proposed criterion that is the FAV divided by two. This is consistent with older summaries that found LC_{50} values for adult salmon and trout were well above the proposed acute criterion (EPA 1985d; Eisler 1998a). Figure 2.4.4.1 shows all acute data NMFS reviewed, for tests in waters with hardness less than 200 mg/L, irrespective of lifestage. (We consider waters

with hardness of less than 200 mg/L more representative of waters in the action area.) Although most of the LC_{50} values are higher than the FAV, a substantial minority are lower. Many of the tests for which the FAV would not be protective fall in two general categories: test waters with low hardness; and waters in which magnesium contributes much of the measured hardness values, that is Ca:Mg ratios are lower than in most of the tests used to develop criteria (Welsh *et al*. 2000a; Naddy *et al*. 2002). However, others appear to capture sensitive life stages or stock. For instance, Chinook salmon exposed to copper in pH 7.7 at hardness 35 mg/L resulted in an LC_{50} of 7.4, which is lower than the hardness adjusted FAV of 13 µg/L. Rainbow trout tested in hardness 25 mg/L at pH 6 yielded a LC_{50} of 2.4 µg/L which is less than the FAV of 9.2 µg/L at hardness 25 mg/L (Fig. 2.4.4.1, data from Stratus (1996;1998).

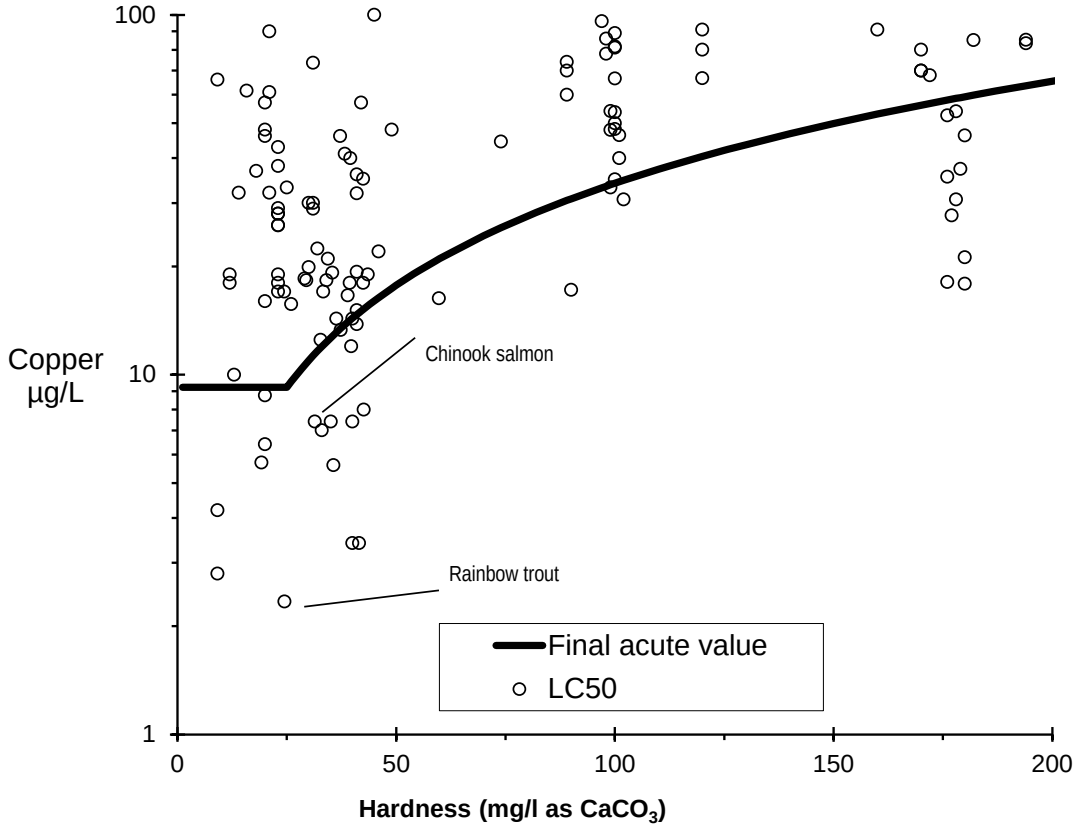


Figure 2.4.4.1. Comparison of 96-hour LC_{50}s for salmonids with copper and the Idaho criterion final acute values, calculated for hardnesses up to 200 mg/L as CaC03. LC_{50}s limited to species within the genera Oncorhynchus, Salvelinus, and Salmo. If all LC_{50} values fell above the line, that would suggest that for the most part, few mortalities would be likely at criterion concentrations.

Chronic Toxicity. Numerous adverse effects have been reported that were attributable to long-term exposures of salmonids and other fish to copper. "Chronic effects" as used here refer to

effects resulting from long-term exposures, and effects from such long-term exposures can include mortality or sublethal effects.

The most sensitive endpoint in some chronic tests with copper and fish was reproductive impairment, as reduced fecundity (Mount 1968; Mount and Stephan 1969; McKim and Benoit 1971; Suter *et al*. 1987). However, with anadromous steelhead and salmon, presumably long-term exposure of adults to copper in freshwater would be unlikely, since adults are either only passing through migratory areas or are exposed on their spawning grounds for a few weeks or less. Thus, the risk of chronic effects from copper is higher for juvenile fish.

Reduced immune response and disease resistance is an effect of copper that appears to be understudied, considering its potential implications. Stevens (1977) reported that pre-exposure to sublethal levels of copper interfered with the immune response and reduced the disease resistance in yearling coho salmon.

Other chronic effects include damage to olfactory tissues, reduced swimming speed, and reduced growth (Table 2.4.4.1).

Growth effects and population-level risks. Comparisons of available chronic copper effects data with salmonids and the Idaho chronic criteria were unfavorable to the criteria. In contrast to the acute LC_{50} data for salmonids with copper where at least most values were higher than the Idaho final acute value, with the Idaho chronic criterion about as many adverse effects were documented to occur at or below the criterion concentrations as above (Figure 2.4.4.2). Relevant studies are described in more detail in Table 2.4.4.1.

A common chronic effect observed with copper exposure has been reduced growth in laboratory toxicity tests with salmonids. In tests in soft water, copper concentrations CCC caused about a 4% to 7.5% reduction in the lengths of Chinook salmon and rainbow trout, depending on the statistical model used to analyze the toxicity data (Table 2.4.4.1). However, the relevance of subtle and sometimes transitory growth reductions under laboratory conditions to natural-origin populations may not be obvious. One study used population modeling to estimate the relevance of subtle and sometimes transitory growth reductions under laboratory conditions to natural-origin populations (Mebane and Arthaud 2010). Demographic data from Marsh Creek, Idaho, was used as a "model" headwaters population of Snake River spring/summer Chinook salmon to develop the population model (Mebane and Arthaud).

The size of juvenile salmon as they first migrate from Marsh Creek is a strong predictor of their survival during the initial part of their seaward migration. Growth reductions in laboratory tests were extrapolated to reduced survival in the wild through the size-survival correelations of migrating juvenile fish. Reductions in growth predict disproportionate reductions in survival of migrating juveniles. For average sized migrants, a 4% to 7.5% length reduction predicts about a 14% to 26% reduction in survival from Marsh Creek to the LGD, the next downstream census

point, 640 km downstream. The study used these changes in juvenile survival rates to adjust the life stage survival rates in the population model, to estimate the population-level consequences of low-level copper stress on juvenile Chinook salmon.

The study projected population-level risks for up to six generations (30 years). Risks of severe decline or quasi-extinction were slightly higher under the copper-influenced scenarios, compared to baseline risks with no copper. Severe declines or quasi-extinction were defined as a 90% reduction of adult spawners or five-consecutive runs with less than 25 spawners each year respectively. Risks of "quasi-extinction" rather than absolute extinction were projected because of biological and mathematical difficulties reaching true zero in the population model. Risks of severe decline occurring in a single spawning run over a 30-year projection were about 75% for the baseline scenario, and 76% to 79% for the copper CCC scenarios. Quasi-extinction risk projections for the same time period averaged 23% for the baseline scenario and 26% to 31% for the copper CCC scenarios (Mebane and Arthaud 2010).

Projections of population recovery times differed more between the scenarios than did the risks of decline. The baseline scenario was projected to meet a relative recovery threshold of 500 adults in about 11 years, and the 4% to 7.5% copper growth reduction scenarios were projected to meet the recovery threshold in the 18 to 28 years (Mebane and Arthaud 2010). The model results mentioned here all assumed density dependence, that is, the population cannot increase above an assumed carrying capacity). While the modeling used a real population to increase realism, all of these risks and population projections should be interpreted in a relative sense in comparison between the scenarios, not as absolute predictions.

Chemosensory and Behavioral Effects. Sensory system effects are generally among the more sensitive fish responses and underlie important behaviors involved in growth, reproduction, and (ultimately) survival (i.e., predator avoidance). Recent experiments on the sensory systems and corresponding behavior of juvenile salmonids contribute to more than 4 decades of research and show that dissolved copper is a neurotoxicant that directly damages the sensory capabilities of salmonids at low concentrations. (Hecht *et al.* 2007). These effects can manifest over a period of minutes to hours and can persist for weeks. To estimate toxicological effect thresholds for dissolved copper in surface waters, Hecht *et al.* (2007) calculated benchmark concentrations (BMCs) for juvenile salmonid olfactory function based on recent data. The BMCs ranged from increases of 0.18 to 2.1 µg/L above background copper concentrations, corresponding to reductions in predator avoidance behavior of approximately 8% to 57%. The BMC examples represent the increases in dissolved copper concentration above background copper concentrations, which were up to 3 µg/L in the tests used to derive the BMCs. These levels are expected to affect the ability of juvenile salmonids to avoid predators in freshwater. These BMCs are much less than the corresponding acute Idaho criteria of 20 µg/L, and even the chronic criteria of 13 µg/L (for a hardness of 120 mg/L for the conditions of a test that was used in the derivation of the BMC, Table 2.4.4.1). These BMCs thresholds for juvenile salmonid sensory and behavioral responses fall within the range of other low sublethal endpoints affected by dissolved copper such as behavior, growth, and primary production, which is around 0.75–2.5 µg/L (Hecht *et al*. 2007).

Studies showing diminished predator avoidance behaviors of juvenile salmon in the presence of elevated copper have subsequently been expanded through predation experiments (McIntyre 2012). Short-term (30 min) copper exposure made prey easier for predators to detect and capture. The primary impact of copper on predator-prey dynamics in her study was faster prey detection, manifested as faster time to attack and time to capture. Cutthroat trout were more effective predators on copper-exposed coho during predation trials, as measured by attack latency, survival time, and capture success rate. The shift in predator-prey dynamics was similar when predators and prey were co-exposed to copper. The onset of these effects occurred at concentrations less than the acute criterion for copper: predatory cutthroat trout captured and ate juvenile coho salmon that had been exposed to 4.5 µg/L copper in only about 1/3 of the time needed to capture and eat coho that had not been exposed to copper (McIntyre 2012). For the water hardness of the test chambers, 56 mg/L, the acute criterion was 10 µg/L.

Hardness and Other Parameters as Predictors of Copper Toxicity. A number of water quality characteristics influence the toxicity of copper. A conclusion that generally seems to hold across most data and studies we reviewed is that in laboratory waters that have low and uniform DOC present, increasing hardness will usually result in alkalinity and pH naturally increasing as well. In this case, decreasing acute copper toxicity will be expected. However, this pattern may not be consistent for chronic copper toxicity in similar laboratory waters, and it most certainly does not hold for natural waters that have variable DOC and pH.

Chakoumakos *et al*. (1979) determined that hardness and alkalinity influenced the LC_{50} of copper to cutthroat trout, whereas pH had greater influence on the speciation of copper involved in toxicity. They recommended that water quality criteria for copper include all three parameters: hardness, alkalinity, and pH. Miller and Mackay (1980) determined that the incipient lethal concentration of copper varied more rapidly with changes in alkalinity in moderately hard (98 mg/L) water than in soft (12 mg/L) water. Conversely, Lauren and McDonald (1986) varied pH, alkalinity, and hardness independently and determined that alkalinity was an important factor reducing copper toxicity to juvenile rainbow trout with no significant influence of increasing hardness. Lauren and MacDonald (1986) argued that the degree of acclimation to ambient hardness levels could explain the difference in results. Meador (1991) found that both pH and DOC were important in controlling copper toxicity to *Daphnia magna*. Welsh *et al*. (1993) evaluated the importance of DOC in affecting the toxicity of copper to fathead minnows and suggested that water quality criteria be reviewed to consider the toxicity of copper in waters of low alkalinity, moderately acidic pH, and low DOC concentrations. Applications of gill models to copper binding also consider complexation by DOC, speciation and competitive effects of pH, and competition by calcium ions. Welsh *et al*. (1993) varied several test water qualities independently and found that pH, hardness, sodium, DOC, and suspended solids have important roles in determining copper toxicity. They also suggested that it may be difficult to sort out the effects of hardness based on simple toxicity experiments.

The data NMFS reviewed also suggested that increasing hardness affords more protection for acute copper exposures than for chronic. Hansen *et al*. (2002b) found a clear relationship between ACRs and water hardness, with lower ACRs at higher hardness levels. Similarly with acute and chronic exposures of copper to *Daphnia magna,* Chapman *et al*. (1980) found that increasing hardness from about 50 to 200 mg/L consistently increased the acute resistance of

Daphnia to copper, but with chronic exposures, resistance only increased with increasing hardness from 50 to 100 mg/L; increasing hardness from 100 to 200 mg/L provided no additional resistance to copper. These results have disturbing implications for a chronic copper criterion because they contradict a fundamental assumption in the criteria derivation (EPA 1985d) that that chronic toxicity is similarly modified by water hardness as acute criteria, and the chronic criterion varies with hardness as a fixed proportion of the acute criteria.

Tests that used natural waters or approximated natural waters by varying DOC along with hardness and other parameters have repeatedly found that hardness is a minor influence on the toxicity of copper to aquatic invertebrates and fish (Appendix C; Hyne *et al*. 2005; Markich *et al*. 2005; Wang *et al*. 2009). The results of these studies indicate that the use of site calcium plus magnesium hardness only as input to an equation to derive a criterion for copper may not be sufficiently protective of listed salmon and steelhead, and that the criteria need to also consider the influences of DOC and pH as key water quality variables that are more important for modulating toxicity. This issue is described in more detail in the Section 2.4.2, "*The Influence of Hardness on Metals Toxicity*" and Appendix C.

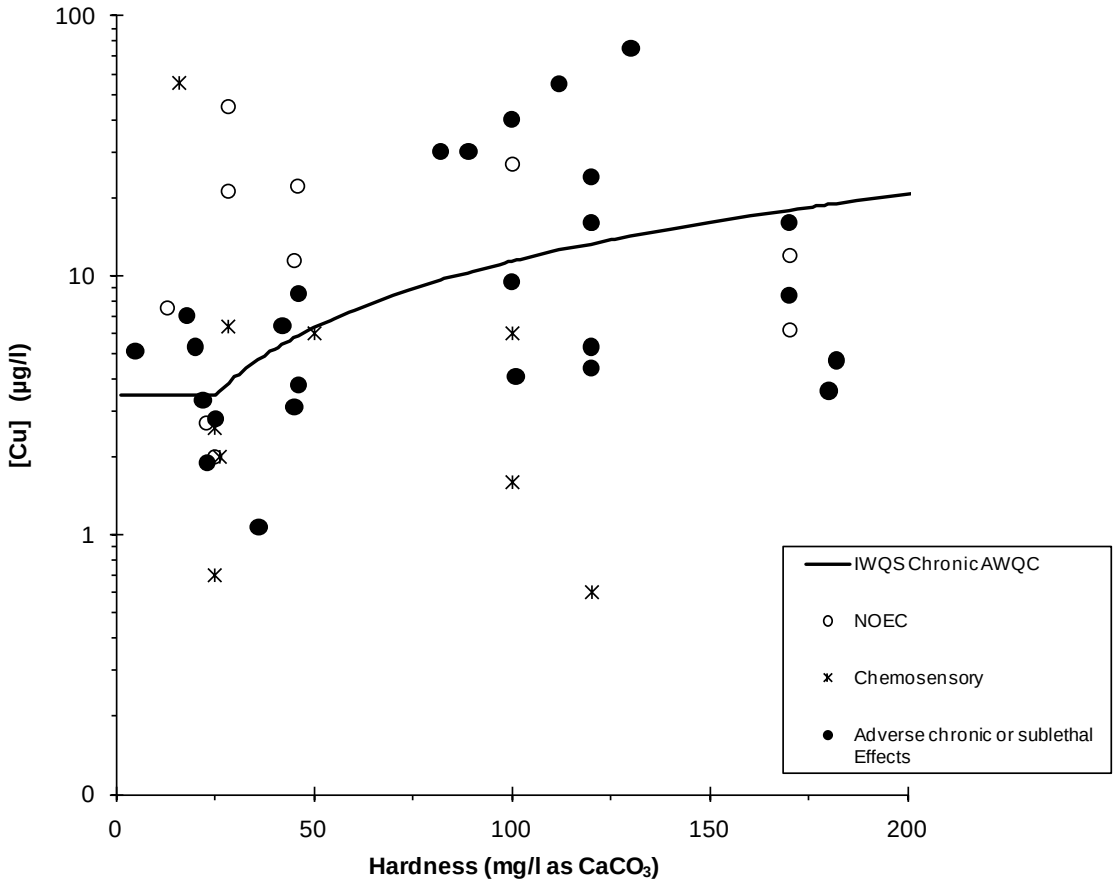


Figure 2.4.4.2. Comparison of the copper Idahochronic criterion and adverse chronic or sublethal effects and estimates of no-effect concentrations to salmonids.

Table 2.4.4.1. Relevant effects and risk ratios of copper to salmonids or other ecosystem components, emphasizing effects that occurred at <u>lower concentrations</u> than the relevant Idaho criteria. Long-term effects (> 4 days to occur) are compared to the chronic criterion, short-term sublethal effects to the Idaho acute criteria, or for acute LC_{50}s, the Idaho final acute value. Risk ratios greater than 1.0 are considered harmful.

Species	Effect	Exposure duration	Hardness (mg/L)	Effect statistic	Effect concen-tration (µg/L)	Criterion (µg/L)	Risk ratio (r=NTR/ effect) concen-tration	Source/ Notes
	Sublethal effects							
Coho salmon (juvenile)	Reduced olfaction and compromised alarm response	3 hours	120	EC10 - EC50	0.18 to 2.1	20.2	112 to 9.6	1
Coho salmon (juvenile)	Reduced olfaction and compromised alarm response	3 hours	120	~EC25	0.6	20.2	34	1
Coho salmon (juvenile)	Shorter time to get captured and eaten	3 hours	56	~EC50	5	10	2	(McIntyre 2012)
Chinook salmon (juvenile)	Avoidance in laboratory exposures	20 minutes	25	LOEC	0.75	4.6	6.1	2
Rainbow trout (juvenile)	Avoidance in laboratory exposures	20 minutes	25	LOEC	1.6	4.6	2.9	2
Chinook salmon (juvenile)	Loss of avoidance ability	21 days	25	LOEC	2	3.5	1.7	2
Atlantic salmon (juvenile)	Avoidance in laboratory exposures	10 minutes	20	LOEC	2	4.6	2.3	3
Coho salmon	Delays and reduced downstream migration of copper exposed juveniles	6 day	95	LOEC	5	10.9	2.2	4
Chinook salmon	Reduced growth (as weight)	120 days	25	EC10	1.9	3.5	1.8	5
Rainbow trout	Reduced growth (as weight)	60 days	25	EC10	2.8	3.5	1.2	6
Rainbow trout	Reduced growth (as weight gain)	56 days	102	EC10	4.1	11.5	2.8	(Hansen *et al*. 2002b)
Rainbow trout	Reduced critical swimming speed, pH 6	30 days	30	EC10	5	4.1	0.8	(Waiwood and Beamish 1978)
Rainbow trout	Reduced growth rate, pH 7.5	30 days	30	EC25	6	4.1	0.8	(Waiwood and Beamish 1978)

Species	Effect	Exposure duration	Hardness (mg/L)	Effect statistic	Effect concen-tration (µg/L)	Criterion (µg/L)	Risk ratio (r=NTR/ effect) concen-tration	Source/ Notes
Rainbow trout	Reduced growth rate, pH 6	30 days	30	EC25	2	4.1	2	(Waiwood and Beamish 1978)
Brook trout	Delayed growth (as weight)	23 weeks	45	EC10	3.1	5.7	1.9	7
Brook trout	Reduced growth (as weight)	3 months	45	EC10	8.5	5.7	0.7	8
Brook trout	Slight mortality	3 months	45	EC10	17	5.7	0.3	7
Brook trout	Complete mortality	22-months	45	EC100	17	5.7	0.3	7
Brook trout	Reduced growth (as weight)	60 days	37	EC10	1.1	4.9	4.4	9
Brook trout	Reduced growth (as weight)	60 days	187	MATC	6.3	19.3	3.1	9
Brook trout	Reduced growth (as weight)	60 days	181	EC10	4.8	18.1	4	(Besser *et al*. 2001a)
	Habitat effects: Adverse effects to ecosystem components							
Ecosystem function	Reduced photosynthesis	~ 1 year	49	LOEC	2.5	6.2	2.5	10
Ecosystem structure	Loss of invertebrate taxa richness in a mountain stream	~ 1 year	49	LOEC	5	6.2	1.2	11
Macroin-vertebrate community	abundance (total individuals)	10-d	60	EC50	6	7.3	1.2	12
Snail, *Leptoxis praerosa*	80% mortality in *in situ* river exposures	114-d	136	LOEC	6.3	14.8	2.3	13
Idaho springsnail	25% mortality	28-d	170	EC25	11	17.9	1.6	15
Bliss Rapids snail	25% mortality	28-d	170	EC25	14	17.9	1.3	15
Snake River pebblesnail	25% mortality	28-d	170	EC25	10	17.9	1.8	15

Species	Effect	Exposure duration	Hardness (mg/L)	Effect statistic	Effect concen-tration (µg/L)	Criterion (µg/L)	Risk ratio (r=NTR/ effect) concen-tration	Source/ Notes
Sculpin, *Cottus bairdi* (MO)	97% mortality	28-d	100	LOEC	7.8	11.4	1.5	14
Sculpin, *Cottus bairdi* (MO)	No mortality or growth effects	28-d	100	NOEC	30	11.4	0.4	14
	Acute Lethality							
Steelhead/ Rainbow trout (fry)	Death (pH 7)	96 h	9.2	LC_{50}	2.8	4.6	3.3	16
Steelhead/ Rainbow trout (fry)	Death (pH 5.7)	96 h	9.2	LC_{50}	4.2	4.6	2.2	16

Table notes (data sources): 1. (Hecht *et al*. 2007; Sandahl *et al*. 2007); 2. (Hansen *et al*. 1999); 3. (Sprague *et al*. 1965); 4. (Lorz and McPherson 1976, 1977); 5. (Chapman 1982); 6. (Marr *et al*. 1996) ; 7. (McKim and Benoit 1971); 8. (McKim and Benoit 1974); 9. (Sauter *et al*. 1976); 10. (Leland and Carter 1985), 11. (Leland *et al*. 1989); 12. (Clements *et al*. 1989); 13. (Reed-Judkins *et al*. 1997); 14. (Besser *et al*. 2009); 15. (Besser *et al*. 2007); 16. (Cusimano *et al*. 1986)

2.4.4.2. Habitat Effects of Copper Criteria

Toxicity to Food Organisms. Copper is highly toxic to many freshwater invertebrates (Kiffney and Clements 2002; Mebane 2002a). 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) list relatively high LC_{50}s, which would apparently indicate that the proposed criteria are usually protective of invertebrates that juvenile salmon and steelhead feed on. However, compilations of short-term LC_{50}s tend to do a poor job of reflecting the sensitivities of metals to invertebrates in field conditions. The compilations indicate that stream invertebrates are not very sensitive to metals, but effects observed in field surveys tend to indicate that stream invertebrates are very sensitive to copper stress (Buchwalter *et al*. 2007). For these reasons, we consider field surveys more relevant indicators of metals effects than acute toxicity testing.

At concentrations less than or near the Idaho chronic criterion, elevated copper in water can adversely affect invertebrate communities that salmonids rely on for food (Table 2.4.4.1; Figure 2.4.4.3). Invertebrate communities in rivers also may be sensitive to elevated copper levels in the sediments. Most commonly, the reported effects to the invertebrate community are changed composition to pollution-tolerant taxa, rather than by reducing overall abundance (Canfield *et al*. 1994; Clements and Kiffney 1994; Beltman *et al*. 1999; Mebane 2002a). However, this might reflect sampling bias, because most invertebrate surveys reviewed were made in the summer. When invertebrates were collected in spring and autumn 1992 in Panther Creek, Idaho, a salmon stream contaminated by copper well in excess of the Idaho chronic criteria, total biomass was

much lower in the copper-influenced areas. A possible explanation for seasonally low biomass is that when the diversity was lower, and then a dominant, pollution tolerant insect taxa hatched and left the stream, the remaining biomass was lower than in unaffected areas with more diverse communities (Mebane 1994). Seasonal differences in copper effects have also been observed in invertebrates in pond communities, where effects of copper were more severe in cold, springtime conditions (6°C to 9.5°C) than in warmer summer (23°C to 28°C) or fall (15°C to 9.5°C) conditions (Winner *et al*. 1990).

Panther Creek, Idaho, has been the subject of detailed analyses of benthic macroinvertebrate communities and copper (among many other analyses). It is emphasized because prior to becoming polluted by copper in the 1960s, Panther Creek supported major runs of Chinook salmon and steelhead. The loss of habitat in Panther Creek resulting from water quality degradation from the Blackbird Mine was specifically cited as a contributing factor leading to the decline of the Snake River spring/summer Chinook salmon species (NMFS 1991). Prior to the mid-1990s, measured copper concentrations in Panther Creek were always well in excess of proposed criteria, so associated biological effects are not directly relevant to the question of whether adverse effects would be expected at criteria concentrations. Since then, restoration efforts have led to pronounced reductions in copper contamination to the point that the Idaho chronic criterion is mostly met. Thus, recent conditions in Panther Creek field surveys are very relevant to the present review because it offers a real-world view of biological conditions in a stream with copper present at close to the criteria concentrations under review.

Metrics calculated for benthic macroinvertebrates from Panther Creek in September 2005 and 2006 are shown in relation to the mean Idaho copper chronic criterion exceedence factors (Figure 2.4.4.3). An exceedence factor is the measured copper concentration at a location divided by the criterion for that sampling effect. The exceedence factors were calculated from chemical sampling from March to September of the year shown. Three measures of the macroinvertebrate community that seemed particularly relevant to their role in the food web of listed salmonids were examined: (1) Stream macroinvertebrate index (SMI) scores; (2) mayfly abundance; and (3) the abundance of organisms that were considered vulnerable to predation by salmonids. The SMI is an additive index comprised of nine measures of community diversity, dominance, or presence of pollution sensitive or intolerant species. It was derived as a measure of similarity or dissimilarity of macroinvertebrates to minimally disturbed reference conditions in the different ecological regions of Idaho (Jessup and Gerritsen 2002). The SMI and its component metrics relates to overall biological condition of stream ecosystems.

Abundance of mayflies was considered separately because mayflies have repeatedly been found to be important in the diets of juvenile salmonids in streams (Sagar and Glova 1987, 1988; Mullan *et al*. 1992; Clements and Rees 1997; Rader 1997; White and Harvey 2007; Syrjänen *et al*. 2011). Because mayflies are often also sensitive to copper, their loss in a stream food web could require shifting to other food items that are less preferred by salmonids. The third metric, abundance of taxa that are vulnerable to predation by juvenile salmonids, is broader than just mayflies. This metric was derived by assigning all organisms collected in the stream samples to one of three broad functional groups (i.e., burrowing, armored, and vulnerable to predation) based on life history traits influencing availability to steelhead fry (Suttle *et al*. 2004).

The comparisons of these metrics with copper exceedence factors in Panther Creek shows that even when copper concentrations were generally lower than the Idaho chronic criteria, the concentration gradient was still correlated with effects on the macroinvertebrate community (Figure 2.4.4.3). If copper only adversely affected macroinvertebrate communities at concentrations above the criteria, no correlation would be expected between copper and the macroinvertebrate metrics across a gradient of sub-criterion levels. The macroinvertebrate-copper exceedence patterns varied between years. In 2005, increasing copper concentrations were correlated with declining SMI scores (Figure 2.4.4.3). In 2005, relations between copper exceedence factors and mayfly abundance or vulnerable prey abundance were weak or nonexistent. In 2006, the pattern was reversed (Figure 2.4.4.3).

Together these comparisons show that relatively low levels of copper apparently affect macroinvertebrate communities, but that relations are more complex than can fully be explained in these simple correlations. For example, the copper gradient in Panther Creek tended to increase upstream to downstream along with temperatures that increased as the elevation dropped. The temperature gradient did not explain the macroinvertebrate patterns as well as the copper gradient; still it is an example of why patterns in field studies may be "noiser" than field or laboratory experiments. The changes in the stream macroinvertebrate communities did not obviously extend to adverse effects to the salmonid fishes, which are of most interest in this evaluation. There were no obvious decreases in various field measures of the salmonid populations at the sites with low-copper influence compared with upstream reference sites (e.g., overall abundances, age-class strength, condition factors of salmonids) (EcoMetrix 2006, 2007).

Sediments with elevated copper that were collected from Chinook salmon and steelhead habitat in Panther Creek, Idaho and tested in a laboratory setting with clean overlying water caused high mortality to *Hyalella azteca*, a freshwater benthic crustacean (Mebane 2002a). The resident benthic invertebrates collected from the same locations as the copper-contaminated sediments had reduced diversity compared to reference collections. Unlike the sediment toxicity tests, adverse effects to the instream invertebrates could not be attributed solely to either copper in the sediments or in water, because copper was elevated in both (Mebane 2002a). Elevated copper in sediments is also associated with elevated copper in benthic invertebrate tissues 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*. 2001b). Uptake and toxicity of copper by invertebrates is strongly influenced by the amount of acid-volatile sulfide in the sediments or by the amount of organic carbon in the sediments (Besser *et al*. 1995; Mebane 2002a).

In summary, habitat effects of elevated copper levels to listed salmon and steelhead include reductions in preferred invertebrate taxa that have been shown to influence the seasonal availability of food for juvenile salmonids. These reductions have been observed even with relatively low concentrations near the Idaho chronic criteria. Logically, reductions or changes in prey availability could translate to adverse effects on juvenile salmonid populations. However, in the Panther Creek field studies that we reviewed in some detail, no obvious extensions of macroinvertebrate effects to the salmonid fishes were observed. This suggests either or both that juvenile salmonids are able to switch prey when preferred prey are diminished, or that the food web effects were too subtle to tease out of the natural variability inherent in field monitoring studies without going to extraordinary means.

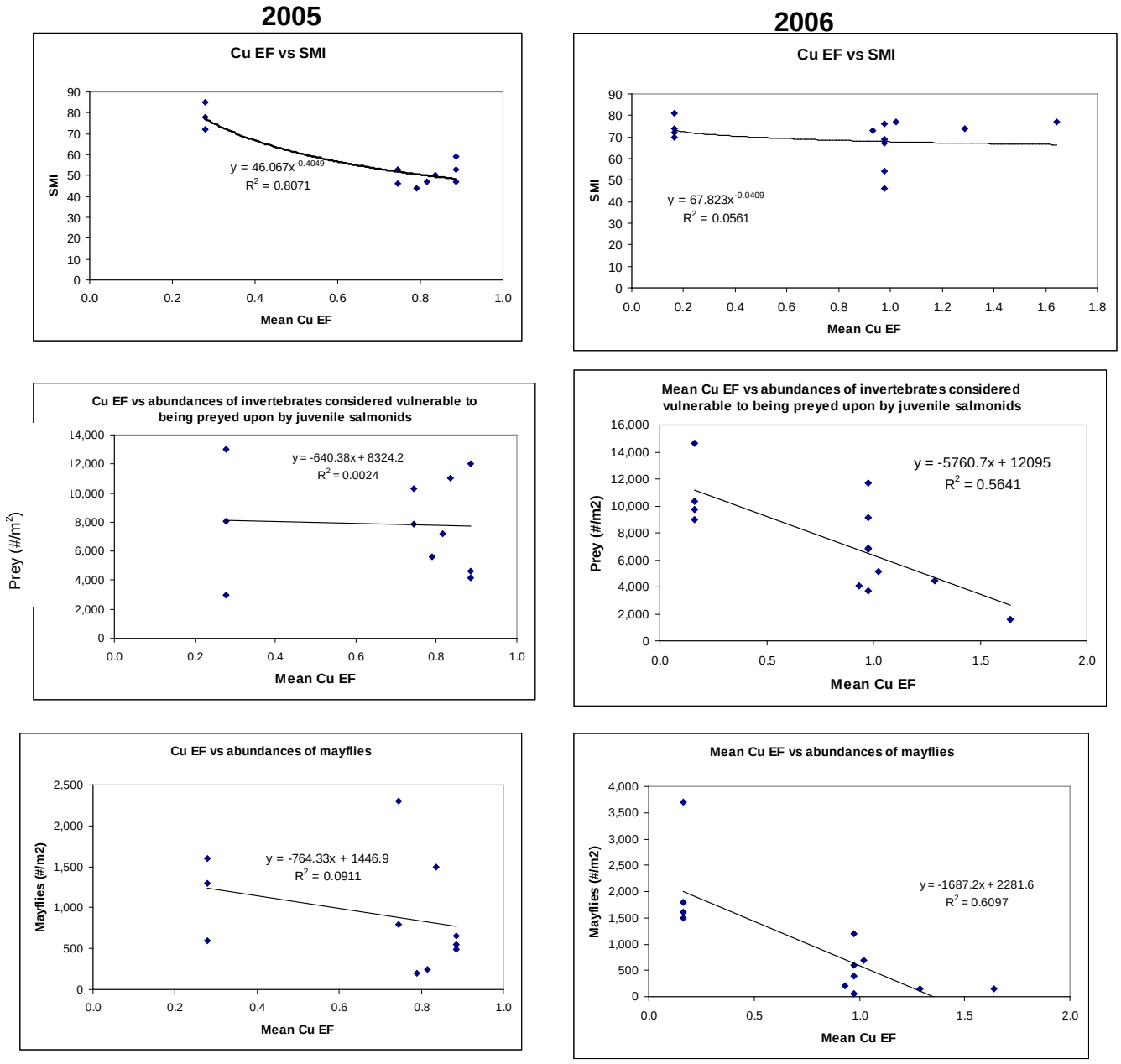


Figure 2.4.4.3. Correlations of relevant macroinvertebrate metrics with mean exceedence factors of the chronic criterion for stations monitored in Panther Creek, Idaho, September 2005 and 2006 (EcoMetrix 2006, 2007).

Bioaccumulation and dietary effects of copper. 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 (Sorensen 1991). 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 (Farag *et al*. 1994; Camusso and Balestrini 1995; Saiki *et al*. 1995; Sorensen 1991; Marr *et al*. 1996). The kidneys and gills are not thought to play a significant role in copper detoxification (Sorensen 1991). Both waterborne and dietary pathways have been associated with bioaccumulation in salmonids.

A series of dietary toxicity studies was conducted that involved feeding young rainbow trout diets prepared from invertebrates collected from the metals-contaminated Clark Fork River in Montana (Woodward *et al*. 1994; 1995; Farag *et al*. 1994). Results of these studies showed that fish fed a diet of pellets prepared from metal enriched invertebrates had reduced growth and physiological abnormalities relative to fish fed similar diets prepared from invertebrates from reference areas or less contaminated portions of the Clark Fork River. The Clark Fork watershed is enriched with several metals, though copper was generally considered to be the metal of greatest concern, and the adverse effects described in these articles were attributed to copper. However, a subsequent feeding study with invertebrates exposed to Clark Fork sediments in a controlled setting again produced adverse effects in rainbow trout but found that the effects were correlated with arsenic but not with copper (Hansen *et al*. 2004). Similar testing with experimentally exposed invertebrates under controlled conditions to single-metal sediment formulations, rather than field-contaminated sediments, also found no adverse effects of dietary copper exposure, but did find reduced growth and survival with the fish exposed to dietary arsenic, at comparable concentrations that had been measured in invertebrate diets from the previous studies with field-collected invertebrates (Erickson *et al*. 2010).

In a substantive review of the issue, Schlekat and others (2005, p. 141) observed that "We found no studies that demonstrate adverse effects resulting from diet-borne metals in systems in which water quality criteria were apparently being met. However, this could be a reflection of poorly designed approaches or a lack of appropriate data rather than an indication that such effects are not possible." [Note: "metals" in this quotation refers to cadmium, copper, lead and zinc; mercury, metalloids such as arsenic, and non-metal inorganics such as selenium were not addressed]. Other studies have reached similar conclusions (Mount *et al*. 1994; Dethloff and Bailey 1998; Taylor *et al*. 2000).

Thus while bioaccumulation of copper could result from dietary exposure near the Idaho chronic criterion concentration, the available information indicates that no appreciable adverse effects from dietary exposure to copper will occur at close to criteria concentrations.

2.4.4.3. Summary for Copper

The results of this analysis suggest that concentrations below the proposed acute and chronic criteria for copper can cause acute and chronic toxicity to salmon and steelhead. At the lower

range of hardness values encountered in Idaho streams and lakes the acute standard could result in injury and death.

Listed salmon and steelhead can experience a variety of adverse effects at or below the chronic Idaho copper criterion. These include:

- Deprivation of chemosensory function which in turn causes maladaptive behaviors including the loss of ability to avoid copper, and the loss of ability to detect chemical alarm signals. Appreciable adverse effects can be expected with increases as small as 0.6 µg/L above background concentrations.

- Reduced growth in juvenile Chinook salmon and rainbow trout under conditions of low hardness and low organic carbon.

- Because survival of juvenile salmon and steelhead in their migration to sea is strongly size-dependent, small reductions in size will result in disproportionately larger reductions in survival during migration to sea. Using population modeling, growth reductions at the chronic copper criterion were projected to result in slight increases in extinction risk and pronounced delays in recovery time in a model Chinook salmon population.

- The diversity and abundance of the macroinvertebrate food base for rearing juvenile salmon and steelhead could be reduced at copper concentrations near or below the Idaho chronic criterion.

While a variety of adverse effects relevant to listed salmonids have been demonstrated at copper concentrations less than the copper criteria under consultation, the most important issue is that the hardness-toxicity equation embedded into the criteria commonly results in fundamentally inaccurate and misleading indications of risk in critical habitats. This is because the best available science indicates that organic carbon is a more important mediator of copper risks than water hardness. During late summer or fall base flow conditions, copper would be expected to be most toxic because organic carbon tends to be low. Yet this is the time of year that hardness tends to be highest, and the hardness-based copper criteria wrongly indicate that copper would be of least risk at this time of year (Appendix C).

2.4.5 The Effects of EPA Approval of the Cyanide Criteria

The cyanide group (CN) includes free cyanide (HCN and CN^-), simple cyanide salts, (e.g. KCN, NaCN), metal-cyanide complexes, and in some organic compounds. The most bioavailable and toxic forms are free cyanide (Gensemer *et al*. 2007). The EPA's (1985e) criteria considered cyanide toxicity to mostly result from HCN but because the cyanide ion CN^- readily converts to HCN at pH values that commonly exist in surface waters, cyanide criteria were stated in terms of free cyanide expressed as CN. Free cyanide is extremely toxic and fast acting, and its fast action was one reason for EPA's (1992) expression of acute criteria based on 1-hour average concentrations. The EPA recommends measuring free cyanide at the lowest occurring pH and also measuring total cyanide during the monitoring of freshwater systems. In cases where total

cyanide concentrations are significantly greater than free cyanide concentrations, EPA recommends evaluating the potential for dissociation of metallocyanide compounds (EPA 1985e).

The criteria being analyzed for cyanide are 22 µg/L for acute exposure and 5.2 µg/L for chronic exposure. A difference between Idaho's cyanide criteria, which is being evaluated in this Opinion, and the cyanide criteria as originally developed by EPA and initially promulgated for Idaho by EPA (1992) is that Idaho's cyanide criteria are defined as Weak Acid Dissociable (WAD) cyanide (EPA 2000a). While not explicitly explained, this definition is probably used because direct measurement of free cyanide was not routinely offered by many environmental test laboratories until fairly recently, and as result a criteria based on free cyanide would be difficult to analytically measure and implement. Interpreting the criteria as total cyanide would include iron-cyanide and other metal-cyanide complexes that are considerably less reactive and toxic than free cyanide. Weak acid dissociable cyanide analyses were a compromise between free and total cyanide measurements and WAD cyanide includes metal-cyanide complexes such as zinc-, nickel-, copper-, and cadmium-cyanide easily dissociate under weakly acidic conditions (pH 5-6).

The relevance of these cyanide definition and analytical testing issues for the present Opinion is that for a given environmental sample collected from an effluent or stream that contains cyanides, analyzing the sample for WAD cyanide would produce a higher value than if it could be analyzed for free cyanide. Likewise, using free cyanide concentrations from a toxicity test cited in this Opinion is more protective than using a WAD cyanide concentration. This adds a degree of conservatism to the present analyses, although the magnitude of this it cannot be quantified because the degree of difference between WAD and free cyanide would depend on the sample.

Temperature and cyanide toxicity. Whereas with metals, water hardness or DOC are often important modifiers of toxicity, with cyanide, temperature has a strong influence on toxicity. A number of tests with different species indicated a marked positive correlation between resistance to HCN and temperature rather than the negative one that might be expected from general stress models. This increased toxicity at lower temperatures has been observed with rainbow trout, brook trout, yellow perch, fathead minnows, and bluegills (Smith *et al*. 1978; Kovacs and Leduc 1982b, 1982a). The most robust dataset was probably from Kovacs and Leduc (1982a) from which a temperature-toxicity relationship for rainbow trout can be estimated as: LC_{50} = (T°C)*3.167+6, $r^2 = 0.97$

When a water quality characteristic such as temperature is apparently related to the toxicity of a substance, the EPA Guidelines (Stephan *et al*. 1985) for developing aquatic life criteria provide two approaches: (1) Direct incorporation of the characteristic into the criteria; or (2) data acceptability. In Approach 1, "*if the acute toxicity of the material to aquatic animals apparently has been shown to be related to a water quality characteristic such as hardness or particulate matter for freshwater animals or salinity or particulate matter for saltwater animals, a Final Acute Equation should be derived based on that water quality characteristic*." (Stephan *et al*. 1985). Examples of this include criteria for ammonia which are based on temperature and pH (EPA 1999a), and most metals criteria that are based on hardness, or EPA's 2007 copper criteria,

based upon multiple water quality characteristics. In Approach 2, "*results of acute tests conducted in unusual dilution water. e.g., dilution water in which total organic carbon [TOC] or particulate matter exceeded 5 mg/L, should not be used [in a criterion dataset], unless a relationship is developed between acute toxicity and organic carbon or particulate matter or unless data show that organic carbon, particulate matter, etc., do not affect toxicity*." (Stephan *et al*. 1985).

While test waters warmer than 6°C could hardly be considered "unusual" (or waters with particulates or >5 mg/L TOC, for that matter), temperature clearly affects the toxicity of cyanide, and the Guidelines are clear that such characteristics should be incorporated in criteria. Why that was not done in the case of cyanide is unexplained in the criteria document.

In cold-temperate climates such as the Idaho action area, it follows that if the cyanide criteria were not adjusted for temperature, only the coldest test results (6°C) should be used. For example, fall-spawning Chinook salmon progeny in the Snake River usually emerge from gravels at water temperatures of about 5.5 to 9°C (Connor *et al*. 2002). If data were available on the effects of cyanide at temperatures of 6°C, 12°C, and 15°C, on the incubation and hatching of eggs from a salmonid with a fall-spawning life history only data from the 6°C exposure would be relied upon. Similarly, since juvenile salmonids from either fall- or spring-spawning species can be expected to be exposed to near-freezing temperatures for long periods (Figure 2.4.5.1), only the LC_{50}s obtained from the coldest tests would be used in a final assessment. For the cyanide data set, these would be the tests conducted around 6°C or below.

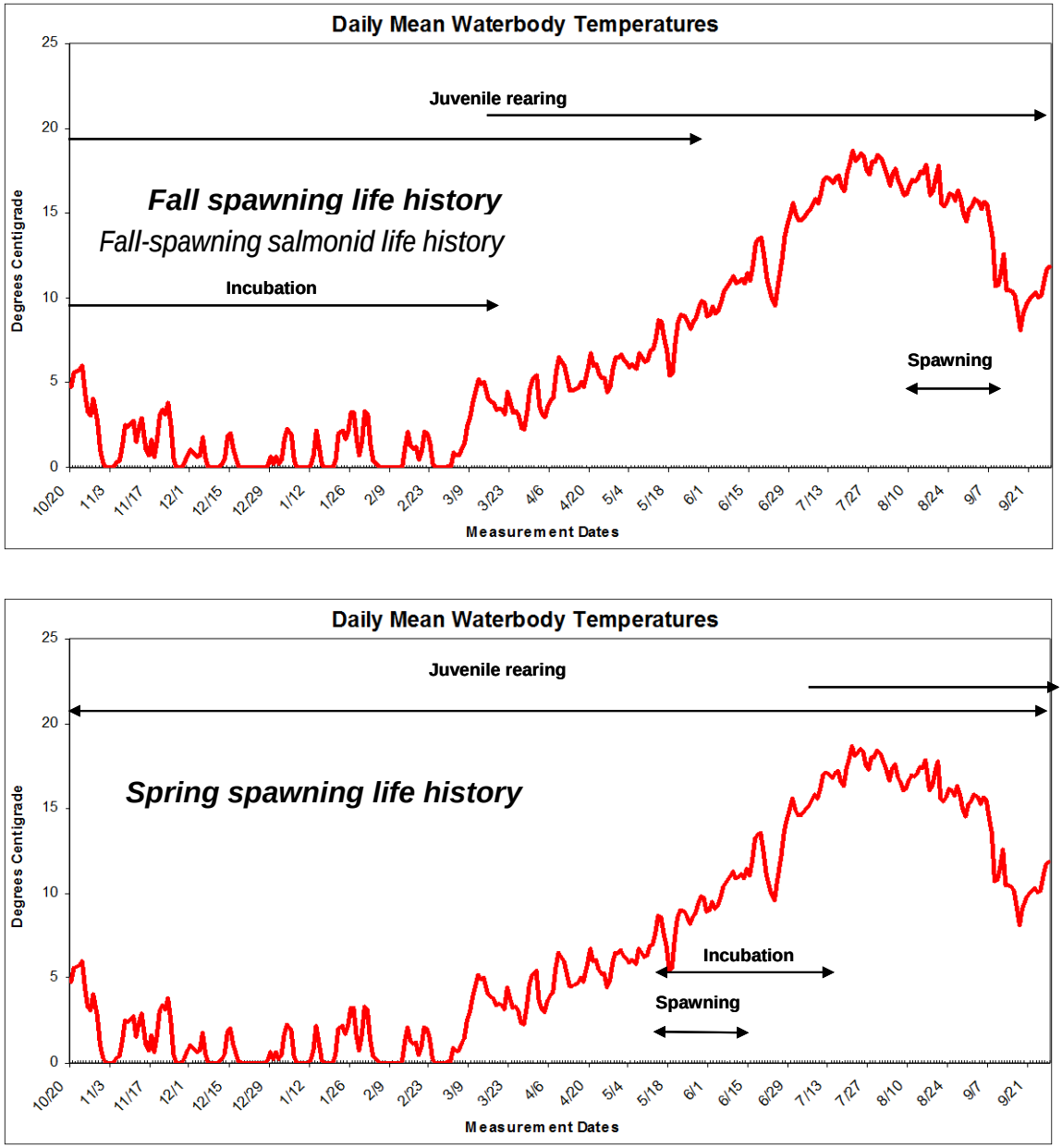


Figure 2.4.5.1. Examples of the occurrence of different salmonid life stages and annual temperature patterns for a coldwater stream: salmonid species with (top) fall-spawning life histories (e.g., Chinook salmon, coho salmon, Atlantic salmon, brown trout, bull trout, brook trout), and (bottom) spring-spawning life histories (e.g., steelhead, rainbow trout, cutthroat trout, most non-salmonid fishes). Temperature data from the Salmon River, Idaho near Sunbeam, Idaho; data from Idaho Department of Environmental Quality, 2002 water year.

Table 2.4.5.1. Contrasting effects of cyanide on salmonids at different temperatures. For lethal effects data, if LC_{50}s are greater than the Final Acute Value of 44 µg/L that is assumed to indicate lack of harm at acute criteria concentrations; for sublethal effects, lowest effects concentrations should be greater than 5.2 µg/L.

Species	Effect	Exposure duration	T (°C)	Effect statistic	Effect concen-tration (µg/L)	Source/ Notes
	Lethal effects					
Rainbow trout	Killed	4 d	6	LC_{50}	27	(Kovacs and Leduc 1982a)
"	Killed	4 d	12	LC_{50}	40	(Kovacs and Leduc 1982a)
"	Killed	4 d	18	LC_{50}	65	(Kovacs and Leduc 1982a)
Rainbow trout	Killed	4-d	10	LC_{50}	57	(Smith *et al*. 1978)
	Sublethal effects					
Rainbow trout	Reduced swimming performance	20 d	6	No effect threshold	<4.8	(Kovacs and Leduc 1982b)
"	Reduced swimming performance	20 d	12	No effect threshold	<9.6	(Kovacs and Leduc 1982b)
"	Reduced swimming performance	20 d	18	No effect threshold	43	(Kovacs and Leduc 1982b)
"	Reduced swimming performance			No effect threshold	<10	(a)
"	Reduced growth	20 d	6	No effect threshold	<4.8	(Kovacs and Leduc 1982b)
"	Reduced growth	20 d	12	No effect threshold	<9.6	(Kovacs and Leduc 1982b)
"	Reduced growth	20 d	18	No effect threshold	24	(Kovacs and Leduc 1982b)
"	Reduced growth in fish forced to exercise	20d	10	LOEC	9.6	(b)
Brook trout	Reduced egg production			18% reduction in spawned eggs/female	5.6	(Koenst *et al*. 1977)
Atlantic salmon	Abnormal embryo and larval development			LOEC	9.6	(Leduc 1978)

(a) EPA 1985e, citing Broderius 1970; (b) EPA 1985e, citing McCracken and Leduc 1980

2.4.5.1. Species Effects of Cyanide Criteria

Acute Cyanide Criterion. The acute criterion under review is 22 µg/L, which is the FAV divided by two. Because the FAV was derived from LC_{50} data, and available acute data for cyanide are LC_{50}s, and a concentration killing 50% of the test population obviously cannot be used directly to judge the protectiveness of the acute criteria. Thus the LC_{50}s are compared to the FAV rather than the acute criterion. Following the assumption that dividing a LC_{50} by two will likely kill few if any fish (Section 2.4.1.6), it also follows that LC_{50}s need to be higher than the FAV (44 µg/L) in order to assume that little mortality would result at the acute criterion. This turns out not to be the case when temperatures were 12°C or less (Table 2.4.5.1), indicating that the acute criterion cannot be considered fully protective under these conditions.

Billard and Roubaud (1985) determined that sperm of rainbow trout had lower fertilization success when they were exposed for 15 minutes directly to 1 µg/L cyanide in a sodium and potassium chloride buffered diluent that kept the sperm immobile. This concentration is below the chronic criterion. However, spermatozoa become motile when released into unbuffered, natural waters and only survive for a few minutes (Billard and Roubaud 1985; Farag *et al*. 2006). Thus effects demonstrated by Billard and Roubaud (1985) may not relate to natural waters.

Chronic Cyanide Criterion. The chronic cyanide criterion is 5.2 µg/L. Kovacs and Leduc (1982) observed chronic toxicity effects on growth in terms of average fat gain and dry weight when juvenile rainbow trout were exposed to 5 µg/L at 6°C. At 12°C, toxicity effects were determined at concentrations greater than or equal to 10 µg/L. As with acute toxicity, chronic effects were inversely related to water temperature in the study. All measures of growth were affected significantly at an exposure concentration of 15 µg/L at all temperatures tested (6°C to 18°C). The results of Kovacs and Leduc (1982) suggest there is potential for reduced growth at the proposed chronic criterion when temperatures are 6°C or lower.

Kovacs and Leduc (1982b) also found that after a 20-day exposure to sublethal cyanide the swimming ability of rainbow trout was reduced at all cyanide concentrations tested in the range of 5 µg/L to 45 µg/L, with the effect increasing at lower temperatures. Although cyanide-exposed fish had returned to normal or near normal growth rates, their swimming impairment suggests biochemical disturbance and perhaps tissue damage as observed by Dixon and Leduc (1981).

Kovacs and Leduc (1982b) noted that at low water temperatures (4°C to 5°C), under conditions where metabolism is depressed, fish are under some stress to maintain their life processes. This is evidenced by a greater water content of fish, less food availability in nature, greater specific dynamic action, assimilation, and food conversion efficiency. Under such conditions, another stressor such as cyanide would have a serious effect on fish production and even on long-term survival. Their study indicated that at 6°C, a concentration as low as 5 µg/L HCN can cause marked reduction in fat synthesis and swimming performance. In Idaho waters, low water temperatures prevail for much of the year. Therefore, for a more realistic appraisal of our water pollution problems, toxicity to fish at low temperatures needs to be evaluated.

We did not locate any tests for reproductive impairments with exposures of listed species or very close surrogates (e.g., other genus *Oncorhynchus* tests) for this analysis. Tests with bluegill and brook trout suggest that fish reproduction can be severely inhibited at concentrations close to the chronic criterion. Kimball *et al*. (1977) tested the effects of long-term cyanide exposure on bluegills and found severe adverse effects at the lowest concentration tested, which was the same as the chronic criterion concentration of 5.2 µg/L. They noted at p. 345 that "*Spawning is completely inhibited at 5.2 µg/L HCN and presumably, is inhibited to some extent at lower level.*"

2.4.5.2. Habitat Effects of Cyanide Criteria

Toxicity to Food Organisms. Although cyanide toxicity varies extensively among invertebrate taxa, available data for the types of aquatic insects and crustaceans that juvenile salmonids feed on indicate that they are similarly or less sensitive to cyanide compared with listed salmon and steelhead (EPA 1980e, 1985e; Eisler 1991). Aquatic invertebrates do not appear to be adversely affected by concentrations that are protective of fish. As documented below, cyanide does not appear to bioaccumulate because of its short-lived nature and the ability of aquatic organisms to depurate the compound. The proposed criteria are likely to be protective of the food sources of listed salmon and steelhead.

Bioaccumulation. There is no evidence of significant bioaccumulation of cyanide in fish at levels below the proposed chronic criterion because the compound is easily metabolized (EPA 1985e). Lanno and Dixon (1996) determined that bioconcentration occurred in juvenile rainbow trout exposed to a cyanide level (8 µg/L) which is close to the chronic criterion, but did not observe any significant toxic effects. Other evidence exists that cyanide levels are elevated in fish tissues when subjected to long-term chronic exposure, but cyanide depuration occurs relatively quickly when fish move to clean water (Eisler 1991; Lanno and Dixon 1996). Therefore, potentially adverse effects related to cyanide bioaccumulation are unlikely to be observed in listed salmon and steelhead.

Water Chemistry. Cyanide in the water column at the proposed acute and chronic criteria concentrations during the colder seasons will result in the water quality being unsuitable for listed salmonids as described above in the temperature and cyanide toxicity section.

2.4.5.3. Summary for Cyanide

The proposed acute and chronic criteria can expose listed salmonids to harmful cyanide concentrations under specific situations. The acute criterion cannot be considered to be reliably protective when water temperatures drop to about 6°C or lower. Further, Leduc (1984) found that cyanide concentrations at the chronic criterion in water colder than 6°C may be associated with chronic toxicity effects. Temperatures in streams within the action area routinely drop below 6°C.

2.4.6. The Effects of EPA Approval of the Mercury Criteria

Mercury is hazardous to fish because of its strong tendency to bioaccumulate in muscle tissue and because it is a potent neurotoxin that causes neurological damage which in turn leads to behavioral effects which in turn lead to reduced growth and reproductive effects (Wiener *et al*. 2003; Weis 2009; Sandheinrich and Wiener 2010; Kidd and Batchelar 2011). Methylmercury is a highly neurotoxic form that readily crosses biological membranes, can be rapidly bioaccumulated through the water, and is taken up primarily through the diet (which accounts for more than 90% of the total amount of methylmercury accumulated by fish). Both organic and inorganic mercury bioaccumulate, but methylmercury accumulates at greater rates than inorganic

mercury. Methylmercury is more efficiently absorbed, and preferentially retained than inorganic mercury (Scheuhammer 1987, Wiener 1995). Methylmercury is biomagnified between trophic levels in aquatic systems and in general proportion to its supply in water (Wattras and Bloom 1992). In fish tissue accumulated mercury consists almost entirely of methylmercury (Bloom 1992; Hammerschmidt *et al*. 1999; Harris *et al*. 2003). Toxicity of methylmercury is therefore particularly important with respect to effects to higher trophic level fish and other organisms (Sorensen 1991; Nichols *et al*. 1999).

Inorganic mercury is absorbed less readily and is eliminated more rapidly than methylmercury. In fact, intestinal absorption of inorganic mercury is limited to a few percent of methylmercury, for which absorption is nearly complete (Scheuhammer 1987; Wiener *et al*. 2003). Inorganic mercury appears to have the greatest effect upon the kidneys, while methylmercury is a potent embryo and nervous system toxicant. Methylmercury readily penetrates the blood brain barrier, produces brain lesions, spinal cord degeneration, and central nervous system dysfunctions. Long-term dietary exposure to mercury has been shown to cause instability, inability to feed, and diminished responsiveness. The central nervous system is the site of the most extensive damage due to mercury exposure.

2.4.6.1. Species Effects of Mercury Criteria

The acute and chronic criteria for dissolved mercury under consultation are 2.1 µg/L and 0.012 µg/L (12 ng/L), respectively (EPA 1985g). The EPA has also developed a human health criterion, in which fish tissue concentrations are not to exceed 0.3 mg/kg ww (66 FR 1344; EPA 2001). This standard was adopted in Idaho in 2005 and is applicable to all designated critical habitats and waters inhabited by listed salmon or steelhead (IDEQ 2005).

Acute Mercury Criterion. The acute mercury criterion is about 175 times higher than the chronic criterion and about 1,000 times higher than typical ambient concentrations (Table 2.4.6.2). All criteria applications contemplated under the Idaho standards (cleanup actions and discharge limits) would also involve application of the chronic criterion. As a practical matter the acute criterion would never be relevant for determining discharge limits to any receiving water since it is hydrologically inconceivable that the critical flows used by EPA with the acute criteria for calculating short-term maximum discharge limits (lowest 1-day average flows in a 10-year period, abbreviated as a 1Q10) would be anywhere close to 175 times lower than the critical flows used for calculating long-term average discharge limits (lowest 7-day average flows occurring in a 10-year period 7Q10). An example is given later in this Opinion in Appendix D, where the question of implementing criteria through limiting effluent volumes is treated in more detail. For Thompson Creek, the 7Q10 is 2.1 cfs which very close to the 1Q10 of 2.05 cfs. Thus the 1Q10 is 1.02 times lower than the 7Q10. The possibility that the 1Q10 and the 7Q10 could differ by 175 is discountable. Nevertheless, even though the acute mercury criterion is unlikely to be applied as a practical matter, the following analysis summarizes the available acute toxicological information for mercury.

Most available data suggest that listed salmon and steelhead are not susceptible to acute toxicity from direct exposure to mercury in water water at concentrations approaching the 2.1 µg/L acute

criterion (Kidd and Batchelar 2011). Many "acute" type of studies NMFS reviewed exposed fish to mercury in water for much longer than the 4 days typical of "acute" exposures. The EPA (1985g) reported LC_{50} values for salmonids exposed to inorganic mercury that ranged between 155 µg/L and 420 µg/L. For organic mercury, reported LC_{50}s ranged from 5 µg/L to 84 µg/L, depending on the chemical form, with a phenylmercuric compound (LC_{50} = 5 µg/L) being the most toxic. Buhl and Hamilton (1991) exposed coho salmon and rainbow trout alevins and parr to mercuric chloride, and determined average LC_{50}s that ranged between 193 µg/L and 282 µg/L. Devlin and Mottet (1992) determined a methylmercury LC_{50} equal to 54 µg/L for coho salmon embryos exposed for 48 days. Niimi and Kissoon (1994) exposed rainbow trout sub-adults to 64 µg/L of mercuric chloride until the fish died. The average time to death was 58 days. In another exposure to 4 µg/L of methylmercury chloride, they determined that the fish lived more than 100 days. The lowest effect level noted from an "acute" type study was an LC10 of 0.9 µg/L following a 28-day exposures of rainbow trout embryo's to mercury, with a no-effect (LC1) estimated of 0.2 µg/L (Birge *et al.* 1980)

Available information on sublethal effects from direct acute exposure is sparse. Rainbow trout were attracted to 0.2 µg/L mercuric chloride in 80 minute exposures, which is about a factor of 10 lower than the acute criterion (Black and Birge 1980).

The reported LC_{50}s for life stages beyond the embryo are well above the acute criterion. The results of these studies suggest collectively that the proposed acute mercury criterion is unlikely to cause mortality. Behavioral alterations at a concentration 10 times lower than the acute criterion were reported, but even that concentration is ~20 times higher than the chronic criterion.

Chronic Mercury Criterion. The EPA's 1984 chronic aquatic life criterion for mercury is something of a misnomer, since its establishment had nothing to do with the chronic effects of mercury on aquatic life. Rather, the criterion was intended to protect the "fishable" uses of aquatic life which in this case is to avoid allowing bioaccumulation in fish at mercury levels that would impair marketability of fish. The chronic criterion was established with the objective of avoiding fish from bioaccumulating mercury to concentrations that were predicted to exceed the Food and Drug Administration's (FDA) (1984) action level of 1 mg/kg fresh weight for the sale of commercially caught fish. "Fresh weight" is synonymous with wet weight, ww, which is more commonly used in the ecotoxicology literature. All tissue residue values for mercury are given as ww unless otherwise indicated.

The marketability approach of setting chronic criterion for mercury replaced EPA's (1980j) approach which was similar to that used for other substances. The EPA (1980j) followed an extrapolated species-sensitivity distribution to obtain a Final Acute Value of 0.0017 µg/L (1.7 ng/L), which was divided by an ACR of 3.0 to obtain a freshwater final chronic value of 0.00057 µg/L (0.57 ng/L).

The physiological effects of direct exposure to mercury at ambient concentrations near the chronic criterion are the result of dietary bioaccumulation. This is due to the strong tendency of mercury to bioaccumulate, discussed further in the next section. In the environment virtually all mercury exposure to fish is from dietary sources, so concentrations in water are not meaningful for direct water-only exposures (Wiener and Spry 1996; Wiener *et al*. 2003). Literature from water borne exposures may be useful; however, in instances where waterborne exposures were used as a means to achieve tissue burdens. However, in these instances the relevant media to evaluate is the tissue burden, not the water concentrations.

Wiener and Spry (1996) noted that water-borne concentrations in natural streams are unlikely to be high enough to result in direct toxicity effects. In a broad survey of mercury in freshwater systems in California and other areas including the lower Columbia River, Gill and Bruland (1990) failed to locate any water bodies containing levels of mercury above or approaching the dissolved criterion although many of these same water bodies were mercury impaired due to elevated concentrations in fish. Similar findings have been reported from other areas (Becker and Bigham 1995; Watras *et al*. 1998; Castro *et al*. 2002; Hope and Rubin 2005; Wiener *et al*. 2006; IDEQ 2007b; Chasar *et al*. 2009; Essig 2010).

Sublethal effects of the proposed chronic criterion may occur from long-term exposure in the natural environment effects, since ambient water mercury concentrations that are near or below the proposed chronic criterion have been associated with bioaccumulation (see below). For example, Davis Creek Reservoir in California is highly contaminated by mercury and has dissolved organo-mercury concentrations around 2.4 ng/L and total dissolved mercury concentrations around 12 ng/L These concentrations of mercury in water are similar in magnitude to the proposed chronic criterion, and were associated with fish tissue concentrations of 2.5 mg/kg ww (Gill and Bruland 1990) that were almost 10 times higher than apparently safe the tissue concentrations of 0.2 to 0.3 mg.kg ww that appear to be safe for fish (later in this section).

Hence, available information suggests that listed salmon and steelhead are unlikely to be killed outright by direct exposure to water concentrations equal to the proposed chronic criterion. However, in all reports from field situations reviewed, effects of direct exposure are likely to be overshadowed by effects from bioaccumulation.

2.4.6.2. Habitat Effects of Mercury Criteria

Toxicity to Food Organisms. Little information was located indicating appreciable risk of adverse effects to invertebrates prey items themselves. Rather, the most significant concern from the perspective of listed salmon and steelhead is bioaccumulation from eating aquatic invertebrates that themselves have elevated mercury levels, not changes in aquatic invertebrate production due to mercury toxicity.

Bioaccumulation. Food chain transfer is by far the most important exposure pathway in aquatic ecosystems (Hall *et al*. 1997; Wiener *et al*. 2003). Aquatic systems have complex food webs including several trophic levels, and primary producers in aquatic systems may themselves

accumulate more mercury from water and sediment than their soil-based counterparts in terrestrial systems. Rates of bacterial methylmercury production in water and sediment ultimately determines the potential of an aquatic system to develop a mercury bioaccumulation problem (EPA 1997b). Aquatic predators including salmonids are most susceptible to bioaccumulating mercury, and thus their tissue concentrations may best reflect the amount of mercury available to aquatic organisms in the environment. For example, in comparisons of bottom feeding fish with fish that feed on plankton, invertebrates, and vertebrates, Wren and MacCrimmon (1986) determined that the greatest mercury concentrations were found in piscivorous fish species and that mercury content increased with higher trophic levels.

Fish store most mercury as methylmercury in their muscle, even when they are exposed to inorganic mercury. Methylmercury both bioconcentrates and biomagnifies across trophic levels, and corresponding, field-measured bioaccumulation factor (BAFs) can be in the millions for top trophic level fish (Nichols *et al*. 1999). Methylmercury accumulates at greater rates than inorganic mercury because it is more efficiently absorbed and is preferentially retained (Scheuhammer 1987; Wiener 1995).

Rates of bioaccumulation are thought to be affected by numerous factors such as the number of trophic levels present, food web structure of the aquatic ecosystem, abundance of sulfur reducing bacteria and concentration of sulfates, amount of dissolved oxygen, water temperature, organic carbon availability, pH, the nature of the mercury source, and other parameters (Porcella *et al*. 1995). The uptake of mercury and methylmercury in fish increases with ambient water concentration, water temperature, size and age of the fish, breeding status, and food ingestion rate. Decreases in pH have also been correlated with increasing methylmercury uptake (Wren and MacCrimmon 1986; Ponce and Bloom 1991).

Diet is the primary route of methylmercury uptake by fish in natural waters, and contributes more than 90% of the amount accumulated. The assimilation efficiency for uptake of dietary methylmercury in fish is probably 65% to 80% or greater. To a lesser extent, fish obtain mercury from water passed over the gills, and fish also methylate inorganic mercury in the gut (Wiener and Spry 1996).

Sediments are an important reservoir for mercury in freshwater systems. Mercury in sediments can become available for food chain transfer, and instances of elevated mercury in sediment corresponding with elevated mercury in fish have been documented (Maret 1995; Clark and Maret 1998; Suchanek *et al*. 2008; Scudder *et al*. 2009; EPA 2011). Mercury may accumulate in bed sediments to levels that greatly exceed levels associated with probable adverse effects to benthic communities even when mercury in surface water was far lower than the chronic criterion of 12 ng/L. One well documented instance was from Onondaga Lake, New York, where dissolved mercury in the epilimnion was about 1 ng/L and mercury in the hypolimnium was up to 10 ng/L (Bloom and Effler 1990). Mercury in sediments were always above 1 mg/kg dw, often above 5 mg/kg dw, and exceeded 25 mg/kg dw in some samples. Mercury in sediments was strongly correlated with mercury in invertebrate tissues (Becker and Bigham 1995). In addition to the role of mercury bound in sediment as an entry point to trophic pathways, direct adverse alterations to benthic communities are probable when mercury in sediment exceeds 1 mg/kg dw (MacDonald *et al*. 2000a).

Toxicity of dietary mercury to fish. Concentrations of mercury that would be expected to elicit ecologically significant adverse effects in fish when ingested as prey were estimated by DePew *et al*. (2012). They concluded that chronic dietary exposure to low concentrations of MeHg may have significant adverse effects on natural-origin fish populations. Adverse effects on behavior resulting from dietary concentrations of mercury usually occurred above 0.5 mg/kg ww. However, adverse effects on reproduction occurred with dietary concentrations of mercury at 0.2 mg/kg ww or lower. DePew *et al*. (2012) noted that although their thresholds were intentionally conservative, they still may underestimate the magnitude of effects experienced by natural-origin fish because their thresholds were derived from laboratory tests conducted under favorable conditions. In the wild, additional environmental stressors related to foraging, predation, temperature fluctuation, and other potentially toxic contaminants are present (Depew *et al*. 2012).

Mercury tissue residues in fish associated with the presence or absence of adverse effects. While the risks of mercury neurotoxicity to humans from eating fish has been the subject of much concern and research, and effects on fish-eating wildlife have been reasonably well documented, there is yet considerable uncertainty regarding effects of mercury on fish themselves. Scheuhammer *et al*. (2007) summarized the state of the knowledge succinctly: "*Compared with humans and mammalian and avian wildlife, relatively little is known of the toxicological significance to fish of environmentally realistic exposures to methylmercury.*" Ranges of estimates of "safe" tissue residues of mercury in various fish tissues are greater than an order of magnitude. Fish can survive in laboratory environments with mercury concentrations in tissues elevated far above those encountered in the wild. In the brain, concentrations of 7 mg/kg ww or greater probably eventually kill fish in laboratory environments, and for mercury sensitive species, brain-tissue concentrations of 3 mg/kg ww or greater probably indicate significant toxic effects. For axial muscle tissue, concentrations of 6 to 20 mg/kg ww have been associated with toxicity in laboratory studies (Wiener and Spry 1996). However, subsequent to the review of Wiener and Spry (1996), more ecologically relevant studies have been devised that have detected effects associated with endocrine disruption, neurotoxicity, and reproductive impairment (Table 2.4.6.1*)*

Given the high neurotoxicity of methylmercury, the exposure levels causing adverse behavioral effects are probably much lower than exposure levels causing overt toxicity. Many fish behaviors are sensitive and ecologically relevant indicators of contaminant toxicity, affected at lower levels than those causing direct mortality. The neurotoxic effects of exposure to sublethal concentrations of methylmercury can impair the ability of fish to locate, capture, and ingest prey and to avoid predators (Wiener *et al*. 2003). For example, Fjeld *et al*. (1998) showed that the feeding efficiency and competitive ability of grayling (*Thymallus thymallus*) exposed as eggs to waterborne methylmercury chloride for 10 days and having yolk-fry with mercury concentrations of 0.27 mg/kg ww or greater, were impaired when fish were tested 3 years later.

The NOEC from Fjeld *et al*'s (1998) study (0.09 mg/kg ww in embryos) would translate to a mean concentration in maternal muscle tissue of about 0.7 (range 0.15 to 1) mg/kg ww based on various ratios of mercury concentrations in eggs or maternal fillets in brook trout reported by McKim *et al*. (1976). Similar calculations by USFWS (2003) and IDEQ using relative mercury concentrations in different tissues of yellow perch or other data resulted in somewhat higher

extrapolations of the 0.09 embryo NOEC concentration to muscle tissue concentrations ranging from 0.45 to 1.8 mg/kg ww. Similarly, estimates of maternal muscle tissue concentrations that would produce an LOEC embryo residue of 0.27 mg/kg ww range from 1.35 to 5.4 mg/kg ww (Fjeld *et al*. 1998; USFWS 2003; IDEQ 2005). These tissue residue effect estimates are roughly similar (within a factor of two) to tissue effects in Atlantic salmon parr. In Atlantic salmon parr, methylmercury concentrations of 0.69 mg/kg ww were associated with brain lesions and behavioral alterations (Berntssen *et al*. 2003).

Mercury tissue residues associated with the presence or absence of adverse effects are summarized in Table 2.4.6.1. Generally, the most sensitive effects of long-term exposures of a variety of fish species to methylmercury have been reproductive or behavioral effects, with concentrations greater than about 0.3 mg/kg ww in whole bodies or axial muscle tissues likely to be harmful to fish (Table 2.4.6.1). However, adverse effects at concentrations lower than this range are possible. Cutthroat trout with whole-body mean mercury burdens of only about 0.05 mg/kg ww collected from a mountain lake had significant changes in metabolic, endocrine, and immune-related genes, compared to fish from lakes with lower mercury concentrations in trout (Moran *et al*. 2007). Possible steroidogenesis effects in white sturgeon collected from the lower Columbia River, as reduced androgen levels in the sperm, were suggested to correspond with a mean muscle mercury concentration of 0.2 mg/kg ww (Webb *et al*. 2006). These reports indicate that a true threshold for the absence of effects from mercury accumulation could be considerably lower than 0.3 mg/kg ww. However, to borrow a phrase from human health care, changes in gene expression or steroid concentrations may be considered "sub-clinical," that is, not of health significance unless further work relates sub-organismless effects such as these to some other organism-level effect such as altered behaviors, reduced growth, or impaired reproduction.

Another recent review reached fairly similar conclusions on what tissue burdens of mercury were unsafe. Sandheinrich and Wiener (2010) concluded that effects on biochemical processes, damage to cells and tissues, and reduced reproduction in fish have been documented at methylmercury concentrations of about 0.3 to 0.7 mg Hg/kg ww in the whole body and about 0.5 to 1.2 mg Hg/kg ww in axial muscle.

Table 2.4.6.1. Examples of mercury tissue residues co-occurring with the presence or absence of adverse effects.

Organism	Residue concentration (mg/kg wet weight, ww)	Tissue	Effect	Source
Edible fish	0.3	Muscle	Acceptable risk for most human consumption of recreationally caught fish (including subsistence), except for vulnerable populations	(EPA 2001b; IDEQ 2005)
Edible fish	1.0	Muscle	Acceptable risk for human consumption of commercially caught fish.	(FDA 1984)
Multiple fish species	0.2	Whole body	Tissue threshold-effect level based largely on sublethal endpoints (growth, reproduction, development, behavior), calculated to be protective of juvenile and adult fish.	(Beckvar *et al*. 2005)
Multiple fish species	0.2	Whole body	5.5% or 33% injury to juvenile or early life stage fish respectively, where injury is limited to endpoints such as survival, reproductive success, and lethal developmental abnormalities.	(Dillon *et al*. 2010)
Grayling, *Thymallus thymallus*	0.27	Yolk-fry	Impaired feeding efficiency and competitive ability 3 years after exposure as yolk-fry	(Fjeld *et al*. 1998)
Grayling, *Thymallus thymallus*	0.09	Yolk-fry	No-observed effects on feeding efficiency and competitive ability 3 years after exposure as yolk-fry	(Fjeld *et al*. 1998)
Grayling, *Thymallus thymallus*	2.2 (0.45 – 3)	Muscle	Estimated range of maternal muscle tissue concentrations that would result in yolk-fry concentrations of 0.27 mg/kg, using organ tissue ratios derived from McKim *et al*. (1976).	This review
Grayling, *Thymallus thymallus*	0.7 (0.15 to 1)	Muscle	Estimated range of maternal muscle tissue concentrations that would result in NOEC yolk-fry concentrations of 0.09mg/kg, using organ tissue ratios derived from McKim *et al*. (1976).	This review
Atlantic salmon	0.69	Brain	Brainstem lesions and behavioral alterations	(Berntssen *et al*. 2003)

Organism	Residue concentration (mg/kg wet weight, ww)	Tissue	Effect	Source
Atlantic salmon	0.61 (0.4 – 0.8)	Muscle	Estimated mean and range of muscle (fillet) concentrations resulting in brain residues of 0.69 mg/kg that caused lesions and behavioral alterations, using organ tissue ratios derived from McKim *et al*. (1976).	This review
Brook trout	0.6 – 0.8	muscle	No-effect (EC0) for 2nd and 3rd generation reduced egg production per female, using regression analysis	(this review, using data from McKim *et al*. 1976)
Brook trout	1.6	muscle	No-effects apparent through 3 generations, based on statistical hypothesis testing	(McKim *et al*. 1976)
Brook trout	4	muscle	No-effects apparent until the progeny attempted to reproduce nearly 2 years after exposure began. Females showed abnormal behavior before spawning and ultimately all died.	(McKim *et al*. 1976)
Cutthroat trout	~0.055	Whole body	Increased expression of genes related to stress response, immune responses, metabolism, and contaminant detoxification.	(Moran *et al*. 2007)
White sturgeon	0.19	muscle	Possible steroidogenesis threshold	(Webb *et al*. 2006)
Mummichogs	0.2 to 0.47	Whole body	Reduced male survival, reduced the ability of offspring to successfully reproduce, and altered sex ratios in offspring	(Matta *et al*. 2001)
Golden shiners	0.5	Whole body	Abnormal behavior in response to a model predatory bird, slower to regroup afterwards	(Webber and Haines 2003)
Fathead minnows	0.86	Whole body	Reduced spawning success	(Drevnick and Sandheinrich 2003)
Fathead minnows	1.5	Whole body	Reduced spawning success	(Hammerschmidt *et al*. 2002)
Fathead minnows	0.7	Whole body	Reduced reproductive behavior and spawning success	(Sandheinrich and Miller 2006)
Walleye	0.25	whole body	Reduced growth and gonadal development	(Friedmann *et al*. 1996)

Coincidentally, this low risk threshold of 0.2 to 0.3 mg/kg ww is almost the same as the 0.3 mg/kg ww water quality standard the IDEQ has adopted subsequent to the present action to protect people eating edible portions of recreationally caught fish. As implemented, because of uncertainty in sampling and analysis of fish, IDEQ (2005) applies a 20% uncertainty factor to fish data, and would consider effluent limits and reductions necessary if average concentrations in the highest trophic level present exceeded 0.24 mg/kg ww. In waters inhabited by threatened or endangered species, the criteria would be applied to the highest trophic level of fish present; elsewhere the criteria would be applied to average trophic level of fish present in the water body (IDEQ 2005). Because the mercury aquatic life criteria are expressed as concentrations in water, but the adverse effects of mercury to fish are related to tissue concentrations, the relations between water and tissue residue concentrations need to be considered.

Factors influencing mercury tissue concentrations in fish. So far, our analysis has shown that concentrations of mercury in fish tissue residues are more meaningful for evaluating risk to fish, and that the lowest thresholds for adverse effects of mercury to fish reported in the literature were around 0.2 to 0.3 mg/kg ww, as measured in muscle fillets, or for small fish, whole carcasses. This leads to two additional and related questions:

1. The Idaho chronic criterion under review is a concentration in water (12 ng/L in filtered samples). If listed salmon and steelhead had long-term exposure to the 12 ng/L chronic criterion, what concentrations would be predicted in the fish tissues?

2. What concentrations in water would likely result in bioaccumulation to the low-risk tissue residue thresholds in fish of about 0.2 to 0.3 mg/kg ww?

Attempting to answer these questions first requires a consideration of the factors that influence mercury concentrations in fish. The bioaccumulation of methylmercury in fish is influenced by an array of abiotic, biotic, and ecological variables. However, because the elimination of methylmercury from the tissues of fish is very slow, as a rule, within a species, older and bigger fish tend to have the highest mercury tissue burdens, and because mercury biomagnifies within food chains within a community, predatory fish (i.e., higher trophic levels) will generally accumulate more mercury than non-predatory fish. Because juvenile salmonids and other fish tend to strictly feed on small invertebrates but may switch to preying on smaller fish as they grow larger, these trophic levels are not rigid within species.

The relationship between mercury bioaccumulation and trophic level may put listed steelhead and salmon at lower risk of mercury toxicity than strictly freshwater fish. Most salmonids only start becoming predominantly piscivorous when they reach about 30 cm in length, although in lakes habitats salmonids tend to start preying on fish at about 15 cm. Most listed steelhead and salmon smolts are less than 20cm in length when they leave their freshwater habitats (Quinn 2005; Mebane and Arthaud 2010). Many studies have examined the interrelationship between trophic level and size or age of fish, to the point that broadscale, predictive models have been developed. The "Environmental Mercury Mapping, Modeling, and Analysis' (EMMMA) project is a statistical model and national data set (31,813 samples) *http://emmma.usgs.gov/* that allows prediction of mercury levels in different fish species by fish length and various sampled locations (Wente 2004). Model results for the Snake River at Lewiston, Idaho, predict that piscivorous

fish such as bass or pikeminnows would exceed 0.3 mg Hg/kg by the time they reach about 20 cm (8 in.). Similar-sized Chinook salmon or rainbow trout would only be expected to have about 0.1 to 0.06 mg Hg/kg respectively (Figure 2.4.6.1). This suggests that in the larger migratory rivers in which the top predators (the highest trophic level) are pikeminnows or centrarchids such as bass or perch, if the 0.3 mg Hg/kg ww water quality standard were met, mercury tissues expected in anadromous steelhead or salmon would be less than the 0.2 mg/kg adverse effect threshold.

In waters where salmonids are the top predators, most evidence suggests that the larger and older non-anadromous fish would be more at risk of mercury toxicity, and that in waters where these fish met the 0.3 mg Hg/kg standard, all the smolt-sized salmonids would be at considerably lower risk. The length-concentration curves the Snake River show this pattern, as do empirical patterns of mercury vs. length in rainbow and brown trout collected from streams and reservoirs in southern Idaho and northern Nevada (Figures 2.4.6.1 and 2.4.6.2). For example, for fish modeled at the Snake River at Lewiston, adult, 15 inch smallmouth bass tend to have at least four times greater mercury tissue concentrations than do 8-inch Chinook salmon or rainbow trout. Thus, if conditions were such that mercury in smallmouth bass was no higher than 0.3 mg/kg, concentrations in smolt-sized fish would be considerably less than 0.3 mg/kg, and would likely be on the order of 0.08 mg/kg or lower (Figure 2.4.6.1). These patterns of higher mercury residues in older fish and fish at higher trophic levels have been repeatedly reported in the literature (Becker and Bigham 1995; Watras *et al*. 1998; Hope and Rubin 2005; McIntyre and Beauchamp 2005; Wiener *et al*. 2006; IDEQ 2007b; Peterson *et al*. 2007; Chasar *et al*. 2009; Scudder *et al*. 2009). Exceptions were noted. In cases where sediments have elevated mercury concentrations, benthic insects can be a greater source of dietary mercury than forage fish, and fish that preyed on benthic insects had higher mercury burdens than exclusively piscivorous fish (MacRury *et al*. 2002). Within the action area, mercury in sediment can be highly elevated in localized areas associated with historic gold mining (Frost and Box 2009), and thus the sediment-insect route of exposure could be locally important. Similarly, in surveys in western streams, juvenile Chinook salmon had unexpectedly high mercury burdens with a mean tissue mercury concentration of 0.30 (0.212 to 0.411) mg/kg ww and a mean length of 330 (range, 260 to 400) mm. The mercury burdens in these Chinook salmon were fifth highest of 75 species sampled across the western United States (Peterson *et al*. 2007). These Chinook salmon were collected from the Klamath River, California, (S. Peterson, personal communication) and these fish were larger than Snake River Chinook salmon smolts, so mercury levels might be expected to be lower in Snake River salmon smolts.

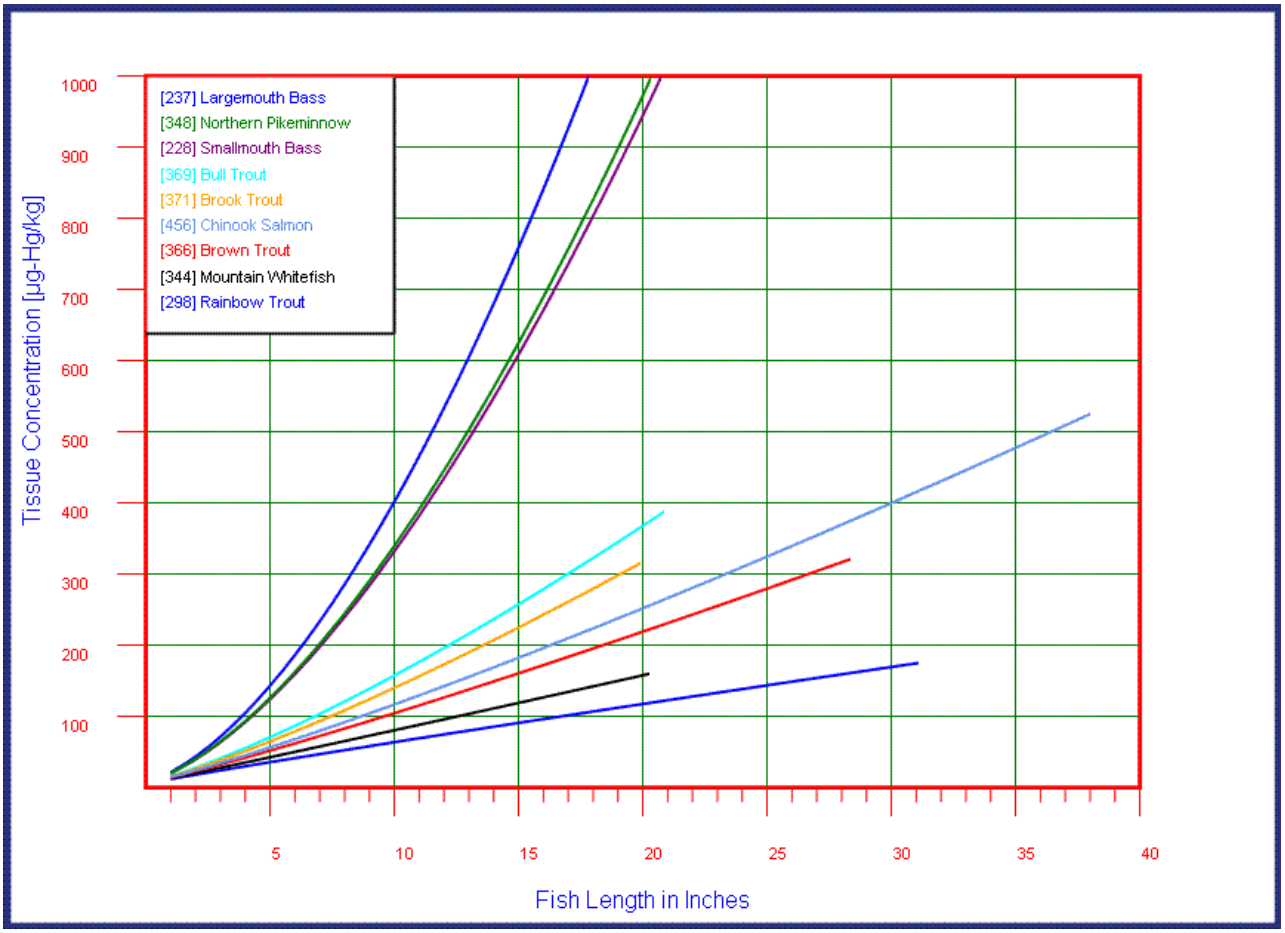


Figure 2.4.6.1. Modeled concentrations of mercury in fish-tissue for the Snake River at Lewiston, standardized by length and species (modeled and figure generated using the Environmental Mercury Mapping, Modeling, and Analysis (EMMMA) website *http://emmma.usgs.gov/*)

A further complexity in understanding risks of mercury toxicity associated with tissue burdens in fish is interactions with selenium. Selenium in fish tissue tends to reduce mercury toxicity when present at greater than a 1:1 molar ratio with mercury. Under this scenario, both the bioaccumulation rates of mercury may be lessened and mercury burdens that do accumulate tend to be less toxic (Chen *et al*. 2001; Belzile *et al*. 2006; Ralston *et al*. 2007). The mechanisms behind these patterns are unclear, and it has both been hypothesized that selenium reduces the activity and toxicity of mercury or that low-levels of mercury make fish and some mammals more susceptible to selenium deficiency (Khan and Wang 2009). Nevertheless, in Idaho and the western United States, the patterns are that in the great majority of instances, selenium is present at a 1:1 or greater molar ratio with mercury (Peterson *et al*. 2009; Essig 2010). This suggests that the risks of adverse effects of mercury in the action area are lower than those observed in conducted at lower selenium to mercury ratios. This observation, in conjunction with: (1) The age and trophic level patterns where juvenile anadromous salmonids tend to have lower exposure to mercury through feeding; and (2) the IDEQ policy of triggering actions for their mercury fish tissue-based water quality standard for existing discharges at 0.24 mg/kg ww (IDEQ 2005) ,

indicate that the 0.3 mg/kg ww fish tissue based water quality criteria for mercury would likely be sufficiently protective against risks of adverse effects to listed salmon and steelhead, and their habitats.

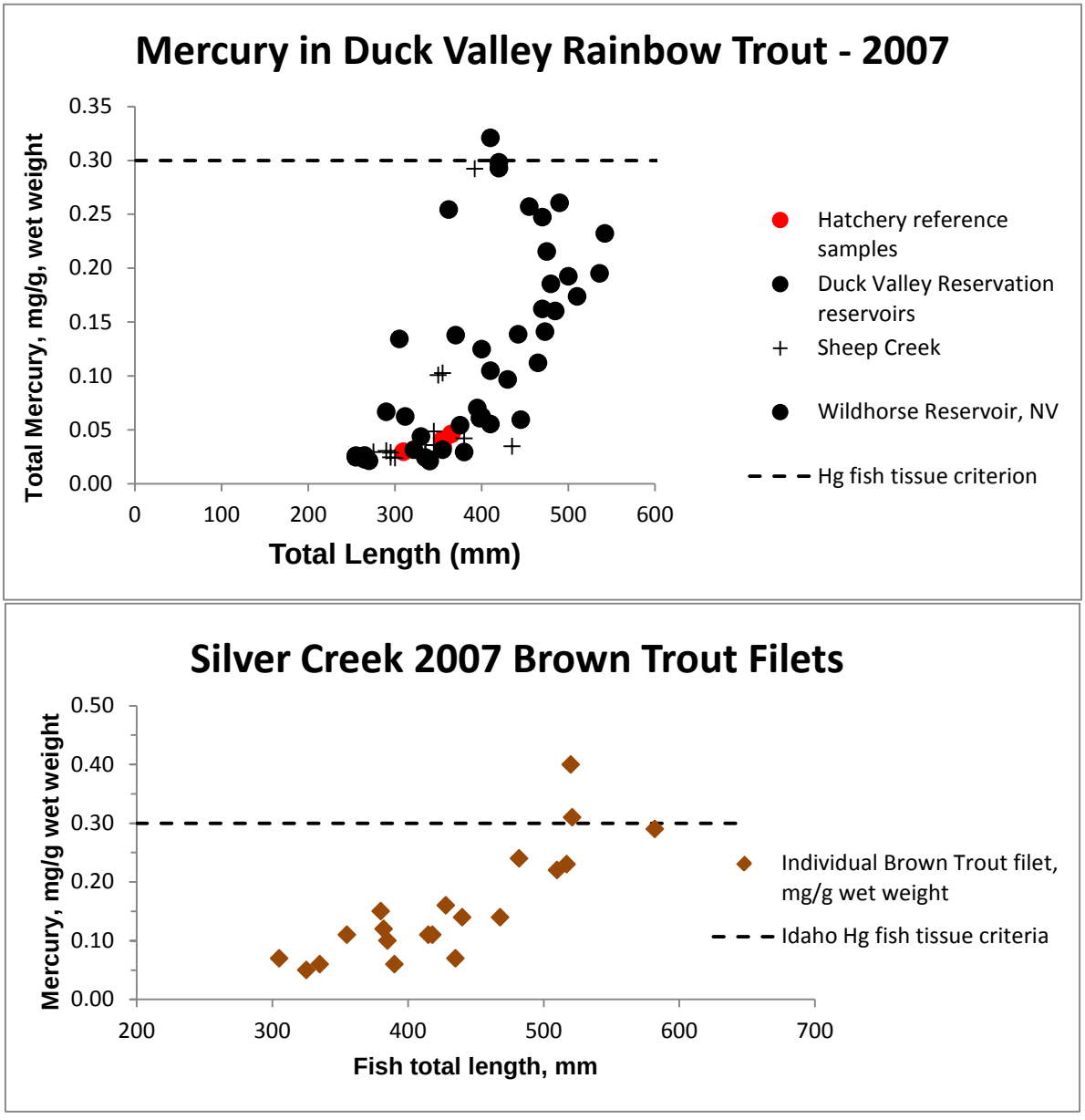


Figure 2.4.6.2. Concentrations of mercury in salmonid tissues from waters in southern Idaho and northern Nevada versus length. Data from Maret and MacCoy (2008)

Table 2.4.6.2. Examples of mercury concentrations in water and or mercury tissue burdens in fish muscle tissue (unless noted otherwise), in relation to the minimal effects threshold of 0.2 mg/kg in tissue and the 12 ng/L criteria in water under consultation.

Location or situation	Hg in unfiltered water (ng/L)	Hg in fish tissue (mg/kg, ww)	Fish species	Source
Idaho rivers, statewide average (range) in rivers	0.94 (<0.15 to 6.8)	0.16 (<0.04 to 1.1)	Various species	(Essig 2010)
Idaho rivers, 90th percentile	1.6	0.34	Various species	(Essig 2010)
Chinook salmon returning to Idaho hatcheries	–	0.149 (0.131 to 0.191)	Chinook fillets	(Essig 2010)
Chinook salmon returning to Idaho hatcheries	–	0.06 (estimated from 0.24 dw)	Chinook, whole carcass	(Felicetti *et al*. 2004)
Yankee Fork Salmon R	3 – 4.6	0.08 – 0.19	Mtn. whitefish, wb	(Rhea *et al*. 2013)
Yankee Fork Salmon R	3 – 4.6	0.08 – 0.17	Shorthead sculpin, wb	Rhea *et al*. 2013)
Yankee Fork Salmon R	3 – 4.6	<0.05	Cutthroat trout	(Mebane 2000, citing unpub. USFWS data)
Lemhi R.	0.7 – 0.92	0.13	Mtn. whitefish	(Essig 2010)
Pahsimeroi R.	0.35 – 0.51	0.10	Mtn. whitefish	(Essig 2010)
Johnson Creek at Yellow Pine, ID (tributary to SF Salmon R)	0.70	–		(Essig 2010)
Camas Creek, ID (tributary to MF Salmon R)	0.68	0.06	Mtn. whitefish	(Essig 2010)
Lochsa R.	0.54	0.05	Cutthroat trout	(Essig 2010)
Selway R.	0.4	0.049 - 0.057	Cutthroat trout	(Essig 2010)
Selway R.	0.4	0.83	Mtn. whitefish	(Essig 2010)
Selway R.	0.4	0.15	Brook trout	(Essig 2010)
Salmon R. ds NF Salmon R	–	1.1	N. pikeminnow	D. Essig, p. comm
Salmon R. ds NF Salmon R	–	0.25	Mtn. whitefish	D. Essig, p. comm
Salmon R. ds SF Salmon R	0.98 – 1.1	0.68	N. pikeminnow	(Essig 2010)
Salmon R. ds SF Salmon R	0.98 – 1.1	0.58	Smallmouth bass	(Essig 2010)

Location or situation	Hg in unfiltered water (ng/L)	Hg in fish tissue (mg/kg, ww)	Fish species	Source
SF Salmon R.	1.4	–		(Essig 2010)
Sugar Creek, tributary to EFSF Salmon River (average and range, 2011 through 2013, n=12)	2520 (12 to 26,300)	Not measured		*http://nwis.waterdata.usgs.gov*; site 13311450
NF Clearwater R	0.23	0.11	Kokanee salmon	(Essig 2010)
Stanley Basin lakes	~ 0.3 (a)	0.11 – 0.16	Bull trout	(Essig and Kosterman 2008)
Snake R. (Brownlee outlet)	1.23	0.47 – 0.77	Smallmouth bass	(Essig and Kosterman 2008)
Portneuf R., downstream of Lava Hot Springs	1.89 – 6.8	0.4 – 1.1 0.25	Brown trout Rainbow trout	(Essig and Kosterman 2008; Essig 2010)
Portneuf R., upstream of Lava Hot Springs	0.21	0.32 – 0.68	Cutthroat/ rainbow hybrid	(Essig 2010)
Salmon Falls Reservoir, ID	2.3 (0.76 - 4.25)	0.7 – 1.4	Walleye	(IDEQ 2007b)
Salmon Falls Reservoir, ID	1.4 – 2.3	0.35	Rainbow trout	(IDEQ 2007b)
Silver Creek, ID	0.15 – 1.45	0.5 –and 0.67 (mean of 10 fish each	Brown trout	(Essig 2010)
Wilderness lakes in the Seven Devils Mtns, ID and the Wallowa Mtns., OR (geometric means, assuming 80% moisture)	Not measured	0.03 0.05 0.05 0.43	Rainbow trout (n=85) Cutthroat trout (n=11) Brook trout (n=230) Lake trout (n=1)	(Eagles-Smith *et al*. 2013)
Lake contaminated by chlor-alkali plant wastes	6 (2 – 12) (filtered)	1.1 0.68 0.48	Piscivores Planktivores Benthivores	(Bloom and Effler 1990; Becker and Bigham 1995)
TMDL target for the Willamette R., OR	0.92	0.3	median for higher trophic level fish	(Hope *et al*. 2007)
Streams with a variety of land uses in OR, WI, and FL	0.6 – 5.7	0.02 – 1.2	Predatory fish	(Chasar *et al*. 2009)
Median for streams across the USA	2.09	0.085	Salmonids	(Scudder *et al*. 2009)

wb – whole-body, converted from dry weight using 27% moisture; Note a – estimated value from NF Payette River near Grandjean (0.28 ng/L) and Big Wood River, near Galena (0.26 ng/L). Both proxy sites drain watersheds with some similarities in geology and land uses as the Stanley Basin lakes.

Concentrations of mercury in water associated with mercury tissue residues of concern. NMFS examined a variety of matched samples of mercury in water and fish tissue to help evaluate concentrations in water that might produce mercury in fish at concentrations that could be adverse (Table 2.4.6.2). The data presented were selected from datasets that would be directly applicable or at least relevant to salmonids in stream or large river habitats that make up most of the action area. Repeatedly, these matched samples show that mercury concentrations in fish commonly approach or exceed the lowest adverse effect threshold of 0.2 ~ 0.3 mg/kg, even though the mercury concentrations in water were commonly an order of magnitude lower than the Idaho chronic mercury criterion.

This observation leads to the question that if the 12 ng/L water criterion for mercury would likely permit too high mercury concentrations in fish, what concentrations in water likely would likely result in low risk to fish? NMFS took two approaches to answering this question, back calculating from tissue to water using BAFs and by using a regression between matched water and tissue concentrations from Essig's (2010) large study of mercury in fish and water in Idaho rivers.

Using data reported by Essig (2010), a linear relationship between total mercury in river water and fish tissues was calculated for this Opinion: (Tissue residue (µg/kg) = 66 (L/ng))•(total Hg (ng/L) +98.9 µg/kg, $r^2 = 0.22$, $p<0.00001$). The regression equation suggests that a water concentration of 0.9 ng/L total dissolved mercury would, on the average, result in a fish tissue concentration of about 300 µg/kg. This estimated water concentration is effectively the same as the 0.92 ng/L concentration of dissolved mercury in water selected as a TMDL target for the Willamette River, Oregon (Hope *et al*. 2007). Both values are a full order of magnitude lower than the Idaho chronic criterion of 12 ng/L under consultation. Estimating the fish tissue concentration that might result if waters actually were at their allowed 12 ng/L dissolved concentration requires extrapolation, since few rivers or lakes approach this water concentration in their surface waters, even among waters with substantial mercury pollution in their food webs, such as Lake Onodaga, New York, Salmon Falls Reservoir, Idaho, or Brownlee Reservoir, Idaho/Oregon (Table 2.4.6.2). If the linear relationship from the Idaho river data held from the maximum measured value (5.5 ng/L) to 12 ng/L, the predicted fish tissue concentrations would be around 0.9 mg/kg (0.67 to 1.9, 95^{th} confidence intervals).

An alternative approach is to use BAFs to estimate potential mercury tissue residues from mercury concentrations in water measured in the field. We examined two sources of BAF estimates; those compiled by DeForest *et al*. (2007) using field data compiled from peer-reviewed literature and technical reports (e.g., USGS Water-Resources Investigations reports); and the Idaho probabilistic survey of mercury species in water and mercury in muscle tissue of edible fish (Essig 2010). DeForest *et al*'s (2007) analysis included BAFs from water concentrations in excess of 12 ng/L, Essig's (2010) BAFs were all developed from a more limited range of mercury in water concentrations, 0.2 to 5.5 ng/L. The BAFs we estimated from these independent data sets were remarkably similar (Table 2.4.6.3). From the BAFs derived

from both studies, on the average, a water body that was at the NTR (Idaho) chronic criterion of 12 ng/L would be expected to eventually bioaccumulate to produce fish mercury residues of around 3 mg/kg ww, with a range of 0.5 to 20 mg/kg or more. Thus, even the lowest BAF estimates used with the 12 ng/L water criterion would predict a muscle tissue residue greater than the 0.3 mg/kg ww threshold selected here. A muscle tissue residue of 3 mg/kg is 10 times higher than the 0.3 mg/kg threshold for risks of adverse effects selected from this review. Adverse effects in fish that have been linked with muscle tissue residues on the order of 3 mg/kg ww include complete reproductive failure, brain damage, and severe behavioral abnormalities (Table 2.4.6.1). If the BAF estimates were used to backcalculate potential total mercury concentrations in water from the 0.3 mg/kg fish tissue concentration, the total mercury in water values would range from about 0.2 to 7 ng/L, with a geometric mean value of about 1.7 ng/L. If rounded to the nearest integer to avoid implying greater precision than one significant digit, this implies on the average if rivers had total mercury concentrations less than 2 ng/L, predicted concentrations of mercury in fish tissue would be expected to be less than 0.3 mg/kg wet weight.

Table 2.4.6.3. Ranges of potential tissue concentrations that would result from (A) applying field-based BAFs to the chronic mercury water quality criterion of 12 ng/l, and (B) ranges of water concentrations that would result from applying BAFs to low-risk tissue concentrations. Calculations showing the laboratory water-only bioconcentration factor (BCF) used in EPA (1985g) to derive the 12 ng/L criterion are also included for comparison.

A. Estimated mercury concentrations resulting in fish if mercury in water were 12 ng/L:

Scenario	Water total Hg (ng/L)	Total Hg BAF	Predicted fish tissue (ng/kg ww)	Predicted fish tissue (mg/kg ww)
DeForest *et al*. (2007) BAFs				
Geometric mean	12	263,362	3,160,344	3.2
Minimum	12	40,857	490,284	0.5
Maximum	12	4,110,638	49,327,656	49.3
Essig (2010) BAFs				
Average	12	249,480	2,993,756	3.0
Geometric mean	12	178,968	2 147 620	2.1
Minimum	12	42,632	511,579	0.5
Maximum	12	1,635,294	19,623,529	19.6
EPA (1985) BCF				
Fathead minnow	12	81,700	1,000,000	1.0

B. Estimated mercury concentrations in water resulting in fish tissue concentrations of 0.3 mg/kg ww:

Scenario	Fish tissue (mg/kg ww)	Fish tissue (ng/kg ww)	Total Hg BAF	Predicted water total Hg (ng/L)
DeForest *et al*. (2007) BAFs				
Geometric mean	0.3	300,000	263,362	1.1
Minimum	0.3	300,000	40,857	7.3
Maximum	0.3	300,000	4,110,638	0.1
Essig (2010) BAF values				
Average	0.3	300,000	249,480	1.2
Geometric mean	0.3	300,000	178,968	1.7
Harmonic mean	0.3	300,000	138,215	2.2
Minimum	0.3	300,000	42,632	7.0
Maximum	0.3	300,000	1,635,294	0.2
EPA (1985) BCF				
Fathead minnow	0.3	300,000	81,700	3.6

2.4.6.3. Summary for Mercury

The 1984 chronic mercury criterion was back calculated from the FDA limit for allowable mercury content in commercially marketed seafood (1.0 mg/kg ww), using a bioconcentration factor derived from a laboratory water-only (aquaria) methylmercury exposures with fathead minnow (USEPA 1985g). Thus, the criterion derivation had no consideration of ecological effects of mercury or effects of mercury to sensitive species. In the 25 plus years since this fish marketability-based criterion was developed, much new information on the effects of mercury on the fish themselves, not just their marketability, has been developed. The newer information both reflects that: (1) The older bioconcentration values considered in the 1984 chronic criterion were about four times lower than the average bioaccumulation factors obtained in field settings; and (2) that adverse developmental effects in fish occur at <1 mg/kg.

Severe adverse effects have been observed in fish that accumulated mercury in their muscle tissue, including brain damage, behavioral abnormalities, and reproductive failure. However, effects of methylmercury on fish are not limited to neurotoxicity, but also include histological changes in the spleen, kidney, liver and gonads. These effects have been observed in multiple species of freshwater fish at tissue concentrations of methylmercury well below 1.0 mg/kg ww (Sandheinrich and Wiener 2010).

2.4.7. The Effects of EPA Approval of the Nickel Criteria

The acute and chronic nickel criteria being consulted on are 470 µg/L and 52 µg/L respectively (Table 1.3.1).

2.4.7.1. Species Effects of Nickel Criteria

Nickel poisoning in fish can cause respiratory stress, convulsions, and loss of equilibrium prior to death. 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, 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). Nickel is thought to have lower inherent toxicity to fish than other criteria metals for which aquatic criteria have been developed (Niyogi and Wood 2004).

Available toxicity test data indicate that juvenile and adult salmon and steelhead are protected from acute effects of nickel at the acute criterion (Figure 2.4.7.1). However, several studies have determined that mortality of salmonid embryos occurs over longer-term exposures to concentrations that are below the Idaho chronic criterion:

> 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.

Eisler (1998b) cite an LC_{10} of 11 µg/L, no hardness given, for rainbow trout embryos exposed from fertilization through hatching.

Birge *et al*. (1981) concluded that nickel concentrations of 10 µg/L would not impair reproduction of most aquatic species although adverse effects at concentrations were not substantially greater than this.

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.

Nebeker *et al*. (1985) found that the sensitivity of rainbow trout to long-term nickel exposures varied depending upon the developmental stage the test was started. Unlike tests with some other metals (cadmium, zinc, and maybe copper and Pb), tests initiated with swim-up fry were much less sensitive than tests started with either eyed or newly fertilized eggs. Newly fertilized eggs were most sensitive with reduced growth observed at the lowest concentration tested, 35 µg/L at a hardness of 27-39 mg/L. The Idaho chronic criterion at this range of hardnesses is 52 to 71 µg/L, which is higher than the lowest concentration causing adverse effects. However the chronic criterion over this hardness range from EPA's 2013 updated action is 17 to 23 µg/L, which is lower than the adverse effects concentration. This is illustrated in Figure 2.4.7.2, where the "Idaho chronic values" shown are the same as EPA's 2013 updated action.

Brix *et al*. (2004) tested newly fertilized rainbow trout eggs using a similar test design to Nebeker *et al*'s (1985) tests, using a higher hardness dilution water of about 91 mg/L. No adverse effects were reported from exposures up to 466 µg/L.

These results suggest that adverse effects could occur to embryos exposed to nickel concentrations that are lower than the Idaho chronic criterion for nickel which was evaluated by EPA (2000a). However, the contrasting responses of the Nebeker *et al*. (1985) and Brix *et al*. (2004) indicate there is yet considerable uncertainty in risks of nickel to aquatic life. Idaho's current criteria for nickel include a chronic criterion for nickel that is lower than Nebeker's adverse effect level; at hardnesses of 27 to 39 mg/L, the 2002 chronic nickel criterion is 17 to 23 µg/L (Table 1.3.1).

Behavioral Effects. One study was located that suggested behavioral avoidance could potentially occur at concentrations that are below the proposed chronic criterion:

Giattina *et al*. (1982) determined that rainbow trout fry avoided a nickel concentration equal to 24 µg/L at a mean water hardness of 28 mg/L. This effect concentration is greater than the updated IWQS chronic criterion (18 µg/L for hardness 28 mg/L), which is reflected in EPA's 2013 updated action.

***Hardness as a Predictor of Nickel Toxicity*.** In meta-analyses of acute toxicity data for nickel with Daphnia magna and rainbow trout Meyer *et al*. (2007b) found that, toxicity tended to decrease with increases in alkalinity, pH, and hardness. However, the relations were fairly weak,

and a similar analysis of data for fathead minnows showed no relationship between hardness and toxicity (Meyer *et al*. 2007b). Deleebeeck *et al*. (2007) investigated: (1) Whether cladocerans living in soft water (< 10 mg $CaCO_3$/L) are intrinsically more sensitive to nickel than cladocerans living in "hard water" (hardness > 25 mg $CaCO_3$/L) in chronic exposures; and (2) whether a single bioavailability model can be used to predict the protective effect of water hardness on the toxicity of nickel to cladocerans in both soft and hard water. Their results found that water hardness significantly reduced nickel toxicity to both the soft and the hard water organisms tested.

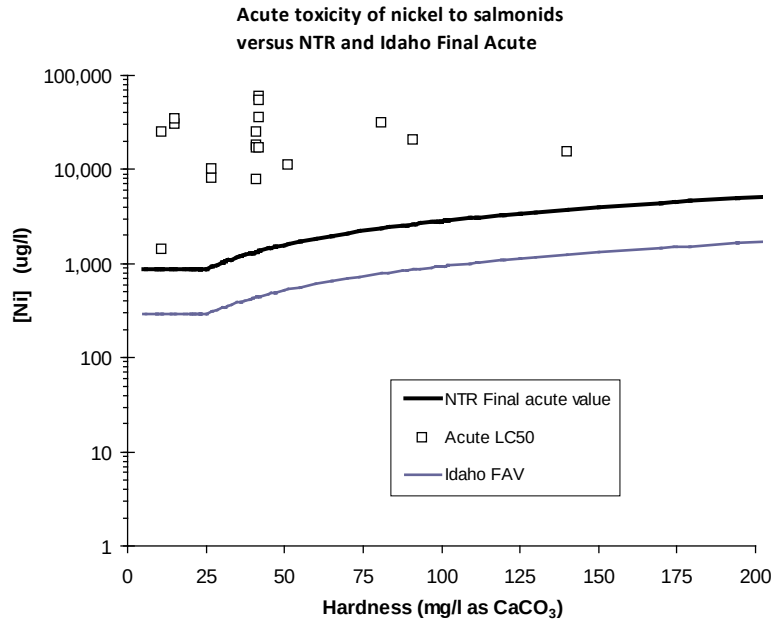


Figure 2.4.7.1 Acute LC_{50}s for nickel with rainbow trout, any life stage (no data on other salmonids) vs. the Idaho and Idaho final acute values (FAVs).

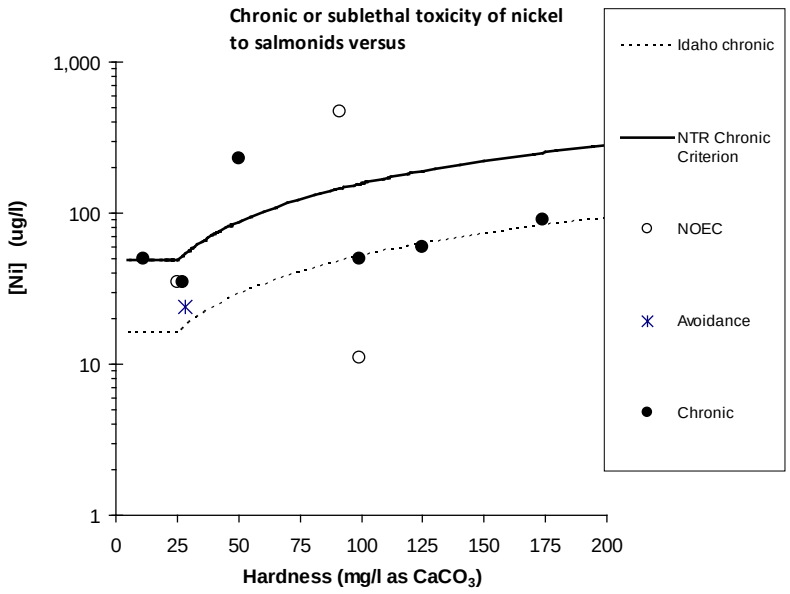


Figure 2.4.7.2. Chronic effects, no-observed effect concentrations, and avoidance concentrations with rainbow trout vs. the NTR and Idaho chronic values for nickel.

2.4.7.2. Habitat Effects of Proposed Nickel Criteria

Toxicity to Food Organisms. In some instances, nickel can be quite toxic to invertebrates such as zooplankton and amphipods. In soft waters, thresholds of effects (EC_{10} values) for zooplankton range from only about 2 to 40 µg/L in waters with hardnesses ranging from about 6 to 43 mg/L (Deleebeeck *et al*. 2007). Lethal concentrations (LC_{50}s) to the freshwater amphipod *Hyalella azteca* ranged from 77 to 147 µg/L in soft and hardwater (18 and 130 mg/L) (Borgmann *et al*. 2005a). This suggests that concentrations causing no or few effects would be in the 20 to 70 µg/L range in hard or soft water, assuming common concentration-response patterns (Section 2.4.1.6). In life cycle tests caddisflies were affected at concentrations greater than 66 µg/L in waters with hardness of about 25 to 30 mg/L (Nebeker *et al*. 1984). Criteria in softwaters comparable to those used in these softwater tests (25 µg/L) would be about 16 µg/L for the state of Idaho's updated criteria (Table 1.3.1). Thus, at least above the "*hardness floor of 25 mg/L*", the state of Idaho's updated chronic nickel criterion would likely be protective of sensitive invertebrates.

Bioaccumulation. Nickel is known to bioaccumulate in salmonids, which can accumulate through both dietary and water-borne exposure routes (EIFAC 1984; Eisler 1998b). Bioconcentration factors vary substantially both within and between species, age of organism, and with exposure concentration. 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 unknown to what extent this is a significant hazard for listed salmon and steelhead.

2.4.7.3. Summary for Nickel

A striking feature of the information reviewed for nickel toxicity is the tremendous range of effects concentrations. Much work, particularly short-term exposures, has shown adverse effects from nickel at concentrations in the milligrams per liter range, which are hundreds or even thousands of times higher than environmentally relevant concentrations. Yet other work has shown nickel to be about as toxic or more toxic, in long-term exposues than metals more commonly considered to pose a risk to sensitive organisms, such as copper or cadmium. No reports were located of adverse effects from short-term (96-hr) toxicity tests using salmonids at concentrations below the final acute value (two times the acute criterion) for nickel.

During this consultation, EPA revised the proposed chronic criterion for nickel resulted in a level that is considerably more protective of listed salmon and steelhead. Potential adverse effects from exposure to nickel at concentrations at or below the criterion in the revised action are expected to be primarily to sensitive invertebrates which may be a food source for listed species. This affect is expected to be very small.

2.4.8. The Effects of EPA Approval of the Selenium Criteria

Selenium in water is a particularly challenging substance to evaluate risks to listed salmon and steelhead because of many contradictions in the available science and controversies of interpretation. Selenium is an essential micronutrient for all animals that have a nervous system, yet it is toxic at not much higher concentrations. At optimal concentrations, selenium is an antioxidant nutrient with positive effects on the immune system in mammals and birds, yet oxidative stress seems to be the principal mechanism of toxicity in animals and may compromise immune function at higher concentrations (Burk 2002; Palace *et al*. 2004; Miller *et al*. 2007; Janz *et al*. 2010). The cell damage caused by oxidative stress in turn can lead to a cascade of symptoms that include edema in developing embryos; teratogenic deformities in offspring; spinal deformities; anemia; cataracts; popeye; pathological alterations in liver, kidney, heart, and ovary; reduced egg viability; and reduced growth of juveniles. Oviparous (egg laying) vertebrates appear to be the most sensitive taxa to selenium toxicity (Lemly 2002; Janz *et al*. 2010). Selenium has been called an insidious threat in waters where it is elevated, because adult fish may appear perfectly healthy, whereas severe effects may be occurring to early life stage fish but not be noticed in routine surveys until a large percentage of the year classes are affected (Lemly 2002). Because of concerns over effects of selenium, a large amount of research has been focused on effects of selenium to wildlife and aquatic life. Over 120 references pertinent to effects of selenium on the freshwater life stages of salmonids were located and reviewed for our analysis.

Idaho's chronic aquatic life criterion for selenium of 5 µg/L is unique in that it is based on "other data" rather than the usual approach that uses the 5th percentile of the SSD in conjunction with an ACR. The "Other Data" provision in EPA's Guidelines for developing aquatic life criteria serves to allow the use of pertinent information that could not be used directly in the usual species ranking, etc. approach. Data from any type of adverse effect that has been shown to be biologically important could be used, such as data from behavioral, biochemical, physiological, microcosm, and field studies. If the "other data" show that a lower criteria value should be used instead of the usual final chronic value, then the CCC would be based on this "other data" (Stephan *et al*. 1985, section X.) To NMFS' knowledge, selenium is the only substance for which the "other data" were sufficiently compelling to adjust a chronic water quality criterion.

The adverse effects attributable to selenium from a well documented field study were both severe (decimation of fish populations in a reservoir with elevated selenium) and occurred at lower selenium concentrations than were calculated from the laboratory studies on toxicity available at the time. In Belews Lake, a reservoir in north-central North Carolina that received fly ash from a coal power plant, selenium concentrations in water reached about 10 µg/L in the main body of the lake. Populations of several fish species suffered recruitment failure and then collapsed. In an arm of the reservoir that had limited circulation with the main body of the lake and selenium in water was below or near the detection limit of 5 µg/L, the fish assemblage was mostly intact. Therefore, EPA set the recommended chronic criterion at the detection limit available during the studies, 5 µg/L. This concentration was EPA's best estimate of a concentration that was intended to be protective, but also generally attainable based upon the information available to them at the time. Subsequently; however, pronounced adverse effects have been discovered in low selenium areas of the reservoir and other locations, at water selenium concentrations down to less than 1 µg/L (EPA 1998). In another twist from the usual approach, the acute criterion was back calculated from the field-based chronic criteria, using a laboratory water based acute:chronic ratio of 8 (EPA 1987a).

The combined notoriety of the Belews Lake and similar cases and the occurrences of severely deformed aquatic bird embryos in western reservoirs and wetlands that received elevated selenium in irrigation return water (e.g., Presser 1994) led to much research on selenium bioaccumulation and toxicity in aquatic organisms. Thus, a large body of knowledge has become available subsequent to EPA's 1987 selenium criteria document.

Recently, several key areas of consensus in the scientific community have formed regarding selenium risks to aquatic life and criteria to protect them:

- Diet is the primary pathway of selenium exposure for both invertebrates and vertebrates.

- Traditional methods for predicting toxicity on the basis of exposure to dissolved concentrations do not work for selenium because the behavior and toxicity of selenium in aquatic systems are highly dependent upon situation-specific factors, including food web structure and hydrology.

- Selenium toxicity is primarily manifested as reproductive impairment due to maternal transfer, resulting in embryotoxicity and teratogenicity in egg laying vertebrates (Janz *et al.* 2010).

Because adverse effects in fish could be better related to selenium residue concentrations in tissues than concentrations in water, recent efforts to evaluate, refine, or develop site-specific aquatic life criteria or thresholds have focused on selenium residues in fish (EPA 1998; DeForest *et al*. 1999; Hamilton 2002, 2003; EPA 2004; deBruyn *et al*. 2008; DeForest 2008; Janz *et al*. 2010). However, while consensus seems to have been reached that an aquatic life criteria for selenium could be based on tissue concentrations, just what number that should be has been the subject of considerable dispute, with proposed threshold values ranging from about 4 to 11 mg/kg, as whole-body dw, part per million (DeForest *et al*. 1999; Hamilton 2003; Skorupa *et al*. 2004; EPA 2004). The EPA (2004) proposed a fish tissue criterion of 7.9 mg/kg dw in fish, with an summer or fall monitoring trigger of 5.8 mg/kg dw, which was primarily based on a dietary toxicity study with bluegill, *Lepomis macrochirus*, under a simulated winter temperature and photoperiod regime. Subsequently, the EPA has conducted additional testing of bluegill under different temperatures and published a call for "scientific information, data, or views on the draft selenium aquatic life criterion" (*http://www.epa.gov/waterscience/criteria/selenium/* and *Regulations.gov docket* (EPA-HQ-OW-2004-0019)). As of April 2009, about 268 responses had been posted to this site, ranging from statements of opinion to complex research reports or original research and interpretative analyses.

This plethora of competing information presented a challenge to resolving the questions of the present review. These include three questions:

1. What concentration of selenium in which fish tissues is a sufficiently low threshold to protect listed salmon and steelhead?

2. Would the effective chronic aquatic life criterion of 5 µg/L selenium in water likely result in bioaccumulation of selenium to levels in tissues that are less than the fish-tissue threshold identified in Question 1 (i.e., is likely protective), or that are greater than the fish-tissue thresholds (i.e., is likely under protective)?

3. If the answer to Question 2 is the latter (underprotective), what concentration in water likely would be protective, i.e., would not result in bioaccumulation to threshold levels in fish?

NMFS reviewed over 120 scientific articles and technical reports in attempts to best answer these questions. These questions are addressed in the "chronic effects" subsection below.

2.4.8.1. Species Effects of Selenium Criteria

The aquatic life criteria for selenium under consultation are an acute criterion of 20 µg/L and a chronic criterion of 5 µg/L, both expressed as "total recoverable" unlike the dissolved criteria for most metals.

Acute Selenium Criterion. Because risks of selenium to aquatic life are via the food chain, the traditional acute toxicity testing database provides no information of value to understanding selenium toxicity in nature. Since a water-based criterion to protect against short-term exposures is environmentally meaningless (Chapman *et al*. 2009; Janz *et al*. 2010; Janz 2011), it is not reviewed in detail here. For example, 96-hour LC_{50} values for juvenile rainbow trout range from 4,200 µg/L to 47,000 µg/L, which are at least 200 times greater than the acute criterion of 20 µg/L (Janz 2011). Unlike all other EPA criteria documents, the acute selenium criterion was not developed from acute toxicity test data, but was back-calculated from the chronic, field-based criterion.

Chronic Selenium Criterion. Because the chronic criterion was derived from a field study where selenium exposure and effects occurred via reproductive failure linked to bioaccumulation from the food web, not water exposures (EPA 1987a; Sorensen 1991; Lemly 1997), and because adverse effects of selenium are associated with selenium in tissues rather than concentrations in water-only exposures of selenium, only toxicity associated with the bioaccumulation of selenium in tissue residues are considered.

2.4.8.2. Habitat Effects of Selenium Criteria

Toxicity to Food Organisms. Macroinvertebrates have typically only been considered dietary sources of selenium to higher trophic levels, in part based on Lemly's (1993a) conclusion that the most important aspect of selenium residues in aquatic food chains is not direct toxicity to the organisms themselves, but rather the dietary source of selenium they provide to fish and wildlife species that feed on them. Lemly (1993a) based his conclusion on review of field and laboratory studies in which he found the lowest threshold adverse effects was reduced growth of adult *Daphnia magna* at tissue residues of 20 mg/kg dw, and reduced reproduction occurred at 30 mg/kg dw. Selenium in the diets of fish can cause adverse effects at less than half this concentration. deBruyn and Chapman (2007) challenged that assumption in a commentary which argued selenium may cause toxic effects to some freshwater invertebrate species at concentrations considered "safe" for their predators. Preliminary results presented by Conley *et al*. (2009) further suggested that if mayflies were exposed to selenium through a more natural feeding regime, maternal transfer to eggs and adverse effects to progeny could occur at dietary concentrations as low as about 11 mg/kg dw. Studies of long-term experimental selenium dosing of experimental streams also noted that elevated selenium concentrations affected the structure of macroinvertebrate communities and were more important to ecosystem structure and function than simply through their role as food for fish and birds. For instance, isopods were depressed following long-term exposures to about 10 and 30 µg/L selenium, which may have resulted in a competitive release that directly supported higher densities of amphipods, and indirectly supported an extremely high population density of baetid mayfly and damselfly nymphs (Swift 2002). Thus, assuming that macroinvertebrates simply act as a conduit of selenium to higher trophic levels may not be accurate. However, the literature NMFS reviewed does not indicate that elevated selenium would lead to profound community-level impacts to macroinvertebrates that would limit food resources. For instance, baetid mayflies and amphipods are probably at least as nutritious food sources for juvenile salmonids as isopods. Thus the primary concern with

selenium in stream food webs does not appear to be one of food limitation but rather as trophic transfer.

Selenium is an essential trace element for fish at dietary concentrations of 0.1 to 0.5 mg Se/kg dw. In fish, selenium toxicity has been reported to occur at dietary concentrations only seven to 30 times greater than those considered essential for proper nutrition (i.e., > 3 mg Se/kg dw) (Janz *et al*. 2010). There have been efforts to use selenium residue concentrations in salmonid fish prey organisms in effects monitoring and assessment targets. For instance, a food web monitoring plan in Thompson Creek, Idaho, a stream that receives mine wastewater effluents with elevated selenium concentration, set a maximum residue guideline of 4 mg/kg dw in aquatic insects or forage fish (Mebane 2000). However, despite the consensus that diet is the sole important route of selenium exposure to fish, the approach of setting dietary guidelines in monitoring and assessment seems to have received little subsequent attention. Instead, most recent research has been aimed at developing protective guidelines for avoiding selenium toxicity in fish and has focused on effects attributable to the residues in the fish themselves, rather than on defining dietary adverse effect thresholds for selenium in diet.

One study we reviewed in detail tested the effects of organic selenium in the diets of juvenile Chinook salmon (Hamilton *et al*. 1990). They fed the salmon using two diets, one contained meal made from low-selenium mosquitofish (collected from a reference site) fortified with SeMe, and a second diet that contained fish meal made from high-selenium mosquitofish collected from a selenium-laden drain (SLD) located in an intensely irrigated agricultural watershed in California. These diets are likely much more biologically relevant than various studies that studied effects of dietary selenium administered through commercial trout chows or other feeds fortified with sodium selenite or other inorganic selenium species. This is because selenium that has been incorporated into living tissues of plants or animals is likely present as organic selenium (Besser *et al*. 1993).

By analyzing growth reductions occurring after 60 days of exposure to a range of SeMe concentrations in the mosquitofish meal diet, a threshold for the onset of effects was estimated at about 7.6 mg/kg selenium in the diet as dw (Figure 2.4.8.1(A)).

The Hamilton *et al*. (1990) feeding studies were conducted for 90 days, but survival in the control groups dropped from 99% at 60 days to 67% at 90 days. Lower survival occurred in all selenium treatments at 90 days compared to 60 days, and effect concentration percentile (ECp) estimates based on growth or survival were lower as well (i.e., were more sensitive). Because other studies have had high survival rates in similar aged Chinook salmon controls (e.g., Chapman 1982), it seems possible that the lower survival with the 90-day results were influenced by some undetected factor such as disease or parasitism. Thus, only the 60-day results are relied upon here.

Tissue concentrations of selenium associated with chronic responses in salmonids. In natural waters and food chains, selenium most commonly occurs as inorganic selenium in two forms. Selenate, SeO_4^{2-}, is an anion that tends to predominate in oxic conditions, and selenite, SeO_3^{2-}, is is an anion that tends to predominate in reducing conditions. Both forms are readily taken up by floating or attached algae (phytoplankton or periphyton) or bacteria and then by aquatic

invertebrates, fish, and birds. Selenium typically biomagnifies strongly from water to algae, with biomagnification factors ranging from the hundreds to >10,000. Selenium is also converted from the inorganic to organic forms by algae or bacteria. Organic selenium is readily bioavailable to higher trophic levels and bioaccumulates from algae to invertebrates to fish or birds. However, further biomagnification is much lower than from the water to primary producers (Besser *et al*. 1993).

The harmful effects of bioaccumulated selenium on fish have generally been detected through two distinct types of studies and effects. The first, maternal transfer of selenium to developing embryos, may follow from the exposure of adult female fish to selenium with resulting embryo/fry teratogenesis, edema, and mortality. Experimentally, these effects are usually detected by either dietary exposure of broodstock to selenium, or by capturing fish exposed in the wild in areas with elevated selenium, stripping eggs and milt, and evaluating the larval development in the laboratory. The second type of effects and studies are growth reductions or mortality resulting from direct exposure of juveniles to selenium (Janz *et al*. 2010).

Of these two study types, only the latter, the direct exposure of juveniles, is considered relevant to the potential exposures of listed anadromous salmon and steelhead within the action area. Because of the nearly total cessation of feeding by anadromous salmon and steelhead as they re-enter freshwater to start their spawning migration, dietary exposure of adult females within freshwater would be very small. A large body of science on the effects of selenium via maternal transfer or reduced fecundity in non-anadromous salmonids or other fish species such as cutthroat trout, rainbow trout, bull trout, brook trout, northern pike, bluegill and minnow did not seem to be very relevant for estimating effects on listed anadromous species and thus is not further considered (e.g., Gillespie and Baumann 1986; Schultz and Hermanutz 1990; Hermanutz 1992; Hermanutz *et al*. 1992; Hermanutz *et al*. 1996; Kennedy *et al*. 2000; Palace *et al*. 2004; de Rosemond *et al*. 2005; Holm *et al*. 2005; Muscatello *et al*. 2006; Van Kirk and Hill 2007; Rudolph *et al*. 2008; Hardy *et al*. 2010).

NMFS evaluated several studies on direct exposure of juvenile salmonids to selenium were evaluated in attempts to estimate thresholds for "safe" or very low, inconsequential, effects of selenium tissue residue on growth or survival. Summaries of the evaluations are presented in Table 2.4.8.1. Analyses of three of the four studies reviewed resulted in low-effect estimates ranging only from about 5 to 7 mg/kg dw, with one considerably higher estimate at 11 mg/kg dw. The latter estimate is considered the least reliable of the values because it required an extrapolation from liver to whole-body residue, using a relationship established with a different species of fish (Table 2.4.8.1.)

Additionally, results from the feeding study with rainbow trout and organic selenium by Vidal *et al*. (2005) were particularly challenging to interpret, because of lack of monotonic response with increasing dietary exposures. Growth was reduced in all dietary exposures, but the lowest growth reductions occurred at the highest dietary exposure. The lowest dietary exposure resulted in a whole body concentration of 0.58 mg/kg ww, about 2.9 mg/kg dw at 90 days, which was similar to the control concentrations (1.6 to 6.2 mg/kg dw). To further complicate matters, whole body selenium residues in all dietary treatments including the controls peaked at 60 days and then declined by 90 days, making it unclear what residue concentration was most associated

with effects. The threshold of adverse effects concentration selected by Vidal *et al*. (2005) is listed in Table 2.4.8.1, although that seems to have been supported by their informal judgments rather than any statistical analyses.

The remaining two tests considered were with rainbow trout and Chinook salmon by Hunn *et al*. (1987) and Hamilton *et al*. (1990) respectively. Of these, we place greater reliance for estimating thresholds of effect on the latter study in which reduced survival and growth occurred in juvenile Chinook salmon fed organic selenium (Hamilton *et al*. 1990). This is because the dietary exposures by Hamilton *et al*. (1990) seemed more relevant to the type of exposures that juvenile salmonids would receive in the wild, the selenium exposures and resulting residues bracketed an environmentally relevant range of concentrations, and the results showed clearly non-adverse and adverse effects, such that robust statistical analyses could be made.

Hamilton *et al*'s (1990) tests included two series in which fish were either fed Oregon moist pellets that had been fortified with organic selenium, as SeMe or a meal made with natural-origin caught forage fish captured from the San Luis Drain, an irrigation wasteway with elevated selenium as well as other contaminants. Using the SeMe treatment effects after 60 days exposure, by logistic regression, we estimated an essentially no-effect concentration for weight reductions at about 3 mg/kg selenium as whole-body dry weight residues. Low-effect thresholds as a 10% reduction in weight and a 4% reduction in length (EC_{10} and EC_{04}) were both similarly estimated at about 7.6 mg/kg (Figure 2.4.8.1 (B and C) 2.4.8.3*)*. The EC_{04} statistic for length reduction was used as an estimate of a threshold for low effects that could be biologically important because in population modeling with Chinook salmon, a 4% length reduction was projected to have low risk for increased extinction risk, although it could result in a delay in population recovery (Mebane and Arthaud 2010).

The effects concentrations estimated from the fish fed the San Luis Drain diet were lower than the SeMe fortified feed (i.e., apparently more sensitive to Se). In the San Luis Drain series, reduced growth occurred in all selenium exposures relative to controls *(*Figure 2.4.8.1(D)*)*. However, inspection of the whole-body selenium and growth curve show that after the first treatment, the slope of the curve is flat with few further reductions in growth until the highest treatment. If the control treatment were excluded from the regression, then the nonlinear curve fit is almost perfect (Figure 2.4.8.1(E)). This suggests that something besides selenium may have been in the wild San Luis Drain diets that contributed to the reduced growth and survival, such as unmeasured pesticides or other farm chemicals that presumably would be present in a drain in an intensely cultivated farm region. Thus we relied on the SeMe test series for selecting no- and low-effects thresholds in preference to the San Luis Drain series.

Table 2.4.8.1. Effects concentration (EC) estimates for selenium whole-body tissue residues on growth or survival of juvenile salmonids, assuming no maternal pre-exposure. Underlined value indicates EC selected for primary effects analysis.

Species	Se, whole-body residues (µg/g dw)	Effects	Exposure	Notes and data sources
Chinook salmon	12	Reduced survival (EC10)	Seleno-methionine (SeMe) in diet, 60 days	Calculated from data reported in Hamilton *et al*. (1990), using threshold sigmoid regression.
Chinook salmon	6.5	No effect on survival (EC0)	SeMe in diet, 60 days	Calculated from data reported in Hamilton *et al*. (1990), using threshold sigmoid regression.
Chinook salmon	**7.6**	Reduced growth (EC10 for weight ≈ EC04 for length	SeMe in diet, 60 days	Calculated from data reported in Hamilton *et al*. (1990), using threshold sigmoid regression
Rainbow trout	7.2	Reduced growth, EC04 for length	Sodium selenite in water, 60 days	Calculated from data reported in Hunn *et al*. (1987), using piecewise regression
Rainbow trout	3.5 5.3	EC0 and, EC10 for survival, respectively	Sodium selenite in water, 60 days	Calculated from data reported in Hunn *et al*. (1987), using threshold sigmoid regression
Rainbow trout	6.0	Reduced growth, LOEC	SeMe in diet, 90 days	LOEC selected by Vidal *et al*.(2005), assuming 20% solids (Jarvinen and Ankley 1999; Essig 2010)
Rainbow trout	11	NOEC for growth or biochemical parameters	Sodium selenite in low carbohydrate diet, 112 days	Estimated from a Se concentration in liver (38 µg/g dw) reported by Hilton and Hodson (1983), using a liver:whole-body regression developed with bluegill (deBruyn *et al*. 2008)

Hunn *et al*. (1987) achieved a gradient of tissue residues in juvenile rainbow trout by exposing them to waterborne selenite. Estimates of effect concentrations linked to a given tissue residue after 60 days results in very similar effects concentration estimates as were obtained from Hamilton *et al*'s (1990) Chinook salmon and SeMe test. At 60 days, an EC_{10} for survival of about 5.3 mg/kg, and an EC01 of 3.5 mg/kg were estimated, and for length reductions, an EC_{04} of around 7.2 mg/kg dw was estimated. These effects concentration estimates are similar to those for reduced growth from the juvenile Chinook. The choice to give these results lesser importance in this analysis than those of Hamilton *et al*. (1990) is admittedly debatable, since even though the selenium was derived from different sources (water vs. spiked diet), it may be that once metabolized into fish tissues, selenium may have the same toxic effects on a gram per gram basis (EPA 1998). However, because the effect values are similar, this issue does not have to be resolved for the present Opinion.

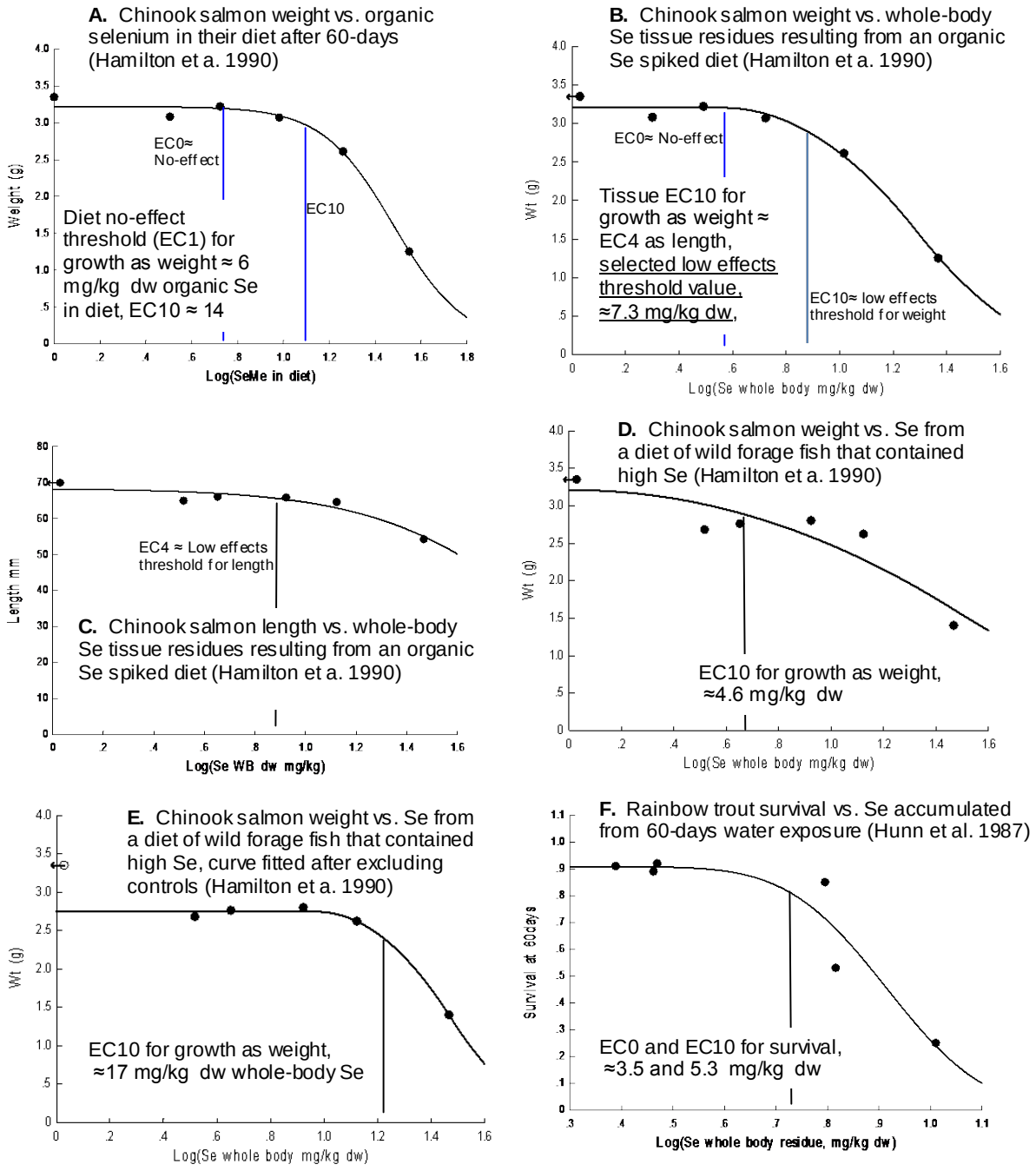


Figure 2.4.8.1. Estimates of thresholds for no- and low-effect concentrations for selenium in diet and whole-body tissues of juvenile Chinook salmon or rainbow trout. Curve fitting and curve fitting and effects concentration percentiles (ECp) were estimated using threshold sigmoid regression (Erickson 2008). EC10=concentration causing a 10% reduction in growth or survival.

Bioaccumulation of selenium through stream food web trophic transfer. Now that thresholds have been estimated for no- and low-effects of selenium in the tissues of rearing anadromous salmonids, the next step of the analysis of risks of selenium in water at the chronic criterion concentration is to estimate what tissue concentrations would be expected from ambient concentrations of selenium in streams. These estimates may be made through food web studies in streams and ecosystem models. Traditionally, BAFs have been used to relate water concentrations of a substance to tissue residues, where the BAF is the ww concentration in tissues divided by the concentration in water. However, BAFs are a crude measure and can be unreliable for a variety of reasons including the following: different aquatic ecosystems have shorter or longer food webs; the source of major biomagnification of selenium, water to algae, varies greatly by water body type; and organisms will regulate internal concentrations of micronutrients such as selenium resulting in higher BAFs at low water concentrations and lower BAFs at higher concentrations (Luoma and Presser 2009; Stewart *et al*. 2010).

In contrast, ecosystem food web models have advantages over the traditional BAFs because food web models can account for some of the interrelated factors that contribute to the widely variable BAFs, namely food web type and length, speciation, and water body type. In particular, selenium uptake may differ greatly by water body type such as estuary, lentic freshwater, or lotic (flowing) freshwaters (Orr *et al*. 2006; Luoma and Presser 2009). Ecosystem models of selenium provide a means for relating selenium concentrations in the water column and concentrations in other food chain components, including selenium residues in fish or fish tissue guidelines to prevent adverse effects. The models can then be run either forwards or backwards to predict concentrations of selenium in fish from a given concentration in water, or for a given concentration in fish such as a tissue-based criterion or guideline, then an associated concentration in water can be estimated (Luoma and Presser 2009; Presser and Luoma 2010). Because in the preceding discussion, a selenium tissue concentration of about 7.6 mg/kg dw was estimated, this concentration in fish "C_{fish}" can be treated as a given, and the model can be used "backwards" to solve for a corresponding estimated selenium concentration in water "C_{water}" (Equation 1):

$$C_{water} = \frac{C_{fish}}{(TTF_{fish})\bullet(TTF_{invertebrate})\bullet(K_d)} \qquad \text{(Equation 1)}$$

where:

C_{water} is the allowable water-column concentration of selenium, given a selenium fish tissue guideline;

C_{fish} is the selenium fish tissue residue guideline, in whole-body or muscle, as dry weight;

K_d is the partitioning coefficient between particulates such as benthic biofilms (i.e., algae and associated living and non-living material, sometimes called aufwuchs) and the water-column selenium concentration, Kd is calculated as $K_d = C_{particulate} \div C_{water\text{-}column}$

$TTF_{invertebrate}$ is the trophic transfer factor from biofilm to aquatic insects that graze on biofilm

TTF_{fish} is the trophic transfer factor from aquatic insects to fish that prey on the aquatic insects.

This form of the model is appropriate for a short coldwater food web where the fish prey exclusively on aquatic insects. In a longer food web with fish preying on other fish, such as bull trout in large rivers, then an additional factor would be added for trophic transfer between the forage fish and top predator. Because trophic transfer factors (TTFs) for fish-to-fish are close to 1, if some portion of the fish's diet included other fish, this would not change the model predictions much.

In order to estimate TTFs and K_d values for streams, reliable data on selenium in food web compartments are needed. For listed Snake River salmon and steelhead, the vast majority of their potential exposure within the action area is in flowing streams and rivers; therefore, NMFS did not consider lentic reservoir scenarios. We evaluated extensive data on selenium concentrations in stream foodwebs from two adjacent watersheds located in the upper Salmon River drainage, Idaho. Additionally, a state-wide probabilistic assessment of selenium concentrations in water and fish tissues was recently completed in Idaho (Essig and Kosterman 2008; Essig 2010). These data are summarized in Table 2.4.8.2. The Idaho statewide assessment used a probabilistic approach where a random draw of all stream segments above a certain size in a global information system dataset was used to select sample sites. This approach allowed more robust statistical analyses of median and ranges of selenium concentrations than could be made if known water bodies of concern were targeted.

We examined two adjacent watersheds in the upper Salmon River, Idaho, watershed, the Yankee Fork and Thompson Creek, because of concerns of suspected elevated selenium, mercury, and other metals as result of mining activities. The Yankee Fork has generally elevated selenium contents in stream sediments and alluvium that reflect the generally high selenium contents in the volcanic rocks that underlay the Yankee Fork and the presence of gold and silver selenides in some of the veins that were exploited in the early phases of mining (Frost and Box 2009). In samples from more than 70 locations throughout the watershed, the highest selenium concentrations were obtained from two samples of undisturbed alluvium, reflecting natural sources. A major open-pit bulk-vat leach gold mining operation, the Grouse Creek Mine, operated on Jordan Creek, a tributary to the Yankee Fork, in the mid-1990s. Selenium concentrations in stream sediments showed no pattern attributable to the Grouse Creek Mine (Frost and Box 2009).

Thompson Creek, Idaho is located just east of the Yankee Fork, sharing a watershed divide. A large-open pit molybdenum mine, the TCM, is partially located within the drainage. Overburden from the pit is dumped into two valley-fill waste rock piles. Two small streams that have elevated selenium concentrations drain from the waste-rock piles into Thompson Creek. These discharges are jointly permitted by EPA and the state of Idaho through the NPDES and Idaho state certification programs. Among many other constituents, selenium in water, biofilm, invertebrates, and fish is systematically monitored by personnel from the mine and consultants pursuant to the NPDES permit and certification (Mebane 2000).

Table 2.4.8.2. Median selenium concentrations in coldwater stream webs relevant to the area of interest, data collected between 2001 and 2008 for all study locations.

Location	Water (µg/L)	Biofilm (Periphyton and detritus, mg/kg dw)	Aquatic insects, mg/kg dw	Sculpin, whole-body, mg/kg dw	Salmonids (WB, mg/kg dw)	Sources & notes
Idaho, statewide median from probabilistic sampling	0.14				1.3	1
Thompson Creek, Idaho - upstream of mine effluents	0.9	2.6	2.7	6.2	6.7	2
Thompson Creek, Idaho - downstream of mine effluents	3.3	5.6	7.2	9.6	7.8	2
Yankee Fork, Idaho - upstream of mine effluents	<0.10	0.4	4.2	6.9	6.2	3
Yankee Fork, Idaho - downstream of mine effluents	0.21	0.4	3.8	5.4	5.6	3
McLeod and Smoky River systems, Alberta - reference streams	0.2	1.0	4.5	–	1.8	4
McLeod and Smoky River systems, Alberta - mining influenced stream	10.7	3.2	10.0	–	8.9	4

Table Notes:

1 – Essig 2010, mean of all collected fish species, muscle tissue, range 0.2 to 14 mg/kg dw, converted from fresh weight using 21.2% solids. Range in water, <0.09 µg/L to 1.75 µg/L.

2 – Water selenium data from Thompson Creek Mining Company, biological data are from August 2003, 2004, and 2007 CEC (CEC 2004b, 2005; GEI 2008). Detection limit for Se in water was 1 µg/L and close to 50% of the samples collected upstream of the mine discharges were <1 µg/L. Maximum likelihood estimates (MLDE) based on the distribution of detected values were used to estimate non-detect concentrations to estimate median concentration (Helsel 2005); however, the resulting estimated median selenium concentration upstream of mine effluents were similar whether the MLE approach or the "data fabrication" approach of using ½ the detection limit for non-detect values was used (0.9 vs. 0.8 µg/L respectively.

2 – Water data from Hecla Mining Company, 2006-2008, detection limit was 0.1 µg/L. Biological data were collected in 2001 and 2002(Rhea *et al*. 2013)

4 – Casey (2007)

Background concentrations of selenium in Thompson Creek are considerably higher than median background concentrations in Idaho, estimated at 0.9 and 0.13 µg/L, respectively. Because no major mining or other human disturbances are known of in the Thompson Creek drainage upstream of the TCM discharges, the elevated instream background selenium concentrations are presumed to be of mostly natural-origin. Concentrations downstream of the discharges from the waste rock dumps are substantially higher, averaging about 3 µg/L and ranging from about 1 to 8 µg/L Se. About 80% of the streams in Idaho are estimated to have baseflow selenium concentrations lower than those in Thompson Creek upstream of mining discharges, and >99% are estimated to have concentrations lower than in Thompson Creek downstream of mine discharge (Table 2.4.8.2.; Essig 2010).

In addition to the three Idaho studies reviewed (statewide, Thompon Creek and Yankee Fork, Table 2.4.8.2), a fourth study relevant to this analysis was from a series of selenium enriched and

reference streams in Alberta that had similar invertebrate and resident salmonid species as did the Idaho streams (Casey 2007). With the exception of the Yankee Fork data, all of the data that we used to estimate TTFs between for different food web positions summarized in Table 2.4.8.2 were from water and biota sampling sites that were matched in space and time. The Yankee Fork water data with low enough detection limits to be useful were not matched in time, but because the water selenium concentrations were low and fairly uniform they are assumed to be representative.

Table 2.4.8.3 Median trophic transfer factors (TTF) and water-biofilm partitioning coefficients of selenium within coldwater stream food webs.

	Trophic transfer factors (TTFs)			Partitioning coefficients
	Aquatic insects	Fish (shorthead sculpin)	Fish (salmonid)	$K_{d\ (water\text{-}biofilm)}$
Thompson Creek, Idaho: upstream of mine effluents (background for this watershed)	2.7	2.2	2.4	3133
Thompson Creek, Idaho: downstream of mine effluents	2.0	1.3	1.0	2188
Yankee Fork, Idaho: upstream of mine effluents	11.0	1.6	1.5	4250
Yankee Fork, Idaho: downstream of mine effluents	10.5	1.4	1.5	1738
McLeod and Smoky River systems, Alberta: reference streams (Deerlick Cr., Cold Cr.)	4.5	–	1.8	5000
McLeod and Smoky River systems, Alberta: mining impacted stream (Luscar Cr.)	2.6	–	2.4	299

The median selenium concentrations in water, biofilm, insects, and fish given in Table 2.4.8.2 can be used to estimate TTFs and water-particulate partition coefficients (K_d) per Equation 1. However, even a casual inspection of the K_d values shows a great range of estimates, with the higher values derived from data collected from reference sites with low selenium, and the low K_d values derived from sites with enriched selenium. This pattern is illustrated in Figure 2.4.8.2 and is reasonably consistent across a gradient of selenium concentrations. This pattern is biologically plausible for a micronutrient for which organisms attempt to regulate internal concentrations and maintain homeostasis by increasing retention when the micronutrient is scarce and increasing uptake when the micronutrient is in excess. This further suggests that risk estimates of selenium in stream food webs could be mistaken if a low K_d from a selenium enriched stream were used to estimate the assimilative capacity for a low-selenium stream using Equation 1, (over predict the amount of selenium that could safely be added). Likewise, if Equation 1 were re-arranged into Equation 2 to predict selenium concentrations in fish resulting from a given water concentration, if the water concentration of interest is much higher than reference conditions such as the

selenium CCC of 5 µg/L which is ~5 to 50X higher than reference selenium concentrations (Table 2.4.8.2), but it used with a K_d value that was derived from reference conditions, then the tissue concentrations in fish would be over-predicted, leading to an overestimation of selenium risks associated with the CCC concentration.

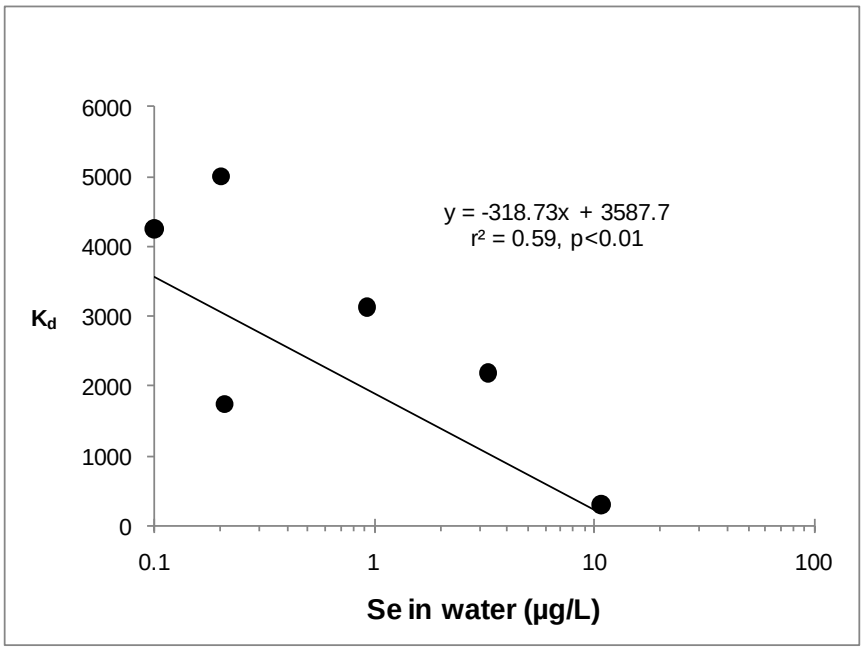


Figure 2.4.8.2. Apparent concentration dependence of selenium water-particulate partitioning coefficients (K_d) from coldwater, salmonid streams.

Based on this information NMFS estimates a concentration in water that would be expected to be transferred through the food web to a given tissue concentration. Using the juvenile Chinook salmon EC_{10} of 7.6 mg/kg dw selenium in whole bodies from Table 2.4.8.1 as the concentration of concern, with the median TTFs and partition coefficient K_d from Thompson Creek, predicts that this concentration in fish could be reached with a selenium water concentration (C_{water}) of about 1.2 µg/L. Median values for Thompson Creek were used in this example because this stream has moderately elevated selenium concentrations that are associated with tissue residues in fish near the Chinook salmon EC_{10} concentration. If the Chinook salmon EC_{10} was instead used with TTFs and K_d from streams with much lower selenium concentrations such as the Yankee Fork or the Alberta reference streams, that would have resulted in a very large denominator and a correspondingly very low projected concentration in water.

$$C_{water} = \frac{7.6\,(C_{fish})}{1.1(TTF_{fish})\bullet 2.2(TTF_{invertebrate})\bullet 2690(K_d)} = 0.00121\ mg/L = 1.2\ \mu g$$

However, the primary purpose of this analysis is to estimate whether a given water concentration of selenium, that is the chronic criterion concentration of 5 µg/L, is likely to lead to trophic transfer to levels in fish likely to cause adverse effects. To estimate likely tissue concentrations from a given water concentration of selenium, equation 1 may be rearranged to predict tissue residues from a given water concentration (Equation 2).

$$C_{fish} = \frac{C_{water}}{(TTF_{fish})\bullet(TTF_{invertebrate})\bullet(K_d)} \qquad \text{(Equation 2)}$$

Using the CCC of 5 µg/L with the K_d estimate of 1994 from the K_d vs. selenium in water regression (Figure 2.4.8.2), a tissue concentration of about 19.5 mg/kg dw in juvenile salmonids would be projected. Using Hamilton's 60 d growth model, this would relate to about a 50% reduction in weight (EC_{50} was 19.3 mg/kg); a 10% reduction in length, and about a 25% reduction in survival.

Similarly, using Equation 2 iteratively to "titrate" down from a severe effects concentration to a low-effects concentration, a selenium concentration in water of about 2 µg/L with a K_d appropriate for that enriched concentration (average of Thompson Creek segments downstream of mine discharges and Yankee Fork downstream of mine effluent) projects a whole-body tissue concentration for stream resident salmonids of about 7.7 mg/kg dw (Equation 3). Although it was estimated independently, this projected tissue concentration is very close to the low-effects whole-body tissue residue of 7.6 mg/kg dw used to evaluate the protectiveness of the 5 µg/L water chronic criterion.

$$C_{fish} = \frac{0.002\ mg/L(C_{water})}{1.0(TTF_{fish})\bullet 2.0(TTF_{invertebrate})\bullet 1963 L/kg(K_d)} = 7.7\ mg/kg \qquad \text{(Equation 3)}$$

The calculated 2 µg/L low risk water concentration corresponds with recommendations of Lemly and Skorupa (2007) for implementing proposed tissue residue-based selenium water quality criteria in a stepped fashion where for waters less than 2 µg/L selenium, dischargers need not be burdened with fish monitoring requirements. The vast majority of streams in the action area have waterborne selenium concentrations <2 µg/L (Table 2.4.8.2). Above 2 µg/L in water, fish tissue monitoring would then be needed to evaluate if selenium was being transferred through the food web to greater than tissue-residue concentrations of concern (7.6 mg/kg dw). If tissue concentrations in fish in a stream influenced by elevated selenium concentrations from point source discharges or non-point sources are greater than this tissue-residue concentration of concern, then actions to reduce anthropogenic selenium loading to the water bodies are presumed necessary. However, it is conceivable that additional site-specific information could indicate that even if tissue residue concentrations in juvenile salmonids exceed 7.6 mg/kg dw, these concentrations are unlikely to be causing adverse effects. This is because in the Yankee Fork and Thompson Creek examples, tissue residue concentrations collected at upstream background

sites were elevated to concentrations only slightly lower than this value (Table 2.4.8.2), and as of 2012, salmonid abundances in mining-influenced sections of Thompson Creek showed no obvious declines that could be attributed to elevated selenium concentrations (Janz *et al*. 2010; GEI 2013).

2.4.8.3. Summary for Selenium

If water concentrations were near the chronic selenium criterion of 5 µg/L indefinitely, selenium would likely be transferred through the food web resulting in selenium concentrations in juvenile salmonids greater than twice as high as a concentration estimated to be low risk for appreciable effects in juvenile salmon or steelhead (~7.6 mg/kg dw in whole bodies). Fish tissue residues resulting from stream food web transfer from a constant water concentration of about 5 µg/L were projected to exceed about 19.5 mg/kg dw in juvenile salmonids. This selenium tissue burden would be projected to result in growth reductions and increased mortality in juvenile anadromous salmonids, on the order of about a 50% reduction in weight, a 10% reduction in length, and about a 25% reduction in survival. Lesser reductions in growth (e.g., a 7.5% reduction) were projected to appreciably increase extinction risks and delay recovery in a modeled Chinook salmon population (Mebane and Arthaud 2010). While their modeling was specific to a Snake River spring/summer Chinook salmon populations from the upper Salmon River, NMFS assumes that the relations between size and survival during downstream migration would also hold for steelhead and sockeye salmon.

2.4.9. The Effects of EPA Approval of the Silver Criteria

Silver, in the free ion form, has been noted to be 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).

2.4.9.1. Species Effects of Silver Criteria

Aquatic life criteria for silver are complicated by the fact that the NTR criteria (EPA 1992) does not follow either of EPA's two published criteria documents for silver (EPA 1980a, 1987b). No attribution for criteria values were given in the NTR. The Idaho hardness-adjusted acute values for the action (Table 1.3.1) match the hardness-adjusted acute values in EPA (1980o), however the chronic value from EPA (1980o) was not used in the NTR nor in Idaho's subsequent water quality criteria. The 1987 criteria version concluded that silver toxicity was affected by chloride speciation, but that hardness was a less important modifier of toxicity. The 1987 criteria retained

the 1980 fixed chronic concentration of 0.12 µg/L, but replaced the 1980 hardness-adjusted acute criterion with a fixed acute criterion concentration of 0.92 µg/L (EPA 1987b). No explanation was provided in the NTR why EPA went back to their older acute criterion or why the chronic criterion was omitted (EPA 1992).

Acute Silver Criterion. Most studies of acute toxicity have used silver nitrate as the test solution, which is highly soluble and is the most toxic form of silver. Hogstrand and Wood (1998) pointed out that because of the strong modifying influence of naturally occurring ligands in ambient waters on silver toxicity (see below), the likelihood is significantly reduced that dissolved silver concentrations approach levels needed to cause acute toxicity. However, regardless of form, at hardness levels of 200 mg/L and less, considered relevant to the action area, the acute silver criterion is sufficiently low to prevent lethality (Figure 2.4.9.1).

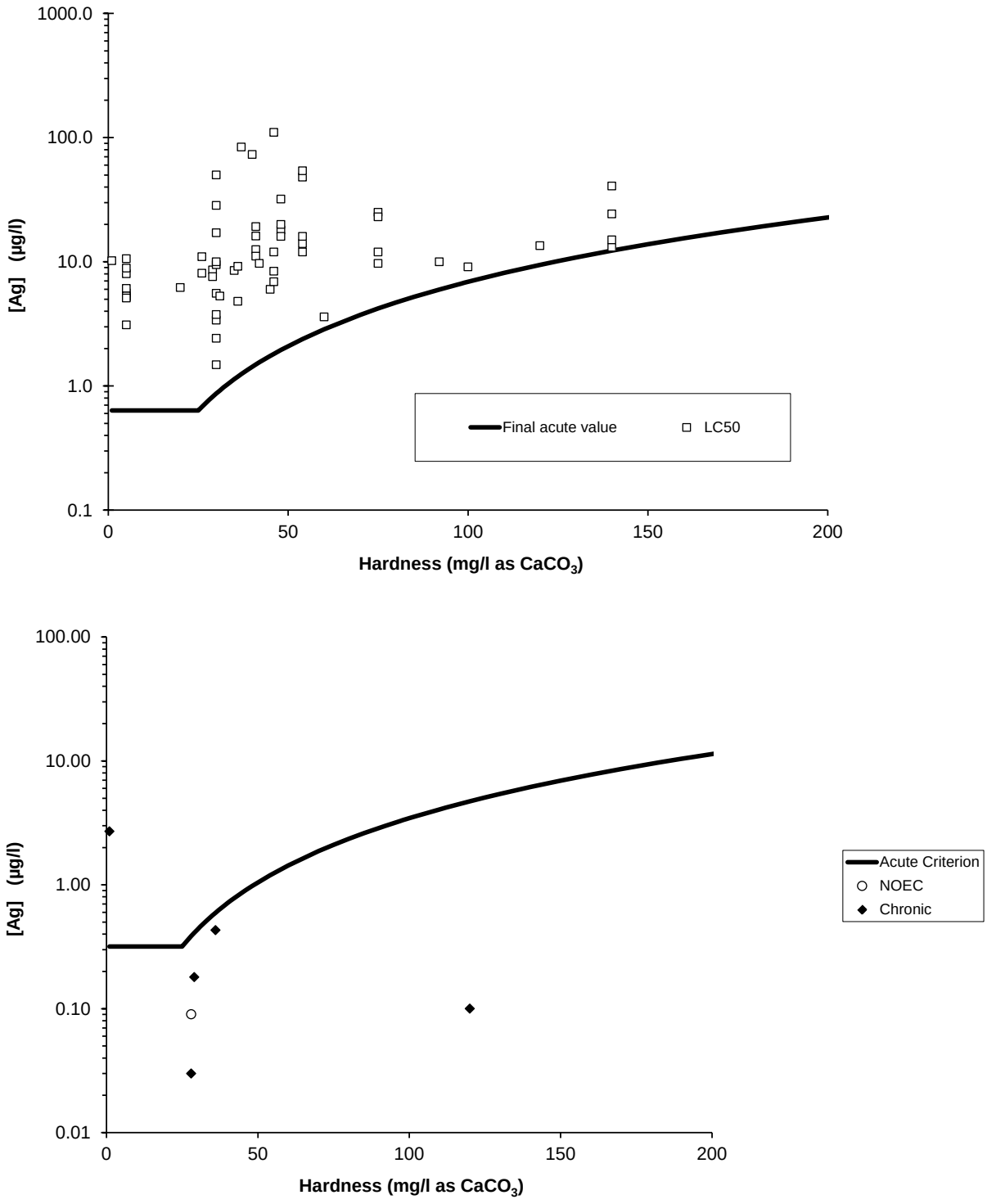


Figure 2.4.9.1. Acute silver criterion in comparison with acute and chronic silver effects data

Chronic Silver Criterion. Chronic criteria for silver are presented in the AWQC documents (EPA 1987b), and EPA (1980o), and these concluded that the "available data indicate that chronic toxicity to freshwater aquatic life may occur at concentrations as low as 0.12 µg/L." However, no chronic silver criterion was included in the National Toxics Rule (EPA 1992) nor in the proposed action. No explanation for this omission was given in EPA 1992, other than "*with this rule, EPA is promulgating its 1980 criteria for silver, because the Agency believes the criteria is protective and within the acceptable range based on uncertainties associated with deriving water quality criteri*a" (EPA 1992, p. 60883). However, although the word "criteria" is plural, there was only an acute criterion and no chronic criterion proposed for approval in the current action. There are sufficient data available that indicate that the acute criterion, which effectively acts as a chronic criterion, does not avoid chronic toxicity that has been determined to occur at concentrations below the acute criterion:

> 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) concluded that the maximum acceptable toxicant concentration 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 absence of a chronic silver criterion implies potential mortality at acute criteria concentrations to listed salmonids based on the data and information reviewed here.

Hardness and Other Parameters as Predictors of Silver Toxicity. The acute and chronic toxicities of silver are influenced by hardness, chloride ion, DOC, sulfide, and thiosulfide concentrations, and with pH and alkalinity (Hogstrand and Wood 1998; Erickson *et al*. 1998). For example, Karen *et al*. (1999) determined that increasing hardness from 30 mg/l to 60 mg/l resulted in significantly reducing silver nitrate toxicity. However, it has been shown that hardness is not as important an influence on silver toxicity as was originally thought and is secondary to other water quality constituents. Specifically, chloride ion and DOC concentrations have a significantly greater influence on toxicity (Galvez and Wood 1997; Hogstrand and Wood 1998; Bury *et al*. 1999b; Karen *et al*. 1999; Wood *et al*. 1999). The presence of chloride ion is protective because silver chloride precipitates out of solution readily, although under certain conditions it is possible to observe the formation of the dissolved $AgCl^0$ complex (Erickson *et al*. 1998). Bury *et al*. (1999a, 1999b) determined that chloride and DOC concentrations ameliorated the silver ion inhibition of Na^+ influx and gill Na^+/K^+-ATPase activity in rainbow trout. Toxicity of silver was found to change very slowly with hardness, where a hundredfold increase in hardness resulted in reducing toxicity only by roughly 50% (Bury *et al*. 1999b) and increased survival time approximately 10 fold (Galvez and Wood 1997). In contrast, only a twofold increase in chloride ion was required to produce toxic effects similar to a hundredfold increase in hardness (Galvez and Wood 1997). Karen *et al*. (1999) observed that DOC was more important than hardness for predicting the toxicity of ionic silver in natural waters to rainbow trout, fathead minnows and *Daphnia magna*. The DOC greatly reduced gill accumulations of silver through complexation. Chloride ion did not reduce gill accumulations of silver because it bound with

free silver (Ag^+) and accumulated in gills as silver chloride, but reduced toxicity because the silver chloride did not enter cells and disrupt ionoregulation.

A key point from the environmental chemistry and aquatic toxicology literature for silver is overwhelming differences in toxicity between free ionic silver and complexed silver compounds. Most laboratory toxicity tests with silver used silver nitrate because it readily disassociates into ionic silver which tends to remain in solution (Hogstrand and Wood 1998). In contrast, in rivers, streams, lakes, and effluents, ionic silver tends to be vanishingly low, and measureable silver in natural waters and effluents occurs as either silver sulfide, silver chloride, silver thiosulfate, or as complexes with natural DOC (Adams and Kramer 1999; Kramer *et al*. 1999). The differences in effects concentrations obtained between tests using silver nitrate and other forms of silver may be on the orders of magnitude. For instance, Hogstrand *et al*. (1996) obtained a 7-day LC_{50} with rainbow trout and silver nitrate of 9 µg Ag/L, but silver chloride and silver thiosulfate LC_{50}s were >100,000 µg Ag/L. Similarly, with fathead minnow, compared to free silver ion resulting from silver nitrate additions, silver chloride complexes were about 300 times less toxic and silver sulfide was at least 15,000 times less toxic (Leblanc *et al*. 1984). When very low and environmentally realistic levels of sulfide were added to a test water (0.0016 mg/L), the LC_{50} of *Daphnia magna* was increased by a factor of 5.5 (Bianchi *et al*. 2002).

2.4.9.2. Habitat Effects of Silver Criteria

Toxicity to Food Organisms. Daphnids appear to be considerably more sensitive to silver than fish, with LC_{50}s reported for cladocerans have been below the acute criterion (EPA 1987b). *Daphnia magna* tested in the absence of sulfide in water with a hardness of about 120 mg/L yielded an LC_{50} of 0.22 µg/L (Bianchi *et al*. 2002); which was 20 times lower than the acute criterion value of 4.7 for that hardness. When tested in the presence of environmentally realistic levels of sulfide, the LC_{50} was increased by about 5.5 times (Bianchi *et al.* 2002). Other invertebrate taxa serving as potential food for juvenile salmonids have been determined to experience mortality only at concentrations that are above the acute criterion, Other observed adverse effects include reductions in growth and inhibition of molting (EPA 1987b; Eisler 1996; Call *et al*. 1999). Reduced growth in mayfly larvae occurred at 2.2 µg/L in hardness 49 mg/L water (Diamond *et al.* 1992), which is greater than the acute criterion of 1.1 µg/L for that hardness. Chronic effects appear to be documented only for daphnids when silver concentrations are below the EPA (1987b) acute 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 salmon and steelhead to accumulate harmful silver concentrations through this exposure pathway (Hirsch 1998a, b).

The proposed silver criteria appear to be protective of salmonid food sources under most circumstances. Adverse effects of the silver criterion to the food organisms of listed salmon and steelhead may be potentially meaningful only when daphnids are a primary food source (e.g., downstream of an impoundment in an otherwise oligotrophic system).

Bioaccumulation. 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).

2.4.9.3. Summary for Silver

In natural waters silver is likely much less toxic than in most published laboratory experiments because of the strong modifying influence of naturally occurring ligands in ambient waters. Because of this, it appears unlikely that acute toxicity to salmonids at criterion concentrations will occur.

Unlike other criteria considered in this Opinion that all had two part values to protect against short-term and indefinite exposures, for silver only a short-term (acute) criterion is proposed. However, adverse chronic effects, including premature hatching, growth inhibition, and chronic mortality, have been observed at in laboratory settings at concentrations below the proposed single silver criterion. Thus, using a single criterion value that was derived using short-term toxicity data to also protect aquatic life from indefinite exposures may be under-protective. The acute criterion is derived as a function of hardness, which is not supported by more current literature which shows chloride, DOC, and sulfide to be more important factors in mitigating silver toxicity. The potential inadequacies and underprotectiveness of the silver criterion are mitigated by the fact that in the environment, silver occurs in a less toxic form than that used in most of the toxicity tests published in the literature. Significant food chain biomagnification by fish is also possible, but all of these effects appear unlikely to occur because of the low silver concentrations typically encountered in the aquatic environment.

2.4.10. The Effects of EPA Approval of the Zinc Criteria

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). There is evidence that zinc may be more toxic to fish at cold winter-like temperatures than at warmer, summer-like temperatures (Hodson and Sprague 1975).

2.4.10.1. Species Effects of Zinc Criteria

Zinc criteria are hardness dependent. At hardness of 100 mg/L, the acute and chronic criterion are 114 and 105 respectively whereas at hardness 25 mg/L, the criteria are 35 and 32 µg/L zinc. The criteria equations are given a floor at hardness 25 mg/L. In effect, the "floor" at hardness 25 mg/L is an implicit assumption that the general relation of zinc being more toxic at lower hardness only holds to a hardness of 25 mg/L and at hardnesses lower than 25 mg/L, zinc is no more toxic than at 25 mg/L.

Acute Zinc Criterion. Toxicity test data indicate that in most instances, the zinc acute criterion concentrations are unlikely to kill juvenile salmonids (Figure 2.4.10.1). Most studies have found toxic effects at concentrations greater than the proposed criterion for adult and early life stages of salmonids (for example, studies reported in EPA 1987c; Chapman 1978a, 1978b; Chapman and Stevens 1978; Stubblefield *et al*. 1999). Two studies were identified where LC_{50} concentrations were less than the FAV for Zn:

> (Mebane *et al*. 2012) reported LC_{50}s for rainbow trout from five of 18 tests that were less than the final acute value for zinc. Two of the tests with effects at zinc concentrations less than the criteria resulted from testing in soft water with hardness less than the Idaho 25 mg/L "hardness floor." Other tests conducted at higher hardnesses had variable results, which were apparently related to some life stages being more sensitive than others.
>
> Hansen *et al*. (2002c) and Stratus (1999) determined 120- and 96-hour LC_{50}s for rainbow and bull trout fry that were below both the acute and chronic criteria in low hardness water (shown as several points in a vertical line all at 30 mg/L as $CaCO_3$; Figure 2.4.10.1). (Both references are to the same study, but the 96-hour toxicity values which are used here for comparisons were only reported in the 1999 source; 96- and 120-hour values were similar)

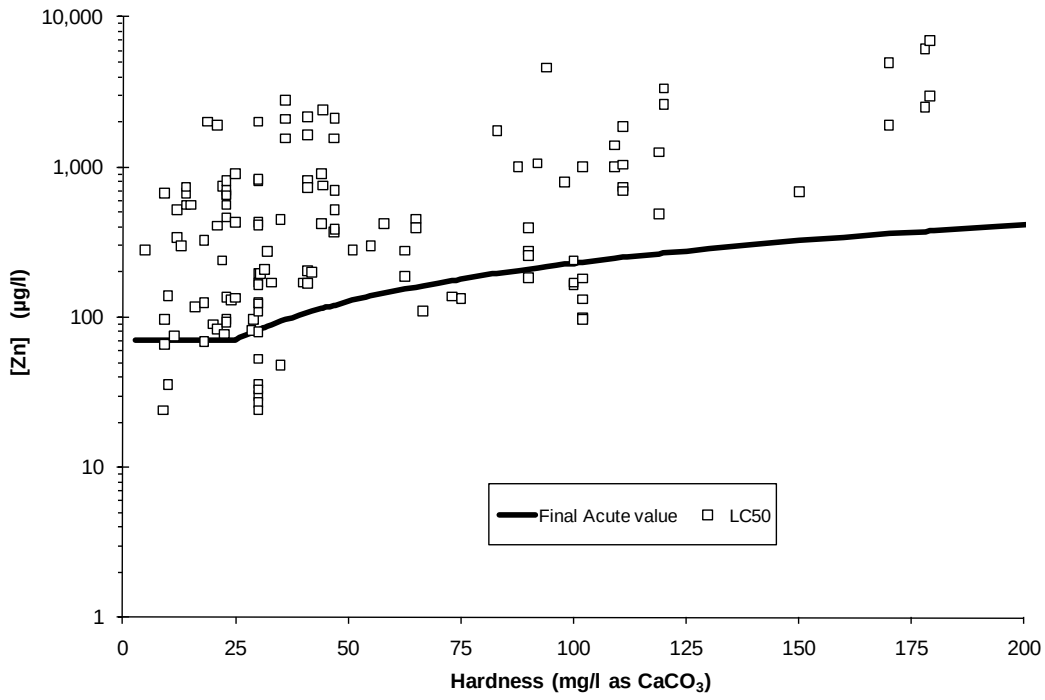


Figure 2.4.10.1. Comparison of reviewed 96-hour LC_{50}s for salmonids with zinc and the Idaho criterion final acute values (FAV), calculated for hardnesses up to 200 mg/L as $CaC0_3$. LC_{50}s limited to species within the genera *Oncorhynchus, Salvelinus,* and *Salmo.* If LC_{50} values fell above the FAV line, that would suggest few if any mortalities would be likely at criterion concentrations.

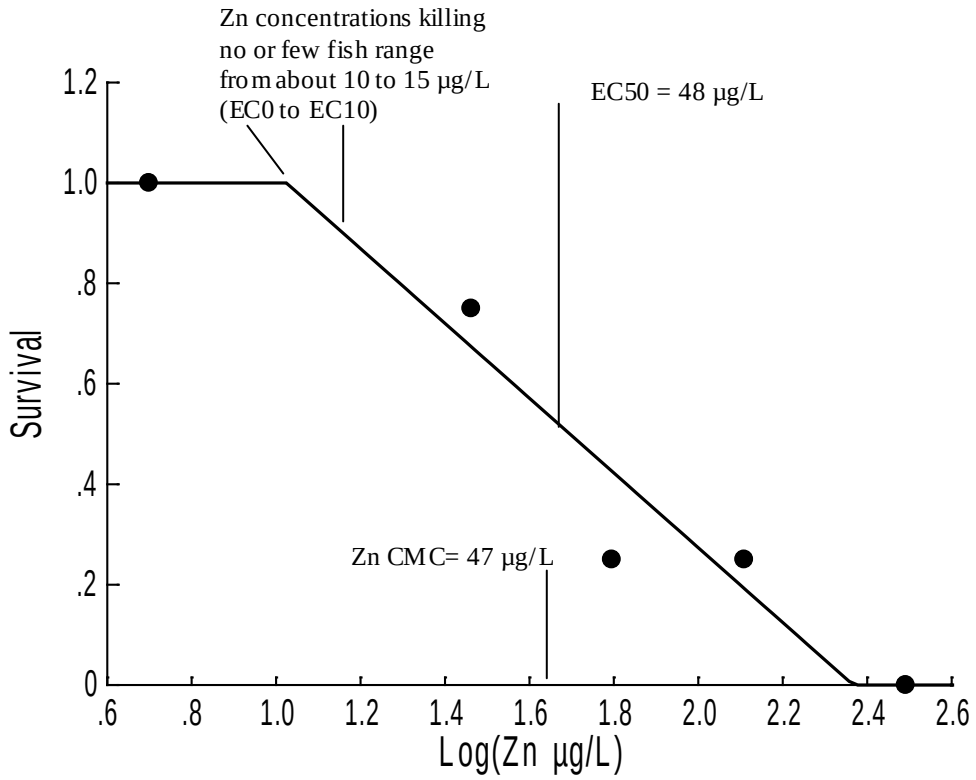


Figure 2.4.10.2. Example of a 96-hour toxicity test with rainbow trout in which zinc at its acute criterion concentration (CMC) killed about half of the fish tested. At the CMC, few if any fish are supposed to be killed. In this instance, the final acute value that the criterion was based on (i.e., the LC_{50} for a hypothetical organism more sensitive than 95% of organisms) was twice as high as the rainbow trout value.) Rainbow trout data from Mebane *et al*. (2012), test hardness 35 mg/L, 0.5g fish, wet wt.

Chronic Zinc Criterion. The proposed chronic criterion is only approximately 10% less than the acute criterion. The similarity of the chronic and acute criteria may not be of great concern with respect to listed salmon and steelhead because fish have naturally elevated zinc levels in their tissues, are able to regulate tissue zinc concentrations over a range of environmental exposure levels, and exhibit increased resistance to elevated zinc levels with acclimation (Sorensen 1991; Eisler 1993). Acclimation likely explains why some chronic tests values are higher than acute values, because chronic tests conducted as ELS exposures begin the exposures during the metals-resistant egg or alevin life stage (Chapman 1985). If during this resistant stage, the fish acclimate to zinc, when they grow older and enter the more sensitive fry stages they may be more resistant than fish that were initially exposed as fry. For instance, Mebane *et al*. (2008) reported a 68-day zinc LC_{50} of 367 µg/L for rainbow trout compared to a 4-day zinc LC_{50} of 130 µg/L, tested in the same dilution water. In this study, the 68-day test began with the egg life stage where whereas the 4-day test began with fry (Mebane *et al*. 2008). Similarly, Brinkman and Hansen (2004) obtained nearly identical 4-day and 30-day LC_{50}s with rainbow trout and zinc when exposures began with fry, but longer ELS exposures that began using eggs yielded much higher (more resistant) values. Chapman (1978b) exposed sockeye salmon to zinc for 21 months beginning with a 3-month adult exposure followed by an 18-mo exposure of embryonic through

smolt stages. Zinc concentrations up to 242 µg/L produced no adverse effects on survival, fertility, fecundity, growth, or on the subsequent survival of smolts transferred to seawater. The sockeye salmon became acclimated to zinc, as Chapman found a 2.2 fold increase in tolerance when he tested some of the zinc exposed sockeye salmon along with naïve fish (Chapman 1978b). Overall, most available data indicated that chronic toxic effects were unlikely at concentrations lower than the chronic criterion (Figure 2.4.10.3)

Acclimation is likely related to why salmonids are sometimes present and in apparent good health in some streams that greatly exceed chronic zinc criteria (Chapman 1978a, 1985; Harper *et al*. 2008). However, the information indicate that acquired protection is transient and may be lost in periods as short as 7 days upon return to toxicant-free water (Bradley *et al*. 1985; Stubblefield *et al*. 1999). Thus, even though acclimation can increase the resistance of fish to zinc by factors of around two to three, the protection by acclimation may not be lasting, and neither acclimation nor the fact that salmonids may be self-sustaining in watersheds with elevated zinc refute the demonstrated effects of zinc in fish that have no prior history of zinc exposures previous to testing.

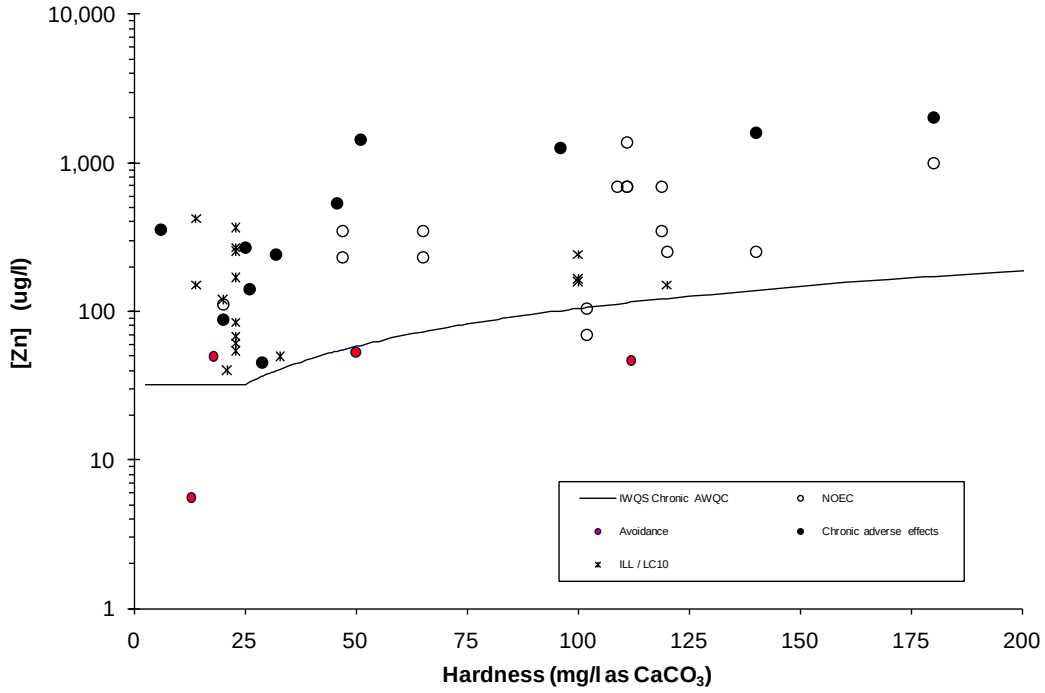


Figure 2.4.10.3. Comparison of the Idaho chronic criterion and adverse chronic or sublethal effects and estimates of no-effect concentrations to salmonids.

Behavioral Effects. Behavioral avoidance reactions have been noted to occur 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 (Black and Birge 1980). Juvenile

cutthroat trout avoidance was documented at 53 µg/L at a hardness of 50 mg/L (Woodward *et al*. 1997).

Little study of behavior effects to adult salmonids in relation to zinc has been conducted. There are insufficient and conflicting data available to identify whether these behavioral effects translate into adverse effects in the field. Sprague *et al*. (1965) and Saunders and Sprague (1967) showed that the upstream migrations of Atlantic salmon were disrupted in the Miramichi River, New Brunswick, when zinc concentrations reached about 150 to 180 µg/L and copper reached about 11 to 15 µg/L. From 1990 to1996, water hardness of the Miramichi River measured monthly averaged 10 mg/L, ranging from 4 to 19 mg/L, and DOC averaged 3.7, ranging about 0.5 to 7 mg/L (Komadina-Douthwright *et al*. 1999). Assuming that the major ion and organic carbon content of the Miramichi River do not greatly change year to year, then these values help relate the thresholds for migratory disruption reported by Saunders and Sprague (1967) to criteria values. At a water hardness of 25 mg/L, the acute zinc criterion would be 36 µg/L, which is considerably lower than the apparent migratory disruption threshold of 150 µg/L.

One study asserted that ambient zinc concentrations in an Idaho river disrupted migration of adult Chinook salmon. In an effort to monitor avoidance responses of salmonids to metals in more realistic conditions, Goldstein *et al*. (1999) monitored adult Chinook salmon movements with radio telemetry in the vicinity of the South Fork and North Fork Coeur d'Alene River confluence. Adult male Chinook salmon were captured from Wolf Lodge Creek (a tributary to Lake Coeur d'Alene) and after harvesting their milt, were trucked to the Coeur d'Alene River and released about 2 km downstream of the confluence of the South Fork and North Fork. Half of the released salmon moved upstream; of the half that moved upstream, 70% ascended the North Fork which had a zinc concentration of about 9 µg/L. Thirty percent ascended the South Fork, which had a zinc concentration of about 2200 µg/L. The authors concluded that their study demonstrated that avoidance of metals can disturb critical spawning migrations and may displace or preclude fish from preferred habitats (Goldstein *et al*. 1999). However, because migrating spawning salmon home on their natal stream by chemical imprinting, and Chinook salmon die within a few weeks after spawning, is unclear what the migratory instincts would be of post-spawning male salmon that had been trucked from their natal stream and released in a different watershed shortly before their deaths. Further, since the North and South Forks make up around 70% and 30% of the Coeur d'Alene River flows respectively, and adult salmon movements in rivers in the absence of any homing cues tend to simply follow larger flows (Anderson and Quinn 2007), the conclusion of the authors is debatable. We determined that they proved feasibility of tracking fish movements in a river, and provide guidance for conducting a more ecologically meaningful study, such as using migratory adult cutthroat prior to spawning.

Hardness as a Predictor of Zinc Toxicity. Zinc toxicity is known to vary with a number of factors other than hardness, such as pH, alkalinity, temperature, and life stage or size. In many situations, the present criteria appear to avoid harm in circumstances when zinc is the only contaminant present at elevated concentrations and perhaps when jointly elevated with cadmium. An exception exists for at least some juvenile salmonids in water with low hardness values of about 35 mg/L or less, as indicated by the results of Hansen *et al*. (2002c) and Mebane *et al*. (2012). Data reported in EPA (1987c) and the large number of other studies depicted in Figure 2.4.10.1 otherwise indicate that the criteria, expressed as a function of hardness, may often be

protective close to the hardness floor of 25 mg/L as $CaCO_3$. Calcium has been determined to reduce zinc uptake directly through both biological acclimation and chemical processes, where protection is additive in nature with increasing calcium concentration (Barron and Albeke 2000). The results of Hansen *et al*. (2002c), however, indicate that some rainbow trout, which are considered a surrogate for listed salmon and steelhead, may be killed at zinc concentrations lower than criteria, particularly as they develop to sensitive sizes during the fry stage.

2.4.10.2. Habitat Effects of Zinc Criteria

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 salmon and steelhead feed on, although in two cases listed in Table 2-1 of EPA (1987c), cladocerans 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). Working with data from streams in northern Idaho, Dillon and Mebane (2002) found that overall insect taxa richness, mayfly richness, and abundance of metals-sensitive mayflies and other taxa were generally highest at streams with low zinc concentrations. The abundance and diversity of aquatic insects generally declined with increasing zinc concentrations. No threshold of response could be determined, and declines may have begun at zinc concentrations less than the Idaho chronic zinc criteria. While these field data were "noisy," severe alterations on the order of 50% reductions in the abundance or diversity of potential prey items were not obvious at zinc concentrations less than about five times or more greater than the chronic zinc criterion. At lower zinc concentrations close to the criteria, it is not clear if lower reductions in the abundance or diversity of potential prey items would be of a magnitude to adversely affect juvenile salmon foraging ability.

Bioaccumulation. Zinc can clearly bioaccumulate in the environment. Zinc has been found to be elevated in benthic invertebrates in field studies conducted in streams with elevated zinc in sediments or water (Ingersoll *et al*. 1994; Woodward *et al*. 1994; Maret *et al*. 2003; Kiffney and Clements 1996). Farag *et al*. (1994) 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. Mount *et al*. (1994) determined that tissue concentrations increased in rainbow trout fry fed a diet containing enriched levels of zinc.

However, the issue of zinc bioaccumulation in salmonids is confounded by naturally high tissue concentrations and the ability of fish to regulate internal concentrations. Alsop *et al*. (1999) 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. Recent reviews have concluded that while zinc is bioaccumulated in the environment, there is no

evidence for biomagnification in the food chain because zinc concentrations in higher trophic levels are not higher than those in lower trophic levels (Hogstrand 2011; Cardwell *et al.* 2013).

2.4.10.3. Summary for Zinc

Zinc is primarily an acute toxin to salmonids, hence the acute criterion is of greater environmental relevance than the chronic criteria. A confusing aspect of the literature on zinc toxicity to salmonids is the great disparity in reported effects between studies. Across different studies, EC_{50} values for rainbow trout with zinc at similar test hardnesses varied by an order of magnitude. Said differently, zinc at criteria concentrations has been found to be highly toxic and killed most of the fish exposed (Figure 2.4.10.2), but in other tests, concentrations well in excess of the criteria killed no fish. This disparity may be due to differences in the sensitivity of fish at different sizes as they develop. While it is commonly assumed that the smallest organisms will be most sensitive (e.g., ASTM 1997), this is clearly not always the case with zinc. Instead for salmonids, the likely pattern is that the newly hatched, smallest fish appear resistant to zinc, lose resistance as they grow during the first and second months after hatching, and then regain resistance as the fish become older and larger. This suggests that even though most of the studies reviewed that addressed zinc toxicity to listed Snake River salmon and steelhead did not show adverse effects below criteria values (Figure 2.4.10.1 and 2.4.10.3c) the risk from exposure to zinc may have been underestemiated because the studies did not distinguish between sensitive life stages, and not examined effects to listed steelhead and salmonids at their most vulnerable post-hatch stages.

Adverse effects were found at sub-criteria values in tests conducted at hardnesses less than 25 mg/L, a few other tests at moderately low hardness of 35 mg/L with the most sensitive size fish tested (Figure 2.4.10.2), and multiple tests reported by Hansen *et al*. (2002c) with rainbow trout. The preponderance of the information reviewed indicate that in waters with hardness less than about 25 mg/L as $CaCO_3$ the Idaho Zn criteria would not be sufficiently protective of listed Snake River salmon and steelhead if they were exposed at their most sensitive life stages. If alternatively, the current IDEQ zinc criteria were determined using the actual water hardness, instead of the assumed hardness of 25 mg/L, most of those data indicate that the criteria would then be sufficient to avoid harm in most of the studies reviewed.

2.4.11. The Effects of EPA Approval of the Chromium III and VI Criteria

Chromium can exist in oxidation states from –II to +VI, but is most frequently found in the oxygenated waters in its hexavalent state (VI). The chromium (III) is oxidized to chromium (VI) and under oxygenated conditions chromium (VI) is the dominant stable species in aquatic systems. The chromium (VI) is highly soluble in water and thus mobile in the aquatic environment.

No single mechanism of impairment has been shown to be responsible for chromium toxicity in fish. The symptoms include changes in tissue histology, temporary reductions in growth, the production of reactive oxygen species, and impaired immune function (Reid 2011).

Comparison of Chromium Criteria. The criteria under review for chromium (III) are 311µg/L acute criterion and 101 µg/L chronic criterion at a hardness of 50 mg/L. Criteria for chromium (VI) are 15µg/L acute, and 10 µg/L chronic and are not hardness dependent. The chromium (III) toxicity is weakly influenced by water hardness. It is unclear if the same if true for chromium (VI), which has been considered more toxic than chromium (III) (EIFAC 1983; Eisler 1986; EPA 1985h). Hexavalent chromium (VI) exists in solution in an anionic rather than cationic form; therefore, calcium competition, one of the main reasons that hardness mitigates toxicity of some metals such as cadmium, nickel, and zinc, does not occur. The acute standards for chromium (III) are unique from analogous standards for the other metals of concern because the total recoverable to dissolved CF (0.316) is substantially smaller.

Baseline Concentrations of Chromium. Although weathering processes result in the natural mobilization of chromium, the amounts added by anthropogenic activities are thought to be far greater. Major sources are the industrial production of metal alloys, atmospheric deposition from urban and industrial centers, and large scale wrecking yards and metals recycling and reprocessing centers (Reid 2011). Because of the rural nature of the action area, transportation costs, and distance to major urban or industrial sources no growth in the business types that discharge chromium is expected in the action area.

The few data on chromium concentrations in Idaho that were located were low relative to aquatic risk concentrations. In the Stibnite Mining District in the EFSFSR basin, total chromium concentrations collected under low flow conditions in September 2011 ranged from <0.2 µg/L to 0.24 µg/L (*http://waterdata.usgs.gov/nwis*, HUC 17060208). In the Blackbird Mining District, concentration of chromium in seeps and adits around the Blackbird Mine were not higher than average background filtered surface water concentrations near the Blackbird Site (<2.9 µg/L) (Beltman and others 1993).

2.4.11.1. Species Effects of Chromium Criteria

There are more toxicity test data available for chromium (VI) than chromium (III). Toxicity tests on salmonid species indicate that adverse effects do not occur to any life stage of salmonids when exposed to ambient dissolved concentrations at or below the chromium (VI) criteria. This includes the results reported by Birge *et al*. (1978, 1981) and Sauter *et al*. (1976) regarding early lifestage survival. We identified only one study of trivalent chromium toxicity to salmonids, and in this test adverse effects were observed at a concentration that was higher than the chronic criterion (LOEC 48 µg/L vs. the chronic criterion of 24 µg/L). The magnitude of effects at these treatments was fairly slight, with 4% reduction in length of ELS fish after 30 days exposures (Stevens and Chapman 1984).

Patton *et al*. (2007) reported that the survival, development, and growth of early life stage fall Chinook salmon were not adversely affected by extended exposures (i.e., 98 day) to hexavalent chromium ranging from 0.79 to 260 µg/L.

Conflicting results have been obtained from fertilization tests of salmonids under exposures to chromium (VI). Billard and Roubaud (1985) determined that the viability of rainbow trout

sperm (but not ova) was adversely affected when exposed directly to a total chromium concentration equal to 5 µg/L, which is below the chronic criterion. Yet Farag *et al*. (2006) found that total chromium concentration ranging from 11 to 266 µg/L or to a chromium (VI) concentration of 130 µg/L did not affect the fertilization process of Chinook salmon or cutthroat trout. Farag *et al*. (2006) suggested that the differences might be because of different species tested, but because cutthroat and rainbow trout are so closely related, the differences seem more likely from the different methodologies used. The time allowed for exposure to chromium during fertilization was 1 minute during Farag *et al*.'s more recent study versus 15 minutes for the study conducted by Billard and Roubard (1985). The shorter time used by Farag *et al*. (2006) more closely mimicked fertilization events that may occur under river conditions where velocities of the water at the substrate are fast and motility of sperm is short-lived. Also, Farag *et al*. (2006) reported that the ova were held in exposure water for 1.5 hours of water hardening after fertilization to more closely mimic natural conditions in which eggs continue to absorb water for approximately 1.5 hours after fertilization. The ova were not exposed to chromium during water hardening in the study performed by Billard and Roubard (1985). Farag *et al.* (2006) concluded that the instantaneous nature of fertilization likely limits the potential effects of chromium on fertilization success. Neither Billard and Roubard (1985) or Farag *et al*. (2006) analyzed chromium speciation for most treatments, but in these oxygenated tests the chromium is expected to be present as chromium (VI) (Reid 2011).

The conflicting results of the Billard and Roubard (1985) and the Farag *et al*. (2006) studies do result in some uncertainty; however, the latter study by Farag *et al*. (2006) seems more persuasive. Thus, the current chronic chromium (VI) criterion of 10 µg/L is likely protective of Chinook salmon fertilization, based on the instantaneous nature of fertilization limiting effects.

Behavioral Effects. Anestis and Neufeld (1986) studied avoidance behavior of juvenile rainbow trout exposed to chromium (VI) and determined a threshold concentration for non-acclimated fish that was equal to 28 µg/L, which is above the acute AWQC. Fish that were acclimated to elevated levels of chromium (VI) required higher concentrations to elicit an observable effect. Dauble *et al*. (2001) describe laboratory avoidance/preference tests that showed that juvenile chinook salmon can detect and avoid chromium at concentrations >=54 µg/L under conditions of 80 mg/L hardness. Thus, there is no evidence of altered behavior in salmonids exposed to chromium (VI) concentrations below either the acute or chronic criterion. We did not locate similar data for chromium (III).

2.4.11.2. Habitat Effects of Chromium Criteria

Toxicity to Food Organisms. The available data suggest that chromium VI may be much more toxic to some aquatic invertebrates than to fish. NMFS did not locate any chronic tests with aquatic insects, but chronic and some acute tests with cladocerans and amphipods were very sensitive, with adverse effects noted at concentrations below the criteria.

The chromium (VI) tested as sodium dichromate chromate, was extremely toxic to the amphipod *Hyalella azteca* in 7-day tests with a LC_{50} of 3.1 µg/L (Borgmann and others 2005a). Hyalella azteca were also found to be highly sensitive to chromium (VI) by Besser *et al*. (2004). They reported the threshold for chromium (VI) toxicity to *H. azteca*, was between 10 and 18 µg/L, with a NOEC of 10 µg/l which is the same as the chronic criterion concentration (Besser and others 2004). Cladocerans have been reported to experience acute and chronic effects at concentrations below the acute and chronic criteria, respectively, for both chromium (III) and (VI). Data in EPA (1985h) 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 and equal to the chronic criterion for each respective valency. Most studies, however, have determined toxicity to daphnids occurs at higher concentrations than the criterion.

Data summarized in EPA (1985h), EIFAC (1983), and Eisler (1986) suggest that other invertebrate taxa that juvenile salmonids may feed on generally experience mortality at chromium (III) and (VI) concentrations that are well above the acute criterion. More recently, Canivet *et al*. (2001) obtained 240-hour chromium (VI) LC_{50}s for larvae of a trichopteran and an ephemeropteran that were well above the acute and chronic criteria.

Salmonid food items appear to be unimpacted by chromium at criteria concentrations under most circumstances. The proposed criteria may only be harmful to food organisms of listed salmon and steelhead if daphnids or amphipods are the primary food source (e.g., downstream of an impoundment in an otherwise oligotrophic system).

Bioaccumulation. There is evidence that invertebrates and salmonids 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 dw were presumptive evidence of chromium contamination, but the biological significance was not clear. Little is known regarding bioaccumulation at concentrations that are below the chronic criteria.

2.4.11.3. Summary for Chromium

Data reviewed by NMFS indicate few direct adverse effects to listed salmonids at concentrations less than the chronic trivalent or hexavalent chromium criteria. Studies on the effects of hexavalent chromium to salmon sperm are contradictory with one test indicating it is toxic at concentrations below the chronic criteria, and a more recent study showing no effects at criteria

concentrations. Because the more recent study that showed no effects appeared to use a more relevant exposure duration, NMFS find it to be more relieable and concludes that direct adverse effects of chromium to listed salmonids are unlikely at or below criteria.

The amphipod *Hyalella azteca* suffered adverse effects at a test concentration below the chronic criterion in one study but not in another. Because so few data on long-term effects of chromium to benthic invertebrates are available, this test is interpreted as suggesting adverse effects to food sources are possible. Bioaccumulation of chromium clearly occurs when water concentrations are high, but relevant data are absent regarding the effects to salmonids when water-borne concentrations are below the chronic criterion. Because adverse effects to the species or critical habitat should never reach the scale where take occurs, the effects of the proposed action for chromium are very minor.

2.4.12. The Effects of EPA Approval of the Lead Criteria

The acute lead (Pb) criterion proposed for approval is 65 µg/L, and the proposed chronic criterion is 2.5 µg/L, as dissolved (filtered) metals at a hardness of 100 mg/L.

Baseline concentrations of lead. In natural waters, lead is usually complexed with particulate matter resulting in much lower dissolved than total concentrations (Mager 2011). For instance, in the lead contaminated Coeur d'Alene River of northern Idaho, dissolved lead concentrations rarely exceed 20 µg/L whereas total concentrations often exceed 100 µg/L. A maximum dissolved lead concentration of 420 µg/L was reported for this location (Clark 2002; Balistrieri and Blank 2008). The Coeur d'Alene River is north of occupied habitat, as is the Clark Fork River, Idaho, where up to 60 µg/L dissolved lead has been reported (Hardy and others 2005). Within the action area, reliable lead data are sparse but the measured concentrations are quite low. The highest lead concentration obtained by the Idaho IDEQ/USGS statewide monitoring program within the action area was from the Hells Canyon reach of Snake River near Anatone, Washington (7 µg/L). All other measurements from within the Clearwater and Salmon River basins and the Snake River downstream of Hells Canyon dam were <1 µg/L (Hardy and others 2005). Mebane (2000) reported lead concentrations in the upper Salmon River near the TCM as high as 2 µg/L, but most values were <0.2 µg/L.

2.4.12.1. Species Effects of Lead Criteria

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 to salmonids and other fish including: spinal curvature and other deformities; anemia; caudal chromatophore degeneration (black tail); caudal fin degeneration; destruction of spinal neurons; aminolevulinic acid dehydratase 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; Mager 2011). 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 in aqueous environments to bed sediments (Eisler 1988b; Sorensen 1991).

Acute Lead Criterion. Available data suggest that toxic effects of lead on salmonids occur above the proposed acute and chronic criteria concentrations. However, the data exhibit wide variation (Figure 2.4.12.1), 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 have been found to be more sensitive to lead in terms of effects to development than older life stages (Sorenson 1991, Mebane *et al*. 2008).

Chronic Lead Criterion. We identified several studies that indicate the chronic criterion is at or below the NOEC level for the early life stage (Figure 2.4.12.2). For example, Sauter *et al*. (1976) determined that the threshold for adverse chronic effects to rainbow trout eggs and fry occurred at a lead concentration between 71 µg/L and 146 µg/L, both of which are well 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 above the proposed chronic criterion. When the eggs were not exposed, effects to sac-fry were found 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 results of Birge *et al*. (1978; 1981) indicated that rainbow trout embryos exposed for more than 4 days can begin to die when lead concentrations are between 2.5 µg/L and 10.3 µg/L, and hardness is 100 mg/L as $CaCO_3$. In contrast, Mebane *et al*. (2008) exposed rainbow trout embryos to lead in low hardness water (20 mg/L) for about 10 days but only noted mortalities at much higher exposures (≥54 µg/L Pb) than did Birge *et al*. (1978; 1981).

Organic forms of lead appear to be much more toxic than inorganic forms, but are not addressed in the proposed criteria. Wong *et al*'s (1981; experiment 1) data indicated a 7-hour LC_{10} of approximately 3.5 µg/L of tetramethyl lead for rainbow trout fry (at hardness of 135 mg/l) and a time-independent LC_{50} (incipient lethal level, ILL) of approximately 24 µg/L for juveniles. A series of field and laboratory tests with brook trout that had been exposed to environmentally relevant concentrations of lead from combustion of leaded fuel had contrasting results to those of Wong (1981). Trout exposed for 3 weeks in melted snow that was contaminated with lead from snowmobile exhaust showed increased bioaccumulation of lead and decreased swimming stamina at waterborne concentrations as low as 12.5 µg/L dissolved lead (Adams 1975).

Table 2.4.12.1. Comparison of the most sensitive chronic endpoints (in µg/L, except hardness in mg/L) from relevant studies with dissolved lead and salmonids or benthic invertebrates (i.e., potential prey) for related species

Species, test type, Endpoint	Duration	Hardness	NOEC	LOEC	MATC	EC10	EC20	Idaho CCC	Note
Mayfly, *Baetis tricaudatus*, EIL-s	10 d	20	103	160	130	37	66	0.54	1
Midge, *Chironomus dilutus*, LC-g	55 d	32	57	75	65	15	28	0.71	1
Midge, EIL-s	27 d	48	109	497	233	108	149	1.1	2
Snail, *Lymnaea stagnalis*, JGS-g	30 d	102	12	16	14	~1	<~2	**2.6**	2
Amphipod, *Hyalella azteca*, LC	42	138	6.3	16	10		2.8 (EC25)	**3.6**	3
Daphnid, *Ceriodaphnia dubia*	7 d	20	51	99	71	nc	nc	0.54	4
Rainbow trout, ELS-s	69 d	20	24	54	36	26	34	0.54	1
Rainbow trout ELS-g	62 d	29	8	18	12	7	>87	0.64	1
Rainbow trout ELS-g	60 d	35	71	146	102	79	99	0.75	5
Rainbow trout ELS-d	1.6 yr	28	nc	nc	nc	8.8	10.5	0.61	6
Rainbow trout JGS-d	1.5 yr	28	nc	nc	nc	8.2	10.5	0.61	6
Brook trout, *Salvelinus fontinalis*, LC-d,g	3 y	44	39	84	57	nc	nc	1.0	7
Fathead minnow, *Pimephales promelas*	30 d	19	17	62	32	nc	21.6	0.54	8

Table abbreviations: d – days, y – years; nc – not calculable, either because treatments were not replicated precluding statistical NOEC/LOECs; inadequate partial responses occurred or because treatment responses were not reported; *Test type*: EIL – early instar larval test; ELS – early-life stage test; JGS –juvenile growth and survival test; LC – life cycle test; *Most sensitive endpoint responses*: s – survival, g – growth, d – spinal deformity, b – biomass, u – unknown. Hardness in mg/L as $CaCO_3$. To improve comparability, 10th percentile effects concentrations (EC10) were calculated

Numbered table notes: 1. Mebane *et al*. 2008*;* 2. Estimated from data graph in Grosell *et al*. (2006a); 3. Besser *et al*. (2005); 4. Jop *et al*. (1995); 5. Sauter *et al*. (1976); 6. Davies *et al*. (1976); 7. Holcombe *et al*. (1976); 8. Grosell *et al*. (2006b); 9. Davies *et al*. (1993)

Hardness as a Predictor of Lead Toxicity. Water hardness is an important influence on inorganic lead toxicity because lead precipitates out of solution as hardness increases. Lead begins to precipitate when hardness reaches 27 mg/L, and toxicity declines significantly as hardness approaches about 50 mg/L (Sorensen 1991). This response is modified somewhat by variation in pH, but hardness appears to be a primary control on lead bioavailability and toxicity. It is unresolved whether lead precipitation in waters with hardness greater than 27 mg/L to 50 mg/L would be associated with adverse effects to aquatic macroinvertebrates, or to the incubation and overwintering life stages of listed salmonids.

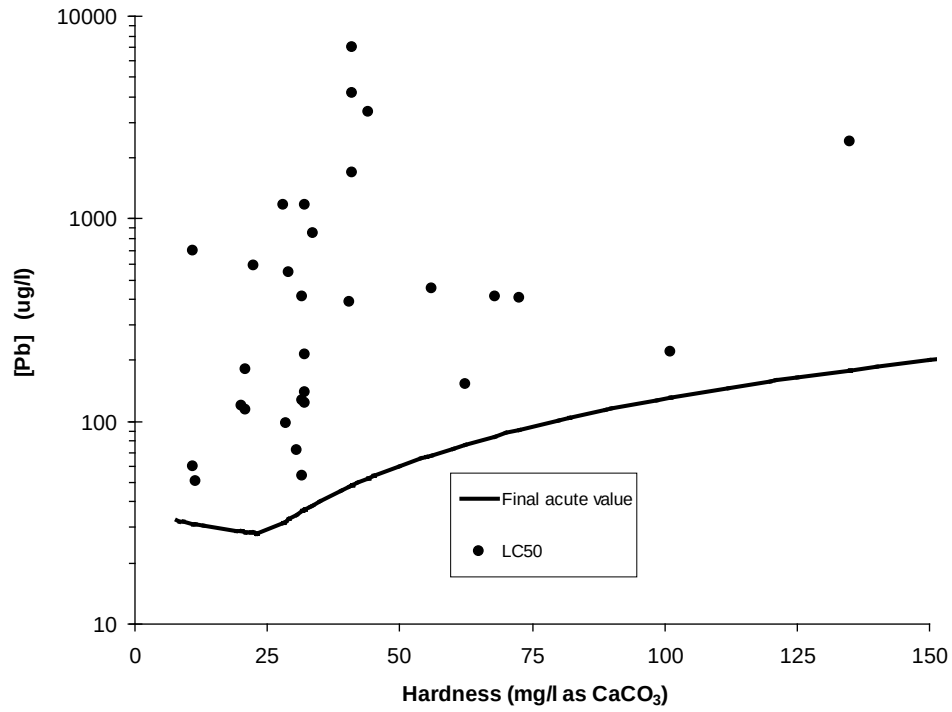


Figure 2.4.12.1. Acute LC_{50}s with salmonids, any life stage vs. the Idaho final acute value for lead.

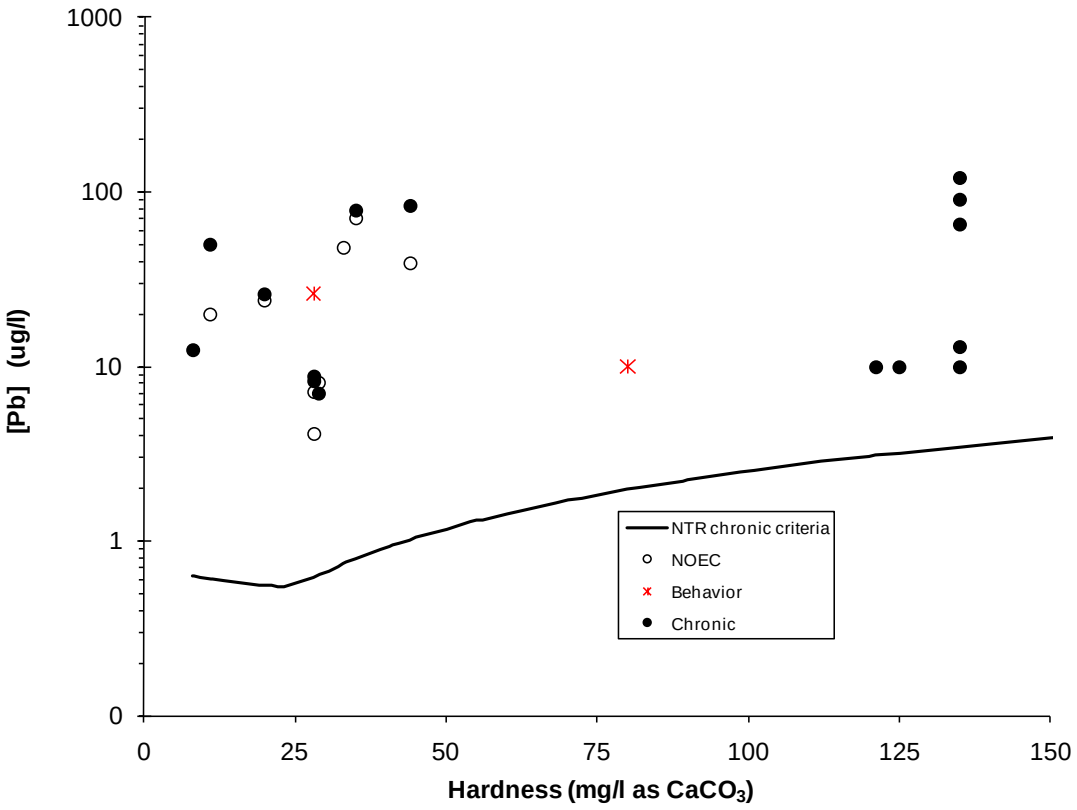


Figure 2.4.12.2. Chronic effects, no-effects, and avoidance concentrations of lead with salmonids vs. the Idaho chronic criterion concentrations for lead.

2.4.12.2. Habitat Effects of Lead Criteria

Toxicity to Food Organisms. Lead toxicity varies considerably among aquatic macroinvertebrates. 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. Some snail taxa are exceptionally sensitive to lead and suffer reduced growth and mortality at lead concentrations well below the chronic criterion. Amphipods may also be quite sensitive to chronic lead exposures compared to other organisms. The approximate thresholds for adverse effects to the amphipod *Hyalella azteca* were lower than the chronic criterion. The chronic effect threshold for a mayfly and a midge were well above the chronic criterion (Table 1.12.2.1). Salmonids are opportunistic feeders and when snails are abundant in stream, they have sometimes been important food items in salmonid diets (McGrath and Lewis 2007; NCASI 1989). However, less armored prey such as mayflies, midges, aquatic insects and amphipods would be preferred prey items for juvenile salmonids in streams if they are abundant (e.g., Karchesky and Bennett 1999; Muir and Coley 1996; Rader 1997; Sagar and Glova 1987; Suttle *et al*. 2004).

Much data on the acute toxicity of lead to coldwater stream invertebrates under conditions of low hardness and low organic carbon that are representative of much of the Clearwater and Salmon

River habitats in Idaho has recently become available. Testing included several species of mayfly, stonefly, caddisfly, true flies, and snails (Mebane *et al.* 2012). The data indicate that acute mortality of the more sensitive taxa occurred at concentrations that are well above the final acute value for lead. However, whether data from this type of short-term, water-only acute tests with aquatic insects to lead and other metals have any relevance to risks of lead to aquatic insects in nature has been challenged (Buchwalter *et al*. 2007).

Ingersoll *et al*. (1994) determined that while the amphipod *Hyalella azteca* accumulated lead from bed sediments, the level of accumulation was not related to the 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.

Bioaccumulation. 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 accumulate lead from water or diet but the effects of lead tissue residues is uncertain. 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 AWQC. Other waterborne or dietary lead exposures and field studies have also shown bioaccumulation, but showed few obvious adverse effects at concentrations near chronic criterion (Adams 1975; Davies *et al*. 1976; Holcombe *et al*. 1976; Mount *et al*. 1994; Farag *et al*. 1999; Erickson *et al*. 2010).

2.4.12.3. Summary for Lead

Potential adverse effects from exposure to lead at concentrations at or below the criterion, are expected to be very minor. The only adverse effects of chronic lead exposures at sub-criteria concentrations were to snails and the amphipod *Hyalella azteca*. In most habitats, listed salmonids would not be expected to be dependent on amphipods and snails for food. Listed salmon and steelhead are unlikely to be injured or killed by exposure to lead concentrations that are at or below the proposed acute or chronic criteria. No evidence of direct adverse sublethal effects occurring at concentrations at or below the chronic criterion to salmonids was found.

2.4.13. Organic Pollutants: General Issues

In addition to the general issues (Section 2.4.1) that apply to all contaminants considered in the proposed action, the following issues specific to organic pollutants may create hazards for listed salmon and steelhead.

Organic Pollutants Toxicity and Exposure. Eisler's series of synoptic reviews, EPA's criteria documents, and the World Health Organization's environmental health criteria documents (e.g.,

WHO 1984) provide a good summary of sources, pathways, and toxic effects of organic pollutants. Most of the organic compounds considered in the proposed action are for organochlorine pesticides (chlordane, dieldrin, aldrin, lindane, heptachlor), used in the past for a variety of agricultural applications, as well as part of measures for controlling insects considered hazardous to human health. The remainder are industrial chemicals (PCBs, PCPs) 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 PCP are still used at all in the United States, and permitted applications for lindane and heptachlor are very limited. For the most part, these organic contaminants are no longer being released directly into the water column. 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 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 furthermore 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 are also typically present. 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 to 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, 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, alter liver and kidney functions, and impair the immune system. Elevated concentrations of many organochlorine compounds can cause growth inhibition, impaired reproduction, and developmental defects that

may affect not only the target organisms themselves, but can also impact the growth and survival of predator species further 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 (e.g., Nimmo 1985; Meador 2006; Rand 1995). Specific examples of these effects are identified for each compound in our analysis.

Proposed Chronic Criteria Are Based on Maximum Tissue Residues For Human or Wildlife Consumption, Not on Health Effects in Aquatic Organisms. For most of the organic contaminants, the chronic ambient water quality criteria are not based on long-term toxic effects in salmonids or other fish species. Instead, they are based on maximum permissible residues for human or wildlife consumption. Numeric criteria that are based on maximum tissue residues considered acceptable for wildlife or human health may not reflect a similar protectiveness of the health of aquatic organisms. Although in some cases these residues may be below those associated with adverse effects in salmonids, adverse effects in fish were not specifically addressed when determining the criteria (EPA 2000d).

Bioconcentration and Bioaccumulation Factors, Used in Determining and Evaluating Proposed Criteria, Associated With High Variability and Uncertainty. An important problem with many chronic criteria for organic pollutants is that the BCFs or BAFs 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 BAFs, 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. However, because a wide range in BCFs appears to exist (EPA 2000d; Meador 2006), such comparisons cannot be discounted and the criteria are evaluated in this Opinion accordingly.

Insufficient Data for Toxicity of Organic Contaminants That are Mixtures of Different Congeners with Varying Modes of Action. Several of the organic contaminants reviewed in this document are not single compounds, but mixtures of a large number of congeners with differing levels of toxicity and modes of action. This is particularly true of PCBs, which are a mixture of over 200 separate congeners, and toxaphene, which is a combination of over 600 isomers. For PCBs, methods such as the calculation of a toxicity equivalence factor (TEF) for those congeners with dioxin-like activity can provide a measure of the overall toxicity of mixtures containing these congeners (Safe *et al*. 1994; Van den Berg *et al*. 1998). For toxaphene, the toxicity of its various isomers is only beginning to be documented (e.g., de Geus *et al*. 1999). However, in neither case are these issues dealt with in existing water quality standards.

Water-borne Exposure from Contaminated Sediments. Because hydrophobic compounds are expected to show a similar or proportional affinity for the lipid of an organism as that for octanol, 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 BCF and K_{ow} is strong (Mackay 1982). The expected ww 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% ww 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 IWQS.

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):

$SQC_{oc} = K_{oc} \text{ X } F_{CV}$
where:

SQC_{oc} = sediment contaminant concentration in µg/kg organic carbon
K_{oc} = partitioning coefficient for sediment organic carbon
F_{cv} = the chronic water quality criterion in µg/L

K_{oc} can be calculated from the octanol/water partitioning coefficient, K_{ow}, using the equation:

$Log_{10}(K_{oc}) = 0.00028 + 0.983 \text{ X } Log_{10}(K_{ow})$

This equation is used in the analysis of effects below for evaluating the potential for water-borne exposure concentrations of organic pollutants that are at or below the Idaho criteria.

Organic Pollutants: Analysis of Individual Chemicals. In the analysis of organic pollutants, the effects of each organic toxic substance of concern are identified, and the proposed criteria are compared with data available to NMFS that describe sample background concentrations and the results of salmonid toxicity tests. Where possible, effects to the food sources of listed salmonids, and effects related to bioaccumulation, are also identified.

The LC_{50}s are used in this Opinion to evaluate criteria, rather than the more germane threshold toxicity concentrations. This reflects standard toxicological procedures, which seldom determine toxicity threshold concentrations. The relation between the two measures of toxicity response is not always linear, so use of a consistent, multiplicative CF is precluded.

The following analysis focuses on exceedences of each parameter individually. Where studies indicate an individual contaminant criterion is not likely to harm listed salmonids, the body of evidence may not support such an indication when several contaminants are near or equal to the proposed criteria in the same water sample. As a case in point, Laetz *et al*. (2009) determined that several combinations of organophosphate pesticides were lethal at concentrations that were sublethal in single-chemical trials. As described in the section on mixture toxicity, the proposed criteria cannot be applied individually to assess the effects of additive or greater that additive toxicity, a significant limitation that can adversely affect listed salmonids.

2.4.14. The Effects of EPA Approval of Pentachlorophenol (PCP) Criteria

Pentachlorophenol is a chlorinated hydrocarbon that is used primarily as an insecticide and fungicide, but also secondarily as an herbicide, molluscicide, and bactericide (Eisler 1989). Its primary application is to protect timber from fungal rot and wood-boring insects. According to the EPA, PCP is a Registered Use Product (RUP) in formulations as a wood preservative. A RUP may be purchased only by a certified applicator. Technical grade PCP is approximately 86% pure and historically has been contaminated with dioxins and hexachlorobenzene. Dioxin contamination is the main reason PCP was reclassified as a RUP in 1987 (EPA 2008).

Commercial forms of PCP that include manufacturing impurities resulted in reduced growth and survival in fathead minnow (*Pimephales promelas*) at PCP concentrations that are approximately five times lower than concentrations of purified PCP that caused similar effects to fish (Cleveland *et al.* 1982). The excess toxicity is presumably due to the impurities that occur in the commercial preparations. These differences should be considered when research studies on toxicity are being evaluated and when environmental concentrations from known sources are compared to criteria values.

Pentachlorophenol has a strong propensity to associate with the organic carbon of sediment and lipid 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 adenosine triphosphate ATP which is fundamental to metabolism 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 (Johansen *et al.* 1985; Brown *et al.* 1985; Eisler 1989).

Pentachlorophenol 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; Dominguez and Chapman 1984; Brown *et al.* 1985). Pentachlorophenol is also considered a probable human carcinogen.

The highest PCP concentrations near the action area were almost three orders of magnitude lower than the most stringent applicable criteria value (0.00047 µg/L in the discharge from Brownlee Dam vs 6.2 µg/L for the fish consumption based water quality criteria) (Table 2.3.1).

2.4.14.1. Species Effects of Pentachlorophenol Criteria

The criteria for PCP established by the EPA are pH dependent (EPA 1986b). 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. To determine the freshwater criteria as a function of pH the following equation is used:

$CMC = \exp^{(1.005 \times pH - 4.83)}$ (*in µg/L*)
$CCC = \exp^{(1.005 \times pH - 5.29)}$ (*in µg/L*)

For example, at a pH of 7.0, the corresponding criteria are 9.1 µg/L and 6.7 µg/L for acute and chronic exposures, respectively. At a pH of 8.0, the corresponding criteria are 25 µg/L and 18 µg/L for acute and chronic exposures, respectively.

Acute Pentachlorophenol Criterion. Data contained in the EPA's AQUIRE database indicates that most 96-hour PCP LC_{50}s for salmonids are in the 10 µg/L to 80 µg/L range, with the lowest reported for cutthroat trout (Mayer and Ellersieck 1986). Van Leeuwen *et al.* (1985) determined the 96-hour LC_{50} to be 18 µg/L at pH 7.2 for early fry of rainbow trout with 95% confidence intervals ranging between 10 µg/L and 32 µg/L. The acute PCP criterion at pH 7.2 is 11 µg/L, suggesting that some mortality could occur at or close to the acute criterion concentration. Other tests with rainbow trout and coho salmon have produced higher (less sensitive) results. Dominguez and Chapman (1984) obtained a 96-hr LC_{50} of 66 µg/L with steelhead fry at pH 7.4. The acute PCP criterion at pH 7.4 is 14 µg/L. Dwyer *et al.* (2005a) tested rainbow trout, two other "standard" test species (fathead and sheepshead minnows) and 17 endangered or threatened species with PCP. They obtained a 96-hr LC_{50} for rainbow trout of about 160 µg/L and LC_{50}s for other listed salmonids ranged from 110 to 170 µg/L. The most sensitive species tested with PCP was the Atlantic sturgeon with an LC_{50} <40 µg/L. All of the salmonid LC_{50}s were well above the acute criterion of 20 µg/L, for the pH 7.8 waters tested (Dwyer *et al.* 2005b). Pacific salmon were not among the species tested, and thus the rainbow trout are probably the closest surrogate from Dwyer's study. Hedtke *et al.* (1982) conducted multiple tests of the acute toxicity of PCP to juvenile coho salmon at different life stages. The smallest swim-up fry were the most sensitive, with LC_{50}s of about 45 µg/L, compared to the acute criterion of 9 µg/L at pH 7 (Hedtke *et al.* 1982). Thus some tests suggest acute toxicity is possible in salmonids when water concentrations are near the acute criterion, yet other results found acute toxicity only at PCP concentrations three times or more greater than the acute criterion. Additionally, because the available data were LC_{50} values, and did not report the actual lower bounds of lethality, the steepness of the dose-response curve is certain, and the lower limit for water concentrations of PCP that may cause mortality in listed salmonids is thus uncertain.

Chronic Pentachlorophenol Criterion. A review of chronic effects with salmonids indicate that with the exception of one study with sockeye salmon, the thresholds of adverse effects are above the chronic criterion.

With sockeye salmon, Webb and Brett (1973) showed that thresholds for decreased growth rates and food conversion efficiencies of approximately 1.74 to 1.8 µg/L at pH 6.8 following 28-day exposures. These effects occurred at PCP concentrations less than the chronic criterion of 4.7 µg/L for test pH of 6.8. From their Figure 5, at the chronic criterion concentration of 4.7 µg/L

PCP, growth rates and food conversions were about 90% of those in the control treatments. In their abstract, Webb and Brett (1973) listed the 1.74 to 1.8 µg/L values not as thresholds of effect (i.e., highest concentrations with no effects) but rather as EC_{50}s for growth and food conversion. A 50% reduction in growth rate or food conversion at concentrations lower than the chronic criterion would be an extremely severe and unacceptable effect. However, further inspection of their results, especially bottom of their p. 504 and their Figure 5, supports a different interpretation. The fish died before growth rates or food conversions were reduced by 50%, and the most severe growth rate and food conversion reductions measured (~ 45% reductions) occurred at about 50 µg/L PCP. No or only low effects were apparent at 1.7 to 1.8 µg/L.

Hodson and Blunt (1981) also observed reduced weight, growth rate, and biomass in rainbow trout exposed 20 µg/L and greater PCP concentrations over 4 weeks from embryo to fry stages, at pH of about 7.8 (chronic criterion = 13 µg/L). Dominguez and Chapman (1984) tested steelhead trout in a 72-day test and found a threshold of effects at about 10 µg/L, which is just above the chronic criterion of 8.6 µg/L for their average test conditions. Besser *et al.* (2005b) tested the effects of PCP on rainbow trout in 60-day tests and found the threshold (EC_{10}) for reduced growth was about 40 µg/L at pH 8.3. This was about twice the chronic criterion of 21 µg/L for pH 8.3.

Blood chemistry changes in juvenile Chinook salmon (altered blood urea and glucose levels) occurred following PCP exposures to 3.9 µg/L of PCP (nominal). No differences in survival, growth, feeding, or schooling behavior were noted (Iwama *et al.* 1986). Exposures to 39 µg/L PCP (nominal) killed all fish after 8 days. Chronic criterion were 3.5 to 5.2 µg/L for the conditions of the tests (pH 6.5 to 6.9). Nagler *et al.* (1986) determined the occurrence of oocyte impairment in rainbow trout at 22 µg/L (pH 7.5).

Behavioral Effects Little *et al.* (1990) examined post-exposure behavioral effects in rainbow trout at exposure concentrations that were from ten to 100 times less than the acute criterion of 20 µg/L. A statistically significant reduction in the percent survival by trout that were preyed on by largemouth bass occurred at an exposure concentration of 0.2 µg/L. A similar response may be expected for salmon. 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* 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* 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 considered 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.

2.4.14.2. Habitat Effects of Pentachlorophenol Criteria

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 3 µg/ to 100 µg/L. It appears that most invertebrates are less sensitive than fish to PCP concentrations in water and therefore may be protected by the proposed criteria. There are, however, studies showing adverse effects to invertebrates exposed to water concentrations below the chronic criterion. Hedtke *et al.* (1986) determined reproductive impairment in a daphnid at 4 µg/L and pH 7.3. Borgmann *et al.* (1989) found that 23 µg/L PCP reduced the amphipod *Gammarus* survival to only 25% of controls, while the amphipod *Hyalella* was only affected at 100 µg/L and above. The chronic criterion for Borgmann's tests ranged from about 19 to 35 µg/L, for pHs ranging from 8.2 to 8.8. Acute responses of amphipods were much higher than acute PCP criteria with LC_{50}s ranging between 92 and 790 µg/L. The corresponding acute criterion values were 6 to 41 µg/L for the test pH's of 6.5 to 8.5.

Bioaccumulation. 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. Metabolism of PCP is relatively rapid in rainbow trout (McKim *et al.* 1986; Glickman *et al.* 1977), and this is likely true in other salmonids as well. Nevertheless, the elimination rate of this compound is sufficiently slow that it takes 11.7 days for tissue concentrations to reach 95% steady state (McKim *et al.* 1986). According to the data provided in McKim *et al.* (1986) a 96-hour exposure will produce tissue concentrations that are only 63% of steady state. Therefore, any assessment of the maximum attainable tissue concentration and resulting biological response for a given exposure concentration must consider a longer time period (e.g., approximately 12 days) to reach that level. An estimate of the steady-state wet-weight BCF for salmonids is 4,600 (~23,000 dw) using the octanol-water partition coefficient for PCP ($\log_{10}$ (K_{ow}) = 5). However, lower than predicted BCF values are common in fish and are likely due to metabolism of PCP.

Bioaccumulation of PCP is pH dependent, 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). Spehar *et al.* (1985) determined the following regression equation relating BCF (wet weight) and pH for PCP uptake by the fathead minnow:
Log BCF = 4.80 - 0.28 x pH.

2.4.14.3. Summary for Pentachlorophenol

Some studies indicate the proposed acute PCP criterion is at the level where some acute toxicity will occur. Other studies showed that LC_{50}s for salmonids were well above the proposed acute water quality standard. Most studies of chronic effects reported the onset of adverse effects occurred at least slightly above the chronic criterion, although a single study found reduced growth in sockeye salmon at lower concentrations than the chronic criterion. Rainbow trout exposed to PCP concentrations far below the chronic criterion showed reduced ability to evade

predators, and reduced ability to capture prey. Both the chronic and acute criteria will likely have some effect on listed species or their food sources.

Pentachlorophenol is not likely to be a component of NPDES discharges, but may be used in the treatment of wood that finds its way into inwater or overwater structures so the exposure risk, while very small, is not discountable

2.4.15. The Effects of EPA Approval of the Aldrin/Dieldrin Criteria

Aldrin and dieldrin are synthetic cyclic chlorinated hydrocarbons called cyclodienes. Although aldrin has been used in higher quantities than dieldrin, both were used extensively in the 1950s and 1960s as soil insecticides. At that time, they 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; 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 (reviewed 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 (reviewed in Biddinger and Gloss 1984). One study, conducted on the environmental fate and transport of dieldrin in the Coralville Reservoir in eastern Iowa, revealed that of the portion of dieldrin that was present specifically in the water column, 74% occurred in fish, 25% was dissolved in water, and less than 1% was adsorbed to suspended solids (Schnoor 1981).

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

Because it is extremely hydrophobic, 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 reenters 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* required 4 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 clearance rate was also highest at the early fry stage.

2.4.15.1. Species Effects of Aldrin/Dieldrin Criteria

The proposed acute criterion for aldrin is 3 µg/L; a chronic criterion has not been proposed. However, the most stringent criteria that applies to all critical habitats and waters occupied by listed species is 0.00014 µg/L, based on protecting human health from consuming exposed fish (Table 1.3.1*)*. For dieldrin, the acute criterion proposed for approval is 2.5 µg/L and the proposed chronic criterion is 0.0019 µg/L (Table 1.3.1*)*. Because aldrin and dieldrin have somewhat different toxicities, they are evaluated separately below.

Acute Aldrin Criterion. Few toxicity studies were found in the literature that reported acute effects of aldrin on salmonids. Ninety-six hour LC_{50} values were 7.5 µg/L for Chinook and 45.9 µg/L for coho salmon (Katz 1961). Sublethal effects on juvenile Atlantic salmon in a 24-hour exposure study were observed at 33 times and 50 times the proposed acute criterion (Peterson 1973). Two other toxicity studies were found that tested the acute effects of aldrin on salmonid species:

Macek *et al*. (1969) reported a 96-hour LC_{50} value of 2.2 µg/L for rainbow trout exposed at 12.7°C, pH 7.1 in a static experiment with a nominal (95%) aldrin concentration.

Katz (1961) reported a 96-hour LC_{50} value of 17.7 µg/L for rainbow trout exposed at 20 ^{o}C, pH 6.8-7.4 in a static experiment with a nominal (88.4%) aldrin concentration.

All of these studies involved exposure in static experiments with nominal aldrin concentrations, a type of experimental design that tends to underestimate the toxicity of a contaminant that can hydrolyze or otherwise degrade (e.g., Stephan *et al*. 1985). Although few studies on the acute effects of aldrin on salmonids could be found in the scientific literature, with none more recent than 1973, the observations that one of them determined acute effects at concentrations below the proposed acute criterion for aldrin, and that all studies likely underestimated the toxicity of

this pesticide, suggest that the proposed acute criterion for aldrin can be lethal to listed salmonids.

***Acute Dieldrin Criterion*.** Only two studies were found reporting acute toxicity testing results on the effects of dieldrin on salmon or steelhead, with one showing adverse effects at concentrations below the proposed acute criterion:

Chadwick and Shumway (1969) reported a 50% mortality rate for steelhead trout fry when exposed to 0.39 µg/L dieldrin for 3 to 7 days at 12°C in a flow-through experiment with a measured dieldrin concentration.

Katz (1961) conducted acute toxicity tests on two salmon species, using 90% dieldrin at 20°C, pH 6.8 to 7.4, in static experiments with a nominal (unmeasured) dieldrin concentration. Ninety-six hour LC_{50}s equal to 6.1 µg/L and 10.8 µg/L were determined for Chinook and coho salmon, respectively.

Available toxicity tests using other salmonid species showed acute effects to rainbow, cutthroat, and brown trout at concentrations below or near the acute criterion:

Shubat and Curtis (1986) reported a 96-hour LC_{50} value of 0.62 µg/L for juvenile rainbow trout exposed at 12°C to 13°C, pH 7.6-8.1, in a flow-through experiment with measured dieldrin concentrations.

Macek *et al*. (1969) reported a 96-hour LC_{50} value of 1.4 µg/L for rainbow trout exposed at 12.7°C, pH 7.1, in a static experiment with a nominal concentration of 85% dieldrin.

Van Leeuwen *et al*. (1985) reported a 96-hour LC_{50} value of 3.1 µg/L for rainbow trout early fry exposed at 9°C to11°C, pH 7.2, in a static experiment with 99% dieldrin.

Shubat and Curtis (1986) reported a 96-hour LC_{100} value of 3.1 µg/L for juvenile rainbow trout exposed at 12°C to 13°C, pH 7.6 to 8.1, in a flow-through experiment with a measured dieldrin concentrations.

The studies outlined above, while including only limited information from the 1960s on the acute toxicity of dieldrin to salmon or steelhead, nonetheless indicate that lethality occurs at levels which are below or slightly above the acute criterion proposed for dieldrin. 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 salmon and steelhead studies. Also, these 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 unmeasured, nominal (target) dieldrin concentrations (Chadwick and Shumway 1969; Macek *et al*. 1969). The more recent and flow-through studies reported lethal concentrations that are below or near the proposed acute criterion for dieldrin, suggesting that this criterion could kill listed salmonid species.

Chronic Aldrin Criterion. The EPA has not determined a chronic freshwater criterion for aldrin, based on a lack of available toxicity information (EPA 1980a), and no chronic criterion has been proposed for aldrin in the current action. However, toxicity testing involving other freshwater fish species suggests the potential exists for adverse effects of chronic exposures. In the freshwater teleost *Puntius conchonius*, exposure to 0.0466 µg/L aldrin for 2 to 4 months resulted in a significant increase in disintegrating oocytes, and reduction in the population of stage 0-II oocytes (Kumar and Pant 1988). Other studies have involved much higher concentrations (Singh and Srivastaya 1992; Singh *et al*. 1996) and thus cannot be used to evaluate effects related to development of a chronic criterion for aldrin.

Chronic Dieldrin Criterion. NMFS did not locate any studies on the chronic toxicity of dieldrin to salmon, but found two studies that reported the results of chronic toxicity experiments on rainbow trout. Concentrations at which adverse effects were noted were 95 and 137 times the proposed chronic criterion of 0.0019 µg/L for dieldrin:

Phillips and Buhler (1979) exposed fingerling rainbow trout to 0.18 µg/L dieldrin for 61 days under flow-through conditions and measured dieldrin concentration. 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.

Shubat and Curtis (1986) reported a 12-day LC_{50} value of 0.26 µg/L for juvenile rainbow trout exposed at 12°C to 13°C, pH 7.6-8.1, in a flow-through experiment with a measured dieldrin concentration.

These limited results suggest that the proposed chronic criterion for dieldrin may avoid harming listed salmon subjected to short-term, water-borne exposure. However, they do not indicate whether the proposed chronic criterion is protective against bioaccumulation-related effects. To address this, several dietary exposure studies were evaluated that reported dieldrin tissue concentrations and chronic effects. If a specific chronic effect is associated with a specific tissue concentration and the BCF for dieldrin is known, then the tissue concentration and BCF can be used to back-calculate an estimate of the aqueous dieldrin exposure concentration resulting in an equivalent tissue concentration (and thus an equivalent chronic effect), in the following manner:

$$\text{BCF} = (\mu\text{g chemical/g tissue} \div \mu\text{g chemical/g water})$$

or,

$$\mu\text{g chemical/g tissue} = \mu\text{g chemical/g water} \times \text{BCF}$$

Two BCF values were identified; 1,700 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) we estimated dieldrin concentrations to be between 26 and 1,926 times the proposed chronic criterion of 0.0019 µg/L for dieldrin:

Hendricks *et al*. (1979) reported repressed growth in juvenile rainbow trout exposed to 5 mg/kg 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 to 430 µg dieldrin/kg body weight/day (0.36 µg to 10.8 µg dieldrin/g of food). The corresponding dieldrin tissue concentration was 0.41 mg/kg to 6.23 mg/kg wet weight. Based on these tissue concentrations, a corresponding concentration for dieldrin in a water-borne only exposure experiment was estimated to be between 0.05 µg/L and 3.66 µg/L. The three effects observed parallel those seen in phenylketonuria, an inherited defect in human phenylalanine metabolism that is also characterized by mental deficiency. Although the study did not address analogous effects, it is possible that fish adaptability, behavior, and survival may be compromised based on biochemical similarities.

There are numerous additional studies on tissue exposure of salmonids to dieldrin. However, they have low utility for the purpose of evaluating the proposed chronic criterion, either because necessary data and findings were not reported, whole body tissue concentration could not be estimated, or test specimens were exposed to a mixture of compounds (e.g., Macek *et al*. 1970; Mehrle and Bloomfield 1974; Poels *et al*. 1980; Shubat and Curtis 1986).

In baseline data from the study area, in fish tissue collected from 33 locations in Idaho, including the lower Snake River below and Salmon River (in the action area), aldrin was <0.005 mg/kg wet weight. The highest concentration found for dieldrin was 0.037 mg/kg ww in carp from Brownlee Reservoir, Idaho (Clark and Maret 1998).

In summary, the reported chronic effects of dieldrin on juvenile salmonids were well above the proposed chronic criterion, suggesting that the chronic criterion for dieldrin is unlikely to injure or kill listed salmonids based on best available information.

***Toxicity in Mixtures*.** Limited information is available on the toxicological interaction of dieldrin with other contaminants. For any interaction between compounds, the route of

exposure, the concentration ratio between the compounds, and the presence of additional compounds in a complex environmental mixture can each cause unique variations in toxicological responses. Water-born and dietary exposure studies conducted on rainbow trout and amphipods indicate the occurrence of synergistic, antagonistic, additive, or independent interactions, depending on the compounds included in the mixture or the biological endpoints tested. These are briefly outlined below.

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 has been shown to vary 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 were found to sustain mortality levels that were about half of that seen 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 of the mixture was similar to that caused by dieldrin alone, thus indicating a reduction in the growth inhibitory effect of Aflatoxin B_1.

2.4.15.2. Habitat Effects of Aldrin/Dieldrin Criteria

Acute Toxicity to Food Organisms: Aldrin. There is a sizable body of scientific literature that provides details on the adverse effects of aldrin on aquatic macroinvertebrates that may serve as salmonid prey species. Only one study was found where LC_{50} values were below or near the proposed acute criterion of 3 µg/L:

Sanders and Cope (1968) reported a 96-hour LC_{50} value of 1.3 µg/L for the stonefly naiad *Pteronarcys californica* exposed at 15.5°C, and pH 7.1 in a static experiment.

The next highest 96-hour LC_{50}s reported were 8 µg/L for the isopod *Asellus brevicaudus* (Sanders 1972), and 9 µg/L for the mayfly *Ephemerella grandis* (EPA 1980a), with both cases involving static experiments using nominal aldrin concentrations. There are numerous additional examples in the toxicological literature that indicate acute toxicity of aldrin to salmonid prey species at much higher levels, with concentrations ranging between 13 to nearly 70,000 times the proposed acute criterion for aldrin (e.g., Jensen and Gaufin 1964; Sanders 1969; Georgacakis *et al*. 1971; Sanders 1972; Khan *et al*. 1973; Kaushik and Kumar 1993). Most salmonid prey species are relatively resistant to the lethal effects of aldrin at the proposed acute criterion.

Acute Toxicity to Food Organisms: Dieldrin. Acute effects of dieldrin on aquatic invertebrates have been noted to occur below or near the proposed acute criterion of 2.5 µg/L in three studies:

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 with a nominal dieldrin concentration.

Other studies have reported acute effects of dieldrin at concentrations that are considerably higher than the proposed acute criterion (from more than three to 720 times the criterion concentration; e.g., Jensen and Gaufin 1964; EPA 1980a; Sanders and Cope 1966; Sanders 1969; Georgacakis *et al*. 1971; Sanders 1972; Santharam *et al*. 1976; Bowman *et al*. 1981; Daniels and Allan 1981). There is apparently a wide range in the level of sensitivity of salmonid prey organisms to dieldrin, but nonetheless there are several studies which demonstrate toxicity responses at concentrations below or near the acute criterion. Thus, this criterion could result in lethal effects to salmonid food organisms.

Chronic Toxicity to Food Organisms: Aldrin. As stated earlier, neither the EPA nor Idaho have proposed a chronic freshwater criterion for aldrin, based on a lack of toxicity information (EPA 1980a). However, available literature indicates that chronic effects of aldrin may occur on at least two salmonid prey species. For the stoneflies *Pteronarcys californica* and *Aeroneuria pacifica*, Jensen and Gaufin (1966) reported 30-day LC_{50} values of 2.5 µg/L and 22 µg/L, respectively.

Chronic Toxicity to Food Organisms Dieldrin. NMFS did not find any reports in the toxicological literature that indicate adverse effects from dieldrin occur to salmonid prey species at levels below the proposed chronic criterion of 0.0019 µg/L. Results for three aquatic insects and three crustaceans demonstrate that adverse effects manifest at the individual or population level only when dieldrin concentrations are much higher, ranging between 105 to at least 5,000

times the criterion (Jensen and Gaufin 1966; Adema 1978; Daniels and Allan 1981; Phipps *et al*. 1995) . This suggests that the proposed chronic criterion for dieldrin is generally protective of salmonid prey.

Bioaccumulation of Aldrin. The tendency of aldrin to bioconcentrate in aquatic organisms generally has not been documented in the scientific literature, probably because metabolic reactions rapidly convert aldrin to dieldrin. However, one study was found in which *Daphnia magna* were exposed for 1 to 2 days at 1.7 µg/L aldrin, with associated BCFs of approximately 1,800 to 9,100 (Metcalf *et al*. 1973).

Bioaccumulation of Dieldrin. Salmonids and other freshwater fish species have been shown to strongly bioaccumulate 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 we found laboratory-derived bioaccumulation information 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 was 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 comparable to BCF 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, 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 (1976) conducted a field study from which reported tissue and water concentrations of dieldrin can be used to calculate BCF values for various insect, crustacean, or fish prey species used by salmonids. Water samples contained 0.004 µg/L to 0.012 µg/L dieldrin, and aquatic organisms had tissue levels ranging from 2 ppb to 61 ppb from the Des Moines River in Iowa in 1973. Corresponding calculations result in BCF values that are on the order of 1,500 for the stonefly *Pteronarcys*, 5,100 for the mayfly *Potamanthus*, 3,500 for chironomidae, 3,600 for trichopterans, 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 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 4 days at 10 µg/L.

Overall, the weight of evidence indicates that both salmonids and their prey bioconcentrate dieldrin from their environment.

Biomagnification. Although no studies could be found that deal directly with salmonids and their prey species, there are a number of published reports involving various food web chains indicating dieldrin does not tend to biomagnify through progressively higher trophic levels. Reinert (1972) conducted a freshwater laboratory study in which they found that direct uptake of dieldrin from water is more likely to occur than uptake through the diet. In the algae-daphnid-guppy (*Poecilia reticulata*) food chain tested, *D. magna* and guppies accumulated more dieldrin directly from water than from their respective food sources exposed to similar water concentrations. Van Sprang *et al*. (1991) determined in another laboratory study using estuarine organisms that biomagnification was not apparent when the estuarine mysid shrimp *Mysidopsis bahia* was fed dieldrin-contaminated *Artemia*. Furthermore, in a field study in the North Sea's Weser Estuary, analysis of dieldrin tissue levels in a cockle-soft clam-brown shrimp-sole food web did not indicate the occurrence of biomagnification of dieldrin in the respective organisms (Goerke *et al*. 1979). This is reflected in literature reviews that have concluded there is little to no evidence to suggest that dieldrin biomagnifies in aquatic food webs (Kay 1984; Suedel *et al*. 1994).

2.4.15.3. Summary for Aldrin/Dieldrin

Aldrin. The limited information available regarding aldrin toxicity to salmonids indicates that 50% mortality can occur when concentrations are below or slightly above the acute criterion.

Similarly, there is evidence that aldrin is toxic to some salmonid prey species when concentrations are below or close to the criterion. This information suggests that the proposed acute criterion for aldrin if found at these levels is reasonably certain to harm listed salmonids or impact their food sources. The limited information available regarding aldrin toxicity indicates that aldrin is toxic to some salmonid prey species when concentrations are below or close to the criterion and is LAA food sources. However, it is unlikely that discharges of aldrin will occur in the action area as no uses are currently approved and levels currently found in the water column are well below the proposed standards.

Additional comments on Aldrin. Although no chronic criterion for aldrin is proposed, available studies demonstrate that chronic effects do occur to freshwater fish at 0.0466 µg/L, and to prey items at 2.5 µg/L. These results suggest that the absence of a chronic criterion could result in adverse chronic effects to listed salmonids and their food source. However, the human-health based aldrin criteria is also applicable to all waters in the action area that are either designated critical habitat for, or are inhabited by listed salmonids. For aldrin this criterion is 0.00014 µg/L (Table 1.3.1). This value is lower than concentrations causing adverse effects to any aquatic prey species, listed species, or surrogate for a listed species reviewed here. Thus although the aldrin acute aquatic life criteria may not be fully protective to listed species and habitats for long-term exposures the application of the fish consumption based water quality standard to protect recreation is protective and is applicable in all waters of Idaho that contain listed species.

Dieldrin. The scientific literature on effects of dieldrin on salmonids reports acute lethal effects at concentrations below or slightly above the proposed acute criterion. These studies included various salmonid species, such as Chinook and coho salmon, steelhead, and rainbow, cutthroat, or brown trout, as well as toxicological information on juveniles and adults. This available information indicates that the proposed acute criterion for dieldrin will likely adversely affect listed salmonid species. The proposed acute criterion is greater than LC_{50}s reported for several important salmonid prey species. However, because acute effects could only come from recent applications, and because the use of dieldrin has been banned since EPA cancelled its registration in 1975, acute effects occurring from release of dieldrin are unlikely. Chronic studies involving juvenile rainbow trout demonstrate that limited adverse effects only occur when ambient concentrations are >95 times the proposed chronic criterion. This information is supplemented by published BCF values and analyses of the results of dietary exposure studies in which estimated aqueous concentrations of dieldrin resulting in reported tissue concentrations was also well above the chronic criterion. These limited studies indicate that the proposed chronic criterion will not result in measurable effects to listed salmonids. Further, no information suggests that prey species may be adversely affected by concentrations below the proposed chronic criterion. Dieldrin was detected in sediment in Brownlee Reservoir of the Snake River (Table 2.3.1). However, levels of dieldrin currently found in Brownlee Reservoir are well below the standard and the reservoir is not occupied by listed species. With no ongoing discharges, the level of dieldrin in sediment in Brownlee Reservoir is likely to decline over time.

2.4.16. The Effects of EPA Approval of the Chlordane Criteria

Chlordane is an organochlorine pesticide that was used in the United States from 1948 to 1988. The commercial formulation is not a single chemical, but a mixture of at least 23 different compounds, the major components being trans-chlordane, cis-chlordane, chlordene, heptachlor, and trans-nonachlor (Kidd and James 1991). For many years it was used as a pesticide on agricultural crops, lawns and gardens, and as a fumigating agent. Because of concerns over cancer risk, evidence of human exposure and accumulation in body fat, persistence in the environment, and danger to wildlife, EPA banned the use of chlordane on food crops in 1978, and phased out other uses over the next 5 years (EPA 1980c). From 1983 to 1988, its only approved use was to control termites in homes. When its application for termite control was banned in 1988, all approved use of chlordane in the United States stopped. However, it continued to be manufactured within the United States for export abroad (ATSDR 1994).

Chlordane is not highly water soluble, and has an octanol/water partition coefficient of $10^{5.54}$. (ATSDR 1992; EPA 1992b). It does not degrade rapidly in water and adsorbs strongly to particles and sediment in the water column, but it can leave aquatic systems by volatilization (Wauchope *et al*. 1992).

Chlordane is absorbed by animals into the body and stored in fatty tissues as well as in the kidneys, muscles, liver, and brain (ATSDR 1989; Smith 1991). It can be released into circulation when these fatty tissues are metabolized, as in the cases of starvation and intense activity (Smith 1991). Excretion of orally administered chlordane is slow and can take days to weeks (ATSDR 1989). Adverse effects of chlordane to mammals occur mainly through the nervous system, the digestive system, and the liver, and can result in convulsions and death (Smith 1991; EPA 1992b; USDHHS 1993a, b; ATSDR 1994). Increased activity of thyroid hormone may also occur (Martin 1971). With chronic exposure, the most frequently observed effects occur to the central nervous system, the liver, and the blood through disorders including aplastic anemia and acute anemia (ATSDR 1989). There is also evidence that exposure to chlordane may be associated with reproductive and developmental effects. Studies indicate that chlordane is weakly or nonmutagenic (Smith 1991), but evidence for carcinogenicity is generally inconclusive (NIOSH 1986; ATSDR 1992, EPA 1993b).

2.4.16.1. Species Effects of Chlordane Criteria

The proposed acute criterion is 2.4 µg/L, while the chronic criterion is 0.0043 µg/L. The acute value was derived from LC_{50} values for nine freshwater fish and five invertebrates and represents the fifth percentile of the mean species values for this group of animals, whereas the chronic criterion is based on the 1980 FDA guidelines for marketability of fish for human consumption (EPA 1980c).

Acute Chlordane Criterion. Reported LC_{50} values for salmonids are well above the proposed acute criterion of 2.4 µg/L. Lethal effects (96-hour LC_{50}) have been observed for acute water-borne exposures to technical-grade chlordane at 56 µg/L to 57 µg/L in coho and Chinook salmon, 45 µg/L to 47 µg/L in brook trout (Cardwell *et al*. 1977), 8 µg/L to 47 µg/L in rainbow

trout (Katz 1961; Mehrle 1974), and higher values have been reported in other studies (e.g., 42 µg/L to 90 µg/L; Kidd and James 1991, HSDB 1995). However, most of these data were from static tests with nominal chlordane concentrations, so it is possible that the compound's actual toxicity was underestimated. In most other species that have been tested, LC_{50}s have been in the 25 µg/L to 100 µg/L range (Cardwell *et al*. 1977, EPA 1980c), although LC_{50}s as low as 3 µg/L have been reported in carp and bass and 7.1 µg/L in bluegill (EPA 1980c). In a more recent study, Moore *et al*. (1998) tested the toxicity of chlordane to fathead minnow in 48-hour tests and determined a mortality rate gradient of approximately 1.68% of mortality per µg/L, so that at the acute criterion of 2.4 µg/L, the predicted mortality would be about 4%. The LC_{50} reported by Moore *et al*. (1998) was 21.4 µg/L, within the range reported for salmonids. This study therefore suggests that a low level of mortality ~5% or less of the exposed fish could occur in salmonids when chlordane concentrations are at the acute criterion.

Chronic Chlordane Criterion. Since the proposed chronic criterion of 0.0043 µg/L proposed for use in the IWQS is not based on chronic toxicological effects on freshwater fish or invertebrates (EPA 1980c), its utility for protecting listed salmonids is not clear. In general, however, chlordane water concentrations associated with biological effects in laboratory exposures appear to be well above the chronic criterion. Cardwell *et al*. (1977) examined the chronic toxicity of water-borne technical chlordane to brook trout (*Salvelinus fontinalis*) under flow-through conditions, and found that the lowest concentration to cause major chronic effects was 0.32 µg/L. Similarly, in a review of chlordane effects on several species, Eisler (1990) stated that 0.2 µg/L to 3 µg/L chlordane can be harmful with long-term exposure to sensitive fish. Other studies examining larval toxicity or sublethal effects of chlordane, such as changes in blood parameters, somatic indices, and ATPase levels, in non-salmonid fish during exposures of 30 to 60 days reported effects at similar or greater concentrations only (Gupta *et al*. 1995; Verma *et al*. 1978; Bansal *et al*. 1980). The lowest of these values were two orders of magnitude above the proposed chronic criterion.

The proposed criterion can also be evaluated by comparing tissue concentrations of chlordane associated with adverse effects with estimated tissue levels of chlordane at the chronic criterion. According to the criteria development documents (EPA 1980c), this concentration should be approximately 0.3 mg/kg in edible tissue, the FDA action level in place at that time. This tissue concentration estimate assumes a normalized BCF value for chlordane of 4,702 and a tissue lipid concentration of 15%, the default value for freshwater fish (EPA 1980c). For natural-origin salmonids, this value may be rather high, as whole body lipid levels of 5% to10% are more typical of adult salmonids (Meador 2002).

Relatively little information is available on tissue concentrations of chlordane associated with biological effects in salmonids. However, data suggest adverse effects in other species at tissue concentrations below those associated with the chronic criterion. For example, Schimmel *et al*. (1976) report increased mortality (25^{th} percentile effect dose (ED_{25})) for spot at concentrations ranging from 0.16 mg/kg to 0.55 mg/kg, and slightly increased mortality (ED_5) in sheepshead minnow at tissue concentrations as low as 0.010 to 0.02 mg/kg. However, other studies report effects at tissue concentrations in the 1 mg/kg to 10 mg/kg range and greater (Parrish *et al*. 1976; Delorme 1998).

It should be noted that BCF values used in calculating the chronic criterion were based primarily on data from fathead and sheepshead minnow, not on studies with salmonids, and thus may not reflect uptake in the species of concern. Also, because these BCFs were determined in the laboratory, they may underestimate chlordane uptake by animals in the field. In the natural environment, major routes of chlordane uptake are likely to be though sediments and diet, and the water quality criteria and laboratory derived BCF do not account for this additional exposure. More realistic assessments of exposure might be obtained from field derived BAFs. Oliver and Niimi (1998) determined a chlordane BAF of about 106 (562,000), or 51,000 normalized for lipid content for salmonid species from Lake Ontario. Using this value, predicted chlordane tissue concentrations would be 1.1 mg/kg to 3.3 mg/kg for the aquatic life criteria and 0.07 mg/kg to 0.44 mg/kg for human health based criteria from Table 1.3.1. For tissue lipid levels in the 5% to 15% range more typical of adult salmonids (Meador 2002), the value would be 0.05 mg/kg to 0.1 mg/kg. This approach is problematic; however, as these values may greatly overestimate exposure for species like anadromous salmon with short residence times in freshwater environments. Whatever the case, the variability in reported BCF and BAF values indicates that there is considerable uncertainty concerning the tissue concentrations that might be expected in fish at the proposed criteria.

These data suggest that exposure to water borne chlordane at concentrations below the chronic criterion should not cause mortality to threatened and endangered salmon. However, even when water concentrations of chlordane are very low, sediment and prey concentrations may be elevated, causing salmonids to accumulate these compounds to levels that are considered adverse (see below). Other harmful effects were not evaluated in the literature.

Behavioral Effects. Behavioral effects may occur at concentrations near or below the acute criterion. Little *et al*. (1990) examined spontaneous swimming activity, swimming capacity, feeding behavior, and vulnerability to predation in rainbow trout (*Oncorhynchus mykiss*) in 96-hour exposures to sublethal concentrations of chlordane. Behavioral changes were consistently demonstrated at concentrations of 2 µg/L.

Factors Affecting the Toxicity of Chlordane. The toxicity of chlordane can vary with temperature, sediment loading, age, condition, and nutritional history of the exposed organism and the formulation and isomer of the chemical. Toxicity is typically greater at higher temperatures (Rai and Mandal 1993). Specific mixtures of chlordanes in the environment may vary from the original mixture of technical-grade chlordane and thus also vary in toxicity. For example, Gooch *et al*. (1990) compared the toxicity of technical-grade chlordane with a mixture isolated from tissues of lake trout. When the toxicity of the residue was evaluated using an acute bioassay and a neuroreceptor binding affinity assay, it was found to be three to five times more toxic than the technical mixture. Chlordane may also interact with other contaminants present in the environment. Gupta *et al*. (1994) for example, found that the fish *Notopterus notopterus* exhibited additive and synergistic toxicity effects when chlordane was combined with the pesticide furadan.

2.4.16.2. Habitat Effects of Chlordane Criteria

Toxicity to Food Organisms. Data for acute and chronic toxicity of chlordane to salmonid prey species are not extensive. Cardwell *et al*. (1977) examined acute and chronic toxicity of water-borne technical chlordane to the cladoceran, *Daphnia magna*, the amphipod *Hyallela azteca*, and the midge *Chironimus sp*. under flow-through conditions. The concentrations of technical chlordane causing 50% immobilization in the cladoceran was 38.4 µg/L. By 96 hours, the amphipod was only slightly affected by the chlordane concentrations tested, and the 168-hour EC_{50} was 97.1 µg/L. While Hall *et al*. (1986) report a much higher LC_{50} of 270 µg/L, the reported LC_{50} values in other studies for these species are more similar to Cardwell *et al*. (1977). For example, EPA (1980c) reported a 48hour LC_{50} of 35 µg/L for *D. magna*. Chlordane 96 hour LC_{50} values of 26 mg/kg and 40 mg/kg have been reported for the amphipods *Gammarus lacustris* and *G. fasciatus*, respectively (Sanders and Cope 1966; Sanders 1969). More recently, Moore *et al*. (1998) performed aqueous 48-hour toxicity tests of chlordane on several invertebrate species, including *D. magna*, *H. azteca*, and *Chironomus tentans Fabricius*. Mortality rate gradients varied from 0.88% mortality per µg/L for *H. azteca* to 2.54% mortality per µg/L for *C. tentans*, with LC_{50}s of 20 µg/L to 57 µg/L. Thus, at the acute criterion of 2.4 µg/L, predicted mortality for these prey species would be between 1% and 6%.

In terms of chronic toxicity, Cardwell *et al*. (1977) reported that the lowest concentrations of technical chlordane found to cause chronic effects on long-term survival, growth, and reproduction were 1.7 µg/L for midges, 11.5 µg/L for amphipods, and 21.6 µg/L for cladocerans, values all well above the chronic criterion.

***Bioaccumulation*.** Chlordane bioconcentrates in both marine and freshwater fish and invertebrates, and studies conducted in the late 1970s showed that the fatty tissues of both land and water wildlife contained large amounts of chlordane and other cyclodiene insecticides (Gobas *et al*. 1988; Isnard and Lambert 1988; ATSDR 1989; HSDB 1995). Bioaccumulation factors in marine waters have been reported to range between 3,000 and 12,000 (Zaroogian *et al*. 1985), and may be as high as 18,500 in freshwater for rainbow trout (Oliver and Niimi 1985). There is some evidence of biotransformation of chlordane in rainbow and cutthroat trout (Albright *et al*. 1980; Pyysalo *et al*. 1981). Albright *et al*. (1980) measured residues in cutthroat trout (*Salmo clarki*) from a lake treated with technical chlordane, and found that trans-nonachlor was the most persistent constituent, accounting for about 50% of the total chlordane remaining in fish collected 3 years after the lake was treated. Other measured constituents (heptachlor, heptachlor epoxide, and chlordene) were non-detectable in less than a year after treatment. The study also indicated that animals appeared to produce oxychlordane from chlordane.

Other studies suggest that the bioconcentration and bioaccumulation of chlordane in nature are complex and may not always follow predictions of octanol-water partitioning. For example, Swackhamer and Hites (1988) examined uptake of chlordane and several other pesticides in different size classes of lake trout and compared the bioconcentration factors with the octanol-water partition coefficient (K_{ow}). However, the correlation was weak ($r^2 = 0.73$) when compared to published relationships based on laboratory data. Factors that can influence bioaccumulation of chlordane in fish include lipid content and trophic positioning (Kidd *et al*. 1998).

In baseline data from the study area, in fish tissue collected from 33 locations in Idaho, including the lower Snake River below and Salmon River (in the action area), chlordane was <0.005 mg/kg wwin fish, except for demersal fish from Brownlee Reservoir. There, using data from carp, largescale suckers, and channel catfish chlordane was detected with a maximum concentration of 0.020 mg/kg ww (Clark and Maret 1998). Chlordane was not detected in any salmonid.

***Uptake and Toxicity Through Alternate Routes of Exposure*.** Because chlordane is no longer in use in the United States, the major source of this compound will not be through point source discharges into surface water bodies, but from repositories of the contaminant that are persistent in sediments. This means that chlordane will not be taken up 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 underestimate actual exposure of organisms in the field.

Because sediments are likely the primary source of chlordane, the sediment chlordane concentration that would result in chlordane concentrations in the water column at or below the proposed criteria can be calculated per Section 2.4.13. For chlordane, $\log_{10}K_{ow} = 5.54$, $\log_{10}K_{oc} = 5.45$, and the aquatic life criterion $F_{CV} = 0.0043$, resulting in an estimated SQC_{oc} = 1.21 µg/g organic carbon. This would mean that for sediment total organic carbon (TOC) levels ranging between 1% to 5%, the chronic aquatic life criterion would be associated with sediment chlordane concentrations ranging between 12 ng/g to 61 ng/g sediment. This exceeds the sediment screening guideline of 10 ng/g dry wet established by the Army Corps of Engineers (COE) for in-water disposal of dredged sediment (COE 1998), and are above the interim Canadian freshwater sediment guidelines of 2.26 ng/g to 4.79 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 (CCME 2001). These data suggest that chlordane could adversely affect the salmonid prey base at concentrations below the proposed criteria, as the COE and the Canadian sediment quality criteria are based primarily on tests with benthic invertebrates. The most stringent applicable criterion in the action area, the fish consumption based AWQC of 0.00057 µg/L that are also applicable to waters occupied by listed species and designated critical habitats, are about eight times lower than the chronic criterion of 0.0043 µg/L for chlordane (Table 1.3.1*)*. When extrapolated to predict sediment concentrations in the same fashion as the chronic criterion, the resulting sediment concentration would be about 1.6 ng/g to 8 ng/g dw sediment, which is less than the COE screening criteria and overlap the Canadian guidelines.

Because there has been very little research on the toxicity of sediment-associated chlordane to salmonids, the sediment concentrations that can cause adverse effects are not well defined. There are a few estimates of biota-sediment accumulation factors (BSAFs) for salmonids. For example, for trans-chlordane, Oliver and Niimi (1988) determined a BSAF of 2.22 for salmonid species from Lake Ontario, with 11% lipid and sediment TOC of 2.7%.

2.4.16.3. Summary for Chlordane

Lethal effects from short-term exposures of salmonids or salmonid invertebrate prey species to chlordane only occurred at concentrations above the acute criterion. There are no current approved uses of chlordane in the United States and no manufacturing of chlordane takes place in Idaho. The levels of chlordane in Idaho detected in Brownlee Reservoir (Table 2.3.1) are well below the proposed criteria and no listed salmon or steelhead are located in or above the reservoir.

Data generally indicate that the proposed chronic criterion for chlordane is likely to avoid harm to listed salmonids. However, many sublethal effects of chronic exposure to chlordane that have been documented in mammals (i.e., neurological damage, altered immune and reproductive function, and increased cancer risk) have not been studied in salmonid species subjected to long-term chlordane exposure at concentrations near or below the criterion. Similarly, few data are available on the sublethal effects of long-term exposure to chlordane on salmonid prey. There are also a few studies suggesting that a risk of increased long-term mortality or sublethal effects at chlordane tissue concentrations close to those that might be expected in fish exposed to chlordane at levels allowed under the chronic aquatic life criteria. Additionally, bioaccumulation can occur in salmonids with chronic exposure to chlordane at levels allowable under the proposed criteria, and exposure is likely to occur not only through the water column but also through diet and contact with sediments. The proposed criteria do not account presently for these other sources of exposure. There is some evidence of risk to benthic invertebrates or through food web uptake associated with bioaccumulation and exposure from sources other than the water column. Based on the strength of evidence considered, the chronic criterion does not appear likely to harm salmonids through water column exposure. If exposure occurs the different exposure pathways may pose some risk for salmon and steelhead, but appear unlikely to result in injury or death. Additionally, there will be no new sources of chlordane and so exposure is unlikely to occur.

2.4.17. The Effects of EPA Approval of the Dichlorodiphenyltrichloroethane Criteria

Dichlorodiphenyltrichloroethane (DDT) is a waxy, odorless or slightly aromatic solid that has been used extensively as an insecticide throughout the world. DDT occurs in three isomeric forms o,p', o,o', and p,p'. The technical product consists primarily of p,p'-DDT (60% to 85%) and o.p'-DDT (15% to 21%), with small amounts of other impurities (NTP 2001). DDT is metabolized to dichlorodiphenylethylene (DDE) and dichlorodiphenyldichloroethane (DDD).

The insecticidal properties of DDT were first discovered in the early 1940s, and the pesticide was used extensively on crops in the United States over the period 1945 to 1972. It was also used as a mosquito larvacide, as a spray for eradication of malaria in dwellings, and as a dust in human delousing programs for typhus control. The EPA banned the use of DDT in food in 1972 and banned non-food uses in 1988, except as an insecticide for public health emergencies. Currently, no United States companies report the production of DDT, but major producers and users of DDT exist outside the country (ATSDR 1994; SRI 1997).

Recent studies (e.g., ATSDR 1994; EPA 1992b) report that DDT (usually expressed as the sum of DDT and its metabolites) can be found at concentrations in the hundreds of ppb in sediment and at ppm levels in fish in many urban areas in the United States. DDT is highly persistent in the environment, with a reported half-life between 2 and 15 years. Volatilization, photolysis, and biodegradation are the main processes for breakdown, but they appear to act very slowly on this compound. This pesticide exhibits a log10 K_{ow} of approximately 6.19, indicating that it has a strong tendency to bioaccumulate in the lipid of organisms. Even though it has been several decades since it was banned in the United States, DDT still persists in the environment and can be found in aquatic sediments.

Chronic exposure to DDT can affect the mammalian nervous system, liver, kidneys, and immune system (ATSDR 1994; WHO 1989). Immunological effects observed in test animals include reduced antibody formation and reduced levels of immune cells in rats and mice at doses ranging from 1 mg/kg/day to 13 mg/kg/day for 3 to 12 weeks. There is also evidence that DDT causes reproductive effects, including sterility and developmental problems, and it is thought that many of these observed effects may be the result of disruptions in the endocrine system. DDT may also be associated with teratogenic effects (ATSDR 1994). The evidence for mutagenicity and genotoxicity of DDT is contradictory (NTP 2001). There is some evidence that DNA exposure may be associated with chromosomal damage, but overall studies suggest that although DDT may have the potential to cause genotoxic effects, it is not strongly mutagenic (ATSDR 1994). Similarly, the evidence regarding the carcinogenicity of DDT is equivocal. It is classified by EPA and International Agency for Research on Cancer as "reasonably anticipated to be a human carcinogen," based on sufficient evidence of carcinogenicity in experimental animals (IARC 1974, 1982). These effects are assumed here to be similar in fish.

2.4.17.1. Species Effects of DDT Criteria

The proposed acute criterion for dissolved concentrations of 4,4'-DDT (p,p'-DDT) is 1.1 µg/L. The proposed chronic criterion for 4,4'-DDT (p,p'-DDT) is 0.001 µg/L, and the also applicable fish consumption based criterion is 0.00059 µg/L *(*Table 1.3.1) No criteria for the DDT metabolites, DDE and DDD, are proposed in this action.

Acute DDT Criterion. The proposed acute criterion is based on toxicity data from 18 freshwater invertebrate species and 24 fish species (EPA 1980f). For invertebrates, LC_{50} values ranged from 0.18 µg/L to 1800 µg/L, while for fish, LC_{50} values ranged from 0.6 µg/L for yellow perch to 180 µg/L for goldfish. The acute criterion of 1.1 µg/L is a value that would be protective of 95% of the species tested. Available data suggest that the acute criterion could expose listed salmonids to lethal DDT concentrations. Studies involving cutthroat trout reported LC_{50}s ranging from 0.85 µg/L to 1.32 µg/L, below or very close to the proposed acute criterion. For rainbow trout, reported LC_{50} values range from 1.7 µg/L to 42 µg/L (Katz 1961; Macek and McAllister 1970; Macek and Sanders 1970; Post and Schroder 1971; Marking 1966). For brown trout, values range from 2 µg/L to 17.5 µg/L (Macek and McAllister 1970; Marking 1966). Other reported 96-hour LC_{50} values range from 4 µg/L to 44 µg/L in coho salmon (Katz 1961; Macek and McAllister 1970; Post and Schroder 1971; Schaumberg *et al*. 1967), 8 µg/L to 20 µg/L for brook trout, and 9.1 µg/L to 9.5 µg/L for lake trout (Marking 1966, Post and

Schroeder 1971). An LC_{50} of 11.5 µg/L was reported for Chinook salmon (Katz 1961). These values are all based on static tests with nominal DDT concentrations.

The only data NMFS found for a flow-through test involved rainbow trout fry, and yielded an LC_{50} of 2.4 µg/L (Tooby *et al*. 1975). In cases where both flow through and static tests were conducted with the same species (shiner perch and dwarf perch), LC_{50} values for the static tests were approximately 20 times higher than those from the flow through tests (4.6 µg/L to 7.6 µg/L vs. 0.26 µg/L to 0.45 µg/L; Earnest and Benville 1971). This suggests that the acute LC_{50}s for salmonids based on static tests could underestimate the toxicity of DDT, and testing values more relevant to a natural stream environment in critical habitat could be an order of magnitude lower, below the proposed acute criterion.

Chronic DDT Criterion. Most available information on DDT effects is based on mammals, or fish species other than salmonids. Chronic exposure of mammals to DDT is known to cause physiological effects in the nervous system, liver, kidneys, endocrine system, and immune system (ATSDR 1994; WHO 1989). There is also evidence that DDT may have the potential to cause genotoxic effects, but it does not appear to be strongly mutagenic (ATSDR 1994).

There are few long-term studies on the effects of water-borne exposure to DDT in salmon, and it is difficult to know how to interpret a number of studies because they were conducted in static systems at nominal DDT concentrations above reported LC_{50} levels. For example, Allison *et al*. (1963) conducted a long-term study in which Snake River cutthroat trout were exposed to DDT in the water for 28 days at concentrations ranging from 10 µg/L to 1000 µg/L. Above 30 µg/L, fish showed increased cumulative mortality and effects on fry survival. However, since acute LC_{50} values for cutthroat trout are reported well below 30 µg/L, the results of Allison *et al*. (1963) do not provide a clear indication of the lower limits of concentrations where chronic effects might occur.

Early life stages of salmonids may be more susceptible to DDT effects than smolts or adults (Hudson *et al*. 1984; WHO 1989), but the reported concentrations where mortality occurred from water-borne exposure were well above the proposed chronic criteria. For example, Halter and Johnson (1974) reported that mean survival times of early life stages of coho salmon were considerably reduced by DDT concentrations above 0.5 µg/L. In another study, Atlantic salmon eggs were exposed to water containing 5 µg/L, 10 µg/L, 50 µg/L, or 100 µg/L of DDT (Dill and Saunders 1974). The hatched fry had balance problems and impaired behavioral development at 50 and 100 µg/L. In a more recent study, Glubokov (1990) reported increased mortality (0.7% to 10% above baseline) of coho salmon during early ontogeny when exposed to DDT over the range of 0.1 µg/L to 10 µg/L.

For studies with water-borne DDT conducted with other species, effect concentrations were also well above the 0.001 µg/L chronic criterion. For example, Pandey *et al*. (1996) exposed the estuarine mullet, *Liza parsia* to DDT at a concentration of 100 µg/L for 15 days, and observed dilation of blood sinusoids, as well as vacuolization, granular degeneration, necrosis and fibrosis in the liver. Weis and Weis (1974) observed increased individual activity and increased school size in goldfish exposed to DDT at 1 µg/L for 7 days. More recently, studies have been conducted to evaluate the estrogenicity of o,p'-DDT, o,p'-DDE, and p,p'-DDE by assessing their

potential to cause the production of estrogen-inducible proteins such as vitellogenin (yolk). Metcalfe *et al*. (2000) exposed medaka embryos to o,p'-DDT for 100 days and found males with testis-ova at nominal concentrations as low as 5 µg/L, or average measured concentrations as low as 1.2 µg/L. Exposure of female medaka to nominal water concentrations of 2.5 µg/L for 2 weeks resulted in progeny with longer hatching times, earlier ovarian development in females, and enhanced vitellogenic response in males exposed to estrogens. Cheek *et al*. (2001) conducted a similar study with medaka in which fish were exposed to water-borne o,p'-DDT in a flow through system. After 2 weeks of exposure, percent hatch and fertilization were reduced at exposure concentrations as low as 0.23 µg/L, while after 8 weeks, vitellogenin induction and effects on fertilization and hatching success were observed at 0.30 µg/L. Because o,p'-DDT typically accounts for about 20% of total DDTs in commercial DDT mixtures, the total DDT concentrations associated with such effects reported by Metcalf *et al*. (2000) and Cheek *et al*. (2001) would probably be in the range of 6 µg/L to 25 µg/L, well above the proposed criteria.

As noted below, the chronic criterion was determined by EPA as an ambient water concentration that would result in fish tissue DDT levels at or below 0.15 mg/kg. There are some problems with this analysis, particularly regarding uncertainty in applying the standardized BCF of 17,870 to salmonids. The range of reported BCFs for salmonids in EPA's water quality documents for DDT include much higher values (EPA 1980f). Reported laboratory-derived BCFs for salmonids for whole body DDT concentrations range from 38,600 in rainbow trout (Reinert *et al*. 1974) to 47,400 in lake trout (Reinert and Stone 1974). Field derived BCFs are higher. Examples are 1,560,000 for coho salmon (Lake Michigan Interstate Pesticide Commission 1972), 1,170,000 for lake trout (Reinert 1970). For muscle tissue only, BCFs range from 11,600 in rainbow trout (Miles and Harris 1973) to 45,400 in brown trout (Miles and Harris 1973) to 458,000 in lake trout (Miles and Harris 1973). These data suggest that the BCF may be unrealistically low for field-collected salmonids. If only salmonid data are used, the geometric mean of the lipid-normalized BCFs in the EPA criteria for DDT (EPA 1980f) is 28,298. Using this BCF, the predicted DDT tissue concentrations in a salmonid at the proposed AWQC of 0.001 mg/L would range from 0.14 mg/kg to 0.42 mg/kg for lipid levels of 5% to15%. Similarly, using the most stringent applicable fish consumption based criterion provides lower values, 0.08 to 0.25 mg/kg. On the other hand, however, these salmonid BCFs could overestimate exposure of listed salmonids with short residence times in Idaho waters.

A number of studies have been conducted in which salmonids and other fish were exposed to DDTs in the diet or through injection, and in some of these, whole body DDT concentrations associated with adverse effects have been measured. Most reported effects of DDTs on salmonids are associated with whole body tissue concentrations in the 1 mg/kg to 3 mg/kg ww range or greater, with some effects on early life stages (e.g., eggs, embryos, and fry) at tissue concentrations in the 0.5 mg/kg ww range (Johnson and Pecor 1969; Poels *et al*. 1980; Burdick *et al*. 1964; Buhler *et al*. 1969; Allison *et al*. 1964, Macek 1968). These concentrations are somewhat higher than the 0.08 to 0.4 mg/kg that were calculated by EPA under the proposed chronic aquatic life or fish consumption criteria. Effects at lower tissue concentrations have been reported in field studies. For example, Vuorinen *et al*. (1997) found correlations between DDT concentrations in muscle of female Baltic salmon and mortality of yolk sac fry. Muscle DDT concentrations in this study ranged from 0.00134 mg/kg to 0.0277 mg/kg, with an average

of 0.00541 mg/kg. However, these are muscle, not whole body residues, and the data are difficult to interpret because PCBs and other organochlorine pesticides were also present.

The estrogenicity of various DDT isomers (o,p'-DDT, o,p'-DDE, and p,p'-DDE) has been tested in salmonids exposed to DDTs through injection or in the diet (Arukwe *et al*. 1998, 2000; Donohoe and Curtis 1996; Celius and Walther 1998). These compounds appear to be estrogenic, but relatively high exposure concentrations were required for effects to be observed. For example, Donohoe and Curtis (1996) observed vitellogenin induction in juvenile rainbow trout after injecting trout at 14 day intervals with single or triplicate doses of o,p'-DDT, or o,p'-DDE (0 mg/kg, 5 mg/kg, 15 mg/kg or 30 mg/kg). Plasma vitellogenin and hepatic estrogen binding site concentrations were significantly elevated by o,p'-DDT and o,p'-DDE (total dose 45 mg/kg and 90 mg/kg). Celius and Walther (1998) and Arukwe *et al*. (1998, 2000) observed induction of eggshell (zona radiata) proteins in Atlantic salmon after injection with o,p'-DDT at a dose of 25 mg/kg body weight twice a week for 3 weeks. If we assume an uptake rate of 50% for dietary exposure, which is the typical value observed in feeding studies with salmonids (Allison *et al*. 1963; Meador 2002), and a 75% uptake rate for injection (Meador 2002), associated tissue concentrations of DDT in the fish in these studies would be approximately 7.5 mg/kg to 19 mg/kg. This is far above the estimated tissue concentration resulting from water-borne exposure under the proposed chronic criterion (0.15 mg/kg).

One non-salmonid study, performed on Atlantic croaker, suggests that DDT concentrations below the chronic criterion could be associated with adverse health effects. Khan and Thomas (1998) reported a stimulatory effect of o,p'-DDT on gonadotropin release and gonadal growth in Atlantic croaker after 3 weeks at dietary concentrations of 0.02 mg/kg and 0.1 mg/kg, or an estimated tissue concentrations of 0.01 mg/kg to 0.05 mg/kg, assuming a 50% uptake rate (Allison *et al*. 1963). However, this result was obtained with o,p'-DDT alone, which accounts for only about 20% of typical DDT mixtures, so tissue body burdens associated with such a result in the environment would probably be closer to the 0.05 mg/kg to 0.25 mg/kg range. These results suggest the potential for subtle effects of DDT on fish reproductive physiology at concentrations below the 0.15 mg/kg concentration allowed under the proposed criterion.

Some additional studies show that chronic exposure to DDTs can threaten fish health through other modes of action, but there is insufficient information to determine the effective doses for these health effects. For example, chronic exposure to DDT may contribute to cancer risk. Nunez *et al*. (1988) determined that DDT in the diet enhanced the risk of hepatocarcinogenesis in rainbow trout treated with carcinogenic PAHs aflatoxin B1 and N-methyl-N'-nitro-N-nitrosoguanidine. However, the concentration in the diet was relatively high (100 mg/kg for 12 months), so would likely result in tissue concentrations well above those associated with the chronic criterion. The minimum DDT exposure associated with increased cancer risk in fish is unknown. There is some evidence that DDT may disrupt cortisol secretion and stress response in salmonids (Benguira and Hontela 2000; Hontela 1997) from *in vitro* experiments, but it is difficult from these studies to determine concentrations of DDTs in ambient water or tissue that would be associated with such effects.

The chronic criterion for DDT of 0.001 µg/L is not based on species effects on aquatic life, but on the highest permissible value for wildlife protection. At the time when these criteria were

developed, chronic toxicity data for DDT were available for only one freshwater fish species, the fathead minnow (Jarvinen *et al*. 1977). The chronic value from this study, which was a life cycle test, was 0.74 µg/L. The value of 0.001 µg/L was obtained by using a maximum permissible tissue concentration of 0.15 mg/kg, based on reduced reproductive output of the brown pelican. The value of 0.15 mg/kg is the lowest reported DDT concentration in the pelican's major food source, the northern anchovy, associated with reduced egg shell thickness and low productivity in the pelican (C). The water quality criterion was calculated by dividing the target tissue concentration (0.15 mg/kg) by geometric mean (17,870) of a group of 80 normalized BCF values derived from field and laboratory studies in freshwater fish and invertebrates, and by an estimated percent lipid value of 8 in the pelican diet (EPA 1980f). Consequently, the data used for the development of this criterion have little bearing on the chronic toxicity of DDTs to listed salmonids or their prey.

Behavioral Effects. A variety of behavioral effects, including changes in temperature selection and exploratory behavior have been observed in salmonids and other fish species following short-term exposure to DDTs (Davy *et al*. 1973; Peterson 1973; Gardner 1973), but exposure concentrations were substantially above the proposed acute criterion (10 µg/L to 50 µg/L).

Factors Affecting DDT Uptake and Toxicity. Several reports indicate that smaller-sized salmonids take up relatively more DDT from the water column and are more sensitive to the action of DDT compared to larger individuals (Buhler and Shanks 1972; WHO 1989; Murphy 1971). Uptake of DDT also increases with temperature (Reinert *et al*. 1974), and decreases with increased salinity (Murphy 1970). In rainbow trout exposed to 0.33 µg/L DDT at temperatures of 5°C, 10°C and 15°C, whole body residues after 12 weeks were 3.8 mg/kg, 5.9 mg/kg, and 6.8 mg/kg respectively (Reinert *et al*. 1974). Murphy (1970) determined that increasing salinity from 0.15% to 10% decreased DDT uptake over 24 hours from 22% of the dose to 18% of the dose (body residues decreased from 658g to 328g).

Some studies suggest that DDT and organophosphate (cholinesterase inhibiting) pesticides can act synergistically to produce greater toxicity to the nervous system and cause higher mortality than either contaminant can alone (WHO 1989). DDT and PCB appear to have an additive relationship that impacts other vertebrate populations, such as contributing to avian eggshell thinning (WHO 1989).

2.4.17.2. Habitat Effects of DDT Criteria

Toxicity to Food Organisms. DDT is highly toxic to many aquatic invertebrate species. Johnson and Finley (1980) reported 96-hour LC_{50}s in various aquatic invertebrates (e.g., stoneflies, midges, crayfish, sow bugs) ranging from 0.18 µg/L to 7.0 µg/L, and 48-hour LC_{50}s of 4.7 µg/L for daphnids. Other reported 96-hour LC_{50}s for various aquatic invertebrate species have been from 1.8 mg/L to 54 mg/L (WHO 1989). In a more recent study, Lotufo *et al*. (2000) examined the relative toxicity of DDTs to several species of freshwater amphipods in water-borne exposures. For *Hyalella azteca*, the LC_{50} for DDT was 0.17 µg/L for a 4-day exposure and 0.1 µg/L for a 10-day exposure. For *Diporeia* spp., the LC_{50} was 2.16 µg/L for 10 days and 0.26 µg/L for 28 days. Using narcosis as an endpoint, the EC_{50} was 0.67 µg/L for 10 days and

0.07 for 28 days. In general, early developmental stages are more susceptible than adults to DDT's effects (WHO 1989). At sub-lethal concentrations, DDT may cause reproductive, developmental, cardiovascular, and neurological changes in aquatic invertebrates (WHO 1989). The reversibility of some effects, as well as the development of some resistance, may be possible in some aquatic invertebrates (Johnson and Finley 1980).

These results suggest that the acute (1.1 µg/L) criterion is probably not protective of gammarid amphipods and related invertebrate salmonid prey, but the chronic aquatic life (0.001 µg/L) standard would likely be protective if the major source of DDT exposure were through the water column. However, because DDTs tend to accumulate in sediment, some reduction in available prey species will likely occur in areas with contaminated sediments.

Bioaccumulation. The chronic exposure to DDTs results in bioaccumulation of these compounds in fish, with most accumulating in the liver and other fatty tissues and relatively little in muscle tissues (WHO 1989). This occurs mainly through the diet from eating contaminated prey, and by uptake from sediment and water (WHO 1989). Developing embryos have been documented to take up DDTs from maternal yolk (WHO 1989).

Bioaccumulation rates vary among fish species. Reported BCFs for DDT range from 1,000 to 1,000,000 in various aquatic species (EPA 1989b), and bioaccumulation may occur in some species at very low environmental concentrations (< 100 pg/L; Johnson and Finley 1980; Oliver and Niimi 1988). The BCFs for salmonids range from ~10,000 to over 1,000,000; Oliver and Niimi (1988) reported field-derived BCFs of over 4,000,000 and over 11,000,000 for salmonid species from Lake Ontario exposed to p,p'-DDT and p,p'-DDE, respectively. The half-life for elimination of DDT from rainbow trout has been estimated to be about 160 days (WHO 1989).

Uptake of DDT in salmon and other fishes can be influenced by a variety of factors. It tends to be greater with increased trophic status and lipid content (Berglund *et al*. 1997, Bentzen *et al*. 1996). Fish uptake of DDT from the water is also size-dependent with smaller fish taking up relatively more than larger fish (WHO 1989). Eutrophication and nutrient loading also tend to increase uptake, probably because of the higher concentration of organic matter and bound DDT in the water (Berglund *et al*. 1997). Muir *et al*. (1994) studied uptake and bioconcentration of p,p'-DDT by rainbow trout at differing levels of DOC. The equilibrium BCFs ranged from 33,300 to 91,000, and bioconcentration tended to be lower with addition of unfiltered humic acid.

Uptake and Toxicity Through Sediments. Because DDT is no longer in use in the United States, the primary source of this compound will not be through point source discharges into surface water bodies, but rather from repositories of the contaminant that are persistent in sediments. This means that DDT will not be taken up 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 underestimate actual exposure of organisms in the field.

Because sediments are the likely the primary potential source of DDT, the sediment DDT concentration that would result in DDT concentrations in the water column at or below the proposed criteria can be calculated per Section 2.4.13. For DDT, $\log_{10}(K_{ow}) = 6.19$, $\log_{10}(K_{oc})$

= 6.08, and F_{cv} = 0.001, resulting in an estimated SQC_{oc} = 1.2 mg/kg organic carbon. This would mean that for sediment TOC levels of 1% and 5%, the proposed criteria would be associated with sediment DDT concentrations ranging from 12 ng/g to 60 ng/g sediment. This level exceeds the sediment screening guideline of 6.9 ng/g dw established by the COE for in-water disposal of dredged sediment (COE 1998), and are above the interim Canadian freshwater sediment guidelines of 1.19 ng/g to 4.77 ng/g dw 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 (CCME 2001). This suggests the potential for impacts on the salmonid prey base, as these guidelines are based primarily on tests with benthic invertebrates.

Because there has been very little research on the toxicity of sediment-associated DDT, the sediment concentrations that can 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 DDT that would be associated with sediment DDT concentrations permissible under the proposed criteria. Without site-specific BSAFs for DDTs in salmonids, it is difficult to determine whether the proposed chronic criterion would be sufficiently protective.

As noted earlier, salmonid invertebrate prey are also likely to take up DDTs from sediments. Results of laboratory and field investigations suggest that thresholds for chronic effects generally occur at total DDT concentrations in sediments of about 2 ng/g dw (Long *et al*. 1995). Similarly, equilibrium partitioning methods predict that chronic effects may occur at DDT concentrations in sediment as low as 0.6 ng/g to 1.7 ng/g dw (Pavlou *et al*. 1987). Chapman (1996) estimated no observed effect levels for sediment DDTs at 8.5 mg/kg dw sediment based on full life cycle tests with the marine polychaete worm *Neanthes arenaceodentata*. If the sediment DDT concentrations associated with the proposed water column concentrations were associated with sediment DDT concentrations of 7 µg/kg to 60 µg/kg, these results suggest that they may not be adequate to protect invertebrate prey species from potential injury.

2.4.17.3. Summary for DDTs

Sediment and fish tissue DDT concentrations from Brownlee Reservoir tended to be the highest found in sampling in various locations in Idaho (Table 2.3.1; Clark and Maret 1998). In water, baseline concentrations of DDT found in Brownlee Reservoir in 2011 were <0.00066 µg/L, which is below the levels where effects would be expected to listed salmon and steelhead. DDT is a banned substance in the United States and so no new or ongoing discharges are expected to occur.

Concentrations of DDT in the action area at the proposed action acute criterion could cause harm to listed fish; however, because there will be no new discharges of DDT and no known hotspots of DDT occur in the action area where listed fish are present these effects are unlikely to occur.

The chronic criteria have risk of sublethal health effects in salmonids if bioconcentration results in tissue concentrations that are higher than those expected by EPA. The proposed chronic criterion may allow substantial bioaccumulation to occur because DDTs are taken up not only from the water column but also from sediments and prey organisms. No reports of direct adverse

effects to listed salmonids were located at concentrations lower than the chronic criterion. While some data are equivocal and there are quite a few uncertainties in interpreting DDT risks to fish, we found no persuasive evidence of adverse effects from DDT at concentrations lower than the chronic criterion concentrations.

2.4.18. The Effects of EPA Approval of the Endosulfan Criteria

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. Endosulfan use is highly restricted in the United States. The EPA cancelled its registration for home and garden use in 2000, and in 2012 banned all uses in the United States[6].

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

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

Most endosulfan toxicity studies on aquatic organisms that have been conducted have evaluated direct water-borne exposure. Studies reported by Barry *et al*. (1995) indicated that, for the cladoceran *Daphnia carinata,* water-borne exposure is in fact the most toxic route. Toxicity towards *D. carinata* was also found to 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.

[6] http://www.epa.gov/pesticides/reregistration/endosulfan/endosulfan-cancl-fs.html

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.

2.4.18.1. Species Effects of Endosulfan Criteria

The proposed acute criterion for endosulfan is 0.22 µg/L and the chronic criterion is 0.056 µg/L.

Acute Endosulfan Criterion. NMFS found only one study that reported acute lethal effects of endosulfan on salmonids during a 96-hour exposure period at a concentrations roughly 0.8 times the proposed acute criterion for endosulfan. These results were reported as LC_{50} values, suggesting that the proposed acute criterion could be lethal to listed salmonids.

Lemke (1980, cited in EPA 1980g) reported a 96-hour LC_{50} value of 0.17 µg/L for rainbow trout exposed in a flow-through experiment in which endosulfan concentration was measured.

Two other studies reported 96-hour LC_{50}s that were near the acute criterion:

Nebeker *et al*. (1983) reported a value of 0.3 µg/L for rainbow trout exposed at 12^{o}C in a flow-through experiment in which endosulfan concentration was measured.

Schoettger (1970) also reported a value of 0.3 µg/L for rainbow trout exposed at 10^{o}C andpH 7.4 in a static experiment with a nominal endosulfan concentration.

Most other studies that were found reported 96-hour LC_{50}s greater than the acute criterion, including:

Sunderam *et al*. (1992) reported a value of 0.7 µg/L for rainbow trout exposed at 12^{o}C, pH 7.5 in a static experiment in which endosulfan concentration was measured.

Faggella *et al*. (1990, cited in Fujimura *et al*. 1991) reported a value of 0.74 µg/L for Chinook salmon fry.

Johnson and Finley (1980) reported a value of 1.2 µg/L for rainbow trout exposed at 13^{o}C, pH 7.2 to 7.5 in a static experiment with a nominal endosulfan concentration.

Macek *et al*. (1969) reported a value of 1.5 µg/L for rainbow trout exposed at 12.7^{o}C, pH 7.1 in a static experiment with a nominal endosulfan concentration.

In rainbow trout exposures to endosulphan lasting minutes to hours, ventilation frequencies were increased; however, the exposure concentrations were more than an order of magnitude above the median LC_{50} for rainbow trout (Patra *et al*. 2009).

It should be noted that half of these studies were performed with nominal concentrations of endosulfan, and most studies were performed under static conditions, both of which tend to underestimate toxicity. Lemke (1980, cited in EPA 1980g) noted that flow-through assays with rainbow trout resulted in three times higher toxicity at the same measured concentration of endosulfan as in static assays.

Chronic Endosulfan Criterion. The available information on the chronic effects of endosulfan on salmonids or other freshwater fish is limited. NMFS found only one study in the literature that reported chronic effects of endosulfan on salmonids. 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 effects such as dilation of intermembranous spaces in mitochondria and deformation of mitochondria were observed. Other 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 types 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 µg/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 on 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 criterion.

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; 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 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 the presence of edema and lifting and hyperplasia of lamellar epithelium in the gills, and also led to an increase in the 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.

These studies collectively indicate the possibility for adverse effects to occur to listed salmonid species under the chronic and acute criteria proposed for endosulfan. Adverse effects of this nature will likely result in appreciable mortality depending on the nature of the exposure. NMFS assumes this will reduce abundance and productivity of any listed salmon and steelhead that are exposed.

Other Water Quality Parameters as Predictors of Endosulfan 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 was found to increase 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.

2.4.18.2. Habitat effects of Endosulfan Criteria

Toxicity to Food Organisms. NMFS found three studies that reported lethal toxicity of endosulfan to aquatic macroinvertebrates at concentrations that were 0.5 to 10 times the acute criterion, suggesting this criterion might not be protective for some salmonid prey species:

Magdza (1983, cited in Sunderam *et al*. 1994) reported a 48-hour EC_{50} value of 0.3 µg/L for the South African freshwater cladoceran *Daphnia longispina*.

Leonard *et al*. (1999) conducted acute toxicity tests on three insect species in static experiments using river water at 26°C and a nominal concentration of endosulfan. A 48-hour LC_{50} value of 0.4 µg/L was determined for a trichopteran larvae, with an LOEC of 0.3 µg/L. Seventy-two hour LC_{50}s of 1.0 µg/L and 0.6 µg/L were determined for two ephemeropteran nymphs, with a corresponding LOEC of 0.3 µg/L.

Sanders and Cope (1968, cited in EPA 1980g) reported an LC_{50} value of 2.3 µg/L for a stonefly species under static conditions with nominal endosulfan concentrations.

However, most toxicity studies indicate lethal effects from endosulfan do not occur on salmonid prey species until concentrations are between 19 to 2,200 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, cited in EPA 1980g); the cladoceran *Daphnia magna*, with 96-hour LC_{50} values of 56 µg/L to 271 µg/L (Schoettger 1970; Nebeker *et al*. 1983; EPA 1976); damselfly naiad 96-hour LC_{50} of 71.8 µg/L to 107 µg/L (Schoettger 1970); and a 48-hour LC_{50} of 215 µg/L for *Moinodaphnia macleayi* or 491 µg/L for *Ceriodaphnia dubia* (Sunderam *et al*. 1994).

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).

In summary, available toxicity data suggest that the proposed chronic criterion may be protective of salmonid prey species relevant to Idaho waters. However, because this collection of reports does not represent the range of salmon prey species, it is impossible to know for certain whether the chronic criterion would avoid population impacts on important prey items, such as insects, copepods, gammarid amphipods, other crustaceans, and molluscs.

***Bioaccumulation*.** Information on uptake, metabolism, and elimination of endosulfan was not available for any salmonid species. However, the following is a brief overview of information available for other freshwater fish species, including *Channa punctata* (Devi *et al*. 1981), *Labeo rohita* (Rao *et al*. 1980), the Indian carp *Catla catla* (Rao 1989), *Anabus testudineus* (Bloch) (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*, western white sucker, and goldfish. This differed somewhat from *Anabus testudineus*, in which both the liver and kidneys were reported as being the principal sites of detoxification.

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

NMFS found only two reports of endosulfan bioaccumulation for salmonid prey species. Sabaliunas *et al*. (1998) exposed the lake mussel *Anodonta piscinalis* to 1.5 µg/L endosulfan in a continuous flow experiment at 10^{o}C with measured contaminant concentration. They noted a whole body concentration factor of 750 under conditions that may not have reached steady state. Finally, a field study was conducted by the Mussel Watch Project (part of the National Oceanic and Atmospheric Administration's National Status and Trends Program) 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). They found that 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.

2.4.18.3 Summary for Endoculfan

Endosulfan has not been found in Idaho waters or sediments at levels that approach the standards as proposed and future discharges of endosulfan are unlikely to occur because the product use has been banned so an acute exposure scenario from an authorized release is unlikely. The proposed acute lethal criterion for endosulfan would likely result in some mortality of listed salmonids. Reported rainbow trout LC_{50}s near or below the proposed acute criterion indicate that appreciable mortality can occur in waters meeting the proposed criterion. Evaluation of the proposed chronic criterion was restricted by the absence of relevant toxicity testing data

involving salmonid species. The limited information that could be gathered on rainbow trout and two other freshwater fish suggests that the proposed chronic criterion can allow chronic physiological damage to listed salmonid species. The physiologic damage was not directly related to "clinically significant" fish health changes. Although there is a paucity of toxicity testing data, the available information suggests that the proposed acute and chronic criteria may protect some invertebrate prey species. Little test data exists for specific salmonid prey species

2.4.19. The Effects of EPA Approval of the Endrin Criteria

Endrin is a chlorinated pesticide that is a steroisomer 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 1992a). It was still used as a toxicant on bird perches for several years, but this use was also banned in 1991 (EPA 1992a). There are no current authorized uses of endrin in the United States

Exposure of rodents to endrin has been noted to result in adverse neurologic, liver, kidney, and miscellaneous endocrine and tissue weight effects (Kavlock *et al*. 1981; Hassan *et al*. 1991; Deichmann *et al*. 1970). There are some indications that endrin may have genotoxic effects, including increased DNA damage in hepatocytes due to oxidative injury (Bagchi *et al*. 1992a,b, 1993c; Hassoun *et al*. 1993). However, most studies suggest that endrin is not carcinogenic (EPA 1980h).

2.4.19.1. Species Effects of Endrin Criteria

The acute criterion for dissolved concentrations of endrin is 0.18 µg/L. The chronic criterion is 0.0023 µg/L (Table 1.3.1) and is based on tissue residue values associated with adverse effects in wildlife (EPA 1980h).

***Acute Endrin Criterion*.** The proposed acute criterion of 0.18 µg/L is below values associated with adverse effects in fish in most studies, but there is evidence in some studies of mortality occurring at concentrations below or near the proposed criterion. Reported LC_{50}s for salmonids range from 0.113 µg/L to 343 µg/L (Post and Schroeder 1971; Katz 1961; Bennett and Wolke 1987a; 1987b; EPA 1980h). While the majority of available studies showed effects at concentrations well above the criterion, in many cases they were nominal concentrations only, not measured concentrations, so their accuracy is not assured. Other fish species have also been found to be sensitive to acute effects when concentrations of endrin that are close to the acute criterion. For example, Jarvinen *et al*. (1988) reported a 96-hour LC_{50} of 0.7 µg/L for fathead minnow larvae (*Pimphales promelas*). They also found that a 48-hour exposure at the same concentration led to a reduction in growth that was detectable within 28 to 30 days. Similarly, Hansen *et al*. (1977) reported an LC_{50} of 0.3 µg/L for juvenile sheepshead minnow.

Chronic Endrin Criterion. There are few data available regarding chronic effects of water-borne exposure to endrin in salmonids. In other species, adverse effects have not been reported unless water concentrations were more than 10 times the proposed chronic criterion of 0.0023 µg/L (e.g., Hansen *et al*. 1977; Jarvinen and Tyo 1978; Jarvinen *et al*. 1988). However, there are some data available on tissue concentrations of endrin associated with a variety of sublethal effects in rainbow 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).

It is difficult to estimate the likely tissue concentrations of endrin in salmonids exposed at ambient water concentrations equivalent to the chronic criterion of 0.0023 µg/L, because no specific BCFs could be found for salmonids. However, for other fish species, reported BCFs range from 1,340 for spot to 15,000 for flagfish, with exposure periods ranging from 4 days to 300 days (EPA, 1980h). Many of these values were derived from field exposures, and thus likely incorporated dietary as well as water uptake. Assuming that this range of BCFs is accurate for salmonids would mean that a water concentration at the chronic criterion would result in estimated tissue concentrations ranging from 0.0033 mg/kg to 0.0345 mg/kg. Data from Grant and Mehrle (1973) suggest the potential for some effects on metabolism, stress response, and growth at water concentrations of endrin at or within 10 times the chronic criterion.

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 that were greater than criteria concentrations (0.12 µg/L to 0.15 µg/L) and found that several immune responses (migration inhibition factor assay), plaque forming cell assay, and serum agglutination titres were inhibited when fish were exposed to *Yersinia ruckeri* O-antigen. Serum cortisol concentrations were found to be significantly elevated in endrin-exposed fish. Fish receiving cortisol in the diet also 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 immunosuppresive effects. Exposure to water-borne endrin from agricultural runoff has been associated with an increased prevalence of parasitic infections in cultured sand goby (Supamataya 1988), but the fish were also exposed at the same time to dieldrin, DDTs, and possibly stress due to changes in dissolved oxygen and water temperature.

Singh and Singh (1980) reported total lipid levels in ovary and liver and cholesterol concentrations in ovary, liver and blood serum in the Asiatic 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 pesticides 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 pesticides at all concentrations. This study suggests that

exposure to endrin concentrations below the proposed chronic criterion could affect lipid and cholesterol balance in other gravid fish, including presumably salmon.

Factors Affecting Toxicity. Studies by Dalela *et al*. (1978) suggest that increases in temperature and pH may increase endrin toxicity, and that smaller fish were more susceptible to adverse effects from a given exposure concentration than larger fish.

2.4.19.2. Habitat Effects of Endrin Criteria

Toxicity to Food Organisms. Invertebrates tend to be more tolerant of endrin than fishes. When Anderson and DeFoe (1980) exposed stoneflies, caddisflies, 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, suggesting that both criteria would likely be protective of salmonid prey species. However, the available information is limited and may not account for exposure through other routes, such as sediments (see below).

Bioaccumulation. Studies show that endrin is bioaccumulated significantly by fish and other aquatic organisms (ATSDR 1996; EPA 1980h; Metcalf *et al*. 1973). Although specific bioconcentration factors 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). For example, in a model laboratory aquatic ecosystem containing algae, snails, water fleas, mosquito larvae, and mosquito fish, Metcalf *et al*. (1973) reported a ratio of biomagnification through the aquatic food chain to bioconcentration by direct uptake from water of 2.0 for endrin compared to 2.5 for DDT.

Uptake and Toxicity Through Alternate Routes of Exposure. 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. For a water concentration of 0.0023 µg/L, the proposed chronic criterion, and a BCF of 15,000, the highest reported for aquatic organisms in EPA's criteria documents (EPA 1980h), the predicted tissue concentration would be only 0.035 mg/kg.

Because endrin is no longer in use in the United States, the primary source of this compound will be from repositories of the contaminant that are persistent in sediments, not through point source discharges into surface water bodies. This means that endrin exposure 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.

Because sediments are likely the primary source of endrin, the sediment endrin concentration that would result in endrin concentrations in the water column at or below the proposed criteria can be calculated per Section 2.4.13. For endrin, where the maximum reported $\log_{10}$ (K_{ow}) is estimated at 5.6, $\log_{10}$ (K_{oc}) would equal 5.5. A value of F_{cv} = 0.0023 results in SQC_{oc} = 736 µg/kg organic carbon. This would mean that for sediment TOC levels of 1% to 5%, the proposed criteria would be associated with sediment endrin concentrations ranging from 7.36 µg/kg to 36.8 µg/kg dw sediment. These levels are within the range of the interim Canadian freshwater sediment guidelines of 2.67 to 62.4 ng/g dw 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 (CCME 2001). This suggests that the proposed criteria are unlikely to reduce the quality or quantity of listed salmon food items, although the data used to develop the criteria may not have been specific to salmon or their prey items.

Because there has been very little research on the toxicity of sediment-associated endrin to salmonids, the sediment concentrations that can cause adverse effects are not well defined. Similarly, BSAFs have not been determined for salmonids, so it is difficult to estimate the likely tissue concentrations of endrin that would be associated with sediment endrin concentrations permissible under the proposed criteria. Without this information, it is difficult to determine whether the proposed chronic criterion would be sufficiently protective. Data on effects of sediment-associated endrin to known salmonid prey species are also lacking. Some marine invertebrates show behavioral effects, such as changes in sediment reworking rates, at sediment endrin concentrations within the 7 µg/kg to 38 µg/kg range (Keilty *et al*. 1988a,b,c). In contrast, effects on mortality or burrowing occurred at much higher concentrations (15 mg/kg to 60 mg/kg dw for burrowing avoidance and about 2500 mg/kg for mortality) (Keilty *et al*. 1988a).

2.4.19.3. Summary for Endrin

Endrin is a banned product in the United State and so new discharges are unlikely to occur. In Idaho levels of endrin in Brownlee Reservoir have been detected at less than the chronic criteria. Most reports of mortality following short-term endrin exposures produced LC_{50}s greater than the acute criterion, although some effects occurred at lower concentrations. Evidence indicates that concentrations at the acute criterion will not harm salmonid prey species.

While data are sparse, most reports of adverse effects from chronic exposures to salmonids or other fish occurred at concentrations higher than the chronic criterion. A report of subclinical reductions in cholesterol and lipids in gravid Asiatic catfish are of ambiguous importance to salmon. Food chain exposure via diet or sediment was estimated by NMFS to mostly result in tissue residues lower than those shown to be harmful to fish.

2.4.20. The Effects of EPA Approval of the Heptachlor Criteria

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

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). Uptake may also occur through the diet or through exposure to contaminated sediments. 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; 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). There is other evidence that heptachlor and heptachlor epoxide are associated with infertility and improper development of offspring (ATSDR 1993; Amita Rani and Krishnakuman 1995; Mestitzova 1967; Oduma *et al*. 1995a,b). On the other hand, heptachlor appears to have limited developmental toxicity, and shows few teratogenic effects in most studies (WHO 1984; ATSDR 1993; Narotsky and Kavlock 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.

2.4.20.1. Species Effects of Heptachlor Criteria

The proposed acute criterion for dissolved concentrations of heptachlor is 0.52 µg/L and the chronic criterion is 0.0038 µg/L. The chronic criterion is based on marketability of fish for human consumption, and is the water concentration of heptachlor estimated to ensure that tissue concentrations are below the FDA action level of 0.34 mg/kg for edible fish (EPA 1980i). This expected tissue concentration is unlikely to represent concentrations that would occur in salmon or steelhead tissues because the BCF of 5,220 was not derived from data on salmon or steelhead. It should also be noted that the most stringent heptachlor criteria that are applicable critical habitats are the human health criteria based on fish consumption rather than the chronic aquatic life criteria. The fish consumption based criteria are 10 times more restrictive than the aquatic life criteria and are applicable to all waters with listed salmon and steelhead.

Acute Heptachlor Criterion. The acute heptachlor criterion of 0.52 µg/L was derived from acute LC_{50} values for 18 species of freshwater fish and invertebrates, based primarily on static laboratory exposure tests, and represents the 5th percentile of the mean species values for this group of animals (EPA 1980i). Heptachlor concentrations in water were not measured in any of these tests; reported exposure concentrations were nominal. Acute toxicity to salmonids occurs generally when concentrations are at least an order of magnitude greater than the proposed acute criterion. For example, LC_{50}s have been reported as 81.9 µg/L, 24.0 µg/L, and 7.4 µg/L to 26.9 µg/L for coho salmon, chinook salmon, and rainbow trout, respectively (Johnson and Finley 1980; EPA 1980i; Macek *et al*. 1969; Katz 1961). Reported 96-hour LC_{50} values in other fish species have ranged from 5 µg/L to 25 µg/L (Johnson and Finley 1980).

Although measured LC_{50} values for salmonids appear to be substantially above the proposed criterion, there is evidence that the corresponding tests, which involved static exposures at nominal concentrations, may have significantly underestimated the toxicity of heptachlor. In the EPA criteria documents for heptachlor (EPA 1980i), LC_{50} values for saltwater fish ranged from 0.85 µg/L to 10.5 µg/L in flow-through, measured concentration tests (Hansen and Parrish 1977; Schimmel *et al*. 1976; Korn and Earnest 1974), but were as high as 194 µg/L in static, unmeasured tests (Eisler 1970). Notably, the saltwater criterion, based on both types of tests, is 0.053 µg/L which is an order of magnitude lower than the freshwater criterion. Thus, the acute toxicity data for salmonids may underestimate actual toxicity of heptachlor. Still, the criterion of 0.52 µg/L is substantially lower than the lowest reported LC_{50} concentration of 10 µg/L, and this difference probably provides an adequate margin of safety against acutely lethal effects of heptachlor.

Chronic Heptachlor Criterion. Little information is available on the sublethal effects of heptachlor in salmonid species. Carr *et al*. (1999) reported that in channel catfish, heptachlor epoxides, and to a lesser extent heptachlor, bind to the gamma-aminobutyric acid 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 bluegill (*Lepomis macrochirus*) liver mitochondria and found that it significantly inhibited ATP hydrolysis in an in-vitro assay. The lowest effective

concentration was 0.00056 g/ml of reaction medium, but how that would compare to water concentrations affecting a live animal is not clear.

Chronic toxicity data are correspondingly limited 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; Radhiah, *et al*. 1986; Radhaiah 1987; Azharbig *et al*. 1990; Konar *et al*. 1970; 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. Tests with non-salmond 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.

2.4.20.2. Habitat Effects of Heptachlor Criteria

Toxicity to Food Organisms. There is little data available on the effects of long-term exposures of heptachlor to salmonid prey. Heptachlor is acutely toxic to freshwater aquatic invertebrates at concentrations comparable to those that are lethal to fish (Johnson and Finley 1980; HSDB 1995). Reported LC_{50} values for freshwater invertebrate species have included 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 daphnids (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.

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. Naik *et al*. (1997) determined that heptachlor induced changes in the rate of oxygen consumption and acetylcholinesterase activity in the central nervous system of a freshwater leech *Poecilobdella viridis* within 2 hours, at concentrations ranging from 0.7 µg/L to 3.5 µg/L, the lowest of which is very close to the current acute criterion of 0.52 µg/L. 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.

Bioaccumulation. Both heptachlor and heptachlor epoxide have been shown to bioconcentrate in aquatic organisms such as fish, mollusks, insects, plankton, and algae (ATSDR 1989). They have been found in the fat of fish, mollusks, and other aquatic species at concentrations of 200 to 37,000 times the concentration of heptachlor in the surrounding waters (WHO 1984; ATSDR 1989). A wide range of BCFs have been determined in laboratory studies using fish (EPA 1980i). No BCF values are available for salmonids, but values for fathead minnow range from 9,500 to 14,400 (Veith *et al*. 1979; EPA 1980i). Goodman *et al*. (1976) reported average bioconcentration factors for heptachlor of 3,600 for sheepshead minnow. Uptake of heptachlor by aquatic organisms is influenced by a number of environmental and water quality factors (Vanderford and Hamelick 1977) including concentrations of organic particulate matter in the water column, turbidity, and season of the year. Residue concentrations may also vary considerably between fish species.

Uptake and Toxicity through Alternate Routes of Exposure. Because heptachlor is no longer in use in the United States, except for selected special applications, the primary potential source of this compound will be from repositories of the contaminant that are persistent in sediments not from point source discharges into surface water bodies. This means that if present, heptachlor and heptachlor epoxide would likely be taken up through direct contact with sediments or through the diet not only through the water column. Thus, studies evaluating the effects of water-borne exposure alone are likely to under-estimate actual exposure of organisms in the field.

Because sediments are likely the primary source of heptachlor, the sediment heptachlor concentration that would result in heptachlor concentrations in the water column at or below the criteria is of interest and can be calculated per Section 2.4.13. 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. This would mean that for sediment TOC levels of 1% to 5%, the sediment heptachlor concentrations would range from 54 ng/g to 269 ng/g sediment. These levels are higher than the sediment screening guideline of 10 ng/g dw established by the COE for in-water disposal of dredged sediment (COE 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 (CCME 2001). 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. Van der Oost *et al*. (1996) examined biota-sediment ratios of heptachlor in feral eel (*Anguilla anguilla*) and found a large variation in BSAF values between different sites, suggesting that inter-site differences in contaminant bioavailability or in the diets of resident fish could have a strong influence on heptachlor uptake. Without site-specific BSAFs for heptachlor in salmonids, it is difficult to determine if the proposed chronic water quality criterion would be sufficiently protective. The highest levels found of heptachlor in Idaho were in Brownlee Reservoir with sediment levels of <.001ng/g.

2.4.20.3. Summary for Helptchlor

Available evidence indicates that listed salmon or steelhead experience acute lethal effects at concentrations much higher than the proposed acute criterion. However, all such evidence is derived from static tests with nominal heptachlor concentrations, a methodology that tends to underestimate toxicity. There is a greater likelihood that heptachlor could harm salmon or steelhead through lethal effects on aquatic invertebrates; however, little information is available on the effects on invertebrate prey species.

Data on chronic effects of heptachlor are sparse, but suggest that the risk of adverse effect through water-borne exposure is likely to be low. Some studies suggest that tissue concentrations that are possible under the chronic criterion could have sublethal or lethal effects on alevins or fry. Bioaccumulation can occur in salmonids with chronic exposure to heptachlor, and when exposure occurs, it is likely to be not only through the water column but through diet and contact with sediments.

2.4.21. The Effects of EPA Approval of Lindane (gamma-BHC) Criteria

On August 2, 2006, EPA announced that the registrants of lindane requested to voluntarily cancel all remaining pesticide registrations of lindane and so there are no remaining uses in the United States.

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 tetrachlorophenols and conjugation with sulfates or glucuronides. Other pathways involve the ultimate formation of mercapturates which are water soluble end-products eliminated via the urine (Smith 1991). Of the isomers, g-HCH is stored to the greatest extent in fat (Smith 1991).

In mammals, the major effects of acute exposure to lindane include central nervous system stimulation, mental and motor impairment, excitation, convulsions, increased respiratory rate or respiratory failure, pulmonary edema, and dermatitis. Effects on the gastrointestinal,

musculoskeletal, liver, kidney, and immune systems have also been reported (Smith 1991; Kidd and James 1991). Chronic exposure to lindane has been associated with effects on the blood (decrease in numbers of red and white blood cells); on the musculoskeletal, immune, and nervous systems; and on the liver and kidney (Smith 1991; Matsumura 1985). Reproductive effects such as decreased sperm count may also be possible (Smith 1991). Available data on the mutagenicity and carcinogenicity of lindane are somewhat contradictory (Smith 1991).

2.4.21.1. Species Effects of Proposed Lindane Criteria

The proposed acute criterion for lindane is 2 µg/L. The proposed chronic criterion is 0.08 µg/L (Table 1.3.1).

Acute Lindane Criterion. Johnson and Finley (1980) reported an LC_{50} value of 1.7 µg/L for brown trout, indicating that the acute criterion could allow mortality to salmonids. For most salmonids and other fish species, however, LC_{50} values are more than an order of magnitude greater than the proposed acute criterion of 2 µg/L. Johnson and Finley (1980) reported 96-hour LC_{50} values of 23 µg/L, 27 µg/L, and 32 µg/L, for coho salmon, rainbow trout, and lake trout, respectively, in static exposure tests. Values for other fish species (goldfish, carp, fathead minnow, black bullhead, channel catfish, green sunfish, bluegill, largemouth bass, and yellow perch) range from 32 µg/L to 131 µg/L (Johnson and Finley 1980). Schimmel *et al*. (1977) conducted flow-through, 96-hour bioassays to determine the acute toxicity of technical grade BHC and lindane to sheepshead minnow (*Cyprinodon variegatus*), and pinfish (*Lagodon rhomboides*). The respective 96-hour LC_{50} values were 104 µg/L and 30.6 µg/L. A few studies show sublethal effects after acute exposure to lindane, but at concentrations well above the proposed acute criterion (e.g., Rozados *et al*. 1991; Soengas *et al*. 1997).

Most data determine LC_{50} values above the proposed acute criteria, although the low LC_{50} for brown trout reported by Johnson and Finley (1980) suggests the need for further testing. This is especially true in light of the fact that Johnson and Finley's (1980) values were based on static exposure tests with nominal (unmeasured) lindane concentrations, which could have under- or overestimated toxicity.

Chronic Lindane Criterion. The proposed chronic criterion for lindane is 0.08 µg/L. This was based on acute:chronic ratios calculated from LC_{50} data and whole life cycle tests fathead minnow, and did not incorporate data on chronic toxicity of lindane to salmonids (EPA 1980q). 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 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 dose causing 50% effects (ED_{50}) for growth effects was 5.22 mg/kg (Schimmel *et al*. 1976).

Tissue concentrations of lindane in fish exposed to the concentrations of lindane in the water column at the proposed criteria concentration can be calculated from EPA's estimated BCFs for lindane. Multiplying the proposed chronic criterion by the geometric mean of BCF values for lindane (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) in salmonids could be relatively close to those predicted based on the proposed chronic criterion (1.68 mg/kg). However, despite this calculations using mean BCFs, the water concentration that actually produced Macek *et al.*'s (1976) tissue residues were far higher than the chronic criterion (16 vs. 0.08 µg/L, above).

Some studies have also been conducted in which lindane was administered through feeding or injection. 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 being reviewed.

***Factors affecting the Toxicity of Lindane*.** Water hardness does not seem to alter the toxicity of lindane to fish. In some experiments, increased temperature caused increased toxicity for some species and decreased toxicity for others (Johnson and Finley 1980).

2.4.21.2. Habitat Effects of Proposed Lindane Criteria

Toxicity to Food Organisms. Available data on the acute toxicity of lindane to aquatic invertebrates suggest that the proposed criterion of 2.0 µg/L may be protective of most types of salmonid invertebrate prey. Reported 96-hour LC_{50} values are on the order of approximately two to three 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 (Ferrando *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 targeted (unmeasured) 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 are more than 25 times the proposed criterion of 0.08 µg/L. For example, for the amphipod, *Hyallela 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 range (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).

Bioaccumulation. 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. 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 1991; ATSDR 1989). Bioconcentration factors determined in laboratory studies with fish have ranged from 35 to 486, with the 486 value determined for rainbow trout (EPA 1980q). No BCF values were found for salmon.

Uptake and Toxicity Through Alternate Routes of Exposure. Because there are no registered uses of lindane in the United States, the only 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.

Because sediments are likely the primary source of lindane, the sediment lindane concentration that would result in lindane concentrations in the water column at or below the proposed criteria can be calculated per Section 2.4.13. For lindane, $\log_{10}$ (K_{ow}) = 3.3, $\log_{10}$ (K_{oc}) = 3.24, and F_{cv} = 0.08, resulting in SQC_{oc} = 0.14 mg/kg organic carbon. This would mean that for sediment TOC levels of 1% to 5%, the sediment lindane concentrations would range from about 1 ng/g to 7 ng/g sediment. These values are about an order of magnitude below the sediment screening guideline of 10 ng/g dry wet established by the COE for in-water disposal of dredged sediment (COE 1998), and are approximately at the level of the interim Canadian Sediment Quality Guidelines (SQG) of 0.32 ng/g to 0.99 ng/g dry wt 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 (CCME 2001). This suggests that the proposed criterion is reasonably likely not to harm salmonids or impact their prey items, although there is some uncertainty since tests used to establish these sediment guidelines were not specific to salmon and their prey.

Data on sediment toxicity of lindane are limited. Most studies suggest that adverse effects to benthic invertebrates that could serve as salmonid prey occur at much higher concentrations. For example, studies show effects on larval growth and adult emergence in chironomids at 2000 ng/g dry wt sediment (Watts and Pascoe 2000). Similarly, Ciarelli *et al*. (1997) reported 10-day LC_{50} values of 780 ng/g to 1490 ng/g dw sediment for the amphipod, *Corophium valutator*.

2.4.21.3. Summary for Lindane

There are not current registered used of lindane in the United States and no known contamination of sites in Idaho at levels that may impact listed salmonids. Most of the available data tend to show adverse effects to listed salmonid species, or their close relatives, or their prey at greater than criteria concentrations. The reliability of a single acute test reporting mortalities at concentrations lower than the acute criterion is uncertain since targeted exposure concentrations were not verified by chemical analysis (i.e., were nominal concentrations).

2.4.22. The Effects of EPA Approval of the Polychlorinated Biphenyl Criterion

Polychlorinated biphenyl (PCBs) were produced by the Monsanto Company and were marketed under the trade name of “Aroclor” using a numbering designation of four digits to identify the different commercial mixtures. For example, "12" was used as the first 2 digits for PCB mixtures and the last two digits identified the percent chlorine by weight of the mixture (e.g., the PCB mixture Aroclor 1254 contains 54% chlorine by weight). Aroclor 1254 is one of the most common PCB mixtures that persists widely as a gobal pollutant. Polychlorinated biphenyls are common in urban waterways and can occur in high concentrations in biota and cause a variety of

biological effects. Polychlorinated biphenyl production in the United States was banned by Congress in 1979.

Many biological responses in laboratory animals have been reported for PCBs, including mortality, impaired growth and reproduction, immune dysfunction, hormonal alterations, enzyme induction, neurotoxicity, behavioral responses, disease susceptibility, and mutagenicity. While some biological responses, such as mortality, growth inhibition, and reproductive impairment, have measurable impacts on a population (Forbes and Calow 1999), other endpoints, such as altered hormone levels or induced enzyme systems, also have adverse physiological effects on species, thereby reducing their fitness. For example, thyroid function is associated with many physiological processes in fish metabolism. As noted by Mayer *et al*. (1977), thyroid metabolism plays a role in respiration, carbohydrate and ammonia metabolism, oxygen consumption, nervous system function, and behavior.

Impairment of these vital functions may affect the ability of fish to tolerate normal environmental fluctuations, including the physiologically demanding process of smoltification. A few studies have demonstrated that PCBs affect the thyroid hormones important for smoltification in salmon (Mayer *et al*. 1977, Folmar *et al*. 1982). Several physiological parameters (e.g., ATPase levels in the gill, thyroid and pituitary hormones, liver glycogen, blood glucose, and lipid metabolism) change during the parr to smolt transformation in salmonids (Wedemeyer *et al*. 1980). Alteration of any associated physiological functions may substantially reduce the chances of successful smoltification and the individual's ability to survive, thrive, and mature in the marine environment.

Variation in the PCB mixture is associated with variation in toxicity response, which is likely due to variable congener makeup and interspecies variation in uptake and elimination rates of the different congeners. Mayer *et al*. (1977) tested three fish species exposed to four different Aroclor mixtures and found a large (10- to 100-fold) range in LC_{50} values depending on the period of exposure and species. This observation was somewhat different from that reported by DeFoe *et al*. (1978) who showed similar LC_{50} values for fathead minnows exposed to Aroclors 1248 and 1260, which may be indicative of the range of species-related differences.

2.4.22.1. Species Effects of PCB Criterion

Idaho has defined a chronic AWQC of (0.014 µg/L), but not an acute criterion. A recreational use criteria based on fish consumption criteria is also applicable to all waters in Idaho with anadromous fish and is more than100 times more restrictive than the chronic aquatic life criterion of (0.000045 µg/L).

Acute PCB Criterion. There is no acute criterion for PCBs.

Chronic PCB Criterion. The proposed chronic criterion for PCBs is 0.014 µg/L in freshwater (Table 1.3.1). Data in the AQUIRE database (EPA 2001) and presented in literature reviews (Niimi 1996; Monosson 2000) indicate that water concentrations in the 0.1 µg/L to 10 µg/L range can be associated with sublethal, adverse effects in fish. One of the lowest response

concentrations for a salmonid was reported by Mauck *et al*. (1978) who demonstrated that backbone composition of phosphorus and hydroxy-proline was altered significantly in brook trout fry exposed to Aroclor 1254 at a concentration of 0.4 µg/L. A slightly higher concentration (0.69 µg/L) also affected collagen and calcium levels in the backbone of fry. In the case of non-salmonids, a study on reproduction in fathead minnows found that larvae were the most sensitive life stage (DeFoe *et al*. 1978). Additionally, when the second generation of fish were examined, mortality and growth were significantly affected at 0.4 µg/L indicating greater sensitivity for offspring of adult fish subjected to chronic exposure.

Factors Affecting the Toxicity of PCBs. In recent work it has been shown that some PCB congeners are considerably more toxic than others, which is primarily a function of the position of the chlorine atoms and their ability to interact with the aryl hydrocarbon (Ah) receptor. This is more a concern for vertebrates, including fish, than invertebrates which generally lack this receptor and are not sensitive to the "dioxin-like" effects of PCBs. The most toxic PCBs are the non-ortho and mono-ortho substituted congeners, which tend to be planar compounds. Some toxicological responses such as developmental and reproductive abnormalities, enzyme induction, and immunosuppression can occur at extremely low concentrations and are likely caused by "dioxin-like" PCB congeners (planar congeners). These planar congeners can occur in the Aroclor mixtures, but usually at low concentrations. The responses caused by the non-planar congeners ("non-dioxin-like") are likely due to different modes of action and include neurotoxicity, hypothyroidism, carcinogenicity, behavioral alteration, and endocrine disruption (Giesy and Kannan 1998).

The TEF approach has been used to determine the relative toxicity of the planar PCB congeners as a fraction of that elicited by 2,3,7,8 Tetrachlorodibenzo-p-dioxin (TCDD). Tissue concentrations of PCB congeners are multiplied by the TEF to generate a toxicity equivalence quantity (TEQ) concentration in terms of its "dioxin-like" potency. These TEQs are then summed to generate a total TEQ concentration for the sample that can be compared to dioxin toxicity results. Ideally, the TEFs should be species and endpoint-specific because of the observed variability (Giesy and Kannan 1998). The TEF approach is not applicable for those "non-dioxin-like" biological responses caused by the non-planar PCB congeners, primarily due to the different modes of action. The TEF approach is not valid for invertebrates because they generally do not contain the aryl hydrocarbon receptor that would cause dioxin-like toxicity.

Most TEFs have been developed for mammals and birds, and only recently have any been developed for fish (Walker and Peterson 1991). The TEFs for fish are somewhat limited because they apply only to ELS mortality in salmonids and enzyme induction (Giesy and Kannan 1998). There are no TEFs for biological effects occurring beyond the embryo/alevin state. For fish, TEFs have been developed for non-ortho PCBs, but not for the ortho-substituted congeners because of a general lack of biological activity (Giesy and Kannan 1998; Van den Berg *et al*. 1998). Table 2.4.22.2 lists TEFs for fish based on ELS mortality due to injection of congeners into eggs (Van den Berg 1998).

Table 2.4.22.2. Reported Toxicity Equivalent Factors (TEFs) for the early life stage of fish

PCB Conger	IUPAC No.	TEF
3,3',4,4'	77	0.0001
3,4,4',5-	81	0.0005
3,3',4,4',5-	126	0.005
3,3',4,4',5,5'	169	0.00005

The values in Table 2.4.22.2 are generally one to two orders of magnitude lower than those reported for mammals and birds (Van den Berg *et al*. 1998). Polychlorinated biphenyl congener data are not available for fish tissue samples, especially eggs. Application of TEFs therefore provides less accurate toxicity response information for this life stage. Walker and Peterson (1991) conducted a dose response study with rainbow trout eggs and determined the TCDD LD_{50} to be 0.23 ng/g, with very low mortality occurring at 0.1 ng/g. Based on this work, a TEQ value above 0.1 ng/g egg may therefore not be protective against ELS mortality. It is uncertain if concentrations below 0.1 ng/g in eggs may lead to adverse effects. This approach could be valid for many fish species, although differences may exist between species (Monosson 2000).

This information could be used to assess toxicity when congener-specific toxicity information becomes available for biological responses relevant to salmonid life stages beyond the early life stages. For example, a recent study demonstrated a significant increase in mortality for adult rainbow trout exhibiting a muscle tissue concentration of 2,3,7,8 TCDD of only 0.00044 ng/g ww (Jones *et al*. 2001).

2.4.22.2. Habitat Effects of PCB Criterion

Toxicity to Food Organisms. One comprehensive study of PCB toxicity to freshwater invertebrates found responses at relatively low concentrations. Nebeker and Puglisi (1974) examined eight Aroclor mixtures and their effects on survival and reproduction in *Daphnia magna*, *Gammarus pseudolimnaeus* (amphipod), and *Tanytarsus dissimilis* (midge), which are all potential prey for salmonids. The midge was the most sensitive invertebrate studied, with 21-day LC_{50} values at 0.63 µg/L for larvae and 0.45 µg/L for pupae (Aroclor 1254). Data contained in the EPA's AQUIRE database for toxic effects of PCBs on aquatic organisms indicate that invertebrates are affected by water concentrations of PCBs in the 0.5 µg/L to 5 µg/L range, which is at least an order of magnitude above the chronic AWQC.

Bioaccumulation. With very high BCFs, it takes only a few µg/L in water to cause tissue concentrations of PCB in the range considered lethal. In addition, many studies have demonstrated that salmonids absorb about 50% of PCBs available in their diet. Madenjian *et al.* (1999) reported the efficiency of retention by coho salmon through dietary uptake of various PCB congeners ranged from 38% to 56%. Similar results were also reported by Gruger *et al*. (1975, 1976) for coho salmon and by Opperhuizen and Schrap (1988) for guppies and other fish species. In a long-term study with rainbow trout, Lieb *et al*. (1974) fed trout PCB-laden pellets for 32 weeks. Fish grew from 0.8 grams to approximately 75 grams and the percent retention of

PCBs was determined to be 68%. The authors also determined that the ratio between the ww PCB concentration in fish and the PCB concentration in dry food was 0.54.

The BCFs, which indicate the relative partitioning between water and tissue, are governed by the balance between the rates of uptake and elimination and can be altered by changes in either rate. Mackay *et al*. (1992) reported an average $\log_{10}$ (BCF) value equal to 4.9 for fish exposed to PCBs (Aroclor 1254). However, because of the large variability in congener hydrophobicity, BCF values for fish range almost four orders of magnitude. The determination of tissue residues from water exposure is consequently extremely uncertain because of the large variability in BCFs for PCB congeners, and there is no one BCF suitable for Aroclor mixtures (Bremle *et al*. 1995).

For example, Berlin *et al*. (1981) reported significantly more mortality in lake trout fry exposed to Aroclor 1254 when tissue concentrations were 1.5 ppm wet weight. Folmar *et al*. (1982) found altered thyroid hormones in coho salmon exposed to 0.1 mg/L of Aroclor 1254 in tissue, which would influence the smoltification process and a smolt's ability to osmoregulate in marine waters. Another study with coho salmon also found effects on thyroid activity, as determined by uptake of iodine, when the whole-body tissue concentration of Aroclor 1254 reached 0.6 mg/kg ww (Mayer *et al*. 1977). If the average BCF noted above (i.e., $\log_{10}$ (BCF) = 4.9) were applied to the data reported in Mayer *et al*. (1977), a water concentration of 0.007 µg/L would be estimated to produce a tissue concentration of 0.6 mg/kg. This water concentration is half the proposed chronic criterion.

The BAF may be modified as a BSAF to include lipid-normalized tissue and organic carbon normalized sediment concentrations with the following equation:

$$BSAF = [tissue]/[sediment] \times f_{oc}/f_{lip}$$

where:

[tissue] and [sediment] are respective concentrations
f_{oc} is the fraction of organic carbon (g/g)
f_{lip} is the fraction of lipid (g/g) (Meador 2006)

Equilibrium partitioning theory was developed to explain and predict the partitioning behavior between sediment, water, and tissue for neutral hydrophobic organic compounds (HOCs), such as PCBs and polycyclic aromatic hydrocarbons (PAHs; McFarland 1984; Di Toro *et al*. 1991). At equilibrium, the BSAF, which represents partitioning between these phases, has a theoretical maximum value of unity (1.0), whereas empirical maximum values range from 2 to 4 (Di Toro *et al*. 1991; EPA/USACOE 1991; Boese *et al*. 1995). For invertebrates and some fish, especially those associated with sediment, the PCB BSAF values are generally close to expected values (2 to 4; Bierman 1990; Tracey and Hansen 1996). The BSAF values for fish can also be close to expected values depending on the exposure time and variability in exposure concentration (Bierman 1990). However, few studies have reported PCB BSAFs for salmon because, for such a highly migratory species, it is exceedingly difficult to determine a relevant exposure concentration. The BSAFs were estimated for migrating juvenile chinook in the Duwamish

estuary system where individuals spend days to weeks feeding on abundant invertebrate populations (Meador *et al*. 2002). The BSAFs over years, location, and natural-origin versus hatchery fish were determined to be relatively consistent, ranging 0.10 to 0.16.

Uptake and Toxicity from Alternate Routes of Exposure. Polychlorinated biphenyls are typically not found in the water column at concentrations of concern because of their high affinity for sediment and biological tissues. It is possible for high sediment concentrations, and water concentrations that are below the chronic criterion or are undetectable, to co-occur in streams. Water quality criteria in such areas thus may have little relevance for assessing impacts to organisms that can accumulate high concentrations of these compounds from their diet. It is more relevant to assess impacts to biota based on tissue or sediment concentrations. Only a few studies have examined this approach for protecting aquatic life based on sediment concentrations, and fewer for tissue concentrations.

There are a number of empirical methods for assessing effects of sediment-associated contaminants and generating SQG (MacDonald *et al*. 2000b). One such method is the "Effects Range" approach, which ranks sediment concentrations associated with adverse effects observed in bioassays. Using a large database of bioassay experiments, the concentration associated with the 10^{th} percentile of all studies is termed the "Effects Range-low". The 50^{th} percentile is called the "Effects Range-median" (Long *et al*. 1995). These values are often used to determine the potential for a sediment concentration to cause adverse effects in biota. MacDonald *et al*. (2000b) have recently reviewed all such approaches and proposed unifying them into a consensus-sediment effect concentration (SEC), using total PCBs as an example. They proposed dividing SECs into three groups, the threshold, midrange, and extreme effect concentrations (TEC, MEC, and EEC, respectively). The TECs are concentrations below which adverse effects on sediment dwelling organisms are not expected, MECs are concentrations above which adverse effects are frequently observed, and EECs are concentrations above which adverse effects are usually or always observed. For freshwater ecosystems the following SECs were generated by MacDonald *et al*. (2000b) for total PCBs in sediment: TEC = 35 µg/kg, MEC = 340 µg/kg, and EEC = 1,600 µg/kg /kg (all in dry weights). Because these values are based on hundreds of bioassay experiments, they should be useful in identifying sediment concentrations in Idaho that may cause adverse effects in sediment-dwelling organisms exposed to total PCBs. In sediment surveys in various locations in the Snake River basin in Idaho, PCBs were less than 50 µg/kg (Table 2.3.1, Clark and Maret 1998). This indicates that PCB concentrations in sediment are likely close to, or below the TEC.

Characterizing the toxic effects caused by PCBs can be simplified by examining tissue concentrations associated with adverse effects. Variation in the toxic response can be reduced because of large differences in time of exposure, makeup of Aroclor mixtures, and differences in toxicokinetic abilities of species. Niimi (1996) and Monosson (2000) provide summaries showing the range in tissue concentrations associated with several different biological responses Niimi (1996) reported that fish tissue concentrations of PCBs greater than 50 mg/kg were associated with adverse effects to growth and reproduction. Monosson (2000) focused on reproductive and developmental effects of Aroclor 1254 and associated tissue concentrations, and determined that concentrations associated with adverse effects ranged from 5 ppm in whole bodies of larvae to 25 mg/kg in liver of adult fish (all wet weights). Additional analyses with

congener 77 indicated an effective concentration of 0.3 mg/kg in eggs. In a critical review of the literature, Meador *et al*. (2002) examined the toxic effects of total PCBs in salmonids and determined that the 10^{th} percentile value of 15 studies considered valid in the determination of a residue effect threshold for salmonids was 2.4 mg/kg lipid. Tissue residues below this were considered to be generally protective of salmonids.

2.4.22.3. Summary for PCBs

In the studies reviewed (above) water borne PCB concentrations close to, or below, the proposed chronic criterion, in concert with predicted bioaccumulation rates, were projected to result in impaired thyroid function in coho salmon and embryo mortality in lake trout. Even though the proposed chronic criterion may result in some effects to listed species, this appears unlikely to occur because the product is banned and no known contamination exists at levels of concern in Idaho areas with listed salmon and steelhead. If discharges do occur the most stringent controlling ambient water quality criterion applicable in designated critical habitats is the fish consumption based human-health criteria, rather than the chronic aquatic life criteria (Table 1.3.1). The fish consumption based criterion is more than100 times more restrictive than the aquatic life criteria. Therefore any effects from the proposed approval of the PCB criterion will have only very small effects on listed species and designated critical habitat.

2.4.23. The Effects of EPA Approval of the Toxaphene Criteria

Toxaphene is a trade name for a man-made organochlorine pesticide that consists of between approximately 177 and 670 congener compounds and has a chlorine content of 67% to 69%. Toxaphene is produced by combining camphene (a pine tree extract) with chlorine, and activating the mixture with ultraviolet radiation and catalysts. Only 26 congeners have been isolated, of which 10 have been identified. The 26 isolated congeners comprise approximately 40% of the toxaphene mixture. Toxaphene is also known as chlorinated camphene and was listed under other trade names including Alltex, Estonox, Motox, Anatox, Penphene, and Geniphene. Toxaphene was first introduced in 1947 and used extensively as an insecticide in the 1970s after DDT was banned. The pesticide was used primarily in the southern United States to control insects on cotton and livestock, and to kill undesirable fish in lakes. Toxaphene was banned for most uses in 1982 and all uses in 1990 in the United States, but is still used on fruit crops in other countries.

Toxaphene exhibits a relatively low $\log_{10}$ octanol-water partition coefficient at 3.3, but is very persistent in the environment, with a reported half-life in soil between 1 and 14 years. In water it will not appreciably hydrolyze, undergo photolysis, or biodegrade. Degradation is faster under anaerobic than aerobic conditions. Evaporation from the aqueous phase is a significant process for toxaphene dispersion, with a half-life of approximately 6 hours. Once it has volatilized, toxaphene can be carried far from the original site.

The EPA has determined that exposure of animals to toxaphene potentially affects the central nervous system, and that chronic exposure also has the potential to affect the liver and kidney,

suppress the immune system, and cause cancer and endocrine disruption. There are reports that it may also have antiestrogenic activity and inhibit the binding of estrogen, progesterone, dexamethasone, and testosterone to their respective receptors (Yang and Chen 1999; ; Hood *et al*. 2000).

2.4.23.1. Species Effects of Toxaphene Criteria

The proposed acute criterion for toxaphene is 0.73 µg/L in freshwater, and the chronic criterion is 0.0002 µg/L.

Acute Toxaphene Criterion. The BA provided by EPA only reported acute toxicity studies on fish that showed effects at concentrations above the proposed acute criterion. Acute effects reported by EPA occurred at 2 µg/L in bass, 2.4 to 29 µg/L in bluegill, 3.1 µg/L in brown trout, and 18.0 µg/L in fathead minnows. Studies not reported in the BA suggest that fish mortality may occur at toxaphene concentrations that are relatively close to the proposed acute criterion, due to the proximity of the LC_{50} values to the proposed criterion. Schimmel *et al*. (1977) reported 96-hour LC_{50}s of 1.1 µg/L and 0.5 µg/L for the sheepshead minnow (*Cyprinodon variegatus*) and pinfish (*Lagodon rhomboides*), respectively. Macek and McAllister (1970) listed 96-hour LC_{50}s for 12 fish species from five families exposed to toxaphene, ranging from 2 µg/L to 14 µg/L. Two salmonids were tested, the brown trout and the coho salmon, with LC_{50} values (and 95% confidence intervals) of 3 µg/L (2 µg/L to 5 µg/L) and 8 µg/L (6 µg/L to 10 µg/L), respectively. Johnson and Finley (1980) reported 96-hour LC_{50}s for toxaphene to coho salmon, rainbow trout, and brown trout of 8.0 µg/L, 10.6 µg/L, and 3.1 µg/L, respectively. Schoettger (1970) reported a 96-hour LC_{50} for Chinook salmon of 1.5 µg/L, which is within a factor or two of the proposed acute criterion. These results are very similar to those reported by Katz (1961) for rainbow trout, chinook and coho salmon.

Chronic Toxaphene Criterion. No studies were found that documented chronic effects when toxaphene concentrations were below the chronic criterion of 0.0002 µg/L. Mayer and Mehrle (1977) and Mayer *et al*. (1975) reported that water concentrations of 0.039 µg/L had significant effects on survival and growth in brook trout fry. The tissue concentrations associated with these responses were only 0.4 mg/kg dw. Other treatments in these studies (0.068 µg/L, 0.14 µg/L, 0.29 µg/L, and 0.5 µg/L) also caused adverse effects in this species. Tissue concentrations for these treatments ranged from 0.2 mg/kg to 8 mg/kg dw. Similar studies by these authors also found adverse effects in fathead minnow at similar water exposure concentrations (Mayer *et al*. 1977; Mayer and Mehrle 1977). An examination of the AQUIRE database on sublethal effects from toxaphene exposure indicated several other studies showing sublethal effects on fish in the 0.03 µg/L to 1 µg/L range (EPA 2001).

Sublethal effects reported by EPA in the BA suggest that reduced reproduction, growth inhibition and histopathology of the kidney and intestinal tract could occur from acute exposure, based on occurrence of these effects in fish at concentrations as low as 0.054 µg/L. Growth inhibition and reduced reproduction were reported in brook trout exposed for 161 days to 0.288 µg/L and 0.068 µg/L, respectively (Mayer *et al.* 1975, 1977a).

Factors Affecting Toxicity. Like PCBs, toxaphene is made up of a large number of congeners, which may vary in toxicity and mode of action, and whose biological effects are not well-characterized. Stelzer and Chan (1999) found differences in the estrogenic activity of a technical toxaphene mixture compared with two congeners that are prominent in humans. Toxaphene may also show interactive effects with other pesticides or environmental contaminants (Chaturvedi 1993).

2.4.23.2. Habitat Effects of Toxaphene Criteria

Toxicity to Food Organisms. Based on available literature, it appears that invertebrates are less sensitive to toxaphene exposure than fish. Results from the AQUIRE database (EPA 2001) show LC_{50}s for freshwater invertebrates ranging from 5 µg/L to 20 µg/L, which is similar to values for saltwater studies in which oysters and shrimp exhibited 96-hour LC_{50}s ranging from 1.4 µg/L to 16 µg/L (Schimmel *et al*. 1977). However, one study with stonefly naiads reported LC_{50} values in the low µg/L range. The 96-hour LC_{50} for *Pteronarcys californica*, *P. badia*, and *Claassenia sabulosa* were 2.3 µg/L, 3.0 µg/L, and 1.3 µg/L, respectively (Sanders and Cope 1968). These values are within a factor of 2 or 3 of the proposed acute criterion, and even closer when the 95% confidence intervals are considered. This study also reported data for the 24- and 48-hour LC_{50}s and found that these values generally decreased by a factor of two for each time point (e.g., 24-hour, 48-hour, and 96-hour LC_{50}s), indicating that steady state had not been reached and that lower LC_{50} values would likely occur with more exposure time. In general, sublethal effects occur at much lower concentrations than those causing mortality. Sanders (1980) reported adverse effects to growth of the amphipod (*Gammarus pseudolimnaeus*) in exposure concentrations of 0.2 µg/L, and a 96-hour LC_{50} of 24 µg/L. Sanders (1980) also reported a reduction in reproduction in *Daphnia magna* at 0.12 µg/L. These chronic values are orders of magnitude higher than the chronic criterion (0.0002 µg/L), indicating that no chronic effects from long-term exposure to toxaphene are expected for invertebrate prey. Toxaphene may cause endocrine-disrupting effects in invertebrate prey species (Hood *et al*. 2000), but the exposure levels associated with these effects have not been quantified. However, based on the acute response data and the small differences between 96-hour LC_{50}s and the CMC value, adverse effects to invertebrates are possible for short-term exposures similar to the CMC.

Bioaccumulation. Bioconcentration factors are very high for toxaphene. Mayer *et al*. (1975) reported BCFs for brook trout ranging from 5,000 to 76,000, and Terriere *et al*. (1966) determined BCFs for rainbow trout to range from 10,000 to 20,000. The AQUIRE database reported BCFs for Atlantic salmon ranging from 4,400 to 11,000 (EPA 2001). Environment Canada (1997) summarized several studies listing BCFs from 3,000 to 76,000 for fish, 400 to 1,200 for some crustaceans, and 7,000 to 10,000 for other groups such as algae and snails. Schimmel *et al*. (1977) also reported BCFs up to 60,000 for juvenile killifish *Fundulus similis*, and a range of 3,100 to 20,600 for other fish.

Toxaphene is biomagnified up the food web by several species (Eisler 2000) and it has been demonstrated in several studies that tissue concentrations increase with trophic level. Evans *et al*. (1991) reported a biomagnification factor of five from plankton to fish.

2.1.23.3. Summary for Toxaphene

Based on available information, toxaphene, under most circumstances, appears unlikely to cause lethal or sublethal effects from direct exposure at toxaphene concentrations in water equal to or below the proposed acute or chronic criteria.

2.5. 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 Act.

According to the most recent census, between 2000 and 2010, the cumulative population in the nine central Idaho counties with anadromous fish increased by 5.8%.[7] NMFS therefore assumes that future private and state actions will continue within the action area with a slight increase from their current rate. Seventy-one percent of the action area is Federally-owned, which somewhat limits possible cumulative effects from private and state actions. However, private land is often clustered in valley bottoms, adjacent to occupied habitat for ESA-listed species.

NMFS is aware of several potential future state and private actions in the action area that may benefit ESA-listed species. The *Draft Recovery Plan for Idaho Snake River Spring/Summer Chinook and Steelhead* recommends habitat restoration projects on private lands throughout the action area. The current draft is posted online at *http://www.westcoast.fisheries.noaa.gov/protected_species/salmon_steelhead/recovery_planning_and_implementation/snake_river/snake_river_salmon_recovery_subdomain.html.* Idaho Department of Lands is working with NMFS to develop a proposed Idaho Forestry Program, which aims to reduce the impacts of state and private forestry on stream habitat through road maintenance and stream buffer measures. The state of Idaho is also working with NMFS and irrigators on measures to reduce the impacts of water withdrawals on stream habitat in watersheds in the Salmon River.

It is reasonable to assume that future mining or municipal development will occur on state, private or tribal lands within the action area that may result in discharges of arsenic, copper, cyanide, mercury, nickel, selenium, silver, chromium III, chromium VI, lead, and zinc to waters of the state that contain listed species. However, many of these activities will be subject to section 7 consultation and are therefore not considered cumulative effects. Additionally, cleanup and closure activities for contaminated sites may also occur in the future and some of these will be on private and state lands and are considered cumulative effects.

Cyanide discharges may also occur as a result of future activities on state, private or tribal lands within the action area from activities like road salting.

[7] U.S. Census Bureau, State and County Quickfacts. Available http://quickfacts.census.gov/qfd/states/16000.html.

Mercury discharges may also occur as a result of future atmospheric deposition.

Pentachlorophenol discharges may occur when treated wood is used in or near water for construction activities or when it is used as a restricted use pesticide for activities on private, state or tribal lands.

Continued agriculture and forestry activities on private land are also likely to occur in the future. This will result in continued use of pesticide and fertilizers. It will also result in continued water diversions for agriculture that reduce flow rates and alter habitat throughout freshwater systems. 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, 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.

NMFS also expects the natural phenomena in the action area (*e.g.*, 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.

2.6. 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 2.4) to the environmental baseline (Section 2.3) and the cumulative effects (Section 2.5) 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 2.2).

Hardness Floor

Exposure of listed Snake River salmon and steelhead to levels of metals in discharges at proposed criteria levels will result in adverse effects. Many of the streams in the Salmon River and Clearwater River drainages of Idaho also have hardness concentrations that average less than 25 mg/L which is the current floor in the hardness equation. For copper and lead, hardness is less important than DOC, but if DOC is low, toxicity does increase below the hardness floor. For nickel, and zinc, acute toxicity to fish rises as hardness declines below the 25 mg/L. For silver, acute toxicity increases modestly in early life stages, below the hardness floor.

The use of a hardness floor of 25 mg/l in calculating metals discharge limits will allow for increased exposures of listed fish to levels of metals that result in adverse effects. These effects range from a direct increase in mortality to decreases in growth and survival of juvenile Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River Sockeye salmon and Snake River Basin steelhead.

It reasonable to assume that listed Snake River spring/summer Chinook salmon and Snake River Basin steelhead will be exposed to levels of metals that are harmful to fish based on exposures to metals that are currently occurring in the action area. These exposures are also described in more detail in the sections that follow for each metal. However, is not possible to estimate within the ESU the number of locations where future metals discharges will overlap with areas that also have low water hardness values. Some examples of current discharges that meet both criteria are shown in Table 2.4.2.1. Two of these discharges are into the mainstem of Panther Creek and Yankee Fork of the Salmon River and have the potential to affect nearly all of the fish that occupy the population due to their location low in the watershed. It is reasonable to assume that future discharges may be located similarly in these areas or in a location that affects a different population in a similar fashion. Because of this, it is reasonable to assume that a large percentage of at least one population of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon or Snake River Basin steelhead within their respective ESUs will be exposed to levels of metal toxicity in early life phases which may reduce egg survival in redds or reduce growth and survival of smolts in the exposed population.

Arsenic

Arsenic occurs in waters in Idaho both naturally and as a discharged pollutant. Arsenic is likely to be discharged in the future through mining or municipal sources so exposure to listed fish and critical habitat is likely to occur.

If only direct water exposures were considered, arsenic would be of minimal concern to listed salmonids at typical ambient concentrations or at the criteria concentrations under review. The risk of harm from short-term water-only exposures to arsenic concentrations at the acute criterion is unlikely enough to be considered a minor risk for short-term exposures.

The chronic criterion appears to avoid chronic adverse effects to the adult and juvenile salmonid life stages from water-only exposures; however, arsenic concentrations below the chronic criterion have been reported to cause mortality in salmonid embryos. The chronic arsenic

criterion is far higher than concentrations of arsenic sufficient to bioaccumulate in invertebrates to concentrations that cause harm to the salmonids that feed on them. Bioaccumulation of arsenic in prey organisms to concentrations that could be harmful to salmonids has occurred in streams at exposures less than 10 µg/L. As such, adverse effects are likely to occur at the chronic criterion, through reduced growth of juveniles via food web transfer.

It is reasonable to assume that listed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead will be exposed to levels of arsenic that are harmful to fish based on the possibility of future mining or municipal activities in the state. However, is not possible to estimate within the ESU the number of locations where future arsenic discharges and exposure may occur. Most likely these locations will be associated with a mine or a municipal discharge. It is also likely that one or more these discharges may be located within the area used by the majority of the fish in a single population, for example Panther Creek is discussed in the analysis and it had multiple discharges into river sections that are used by the entire Panther Creek population of Chinook at some point in their life cycle. Because of this it is reasonable to assume that a large percentage of at least one population of listed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead will be exposed to levels of arsenic approaching the chronic criteria during early life phases which will reduce egg survival in redds and reduce growth and survival of smolts in the exposed population.

Copper

Sources of copper such as mines, municipalities and stormwater runoff from highways exist in the action area currently and will likely be present in the future. It is also likely that copper will be found in new discharges or will be present in water bodies related to past activities.

The results of this analysis suggest that concentrations below the proposed acute and chronic criteria for copper can cause acute and chronic toxicity to salmon and steelhead. At the lower range of hardness values encountered in Idaho streams and lakes the acute standard could result in injury and death.

Listed salmon and steelhead can experience a variety of adverse effects at or below the chronic Idaho copper criterion. These include:

- Deprivation of chemosensory function which in turn causes maladaptive behaviors including the loss of ability to avoid copper, and the loss of ability to detect chemical alarm signals. Appreciable adverse effects can be expected with increases as small as 0.6 µg/L above background concentrations.

- Reduced growth in juvenile Chinook salmon and rainbow trout under conditions of low hardness and low organic carbon.

- Because survival of juvenile salmon and steelhead in their migration to sea is strongly size-dependent, small reductions in size will result in disproportionately larger reductions in survival during migration to sea. Using population modeling, growth reductions at the

chronic copper criterion were projected to result in slight increases in extinction risk and pronounced delays in recovery time in a model Chinook salmon population.

- The diversity and abundance of the macroinvertebrate food base for rearing juvenile salmon and steelhead could be reduced at copper concentrations near or below the Idaho chronic criterion.

While a variety of adverse effects relevant to listed salmonids have been demonstrated at copper concentrations less than the copper criteria under consultation, the most important issue is that the hardness-toxicity equation embedded into the criteria commonly results in fundamentally inaccurate and misleading indications of risk in critical habitats. This is because the best available science indicates that organic carbon is a more important mediator of copper risks than water hardness. During late summer or fall base flow conditions, copper would be expected to be most toxic because organic carbon tends to be low. Yet this is the time of year that hardness tends to be highest, and the hardness-based copper criteria wrongly indicate that copper would be of least risk at this time of year (Conclusion Section; Appendix C).

It reasonable to assume that listed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead will be exposed to levels of copper that are harmful to fish based on the possibility of future mining or municipal activities in the state. However, is not possible to estimate within the ESU the number of locations where future copper discharges and exposure may occur. Most likely these locations will be associated with a mine or a municipal discharge. It is also likely that one or more of these discharges may be located within the area used by the majority of the fish in a single population, for example Panther Creek is discussed in the analysis and it had multiple discharges into river sections that are used by the entire Panther Creek population of Chinook at some point in their life cycle. Because of this it is reasonable to assume that a large percentage of at least one population of Snake River spring/summer Chinook, Snake River fall Chinook salmon, Snake River sockeye salmon or Snake River Basin steelhead within their respective ESUs will be exposed to levels of copper approaching the acute or chronic criteria during early life phases which will reduce growth and survival of smolts in the exposed population.

Cyanide

It is likely that cyanide will be found in new discharges or will already be present in water bodies. Potential sources such as mines and forest fires exist in the action area currently and will be present in the future.

The proposed acute and chronic criteria can expose listed salmonids to harmful cyanide concentrations under specific situations. The acute criterion is not reliably protective when water temperatures drop to about 6°C or lower. Further, Leduc (1984) found that cyanide concentrations at the chronic criterion in water colder than 6°C may be associated with chronic toxicity effects. Temperatures in streams within the action area routinely drop below 6°C.

It reasonable to assume that listed Snake River spring/summer Chinook salmon and Snake River Basin steelhead will be exposed to levels of cyanide that are at or near the proposed standard.

However, it is not possible to estimate the number of locations where future cyanide discharges and exposure may occur. Most likely these locations will be associated with mining activities but other sources may also occur. It is also likely that one or more these discharges may be located within the area used by the majority of the fish in a single population; for example, Jordan Creek is discussed in the analysis and it had multiple discharges into river sections that were used by the entire Yankee Fork population of spring/summer Chinook at some point in their life cycle. Because of this it is reasonable to assume that a large percentage of at least one population of Snake River spring/summer Chinook or Snake River Basin steelhead within their respective ESUs will be exposed to levels of cyanide approaching the chronic criteria during early life phases which will reduce survival or juveniles as they overwinter and this will reduce abundance of smolts in the exposed population.

In separate reviews, USFWS (2010) and NMFS (2010b) evaluated the same cyanide criteria from a national perspective. Both described scenarios in which impaired reproduction from diverse species was extrapolated to effects on listed anadromous salmonids, through the use of interspecies correlation estimates of acute toxicity. Under these scenarios, adverse effects were considered by USFWS and NMFS as likely to jeopardize the continued existence of a variety of species, including Snake River salmon and steelhead. The findings and conclusions in the earlier draft biological opinions are similar to those reached here.

Mercury

Mercury toxicity in fish occurs by bioaccumulation through the food web. Direct toxicity from exposure to mercury in the water column is unlikely in the natural environment.

The chronic mercury criterion in the proposed action is based upon EPA's 1984 chronic criterion value (EPA 1985g). The 1984 chronic mercury criterion was back calculated from the FDA limit for allowable mercury content in commercially marketed seafood (1.0 mg/kg ww), using a bioconcentration factor derived from a laboratory water-only (aquaria) methylmercury exposures with fathead minnow (EPA 1985g). Thus, the criterion derivation had no consideration of ecological effects of mercury or effects of mercury to sensitive species. In the 25 plus years since this fish marketability-based criterion was developed, much new information on the effects of mercury on the fish themselves, not just their marketability, has been developed. The newer information both reflects that: (1) The older bioconcentration values considered in the 1984 chronic criterion were about four times lower than the average bioaccumulation factors obtained in field settings; and (2) that adverse developmental effects in fish occur at <1 mg/kg.

Severe adverse effects have been observed in fish that accumulated mercury in their muscle tissue, including brain damage, behavioral abnormalities, and reproductive failure. However, effects of methylmercury on fish are not limited to neurotoxicity, but also include histological changes in the spleen, kidney, liver and gonads. These effects have been observed in multiple species of freshwater fish at tissue concentrations of methylmercury well below 1.0 mg/kg ww (Sandheinrich and Wiener 2010).

The EPA has developed a fish-tissue based water quality criterion of 0.3 mg/kg for mercury to reduce human risks of eating mercury-tainted fish. Idaho has adopted this criterion, and is

implementing it as a 0.24 mg/kg a triggering residue concentration for existing dischargers, using an uncertainty (safety factor) of 0.8 times (IDEQ 2007a). This fish tissue-based criterion is unlikely to result in adverse effects to listed salmon and steelhead and their habitats. However, if mercury concentrations in rivers or lakes were allowed to approach the chronic water-based criteria of 12 ng/L, resulting mercury residues in fish could be about an order of magnitude higher than the selected threshold (~3 mg/kg ww).

It is reasonable to assume that listed Snake River salmon and steelhead will be exposed to levels of mercury that are harmful based on fish tissue information collected from other fish species within the state. Most likely these locations will be associated with mining or atmospheric deposition. Because of this it is reasonable to assume that a large percentage of at least one population within the ESU will be exposed to levels of mercury that will bioaccumulate and cause severe adverse effects including neurotoxicity and histological changes resulting in reduced abundance and productivity.

Nickel

Although nickel is rarely found in Idaho waters it does occur in some streams large enough to contain listed salmon and steelhead like the Panther Creek watershed. Therefore it is reasonable to assume that it has the potential to occur in other areas of the state where mining activities are likely to occur.

A striking feature of the information reviewed for nickel toxicity is the tremendous range of effects concentrations. Much work, particularly short-term exposures, has shown adverse effects from nickel at concentrations in the milligrams per liter range, which are hundreds or even thousands of times higher than environmentally relevant concentrations. Yet other work has shown nickel to be about as toxic or more toxic, in long-term exposures than metals more commonly considered to pose a risk to sensitive organisms, such as copper or cadmium. No reports were located of adverse effects from short-term (96-hr) toxicity tests using salmonids at concentrations below the final acute value (two times the acute criterion) for nickel.

During this consultation, EPA revised the proposed chronic criterion for nickel resulting in a level that is considerably more protective of listed salmon and steelhead. Potential adverse effects from exposure to nickel at concentrations at or below the criterion in the revised action are expected to be primarily to sensitive invertebrates which may be a food source for listed species. This affect is expected to be small.

Selenium

The acute criteria for selenium is of little relevance because selenium in the water column is not expected to affect listed salmon and steelhead directly through ventilation. The primary concern with selenium is build up in the muscle tissues as trophic transfer from prey species.

If water concentrations were near the chronic selenium criterion of 5 µg/L indefinitely, selenium would likely be transferred through the food web resulting in selenium concentrations in juvenile salmonids greater than twice as high as a concentration estimated to be low risk for appreciable

effects in juvenile salmon or steelhead (~7.6 mg/kg dw in whole bodies). Fish tissue residues resulting from stream food web transfer from a constant water concentration of about 5 µg/L were projected to exceed about 19.5 mg/kg dw in juvenile salmonids. This selenium tissue burden would be projected to result in growth reductions and increased mortality in juvenile anadromous salmonids, on the order of about a 50% reduction in weight, a 10% reduction in length, and about a 25% reduction in survival. Lesser reductions in growth (e.g., a 7.5% reduction) were projected to appreciably increase extinction risks and delay recovery in a modeled Chinook salmon population (Mebane and Arthaud 2010). While their modeling was specific to a Snake River spring/summer Chinook salmon populations from the upper Salmon River, NMFS assumes that the relations between size and survival during downstream migration would also hold for steelhead and sockeye salmon,

It is reasonable to assume that listed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead will be exposed to levels of selenium that are harmful based future mining activities. However, it is not possible to estimate within the ESU the number of locations where future selenium discharges or exposure may occur. Most likely these locations will be associated with a mine or highly mineralized areas. Because of this it is reasonable to assume that a large percentage of at least one population within the ESU will be exposed to levels of selenium approaching the chronic standard during some phase of life which could result in mortality primarily due to reduced growth and survival of juveniles. This could significantly reduce the abundance and productivity of that population of salmon or steelhead which will prevent the ESU from achieving recovery.

Silver

No concentrations of silver at or near the acute or chronic criteria have been identified in Idaho, even in areas where silver mining occurred for extended periods of time and significant environmental damage was caused by other substances related to the mining activities.

In natural waters silver is likely much less toxic than in most published laboratory experiments because of the strong modifying influence of naturally occurring ligands in ambient waters. Because of this, it appears unlikely that acute toxicity to salmonids at criterion concentrations will occur.

Unlike other criteria considered in this Opinion that had two part values to protect against short term and longer exposures, for silver only a short-term (acute) criterion is proposed. However, adverse chronic effects, including premature hatching, growth inhibition, and chronic mortality, have been observed at in laboratory settings at concentrations below the proposed single silver criterion. Thus, using a single criterion value that was derived using short-term toxicity data to also protect aquatic life from indefinite exposures may be under-protective. The acute criterion is derived as a function of hardness, which is not supported by more current literature which shows chloride, DOC, and sulfide to be more important factors in mitigating silver toxicity. The potential inadequacies and underprotectiveness of the silver criterion are mitigated by the fact that in the environment, silver occurs in a less toxic form than that used in most of the toxicity tests published in the literature. Significant food chain biomagnification by fish is also possible,

but all of these effects appear unlikely to occur because of the low silver concentrations typically encountered in the aquatic environment.

Zinc

Zinc is primarily an acute toxin to salmonids, hence the acute criterion is of greater environmental relevance than the chronic criteria. A confusing aspect of the literature on zinc toxicity to salmonids is the great disparity in reported effects between studies. Across different studies, EC_{50} values for rainbow trout with zinc at similar test hardnesses varied by an order of magnitude. Said differently, zinc at criteria concentrations has been found to be highly toxic and killed most of the fish exposed (Figure 2.4.10.2), but in other tests, concentrations well in excess of the criteria killed no fish. This disparity may be due to differences in the sensitivity of fish at different sizes as they develop. While it is commonly assumed that the smallest organisms will be most sensitive (e.g., ASTM 1997), this is clearly not always the case with zinc. Instead for salmonids, the likely pattern is that the newly hatched, smallest fish appear resistant to zinc, lose resistance as they grow during the first and second months after hatching, and then regain resistance as the fish become older and larger. This suggests that even though most of the studies reviewed that addressed zinc toxicity to listed Snake River salmon and steelhead did not show adverse effects below criteria values (Figure 2.4.10.1 and 2.4.10.3c) the risk from exposure to zinc may have been underestimated because the studies did not distinguish between sensitive life stages, and did not examine effects to listed steelhead and salmonids at their most vulnerable post-hatch stages.

Adverse effects were found at sub-criteria values in tests conducted at hardnesses less than 25 mg/L, a few other tests at moderately low hardness of 35 mg/L with the most sensitive size fish tested (Figure 2.4.10.2), and multiple tests reported by Hansen *et al*. (2002c) with rainbow trout. The preponderance of the information reviewed indicate that in waters with hardness less than about 25 mg/L as $CaCO_3$ the Idaho zinc criteria would not be sufficiently protective of listed Snake River salmon and steelhead if they were exposed at their most sensitive life stages. If alternatively, the current IDEQ zinc criteria were determined using the actual water hardness, instead of the assumed hardness of 25 mg/L, most of those data indicate that the criteria would then be sufficient to avoid harm in most of the studies reviewed. This would be sufficient to avoid population level effects to Snake River salmon and steelhead.

Pentachlorophenol

Some studies indicate the proposed acute PCP criterion is at the level where some acute toxicity will occur. Other studies showed that LC_{50}s for salmonids were well above the proposed acute water quality standard. Most studies of chronic effects reported the onset of adverse effects occurred at least slightly above the chronic criterion, although a single study found reduced growth in sockeye salmon at lower concentrations than the chronic criterion. Rainbow trout exposed to PCP concentrations far below the chronic criterion showed reduced ability to evade predators, and reduced ability to capture prey. Both the chronic and acute criteria will likely have some effect on listed species or their food sources.

Pentachlorophenol is not likely to be a component of NPDES discharges, but may be used in the treatment of wood that finds its way into inwater or overwater structures so the exposure risk, while small, is not discountable.

Chromium III and VI

There are no known concentrations of chromium that approach the proposed standards in water bodies in the action area, and no current discharges of chromium into water bodies in the action area. Because new permits are also unlikely to reach concentrations of chromium where effects to listed species have been identified, adverse effects are unlikely to occur.

Data reviewed by NMFS indicate few direct adverse effects to listed salmonids at concentrations less than the chronic trivalent or hexavalent chromium criteria. Studies on the effects of hexavalent chromium to salmon sperm are contradictory with one test indicating it is toxic at concentrations below the chronic criteria, and a more recent study showing no effects at criteria concentrations. Because the more recent study that showed no effects appeared to use a more relevant exposure duration, it is relied upon in concluding that direct adverse effects of chromium to listed salmonids are unlikely at or below criteria.

The amphipod *Hyalella azteca* suffered adverse effects at a test concentration below the chronic criterion in one study but not in another. Because so few data on long-term effects of chromium to benthic invertebrates are available, this test is interpreted as suggesting adverse effects to food sources are possible. Bioaccumulation of chromium clearly occurs when water concentrations are high, but relevant data are absent regarding the effects to salmonids when water-borne concentrations are below the chronic criterion. Because adverse effects to the species or critical habitat should never reach the scale where take occurs, the effects of the proposed action for chromium are very minor.

Lead

Potential adverse effects from exposure to lead at concentrations at or below the acute or chronic criteria, to listed salmon and steelhead and their critical habitat are likely to be very minor. The only adverse effects of chronic lead exposures at sub-criteria concentrations were to snails and the amphipod *Hyalella azteca*. In most habitats, listed salmonids would not be expected to be dependent on amphipods and snails for food. Listed salmon and steelhead are unlikely to be injured or killed by exposure to lead concentrations that are at or below the proposed acute or chronic criteria. No evidence of direct adverse sublethal effects occurring at concentrations at or below the chronic criterion to salmonids was found.

Aldrin/Dieldrin

Aldrin. The limited information available regarding aldrin toxicity to salmonids indicates that 50% mortality can occur when concentrations are below or slightly above the acute criterion. Similarly, there is evidence that aldrin is toxic to some salmonid prey species when concentrations are below or close to the criterion. This information suggests that the proposed acute criterion for aldrin if found at these levels is reasonably certain to harm listed salmonids or

impact their food sources. The limited information available regarding aldrin toxicity indicates that aldrin is toxic to some salmonid prey species when concentrations are below or close to the criterion and is likely to adversely affect food sources. However, it is unlikely that discharges of aldrin will occur in the action area as no uses are currently approved and levels found in the water column are well below the proposed standards. Because of this NMFS finds that adverse affects are unlikely to occur.

Additional comments on Aldrin. Although no chronic criterion for aldrin is proposed, available studies demonstrate that chronic effects do occur to freshwater fish at 0.0466 µg/L, and to prey items at 2.5 µg/L. These results suggest that the absence of a chronic criterion could result in adverse chronic effects to listed salmonids and their food source. However, the human-health based aldrin criteria is also applicable to all waters in the action area that are either designated critical habitat for, or are inhabited by listed salmonids. For aldrin this criterion is 0.00014 µg/L (Table 1.3.1). This value is lower than concentrations causing adverse effects to any aquatic prey species, listed species, or surrogate for a listed species reviewed here so lack of a chronic criteria does not pose a risk to listed salmon and steelhead.

Dieldrin. The scientific literature on effects of dieldrin on salmonids reports acute lethal effects at concentrations that are below or slightly above the proposed acute criterion. These studies included various salmonid species, such as Chinook and coho salmon, steelhead, and rainbow, cutthroat, or brown trout, as well as toxicological information on juveniles and adults. This available information indicates that the proposed acute criterion for dieldrin will likely adversely affect listed salmonid species. The proposed acute criterion is greater than LC_{50}s reported for several important salmonid prey species. However, because acute effects could only come from recent applications, and because the use of dieldrin has been banned since EPA cancelled its registration in 1975, acute effects occurring from an authorized release are unlikely. Chronic studies involving juvenile rainbow trout demonstrate that limited adverse effects only occur when ambient concentrations are >95 times the proposed chronic criterion. This information is supplemented by published BCF values and analyses of the results of dietary exposure studies in which estimated aqueous concentrations of dieldrin resulting in reported tissue concentrations was also well above the chronic criterion. These limited studies indicate that the proposed chronic criterion will not result in measurable effects to listed salmonids. Further, no information suggests that prey species may be adversely affected by concentrations below the proposed chronic criterion. Dieldrin was detected in sediment in Brownlee Reservoir of the Snake River (Table 2.3.1) and these are likely the highest levels that will be found in the state based on the location of the reservoir. However, levels of dieldrin currently found in Brownlee Reservoir are well below the standard and the reservoir is not occupied by listed species. With no ongoing discharges, the level of dieldrin in sediment in Brownlee Reservoir is likely to decline over time.

Chlordane

Lethal effects from short-term exposures of salmonids or salmonid invertebrate prey species to chlordane only occurred at concentrations above the acute criterion. There are no current approved uses of chlordane in the United States and no manufacturing of chlordane takes place in Idaho. Chlordane was detected in Brownlee Reservoir (Table 2.3.1) and these are likely the

highest levels that will be found in the state based on the location of the reservoir. The levels detected were well below the proposed criteria and no listed salmon or steelhead are located in or above the reservoir.

Data generally indicate that the proposed chronic criterion for chlordane is likely to avoid harm to listed salmonids. However, many sublethal effects of chronic exposure to chlordane that have been documented in mammals (i.e., neurological damage, altered immune and reproductive function, and increased cancer risk) ; we found no studies of salmonid species subjected to long-term chlordane exposure at concentrations near or below the criterion. Similarly, few data are available on the sublethal effects of long-term exposure to chlordane on salmonid prey. There are also a few studies suggesting that a risk of increased long-term mortality or sublethal effects at chlordane tissue concentrations close to those that might be expected in fish exposed to chlordane at levels allowed under the chronic aquatic life criteria. Additionally, bioaccumulation can occur in salmonids with chronic exposure to chlordane at levels allowable under the proposed criteria, and exposure is likely to occur not only through the water column but also through diet and contact with sediments. There is some evidence of risk to benthic invertebrates or through food web uptake associated with bioaccumulation and exposure from sources other than the water column. Based on the strength of evidence considered, the chronic criterion does not appear likely to harm salmonids through water column exposure. The other exposure pathways may pose some risk for salmon and steelhead, but appear likely to result in only minor effects.

DDTs

Sediment and fish tissue DDT concentrations from Brownlee Reservoir tended to be the highest found in sampling in various locations in Idaho (Table 2.3.1; Clark and Maret 1998). In water, baseline levels for DDT found in Brownlee Reservoir in 2011 were <0.00066 µg/L, which is below the levels where effects would be expected to listed salmon and steelhead. DDT is a banned substance in the United States and so no new or ongoing discharges are expected to occur.

Concentrations of DDT in the action area at the proposed action acute criterion could cause harm to listed fish; however, the effects of the EPA approval of the acute criterion is discountable because DDT is extremely unlikely to occur at that concentration because there will be no new discharges of DDT and no known hotspots of DDT occur in the action area where listed fish are present.

The chronic criteria have risk of sublethal health effects in salmonids if bioconcentration results in tissue concentrations that are higher than those expected by EPA. The proposed chronic criterion may allow substantial bioaccumulation to occur because DDTs are taken up not only from the water column but also from sediments and prey organisms. No reports of direct adverse effects to listed salmonids were located at concentrations lower than the chronic criterion. While some data are equivocal and there are quite a few uncertainties in interpreting DDT risks to fish, NMFS found no persuasive evidence of appreciable adverse effects from DDT at concentrations lower than the chronic criterion concentrations.

Endosulfan

Endosulfan has not been found in Idaho waters or sediments at levels that approach the standards as proposed and future discharges of endosulfan are unlikely to occur because the product use has been banned so an acute exposure scenario from an authorized release is unlikely. The proposed acute lethal criterion for endosulfan would likely result in some mortality of listed salmonids. Reported rainbow trout LC_{50}s near or below the proposed acute criterion indicate that appreciable mortality can occur in waters meeting the proposed criterion. Evaluation of the proposed chronic criterion was restricted by the absence of relevant toxicity testing data involving salmonid species. The limited information that could be gathered on rainbow trout and two other freshwater fish suggests that the proposed chronic criterion can allow chronic physiological damage to listed salmonid species. The physiologic damage was not directly related to "clinically significant" fish health changes. Although there is a paucity of toxicity testing data, the available information suggests that the proposed acute and chronic criteria may protect some invertebrate prey species. Little test data exist for specific salmonid prey species.

Endrin

Endrin is a banned product in the United State so new discharges are likely to occur. Endrin was detected in Brownlee Reservoir which would likely contain the highest levels in the state due to its location. Concentrations elsewhere in Idaho are likely to be lower than those in Brownlee Reservoir (Table 2.3.1). The levels detected were lower than the chronic criteria. Most reports of mortality following short-term endrin exposures produced LC_{50}s greater than the acute criterion, although some effects occurred at lower concentrations. Evidence indicates that concentrations at the acute criterion will not harm salmonid prey species.

While data are sparse, most reports of adverse effects from chronic exposures to salmonids or other fish occurred at concentrations higher than the chronic criterion. A report of subclinical reductions in cholesterol and lipids in gravid Asiatic catfish are of ambiguous importance to salmon. Food chain exposure via diet or sediment was estimated by NMFS to mostly result in tissue residues lower than those shown to be harmful to fish.

Heptachlor

Currently heptachlor is not used in Idaho because the only remaining use is to control fire ants which are not present in Idaho. The only information regarding existing concentrations of heptachlor in Idaho is from Brownlee Reservoir which is the reservoir most likely to contain contaminated sediments. The levels measured at Brownlee are well below levels found to cause acute or chronic effects and should decline over time.

Available evidence indicates that listed salmon or steelhead experience acute lethal effects at concentrations much higher than the proposed acute criterion. However, all such evidence is derived from static tests with nominal heptachlor concentrations, a methodology that tends to under estimate toxicity. There is a greater likelihood that heptachlor could harm salmon or steelhead through lethal effects on aquatic invertebrates; however, little information is available on the effects on invertebrate prey species.

Data on chronic effects of heptachlor are sparse, but suggest that the risk of adverse effect through water-borne exposure is likely to be low. Some studies suggest that tissue concentrations that are possible under the chronic criterion could have sublethal or lethal effects on alevins or fry. Bioaccumulation can occur in salmonids with chronic exposure to heptachlor, and when exposure occurs, this is could occur through the water column, diet and contact with sediments.

Lindane

There are no current registered uses of lindane in the United States and no known contamination of sites in Idaho at levels that may impact listed salmonids. Most of the available data tended to show adverse effects to listed salmonid species, or their close relatives, or their prey at levels greater than the proposed criteria concentrations. The reliability of a single acute test reporting mortalities at concentrations lower than the acute criterion is uncertain since targeted exposure concentrations were not verified by chemical analysis (i.e., were nominal concentrations).

PCBs

Water borne PCB concentrations close to, or below, the proposed chronic criterion, in concert with predicted bioaccumulation rates, were projected to result in impaired thyroid function in coho salmon and embryo mortality in lake trout. However, polychlorinated biphenyls are no longer manufactured in the United States, and PCB contamination of surface waters in Idaho is not recorded in the impaired waters list for Idaho and no known cleanup or sediment concerns that might impact listed fish were identified. This makes the risk of exposure to listed salmon or steelhead unlikely. If discharges do occur the most stringent controlling ambient water quality criterion applicable in designated critical habitats is the fish consumption based human-health criteria, rather than the chronic aquatic life criteria (Table 1.3.1). The fish consumption based criterion is more than100 times more restrictive than the aquatic life criteria. Therefore any effects from the proposed approval of the PCB criterion will be very small on listed species and designated critical habitat.

Toxaphene

Toxaphene appears unlikely to cause adverse effects to habitat or listed salmon or steelhead from exposure at concentrations in water equal to or below the proposed acute or chronic criteria. The risk of exposure is also very small.

2.6.1. Integration and Synthesis Summary for Each Affected Species

For Snake River A run steelhead the existing populations may have achieved “maintained” or “moderate risk” status based population estimates using an aggregate of the returns over LGD. However, none of the populations have attained the “low risk” or “viable” status needed to attain the recovery goal for the ESU. To achieve recovery goals, improvement in abundance and productivity is necessary in a least half of the populations. For Snake River B run steelhead populations all of the populations remain at high risk for abundance and productivity.

Abundance and productivity for all populations must improve for the DPS to attain its recovery goal (Ford 2011). The proposed action for the hardness floor, arsenic, copper, cyanide, selenium and mercury is likely to cause adverse modification to critical habitat or lethal and sublethal effects to a large portion of one or more populations, and the reduction in, or loss of, that population will result in jeopardy for the Snake River steelhead DPS.

Snake River spring/summer Chinook populations all remain at high risk for abundance and productivity. Abundance and productivity for all populations must improve for the ESU to attain its recovery goal (Ford 2011). The proposed action for the hardness floor, arsenic, copper, cyanide, selenium and mercury is likely to cause adverse modification to critical habitat or lethal and sublethal effects to a large portion of one or more populations, and the reduction in, or loss of, that population will result in jeopardy for the Snake River spring summer Chinook salmon ESU.

Snake River fall Chinook salmon are at moderate risk for abundance and productivity and have not attained the recommended level of "very low risk" for abundance and productivity necessary for a single population MPG to achieve its recovery goal (Ford 2011). The proposed action for the hardness floor, arsenic, copper, cyanide, selenium and mercury is likely to cause adverse modification to critical habitat or lethal and sublethal effects the population and will result in jeopardy for the Snake River fall Chinook salmon ESU.

Snake River sockeye salmon are currently at high risk for abundance and productivity the Redfish Lake Population must improve for the ESU to achieve its recovery goal (Ford 2011). The proposed action for the hardness floor, arsenic, copper, cyanide, selenium and mercury is likely to cause adverse modification to critical habitat or lethal and sublethal effects to a large portion of the population, and will result in jeopardy for the Snake River sockeye salmon ESU.

2.7. Conclusion

After reviewing the current status of the listed species, the environmental baseline within the action area, the effects of the proposed action, and cumulative effects, NMFS has made the follow determinations in its Opinion.

Hardness Floor

Metal limits for discharges that are calculated using the current equation with the 25 mg/L hardness floor may result unacceptable declines in abundance and productivity for any exposed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead population and prevent the population from achieving the minimum level of abundance and productivity needed for the ESU or DPS to achieve its recovery goal. NMFS concludes that the potential effects of using the hardness floor in applying the proposed IWQS will rise to the level of jeopardizing the Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead.

Permitting of new or existing discharges using the current equations containing a floor of 25mg/L for calculating metal discharge limits will allow some metals to reduce water quality, accumulate in sediments, periphyton, and in aquatic macroinvertebrate tissues in concentrations that will be detrimental to aquatic macroinvertebrate communities. NMFS concludes this will result in the adverse modification of habitat within designated critical habitat for Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon, or Snake River Basin steelhead.

Arsenic

If sustained concentrations of arsenic in a surface water within the action area exceeds 10 µg/L, which is much lower than the proposed chronic criterion of 150 µg/L dissolved arsenic in water, then accumulation of arsenic would be expected in sediments, periphyton, and in aquatic macroinvertebrate tissues at concentrations that would be harmful in diets of salmonids. This would also likely create a reserve of arsenic in sediment resulting in a contaminated food source for listed species over an extended timeframe. This will result in unacceptable declines in population abundance and productivity for an exposed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead population and will prevent the populations from achieving the minimum level of abundance and productivity needed for the ESU or DPS to achieve its recovery goals. NMFS concludes that the potential effects from the proposed chronic arsenic criteria would jeopardize Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead.

If new or existing discharges containing concentrations of arsenic in surface water within the action area approach the chronic criterion of 150 µg/L dissolved arsenic in water the accumulation of arsenic is expected in sediments, periphyton, and in aquatic macroinvertebrate tissues to concentrations that will be detrimental to aquatic macroinvertebrate communities. Continued exposure may result in a reserve of arsenic in sediment that may take years to dissipate resulting in an ongoing effect to prey species over a number of years. Because of the likelihood of a new discharge, or continuation of existing discharges, being located within critical habitat for listed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead, NMFS has determined the chronic standard for arsenic is likely to result in an adverse modification of designated critical habitat for these species.

The proposed acute criterion for arsenic is not likely to result in adverse effects to listed salmon and steelhead in Idaho because the exposure to concentrations of dissolved arsenic at the proposed standards is not expected to result in significant toxic effects to individual fish or populations.

Copper

Continued exposure to copper at the proposed acute or chronic criteria levels will result in adverse effects including mortality and reduced growth in juvenile fish. NMFS concludes that

these potential effects are likely to jeopardize the four listed salmon and steelhead ESUs or DPSs because of predicted effects to growth, reproduction and survival that would increase the extinction risk for a DPS or ESU.

New or existing discharges containing concentrations of copper in surface water that approach either the acute or chronic criterion will compromise the diversity and abundance of the macroinvertebrate food base for rearing juvenile salmon and steelhead. It is likely that a new discharge will be located within critical habitat for listed species, thus NMFS has determined the proposed acute and chronic criteria for copper are likely to result in an adverse modification of critical habitat for Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead.

Cyanide

Juvenile Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon or Snake River steelhead will likely die if exposed to cyanide concentrations at the proposed chronic criteria (in water temperatures below 6°C). NMFS concludes that the loss of juveniles will negatively affect the exposed population, and prevent attainment of viability criteria for the exposed DPS or ESU. Therefore, NMFS concludes that the proposed cyanide criteria will jeopardize Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead.

Cyanide in the water column at the proposed chronic criteria concentrations during the colder seasons will result in the water quality being unsuitable for listed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead resulting in an adverse modification of designated critical habitat for these species.

The proposed acute criterion for cyanide is not likely to result in adverse effects to listed salmon and steelhead in Idaho because the exposure to concentrations of cyanide at the proposed standards is not expected to result in significant toxic effects to individual fish or populations.

Mercury

Risks of mercury toxicity result primarily from bioaccumulation occurring from exposure to mercury in the diet. In reviewing the proposed chronic mercury criterion, NMFS concludes that these potential dietary effects impair the ability of listed fish to locate, capture, and ingest prey, and to avoid predators, as well as impaired reproduction. These effects can reduce survival of individual fish and reduce the viability of a population. Therefore, NMFS concludes that the proposed chronic criteria for mercury will jeopardize Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead. Because the nature of effects is through ingestion of prey with a body burden of mercury, NMFS also concludes the proposed chronic criterion will adversely modify designated critical habitat for rearing Snake River salmon and steelhead.

NMFS concludes that exposure of listed salmon and steelhead to mercury at the acute criterion is unlikely to result in death or sub-lethal effects that result in injury or reduced survival.

Selenium

Continued exposure to selenium at the chronic criterion level will result in transfer of selenium through prey species to listed juvenile Snake River Basin steelhead, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River spring/summer Chinook salmon creating a selenium tissue burden that reduces growth and survival. These effects will translate to reduced viability of the exposed population. Thus NMFS concludes that the proposed chronic criterion will jeopardize Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead. NMFS also concludes that these potential dietary effects will adversely modify critical habitat for rearing Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead.

The proposed acute criterion for selenium is not likely to result in adverse effects to listed salmon and steelhead in Idaho because the exposure to concentrations of selenium at the proposed standards is not expected to result in significant toxic effects to individual fish or populations.

Nickel

The available information indicates that the risk of adverse effects from exposure to the chronic criterion for nickel to listed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River Basin steelhead and Snake River Sockeye or their habitat at concentrations at or below the criterion, are likely to adversely affect listed species because it may result in reduced survival and growth of salmonid embryos. However, the effect is not expected to reduce the viability of the exposed population. Therefore, NMFS concludes that the proposed chronic criterion for nickel is not likely to jeopardize the species or adversely modify designated critical habitat for listed Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River Basin steelhead or Snake River Sockeye.

The proposed acute criterion for nickel is not likely to result in adverse effects to listed salmon and steelhead or their designated critical habitat in Idaho because the exposure to concentrations of nickel at the proposed standards is not expected to result in significant effects.

Silver

The available information indicates that when salmonids or their habitat are exposed to silver (as silver nitrate) over the long-term, mortality and reduced reproduction could occur at concentrations below the acute silver criteria. However, because silver in the environment is expected to form complexes with chloride, DOC, or sulfide that have less toxicity than silver nitrate, these effects are unlikely to affect the viability of populations in the listed ESUs, and therefore, are unlikely to jeopardize Snake River spring/summer Chinook salmon, Snake River

fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead or adversely modify designated critical habitat for these species.

Zinc

Some available studies have shown some adverse effects from exposure to zinc by listed salmon and steelhead at concentrations at or below the proposed acute or chronic criteria. However, many other studies show adverse effects only at concentrations higher than the criteria. NMFS concludes that any effects that may occur are unlikely to decrease the viability of affected populations, and therefore are not likely to jeopardize the Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead.

Exposure to zinc at concentrations below the proposed criteria for zinc may change the composition of prey species for listed salmon. However, the overall abundance of prey availability is unlikely to result in adverse effects to listed populations, and therefore, exposure to zinc at the proposed criteria is unlikely to result in the adverse modification of designated critical habitat for these species.

Pentachlorophenol

Discharges of PCP in Idaho are only expected from the use of treated wood in construction in or around surface water. Therefore any effects from the proposed approval of the PCP chronic or acute criteria are expected to be short-term events and have only minor effects on listed species and designated critical habitat. Therefore, NMFS concludes that the proposed criteria for PCP are not likely to jeopardize Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead, or result in the adverse modification of designated critical habitat.

Chromium III and Chromium VI

The proposed criterion for chromium III and chromium VI are not likely to result in adverse effects to Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead in Idaho because the exposure to concentrations of chromium III and chromium VI at the proposed standards are not expected to result in significant toxic effects to individual fish or fish populations.

The proposed criterion for chromium III and chromium VI are not likely to result in adverse effects to designated critical habitat for Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead in Idaho.

Lead

The proposed criterion for lead are not likely to result in adverse effects to Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead in Idaho because the exposure to concentrations of lead at the

proposed standards are not expected to result in significant toxic effects to individual fish or fish populations.

The proposed criterion for lead are not likely to result in adverse effects to designated critical habitat for Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead in Idaho.

Aldrin

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to Aldrin is very unlikely. This is based on the inability to use Aldrin as a pesticide, the lack of other discharges and the lack of known contamination in waters containing listed salmon or steelhead. An additional safety factor is provided by the applicability of a lower recreation criterion based on fish consumption.

Dieldrin

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to Dieldrin very unlikely. This is based on the inability to use Deildrin as a pesticide, the lack of other discharges and the lack of known contamination in waters containing listed salmon or steelhead. An additional safety factor is provided by the applicability of a lower recreation criterion based on fish consumption.

Chlordane

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to Chlordane very unlikely. This is based on the inability to use Chlordane as a pesticide, the lack of other discharges and the lack of known contamination in waters containing listed salmon or steelhead. An additional safety factor is provided by the applicability of a lower recreation criterion based on fish consumption.

DDT

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to DDT is unlikely. This is based on the inability to use DDT as a pesticide and the lack of other new discharges. An additional safety factor is provided by the applicability of a lower recreation criterion based on fish consumption. The more meaningful exposure scenario for DDT is chronic exposure to low level concentrations that have persisted in sediments of rivers, reservoirs, and lakes. On the whole, the available information indicates that the risk of adverse effects from exposure to DDT by listed Snake River salmon and Snake River Basin steelhead at concentrations at or below the chronic criterion is very low and because no new discharges will occur and any potential exposure from existing contamination will be reduced over time.

Endosulfan

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to Endosulfan is very unlikely. This is based on the inability to use Endosulfan as a pesticide, the lack of other discharges and the lack of known contamination in waters containing listed salmon or steelhead.

Endrin

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to endrin is very unlikely. This is based on the inability to use endrin as a pesticide, the lack of other discharges and the lack of known contamination in waters containing listed salmon or steelhead. Even if fish are exposed, few effects at sub-criterion concentrations have been documented.

Heptachlor

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to heptachlor is very unlikely. This is based on the inability to use heptachlor as a pesticide, the lack of other discharges and the lack of known contamination in waters containing listed salmon or steelhead. An additional safety factor is provided by the applicability of a lower recreation criterion based on fish consumption.

Lindane

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to lindane is very unlikely. This is based on the inability to use lindane as a pesticide, the lack of other discharges and the lack of known contamination in waters containing listed salmon or steelhead.

PCBs

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to PCBs is very unlikely. There are no registered uses of PCBs in the state, and no known contamination in waters containing listed salmon or steelhead. If discharges do occur the most stringent controlling ambient water quality criterion that is applicable to designated critical habitats are the fish consumption based human-health criteria, rather than the chronic aquatic life criteria (Table 1.3.1). The fish consumption based criteria are more than100 times more restrictive than the aquatic life criteria. Therefore any effects from the proposed approval of the PCB criteria will have only very small effects on listed species and designated critical habitat.

Toxaphene

Exposure of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon and Snake River Basin steelhead and their designated critical habitat to toxaphene is very unlikely. This is based on the inability to use toxaphene as a pesticide, the lack of other discharges and the lack of known contamination in waters containing listed salmon or steelhead. Even if fish are exposed few effects at sub-criterion concentrations have been documented.

2.8. Reasonable and Prudent Alternatives (RPAs) and Analysis of Effects of the RPAs

"Reasonable and prudent alternatives" (RPAs) refer to alternative actions identified during formal consultation that can be implemented in a manner consistent with the intended purpose of the action, that can be implemented consistent with the scope of the Federal agency's legal authority and jurisdiction, that are economically and technologically feasible, and that 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).

The EPA's authorities include the responsibility to review and approve or disapprove state revisions of their water quality standards; states are to review their water quality standards, at least once every 3 years (40 CFR sections 131.20 through 131.21). If EPA disapproves a state's new or revised water quality criteria and the state does not adopt specified changes, the EPA Administrator has the responsibility and authority to promptly propose and promulgate such standard 40 CFR section 131.22). The water quality standards considered in this action are implemented in part through wastewater discharge permits, administered by EPA through the National Pollutant Discharge Elimination System (NPDES). Monitoring, including biological monitoring, may be required of dischargers as part of their permit conditions (40 CFR 122.48). When the ESA is applicable and requires consideration or adoption of particular permit conditions, those requirements must be followed (40 CFR 122.49).

2.8.1. The RPA for the Hardness Floor

2.8.1.1. New Aquatic Life Criteria

The EPA shall recommend that the state of Idaho adopt, and EPA will promulgate if necessary, removal of the low end hardness floor on the hardness dependent metals criteria equations within 3 years of the date of this Opinion.

2.8.2. The RPAs for Arsenic

2.8.2.1. Interim Protection for Listed Species

Until a new chronic criterion for arsenic is adopted, EPA shall ensure that the 10 µg/L recreational use standard is applied in all Water Quality Based Effluent Limitations (WQBELs) and Reasonable Potential to Exceed Calculations using the human health criteria and the current methodology for developing WQBELs to protect human health. The recreational use standard is interpreted to apply as inorganic, unfiltered, arsenic.

2.8.2.2. New Chronic Aquatic Life Criterion for Arsenic

The EPA shall ensure, either through EPA promulgation of a criterion or EPA approval of a state-promulgated criterion, that a new chronic criterion for arsenic is in effect in Idaho within 7 years of the date of this Opinion. The new criterion shall be protective of listed salmon and steelhead, consistent with the discussion and analysis in this Opinion. If ESA consultation is required for the new criterion, EPA shall provide an adequate biological evaluation to NMFS and initiate consultation within 6 years of the date of this Opinion, unless NMFS and EPA mutually agree to a different time-frame, to allow for consultation to be completed prior to EPA progmulgation or approval of the new criterion.

2.8.3. The RPAs for Copper

2.8.3.1. Interim Protection for Listed Species

Until new criteria are adopted, a zone of passage must be maintained around any mixing zone for discharges that include copper, sufficient to allow unimpeded passage of adult and juvenile salmonids as defined in Appendix F Salmonid Zone of Passage Considerations.

Permits for new discharges must ensure a zone of passage persists under seasonal flow conditions (see Appendix D). If the regulatory mixing zone is limited to less than or equal to 25% of the seasonal flow conditions, then a sufficient zone of passage is presumed to be present.

Permits reissued for existing discharges must ensure a zone of passage persists under seasonal flow conditions. If the regulatory mixing zone is limited to less than or equal to 25% of the volume of a stream, then sufficient zone of passage is presumed to be present. If existing discharges were calculated using greater than 25% of the seasonal flow conditions for applying aquatic life criteria the mixing zone must be reduced to 25% unless one of the following conditions exists:

1. An evidence-based "Salmonid Zone of Passage Demonstration" (see Appendix F) indicates that impeding fish movements is unlikely, or;

2. Biological monitoring of aquatic communities in the downstream receiving waters shows no appreciable adverse effects relative to reference conditions as described in Appendix E Biomonitoring of Effects, and biological whole-effluent toxicity testing is consistently negative, defined as follows:

a. Whole effluent toxicity (WET) testing shall be required, using at least the 7-day *Ceriodaphnia dubia* 3-brood test and the 7-day fathead minnow growth and survival test. If previous testing of a facility's effluents have demonstrated that one test is more sensitive, at EPA's discretion it is acceptable to base further testing on only the more sensitive test. Toxicity trigger concentrations for WET tests shall also be established using dilution series based upon no more than 25% of the applicable critical flow volume. The dilution series for WET testing (7Q10) shall be designed such that one treatment consists of 100% effluent, and at least one treatment is more dilute than the targeted critical flow conditions. Receiving waters upstream of the effluent discharge should be used as dilution water.

   The "critical concentration" is defined here as the condition when the smallest permitted dilution factor occurs, modified by a 25% mixing zone fraction. For example, if the minimum effluent dilution occurring at a site is a 1:4 ratio (one part effluent to four parts streamwater), then because only 25% of the measured streamflow is authorized for dilution; then the dilution factor for effluent testing is likewise reduced to 1:1. The critical concentration would then be 50% effluent, i.e., one part each effluent and dilution water.

   WET tests results need to be consistently negative to indicate the absence of appreciable instream toxicity in test conditions that reflect the critical effluent concentration, above. A "negative test result" is produced by a test meeting the performance objectives of a passing test according to EPA (2002c) or EPA (2010c). Test results are considered to be consistently negative if the failure rate is less than one in 20.

b. If instream biological monitoring shows adverse effects or if WET tests are not consistently negative, then a toxicity identification evaluation and toxicity reduction evaluation (TIE/TRE) must be undertaken to identify and remedy the causes of toxicity, which may include reducing effluent limits as warranted. Because considerable judgment may be involved in designing and carrying out a TIE/TRE, and because the results are performance-based (no detectable toxicity per A.2), more specific guidance is inappropriate to provide here. Mount and Hockett (2000) provide one example of a TIE/TRE.

2.8.3.2. New Acute and Chronic Aquatic Life Criteria for Copper

The EPA shall ensure, either through EPA promulgation of criteria or EPA approval of a state-promulgated criteria, that new acute and chronic criteria for copper are in effect in Idaho within 3 years of the date of this Opinion. The new criteria shall be no less stringent than the Clean Water Act section 304(a) 2007 national recommended aquatic life criteria (i.e. the BLM Model) for copper. NMFS does not anticipate that additional consultation will be required if the 2007 national recommended aquatic life criteria for copper are adopted.

2.8.4. The RPAs for Mercury

2.8.4.1. Interim Protection for Listed Species

1. Until a new chronic criterion is adopted EPA will use the 2001 EPA/2005 Idaho human health fish tissue criterion of 0.3 mg/kg wet weight for WQBELs and reasonable potential to exceed criterion calculations using the current methodology for developing WQBELs to protect human health. Implementation of the Idaho methylmercury criterion shall be guided by EPA's (EPA 2010a) methylmercury water quality criteria implementation guidance or IDEQ's (IDEQ 2005) methylmercury water quality criteria implementation guidance, (or)

2. For water bodies for which appropriate fish tissue data are not available, if the geometric mean of measured concentrations of total mercury in water is less than 2 ng/L, then the water body will be presumed to meet the fish tissue criterion of 0.3 mg/kg wet weight. If the water column concentration is greater than 2 ng/L, fish tissue data shall be collected.

2.8.4.2. New Chronic Aquatic Life Criteria for Mercury

The EPA shall ensure, either through EPA promulgation of a criterion or EPA approval of a state-promulgated criterion, that a new chronic criterion for mercury is in effect in Idaho within 7 years of the date of this Opinion. The new criterion shall be protective of listed salmon and steelhead, consistent with the discussion and analysis in this Opinion. If ESA consultation is required for the new criterion, EPA shall provide an adequate biological evaluation to NMFS and initiate consultation within 6 years of the date of this Opinion, unless NMFS and EPA mutually agree to a different time-frame, to allow for consultation to be completed prior to EPA progmulgation or approval of the new criterion.

2.8.5. The RPA for Cyanide

Calculation of effluent limits for cyanide shall be made using the receiving water mixing zone limitations described in "RPAs for Copper", in the *Interim Measures, Zone of Passage* section.

2.8.6. The RPAs for Selenium

2.8.6.1. Interim Protection for Listed Species

Until a new chronic criterion is adopted, EPA shall ensure that all effluent discharges located within critical habitats or habitats of Snake River listed salmonids that are regulated under the NPDES program apply the following terms:

1. At discharge locations where at the edge of the mixing zone, selenium concentrations are measured or projected to be higher than natural background for the locale and annual

geometric mean concentrations are higher than 2 µg/L, whole body fish tissue should be monitored in locations downstream of the discharge and in reference locations. The results shall be reported as an NPDES permit condition.

2. If fish tissue concentrations exceed the screening risk concentration of 7.6 mg/kg dw and are higher than reference concentrations, then the issuance of an NPDES permit shall include provisions to reduce selenium loading in order to reduce impairment of aquatic life uses. These provisions are not required if fish population surveys using surrogate species such as rainbow trout show that appreciable adverse effects are not present, as defined in Appendix E Biomonitoring of Effects.

2.8.6.2. New Chronic Aquatic Life Criterion for Selenium

The EPA shall ensure, either through EPA promulgation of a criterion or EPA approval of a state-promulgated criterion, that a new chronic criterion for selenium is in effect in Idaho within 4 years of the date of this Opinion. The new criterion shall be protective of listed salmon and steelhead, consistent with the discussion and analysis in this Opinion. If ESA consultation is required for the new criterion, EPA shall provide an adequate biological evaluation to NMFS and initiate consultation within 3 years of the date of this Opinion, unless NMFS and EPA mutually agree to a different time-frame, to allow for consultation to be completed prior to EPA progmulgation or approval of the new criterion.

2.8.7. Notification of EPA Final Decision

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 alternatives.

2.8.8. Analysis of the RPAs

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 and destruction or adverse modification of critical habitat is set out in sections below.

In determining the time frame for implementing the RPAs in this Opinion NMFS recognized that EPA needs to complete consultation with USFWS on these water quality standards to make sure that they are protective of other listed species. This consultation is scheduled to be completed in early 2015. After that, promulgation of rules under either the state or Federal process will require a minimum of 2 years to complete. For most water quality standards the state of Idaho will likely take the lead and promulgate state rules that require approval by the Idaho Board of Environmental Quality. Additionally, before becoming effective the rules will be reviewed by

the Idaho Legislature. Finally, EPA approval of the new rules must also occur. Based on this process we have assumed that the soonest new rules can be completed is 3 years and have used 3 years for the implementation time frame for the RPAs that will not require additional analysis to derive new criteria (i.e., hardnes floor, 2007 BLM copper criteria). The RPA for cyanide can be implemented immediately and therefore does not include an implementation period.

For the other RPAs, EPA and/or the state will likely require additional time to conduct the analyses necessary to support new criteria (arsenic, mercury, selenium). These RPAs therefore provide a longer implementation period of 4 to 7 years. To ensure that the listed species are not adversely affected during the implementation period, these RPAs include interim protective meaures that NMFS expects will adequately reduce any interim risk of harm to the species or their critical habitats. In addition, EPA consults with NMFS over each new or reissued NPDES permit in Idaho to ensure that it will not cause jeopardy to the species or adverse modification to critical the habitat. These factors, when considered together, will minimize any adverse during the implementation period while new criteria are developed and adopted.

2.8.8.1. Analysis of the Reasonable and Prudent Alternative for the Hardness Floor

Use of rules and guidance that require hardness-dependent metals criteria to be implemented using ambient water hardness without a hardness floor was analyzed as being protective in this Opinion. The RPA requires that the hardness floor be removed within 3 years. In the interim, within the action area, only one major discharger is known to be permitted to discharge metals into a water body with water hardness values that are consistently lower than the 25 mg/L hardness floor. This facility, the Beartrack Mine discharges to Napias Creek upstream of a waterfall which is considered to be impassible by Snake River Chinook salmon and thus excluded from critical habitat for Snake River salmon or steelhead (50 CFR §226.205, 226.212). The facility discharges high in the Napias Creek watershed; in lower Napias Creek, where it becomes designated as critical habitat downstream of Napias Creek falls, streamflows are estimated to increase by a factor of about 1.9 (USGS 2012). Thus, assuming discharges from the Beartrack Mine resulted in instream metals concentrations that approached adverse effects thresholds, i.e., criteria constrained by the hardness floor, this increase in dilution would effectively result in reducing metals concentrations by 0.54 times, assuming no intervening sources in the Napias Creek drainage. Because the amount of critical habitat downstream of Napias Creek Falls is small (less than 2 linear miles), in the interim time before the hardness floor is removed it is unlikely to result in appreciable reductions of any of the listed species' survival or recovery. The likelihood of new, major facilities coming online and discharging metals into low-hardness waters within this 3 year time-frame is considered unlikely.

2.8.8.2. Analysis of the Reasonable and Prudent Alternative for Arsenic

An interim protection for arsenic is available through use of the human health criterion, which is 10 µg/L. This criterion is applicable to all waters in the action area. Because it is more stringent than the chronic criterion of 150 µg/L, the criterion for the protection of human health is the controlling criterion for permitting actions. While bioaccumulation has been found in salmonid

prey from exposures at concentrations at or near 10 µg/L the application of this lower standard, coupled with biological monitoring, will provide adequate information to review effects in a site specific manner. Because any new or reissued permits will be subject to individual ESA consultation to assure they avoid jeopardy or adverse modification of habitat, EPA will make adjustments as necessary during the NPDES permitting cycle taking into account local conditions to avoid measureable direct effects to the listed species. Some minor adverse effects, as described in the effects section, may still occur during the early life history phases for all listed Snake River salmon and steelhead. Use of the human health criterion will provide adequate protection in the interim to avoid jeopardy.

The adoption of a new chronic aquatic life criterion within the next 7 years will be subject to ESA consultation to ensure that the new criterion will be adequately protective. Therefore, NMFS concludes that the arsenic RPA will not jeopardize any of the listed species considered in this Opinion or adversely modify their critical habitats.

2.8.8.3. Analysis of the Reasonable and Prudent Alternative for Copper

For the next 3 years, the interim requirement of assuring an adequate zone of passage for any permits that contain copper discharge limits as described in the copper RPA will minimize adverse effects to listed salmon and steelhead. Any new permits will also be subject to individual consultation to assure they avoid jeopardy or adverse modification of habitat. NMFS found five existing permits that contain copper limits and these will be updated to the new criteria when they are reissued. Over the next permitting cycle this should reduce the adverse effects described in the effects section to acceptable levels.

In Appendix C of this Opinion, we analyze implementation of the 2007 BLM EPA copper criteria and conclude that they will be adequately protective to avoid jeopardy to the listed species or critical habitat considered in this Opinion.

Therefore, NMFS concludes that the copper RPA will not jeopardize any of the listed species considered in this Opinion or adversely modify their critical habitats.

2.8.8.4. Analysis of the Reasonable and Prudent Alternative for Cyanide

The propsed cyanide criteria are likely to advesley affect the listed salmonids species in specific situations, primarily where water temperatures are at of below 6◦C. Implementation of the more restrictive practices in developing cyanide discharge limits as described in the cyanide RPA will suffice to minimize the adverse effects to listed salmon and steelhead. These practices will assure an adequate zone of passage exists for the fish, under all flow conditions, and provide biological monitoring and whole-effluent toxicity testing to assure the permit limits are protective of listed fish and prey species. This will be done at each discharge site by taking into account the localized conditions that affect toxicity of cyanide. Based on development of these site specific limits and the associated monitoring of discharge levels, combined with the fact that NMFS consults with EPA over each new or reissued NPDES permit, we expect only minor

adverse effects. Therefore, NMFS concludes that the cyanide RPA will not jeopardize any of the listed species considered in this Opinion or adversely modify their critical habitats.

2.8.8.5. Analysis of the Reasonable and Prudent Alternative for Mercury

The interim requirement of using a human health criterion that consists of a fish-tissue based water quality criterion of 0.3 mg/kg for mercury to determine permit limits will be followed. Idaho has adopted this criterion, and is implementing it as a 0.24 mg/kg a triggering residue concentration for existing dischargers, using an uncertainty (safety factor) of 0.8 times (IDEQ 2007a). This fish tissue-based criterion is close to being a threshold below which adverse effects are unlikely and is sufficient to protect listed salmon and steelhead species and their habitats.

The adoption of a new chronic aquatic life criterion for mercury within the next 7 years will be subject to ESA consultation to ensure that the new criterion will be adequately protective. Therefore, NMFS concludes that the mercury RPA will not jeopardize any of the listed species considered in this Opinion or adversely modify their critical habitats.

2.8.8.6. Analysis of the Reasonable and Prudent Alternative for Selenium

The interim requirement of monitoring fish tissues and taking corrective action when fish tissues exceed 7.6 mg/kg dw or 2 µg/L in the water column will be sufficiently protective to minimize any food web transfer to concentrations in listed salmon and steelhead. There is one known existing facility within the action area that currently discharges selenium to a stream within critical habitat for Snake River salmon or steelhead at concentrations that approach those described in the RPA (Thompson Creek Mine's discharges to Thompson Creek). As evaluated in Section 2.4.8 (Analysis of Effects), as of 2012, conditions had not resulted in appreciable harm to aquatic life. Any new permits containing discharges of selenium will be subject to individual consultation to assure that jeopardy or adverse modification do not occur. Based on these protective interim practices and the low number of discharges, the continued use of the existing selenium standard during the implementation period will result in only minor adverse effects.

The adoption of a new chronic aquatic life criterion within the next 4 years will be subject to ESA consultation to ensure that the new criterion will be adequately protective. Therefore, NMFS concludes that the selenium RPA will not jeopardize any of the listed species considered in this Opinion or adversely modify their critical habitats

2.9. Incidental Take Statement

Section 9 of the ESA and Federal regulation 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 purposes of 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.[8] 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 ITS.

2.9.1. Amount or Extent of Take

The NPDES permits issued or approved under the rules evaluated in this action are reasonably certain to affect the water quality within critical habitats and subsequently result in incidental take. As such, the proposed action creates the framework through which incidental take will occur. This take, however, is indirect and will only occur through implementation of the WQS analyzed in this Opinion through NPDES permits, TMDLs, and clean-up actions. Because of the future and indirect nature of this take, and due to the large degree of variability in effects caused by future implementation of the criteria, it is not possible for NMFS to attempt to quantify the amount of take with any accuracy. However, the extent of critical habitats with foreseeable water quality changes can be described, which can serve as a surrogate measure of the extent of take (as habitat) rather than the amount of take (as fish). Additionally, a more precise measure of the amount and extent of take will be assessed with each individual NPDES consultation, TMDL or CERCLA cleanup, which occurs subsequent to this consultation.

Although calculating the amount of take has substantial inherent uncertainties, it is reasonably certain that some incidental take will occur. Fish will be present in waters that are affected by discharges permitted under the standards reviewed in this consultation and, in some instances, harm to those fish will occur. As described above, the extent of take likely to occur as a result of the proposed action will be evaluated in individual consultations on a site-specific basis for each NPDES permit issued in the action area. NMFS anticipates the upper bounds of the extent of take through the following assumptions and calculations as follows:

1. Incidental take will occur in the immediate proximity of discharges, i.e., in mixing zones, from permitted active or inactive mining facilities. Some smaller amount of incidental take will also likely occur downstream of the mixing zones but the amount of take will decrease in a downstream direction. The level of take downstream of the mixing zones will be proportional to the level of take within the mixing zones.

[8] 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 NMFS adopts in this consultation is consistent with our understanding of the dictionary definition of harass and is consistent with the U.S. Fish and Wildlife interpretation of the term.

2. As of 2014, there were five such facilities located in the action area (EPA 2010a; NMFS 2011a).

3. To be conservative we doubled the existing number of operating NPDES dischargers that could be operating at one time and assume they are all mines. This would mean up to 10 mining facilities could discharge into critical habitats.

4. Each facility is assumed to have three outfalls with mixing zones located in critical habitats, and each mixing zone persists for 50 m and thus impinges on 50 m of habitat.

The rationales for these assumptions include the following: (1) Mixing zones are a place where criteria are allowed to be exceeded. Take resulting from exposure to toxic substances would be from metals or cyanide because the organic chemicals considered in this Opinion are extremely unlikely to be present in discharges; (2) the conclusion that discharges at criteria concentrations of metals will occur at metals mining facilities, and not the urban or other industrial facilities was based on discussions and analyses regarding EPA's separate consultation on revised cadmium criteria (EPA 2010a; NMFS 2011a) and independent review of recent NPDES monitoring and permitting results that were available online (*epa.gov/r10earth/waterpermits.htm*); (3) it would be conservative to assume an increase in discharges. Since EPA's (2010a) review, at least one new mining discharge has been permitted for the Idaho Cobalt Project, which will discharge into a tributary upstream of critical habitat into Big Deer Creek, a tributary to Panther Creek. Further, active exploration in advance of a potential new mine is going on at the old Stibnite Mine, on the East Fork of the South Fork Salmon River, and NMFS presumes for the purpose of this estimate of potential take that there could be other new exploration and development; and (4) the distance it takes for effluent plumes released from wastewater outfalls to fully mix will vary based on various factors such as the relative flows of the receiving water and effluents, temperature, configuration of the outfall, and channel and substrate characteristics. However, in mountain streams, dye studies and simulation studies have shown that thorough mixing usually occurs within 50 m (or 0.05 km) (IDEQ 1999; Mebane 2000).

Following these four assumptions, an estimate of the extent of take, as stream habitat is 10 facilities with three mixing zones per facility for a total of 30 mixing zones. Each mixing zone is 50 m long and 2.5 meters wide or 125 square meters. This results in a total area of 3,750 square meters. The extent of take authorized under this Opinion will be exceeded if total mixing zone areas exceed 3,750 square meters.

No additional take is authorized for new anthropogenic nonpoint sources of contaminents. These are most likely to occur in highly mineralized areas as a result of mining activities and the most likely response is to require removal, isolation, or treatment of water prior to a discharged occurring. Water treatment may lead to a new NPDES permit and based on that permit may be included in the mixing zone take analysis above.

2.9.2. Effect of the Take

After reviewing the status of Snake River spring/summer Chinook salmon, Snake River fall Chinook salmon, Snake River sockeye salmon, and Snake River Basin steelhead, the status of designated critical habitats, the environmental baseline for the action area, the effects of the proposed action as revised by the RPA, and cumulative effects, NMFS concludes that this level of anticipated take is not likely to result in jeopardy to these species.

2.9.3. Reasonable and Prudent Measures and Terms and Conditions

2.9.3.1. Reasonable and Prudent Measures

"Reasonable and prudent measures" are nondiscretionary measures to minimize the amount or extent of incidental take (50 CFR 402.02). "Terms and conditions" implement the reasonable and prudent measures (50 CFR 402.14). These must be carried out for the exemption in section 7(o)(2) to apply.

NMFS believes the RPMs and terms and conditions described below, are necessary and appropriate to minimize the likelihood of incidental take of ESA-listed species due to implementation of the proposed action.

The EPA shall:

1. Minimize the potential for mixture toxicity in discharges.

2. Minimize the potential adverse effects that occur when discharging under NPDES permits.

3. Minimize exposure of aquatic life to PCP.

4. Use updated procedures for calculating any WERs developed for determining discharge limits.

5. Ensure completion of a monitoring and reporting program to confirm that the terms and conditions in this ITS are effective in avoiding and minimizing incidental take from permitted activities and ensure the amount of incidental take is not exceeded.

2.9.3.2. Term and Conditions

1. To implement RPM No. 1 (minimize the effects of toxicity resulting from simultaneous exposure to mixtures), the EPA shall:

    a. For all discharges that are expected to simultaneously contain two or more toxic substances evaluated in this opinion, or cadmium, this section shall apply to prevent mixture toxicity.

    If discharges and the permit limits are authorized such that >1 cumulative criterion units (CCU) would be calculated to be allowed in receiving waters, then WET testing and biomonitoring shall be included in the permit provisions as described in Appendix E Biomonitoring of Effects. Cumulative criterion units are defined for this purpose as CCU $=\sum(C_d \div CCC)$ where C_d is the projected authorized concentration in the fully mixed receiving waters downstream of the effluent discharge, the CCC is the applicable chronic criterion concentration of each regulated constituent calculated for that location.

2. To implement RPM No. 2 (minimize the potential adverse effects when discharging under NPDES Permits.) for discharges that include silver, nickel and zinc, the EPA shall:

    a. For new discharges: Ensure a zone of passage exists under seasonal flow conditions (see Appendix D). If the regulatory mixing zone is limited to less than or equal to 25% of the volume of a stream, then sufficient zone of passage is presumed to be present.

    b. For existing discharges: When permits are renewed, ensure a zone of passage under seasonal flow conditions. If the regulatory mixing zone is limited to less than or equal to 25% of the volume of a stream, then sufficient zone of passage is presumed to be present. If existing discharges were calculated using greater than 25% of the applicable seasonal flow conditions for applying aquatic life criteria the mixing zone must be reduced to 25% unless one of the following conditions exist:

        (1) An evidence-based "Salmonid Zone of Passage Demonstration" indicates that impeding fish movements is unlikely, or;

        (2) Biological monitoring of aquatic communities in the downstream receiving waters show no appreciable adverse effects relative to reference conditions as described in the Appendix E Biomonitoring of Effects, and biological whole-effluent toxicity testing is consistently negative, defined as follows:

            (a) Whole effluent toxicity (WET) testing shall be required, using at least the 7-day *Ceriodaphnia dubia* three-brood test and the 7-day fathead minnow growth and survival test. If previous testing of a facility's effluents have demonstrated that one test is more sensitive than the other, at EPA's discretion it is acceptable to base further testing on only the more sensitive test. Toxicity

trigger concentrations for WET tests shall also be established using dilution series based upon no more than 25% of the applicable critical flow volume. The dilution series for WET testing (7Q10) shall be designed such that one treatment consists of 100% effluent, and at least one treatment is more dilute than the targeted seasonal flow conditions. Receiving waters upstream of the effluent discharge should be used as dilution water.

The "critical concentration" is defined here as the condition when the smallest permitted dilution factor occurs, modified by a 25% mixing zone fraction. For example, if the minimum effluent dilution occurring at a site is a 1:4 ratio (one part effluent to four parts streamwater), then because only 25% of the measured streamflow is authorized for dilution; then the dilution factor for effluent testing is likewise reduced to 1:1. The critical concentration would then be 50% effluent, i.e., one part each effluent and dilution water.

The WET tests results need to be consistently negative to indicate the absence of appreciable instream toxicity in test conditions that reflect the critical effluent concentration, above. A "negative test result" is produced by a test meeting the performance objectives of a passing test according to EPA (2002c) or EPA (2010c). Test results are considered to be consistently negative if the failure rate is less than one in 20.

(b) If instream biological monitoring shows adverse effects or if WET tests are not consistently negative, then a TIE/TRE must be undertaken to identify and remedy the causes of toxicity, which may include reducing effluent limits as warranted. Because considerable judgment may be involved in designing and carrying out a TIE/TRE, and because the results are performance-based (no detectable toxicity per this subsection, more specific guidance is inappropriate to provide here. Mount and Hockett (2000) provide one example of a TIE/TRE.

3. To implement RPM No. 2 (minimize the potential adverse effects that occur when discharging under NPDES permits.)

   a. For purposes of calculating effluent limits, the effluent discharge volumes and receiving streamflows shall apply the "conservative assumptions" described in Appendix D.

4. To implement RPM No.3 (minimize exposure to pentachlorophenol) the EPA shall:

   a. Whenever possible require wood structures being installed with treated wood should be installed in accordance with the BMPs described in *The Use of Treated Wood Products in Aquatic Environments: Guidelines to West Coast NOAA Fisheries staff for Endangered species Act and Essential Fish Habitat Consultations in Alaska, Northwest and Southwest regions*. October 12, 2009.

5. To implement RPM No. 4 (use WERs conservatively) EPA shall:

    a. Calculate the WER using the lesser of: (1) The site water EC_{50}/ lab water EC_{50} ratios; or, (2) the ratio of site water EC_{50} divided by the (SMAV) for that test organism (e.g., Ceriodaphnia dubia, fathead minnow, or rainbow trout) from an updated criteria dataset as described by EPA (2001a); or,

    b. In the case of copper, the WER should be calculated as the site water BLM-based copper criterion ÷ Hardness-based copper criterion for the same hardness.

6. To implement RPM No. 5 (monitoring and reporting), the EPA shall monitor and report as described below. The goal of the monitoring program is to assure that the level of take described in this opinion in not exceeded by monitoring the extent of take.

    a. Monitoring and Reporting the Extent of Take. To insure that the amount of take is not exceeded EPA shall monitor and report on the amount of take as a term and condition of this Incidental Take Statement. The reporting shall be done each time a new NPDES permit is issued that discharges a toxic substance evaluated in this Opinion into waters containing Snake River salmon or steelhead or their critical habitat.

        The reporting shall include the following:

        (1) A copy of the NPDES permit issued to the facility either as a draft or final permit.

        (2) A calculation of the total area of mixing zones granted for the new permit and for existing permits that discharge a toxic substance into waters occupied by listed salmon or steelhead.

7. To implement RPM No. 5 (monitoring and reporting) Biomonitoring of Effects (in situ biological monitoring and assessment) EPA shall require biomonitoring as described in Appendix E Biomonitoring of Effects. The goal of this monitoring is to assure that the nature of the effects occurring are not greater that those described in the effects section of the Opinion.

2.10. Conservation Recommendations

Section7(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. Specifically, conservation recommendations 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 (50CFR 402.02).

2.10.1. Conservation Recommendation for Arsenic

With an emphasis on arsenic, develop an approach to assess the risk to fish at metals-contaminated sites which addresses exposure to multiple contaminants via both water and dietary routes, and which define samples (e.g., invertebrates, sediment, water), metals, and analyses (total metal, speciated metal) necessary to appropriately quantify risk.

2.10.2. Conservation Recommendations for Silver

Publish updated aquatic life criteria for silver that include a chronic criterion value, using a biotic ligand model (BLM) to account for factors that modify toxicity. Much of the fundamental research into the proof of principal, refinement and validation of the BLM-approaches to define metals bioavailability and toxicity was with silver (Di Toro *et al.* 2001; Paquin *et al.* 2002). As result, the BLMs available for silver may be more mature than those for any other metal except for copper (Niyogi and Wood 2004; this Opinion).

Based on the material reviewed to prepare this opinion, NMFS also believes that adequate data exist to derive BLM-based aquatic life criteria for silver including the development of a chronic criterion.

2.10.3. Conservation Recommendation for Cyanide

Revise the cyanide aquatic life criteria to include temperature dependence. Doing so could alleviate the concern about under protectiveness at temperatures less than 6°C and would be consistent with the EPA Guidelines: *"If the acute toxicity of the material to aquatic animals apparently has been shown to be related to a water quality characteristic such as hardness or particulate matter for freshwater animals or salinity or particulate matter for saltwater animals, a Final Acute Equation should be derived based on that water quality characteristic*" (Stephan *et al*. 1985).

2.10.4. Conservation Recommendation for use of bioassessment data in permitting decisions

NMFS recognizes that EPA's WQBEL strategy and biocriteria efforts have long appreciated that well informed field bioassessments complement single-chemical numerical criteria and whole-effluent toxicity testing. In fact due to the nearly infinite permutations of chemical mixtures possible, field assessments may be one of the few practical means for addressing the issue of interactions, mixture effects and multiple stressors. However, there has been little implementation of bioassessment into permitting decisions. Guidance on how bioassessments could be used with point or non-point source implementation has been developed through EPA's Stressor Identification manual, and the State of Idaho has developed extensive biological monitoring databases and interpretive assessment methodologies. Bioassessment of receiving waters has been required as a monitoring element for receiving waters in NPDES permits issued by EPA in Idaho; however, to our knowledge, the data collected has never been a factor in

determining the adequacy of permit limits in renewal applications. NMFS recommends that EPA develop an approach to effectively use bioassessment data in permitting decisions.

2.11. Reinitiation of Consultation

As provided for in 50 CFR 402.16, reinitiation of formal consultation is required where discretionary Federal agency involvement or control over the action has been retained (or is authorized by law) and if: (1) The amount or extent of take is exceeded; (2) new information reveals effects of the agencies action on listed species or designated critical habitat in a manner or to an extent not considered in this opinion; (3) the agencies 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 is designated that may be affected by the action.

2.12. Summary of Conclusions

Tables 2.12.1 and 2.12.2 provide a summary conclusions, reasonable and prudent alternatives and reasonable and prudent measures. Table 2.12.3 provides a summary of conclusions for organic chemicals.

Table 2.12.1. Aspects of the action that would or would not likely contribute to "adverse modifications" of critical habitat or "jeopardy".

Category	Provisions or chemicals (unless otherwise specified, applies to both acute and chronic criteria)
A. Adverse modifications or jeopardy: Elements of the action that will likely contribute to jeopardizing the continued existence of listed Snake River anadromous salmonids or adversely modify critical habitat.	
General aspects	"*Hardness floor*"
Specific chemical criteria	*Arsenic (chronic), Copper, Cyanide, Selenium (chronic), Mercury (chronic),*
B. No jeopardy or adverse modifications of critical habitats: Elements of the action that are likely to adversely affect listed species, but the magnitude of potential take is unlikely to reach levels that jeopardize the continued existence of listed Snake River anadromous salmonids or adversely modify critical habitat.	
Chemical criteria	*Zinc, PCP, Silver , Nickel, Chromium III, Chromium VI, Lead, Aldrin, Dieldrin, Chlordane, DDTs, Endosulfan, Endrin, Heptachlor, Lindane, PCBs, Toxaphene*

Table 2.12.2. Summary of conclusions on the protectiveness of the Idaho Toxics aquatic life criteria for inorganic chemicals.

Constituent	Criteria	EPA's BA Conclusion for Salmonids	Biological Opinion Conclusion	Likely outcomes if water quality in critical habitats were allowed to be at criteria	Synopsis of reasonable and prudent alternatives or measures (see full text for details)
Arsenic	Acute	Not likely to adversely affect listed species (NLAA)	Unlikely to jeopardize the continued existence of listed species or to result in an adverse modification of critical habitat ("No jeopardy or adverse mod.")	Appreciable adverse effects unlikely	(1) Until a new chronic criterion for arsenic is adopted, ensure that the 10 µg/L recreational use standard is applied in all Water Quality Based Effluent Limitations; and (2) develop a new aquatic life criteria for arsenic that incorporates dietary exposure;
	Chronic	NLAA	Likely to jeopardize the continued existence of listed species and to result in an adverse modification of critical habitat ("Adverse mod. and jeopardy")	Chronic criterion concentrations could lead to food chain contamination and adversely affect growth and survival of salmonids	
Chromium (III) & (VI)	Acute	NLAA	No jeopardy or adverse mod.	Appreciable adverse effects unlikely	None
	Chronic	NLAA	No jeopardy or adverse mod.	Risk to sensitive benthic invertebrates, but effects of a magnitude that would fundamentally alter benthic communities and food webs seems very low.	
Copper	Acute	NLAA	Adverse mod. and jeopardy	Adverse effects predicted from abnormal behavior resulting from loss of sense of smell;	(1) Ensure appropriate biological monitoring is conducted and that an adequate zone of

Constituent	Criteria	EPA's BA Conclusion for Salmonids	Biological Opinion Conclusion	Likely outcomes if water quality in critical habitats were allowed to be at criteria	Synopsis of reasonable and prudent alternatives or measures (see full text for details)
Copper	Chronic	NLAA	Adverse mod. and jeopardy	Adverse effects predicted from abnormal behavior resulting from loss of sense of smell; reduced growth is predicted to result in reduced survival during migration. Habitat effects possible from altered invertebrate communities. Adverse effects of copper at concentrations lower than criteria are more likely in high-calcium waters with low organic carbon concentrations.	passage exists; and (2) adopt EPA's 2007 national recommended aquatic life criteria
Cyanide	Acute	NLAA	Adverse mod. and jeopardy	Lethality to listed salmonids possible under winter conditions.	Conduct appropriate biological monitoring and ensure an adequate zone of passage exists
	Chronic	NLAA	Adverse mod. and jeopardy	Criterion is close to threshold for adverse effects to salmonid reproduction, and swimming ability.	
Lead	Acute	NLAA	No jeopardy or adverse mod.	Appreciable adverse effects unlikely	None
	Chronic	NLAA	No jeopardy or adverse mod.	Appreciable direct adverse effects unlikely; some effects to snails and sensitive benthic invertebrates exposed through both diet and water.	None
Mercury	Acute	NLAA	No jeopardy or adverse mod.	Appreciable adverse effects unlikely	Use EPA's 2001 fish tissue criterion as adopted by IDEQ in 2005.
	Chronic	Likely to adversely affected listed species (LAA)	Adverse mod. and jeopardy	Idaho's aquatic life criterion predicted to result in bioaccumulation in salmonids to levels impairing reproduction and neurological/behavioral problems	

Constituent	Criteria	EPA's BA Conclusion for Salmonids	Biological Opinion Conclusion	Likely outcomes if water quality in critical habitats were allowed to be at criteria	Synopsis of reasonable and prudent alternatives or measures (see full text for details)
Nickel	Acute	NLAA	No jeopardy or adverse mod.	Appreciable adverse effects unlikely	Conduct appropriate biological monitoring and ensure an adequate zone of passage exists.
	Chronic	NLAA	No jeopardy or adverse mod.	Appreciable direct adverse effects unlikely; some effects to snails and sensitive benthic invertebrates exposed through both diet and water.	
Selenium	Acute	NLAA	No jeopardy or adverse mod.	Appreciable adverse effects unlikely	If receiving water concentrations are >2 µg/L then fish tissue monitoring is needed.
Selenium	Chronic	LAA	Adverse mod. and jeopardy	Predicted to bioaccumulate via food chain transfer to burdens linked to reduced growth and survival of juvenile salmonids.	If whole-body fish tissues in juvenile salmonids or adult sculpin are greater than 7.6 mg/kg dw, then remedial steps to reduce Se loading are needed.
Silver	Acute	NLAA	No jeopardy or adverse mod.	Reduced survival from short or long-term exposures.	Conduct appropriate biological monitoring and ensure an adequate zone of passage exists.
	Chronic			No separate chronic criterion; acute criterion was assumed by EPA to protect against chronic effects	
Zinc	Acute	NLAA	No jeopardy or adverse mod.	Some risk of lethality for sensitive life stages and sizes under some water chemistry conditions	Conduct appropriate biological monitoring and ensure an adequate zone of passage exists.
	Chronic	NLAA	No jeopardy or adverse mod.	Species adverse effects unlikely, Habitat indirect effects to co-occurring species possible	

Table 2.12.3. Summary of conclusions on the protectiveness of Idaho aquatic life criteria for organic chemicals

Chemical	EPA's BA Conclusion for Salmonids	Biological Opinion Conclusion	Reasonable and Prudent Measures to Minimize Take (RPMs)	Notes
Organics				
Aldrin	NLAA	No jeopardy or adverse mod.	None	
Dieldrin	NLAA	No jeopardy or adverse mod.	None	
Chlordane	NLAA	No jeopardy or adverse mod.	None	
DDTs	NLAA	No jeopardy or adverse mod.	None	
Endosulfan	NLAA	No jeopardy or adverse mod.	None	Sub-criteria adverse effects documented, but no registered uses.
Endrin	NLAA	No jeopardy or adverse mod.	None	
Heptachlor	NLAA	No jeopardy or adverse mod.	None	Some uncertainty about chronic criterion protectiveness from bioaccumulation but fish consumption based criteria is also applicable and is sufficiently low to make risks of harm via bioaccumulation unlikely.
Lindane	NLAA	No jeopardy or adverse mod.	None	

Chemical	EPA's BA Conclusion for Salmonids	Biological Opinion Conclusion	Reasonable and Prudent Measures to Minimize Take (RPMs)	Notes
PCBs	NLAA	No jeopardy or adverse mod.	None	Product not manufactured in the USA. Human-health (HH) based criteria is lower than aquatic life criterion (ALC). HH criterion is likely sufficiently protective for all life stages and prey
PCP	NLAA	No jeopardy or adverse mod.	Use appropriate BMPs for construction in or around water.	At subcriteria concentrations, maladaptive behavior in rainbow trout occurred, and reduced growth in sockeye salmon
Toxaphene	NLAA	No jeopardy or adverse mod.	None	

3. MAGNUSON-STEVENS FISHERY CONSERVATION AND MANAGEMENT ACT ESSENTIAL FISH HABITAT CONSULTATIONS

The consultation requirement of section 305(b) of the MSA directs Federal agencies to consult with NMFS on all actions or proposed actions that may adversely affect EFH. The MSA (section 3) defines EFH as "those waters and substrate necessary to fish for spawning, breeding, feeding, or growth to maturity." Adverse effects include the direct or indirect physical, chemical, or biological alterations of the waters or substrate and loss of, or injury to, benthic organisms, prey species and their habitat, and other ecosystem components, if such modifications reduce the quality or quantity of EFH. Adverse effects on EFH may result from actions occurring within EFH or outside EFH, and may include site-specific or EFH-wide impacts, including individual, cumulative, or synergistic consequences of actions (50 CFR 600.810). Section 305(b) also requires NMFS to recommend measures that can be taken by the action agency to conserve EFH.

This analysis is based, in part, on the BA provided by the EPA and descriptions of EFH for Pacific coast salmon (PFMC 1999) contained in the fishery management plans developed by the Pacific Fishery Management Council (PFMC) and approved by the Secretary of Commerce.

3.1. Essential Fish Habitat Affected by the Project

The proposed action and action area for this consultation are described in Section 1.4 of this document. Juvenile (rearing and migratory) and adult (migratory and spawning) spring/summer Chinook salmon EFH will be affected by the proposed action. Based on information provided in the BA and the analysis of effects presented in the ESA portion of this document, NMFS concludes that the proposed action would adversely affect Pacific Coast salmon EFH. The affected habitat potentially includes all of the critical habitat in Idaho as described in Table 1.4.1. NMFS has considered areas designated as critical habitat under the ESA to be synonymous with EFH. However, as a practical matter our EFH discussion in this section we will limited our description to existing mixing zones where discharges are currently occurring as described in the incidental take statement in Section 2.9. Together these mixing zones represent approximately 1 acre of EFH.

3.2. Adverse Effects on Essential Fish Habitat

Because the action area's designated critical habitat is nearly identical to EFH for the effects are also the same. Effects to critical habitat were discussed in the previous Opinion (Section 2.4) and are incorporated by reference for the effects to EFH. In the preceding opinion, NMFS determined the action's effects to critical habitat, and thus to EFH, will have the following adverse effects on EPH designated for salmon.

1. Disharges of toxic substances into EFH will result in reduced water quality in the water column and accumulate in sediments at levels that directly affects the suitability of the Habitat for listed species.

2. Discharges of toxic substances into EFH will result increased concentrations in macroinvertebrate tissues at concentrations that make them harmful in the diets of salmonids.

3. Discharges of toxic substances into EFH will result will result in fewer macroinvertebrates in the habitat resulting in reduced food sources in the habitat.

3.3. Essential Fish Habitat Conservation Recommendations

1. For mercury and arsenic use the recreational use standard, human health criteria for all water quality based effluent limitations in permits until new criteria can be promulgated.

2. For copper, cyanide and selenium assure that any permitting decisions allowing discharges contain an adequate zone of passage and include any other provisions available to reduce contaminant loading in the discharges until new criteria can be promulgated.

3. Provide adequate monitoring in NPDES permits to assure that mixture toxicity and bioaccumulation is not occurring in either the habitat or species.

NMFS expects that full implementation of these EFH Conservation Recommendations will protect, by avoiding or minimizing the adverse effects described in Section 3.2 above, on approximately 1 acre of designated EFH for Pacific coast salmon.

3.4. Statutory Response Requirement

As required by section 305(b)(4)(B) of the MSA, the Federal agency must provide a detailed response in writing to NMFS within 30 days after receiving an EFH Conservation Recommendation from NMFS. Such a response must be provided at least 10 days prior to final approval of the action if the response is inconsistent with any of NMFS' EFH Conservation Recommendations, unless NMFS and the Federal agency have agreed to use alternative time frames for the Federal agency response. The response must include a description of measures proposed by the agency for avoiding, mitigating, or offsetting the impact of the activity on EFH. In the case of a response that is inconsistent with NMFS Conservation Recommendations, the Federal agency must explain its reasons for not following the recommendations, including the scientific justification for any disagreements with NMFS over the anticipated effects of the action and the measures needed to avoid, minimize, mitigate, or offset such effects [50 CFR 600.920(k)(1)].

In response to increased oversight of overall EFH program effectiveness by the Office of Management and Budget, NMFS established a quarterly reporting requirement to determine how many conservation recommendations are provided as part of each EFH consultation and how many are adopted by the action agency. Therefore, NMFS asks that in your statutory reply

to the EFH portion of this consultation, you clearly identify the number of conservation recommendations accepted.

3.5. Supplemental Consultation

The EPA must reinitiate EFH consultation with NMFS if the proposed action is substantially revised in a way that may adversely affect EFH, or if new information becomes available that affects the basis for NMFS' EFH conservation recommendations [50 CFR 600.920(l)].

4. 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 [DQA]) specifies three components contributing to the quality of a document. They are utility, integrity, and objectivity. This section of the Opinion addresses these DQA components, documents compliance with the DQA, and certifies that this Opinion has undergone pre-dissemination review.

4.1. Utility

"Utility" principally refers to ensuring that the information contained in this consultation is helpful, serviceable, and beneficial to the intended users. This ESA consultation concludes that the proposed project will not jeopardize the affected Snake River Basin steelhead and Snake River spring/summer Chinook salmon. Therefore, the EPA can implement this action in accordance with their authorities under the CWA and CERCLA. The intended users of this Opinion are the EPA and any of their cooperators, contractors, or permittees. Individual copies of this Opinion were provided to the agencies. This Opinion will be posted on the NMFS West Coast Region web site (http://www.westcoast.fisheries.noaa.gov) . The format and naming adheres to conventional standards for style.

4.2. 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.

4.3. 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, ESA regulations, 50 CFR 402.01, et seq., and the MSA implementing regulations regarding EFH, 50 CFR 600.

Best Available Information: This consultation and supporting documents use the best available information, as referenced in the References section. The analyses in this Opinion/EFH consultation contain 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 and MSA implementation, and reviewed in accordance with West Coast Region ESA quality control and assurance processes.

5. References

Abel, P.D. 1980. Toxicity of g-hexachlorocyclohexane (lindane) to *Gammarus pulex*: Mortality in relation to concentration and duration of exposure. Freshwater. Biol. 10:251B259.

Adams, E.S. 1975. Effects of lead and hydrocarbons from snowmobile exhaust on brook trout (*Salvelinus fontinalis*). *Transactions of the American Fisheries Society*. 104(2): 363–373

Adams, N.W.H. and J.R. Kramer. 1999. Silver speciation in wastewater effluent, surface waters, and pore waters. *Environmental Toxicology and Chemistry*. 18(12): 2667-2673. *http://dx.doi.org/10.1002/etc.5620181203*

Adema, D.M.M. 1978. Daphnia magna as a test animal in acute and chronic toxicity tests. Hydrobiol. 59: 125-134.

Albright, L.J., P.C. Oloffs, and S.Y. Szeto. 1980. Residues in cutthroat trout (Salmo clarki) and California newts (Tarichia tarosa) from a lake treated with technical chlordane. J. Environ. Sci. Health, Part B., 15:333-349.

Aldegunde, M., J. Soengas, C. Ruibal, and M. Andrés. 1999. Effects of chronic exposure to γ-HCH (lindane) on brain serotonergic and gabaergic systems, and serum cortisol and thyroxine levels of rainbow trout, Oncorhynchus mykiss. *Fish Physiology and Biochemistry*. 20(4): 325-330Aldenberg, T. and J.S. Jaworska. 2000. Uncertainty of the hazardous concentration and fraction affected for normal species sensitivity distributions. *Ecotoxicology and Environmental Safety*. 46(1): 1-18

Aldenberg, T. and J.S. Jaworska. 2000. Uncertainty of the hazardous concentration and fraction affected for normal species sensitivity distributions. *Ecotoxicology and Environmental Safety*. 46(1): 1-18

Allison, D. et al. 1963. Insecticides: Effects on cutthroat trout of repeated exposure to DDT. Science 142:958.

Allison, D., B.J. Kallman, O.B. Cope, and C.C. Van Vallin. 1964. Some chronic effects of DDT on cutthroat trout. Washington DC, U.S. Department of the Interior, Bureau of Sport, Fisheries, and Wildlife, pp. 1-30. (Resource Report No. 64).

Alsop, D.H., J.C. McGeer, D.G. McDonald, and C.M. Wood. 1999. Costs of chronic water-borne zinc exposure and the consequences of zinc acclimation on the gill/zinc interactions of rainbow trout in hard and soft water. Environ. Toxicol. Chem. 18:1014-1025.

Amita Rani, B. and M. Krishnakumari. 1995. Prenatal toxicity of heptachlor in albino rats. *Pharmacology & toxicology*. 76(2): 112-114

Anderson, R.L., and D.L. DeFoe. 1980. Toxicity and bioaccumulation of endrin and methoxychlor in aquatic invertebrates and fish. Environ. Pollut. 22A(2): 111-121.

Anderson, J.H. and T.P. Quinn. 2007. Movements of adult coho salmon (Oncorhynchus kisutch) during colonization of newly accessible habitat. *Canadian Journal of Fisheries and Aquatic Sciences*. 64(8): 1143-1154. *http://dx.doi.org/10.1139/f07-087*

Anderson, P.D. and P.A. Spear. 1980. Copper pharmacokinetics in fish gills—II body size relationships for accumulation and tolerance. *Water Research*. 14(8): 1107-1111

Anderson, P.D. and L.J. Weber. 1975. Toxic response as a quantitative function of body size. *Toxicology and Applied Pharmacology*. 33(3): 471-483

Anestis, I, and R.J. Neufeld. 1986. Avoidance-preference reactions of rainbow trout (*Salmo gairdneri*) after prolonged exposure to chromium (VI). Wat. Res. 20: 1233-1241.

Apperson, K.A., and P.J. Anders. 1990. Kootenal River white sturgeon investigations and experimental culture. Annual progress report, FY 1989. Idaho Dept. of Fish and Game, Boise (USA). Fisheries Research Sect. U.S. Dept. of Energy Report. 66 pp

Arkoosh, M.R., E. Casillas, E. Clemons, A.N. Kagley, R. Olson, P. Reno, and J.E. Stein. 1998a. Effect of pollution on fish diseases: Potential impacts on salmonid populations. *Journal of Aquatic Animal Health*. 10(2): 182-190

Arkoosh, M.R., E. Casillas, P. Huffman, E. Clemons, J. Evered, J.E. Stein, and U. Varanasi. 1998b. Increased susceptibility of juvenile chinook salmon (*Oncorhynchus tshawytscha*) from a contaminated estuary to the pathogen Vibrio anguillarum. Trans. Amer. Fish. Soc. 127:360-374.

Arnold, H., H-J. Pluta, and T. Braunbeck. 1996. Cytological alterations in the liver of rainbow trout Oncorhynchus mykiss after prolonged exposure to low concentrations of waterborne endosulfan. Dis. Aquat. Org. 25:39-52.

Arthaud, D.L., C.M. Greene, K. Guilbault, and J.V. Morrow. 2010. Contrasting life-cycle impacts of stream flow on two Chinook salmon populations. Hydrobiologia. 655(1): 171-188. http://dx.doi.org/10.1007/s10750-010-0419-0

Arukwe, A., T. Celius, B.T. Walther, and A. Goksoeyr. 2000. Effects of xenoestrogen treatment on zona radiata protein and vitellogenin expression in Atlantic salmon (Salmo salar). Aquat. Toxicol. 49:159-170.

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

Asfaw, A., M.R. Ellersieck, and F.L. Mayer. 2004. Interspecies correlation estimations (ICE) for acute toxicity to aquatic organisms and wildlife, II. User manual and software. U.S. Environmental Protection Agency, EPA/600/R-03/105. 20 pp.

ASTM (American Society for Testing and Materials). 1997. Standard guide for conducting acute toxicity tests on test materials with fishes, macroinvertebrates, and amphibians. Method E729-96. Pages 22 *in Annual Book of ASTM Standards*, volume 11.04. American Society for Testing and Materials, West Conshohocken, PA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1989. U.S. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Chlordane (ATSDR/TP-89/06). U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. Toxicological Profile for Chlordane (Draft). U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Toxicological profile for heptachlor and heptachlor epoxide. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

ATSDR (Agency for Toxic Substances and Diseases Registry). 1994. Toxicological Profile for 4,4'-DDT, 4,4'-DDE, 4, 4'-DDD (Update). U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1996. Toxicological profile for endrin. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

Azharbig, M, K. Vijay Joseph, P. Madhavilatha, K. Jayantharao. 1990. Effect of heptachlor on freshwater fish Channa punctatus and ATPase activity in functionally different muscles. Environment and Ecology 8:480-481.

Bagchi, D., M. Bagchi, E. Hassoun, and S.J. Stohs. 1992a. Endrin-induced urinary excretion of formaldehyde, acetaldehyde, malondialdehyde and acetone in rats. *Toxicology*. 75(1): 81-89. http://dx.doi.org/10.1016/0300-483X(92)90128-2

Bagchi, M., E.A. Hassoun, D. Bagchi, and S.J. Stohs. 1992b. Endrin-induced increases in hepatic lipid peroxidation, membrane microviscosity, and DNA damage in rats. *Archives of Environmental Contamination and Toxicology*. 23(1): 1-5

Bagchi, D., E.A. Hassoun, M. Bagchi, and S.J. Stohs. 1993. Protective effects of antioxidants against endrin-induced hepatic lipid peroxidation, DNA damage, and excretion of urinary lipid metabolites. *Free Radical Biology and Medicine*. 15(2): 217-222. http://dx.doi.org/10.1016/0891-5849(93)90062-Y

Baldwin, D.H., J.A. Spromberg, T.K. Collier, and N.L. Scholz. 2009. A fish of many scales: extrapolating sublethal pesticide exposures to the productivity of wild salmon populations. *Ecological Applications*. 19(8): 2004-2015. *http://dx.doi.org/10.1890/08-1891.1*

Balistrieri, L.S. and R.G. Blank. 2008. Dissolved and labile concentrations of Cd, Cu, Pb, and Zn in the South Fork Coeur d'Alene River, Idaho: Comparisons among chemical equilibrium models and implications for biotic ligand models. *Applied Geochemistry*. 23(12): 3355-3371. *http://dx.doi.org/10.1016/j.apgeochem.2008.06.031*

Balistrieri, L.S., D.A. Nimick, and C.A. Mebane. 2012. Assessing time-integrated dissolved concentrations and predicting toxicity of metals during diel cycling in streams. *Science of the Total Environment*. 425: 155–168. *http://dx.doi.org/10.1016/j.scitotenv.2012.03.008*

Ball, A.L., U. Borgmann, and D.G. Dixon. 2006. Toxicity of a cadmium-contaminated diet to *Hyalella azteca*. *Environmental Toxicology and Chemistry*. 25(9): 2526–2532

Bansal, S.K., S.R. Verma, A.K. Gupta, and R.C. Dalela. 1980. Predicting long-term toxicity by subacute screening of pesticides with larvae and early juveniles of four species of freshwater major carp. Ecotoxicol. Environ. Safety. 4(3): 224-231.

Barata, C. and D.J. Baird. 2000. Determining the ecotoxicological mode of action of chemicals from measurements made on individuals: results from instar-based tests with *Daphnia magna* Straus. *Aquatic Toxicology*. 48(2/3): 195-209

Barber, T.R., C.C. Lutes, M.R.J. Doorn, P.C. Fuchsman, H.J. Timmenga, and R.L. Crouch. 2003. Aquatic ecological risks due to cyanide releases from biomass burning. *Chemosphere*. 50(3): 343-348

Barnthouse, L.W., G.W. Suter, II, and A.E. Rosen. 1989. Inferring population-level significance from individual-level effects: An extrapolation from fisheries science to ecotoxicology. Pages 289-300 *in Aquatic Toxicology and Hazard Assessment: Eleventh Volume, ASTM STP 1007*. American Society for Testing and Materials (ASTM), Philadelphia, PA.

Barron, M.G., and S. Albeke. 2000. Calcium control of zinc uptake in rainbow trout. Aq. Toxicol. 50: 257-264.

Barry, M. J., D.C. Logan, J.T. Ahokas, and D.A. Holdway. 1995. Effect of algal food concentration on toxicity of two agricultural pesticides to Daphnia carinata. Ecotoxicol. Environ. Saf. 32:273-279.

Baxter, C.V. 2002. Fish movement and assemblage dynamics in a Pacific Northwest riverscape. Ph.D dissertation. 188 pp. Department of Fisheries and Wildlife, Oregon State University, Corvallis.

Becker, D.S. and G.N. Bigham. 1995. Distribution of mercury in the aquatic food web of Onondaga Lake, New York. *Water, Air, and Soil Pollution*. 80(1-4): 563-571

Beckvar, N., T.M. Dillon, and L.B. Read. 2005. Approaches for linking whole-body fish tissue residues of mercury or DDT to biological effects thresholds. *Environmental Toxicology and Chemistry*. 24(8): 2094-2105

Bedford, J.W, and M.J. Zabik. 1973. Bioactive Compounds in the Aquatic Environment: Uptake and Loss of DDT and Dieldrin by Freshwater Mussels. Arch. Environ. Contam. Toxicol. 1:97-111.

Behnke, R.J. and J.R. Tomelleri. 2002. Trout and Salmon of North America. Simon and Schuster, New York. 384 pp

Bell, M.C. 1986. Fisheries handbook of engineering requirements and biological criteria. Fish passage development and evaluation program, Corps of Engineers, North Pacific Division, Portland, Oregon. 290 pages.

Beltman, D.J., J. Holmes, J. Lipton, A.S. Maest, and J. Schardt. 1993. Blackbird Mine site source investigation - Field sampling report. Prepared for the State of Idaho. RCG/Hagler, Bailly, Inc., Boulder, CO April 21, 1993. 244 pp.

Beltman, D.J., J. Lipton, D. Cacela, and W.H. Clements. 1994. Benthic invertebrate injury studies, Blackbird Mine, Idaho. Prepared for the State of Idaho. RCG/Hagler Bailly and Colorado State University. 195 pp.

Beltman, D.J., J. Lipton, D. Cacela, and W.H. Clements. 1999. Benthic invertebrate metals exposure, accumulation, and community-level effects downstream from a hard-rock mine site. *Environmental Toxicology and Chemistry*. 18(2): 299-307. *http://dx.doi.org/10.1002/etc.5620180229*

Belzile, N., Y.-W. Chen, J.M. Gunn, J. Tong, Y. Alarie, T. Delonchamp, and C.-Y. Lang. 2006. The effect of selenium on mercury assimilation by freshwater organisms. *Canadian Journal of Fisheries and Aquatic Sciences*. 63(1): 1-10

Benguira, S., and A. Hontela. 2000. Adrenocorticotrophin- and cyclic adenosine 3', 5' monophosphate-stimulated cortisol secretion in interregnal tissue of rainbow trout exposed in vitro to DDT compounds. Environmental Toxicology and Chemistry 194:842-847.

Bennett, R.O., and R.E. Wolke. 1987a. The effect of sublethal endrin exposure on rainbow trout, Salmo gairdneri Richardson. 1. Evaluation of serum cortisol concentrations and immune responsiveness. Jour. Fish. Biol. 31:375-385.

Bennett, R.O., and R.E. Wolke. 1987b. The effect of sublethal endrin exposure on rainbow trout, Salmo gairdneri Richardson. 2. The effect of altering serum cortisol concentrations on the immune response. Jour. Fish. Biol. 31:387-394.

Bentzen, E., D.R.S. Lean, W.D. Taylor, and D. Mackay. 1996. Role of food web structure on lipid and bioaccumulation of organic contaminants by lake trout (Salvelinus namaycush). *Canadian Journal of Fisheries and Aquatic Sciences*. 53(11): 2397-2407

Berejikian, B.A., R.J.F. Smith, E.P. Tezak, S.L. Schroder, and C.M. Knudsen. 1999. Chemical alarm signals and complex hatchery rearing habitats affect antipredator behavior and survival of chinook salmon (*Oncorhynchus tshawytscha*) juveniles. *Canadian Journal of Fisheries and Aquatic Sciences*. 56(5): 830-838

Berglund, O., P. Larsson, C. Bronmark, L. Greenberg, A. Eklov, and L. Okla. 1997. Factors influencing organochlorine uptake in age-O brown trout (Salmo trutta) in iotic environments. *Canadian Journal of Fisheries and Aquatic Sciences*. 54(12): 2767-2774

Bergman, H.L., and E.J. Dorward-King. 1997. Reassessment of Metals Criteria for Aquatic Life Protection. SETAC Technical Publication Series. Society of Environmental Toxicology and Chemistry, Pensacola, Florida.

Berlin, W.H., Hesselberg R.J., and M.J. Mac. 1981. Chlorinated hydrocarbons as a factor in the reproduction and survival of lake trout (*Salvelinus namaycush*) in Lake Michigan. U.S. Fish and Wildlife Service Technical Papers, Number 105, 11-22.

Berman E., M. Schlicht, V.C. Moser, and R.C. MacPhail. 1995. A multidisciplinary approach to toxicology screening. I. Systemic toxicity. J Toxicol Environ Health 45:127-143.

Berntssen, M.H.G., A. Aatland, and R.D. Handy. 2003. Chronic dietary mercury exposure causes oxidative stress, brain lesions, and altered behaviour in Atlantic salmon (*Salmo salar*) parr. *Aquatic Toxicology*. 65(1): 55-72

Besser, J.M., T.J. Canfield, and T.W. La Point. 1993. Bioaccumulation of organic and inorganic selenium in a laboratory food chain. *Environmental Toxicology and Chemistry*. 12(1): 57-72. *http://dx.doi.org/10.1002/etc.5620120108*

Besser, J. M., J.A. Kubitz, C.G. Ingersoll, W.E. Braselton and J.P. Giesy. 1995. Influences on copper bioaccumulation, growth, and survival of the midge, *Chironomus tentans*, in metal-contaminated sediments. Journal of Aquatic Ecosystem Health 4: 157-168.

Besser, J. M., W.G. Brumbaugh, T.W. May, S.E. Church, and B.A. Kimball. 2001a. Bioavailability of metals in stream food webs and hazards to brook trout (*Salvelinus fontinalis*) in the upper Animas River watershed, Colorado. Arch. Environ. Contam. Toxicol. 40: 48-59.

Besser, J.M., A.L. Allert, D.K. Hardesty, C.G. Ingersoll, J.T. May, N. Wang, and K.J. Lieb. 2001b. Evaluation of metal toxicity in streams of the upper Animas River watershed, Colorado. U.S. Geological Survey, Biological Science Report 2001–001.

Besser, J.M., W.G. Brumbaugh, N.E. Kemble, T.W. May, and C.G. Ingersoll. 2004. Effects of sediment characteristics on the toxicity of chromium(III) and chromium(VI) to the amphipod,*Hyalella azteca*. *Environmental Science and Technology*. 38(23). *http://dx.doi.org/10.1021/es049715i*

Besser, J.M., W.G. Brumbaugh, E.L. Brunson, and C.G. Ingersoll. 2005a. Acute and chronic toxicity of lead in water and diet to the amphipod *Hyalella azteca*. *Environmental Toxicology and Chemistry*. 24(7): 1807–1815. *http://dx.doi.org/10.1897/04-480R.1*

Besser, J.M., N. Wang, F.J. Dwyer, F.L. Mayer, and C.G. Ingersoll. 2005b. Assessing contaminant sensitivity of endangered and threatened fishes: 2. Chronic toxicity of copper and pentachlorophenol to two endangered species and two surrogate species. *Archives of Environmental Contamination and Toxicology*. 48(2): 155-165

Besser, J.M., C.A. Mebane, D.R. Mount, C.D. Ivey, J.L. Kunz, E.I. Greer, T.W. May, and C.G. Ingersoll. 2007. Relative sensitivity of mottled sculpins (*Cottus bairdi*) and rainbow trout (*Oncorhynchus mykiss*) to toxicity of metals associated with mining activities. *Environmental Toxicology and Chemistry*. 26(8): 1657–1665 *http://dx.doi.org/10.1897/06-571R.1*

Besser, J.M., W.G. Brumbaugh, C.D. Ivey, C.G. Ingersoll, and P.W. Moran. 2008. Biological and chemical characterization of metal bioavailability in sediments from Lake Roosevelt, Columbia River, Washington, USA. *Archives of Environmental Contamination and Toxicology*. 54(4): 557–570

Besser, J.M., D.K. Hardesty, E.I. Greer, and C.G. Ingersoll. 2009. Sensitivity of freshwater snails to contaminants: chronic toxicity tests with endangered species and surrogates. U.S. Geological Survey, Columbia Environmental Research Laboratory, Administrative Report to the U.S. Environmental Protection Agency, Columbia, MO. 47 pp.

Bianchi, A., K.C. Bowles, C.J. Brauner, J.W. Gorsuch, J.R. Kramer, and C.M. Wood. 2002. Evaluation of the effect of reactive sulfide on the acute toxicity of silver (I) to *Daphnia magna*. part 2: toxicity results. *Environmental Toxicology and Chemistry*. 21(6): 1294–1300

Biddinger, G.R., and S.P. Gloss. 1984. The importance of trophic transfer in the bioaccumulation of chemical contaminants in aquatic ecosystems. Res. Review 91:103-145.

Bierman, V.J. 1990. Equilibrium partitioning and biomagnification of organic chemicals in benthic animals. Environ. Sci. Technol. 24:1407-1412.

Billard, R., and P. Roubaud. 1985. The Effect of Metals and Cyanide on Fertilization in Rainbow Trout. Water Res. 19:209-214.

Birge, W.J., J.E. Hudson, J.A. Black, and A.G. Westerman. 1978. Embryo-larval bioassays on inorganic coal elements and in situ biomonitoring of coal-waste effluents. Pages 97-104 in: Samuel, D.E., J.R. Stauffer, C.H. Hocutt, and W.T. Mason. [Eds.]. Surface mining and fish/wildlife needs in the eastern United States: Proceedings of a symposium December 3-6, 1978. FWS/OBS-78/81.

Birge, W.J., J.A. Black, A.G. Westerman, and J.E. Hudson. 1980. Aquatic toxicity tests on inorganic elements occurring in oil shale. Pages 519-534 *in* D. E. Samuel, J. R. Stauffer, C. H. Hocutt, and W. T. Mason, editors. *Oil shale symposium: Sampling, analysis equality assurance, March 1979, EPA 600/9-80-022*. U.S. Environmental Protection Agency, Cincinatti, OH.

Birge, W.J., J.A. Black, and B.A. Ramey. 1981. The reproductive toxicology of aquatic contaminants. P. 59-115 in: Saxena, J., and F. Fisher [Eds.]. Hazard assessment of chemicals: Current developments. Academic Press, New Yory, NY.

Bjornn, T.C. and N. Horner. 1980. Biological Criteria for classification of Pacific salmon and steelhead as threatened or endangered under the Endangered Species Act. Idaho Cooperative Fishery Research Unit, National Marine Fisheries Service, Moscow, ID

Bjornn, T.C. and D.W. Reiser. 1991. Habitat requirements of salmonids in streams. Pages 83-138 *in* W. R. Meehan, editor. *Influences of forest and rangeland management on salmonid fishes and their habitats. American Fisheries Society Special Publication 19*, Bethesda, MD.

Bjornn, T.C., D.R. Craddock, and D.R. Corley. 1968. Migration and survival of Redfish Lake, Idaho, sockeye salmon, *Oncorhynchus nerka*. Transactions of the American Fisheries Society 97:360-373.

Black, J.A. and W.J. Birge. 1980. An Avoidance Response Bioassay for Aquatic Pollutants. University of Kentucky Water Resources Research Institute Research Report 123. 40 pp.

Blackwood, L.G. 1992. The lognormal distribution, environmental data, and radiological monitoring. *Environmental Monitoring and Assessment*. 21(3): 193-210. *http://dx.doi.org/10.1007/BF00399687*

Blockwell, S.J., D. Pascoe, and E.J. Taylor. 1996. Effects of lindane on the growth of the freshwater amphipod Gammarus pulex (L.) Chemosphere 32:1795-1803.

Blockwell, SJ; Maund, SJ; Pascoe, D. 1998. The acute toxicity of lindane to Hyalella azteca and the development of a sublethal bioassay based on precopulatory guarding behavior. Archives Of Environmental Contamination and Toxicology; 35:432-440.

Bloom, N.S. 1992. On the chemical form of mercury in edible fish and marine invertebrate tissue. *Canadian Journal of Fisheries and Aquatic Sciences*. 49(5): 1010-1017. *http://dx.doi.org/10.1139/f92-113*

Bloom, N.S. and S.W. Effler. 1990. Seasonal variability in the mercury speciation of Onondaga Lake (New York) *Water, Air, and Soil Pollution*. 53(3-4): 251-265

Boese, B.L., M. Winsor, H. Lee II, S. Echols, J. Pelletier, and R. Randall. 1995. PCB congeners and hexachlorobenzene biota sediment accumulation factors for *Macoma nasuta* exposed to sediments with different total organic carbon contents. Environ. Toxicol. Chem. 14:303-310.

Booth, D.B., D. Hartley, and R. Jackson. 2002. Forest cover, impervious-surface area, and the mitigation of stormwater impacts. *JAWRA Journal of the American Water Resources Association*. 38(3): 835-84

Borgert, C.J. 2004. Chemical mixtures: An unsolvable riddle? *Human and Ecological Risk Assessment*. 10(4): 619-629

Borgmann, U., K.M. Ralph, and W.P. Norwood. 1989. Toxicity test procedures for *Hyalella azteca*, and chronic toxicity of cadmium and pentachlorophenol to *H. azteca, Gammarus fasciatus,* and *Daphnia magna*. *Archives of Environmental Contamination and Toxicology*. 18(5): 756-764

Borgmann, U., Y. Couillard, P. Doyle, and D.G. Dixon. 2005a. Toxicity of sixty-three metals and metalloids to *Hyalella azteca* at two levels of water hardness. *Environmental Toxicology and Chemistry*. 24(3): 641–652

Borgmann, U., M. Nowierski, and D.G. Dixon. 2005b. Effect of major ions on the toxicity of copper to *Hyalella azteca* and implications for the biotic ligand model. *Aquatic Toxicology*. 73(3): 268-287

Bowler, B. 1990. Additional information on the status of Snake River sockeye salmon. Report submitted to ESA Administrative Record for sockeye salmon. Idaho Department of Fish and Game, 600 South Walnut Street, Boise, Idaho 83707. 23 pages. December 1990.

Bowman, M.C., W.L Oller, and T. Cairns. 1981. Stressed Bioassay Systems for Rapid Screening of Pesticide Residues. Part I: Evaluation of Bioassay Systems. Arch. Environm. Contam. Toxicol. 10:9-24.

Boyle, D., K.V. Brix, H. Amlund, A.-K. Lundebye, C. Hogstrand, and N.R. Bury. 2008. Natural arsenic contaminated diets perturb reproduction in fish. *Environmental Science and Technology*. 42(14): 5354 - 5360. *http://dx.doi.org/10.1021/es800230w*

Bradley, R.W., C. DuQuesnay, and J.B. Sprague. 1985. Acclimation of rainbow trout, *Salmo gairdneri* Richardson, to zinc: kinetics and mechanism of enhanced tolerance induction. *Journal of Fish Biology*. 27: 367-379

Bremle, G., L. Okla, and P. Larsson. 1995. Uptake of PCBs in fish in a contaminated river system: bioconcentration factors measured in the field. Environ. Sci. Technol. 29: 2010-2015.

Brinkman, S.F. and D. Hansen. 2004. Effect of hardness on zinc toxicity to Colorado River cutthroat trout (*Oncorhynchus clarki pleuriticus*) and rainbow trout (*Oncorhynchus mykiss*) embryos and fry. Pages 22-35 *in Water Pollution Studies, Federal Aid in Fish and Wildlife Restoration Project F-243-R11*. Colorado Division of Wildlife, Fort Collins, *http://wildlife.state.co.us/Research/Aquatic/Publications/*.

Brinkman, S.F. and D. Hansen. 2007. Toxicity of cadmium to early life stage brown trout (*Salmo trutta*) at multiple hardnesses. *Environmental Toxicology and Chemistry*. 26(8): 1666–1671. *http://dx.doi.org/10.1897/06-376*

Brix, K.V., J. Keithly, D.K. DeForest, and J. Laughlin. 2004. Acute and chronic toxicity of nickel to rainbow trout (*Oncorhynchus mykiss*). *Environmental Toxicology and Chemistry*. 23(9): 2221-2228. *http://dx.doi.org/10.1897/03-38*

Brix, K.V., D.K. DeForest, M. Burger, and W.J. Adams. 2005. Assessing the relative sensitivity of aquatic organisms to divalent metals and their representation in toxicity datasets compared to natural aquatic communities. *Human and Ecological Risk Assessment*. 11(6): 1139-1156

Brix, K.V., D.K. DeForest, and W.J. Adams. 2011. The sensitivity of aquatic insects to divalent metals: A comparative analysis of laboratory and field data. *Science of the Total Environment*. 409(20): 4187-4197. *http://dx.doi.org/10.1016/j.scitotenv.2011.06.061*

Brown, V.M., T.L. Shaw, and D. Shurben. 1974. Aspects of water quality and the toxicity of copper to rainbow trout. *Water Research*. 8(10): 797-803

Brown J.A., P.H. Johansen, P.W. Colgan, R.A. Mathers. 1985. Changes in the predator-avoidance behaviour of juvenile guppies (*Poecilia reticulata*) exposed to pentachlorophenol. Canadian Journal of Zoology 63:2001-2005.

Buchwalter, D.B., D.J. Cain, W.H. Clements, and S.N. Luoma. 2007. Using biodynamic models to reconcile differences between laboratory toxicity tests and field biomonitoring with aquatic insects. *Environmental Science and Technology*. 41(13): 4821-4828. *http://dx.doi.org/10.1021/es070464y*

Buck, W.B., R.D. Radeleff, J.B. Jackson. 1959. Oral toxicity studies with hepptachlor and heptachlor epoxide in young calves. J. Entomo. 52:1127-1129.

Budy, P., C. Luecke, and W.A. Wurtsbaugh. 1998. Adding nutrients to enhance the growth of endangered sockeye salmon: Trophic transfer in an oligotrophic lake. *Transactions of the American Fisheries Society*. 127(1): 19-34

Buhl, K.J., and S.J. Hamilton. 1990. Comparative toxicity of inorganic contaminants released by placer mining to early life stages of salmonids. Ecotoxicology and Environmental Safety 20:325-342.

Buhl, K.J., and S.J. Hamilton. 1991. Relative sensitivity of early life stages of Arctic grayling, coho salmon and rainbow trout to nine inorganics. Ecotoxicology and Environmental Safety 22:184-197.

Buhler, D.R., and W. E. Shanks. 1972. Influence of body weight on chronic oral DDT toxicity in coho salmon. J. Fish. Res. Board Can. 27:347.

Buhler, D.R., M.E. Rasmusson, and W.E. Shanks. 1969. Chronic oral DDT toxicity in juvenile coho and Chinook salmon. Toxicol. Appl. Pharmacol. 14:535

Burdick, G.E., E.J. Harris, H.J. Dean, T.M. Walker, J. Skea, and D. Colby. 1964. The accumulation of DDT in lake trout and the effect on reproduction. Trans. Am. Fish. Soc. 93:127.

Burk, R.F. 2002. Selenium, an antioxidant nutrient. *Nutrition in clinical care*. 5(2): 75-79. *http://dx.doi.org/10.1046/j.1523-5408.2002.00006.x*

Bury, N.R., J.C. McGeer, and C.M. Wood. 1999*a*. Effects of altering freshwater chemistry on physiological responses of rainbow trout to silver exposure. Environ. Toxicol. Chem. 18:49-55.

Bury, N.R., F. Galvez, and C.M. Wood. 1999*b*. Effects of chloride, calcium, and dissolved organic carbon on silver toxicity: comparison between rainbow trout and fathead minnows. Environ. Toxicol. Chem. 18:56-62.

Busack, C. 1991. Genetic evaluation of the Lyons Ferry Hatchery stock and wild Snake River fall Chinook. Report submitted to the ESA Administrative Record fro fall Chinook salmon, May 1991, 59 pages. Available Washington Department of Fisheries, 115 General Administration Bldg., Olympia, WA 98504.

Cairns, J.J. 1986. The myth of the most sensitive species. *BioScience*. 36:670-672

Calamari, D., G.F. Gaggino, and G. Pacchetti. 1982. Toxicokinetics of low levels of Cd, Cr, and Ni and their mixture in long-term treatment of *Salmo gairdneri* Rich. Chemosphere 11:59-70.

Call, D.J., and six others. 1999. Silver toxicity to *Chironomus tentans* in two freshwater sediments. Environ. Toxicol. Chem. 18:30-39.

Camusso M.L., and R. Balestrini. 1995. Bioconcentration of Trace Metals in Rainbow Trout: A Field Study. Ecotoxicol. Environ. Saf. 31:133-141.

Canfield, T.J., N.E. Kemble, W.G. Brumbaugh, F.J. Dwyer, C.G. Ingersoll, and J.F. Fairchild. 1994. Use of benthic invertebrate community structure and the sediment quality triad to evaluate the metals-contaminated sediment in the upper Clark Fork River, Montana. *Environmental Toxicology and Chemistry*. 13(12): 1999-2012

Canivet, V., P. Chambon, and J. Gilbert. 2001. Toxicity and bioaccumulation of arsenic and chromium in epigean and hypogean freshwater macroinvertebrates. Arch. Environ. Contam. Toxicol. 40: 345-354.

Cardwell, R.D., D.G. Foreman, T.R. Payne, D.J. Wilbur. 1977. Acute and chronic toxicity of chlordane to fish and invertebrates. EPA 600/3-77-019. EPA Ecol. Res. Series, U.S. Environ. Prot. Agency. Duluth, Minnesota.

Cardwell, R.D., D.K. DeForest, K.V. Brix, and W.J. Adams. 2013. Do Cd, Cu, Ni, Pb, and Zn Biomagnify in Aquatic Ecosystems? *Reviews of Environmental Contamination and Toxicology*. 226: 101-122. http://dx.doi.org/10.1007/978-1-4614-6898-1_4

Carlisle, D.M. and W.H. Clements. 1999. Sensitivity and variability of metrics used in biological assessments of running waters. *Environmental Toxicology and Chemistry*. 18(2): 285-291

Carlson, A.R., W.A. Brungs, G.A. Chapman, and D.J. Hansen. 1984. Guidelines for deriving numerical aquatic site-specific water quality criteria by modifying national criteria. U.S. Environmental Protection Agency, EPA-600/3-84-099 PB85-121101, Washington, DC.

Carney, M., J. Lipton, S.F. Brinkman, D. Cacela, S. Humphries, P. Craig, and J.T. Oris. 2008. Responses to copper challenges by brown trout fry acclimated, de-acclimated, and naïve to waterborne copper. Society of Environmental Toxicology and Chemistry, SETAC 29th Annual Meeting, Tampa, FL.

Carr, R.L., T.A. Couch, J. Liu, J.R. Coats, and J.E. Chambers. 1999. The interaction of chlorinated alicyclic insecticides with brain GABA(A) receptors in channel catfish (Ictalurus punctatus). J Toxicol Environ Health 56:543-553.

Carroll, J.J., S.J. Ellis, and W.S. Oliver. 1979. Influences of hardness constituents on the acute toxicity of cadmium to brook trout (*Salvelinus fontinalis*). *Bulletin of Environmental Contamination and Toxicology*. 22: 575–581

Casey, R. 2007. Results of aquatic studies in the McLeod and upper Smoky River systems. Alberta Environment, Pub. No: T/785. 72 pp. *http://environment.gov.ab.ca/info/library/7743.pdf* [Accessed 27 June, 2009].

Castro, M.S., E.N. McLaughlin, S.L. Davis, and R.P. Morgan, II. 2002. Total Mercury Concentrations in Lakes and Fish of Western Maryland, USA *Archives of Environmental Contamination and Toxicology*. 42(4): 454-462

CCME (Canadian Council of Ministers of the Environment). 2001. Canadian sediment quality guidelines for protection of aquatic life: summary tables. Publication 1299. Winnipeg.

CEC. 2004a. [Review of the proposal to remove the hardness "cap" of 25 mg/L for calculation of standards of hardness-based metals criteria in Idaho]. Prepared for the Hecla Mining Company, Challis, Idaho and the Thompson Creek Mining, Challis, Idaho. Report by Chadwick Ecological Consultants, Inc., Littleton, Colorado. 24 pp.

CEC. 2004b. Selenium bioaccumulation monitoring in Thompson Creek, Custer County, Idaho 2003. Prepared for Thompson Creek Mining, Challis, Idaho. Report by Chadwick Ecological Consultants, Inc., Littleton, Colorado. 32 pp.

CEC. 2005. Selenium bioaccumulation monitoring in Thompson Creek, Custer County, Idaho 2004. Prepared for Thompson Creek Mining, Challis, Idaho. Report by Chadwick Ecological Consultants, Inc., Littleton, Colorado. 30 pp.

Celius, T., and B.T. Walther. 1998. Differential sensitivity of zonagenesis and vitellogenesis in Atlantic salmon (Salmo salar L) to DDT pesticides. J. Exp. Zool. 281:346-353.

CH2M Hill. 2002. Boise River water-effect ratio project [for copper and lead]. Prepared for the City of Boise, Boise, ID, CH2M Hill, Boise, ID 44 pp. *http://www.deq.state.id.us/water/data_reports/surface_water/monitoring/site_specific_criteria.cfm*, accessed December 2004 [Accessed (*http://www.deq.state.id.us/water/data_reports/surface_water/monitoring/site_specific_criteria.cfm* December 2004)].

Chadwick, G.G., and D.L. Shumway. 1969. Effects of Dieldrin on the Growth and Development of Steelhead Trout. In: The Biological Impact of Pesticides in the Environment. Environ. Health Sci. Ser. No. 1, Oregon St. Univ. p.90-96.

Chakoumakos, C., R.C. Russo, and R.V. Thruston. 1979. Toxicity of copper to cutthroat trout (*Salmo clarki*) under different conditions of alkalinity, pH, and hardness. Environ. Sci. Tech. 13: 213-219.

Chambers, J.E., and J.D. Yarbrough. 1976. Xenobiotic Biotransformation Systems in Fish. Comp. Biochem. Physiol. 55C:77-84.

Chapman, G.A. 1975. Toxicity of copper, cadmium, and zinc to Pacific Northwest salmonids. U.S. Environmental Protection Agency, Western Fish Toxicology Station, National Water Quality Laboratory, Corvallis, OR. 27 pp.

Chapman, G.A. 1978a. Effects of continuous zinc exposure on sockeye salmon during adult-to-smolt freshwater residency. *Transactions of the American Fisheries Society*. 107(6): 828–836.

Chapman, G.A. 1978b. Toxicities of cadmium, copper, and zinc to four juvenile stages of chinook salmon and steelhead. *Transactions of the American Fisheries Society*. 107(6): 841-847.

Chapman, G.A. 1982. [Chinook salmon early life stage tests with cadmium, copper, and zinc]. U.S. Environmental Protection Agency, Environmental Research Laboratory, Letter of December 6, 1982 to Charles Stephan, U.S. EPA Environmental Research Laboratory, Duluth, Corvallis, Oregon

Chapman, G.A. 1983. Do organisms in laboratory toxicity tests respond like organisms in nature? Pages 315-327 *in* W. Bishop, R. Cardwell, and B. Heidolph, editors. *Aquatic Toxicology and Hazard Assessment: Sixth Symposium (STP 802)*, volume STP 802. American Society for Testing and Materials (ASTM), Philadelphia.

Chapman, G.A. 1985. Acclimation as a factor influencing metal criteria. Pages 119–136 *in* R. C. Bahner, and D. J. Hansen, editors. *Aquatic Toxicology and Hazard Assessment: Eighth Symposium (STP 891-EB)*, volume STP 891. American Society for Testing and Materials (ASTM), Philadelphia.

Chapman, G.A. 1994. Unpublished data on effects of chronic copper exposures with steelhead - acclimation, life stage differences, and behavioral effects. Letter of July 5, 1994 to Chris Mebane, [NOAA liaison to] EPA Region X, Seattle, Wash. U.S. EPA Coastal Ecosystems Team, Newport, Oregon.

Chapman, P.M. 1996. Test of sediment effects concentrations: DDT and PCB in the Southern California Bight. Environ. Toxicol. Chem. 15:1197-1198.

Chapman, D.W. and T.C. Bjornn. 1969. Distribution of salmonids in streams with special reference to food and feeding. Pages 153-176 *in* T. G. Northcote, editor. *Symposium on salmon and trout in streams. H.R. MacMillan Lectures in fisheries.* University of British Columbia, Vancouver.

Chapman, W.M. and E. Quistorff. 1938. The food of certain fishes of north central Columbia River drainage, in particular, young Chinook salmon and steelhead trout. Washington Department of Fisheries, Biological Report 37A. 14 pp.

Chapman, G.A. and D.G. Stevens. 1978. Acutely lethal levels of cadmium, copper, and zinc to adult male coho salmon and steelhead. *Transactions of the American Fisheries Society*. 107(6): 837-840. *http://dx.doi.org/10.1577/1548-8659(1978)107<837:ALLOCC>2.0.CO;2*

Chapman, G.A., S. Ota, and F. Recht. 1980. Effects of water hardness on the toxicity of metals to *Daphnia magna*. U.S. EPA, Office of Research and Development, Corvallis, Oreg. 23 pp.

Chapman, P.M., W.J. Adams, M.L. Brooks, C.G. Delos, S.N. Luoma, W.A. Maher, H.M. Ohlendorf, T.S. Presser, and D.P. Shaw, editors. 2009. *Ecological assessment of selenium in the aquatic environment: Summary of a SETAC Pellston Workshop. Pensacola FL (USA)*. Society of Environmental Toxicology and Chemistry (SETAC), *http://www.setac.org/node/265*.

Chasar, L.C., B.C. Scudder, A.R. Stewart, A.H. Bell, and G.R. Aiken. 2009. Mercury cycling in stream ecosystems. 3. trophic dynamics and methylmercury bioaccumulation. *Environmental Science and Technology*. DOI: 10.1021/es8027567. *http://dx.doi.org/DOI:* 10.1021/es8027567

Chaturvedi, A.K. 1993. Toxicological evaluation of mixtures of ten widely used pesticides. J Appl Toxicol 13:183-188

Cheek, A.O., T.H. Brouwer, S. Carroll, S. Manning, J.A. McLachlan, and M. Brouwer. 2001. Experimental evaluation of vitellogenin as a predictive biomarker for reproductive disruption. Environ. Health Perspect. 109(7):681-690.

Chen, Y.-W., N. Belzile, and J.M. Gunn. 2001. Antagonistic effect of selenium on mercury assimilation by fish populations near Sudbury metal smelters? *Limnology and Oceanography*. 46(7): 1814-1818

Ciarelli, S., W.A.P.M.A. Vonck, and N.M. van Straalen. 1997. Reproducibility of spiked-sediment bioassays using the marine benthic amphipod, Corophium volutator. Mar. Environ. Res. 43:329-343.

Cichosz, T., C. Rabe, A. Davidson, and D. Saul. 2003. Draft Clearwater subbasin assessment. Available: *http://www.nwcouncil.org/fw/subbasinplanning/clearwater/plan/Default.htm*. (August 2011).

CIG (Climate Impacts Group). 2004. Overview of climate change impacts in the U.S. Pacific Northwest (July 29, 2004, updated August 17, 2004). Climate Impacts Group, University of Washington, Seattle, Washington. Available: *http://cses.washington.edu/db/pdf/cigoverview353.pdf*. (August 2011).

Clark, G.M. 2002. Occurrence and transport of cadmium, lead, and zinc in the Spokane River basin, Idaho and Washington, water years 1999-2001. U.S. Geological Survey, WRIR 02-4183, Boise, Idaho. 37 pp. *http://id.water.usgs.gov/PDF/wri024183/* [Accessed December 2011].

Clark, D.W., and D.M. Dutton. 1996. Quality of groundwater and surface water in intermontane basins of the northern Rocky Mountains, Montana and Idaho. USGS Hydrol. Invest. Atlas HA-738-C.

Clark, G.M. and T.R. Maret. 1998. Organochlorine Compounds and Trace Elements in Fish Tissue and Bed Sediments in the Lower Snake River Basin, Idaho and Oregon. U.S. Geological Survey, Water-Resources Investigation Report 98-4103 35 pp. *http://id.water.usgs.gov/PDF/wri984103/index.html* [Accessed August 2011].

Clayton, J.L. 1998. Alkalinity generation in snowmelt and rain runoff during short distance flow over rock. Rocky Mountain Research Station, U.S. Forest Service, Research Paper RMRS-RP-12, Boise, ID. 7 pp. *http://www.fs.fed.us/rm/pubs/rmrs_rp012.pdf*.

Clements, W.H., and P.M. Kiffney. 1994. Integrated laboratory and field approach for assessing impacts of heavy metals at the Arkansas River, Colorado. Environ. Toxicol. Chem. 13:397-404.

Clements, W.H. and P.M. Kiffney. 1996. Validation of whole effluent toxicity tests: integrated studies using field assessments, microcosms, and mesocosms. Pages 229-244 *in* D. L. Grothe, K. L. Dickson, and D. K. Reed-Judkins, editors. *Whole effluent toxicity testing: an evaluation of methods and prediction of receiving system impacts*. SETAC Press, Pensacola, FL.

Clements, W.H. and D.E. Rees. 1997. Effects of heavy metals on prey abundance, feeding habits, and metal uptake of brown trout in the Arkansas River, Colorado. *Transactions of the American Fisheries Society*. 126(5): 774–785. *http://dx.doi.org/10.1577/1548-8659(1997)126<0774:EOHMOP>2.3.CO;2*

Clements, W.H., J.L. Farris, D.S. Cherry, and J. Cairns, Jr. 1989. The influence of water quality on macroinvertebrate community responses to copper in outdoor experimental streams. *Aquatic Toxicology*. 14(3): 249-262

Cleveland L., D.R. Buckler, F.L. Mayer, and D.R. Bransom. (1982). Toxicity of three preparations of pentachlorophenol to fathead minnows B A comparative study. Environmental Toxicology and Chemistry 1:205-212.

Cockell, K.A., J.W. Hilton, and W.J. Bettger. 1991. Chronic toxicity of dietary disodium arsenate heptahydrate to juvenile rainbow trout (*Oncorhynchus mykiss*) *Archives of Environmental Contamination and Toxicology*. 21(4): 518-527

Cockell, K.A., J.W. Hilton, and W.J. Bettger. 1992. Hepatobiliary and Hematological Effects of Dietary Disodium Arsenate Heptahydrate in Juvenile Rainbow Trout. Comp. Biochem. Physiol. 103C:453-458.

COE (United States Army Corps of Engineers). 1998. Dredged Material Evaluation Framework. Lower Columbia River Management Area. Prepared by the US Army Corps of Engineers, Northwest Division, EPA Region 10, the Oregon Department of Natural Resources, and the Oregon Department of Environmental Quality. November 1998.

Conley, J.M., D.H. Funk, and D.B. Buchwalter. 2009. Selenium bioaccumulation and maternal transfer in the mayfly *Centroptilum triangulifer* in a life-cycle, periphyton-biofilm trophic assay. *Environmental Science and Technology*. 43(20): 7952–7957

Connor, W.P., H.L. Burge, and R. Waitt. 2002. Juvenile life history of wild fall Chinook salmon in the Snake and Clearwater Rivers. *North American Journal of Fisheries Management*. 22(3): 703–712

Coutant, C.C. 1999. Perspectives on temperature in the Pacific Northwest's fresh waters. Oak Ridge National Laboratory, Report ORNL/TM-1999/44, Oak Ridge, Tennessee

Covich, A.P., M.A. Palmer, and T.A. Crowl. 1999. The role of benthic invertebrate species in freshwater ecosystems. *BioScience*. 49(2): 119-127

Crane, M. and M.C. Newman. 2000. What level of effect is a no observed effect? *Environmental Toxicology and Chemistry*. 19(2): 516-519

Cusimano, R.F., D.F. Brakke, and G.A. Chapman. 1986. Effects of pH on the toxicities of cadmium, copper, and zinc to steelhead trout (*Salmo gairdneri*). *Canadian Journal of Fisheries and Aquatic Sciences*. 43(8): 1497-1503.

Dabrowski, K.R. 1976. The effect of Arsenic on embrional development of rainbow trout (*Salmo gairdneri*, RICH.). Water Research 10:793-796.

Dalela, R.C.; S.R. Verma,; M.C. Bhatnagar. 1978. Acta Hydrochim. Hydrobiol. 6(1), 15-25.

Daniels, R.E., and J.D. Allan. 1981. Life Table Evaluation of Chronic Exposure to a Pesticide. Can. J. Fish. Aquat. Sci. 38:485-494.

Dauble, D.D., G.W. Patton, T.M. Poston, and R.E. Peterson. 2001. Evaluation of the effects of chromium to fall Chinook salmon in the Hanford Reach of the Columbia River: integration of recent toxicity test results. Pacific Northwest National Laboratory, PNNL-14008, Richland, Washington. 65 pp.

Dauble D. D., T. P. Hanrahan, D. R. Geist, and M. J. Parsley. 2003. Impacts of the Columbia River hydroelectric system on mainstem habitats of fall Chinook salmon. North American Journal of Fisheries Management 23:641-659.

Davies, P.H. 1982. Water Quality Technical Report for the Blackbird Project. *in Final Environmental Impact Statement, Blackbird Cobalt-Copper Project, Lemhi County, Idaho*. U.S. Forest Service, Salmon National Forest, Region 4, Salmon, Idaho.

Davies, P.H. and S.F. Brinkman. 1994. Cadmium toxicity to rainbow trout: bioavailability and kinetics in waters of high and low complexing capacities. Pages II-33 - II-59 (Appendix II) *in* P. H. Davies, editor. *Water Pollution Studies, Federal Aid in Fish and Wildlife Restoration, Project #33*. Colorado Division of Wildlife, Fort Collins, Colo

Davies, P.E. and L.S.J. Cooke. 1993. Catastrophic macroinvertebrate drift and sublethal effects on brown trout, *Salmo trutta*, caused by cypermethrin spraying on a Tasmanian stream. *Aquatic Toxicology*. 27(3-4): 201-224.

Davies, P.H., J.P. Goettl Jr., J.R. Sinley, and N.F. Smith. 1976. Acute and chronic toxicity of lead to rainbow trout *Salmo gairdneri*, in hard and soft water. Water Research 10:199-206.

Davies, P.H., J.P. Goettl Jr., and J.R. Sinley. 1978. Toxicity of silver to rainbow trout (*Salmo gairdneri*). Water Research 12:113-117.

Davies, P.H., Gorman, W.C., Carlson, C.A. and S.F. Brinkman. 1993. Effect of hardness on bioavailability and toxicity of cadmium to rainbow trout. Chemical Speciation and Bioavailability 5(2):67-77.

Davy, F.B., H. Kleerekoper, and J.H. Matis. 1973. Effects of exposure to sublethal DDT on the exploratory behavior of goldfish (Carassius auratus). Water Resour. Res. 9:900-905.

Dayal H., W. Gupta, N. Trieff, D. Maierson, and D. Reich. 1995. Symptom clusters in a community with chronic exposure to chemicals in two superfund sites. Arch Environ Health 50:108-11.

De Boeck, G., K. van der Ven, J. Hattink, and R. Blust. 2006. Swimming performance and energy metabolism of rainbow trout, common carp and gibel carp respond differently to sublethal copper exposure. *Aquatic Toxicology*. 80(1): 92-100

de Geus H.J., H. Besselink, A. Brouwer, J. Klungsoyr, B. McHugh, E. Nixon, G. Rimkus, P.G. Wester, J. de Boer. 1999. Environmental occurrence, analysis, and toxicology of toxaphene compounds. Environ Health Perspect 107 Suppl 1:115-44

de Rosemond, S.C., K. Liber, and A. Rosaasen. 2005. Relationship between embryo selenium concentration and early life stage development in white sucker (*Catostomus commersoni*) from a northern Canadian lake. *Bulletin of Environmental Contamination and Toxicology*. 74(6): 1134 - 1142

De Schamphelaere, K.A.C. and C.R. Janssen. 2004. Bioavailability and chronic toxicity of zinc to juvenile rainbow trout (*Oncorhynchus mykiss*): comparison with other fish species and development of a Biotic Ligand Model. *Environmental Science and Technology*. 38(23): 6201 -6209. *http://dx.doi.org/10.1021/es049720m*

de Vlaming, V. and T.J. Norberg-King. 1999. A review of single species toxicity tests: Are the tests reliable predictors of aquatic ecosystem community response? U.S. Environmental Protection Agency, EPA 600/R/97/114, Duluth, MN.

de Vlaming, V., V. Connor, C. DiGiorgio, H.C. Bailey, L.A. Deanovic, and D.E. Hinton. 2000. Application of whole effluent toxicity test procedures to ambient water quality assessment *Environmental Toxicology and Chemistry*. 19(1): 42–62

deBruyn, A.M.H. and P.M. Chapman. 2007. Selenium toxicity to invertebrates: will proposed thresholds for toxicity to fish and birds also protect their prey. *Environmental Science and Technology*. 41(5): 1766 -1770, 2007. 10.1021/es062253j S0013-936X(06)02253-X

deBruyn, A.M.H., A. Hodaly, and P.M. Chapman. 2008. Part I. Selection of tissue types for the development of a meaningful selenium tissue threshold in fish. Pages 8-49 *in Selenium Tissue Thresholds: Tissue Selection Criteria, Threshold Development Endpoints, and Potential to Predict Population or Community Effects in the Field*. Report by GEI Consultants, Golder Associates, Parametrix, and University of Saskatchewan Toxicology Centre to the North America Metals Council – Selenium Working Group, Washington, D.C., *http://www.namc.org/selenium.html*.

DeFoe, D.L., G.D. Veith, and R.W. Carlson. 1978. Effects of Aroclor 1248 and 1260 on the fathead minnow (*Pimephales promelas*). Jour. Fish Res. Bd. Can. 35:997-1002.

DeForest, D.K. 2008. Part II: Review of selenium tissue thresholds for fish: evaluation of the appropriate endpoint, life stage, and effect level and recommendation for a tissue-based criterion. Pages 50-75 *in Selenium Tissue Thresholds: Tissue Selection Criteria, Threshold Development Endpoints, and Potential to Predict Population or Community Effects in the Field*. Report by GEI Consultants, Golder Associates, Parametrix, and University of Saskatchewan Toxicology Centre to the North America Metals Council – Selenium Working Group, Washington, D.C., *http://www.namc.org/selenium.html*.

DeForest, D.K., K.V. Brix, and W.J. Adams. 1999. Critical review of proposed residue-based selenium toxicity thresholds for freshwater fish. *Human and Ecological Risk Assessment*. 5(6): 1187-1228

DeForest, D.K., K.V. Brix, and W.J. Adams. 2007. Assessing metal bioaccumulation in aquatic environments: The inverse relationship between bioaccumulation factors, trophic transfer factors and exposure concentration. *Aquatic Toxicology*. 84(2): 236-246

Deichmann, W.B., W. MacDonald, E. Blum, M. Bevilacqua, J. Radomski, M. Keplinger, and M. Balkus. 1970. Tumorigenicity of aldrin, dieldrin and endrin in the albino rat. *IMS, Industrial medicine and surgery*. 39(10): 426-434

Deleebeeck, N.M.E., B.T.A. Muyssen, F. De Laender, C.R. Janssen, and K.A.C. De Schamphelaere. 2007. Comparison of nickel toxicity to cladocerans in soft versus hard surface waters. *Aquatic Toxicology*. 84(2): 223-235.

Delorme, P.D. 1998. The Effects of Toxaphene, Chlordane and 2,3,4,7,8 pentachlorodibenzofuran On Lake Trout and White Sucker in An Ecosystem Experiment and the Distribution and Effects of 2,3,4,7,8- Pentachlorodibenzofuran On White Suckers and Broodstock Rainbow Trout in Laboratory Experiments. Dissertation Abstracts International Part B: Science and Engineering [Diss. Abst. Int. Pt. B - Sci. & Eng.], University Microfilms International, May 1998, vol. 58, no. 11, p. 5756

Delos, C.G. 2008. Modeling framework applied to establishing an allowable frequency for exceeding aquatic life criteria. U.S. Environmental Protection Agency, Office of Water, 4304, Washington, D.C. 158 pp.

DeMaster, D., R. Angliss, J. Cochrane, P. Mace, R. Merrick, M. Miller, S. Rumsey, B.E. Taylor, G.G. Thompson, and R.S. Waples. 2004. Recommendations to NOAA Fisheries: ESA Listing Criteria by the Quantitative Working Group. National Marine Fisheries Service, NOAA, NOAA Technical Memorandum NMFS-F/SPO-67. 85 pp. *http://spo.nmfs.noaa.gov/tm/tm67.pdf*.

Depew, D.C., N. Basu, N.M. Burgess, L.M. Campbell, E.W. Devlin, P.E. Drevnick, C.R. Hammerschmidt, C.A. Murphy, M.B. Sandheinrich, and J.G. Wiener. 2012. Toxicity of dietary methylmercury to fish: Derivation of ecologically meaningful threshold concentrations. *Environmental Toxicology and Chemistry*. 31(7): 1536-1547. *http://dx.doi.org/10.1002/etc.1859*

Detenbeck, N.E., P.W. DeVore, G.J. Niemi, and A. Lima. 1992. Recovery of temperate-stream fish communities from disturbance - a review of case studies and synthesis of theory. *Environmental Management*. 16(1): 33-53. *http://dx.doi.org/10.1007/BF02393907*

Dethloff, G.M., and H.C. Bailey. 1998. Effects of copper on immune system parameters of rainbow trout (*Oncorhynchus mykiss*). Environ. Toxicol. Chem. 17:1807-1814.

Devi, A.P., D.M.R. Rato, K.S. Tilak, and A.S. Murty. 1981. Relative toxicity of the technical grade material, isomers, and formulations of endosulfan to the fish Channa punctata. *Bulletin of Environmental Contamination and Toxicology*. 27(1): 239-243. http://dx.doi.org/10.1007/BF01611015

Devlin, E. W., and N. K. Mottet. 1992. Embryotoxic action of methyl mercury on coho salmon embrys. Bull. Environ. Contam. Toxicol. 49:449-454.

Di Toro, D. M., C. S. Zarba, D. J. Hansen, W. J. Berry, R. C. Swartz, C. E. Cowan, S. P. Pavlou, H. E. Allen, N. A. Thomas, and P. R. Paquin. 1991. Technical basis for establishing sediment quality criteria for nonionic organic chemicals using equilibrium partitioning. Environmental Toxicology and Chemistry 10:1541-1583.

Di Toro, D.M., H.E. Allen, H.L. Bergman, J.S. Meyer, P.R. Paquin, and R.C. Santore. 2001. Biotic ligand model of the acute toxicity of metals. 1. Technical basis. *Environmental Toxicology and Chemistry*. 20(10): 2383-2396.

Diamond, J. and C. Daley. 2000. What is the relationship between whole effluent toxicity and instream biological condition? Environmental Toxicology and Chemistry. 19(1): 158-168

Diamond, J.M., E.L. Winchester, D.G. Mackler, and D. Gruber. 1992. Use of the mayfly *Stenonema modestum* (Heptageniidae) in subacute toxicity assessments. *Environmental Toxicology and Chemistry*. 11(3): 415-425

Diamond, J.M., S.J. Klaine, and J.B. Butcher. 2006. Implications of pulsed chemical exposures for aquatic life criteria and wastewater permit limits. *Environmental Science and Technology*. 40(16): 5132-5138

Dill, P.A., and R. C. Saunders. 1974. Retarded behavioral development and impaired balance in Atlantic salmon (Salmo salar) alevins hatched from gastrulae exposed to DDT. Jour. Fish. Res. Board Can. 31:1936.

Dillon, F.S. and C.A. Mebane. 2002. Development of site-specific water quality criteria for the South Fork Coeur d'Alene River, Idaho: application of site-specific water quality criteria developed in headwater reaches to downstream waters. Prepared for and in conjunction with the Idaho Department of Environmental Quality. Windward Environmental, Seattle, WA. 95 pp. *http://www.deq.state.id.us/water/data_reports/surface_water/monitoring/site_specific_criteria.cfm*.

Dillon, T., N. Beckvar, and J. Kern. 2010. Residue-based mercury dose–response in fish: An analysis using lethality-equivalent test endpoints. *Environmental Toxicology and Chemistry*. 29(11): 2559-2565. *http://dx.doi.org/10.1002/etc.314*

Dixon, D.G. and J.W. Hilton. 1985. Effects of available dietary carbohydrate and water temperature on the chronic toxicity of waterborne copper to rainbow trout (*Salmo gairdneri*). *Canadian Journal of Fisheries and Aquatic Sciences*. 42(5): 1007-1013

Dixon, D.G. and G. Leduc. 1981. Chronic cyanide poisoning of rainbow trout and its effects on growth, respiration, and liver histopathology. *Archives of Environmental Contamination and Toxicology*. 10(1): 117-131

Dixon, D. G., and J. B. Sprague. 1981. Acclimation-induced changes in toxicity of arsenic and cyanide to rainbow trout, *Salmo gairdneri* Richardson. Journal of Fish Biology 18:579-589.

Dominguez S.E., and G.A. Chapman. 1984. Effect of pentachlorophenol on the growth and mortality of embryonic and juvenile steelhead trout. Archives of Environmental Contamination and Toxicology 13:739-743.

Donohoe, R.M. and L. R. Curtis. 1996. Estrogenic activity of chlordecone, o,p'-DDT and o,p' DDE in juvenile rainbow trout: Induction of vitellogenesis and interaction with hepatic estrogen binding sites. Aquat. Toxicol 36:31-52.

Drevnick, P.E. and M.B. Sandheinrich. 2003. Effects of dietary methylmercury on reproductive endocrinology of fathead minnows. *Environmental Science and Technology*. 37(19): 4390-4396

Duboudin, C., P. Ciffroy, and H. Magaud. 2004. Effects of data manipulation and statistical methods on species sensitivity distributions. *Environmental Toxicology and Chemistry*. 23(2): 489-499

Dunier, M., A.K. Siwicki, J. Scholtens, S.D. Molin, C. Vergnet, and M. Studnicka. 1994. Effects of Lindane Exposure on Rainbow Trout (Oncorhynchus mykiss) Immunity: III. Effect on Nonspecific Immunity and B Lymphocyte Functions. *Ecotoxicology and environmental safety*. 27(3): 324-334

Dunier, M., C. Vergnet, A.K. Siwicki, and V. Verlhac. 1995. Effect of Lindane Exposure on Rainbow Trout (Oncorhynchus mykiss) Immunity IV. Prevention of Nonspecific and Specific Immunosuppression by Dietary Vitamin C (Ascorbate-2-polyphosphate). *Ecotoxicology and environmental safety*. 30(3): 259-268

Dussault, È.B., R.C. Playle, D.G. Dixon, and R.S. McKinley. 2008. Effects of soft-water acclimation on the physiology, swimming performance, and cardiac parameters of the rainbow trout, *Oncorhynchus mykiss*. *Fish Physiology and Biochemistry*. 34(4): 313-322. *http://dx.doi.org/doi:* 10.1007/s10695-007-9190-1

Dwyer, F.J., D.K. Hardesty, C.E. Henke, C.G. Ingersoll, D.W. Whites, T. Augspurger, T.J. Canfield, D.R. Mount, and F.L. Mayer. 2005a. Assessing contaminant sensitivity of endangered and threatened fishes: III. Effluent toxicity tests. *Archives of Environmental Contamination and Toxicology*. 48(2): 174-183

Dwyer, F.J., F.L. Mayer, L.C. Sappington, D.R. Buckler, C.M. Bridges, I.E. Greer, D.K. Hardesty, C.E. Henke, C.G. Ingersoll, J.L. Kunz, D.W. Whites, D.R. Mount, K. Hattala, and G.N. Neuderfer. 2005b. Assessing contaminant sensitivity of endangered and threatened fishes: I. Acute toxicity of five chemicals. *Archives of Environmental Contamination and Toxicology*. 48(2): 143-154. *http://dx.doi.org/10.1007/s00244-003-3038-1*

Eagles-Smith, C.A., G. Herring, B.L. Johnson, and R. Graw. 2013. Mercury bioaccumulation in fishes from subalpine lakes of the Wallowa-Whitman National Forest, northeastern Oregon and western Idaho. U.S. Geological Survey Open-File Report 2013-1148. 47 pp. *http://pubs.usgs.gov/of/2013/1148/* [Accessed 2 Jul 2013].

Earnest, R.D., and P.E. Benville 1971. Correlation of DDT and lipid levels for certain San Francisco Bay fish. Pestic. Monitor. Jour. 5:235.

EcoMetrix. 2005. Biomonitoring study, Panther Creek watershed, September 2004. Prepared by EcoMetrix Incorporated for the Blackbird Mine Site Group, Salmon, Idaho, Brampton, Ontario. 427 pp.

EcoMetrix. 2006. Biomonitoring study, Panther Creek watershed, September 2005. Prepared by EcoMetrix Incorporated, Brampton, Ontario, for the Blackbird Mine Site Group, Salmon, Idaho. 105 pp.

EcoMetrix. 2007. Biomonitoring study, Panther Creek watershed, September 2006. Prepared by EcoMetrix Incorporated, Mississauga, Ontario, for the Blackbird Mine Site Group, Salmon, Idaho. 512 pp.

EcoMetrix. 2008. Biomonitoring study, Panther Creek watershed, September 2007. Prepared by EcoMetrix Incorporated, Brampton, Ontario, for the Blackbird Mine Site Group, Salmon, Idaho. 587 pp.

EcoMetrix. 2009. Biomonitoring study, Panther Creek watershed, September 2008. Prepared by EcoMetrix Incorporated, Mississauga, Ontario, for the Blackbird Mine Site Group, Salmon, Idaho. 595 pp.

EcoMetrix. 2010. Biomonitoring study, Panther Creek watershed, September 2009. Prepared by EcoMetrix Incorporated, Brampton, Ontario, for the Blackbird Mine Site Group, Salmon, Idaho. 603 pp.

EcoMetrix. 2011. Biomonitoring study, Panther Creek watershed, September 2010. Prepared by EcoMetrix Incorporated, Brampton, Ontario, for the Blackbird Mine Site Group, Salmon, Idaho. 151 pp.

Ecovista. 2004a. “Clearwater Subbasin Assessment”. In Intermountain Subbasin Plan prepared for the Northwest Power and Conservation Council. Portland, Oregon, May 2004. http://www.nwcouncil.org/fw/subbasinplanning/admin/level2/intermtn/plan/

Ecovista. 2004b. “Salmon Subbasin Assessment”. In Intermountain Subbasin Plan, prepared for the Northwest Power and Conservation Council. Portland, Oregon, May 2004. http://www.nwcouncil.org/fw/subbasinplanning/admin/level2/intermtn/plan/

EIFAC (European Inland Fisheries Advisory Commission). 1983. Water quality criteria for European freshwater fish: Report on chromium and freshwater fish. Technical Paper No. 43. Food and Agriculture Organization, Rome, Italy.

EIFAC. 1984. Water quality criteria for European freshwater fish: Report on nickel and freshwater fish. Technical Paper No. 45. Food and Agriculture Organization, Rome, Italy.

Eisler, R. 1970. Acute toxicities of organochlorine and organophosphorous insecticides to estuarine fishes. U.S. Dept. Inter. Bur. Sport fish. Wildl. Tech. Pap 46.

Eisler, R. 1986. Polychlorinated biphenyl hazards to to fish, wildlife, and invertebrates: A synoptic review. U. S. Geological Survey, Biological Science Report 85(1.7). Contaminant Hazard Reviews, April 1986. Report No. 7.

Eisler, R. 1988a. Arsenic hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Fish and Wildlife Service, Biological Report 85(1.12).

Eisler, R. 1988b. Lead hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Fish and Wildlife Service, Biological Report 85(1.14).
Eisler R. 1989. Pentachlorophenol Hazards to Fish, Wildlife and Invertebrates: A synoptic review. Biological Report 85 (1.17). Contaminant Hazard Reviews Report No. 17. U.S. Department of the Interior, Fish and Wildlife Service. Laurel, Maryland, 72 pp.
Eisler, R. 1990. Chlordane hazards to fish, wildlife, and invertebrates: a synoptic review. U. S. Fish and Wildlife Service Biological Report 85 (1.21). 49 pp.
Eisler, R. 1991. Cyanide hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 85(1.23). Fish and Wildlife Service Contaminant Hazard Review.
Eisler, R. 1993. Zinc hazards to fish, wildlife, and invertebrates: A synoptic review. U. S. Fish and Wildlife Service, Biological Report 10, Contaminant Hazard Reviews Report 26.
Eisler, R. 1996. Silver hazards to fish, wildlife, and invertebrates: A synoptic review. U. S. Dept. Interior National Biological Service, Biological/Contaminant Hazard Reviews Report 32.
Eisler, R. 2000. Toxaphene. In: Handbook of Chemical Risk Assessment. Health Hazards to Humans, Plants, and Animals. Lewis Pubs. Vol. 2. pp. 1459 B 1481.
Eisler, R. 1986. Chromium hazards to fish, wildlife, and invertebrates: A synoptic review. U. S. Fish and Wildlife Service Biological Report 85 (1.6). 60 pp.
Eisler, R. 1998a. Copper hazards to fish, wildlife, and invertebrates: A synoptic review. U. S. Geological Survey, Biological Science Report USGS/BRD/BSR--1998-0002. Contaminant Hazard Reviews Report 33.
Eisler, R. 1998b. Nickel hazards to fish, wildlife, and invertebrates: A synoptic review. U. S. Geological Survey, Biological Science Report USGS/BRD/BSR--1998-0001. Contaminant Hazard Reviews Report 34.
Eisler, R. 2000. Pentachlorophenol. in Handbook of Chemical Assessment, Health Hazards to Humans, Plants, and Animals, Volume 2, pp. 1193 B 1235.
Eisler, R. 2000. Polychlorinated Biphenyls. in Handbook of Chemical Assessment, Health Hazards to Humans, Plants, and Animals, Volume 2, pp. 1237 B 1341.
Environment Canada. 1997. Toxaphene. In: Toxic Substances Management Policy. Scientific Justification. En40-230/4-1997E.
EPA (U.S. Environmental Protection Agency). 1976. Toxicity of Four Pesticides to Water Fleas and Fathead Minnows. EPA-600/3-76-099.
EPA. 1980*a*. Ambient Water Quality Criteria for Aldrin/Dieldrin. EPA Report 440/5-80-019.
EPA. 1980*c*. Ambient Water Quality Criteria for Chlordane. EPA Report 440/5-80-027.
EPA. 1980*e*. Ambient Water Quality Criteria for Cyanides. EPA Report 440/5-80-037.
EPA. 1980*f*. Ambient Water Quality Criteria for DDT. EPA Report 440/5-80-038.
EPA. 1980*g*. Ambient Water Quality Criteria for Endosulfan. EPA Report 440/5-80-046.
EPA. 1980*h*. Ambient Water Quality Criteria for Endrin. 1980. EPA Report 440/5-80-04.
EPA. 1980*i*. Ambient Water Quality Criteria for Heptachlor. EPA Report 440/5-80-052.
EPA. 1980*j*. Ambient Water Quality Criteria for Mercury. EPA Report 440/5-80-058.
EPA. 1980*o* Ambient Water Quality Criteria for Silver. EPA Report 440/5-80-071.
EPA. 1980*q*. Ambient Water Quality Criteria for Hexachlorocyclohexane. Report 440/5-80-054.
EPA. 1984a. Ambient water quality criteria for cadmium. U.S. Environmental Protection Agency, EPA 440/5-84-032, Duluth, Minn. 133 pp. *http://epa.gov/waterscience/criteria/aqlife.html*.

EPA. 1984b. Ambient water quality criteria for lead. U.S. Environmental Protection Agency, EPA 440/5-84-027, Duluth, MN. 89 pp.
EPA. 1985. Ambient water quality criteria for copper - 1984. U.S. Environmental Protection Agency, EPA 440/5-84-031, Duluth, MN. 142 pp. *http://water.epa.gov/scitech/swguidance/standards/criteria/ambientwqc_index.cfm* [Accessed December 2012].
EPA. 1985a. Ambient Water Quality Criteria for Arsenic. EPA Report 440/5-84-033.
EPA. 1985d. Ambient Water Quality Criteria for Copper-1984. EPA Report 440/5-84-031.
EPA. 1985e. Ambient Water Quality Criteria for Cyanide - 1984. EPA Report 440/5-84-028.
EPA. 1985f. Ambient Water Quality Criteria for Lead - 1984. EPA Report 440/5-84-027.
EPA. 1985g. Ambient Water Quality Criteria for Mercury - 1984. EPA Report 440/5-84-026.
EPA. 1985h. Ambient Water Quality Criteria for Chromium - 1984. EPA Report 440/5-84-027.
EPA. 1986b. Ambient Aquatic Life Criteria for Pentachlorophenol. Report 440/5-86-009.
EPA. 1987a. Ambient water quality criteria for selenium - 1987. U.S. Environmental Protection Agency, EPA 440/5-87-0006, NTIS PB88-142237, Duluth, Minn. *http://epa.gov/waterscience/criteria/aqlife.html*.
EPA. 1987b. Ambient Aquatic Life Water Quality Criteria for Silver. EPA Report 440/5-87-011.
EPA. 1987c. Ambient Water Quality Criteria for Zinc. EPA Report 440/5-87-003.
EPA. 1989b. Health Advisory Summary: Chlordane. Office of Drinking Water, Washington, DC.
EPA. 1991. Technical support document for water quality-based toxics control. Office of Water, U.S. Environmental Protection Agency, EPA 505/2-90-001, Washington, D.C. 143 pp. *http://www.epa.gov/npdes/pubs/owm0264.pdf*.
EPA. 1992. National Toxics Rule. *Federal Register*. 57(246): 60848-60910
EPA. 1992a. National Study of Chemical Residues in Fish. Vol. II. EPA 823-R-92-0086.
EPA. 1992b. Handbook of RCRA Ground-Water Monitoring Constituents: Chemical and Physical Properties (Appendix IX to 40 CFR Part 254). Office of Solid Waste, Washington, DC.
EPA. 1993b. Integrated Risk Information System (IRIS) on Chlordane. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH. EPA (U. S. Environmental Protection Agency). 1994, Water Quality Standards Handbook: Second Edition. EPA-823-B-94-005a.
EPA. 1994. Water Quality Standards Handbook. U.S. Environmental Protection Agency, EPA-823-B-94-005a, Washington, D.C. *http://www.epa.gov/ost/standards/handbook/*.
EPA. 1996. 1995 updates: water quality criteria documents for the protection of aquatic life in ambient water. U.S. Environmental Protection Agency, EPA 820-B-96-001, Washington, D.C.
EPA. 1997b. Mercury Study Report to Congress, Volume VI [Final], An Ecological Assessment for Anthropogenic Mercury Emissions in the United States. EPA-425/R-97-008.
EPA. 1998. Report on the Peer Consultation Workshop on Selenium Aquatic Toxicity and Bioaccumulation. U.S. Environmental Protection Agency, EPA 822-R-98-007, Washington, DC. *http://nepis.epa.gov*.

EPA. 1999a. 1999 update of ambient water quality criteria for ammonia. U.S. Environmental Protection Agency, EPA/822/R-99-014, Washington, D.C. 147 pp.
EPA. 1999b. National recommended water quality criteria-correction. U.S. Environmental Protection Agency, EPA/822/Z-99-001, Washington, D.C.
EPA. 2000a. Biological Assessment of the Idaho Water Quality Standards for Numeric Water Quality Criteria for Toxic Pollutants (final 8-4-2000). U.S. Environmental Protection Agency, Seattle, WA. 208 pp.
EPA. 2000b. [Fact sheet to Reissue a Wastewater Discharge Permit to Thompson Creek Mining, Clayton, ID, NPDES Permit Number: ID-002540-2]. U.S. Environmental Protection Agency, Office of Water, Seattle, WA. 61 pp. *http://yosemite.epa.gov/r10/WATER.NSF/NPDES+Permits/Permits+Homepage* [Accessed 05 February 2008].
EPA. 2000c. An SAB Report: Review of the Biotic Ligand Model of the Acute Toxicity of Metals. U.S. Environmental Protection Agency, Washington, D.C. *http://www.epa.gov/sab/pdf/epec0006.pdf*.
EPA. 2001. ECOTOX User Guide: ECOTOXicology Database System. Version 2.0. http://www.epa.gov/ecotox.
EPA. 2001a. Streamlined water-effect ratio procedure for discharges of copper. U.S. Environmental Protection Agency, EPA-822-R-01-005, Washington, DC. 41 pp. *http://www.epa.gov/waterscience/criteria/copper/copper.pdf*.
EPA. 2001b. Water quality criterion for the protection of human health: methylmercury. U.S. Environmental Protection Agency, EPA-823-R-01-001, Washington, DC. 303 pp. *http://www.epa.gov/waterscience/criteria/methylmercury/*.
EPA. 2002a. Methods for measuring the acute toxicity of effluents and receiving waters to freshwater and marine organisms, 5th edition. U.S. Environmental Protection Agency, EPA-821-R-02-012, Cincinnati, OH. 275 pp. *http://www.epa.gov/waterscience/WET/*.
EPA. 2002b. National recommended water quality criteria: 2002. U.S. Environmental Protection Agency, EPA-822-R-02-047, Washington, DC. 36 pp. *http://epa.gov/waterscience/criteria/wqcriteria.html*.
EPA. 2002c. Short-term methods for estimating the chronic toxicity of effluents and receiving waters to freshwater organisms, 4th edition. U.S. Environmental Protection Agency, EPA-821-R-02-013, Cincinnati, OH. 350 pp. *http://www.epa.gov/waterscience/WET/*.
EPA. 2003. Record of Decision: Blackbird Mine Superfund Site, Lemhi County, Idaho. U.S. Environmental Protection Agency, Seattle, WA. 159 pp. *http://yosemite.epa.gov/R10/CLEANUP.NSF/sites/Blackbird*.
EPA. 2004. Draft Aquatic Life Water Quality Criteria for Selenium - 2004. U.S. Environmental Protection Agency, EPA-822-D-04-001, Washington, DC. 334 pp. *http://www.epa.gov/waterscience/criteria/aqlife.html*.
EPA. 2007a. Aquatic life ambient freshwater quality criteria - copper, 2007 revision. U.S. Environmental Protection Agency, EPA-822-R-07-001 (March 2, 2007), Washington, DC. 208 pp. *http://www.epa.gov/waterscience/criteria/copper/* [Accessed 30 March 2008].
EPA. 2007b. The Biotic Ligand Model: Technical support document for its application to the evaluation of water quality criteria for copper. U.S. Environmental Protection Agency, Office of Science and Technology, EPA 822-R-03-027, Washington, D.C. 72 pp. *http://www.epa.gov/waterscience/criteria/copper/2007/blm-tsd.pdf*.

EPA. 2008. Pentachlorophenol and its Use as a Wood Preservative. U.S. Environmental Protection Agency, Office of Pesticide Programs. *http://www.epa.gov/pesticides/factsheets/chemicals/pentachlorophenol_main.htm* , accessed 25March2014

EPA. 2010a. Biological evaluation of the Idaho water quality criteria for cadmium with revised hardness cap (September 2, 2010). U.S. Environmental Protection Agency, September 2, 2010, Seattle, WA. 194 pp.

EPA. 2010b. Guidance for implementing the January 2001 methylmercury water quality criteria. U.S. Environmental Protection Agency, EPA-823-R-10-001. 221 pp. *http://www.epa.gov/waterscience/criteria/methylmercury/*.

EPA. 2010c. National Pollutant Discharge Elimination System: Test of Significant Toxicity Implementation Document. U.S. Environmental Protection Agency, EPA 833-R-10-003, Washington, D.C. 73 pp. *http://www.epa.gov/npdes/permitbasics*.

EPA. 2011. Revised Mercury TMDL for Jordan Creek. U.S. Environmental Protection Agency, Portland, OR. 55 pp. *http://yosemite.epa.gov/r10/water.nsf/tmdls/jordancreek*.

EPA/USACOE. 1991. Evaluation of dredged material proposed for ocean disposal (testing manual). EPA-503/8-91/001, EPA Office of Marine and Estuarine Protection, Washington, DC.

Erickson, R.J. 2008. Toxicity Response Analysis Program, version 1.2. U.S. Environmental Protection Agency, National Health and Environmental Research Laboratory, Mid-Continent Ecological Division, Duluth, Minnesota. *http://www.epa.gov/med/prods_pubs.htm* [Accessed December 2010].

Erickson, R.J., D.A. Benoit, and V.R. Mattson. 1987. A prototype toxicity factors model for site specific copper water quality criteria (Revised September 5, 1996). U.S. Environmental Protection Agency, Environmental Research Laboratory, Duluth, Minnesota. 32 pp.

Erickson, R.J., D.A. Benoit, V.R. Mattson, H.P. Nelson, and E.N. Leonard. 1996. The effects of water chemistry on the toxicity of copper to fathead minnows. *Environmental Toxicology and Chemistry*. 15(2): 181-193.

Erickson, R.J., L.T. Brooke, M.D. Kahl, F. Vende Venter, S.L. Harting, T.P. Markee, and R.L. Spehar. 1998. Effects of laboratory test conditions on the toxicity of silver to aquatic organisms. *Environmental Toxicology and Chemistry*. 17(4): 572–578

Erickson, R.J., C.E. Stephan, and D.R. Mount. 1999. Appendix A2: Derivation of recommended acute copper TRVs [toxicity reference values] for salmonids. Pages 59-115 *in Clark Fork River Ecological Risk Assessment*. U.S. Environmental Protection Agency, Helena, MT, *http://www.epa.gov/region8/superfund/sites/mt/milltowncfr/millterap.html*, accessed December 2004, however by October 2008 the site was not found.

Erickson, R.J., D.R. Mount, T.L. Highland, J.R. Hockett, E.N. Leonard, V.R. Mattson, T.D. Dawson, and K.G. Lott. 2010. Effects of copper, cadmium, lead, and arsenic in a live diet on juvenile fish growth. *Canadian Journal of Fisheries and Aquatic Sciences*. 67(11): 1816-1826. *http://dx.doi.org/10.1139/F10-098*

Erickson, R.J., D.R. Mount, J.D. Fernandez, T.L. Highland, J.R. Hockett, D.J. Hoff, and C.T. Jenson. 2011. Arsenic Toxicity to Juvenile Fish: Effects of Exposure Route, Arsenic Speciation, and Fish Species [platform presentation]. *in* Abstracts, SETAC North America 32nd Annual Meeting, November 16, 2011, Boston, MA. Society of Environmental Toxicology and Chemistry (SETAC), *http://boston.setac.org/*.

Essig, D.A. 2010. Arsenic, mercury, and selenium in fish tissue and water from Idaho's major rivers: a statewide assessment. Idaho Department of Environmental Quality, Boise, ID. *http://www.deq.idaho.gov/water-quality/surface-water/mercury.aspx* [Accessed November 2011].

Essig, D.A. and M.A. Kosterman. 2008. Arsenic, mercury, and selenium in fish tissue from Idaho lakes and reservoirs: a statewide assessment. Idaho Department of Environmental Quality, Boise, ID. 80 pp. *http://www.deq.idaho.gov/water/data_reports/surface_water/monitoring/publications.cfm#standards*.

Evans, M.S., G.E. Noguchi, and C.P. Rice. 1991. The biomagnification of polychlorinated biphenyls, toxaphene and DDT compounds in a Lake Michigan offshore food web. Arch. Environ Contam. Toxicol. 20:87-93.

Everest, F. H. and D. W. Chapman. 1972. Habitat selection and spatial interaction by juvenile Chinook salmon and steelhead trout in two Idaho streams. Journal of the Fisheries Research Board of Canada 29(1):91-100.

Evermann, B. W. 1895. A preliminary report upon salmon investigations in Idaho in 1894. U.S. Fish Commission Bulletin. 15:253-284.

Fagella G., O. Brandt, and B. Finlayson. 1990. Standardized Testing Program B 1988 Progress Report, California Department of Fish and Game, Aquatic Toxicology Laboratory, Elk Grove, Calif.

Farag, A.M., C.J. Boese, D.F. Woodward, and H.L. Bergman. 1994. Physiological changes and tissue metal accumulation in rainbow trout exposed to foodborne and water-borne metals. Environ. Toxicol. Chem. 13:2021-2029.

Farag, A.M., D.F. Woodward, J.N. Goldstein, W.G. Brumbaugh, and J.S. Meyer. 1998. Concentrations of metals associated with mining waste in sediments, biofilm, benthic macroinvertebrates, and fish from the Coeur d'Alene River Basin, Idaho. *Archives of Environmental Contamination and Toxicology*. 34(2): 119-127. *http://dx.doi.org/10.1007/s002449900295*

Farag, A.M., D.F. Woodward, W.G. Brumbaugh, J.N. Goldstein, E. McConnell, C. Hogstrand, and F.T. Barrows. 1999. Dietary effects of metals-contaminated invertebrates from the Coeur d'Alene River, Idaho on cutthroat trout. *Transactions of the American Fisheries Society*. 129: 578-592

Farag, A.M., D.D. Harper, L. Cleveland, W.G. Brumbaugh, and E.E. Little. 2006. The potential for chromium to affect the fertilization process of Chinook salmon (*Oncorhynchus tshawytscha*) in the Hanford Reach of the Columbia River, Washington, USA. *Archives of Environmental Contamination and Toxicology*. 50(4): 575-579. *http://dx.doi.org/10.1007/s00244-005-0010-2*

FDA (Food and Drug Administration). 1984. Action level for methyl mercury in fish. *Federal Register*. 49: 45663 (November 19, U.S. Food and Drug Administration)

Felicetti, L.A., C.C. Schwartz, R.O. Rye, K.A. Gunther, J.G. Crock, M.A. Haroldson, L. Waits, and C.T. Robbins. 2004. Use of naturally occurring mercury to determine the importance of cutthroat trout to Yellowstone grizzly bears. *Canadian Journal of Zoology*. 82(3): 493–501

Feroz, M., and M. A. Q. Khan. 1979. Metabolism of SUP-14 C-heptachlor in goldfish (Carassius aurarus) Arch. Environ. Contam. Toxicol. 8:519-531.

Ferrando, M., E. Sancho, and E. Andreu-Moliner. 1995. Effects of lindane on Daphnia magna during chronic exposure. *Journal of Environmental Science & Health Part B.* 30(6): 815-825

Finlayson, B. J., and K. M. Verrue. 1982. Toxicities of copper, zinc, and cadmium mixtures to juvenile chinook salmon. Trans. Amer. Fish. Society 111:645-650.

Fjeld, E., T.O. Haugen, and L.A. Vøllestad. 1998. Permanent impairment in the feeding behavior of grayling (*Thymallus thymallus*) exposed to methylmercury during embryogenesis. *Science of the Total Environment*. 213(1-3): 247-254

Fliedner, A., and W. Klein, W. 1996. Effects of lindane on the planktonic community in freshwater microcosms. Ecotoxicol. Environ. Saf. 33:228-235.

Foerster, R.E. 1968. The sockeye salmon. Bulletin of the Fisheries Research Board of Canada, 162. 422 pages.

Folmar, L.C., W.W. Dickhoff, W.S. Zaugg, and H.O. Hodgins. 1982. The effects of Aroclor 1254 and no. 2 fuel oil on smoltification and seawater adaptation of coho salmon (*Oncorhynchus kisutch*). Aquat. Toxicol. 2: 291-299.

Forbes, T.L. and V.E. Forbes. 1993. A critique of the use of distribution-based extrapolation models in ecotoxicology. *Functional Ecology*. 7(3): 249-254

Forbes, V. E., and P. Calow. 1999. Is the per capita rate of increase a good measure of population-level effects in ecotoxicology? Environ. Toxicol. Chem. 18:1544-1556.

Forbes, V.E. and P. Calow. 2002. Species sensitivity distributions revisited: a critical appraisal. *Human and Ecological Risk Assessment*. 8(3): 473-492

Forbes, V.E., P. Calow, and R.M. Sibly. 2008. The extrapolation problem and how population modeling can help. *Environmental Toxicology and Chemistry*. 27(10): 1987-1994. *http://dx.doi.org/DOI:* 10.1897/08-029.1

Ford M.J. (ed) 2011 Status Review update for Pacific salmon and steelhead listed under the Endangered Species Act: Pacific Northwest. U.S. Dept. Commer., NOAA TechMemo. NMFS-NWFSC-113, 281 p.

FPC (Fish Passage Center) . 2011a. Query of annual adult sockeye salmon passage data at Lower Granite dam (1975 – 2010). Available: http://www.fpc.org/adultsalmon/adultqueries/Adult_Annual_Totals_Query_form.html. (October 2011).

FPC. 2011b. Query of annual adult Chinook salmon passage data at Lower Granite dam (1975-2010). Available: http://www.fpc.org/adultsalmon/adultqueries/Adult_Annual_Totals_Query_form.html. (July 2011).

FPC. 2011c. Query of annual adult steelhead passage data at Lower Granite dam (1975 – 2010). Available: *http://www.fpc.org/adultsalmon/adultqueries/Adult_Annual_Totals_Query_form.html*. (August 2011).

Friedmann, A.S., M.C. Watzin, T. Brinck-Johnsen, and J.C. Leiter. 1996. Low levels of dietary methylmercury inhibit growth and gonadal development in juvenile walleye (*Stizostedion vitreum*). *Aquatic Toxicology*. 35(3-4): 265-278

Frost, T.P. and S.E. Box. 2009. Stream-Sediment Geochemistry in Mining-Impacted Drainages of the Yankee Fork of the Salmon River, Custer County, Idaho. U.S. Geological Survey, Scientific Investigations Report 2008-5115, Reston, VA. *http://pubs.usgs.gov/sir/2009/5115/.*

Fujimura, R., B. Finlayson, and G. Chapman. 1991. Evaluation of acute and chronic toxicity tests with larval striped bass. Aquat. Toxicol. Risk Assess. 14:193-211.

Fulton, L.A. 1968 Spawning areas and abundance of Chinook salmon (Oncorhynchus tshawytscha) in the Columbia River Basin: past and present. United States Fish and Wildlife Service, Special Scientific Report--Fisheries No. 571, 28 pp.

Fulton. 1970. Spawning Areas and Abundance of Steelhead trout and Coho, Sockeye, and Chum Salmon in the Columbia River Basin – Past and Present. U.S. Department of Commerce, NMFS Special Scientific Report – Fisheries No. 618.

Gaillardet, J., J. Viers, and B. Dupre. 2007. Trace Elements in River Waters. Pages 225-272 *in Treatise on Geochemistry*, volume 5. Elsevier, *http://www.sciencedirect.com/science/referenceworks/9780080437514*.

Gakstatter, J.H. and C.M. Weiss. 1967. The elimination of DDT-C14, dieldrin-C14, and lindane-C14 from fish following a single sublethal exposure in aquaria. *Transactions of the American Fisheries Society*. 96(3): 301-307

Galvez, F., and C.M. Wood. 1997. The relative importance of water hardness and chloride levels in modifying the acute toxicity of silver to rainbow trout (*Oncorhynchus mykiss*). Env. Tox. Chem. 16:2363-2368.

Galvez, F., and C.M. Wood. 1999. Physiological effects of dietary silver sulfide exposure in rainbow trout. Env. Tox. Chem. 18:84-88.

Gardner, D. R. 1973. The effect of some DDT and methoxychlor analogs on temperature selection and lethality in brook trout fingerlings. Pestic. Biochem. Physiol. 2:437-446.

Gearheard, M.F. 2008. EPA Disapproval of Idaho's Removal of Mercury Acute and Chronic Freshwater Aquatic Life Criteria, Docket No. 58-0102-0302. Letter of 12 December from Michael F. Gearheard, EPA Region 10, Office of Water and Watersheds, Seattle, to Barry Burnell, Water Quality Program Administrator, Idaho Department of Environmental Quality, Boise. 6 pp. *http://www.deq.idaho.gov/media/451688-epa_letter_mercury_criterion_disapproval.pdf* [Accessed November 2011].

Geckler, J.R., W.B. Horning, T.M. Nieheisel, Q.H. Pickering, E.L. Robinson, and C.E. Stephan. 1976. Validity of laboratory tests for predicting copper toxicity in streams. U.S. EPA Ecological Research Service, EPA 600/3-76-116, Cincinnati, OH. 208 pp.

GEI (GEI Consultants). 2008. Selenium bioaccumulation evaluation Thompson Creek, Custer County, Idaho, 2007. GEI Consultants, Littleton, CO. 43 pp.

GEI. 2013. Aquatic biological monitoring of Thompson Creek and Squaw Creek, Custer County, Idaho, 2012. GEI Consultants, Littleton, CO. 123 pp.

Gensemer, R.W., R.B. Naddy, W.A. Stubblefield, J.R. Hockett, R.C. Santore, and P.R. Paquin. 2002. Evaluating the role of ion composition on the toxicity of copper to *Ceriodaphnia dubia* in very hard waters. *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology*. 133(1-2): 87-97

Gensemer, R.W., D.K. DeForest, R.D. Cardwell, D. Dzombak, and R.C. Santore. 2007. Scientific review of cyanide ecotoxicology and evaluation of ambient water quality criteria. Water Environment Research Foundation, 01-ECO-01, Alexandria, Virginia. 124 pp.

Geobel H., S. Gorbach, W. Knauf, R. H. Rimpau, and H. Hüttenbach. 1982. Properties, Effects, Residues and Analytics of the Insecticide Endosulfan. Residue Rev. 83:1-165.

Georgacakis, E., S.R. Chadran, and M.A.Q. Khan. 1971. Toxicity-Metabolism Relationship of the Photoisomers of Cyclodiene Insecticides in Freshwater Animals. Bull. Environ. Contam. Toxicol. 6:535-538.

Giattina, J.D., R.R. Garton, and D.G. Stevens. 1982. Avoidance of copper and nickel by rainbow trout as monitored by a computer-based data acquisition system. Trans. Am. Fish. Soc. 111:491-504.

Gibson, H.R. and D.W. Chapman. 1972. Effects of Zectran insecticide on aquatic organisms in Bear Valley Creek, Idaho. *Transactions of the American Fisheries Society*. 101(2): 330-344. *http://dx.doi.org/10.1577/1548-8659(1972)101<330:eozioa>2.0.co;2*

Giesy, J.P., and K. Kannan. 1998. Dioxin-like and non-dioxinBlike toxic effects of polychlorinated biphenyls (PCBs): implications for risk assessment. Crit. Rev. Toxicol. 28: 511-569.

Gill, G.A., and K.W. Bruland. 1990. Mercury speciation in surface freshwater systems in California and other areas. Environ. Sci. Technol. 24(9):1392B1399.

Gillespie, R.B. and P.C. Baumann. 1986. Effects of high tissue concentrations of selenium on reproduction by bluegills. *Transactions of the American Fisheries Society*. 115(2): 208–213

Glenn, D.W., III and J.J. Sansalone. 2002. Accretion and Partitioning of Heavy Metals Associated with Snow Exposed to Urban Traffic and Winter Storm Maintenance Activities. II. *Journal of Environmental Engineering*. 128(2): 167-185

Glickman A.H., C.N. Statham, A. Wu, J.J. Lech. (1977). Studies on the uptake, metabolism, and disposition of pentachlorophenol and pentachloroanisole in rainbow trout. Toxicology and Applied Pharmacology 41:649-658.

Glubokov, AI. 1990. Growth of three species of fish during early ontogeny under normal and toxic conditions. J. Ichthyol. 30:51-59.

Gobas, F.A., D.C. Muir, and D. Mackay. 1988. Dynamics of dietary bioaccumulation and faecal elimination of hydrophobic organic chemicals in fish. *Chemosphere*. 17(5): 943-962

Goebel, H., S. Gorbach, W. Knauf, R.H. Rimpau, and H. Huttenbach. 1982. Properties, effects, residues, and analytics of the insecticide Endosulfan. *Residue Reviews*. 83: 1-165. http://dx.doi.org/10.1007/978-1-4612-5712-7_1

Goerke, H., G. Eder, K. Weber, and W. Ernst. 1979. Patterns of Organochlorine Residues in Animals of Different Trophic Levels from the Weser Estuary. Mar. Pollut. Bull. 10:127-133.

Golder. 2003. Blackbird Mine Site 2002 Monitoring Report, Lemhi County, Idaho. Golder Associates, Inc., Report to the Blackbird Mine Site Group, Redmond, WA. various pp.

Golder. 2009. Blackbird Mine Site 2008 Monitoring Report, Lemhi County, Idaho. Golder Associates, Inc., Report to the Blackbird Mine Site Group, Redmond, WA. 347 pp.

Goldstein, J.N., D.F. Woodward, and A.M. Farag. 1999. Movements of adult chinook salmon during spawning migration in a metals-contaminated system, Coeur d'Alene River, Idaho. *Transactions of the American Fisheries Society*. 128(1): 121–129

Goldstein, J.N., W.A. Hubert, D.F. Woodward, A.M. Farag, and J.S. Meyer. 2001. Naturalized salmonid populations occur in the presence of elevated trace element concentrations and temperatures in the Firehole River, Yellowstone National Park, Wyoming, USA. *Environmental Toxicology and Chemistry*. 20: 2342–2352

Gooch, J. W., F. Matsumura, and M. J. Zabik. 1990. Chlordane residues in Great Lakes lake trout: Acute toxicity and interaction at the GABA receptor of rat and lake trout brain. Chemosphere 21:393-406.

Good, T.P., R.S. Waples, and P. Adams (editors). 2005. Updated status of Federally listed ESUs of West Coast salmon and steelhead. National Marine Fisheries Service, Northwest Fisheries Science Center, NOAA Technical Memorandum, NMFS-NWFSC-66, 598 pages. *http://www.nwr.noaa.gov/Publications/Biological-Status-Reviews/upload/SR2005-allspecies.pdf*

Goodman, L.R., D.J. Hansen, J. A. Couch, and J. Forester. 1976. Effects of heptachlor and toxaphene on laboratory-reared embryos and fry of the sheepshead minnow. Proc., Annu. Conf., Southeast. Assoc. Game Fish Comm, 30:192-202.

Grant, B.R., and P.M. Mehrle. 1973. Endrin toxicosis in rainbow trout (Salmo gairdneri). J. Fish. Res. Board Can., vol. 30:31-40.

Greco, A.M., K.M. Gilmour, J.C. Fenwick, and S.F. Perry. 1995. The effects of softwater acclimation on respiratory gas transfer in the rainbow trout *Oncorhynchus mykiss*. *The Journal of Experimental Biology*. 198: 2557-2567

Grosell, M., R.M. Gerdes, and K.V. Brix. 2006a. Chronic toxicity of lead to three freshwater invertebrates - *Brachionus calyciflorus, Chironomus tentans,* and *Lymnaea stagnalis*. *Environmental Toxicology and Chemistry*. 25(1): 97–104

Grosell, M.H., R.M. Gerdes, and K.V. Brix. 2006b. Influence of Ca, humic acid and pH on lead accumulation and toxicity in the fathead minnow during prolonged water-borne lead exposure. *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology*. 143(4): 473-483

Grothe, D.L., K.L. Dickson, and D.K. Reed-Judkins, editors. 1996. *Whole effluent toxicity testing: an evaluation of methods and prediction of receiving system impacts*. SETAC Press, Pensacola, FL, 346 pp.

Gruger, E.H., N.L. Karrick, A.I. Davidson, and T. Hruby. 1975. Accumulation of 3,4,3',4' tetrachlorobiphenyl, 2,4,5,2',4',5' and 2,4,6,2',4',6' hexachlorobiphenyl in juvenile coho salmon. Environ. Sci. Technol. 9:121-127.

Gruger, E.H., T. Hruby, and N.L. Karrick. 1976. Sublethal effects of structurally related tetrachloro-, pentachloro-, and hexachlorobiphenyl on juvenile coho salmon. Environ. Sci. Technol. 10:1033-1037.

Gupta, A.K., D. Dutt, M. Anand, and R.C. Dalela. 1994. Combined toxicity of chlordane, malathion and furadan to a test fish Notopterus notopterus (Mor). Journal of Environmental Biology 15:1-6.

Gupta, A.K. A. Muni, S. Ranjana, and R.C. Dalela. 1995. Toxic effects of chlordane and malathion on certain haematological parameters of a freshwater teleost, Notopterus notopterus. Journal of Environmental Biology 16:219-223.

Hall, W.S., K. Dickson, F. Saleh, and J. Rodgers, Jr. 1986. Effects of suspended solids on the bioavailability of chlordane toDaphnia magna. *Archives of Environmental Contamination and Toxicology*. 15(5): 529-534. http://dx.doi.org/10.1007/bf01056566

Hall, B.D., R.A. Bodaly, R.J.P. Fudge, J.W.M. Rudd, and D.M. Rosenberg. 1997. Food as the dominant pathway of methylmercury uptake by fish. *Water, Air, and Soil Pollution*. 100(1-2): 13-24

Halter, M. T., and H. E. Johnson. 1974. Acute toxicities of a polychlorinated biphenyl (PCB and DDT alone and in combination to early life stages of coho salmon (Oncorhynchus kisutch). J. Fish. Res. Board Can. 31:1543-1547.

Hamilton, S.J. 2002. Rationale for a tissue-based selenium criterion for aquatic life. *Aquatic Toxicology*. 57(1-2): 85-100

Hamilton, S.J. 2003. Review of residue-based selenium toxicity thresholds for freshwater fish. *Ecotoxicology and Environmental Safety*. 56(2): 201-210

Hamilton, S.J., K.J. Buhl, N.L. Faerber, R.H. Wiedmeyer, and F.A. Bullard. 1990. Toxicity of organic selenium in the diet to chinook salmon. *Environmental Toxicology and Chemistry*. 9(3): 347–358

Hammerschmidt, C.R., J.G. Wiener, B.E. Frazier, and R.G. Rada. 1999. Methylmercury content of eggs in yellow perch related to maternal exposure in four Wisconsin lakes. *Environmental Science and Technology*. 33(7): 999-1003. *http://dx.doi.org/10.1021/es980948h*

Hammerschmidt, C.R., M.B. Sandheinrich, J.G. Wiener, and R.G. Rada. 2002. Effects of dietary hethylmercury on reproduction of fathead minnows. *Environmental Science and Technology*. 36(5): 877-883

Hansen, D.J., and P.R. Parrish. 1977. Suitabiity of sheepshead minnows (Cyprinodon variegatur) for life-cycle toxicity tests: In: F.L. Meyer and J.L. Hamelink (eds.), Toxicology and Hazard Evaluation. ASTM STP 634, Am. Soc. Test. Mater. p. 117.

Hansen, D.J., S.C. Schimmel, and J. Forester. 1977. Endrin: effects on the entire life cycle of a saltwater fish, Cyprinodon variegates. J. Toxicol. Environ. Health 3:721-733.

Hansen, J.A., J.C.A. Marr, J. Lipton, and H.L. Bergman. 1999. Differences in neurobehavioral responses of chinook salmon *(Oncorhynchus tshawytscha)* and rainbow trout *(Oncorhynchus mykiss)* exposed to copper and cobalt: behavioral avoidance. *Environmental Toxicology and Chemistry*. 18(9): 1972-1978

Hansen, J.A., J. Lipton, and P.G. Welsh. 2002a. Relative sensitivity of bull trout (*Salvelinus confluentus*) and rainbow trout (*Oncorhynchus mykiss*) to acute copper toxicity. *Environmental Toxicology and Chemistry*. 21(3): 633–639. *http://dx.doi.org/10.1002/etc.5620210324*

Hansen, J.A., J. Lipton, P.G. Welsh, J. Morris, D. Cacela, and M.J. Suedkamp. 2002b. Relationship between exposure duration, tissue residues, growth, and mortality in rainbow trout (*Oncorhynchus mykiss*) juveniles sub-chronically exposed to copper. *Aquatic Toxicology*. 58: 175-188

Hansen, J.A., P.G. Welsh, J. Lipton, D. Cacela, and A.D. Dailey. 2002c. Relative sensitivity of bull trout *(Salvelinus confluentus)* and rainbow trout *(Oncorhynchus mykiss)* to acute exposures of cadmium and zinc. *Environmental Toxicology and Chemistry*. 21(1): 67–75.

Hansen, J.A., J. Lipton, P.G. Welsh, D. Cacela, and B. MacConnell. 2004. Reduced growth of rainbow trout (*Oncorhynchus mykiss*) fed a live invertebrate diet pre-exposed to metal-contaminated sediments. *Environmental Toxicology and Chemistry*. 23(8): 1902–1911

Hardy, M.A., D.J. Parliman, and I. O'Dell. 2005. Status of and changes in water quality monitored for the Idaho statewide surface-water-quality network, 1989-2002. U.S. Geological Survey, Scientific Investigations Report, Scientific Investigations Report 2005–5033, Boise, Idaho. 105 pp. *http://id.water.usgs.gov/PDF/sir20055033/.*

Hardy, R.W., L. Oram, and G. Möller. 2010. Effects of dietary selenomethionine on cutthroat trout (*Oncorhynchus clarki bouvieri*) growth and reproductive performance over a life cycle. *Archives of Environmental Contamination and Toxicology*. 58(1): 237-245. *http://dx.doi.org/DOI:* 10.1007/s00244-009-9392-x

Harper, D.D., A.M. Farag, and W.G. Brumbaugh. 2008. Effects of acclimation on the toxicity of stream water contaminated with zinc and cadmium to juvenile cutthroat trout. *Archives of Environmental Contamination and Toxicology*. 54(4): 697-704. *http://dx.doi.org/10.1007/s00244-007-9063-8*

Harper, D.D., A.M. Farag, C. Hogstrand, and B. MacConnell. 2009. Trout density and health in a stream with variable water temperatures and trace element concentrations: does a cold-water source attract trout to increased metal exposure? *Environmental Toxicology and Chemistry*. 28(4): 800–808. *http://dx.doi.org/10.1897/08-072R.1*

Harris, H.H., I.J. Pickering, and G.N. George. 2003. The chemical form of mercury in fish. *Science*. 301(5637): 1203. *http://dx.doi.org/10.1126/science.1085941*

Hart, J.L. 1973. Pacific Fishes of Canada. Bulletin of the Fisheries Research Board of Canada 180: 730pages.

Hartt, A.C. and M.B. Dell. 1986. Early ocean migrations and growth of juvenile Pacific salmon and steelhead trout. Bulletin of the International North Pacific Fisheries Commission. 46.105 pages.

Hassan, M.Q., I.T. Numan, N. Al-Nasiri, and S.J. Stohs. 1991. Endrin-induced histopathological changes and lipid peroxidation in livers and kidneys of rats, mice, guinea pigs and hamsters. *Toxicologic pathology*. 19(2): 108-114

Hassan, M.Q., I.T. Numan, N. Al-Nasiri, and S.J. Stohs. 1991. Endrin-induced histopathological changes and lipid peroxidation in livers and kidneys of rats, mice, guinea pigs and hamsters. *Toxicologic Pathology*. 19(2): 108-114

Hassoun, E., M. Bagchi, D. Bagchi, and S.J. Stohs. 1993. Comparative studies on lipid peroxidation and DNA-single strand breaks induced by lindane, DDT, chlordane and endrin in rats. *Comparative Biochemistry and Physiology. Part C, Comparative*. 104(3): 427-431

Healey, M.C. 1991. The life history of Chinook salmon (*Oncorhynchus tshawytscha*). *In* C. Groot and L. Margolis (eds.), Life history of Pacific salmon, p. 311-393. Univ. B.C. Press, Vancouver, B.C.

Hebdon, J.L., P. Kline, D. Taki, and T.A. Flagg. 2004. Evaluating reintroduction strategies for Redfish Lake sockeye salmon captive broodstock strategy. Pages 401-413 *in* M. J. Nickum, P.M. Mazik, J. G. Nickum, and D.D. MacKinlay, editors. Propagated fish in resource management. American Fisheries Society, Symposium 44, American Fisheries Society, Bethesda, Maryland.

Hecht, S.A., D.H. Baldwin, C.A. Mebane, T. Hawkes, S.J. Gross, and N.L. Scholz. 2007. An overview of sensory effects on juvenile salmonids exposed to dissolved copper: Applying a benchmark concentration approach to evaluate sublethal neurobehavioral toxicity. National Marine Fisheries Service, NOAA Technical Memorandum NMFS-NWFSC-83, Seattle, WA. 51 pp. *http://www.nwfsc.noaa.gov/publications/scipubs/index.cfm*.

Hedtke, J.L., E. Robinson-Wilson, and L.J. Weber. 1982. Influence of body size and developmental stage of coho salmon (*Oncorhynchus kisutch*) on lethality of several toxicants. *Fundamental and Applied Toxicology*. 2(2): 67-72

Hedtke, S.F., C.W. West, K.N. Allen, T.J. Norberg-King, and D.I. Mount. 1986. Toxicity of pentachlorophenol to aquatic organisms under naturally varying and controlled environmental conditions. *Environmental Toxicology and Chemistry*. 5(6): 531-542.

Heijerick, D.G., K.A.C. De Schamphelaere, and C.R. Janssen. 2002. Predicting acute zinc toxicity for *Daphnia magna* as a function of key water chemistry characteristics: development and validation of a biotic ligand model. *Environmental Toxicology and Chemistry*. 21(6): 1309–1315

Helsel, D.R. 2005. Nondetects and data analysis: statistics for censored environmental data. Wiley Interscience, Hoboken, NJ

Helsel, D.R. and R.M. Hirsch. 2002. Statistical methods in water resources. Pages 524 *in Techniques of Water-Resources Investigations of the United States Geological Survey, Book 4, Hydrologic Analysis and Interpretation, Chapter A3*. U.S. Geological Survey, *http://pubs.usgs.gov/twri/twri4a3/*.

Hendricks, J.D., T.P. Putnam, and R.O. Sinnhuber. 1979. Effect of Dietary Dieldrin on Aflatoxin B1 Carcinogenesis in Rainbow Trout (Salmo gairdneri). J. Environ. Pathol. Toxicol. 2:719-728.

Henjum, M.G., J.R. Karr, D.L. Bottom, D.A. Perry, J.C. Bednarz, S.G. Wright, S.A. Beckwitt and E. Beckwitt. 1994. Interim Protection for Late-successional Forests, Fisheries and Watersheds. National Forests East of the Cascade Crest, Oregon and Washington. A Report to the United States Congress and the President. The Wildlife Society, Bethesda, MD.

Hermanutz, R.O. 1992. Malformation of the fathead minnow (*Pimephales promelas*) in an ecosystem with elevated selenium concentrations. *Bulletin of Environmental Contamination and Toxicology*. 49(2): 290-294

Hermanutz, R.O., K.N. Allen, T.H. Roush, and S.F. Hedtke. 1992. Effects of elevated selenium concentrations on bluegills (*Lepomis macrochirus*) in outdoor experimental streams. *Environmental Toxicology and Chemistry*. 11(2): 217-224

Hermanutz, R.O., K.N. Allen, N. Detenbeck, and C.E. Stephan. 1996. Exposure of Bluegills (*Lepomis macrochirus*) to Selenium in Outdoor Experimental Streams. U.S. Environmental Protection Agency, National Health and Environmental Research Laboratory, Mid-Continent Ecological Division, Duluth. 43 pp.

Hiltibran, R.C. 1982. Effects of insecticides on the metal-activated hydrolysis of adenosine triphosphate by bluegill liver mitochondria. Archives of Environmental Contamination and Toxicology 11:709-717.

Hilton, J.W. and P.V. Hodson. 1983. Effect of increased dietary carbohydrate on selenium metabolism and toxicity in rainbow trout (*Salmo gairdneri*). *Journal of Nutrition*. 113(6): 1241-1248

Hirsch, M.P. 1998a. Toxicity of silver sulfide-spiked sediments to the freshwater amphipod (*Hyalella azteca*). Environ. Toxicol. Chem. 17: 601-605.

Hirsch, M.P. 1998b. Bioaccumulation of silver from laboratory -spiked sediments in the oligochaete (*Lumbricus varietgatus*). Environ. Toxicol. Chem. 17: 605-609.

Hodson P.V., and B.R. Blunt. 1981. Temperature-induced changes in pentachlorophenol chronic toxicity to early life stages of rainbow trout. Aquatic Toxicology 1:113-127.

Hodson, P.V. and J.B. Sprague. 1975. Temperature-induced changes in acute toxicity of zinc to Atlantic salmon (*Salmo salar*). *Journal of the Fisheries Research Board of Canada*. 33(1): 1-10

Hodson, P.V., D.G. Dixon, D.J. Spry, D.M. Whittle, and J.B. Sprague. 1982. Effect of growth rate and size of fish on rate of intoxication by water-borne lead. Can. J. Fish. Aquat. Sci. 39:1243-1251.

Hoff, D.J., T.L. Highland, J.R. Hockett, C.T. Jenson, and M. Poe. 2011. Dietary Arsenic Toxicity in Subadult Rainbow Trout: Growth Effects, Nutrient Absorption, and Tissue Bioaccumulation [poster]. *in* Abstracts, SETAC North America 32nd Annual Meeting, November 16, 2011, Boston, MA. Society of Environmental Toxicology and Chemistry (SETAC), *http://boston.setac.org/*.

Hogstrand, C. 2011. Zinc. *Fish Physiology: Homeostasis and Toxicology of Essential Metals*. 31(PART A): 135-200. http://dx.doi.org/10.1016/S1546-5098(11)31003-5

Hogstrand, C. and C.M. Wood. 1998. Toward a better understanding of the bioavailability, physiology, and toxicity of silver in fish: Implications for water quality criteria. *Environmental Toxicology and Chemistry*. 17(4): 547-561. *http://dx.doi.org/10.1002/etc.5620170405*

Hogstrand, C., F. Galvez, and C.M. Wood. 1996. Toxicity, silver accumulation and metallothionein induction in freshwater rainbow trout during exposure to different silver salts. *Environmental Toxicology and Chemistry*. 15(7): 1102-1108. *http://dx.doi.org/10.1002/etc.5620150713*

Holcombe, G.W., D.A. Benoit, E.N. Leonard, and J.M. McKim. 1976. Long-term effects of lead exposure on three generations of brook trout (*Salvelinus fontinalis*). *Journal of the Fisheries Research Board of Canada*. 33(8): 1731-1741

Holcombe, G.W., G.L. Phipps, and J.T. Fiandt. 1983. Toxicity of selected priority pollutants to various aquatic organisms. *Ecotoxicology and Environmental Safety*. 7(4): 400-409

Hollis, L., J.C. McGeer, D.G. McDonald, and C.M. Wood. 1999. Cadmium accumulation, gill Cd binding, acclimation, and physiological effects during long term sublethal Cd exposure in rainbow trout. *Aquatic Toxicology*. 46(2): 101-119

Holm, J., V.P. Palace, K. Wautier, R.E. Evans, C.L. Baron, C.L. Podemski, P. Siwik, and G.L. Sterling. 2005. Developmental effects of bioaccumulated selenium in eggs and larvae of two salmonid species. *Environmental Toxicology and Chemistry*. 24(9): 257-273

Hontela, A. 1997. Endocrine and physiological responses of fish to xenobiotics: Role of glucocorticosteroid hormones. *Reviews in Toxicology*. 1(5-6): 1-46

Hood T.E., E.J. Calabrese, and B.M. Zuckerman. 2000. Detection of an estrogen receptor in two nematode species and inhibition of binding and development by environmental chemicals. Ecotoxicol Environ Saf 47(1):74-81

Hope, B.K. and J.R. Rubin. 2005. Mercury Levels and Relationships in Water, Sediment, and Fish Tissue in the Willamette Basin, Oregon. *Archives of Environmental Contamination and Toxicology*. 48(3): 367-380

Hope, B.K., A. Lut, G. Aldrich, and J. Rubin. 2007. Environmental management with knowledge of uncertainty: a methylmercury case study. *Integrated Environmental Assessment and Management*. 3(1): 144–149

Hopkin, S.P. 1993. Ecological implicatons of the "95% protection levels" for metals in soils. *Oikos*. 66: 137-141

Howarth, R.S. and J.B. Sprague. 1978. Copper lethality to rainbow trout in waters of various hardness and pH. *Water Research*. 12(7): 455-462.

HSDB (U.S. National Library of Medicine. Hazardous Substances Data Bank). Bethesda, MD, 1995.

Hudson, R.H., R.K. Tucker, and K. Haegele. 1984. Handbook of Acute Toxicity of Pesticides to Wildlife. Resource Publication 153. U.S. Dept. of Interior, Fish and Wildlife Service, Washington, DC. .

Hunn, J.B., S.J. Hamilton, and D.R. Buckler. 1987. Toxicity of sodium selenite to rainbow trout fry. *Water Research*. 21(2): 233-238

Hyne, R.V., F. Pablo, M. Julli, and S.J. Markich. 2005. Influence of water chemistry on the acute toxicity of copper and zinc to the cladoceran *Ceriodaphnia* cf *dubia*. *Environmental Toxicology and Chemistry*. (7): 1667–1675. *http://dx.doi.org/10.1897/04-497R.1*

IARC (International Agency for Research on Cancer. 1974. IARC monographs on the evaluation of the carcinogenic risk of chemicals to man. Some organochlorine pesticides. Vol. 5, 241 pp. Lyon, France.

IARC. 1982. IARC monographs on the evaluation of the carcinogenic risk of chemicals to man. Chemicals, industrial processes and industries associated with cancer in humans. Suppl. 4. 242 pp. Lyon, France.

ICTRT (Interior Columbia Basin Technical Recovery Team). 2003. Independent populations of Chinook, steelhead, and sockeye for listed evolutionarily significant units within the Interior Columbia River domain. Working Draft. *www.nwfsc.noaa.gov/trt/col_docs/independentpopchinsteelsock.pdf*

ICTRT. 2007. Viability criteria for application to interior Columbia basin salmonid ESUs – review draft. Seattle, WA. Available: *http://www.nwfsc.noaa.gov/trt/trt_documents/ictrt_viability_criteria_reviewdraft_2007_complete.pdf*. (October 2011).

Ide, F.P. 1957. Effect of Forest Spraying with DDT on Aquatic Insects of Salmon Streams. *Transactions of the American Fisheries Society*. 86(1): 208-219. *http://dx.doi.org/10.1577/1548-8659(1956)86[208:eofswd]2.0.co;2*

IDEQ (Idaho Department of Environmental Quality). 1999. Mixing zone recommendations for proposed Grouse Creek Mine discharges into the Yankee Fork of the Salmon River, Idaho. Technical Services Division, Idaho Department of Environmental Quality, Boise. 27 pp.

IDEQ. 2003. Upper Salmon River Subbasin Assessment and TMDL. 232 pp. *www.deq.idaho.gov/media/454912-salmon_river_upper_entire.pdf*.

IDEQ. 2005. Implementation Guidance for the Idaho Mercury Water Quality Criteria. Idaho Department of Environmental Quality, Boise, ID. 212 pp. *http://www.deq.state.id.us/water-quality/surface-water/mercury.aspx.*

IDEQ. 2007a. Rules of the Department of Environmental Quality, IDAPA 58.01.02, "Water Quality Standards". revised March 30, 2007. *http://adm.idaho.gov/adminrules/rules/idapa58/0102.pdf* [Accessed August 2011].

IDEQ. 2007b. Salmon Falls Creek Subbasin Assessment and TMDL. Idaho Department of Environmental Quality, Twin Falls, ID. 413 pp. *http://www.deq.state.id.us/WATER/data_reports/surface_water/tmdls/salmon_falls_creek/salmon_falls_creek.cfm#SBA*.

IDEQ. 2010. Idaho's 2010 Integrated Report. Idaho Department of Environmental Quality. 776 pp. http://www.deq.idaho.gov/water-quality/surface-water/monitoring-assessment/integrated-report.aspx [Accessed March 2014].

IDEQ. 2011 Idaho Department of Environmental Quality. Rules of the Department of Environmental Quality, IDAPA 58.01.02, "Water Quality Standards". revised March 18, 2011. *http://adm.idaho.gov/adminrules/rules/idapa58/0102.pdf* [Accessed August 2011].

IDFG (Idaho Fish and Game); Nez Perce Tribe and Shoshone-Bannock Tribes. 1990. Salmon River Subbasin Salmon and Steelhead Production Plan, September 1, 1990. Northwest Power Planning Council, Portland, Oregon.

IDFG. 2011. Sockeye recovery and status: 12-year hatchery returns. Available: *http://fishandgame.idaho.gov/public/fish/?getPage=149*. (October 2011).

Independent Science Group. 2000. *Return to the River: Restoration of Salmonid Fishes in the Columbia River Ecosystem*. Council document 2000-12. For the Northwest Power Planning Council, Portland, Oregon. http://www.nwcouncil.org/library/return/2000-12.htm

Ingersoll, C.G. and R.W. Winner. 1982. Effect on *Daphnia pulex* (De Geer) of daily pulse exposures to copper or cadmium. *Environmental Toxicology and Chemistry*. 1(4): 321-327. *http://dx.doi.org/10.1002/etc.5620010407*

Ingersoll, C.G., W.G. Brumbaugh, F.J. Dwyer, and N.E. Kemble. 1994. Bioaccumulation of metals by *Hyalella azteca* exposed to contaminated sediments from the upper Clark Fork River, Montana. Environ. Toxicol. Chem. 13:2013-2020.

Ingersoll, C.G., E.L. Brunson, F.J. Dwyer, D.K. Hardesty, and N.E. Kemble. 1998. Use of sublethal endpoints in sediment toxicity tests with the amphipod *Hyalella azteca*. *Environmental Toxicology and Chemistry*. 17(8): 1508-1523. *http://dx.doi.org/10.1002/etc.5620170811*

Irving, E.C., D.J. Baird, and J.M. Culp. 2003. Ecotoxicological responses of the mayfly *Baetis tricaudatus* to dietary and waterborne cadmium: implications for toxicity testing. *Environmental Toxicology and Chemistry*. 22(5): 1058-1064

Irving, E.C., R.B. Lowell, J.M. Culp, K. Liber, Q. Xie, and R. Kerrich. 2008. Effects of arsenic speciation and low dissolved oxygen condition on the toxicity of arsenic to a lotic mayfly. *Environmental Toxicology and Chemistry*. 27(3): 583–590

ISAB (Independent Scientific Advisory Board). 2007. Climate change impacts on Columbia River Basin fish and wildlife. ISAB Climate Change Report, ISAB 2007-2, Northwest Power and Conservation Council, Portland, Oregon.

Isnard, P. and S. Lambert. 1988. Estimating bioconcentration factors from octanol-water partition coefficient and aqueous solubility. *Chemosphere*. 17(1): 21-34

Iwama G.K., G.L. Greer, and D.J. Randall. (1986). Changes in selected haematological parameters in juvenile chinook salmon subjected to a bacterial challenge and a toxicant. Journal of Fish Biology 28:563-572.

Iwasaki, Y., T. Kagaya, K.-i. Miyamoto, and H. Matsuda. 2009. Effects of heavy metals on riverine benthic macroinvertebrate assemblages with reference to potential food availability for drift-feeding fishes. *Environmental Toxicology and Chemistry*. 28(2): 354–363. *http://dx.doi.org/doi:* 10.1897/08-200.1

Janssen, C.R., G. Persoone, and T.W. Snell. 1994. Cyst-based toxicity tests. VIII. Short-chronic toxicity tests with the freshwater rotifer Brachionus calyciflorus. *Aquatic Toxicology*. 28(3-4): 243-258. http://dx.doi.org/10.1016/0166-445X(94)90036-1

Janz, D.M., D.K. DeForest, M.L. Brooks, P.M. Chapman, G. Gilron, D. Hoff, W.A. Hopkins, D.O. McIntyre, C.A. Mebane, V.P. Palace, J.P. Skorupa, and M. Wayland. 2010. Selenium toxicity to aquatic organisms. Pages 139-230 *in* P. M. Chapman, W. J. Adams, M. L. Brooks, C. G. Delos, S. N. Luoma, W. A. Maher, H. M. Ohlendorf, T. S. Presser, and D. P. Shaw, editors. *Ecological Assessment of Selenium in the Aquatic Environment*. Society of Environmental Toxicology and Chemistry (SETAC), Pensacola, Florida, *http://dx.doi.org/10.1201/EBK1439826775-c6*.

Janz, D.M. 2011. Selenium. *Fish Physiology: Homeostasis and Toxicology of Essential Metals*. 31(PART A): 327-374. http://dx.doi.org/10.1016/S1546-5098(11)31007-2

Jarvinen, A.W. and G.T. Ankley. 1999. Linkage of effects to tissue residues: development of a comprehensive database for aquatic organisms exposed to inorganic and organic chemicals. SETAC Press, Pensacola, Fla. 364 pp

Jarvinen, A.W., and R.M. Tyo. 1978. Toxicity to fathead minnows of endrin in food and water. Arch. Environ. Contam. Toxicol. 7(4):409-421.

Jarvinen, A.W., M. J. Hoffman, and T. W. Thorslund. 1977. Long-term toxic effects of DDT food and water exposure on fathead minnows (Pimphales promelas) Jour. Fish. Res. Board Can. 34:2089.

Jarvinen AW, Tanner DK, Kline ER. 1988. Toxicity of chlorpyrifos, endrin, or fenvalerate to fathead minnows following episodic or continuous exposure. Ecotoxicol Environ Saf 5(1):78-95.

Jenkins, T.M., C.R. Feldmeth, and J.V. Elliott. 1970. Feeding of rainbow trout (*Salmo gairdneri*) in relation to abundance of drifting invertebrates in a mountain stream. *Journal of the Fisheries Research Board Canada*. 27(12): 2356-2361. *http://dx.doi.org/10.1139/f70-264*

Jensen, L.D., and A.R. Gaufin. 1964. Effects of ten organic insecticides on two species of stonefly naiads. Trans. Am. Fish. Soc. 93:27.

Jensen, L.D., and A.R. Gaufin. 1966. Acute and long-term effects of organic insecticides on two species of stonefly naiads. Jour. Water Pollut. Control Fed. 38:1273.

Jessup, B. and J. Gerritsen. 2002. Chapter 3. Stream Macroinvertebrate Index. Pages 3-1 to 3-45 *in* C. S. Grafe, editor. *Idaho Small Stream Ecological Assessment Framework: an Integrated Approach*. Idaho Department of Environmental Quality, Boise, *http://www.deq.idaho.gov/water-quality/surface-water/monitoring-assessment.aspx*.

Johansen P.H., R.A.S. Mathers, J.A. Brown, and Colgan. 1985. Mortality of early life stages of largemouth bass, *Micropterus salmoides* due to pentachlorophenol exposure. Bulletin of Environmental Contamination and Toxicology 34:377-384.

Johnson, D.H. 1999. The insignificance of statistical significance testing. *Journal of Wildlife Management*. 63(3): 763–772

Johnson, W. W., and Finley, M. T. 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

Johnson, H.E., and C. Pecor. 1969. Coho salmon mortality and DDT in Lake Michigan. Trans. N. Am. Wildl. Nat. Resourc. Conf. 34:159.

Jones, R. 1991. The effect of Sunbeam Dam on sockeye salmon in the Salmon River, Idaho. Memo to ESA Administrative Record for sockeye salmon, March 1991, 6 p. Available from U.S. Department of Commerce, National Marine Fisheries Service, Portland, Oregon 97232.

Jones, P.D., K. Kannan, J.L. Newsted, D.E. Tillitt, L.L. Williams, and J.P. Giesy. 2001. Accumulation of 2,3,7,8-tetrachlorodibenzo-p-dioxin by rainbow trout (*Oncorhynchus mykiss*) at environmentally relevant dietary concentrations. Environ. Tox. Chem. 20: 344-350.

Jonsson, C. M., and M. C. F. Toledo. 1993. Bioaccumulation and elimination of endosulfan in fish yellow tetra (Hyphessobrycon bifasciatus) Bull. Environ. Contam. Toxicol. 50:572-577.

Jop, K.M., A.M. Askew, and R.B. Foster. 1995. Development of a water-effect ratio for copper, cadmium, and lead for the Great Works River in Maine, using *Ceriodaphnia dubia* and *Salvelinus fontinalis*. *Bulletin of Environmental Contamination and Toxicology*. 54: 29-35

Karchesky, C.M. and D.H. Bennett. 1999. Dietary overlap between introduced fishes and juvenile salmonids in lower Granite Reservoir, Idaho-Washington. *in* Abstracts, In ODFW and NMFS. 1999. Management Implications of Co-occurring Native and Introduced Fishes: Proceedings of the Workshop. October 27-28, 1998, Portland, Oregon., 145-154 pp.

Karen, DJ., and six others. 1999. Influence of water quality on silver toxicity to rainbow trout (*Oncorhynchus mykiss*), fathead minnows (*Pimephales promelas*), and water fleas (*Daphnia magna*). Environ. Toxicol. Chem. 18:63-70.

Karnak, R.E., and W.J. Collins. 1974. The Susceptibility to Selected Insecticides and Acetylcholinesterase Activity in a Laboratory Colony of Midge Larvae, Chironomus Tentans (Diptera: Chironomidae). Bull. Environ. Contam. Toxicol. 12:62-69.

Katz, M. 1961. Acute toxicity of some organic insecticides to three species of salmonids and to the threespine stickleback. Trans Amer. Fisheries Soc. 90:264-268.

Kaushik, N., and A.S. Kumar. 1993. Susceptibility of the freshwater crab Paratelphusa masoniana (Henserson) to three pesticides, singly and in combination. Environ. Ecol. 11:560-564.

Kavlock, R.J., N. Chernoff, R.C. Hanisch, J. Gray, E. Rogers, and L.E. Gray Jr. 1981. Perinatal toxicity of endrin in rodents. II. Fetotoxic effects of prenatal exposure in rats and mice. *Toxicology*. 21(2): 141-150

Kay, S.H. 1984. Potential for biomagnification of contaminants within marine and freshwater food webs. Technical Report D-84-7, U.S. Army Corps of Engineers Waterways Experiment Station. Vicksburg, MS.

Keefer, M. L., C. A. Peery, and M. J. Henrich. 2008. Temperature mediated en route migration mortality and travel rates of endangered Snake River sockeye salmon. Ecology of Freshwater Fish. 17:136-145.

Keeley, E.R. and J.W.A. Grant. 1997. Allometry of diet selectivity in juvenile Atlantic salmon (*Salmo salar*). *Canadian Journal of Fisheries and Aquatic Sciences*. 54(8): 1894-1902. *http://dx.doi.org/10.1139/f97-096*

Keeley, E.R. and J.W.A. Grant. 2001. Prey size of salmonid fishes in streams, lakes, and oceans. *Canadian Journal of Fisheries and Aquatic Sciences*. 58(6): 1122-1132

Keilty, T.J., D.S. White, and P.F. Landrum. 1988a. Short-term lethality and sediment avoidance assays with endrin-contaminated sediment and two oligochaetes from Lake Michigan. Archives of Environmental Contamination and Toxicology 17: 95-101.

Keilty, T.J., D.S. White, and P.F. Landrum. 1988b. Sublethal responses to endrin in sediment by Stylodrilus heringianus (Lumbriculidae) as measured by a super(137)cesium marker layer technique. Aquatic Toxicology 13:251-270.

Kellogg, R.L., and R.V. Bulkley. 1976. Seasonal Concentrations of Dieldrin in Water, Channel Catfish, and Catfish-Food Organisms, Des Moines River, Iowa B 1971-73. Pest. Monitor. J. 9:186-194.

Kemble, N.E., and six others. 1994. Toxicity of metal-contaminated sediments from the upper Clark Fork River, Montana, to aquatic invertebrates and fish in laboratory exposures. Environ. Toxicol. Chem. 13:1985-1997.

Kennedy, C.J., L.E. McDonald, R. Loveridge, and M.M. Strosher. 2000. The effect of bioaccumulated selenium on mortalities and deformities in the eggs, larvae, and fry of a wild population of cutthroat trout *(Oncorhynchus clarki lewisi)*. *Archives of Environmental Contamination and Toxicology*. 39(1): 46-52

Khan, H.M., and M.A.Q. Khan. 1974. Biological Magnification of Photodieldrin by Food Chain Organisms. Arch. Environ. Contam. Toxicol. 2:289-301.

Khan, A, and P. Thomas. 1998. Estradiol-17 beta and o,p'-DDT stimulate gonadotropin release in Atlantic croaker. Mar. Environ. Res. 46:149-152.

Khan, M.A.K. and F. Wang. 2009. Mercury–selenium compounds and their toxicological significance: toward a molecular understanding of the mercury–selenium antagonism. *Environmental Toxicology and Chemistry*. 28(8): 1567-1577. *http://dx.doi.org/10.1897/08-375.1*

Khan, M.A.Q, R.H. Stanton, D.J. Sutherland, J.D. Rosen, and N. Maitra. 1973. Toxicity-Metabolism Relationship of the Photoisomers of Certain Chlorinated Cyclodiene Insecticide Chemicals. Arch. Environ. Contam. Toxicol. 1:159-169.

Khan, H.M., S. Neudorf, and M.A.Q. Khan. 1975. Absorption and Elimination of Photodieldrin by Daphnia and Goldfish. Bull. Environ. Contam. Toxicol. 13:582-587.

Kidd, K.A. and K. Batchelar. 2011. Mercury. *Fish Physiology: Homeostasis and Toxicology of Non-Essential Metals*. 31(PART B): 237-295. *http://dx.doi.org/10.1016/S1546-5098(11)31027-8*

Kidd, H., and James, D. R., Eds. The Agrochemicals Handbook, Third Edition. Royal Society of Chemistry Information Services, Cambridge, UK, 1991 (as updated).6-10

Kidd, K. A., D.W. Schindler, R. H. Hesslein, and D. C. G. Muir. 1998. Effects of trophic position and lipid on organochlorine concentrations in fishes from subarctic lakes in Yukon Territory. Canadian Journal of Fisheries and Aquatic Sciences 55:869-881.

Kieffer, J.D., A.M. Rossiter, C.A. Kieffer, K. Davidson, and B.L. Tufts. 2002. Physiology and survival of Atlantic salmon following exhaustive exercise in hard and softer water: implications for the catch-and-release sport fishery. *North American Journal of Fisheries Management*. 22(1): 132–144

Kiffney, P.M., and W.H. Clements. 1994. Structural responses of benthic macroinvertebrate communities from different stream orders to zinc. Environ. Toxicol. Chem. 13:389-395.

Kiffney, P.M., and W.H. Clements. 1996. Size-dependent response of macroinvertebrates to metals in experimental streams. Environ. Toxicol. Chem. 15:1352-1356.

Kiffney, P.M. and W.H. Clements. 2002. Ecological effects of metals on benthic invertebrates. Pages 135-154 *in* T. P. Simon, and P. M. Stewart, editors. *Biological Response Signatures: Multimetric index patterns for assessment of freshwater aquatic assemblages*. CRC Press, Boca Raton, FL.

Kilbey, M.M., G.E. Fritchie, and D.M. McLendon. 1972. Phenylalanine Metabolism altered by Dietary Dieldrin. Nature 238:462-465.

Kimball, G.L., L.L. Smith, and S.J. Broderius. 1977. Chronic toxicity of hydrogen cyanide to the bluegill. *Transactions of the American Fisheries Society*. 107(2): 341-345

Kingsbury, P.D. and D.P. Kreutzweiser. 1987. Permethrin treatments in canadian forests. Part 1: Impact on stream fish. *Pesticide Science*. 19(1): 35-48. *http://dx.doi.org/10.1002/ps.2780190106*

Kiser, T., J.A. Hansen, and B.P. Kennedy. 2010. Impacts and pathways of mine contaminants to bull trout (*Salvelinus confluentus*) in an Idaho watershed. *Archives of Environmental Contamination and Toxicology*. 59(2): 301-311. *http://dx.doi.org/10.1007/s00244-009-9457-x*

Koenst, W.M., L.L. Smith, and S.J. Broderius. 1977. Effect of chronic exposure of brook trout to sublethal concentrations of hydrogen cyanide. *Environmental Science and Technology*. 11(9): 883-887

Komadina-Douthwright, S.M., T. Pollock, D. Caissie, R.A. Cunjak, and P. Hardie. 1999. Water quality of Catamaran Brook and the Little Southwest Miramichi River, New Brunswick (1990 -1996). Canadian Data Report of Fisheries and Aquatic Sciences 1051. 32 pp.

Konar, S. 1970. Toxicity of heptachlor to aquatic life. *Journal (Water Pollution Control Federation)*. R299-R303

Korn, S. and, R. Earnest. 1974. Acute toxicity of twenty insecticides to striped bass, *Morone saxtilis*. Calif. Fish Game 60:128-131.

Kovacs, T.G., and G. Leduc. 1982. Sublethal toxicity of cyanide to rainbow trout (*Salmo gairdneri*) at different temperatures. Can. J. Fish. Aquat. Sci. 39:1389-1395.

Kovacs, T.G. and G. Leduc. 1982a. Acute toxicity of cyanide to rainbow trout (*Salmo gairdneri*) acclimated at different temperatures. *Canadian Journal of Fisheries and Aquatic Sciences*. 39(10): 1426-1429

Kovacs, T.G. and G. Leduc. 1982b. Sublethal toxicity of cyanide to rainbow trout (*Salmo gairdneri*) at different temperatures. *Canadian Journal of Fisheries and Aquatic Sciences*. 39(10): 1389-1395

Kramer, J.R., N.W.H. Adams, H. Manolopoulos, and P.V. Collins. 1999. Silver at an old mining camp, Cobalt, Ontario, Canada. *Environmental Toxicology and Chemistry*. 18(1): 23-29. *http://dx.doi.org/10.1002/etc.5620180104*

Kumar, S., and S.C. Pant. 1988. Comparative Sublethal Ovarian Pathology of Some Pesticides in the Teleost, Puntius conchonius Hamilton. Bull. Environ. Contam. Toxicol. 41:227-232.

Kunwar, P.S., C. Tudorachea, M. Eyckmansa, R. Blust, and G. De Boeck. 2009. Influence of food ration, copper exposure and exercise on the energy metabolism of common carp (*Cyprinus carpio*). *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology*. 149(1): 113-119

Labenia, J.S., D.H. Baldwin, B.L. French, J.W. Davis, and N.L. Scholz. 2007. Behavioral impairment and increased predation mortality in cutthroat trout exposed to carbaryl. *Marine Ecology Progress Series*. 329: 1-11

Laetz, C.A., D.H. Baldwin, T.K. Collier, V. Hebert, J.D. Stark, and N.L. Scholz. 2009. The synergistic toxicity of pesticide mixtures: implications for risk assessment and the conservation of endangered Pacific salmon. *Environmental Health Perspectives*. 117(3): 348-353. *http://dx.doi.org/10.1289/ehp.0800096*

Lake Michigan Interstate Pesticide Committee. 1972. An evaluation of DDT and dieldrin in Lake Michigan. EPA-R3-72-003. U.S. Environmental Protection Agency, Cincinnati, Ohio.

Lanno, R.P., and D.G. Dixon. 1996. The comparative chronic toxicity of thiocyanate and cyanide to rainbow trout. Aquat. Toxicol. 36:177-187.

Lauren, D. J., and D. G. McDonald. 1986. Influence of water hardness, pH, and alkalinity on the mechanisms of copper toxicity in juvenile rainbow trout, *Salmo gairdneri*. Can. J. Fish. Aquat. Sci. 43:1488-1496.

Lazorchak, J.M. and M.E. Smith. 2007. Rainbow Trout (*Oncorhynchus mykiss*) and Brook Trout (*Salvelinus fontinalis*) 7-Day Survival and Growth Test Method. *Archives of Environmental Contamination and Toxicology*. 53(3): 397-405. *http://dx.doi.org/10.1007/s00244-006-0227-8*

Leblanc, G.A. 1995. Trophic-level differences in the bioconcentration of chemicals: implications in assessing environmental biomagnification. *Environmental science & technology*. 29(1): 154-160

Leblanc, G.A., J.D. Mastone, A.P. Paradice, B.F. Wilson, H.B. Lockhart, Jr., and K.A. Robillard. 1984. The influence of speciation on the toxicity of silver to fathead minnow *(Pimephales promelas*). *Environmental Toxicology and Chemistry*. 3(1): 37-46. *http://dx.doi.org/10.1002/etc.5620030106*

Leduc, G. 1978. Deleterious Effects of Cyanide on Early Life Stages of Atlantic Salmon (*Salmo salar*). *Journal of the Fisheries Research Board of Canada*. 35: 166-174

Leduc, G. 1984. Cyanides in water: Toxicological significance. P. 153-224 in: Weber, L.J. [Ed.]. Aquatic toxicology, Volume 2. Raven Press, New York, NY.

Lee, D. C., J. R. Sedell, B. E. Rieman, R. F. Thurow, and J. E. Williams. 1997. Broadscale Assessment of Aquatic Species and Habitats. Volume III, Chapter 4. U.S. For. Serv., Gen. Tech. Rep. PNW-GTR-405. Portland, Oregon.

Lehotay, S. J., J. A. Harman-Fetcho, and L. L. McConnell. 1999. Agricultural pesticide residues in oysters and water from two Chesapeake Bay tributaries. Mar. Pollut. Bull. 37:32-44.

LeJeune, K., J. Lipton, W.A. Walsh, D. Cacela, S. Jensen, and W.S. Platts. 1995. Fish population survey, Panther Creek, Idaho. Report by RCG/Hagler Bailly, Boulder, CO to the State of Idaho and National Oceanic and Atmospheric Administration. 214 pp.

Leland, H.V. and J.L. Carter. 1985. Effects of copper on production of periphyton, nitrogen fixation and processing of leaf litter in a Sierra Nevada, California, stream. *Freshwater Biology*. 15(2): 155-173

Leland, H.V., S.V. Fend, T.L. Dudley, and J.L. Carter. 1989. Effects of copper on species composition of benthic insects in a Sierra Nevada, California, stream. *Freshwater Biology*. 21(2): 163-179

Lemke A. E. 1980. Comprehensive Report. Interlaboratory comparison acute testing set. U.S. Environmental Protection Agency, Environ. Res. Lab., Duluth, Minnesota.

Lemly, A.D. 1993a. Guidelines for evaluating selenium data from aquatic monitoring and assessment studies. *Environmental Monitoring and Assessment*. 28: 83-100.

Lemly, A.D. 1993b. Metabolic stress during winter increases the toxicity of selenium to fish. *Aquatic Toxicology*. 27(1-2): 133-158

Lemly, A.D. 1997. Ecosystem recovery following selenium contamination in a freshwater reservoir. *Ecotoxicology and Environmental Safety*. 36(3): 275-281

Lemly, A.D. 2002. Symptoms and implications of selenium toxicity in fish: the Belews Lake case example. *Aquatic Toxicology*. 57(1-2): 39-49. *http://dx.doi.org/10.1016/S0166-445X(01)00264-8*

Lemly, A.D. and J.P. Skorupa. 2007. Technical issues affecting the implementation of US Environmental Protection Agency's proposed fish tissue–based aquatic criterion for selenium. *Integrated Environmental Assessment and Management*. 3(4): 552–558. *http://dx.doi.org/10.1897/IEAM_2007-024.1*

Leonard, A. W., R. V. Hyne, R. P. Lim, and J. C. Chapman. 1999. Effect of Endosulfan Runoff from Cotton Fields on Macroinvertebrates in the Namoi River. Ecotoxicol. Environ. Saf. 42:125-134.

Leth, B., T. Petering, D.T. Vidergar, and P. Kline. 2004. LSRCP [Lower Snake River Compensation Plan] Hatchery Evaluation Studies in Idaho Part 1: Chinook Salmon, 2001 Annual Report, October 1, 2000 to September 30, 2001. Idaho Department of Fish and Game, IDFG Report Number 04-37, Boise, ID. 100 pp. *http://www.fws.gov/lsnakecomplan/Reports/IDFG/Eval/04-37%20Leth%202000-2001%20Chinook.pdf*.

Leung, S-Y.T., R.V. Bulkley, and J.J. Richard. 1981. Persistence of Dieldrin in Water and Channel Catfish from the Des Moines River, Iowa, 1971-73 and 1978. Pestic. Monitor. J. 15:98-102.

Lieb, A.J., D.D. Bills, and R.O Sinnhuber. 1974. Accumulation of dietary polychlorinated biphenyls (Aroclor 1254) by rainbow trout (*Salmo gairdneri*). Jour. Ag. Fd. Chem. 22: 638-642.

Limpert, E., W.A. Stahel, and M. Abbt. 2001. Log-normal distributions across the sciences: Keys and clues. *BioScience*. 51(5): 341–352. *http://dx.doi.org/10.1641/0006-3568(2001)051[0341:LNDATS]2.0.CO;2*

Lipton, J., H. Browning, E. Buccampuso, and K. Kangas. 2004. Evaluation of Low Hardness Extrapolation of Water Quality Criteria. Report to the Idaho Department of Environmental Quality. Stratus Consulting and the Colorado School of Mines. 20 pp.

Little E.E., R.D. Archeski , B.A. Flerov, and V.I Kozlovskaya. 1990. Behavioural indicators of sublethal toxicity in rainbow trout. Archives of Environmental Contamination and Toxicology 19:380-385.

Logerwell, E.A., N. Mantua, P.W. Larson, R.C. Francis, and V.N. Agostini. 2003. Tracking environmental processes in the coastal zone for understanding and predicting Oregon coho (*Oncorhynchus kisutch*) marine survival. *Fisheries Oceanography* 12:554-568.

Long, E.R., D.D. MacDonald, S.L. Smith, F.D. Calder. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. Environ. Manage. 19:81-97.

Long, K.E., E.J. Van Genderen, and S.J. Klaine. 2004. The effects of low hardness and pH on copper toxicity to *Daphnia magna*. *Environmental Toxicology and Chemistry*. 23(1): 72–75

Lorz, H.W. and B.P. McPherson. 1976. Effects of copper or zinc in fresh water on the adaptation to sea water and ATPase activity, and the effects of copper on migratory disposition of the coho salmon (*Oncorhynchus kisutch*). *Journal of the Fisheries Research Board of Canada*. 33(9): 2023-2030

Lorz, H.W. and B.P. McPherson. 1977. Effects of copper and zinc on smoltification of coho salmon. Oregon Department of Fish and Wildlife and U.S. EPA Environmental Research Laboratory., EPA 600/3-77-032, Corvallis, Oreg. 69 pp.

Lotufo, G.R., P.F. Landrum, M.L. Gedeon, E.A. Tigue, and L.R. Herche. 2000. Comparative toxicity and toxicokinetics of DDT and its metabolites in freshwater amphipods. Environ. Toxicol. Chem. 19:368-379.

Lunn, C.R., D.P. Toews, and D.J. Pree. 1976. Effects of three pesticides on respiration, coughing, and heart rates of rainbow trout (Salmo gairdneri Richardson). Can. J. Zool. 54:214-219.

Luoma, S.N. and T.S. Presser. 2009. Emerging opportunities in management of selenium contamination. *Environmental Science and Technology*. DOI 10.1021/es900828h. *http://dx.doi.org/DOI* 10.1021/es900828h

MacDonald, D.D., C.G. Ingersoll, and T.A. Berger. 2000a. Development and evaluation of consensus-based sediment quality guidelines for freshwater ecosystems. *Archives of Environmental Contamination and Toxicology*. 39(1): 20-31. *http://dx.doi.org/10.1007/s002440010075*

MacDonald, D.D., L.M. Dipinto, J. Field, C.G. Ingersoll, E.R. Long, and R.C. Swartz. 2000b. Development and evaluation of consensus-based sediment effect concentrations for polychlorinated biphenyls. Environ. Tox. Chem. 19:1403-1413.

Macek, K.J. 1968. Reproduction in brook trout (Salvelinus fontinalis) fed sublethal concentrations of DDT. Jour. Fish. Res. Board. Can. 25:1787.

Macek, K.J., and W.A. McAllister. 1970. Insecticide susceptibility of some common fish family representatives. Trans Amer. Fish. Soc. 99:20-27.

Macek, K.J., and H.O. Sanders 1970. Biological variation in the susceptibility of fish and aquatic invertebrates to DDT. Trans. Am. Fish. Soc. 99:89.

Macek K. J., C. Hutchinson, and O. B. Cope. 1969. The effects of temperature on the susceptibility of bluegills and rainbow trout to selected pesticides. Bull. Environ. Contam. Toxicol. 4:174-183.

Macek, K.J., C.R. Rodgers, D.L. Stalling, and S. Korn. 1970. The Uptake, Distribution and Elimination of Dietary 14C-DDT and 14C-Dieldrin in Rainbow Trout. Trans. Amer. Fish. Soc. 99:689-695.

Macek, K. J., M. A. Lindberg, S. Sauter, K. S. Buxton, and P. A. Costa. 1976. Toxicity of Four Pesticides to Water Fleas and Fathead Minnows. EPA-600/3-76-099.

Mackay, D. 1982. Correlation of bioconcentration factors. Environ. Sci. Technol. 16:274-278.

Mackay, D., W.Y. Shiu, and K.C. Ma. 1992. Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals. Monoaromatic hydrocarbons, chlorobenzenes, and PCBs, volume 1. Lewis Publs. Boca Raton.

MacRae, R.K., D.E. Smith, N. Swoboda-Colberg, J.S. Meyer, and H.L. Bergman. 1999. Copper binding affinity of rainbow trout (*Oncorhynchus mykiss*) and brook trout (*Salvelinus fontinalis*) gills: implications for assessing bioavailable metal. *Environmental Toxicology and Chemistry*. 18(6): 1180–1189

MacRury, N.K., B.D.S. Graeb, B.M. Johnson, and W.H. Clements. 2002. Comparison of dietary mercury exposure in two sympatric top predator fishes, largemouth bass and northern pike: a bioenergetics modeling approach. *Journal of Aquatic Ecosystem Stress and Recovery*. 9(3): 137-147

Madenjian, C.P., L.J. Schmidt, S.M. Chernyak, R.F. Elliott, T.J. Desorcie, R.T. Quintal, L.J. Begnoche, and R.J. Hesselberg. 1999. Variation in net trophic transfer efficiencies among 21 PCB congeners. Environ. Sci. Technol. 33: 3768-3773.

Maest, A.S., D.J. Beltman, D. Cacela, J. Lipton, and J. Schardt. 1994. 1993 surface water resource injury assessment: Blackbird Mine site NRDA. Prepared by: RCG/Hagler Bailly, Boulder, CO. Submitted to: State of Idaho and National Oceanic and Atmospheric Administration. 338 pp.

Maest, A., D.J. Beltman, D. Cacela, J. Lipton, J. Holmes, K. LeJeune, and T. Podrabsky. 1995. Spring 1994 surface water injury assessment report: Blackbird Mine site NRDA. Submitted by: RCG/Hagler Bailly, Boulder, CO. Submitted to: State of Idaho and National Oceanic and Atmospheric Administration. 214 pp.

Magdza C. H. D. 1983. Toxicity of endosulfan to some aquatic organisms of Southern Africa. Zimbabwe J. Agric. Res. 21: 159-165.

Mager, E.M. 2011. Lead. *Fish Physiology: Homeostasis and Toxicology of Non-Essential Metals*. 31(Part B): 185-236. *http://dx.doi.org/10.1016/S1546-5098(11)31026-6*

Mager, E.M., K.V. Brix, R.M. Gerdes, A.C. Ryan, and M. Grosell. 2011a. Effects of water chemistry on chronic lead toxicity to the cladoceran, *Ceriodaphnia dubia*. *Ecotoxicology and Environmental Safety*. 74(3): 238-243. *http://dx.doi.org/10.1016/j.ecoenv.2010.11.005*

Mager, E.M., A.J. Esbaugh, K.V. Brix, and M. Grosell. 2011b. Influences of water chemistry on the acute toxicity of lead to *Pimephales promelas* and *Ceriodaphnia dubia*. *Comparative Biochemistry and Physiology - C Toxicology and Pharmacology*. 53(1): 82–90. *http://dx.doi.org/doi:10.1016/j.cbpc.2010.09.004*

Majewski H.S., J.F. Klaverkamp, and D.P. Scott. (1978). Acute lethality and sublethal effects of acetone, ethanol, and propylene glycol on the cardiovascular and respiratory system of rainbow trout (*Salmo gairdneri*). Water Research 12: 217-221.

Mallet, J. 1974. Inventory of salmon and steelhead resources, habitats, use and demands. Job performance report. Proj. F-58-R-1. Idaho Department of Fish and Game, Boise, Idaho.

Maltby, L., N. Blake, T.C.M. Brock, and P.J. Van den Brink. 2005. Insecticide species sensitivity distributions: importance of test species selection and relevance to aquatic ecosystems. *Environmental Toxicology and Chemistry*. 24(2): 379-388

Marcelle, C. and J. Thome. 1983. Acute toxicity and bioaccumulation of lindane in gudgeon, Gobio gobio (L.). *Bulletin of environmental contamination and toxicology*. 31(4): 453-458

Maret, T.R. 1995. Mercury in streambed sediment and aquatic biota in the Upper Snake River Basin, Idaho and western Wyoming, 1992. U.S. Geological Survey Water Fact Sheet, Open-File Report, FS-089-95. 2 pp. *http://id.water.usgs.gov/nawqa/factsheets/MARET.089.html*, accessed October 2006.

Maret, T.R. and D.E. MacCoy. 2002. Fish assemblages and environmental variables associated with hard-rock mining in the Coeur d'Alene River basin, Idaho. *Transactions of the American Fisheries Society*. 131(5): 865–884

Maret, T.R. and D.E. MacCoy. 2008. Mercury in Aquatic Ecosystems. *http://id.water.usgs.gov/*.

Maret, T.R., C.T. Robinson, and G.W. Minshall. 1997. Fish assemblages and environmental correlates in least-disturbed streams of the upper Snake River basin. *Transactions of the American Fisheries Society*. 126(2): 200-216. *http://dx.doi.org/doi:* 10.1577/1548-8659(1997)126<0200:FAAECI>2.3.CO;2

Maret, T.R., D.J. Cain, D.E. MacCoy, and T.M. Short. 2003. Response of benthic invertebrate assemblages to metals exposure and bioaccumulation associated with hard-rock mining in northwestern streams, U.S.A. *Journal of the North American Benthological Society*. 22(4): 598-620.

Maret, T.R., J.E. Hortness, and D.S. Ott. 2006. Instream flow characterization of upper Salmon River basin streams, central Idaho, 2004. U.S. Geological Survey, Scientific Investigations Report 06-5230, Boise, Idaho. *http://id.water.usgs.gov/projects/salmon_streamflow/index.html* and *http://pubs.usgs.gov/sir/2006/5230/*.

Markich, S.J., G.E. Batley, J.L. Stauber, N.J. Rogers, S.C. Apte, R.V. Hyne, K.C. Bowles, K.L. Wilde, and N.M. Creighton. 2005. Hardness corrections for copper are inappropriate for protecting sensitive freshwater biota. *Chemosphere*. 60(1): 1-8

Marking, L.L. 1966. Evaluation of p,p=-DDT as a reference toxicant in bioassays. In: Investigations in Fish Control, U.S. Fish. Wildl. Serv. Resour. Publ. U.S. Dept. Inter. 14:10

Marking, L.L. 1985. Toxicity of chemical mixtures. Pages 164-176 *in* G. M. Rand, and S. R. Petrocelli, editors. *Fundamentals of Aquatic Toxicology: Methods and Applications*. Hemisphere Publishing, New York, NY.

Marr, J.C.A., H.L. Bergman, M. Parker, J. Lipton, D. Cacela, W. Erickson, and G.R. Phillips. 1995a. Relative sensitivity of brown and rainbow trout to pulsed exposures of an acutely lethal mixture of metals typical of the Clark Fork River, Montana. *Canadian Journal of Fisheries and Aquatic Sciences*. 52(9): 2005-2015

Marr, J.C., H.L. Bergman, J. Lipton, and C. Hogstrand. 1995*a*. Differences in relative sensitivity of naive and metals-acclimated brown and rainbow trout exposed to metals representative of the Clark Fork River, Montana. Can. J. Fish. Aq. Sci. 32:2016-2030.

Marr, J.C.A., J. Lipton, D. Cacela, M.G. Barron, D.J. Beltman, C. Cors, K. LeJeune, A.S. Maest, T.L. Podrabsky, H.L. Bergman, J.A. Hansen, J.S. Meyer, and R.K. MacRae. 1995b. Fisheries toxicity injury studies, Blackbird Mine site, Idaho. Prepared by RCG/Hagler Bailly and the University of Wyoming for the National Oceanic and Atmospheric Administration, Boulder, CO and Laramie, WY. 125 pp.

Marr, J.C.A., J. Lipton, D. Cacela, J.A. Hansen, H.L. Bergman, J.S. Meyer, and C. Hogstrand. 1996. Relationship between copper exposure duration, tissue copper concentration, and rainbow trout growth. *Aquatic Toxicology*. 36(1): 17-30

Marr, J.C.A., J. Lipton, D. Cacela, J.A. Hansen, J.S. Meyer, and H.L. Bergman. 1999. Bioavailability and acute toxicity of copper to rainbow trout (*Oncorhynchus mykiss*) in the presence of organic acids simulating natural dissolved organic carbon. *Canadian Journal of Fisheries and Aquatic Sciences*. 56(8): 1471-1483

Martin, E.W. 1971. Hazards of Medication: A manual on Drug Interactions, Incompatibilities, Contraindications, and Adverse Effects. Lippincott Press, Philadelphia, PA.

Maser, Chris & James R. Sedell. 1994. From the Forest to the Sea: The Ecology of Wood in Streams, Rivers, Estuaries, and Oceans. St. Lucie Press, Delray Beach, Florida.

Matsumura, F. 1985. Classification of insecticides. Pages 45-109 *in Toxicology of insecticides*. Springer.

Matta, M.B., J. Linse, C. Cairncross, L. Francendese, and R.M. Kocan. 2001. Reproductive and transgenerational effects of methylmercury or Arochlor 1268 on *Fundulus heteroclitus*. *Environmental Toxicology and Chemistry*. 20(2): 327-335

Matthews, G. M. and R. S. Waples. 1991. Status Review for Snake River Spring and Summer Chinook Salmon. NMFS F/NWC-200. Available from NMFS, Northwest Fisheries Science Center, Coastal Zone and Estuaries Studies Division, 2725 Montlake Blvd. E., Seattle, Washington 98112-2097. 75 pages. *http://www.nwfsc.noaa.gov/publications/techmemos/tm200/tm200.htm*

Mauck W.L., P.M. Mehrle, and F.L. Mayer. 1978. Effects of the polychlorinated biphenyl Aroclor 1254 on growth, survival, and bone development in brook trout (*Salvelinus fontinalis*). Jour. Fish. Res. Bd. Can. 35: 1084-1088.

Mayer, F.L.J., and M.R. Ellersieck. 1986. Manual of Acute Toxicity: Interpretation and Data Base for 410 Chemicals and 66 Species of Freshwater Animals Resour. Publ. No. 160, U.S. Dept. Interior, Fish Wildl. Serv., Washington, DC.

Mayer, F.L. Jr., and P.M. Mehrle. 1977. Toxicological aspects of toxaphene in fish: A summary. Transactions of North American Wildlife Natural Resource Conference 42:365-373.

Mayer, F.L., J.C. Street, and J.M. Neuhold. 1972. DDT Intoxication in Rainbow Trout as Affected by Dieldrin. Toxicol. Appl. Pharmacol. 22:347-354.

Mayer, F.L., Jr., P.M. Mehrle, and W.P. Dwyer. 1975. Toxaphene effects on reproduction, growth and mortality of brook trout. U.S. Environmental Protection Agency Report. EPA-600/3-75-013.

Mayer, F.L., P.M. Mehrle, and W.P. Dwyer. 1977a. Toxaphene: chronic toxicity to fathead minnows and channel catfish. U.S. Environmental Protection Agency Report. EPA-600/3-77-069.

Mayer, F.L., P.M. Mehrle, H.O. Sanders. 1977b. Residue dynamics and biological effects of polychlorinated biphenyls in aquatic organisms. Arch. Environ. Contam. Toxicol. 5: 501-511.

McCarthy, M.A., D. Keith, J. Tietjen, M.A. Burgman, M. Maunder, L. Master, B.W. Brook, G. Mace, H.P. Possingham, R. Medellin, S. Andelman, H. Regan, T. Regan, and M.H. Ruckelshaus. 2004. Comparing predictions of extinction risk using models and subjective judgement. *Acta Oecologica*. 26(2): 67-74.

McCarty L.S. (1986). The relationship between aquatic toxicity QSARs and bioconcentration for some organic chemicals. Environmental Toxicology and Chemistry 5:1071-1080.

McClure, M., T. Cooney, and ICTRT. 2005. Updated population delineation in the interior Columbia Basin Memorandum to NMFS NW Regional Office, Co-managers, and other interested parties. May 11. 14 pages.

McElhany, P., M. Ruckleshaus, M.J. Ford, T. Wainwright, and E. Bjorkstedt. 2000. Viable salmon populations and the recovery of evolutionarily significant units. National Marine Fisheries Service, Northwest Fisheries Science Center, NOAA Technical Memorandum NMFS-NWFSC-42. 156 pages. *http://www.nwfsc.noaa.gov/publications/techmemos/tm42/tm42.pdf*

McFadden, J.T. and E.L. Cooper. 1962. An ecological comparison of six populations of brown trout (*Salmo trutta*). *Transactions of the American Fisheries Society*. 91(1): 53-62

McFarland, V.A. 1984. Activity-based evaluation of potential bioaccumulation from sediments. In: R.L. Montgomery and J.W. Leach (eds) Dredging and Dredged Material Disposal. Proceedings of the Conference, Dredging =84 Amer. Soc. of Civil Engineers New York, NY 1:461-466.

McGeachy, S.M and D.G. Dixon 1989. The impact of temperature on the acute toxicity of arsenate and arsenite to rainbow trout (*Salmo gairdneri*). Ecotoxicology and Environmental Safety. 17:86–93

McGeachy, S. M., and D. G. Dixon. 1990. The effect of temperature on the chronic toxicity of arsenate to rainbow trout (*Salmo gairdneri* Richardson). Can. J. Fish. Aq. Sci. 47:2228-2234.

McGeer, J.C., C. Szebedinszky, D.G. McDonald, and C.M. Wood. 2000. Effects of chronic sublethal exposure to water-borne Cu, Cd, or Zn in rainbow trout 2: Tissue specific metal accumulation. Aq. Toxicol. 50:245-256.

McGeer JC, Szebedinszky C, McDonald DG, and Wood CM. 2002. The role of dissolved organic carbon in moderating the bioavailability and toxicity of Cu to rainbow trout during chronic waterborne exposure. *Comp Biochem Physiol C Toxicol Pharmacol* **133**: 147-160.

McGowan, C.P. and M.R. Ryan. 2009. A quantitative framework to evaluate incidental take and endangered species population viability. *Biological Conservation*. 142(12): 3128-3136. *http://dx.doi.org/10.1016/j.biocon.2009.08.012*

McGowan, C.P. and M.R. Ryan. 2010. Arguments for using population models in incidental take assessments for endangered species. *Journal of Fish and Wildlife Management*. 1(2): 183-188. *http://dx.doi.org/10.3996/062010-jfwm-014*

McGrath, C.C. and W.M.J. Lewis. 2007. Competition and predation as mechanisms for displacement of greenback cutthroat trout by brook trout. *Transactions of the American Fisheries Society*. 136(5): 1381–1392

McIntosh, B.A., J.R. Sedell, J.E. Smith, R.C. Wissmar, S.E. Clarke, G.H. Reeves, and L.A. Brown. 1994. Management History of Eastside Ecosystems: Changes in Fish Habitat Over 50 Years, 1935 to 1992. USDA Forest Service, Pacific Northwest Research Station, General Technical Report PNW-GTR-321. February. http://www.fs.fed.us/pnw/publications/gtr321/

McIntyre, J.K. and D.A. Beauchamp. 2005. Age and trophic position dominate bioaccumulation of mercury and organochlorines in the food web of Lake Washington. *Science of the Total Environment*. 371(2-3): 571-584

McIntyre, D.O. and T.K. Linton. 2011. Arsenic. *Fish Physiology: Homeostasis and Toxicology of Non-Essential Metals*. 31(PART B): 297-349. *http://dx.doi.org/10.1016/S1546-5098(11)31028-X*

McIntyre, J.K., D.H. Baldwin, D.A. Beauchamp, and N.L. Scholz. 2012. Low-level copper exposures increase visibility and vulnerability of juvenile coho salmon to cutthroat trout predators. *Ecological Applications*. 22(5): 1460–1471. http://dx.doi.org/10.1890/11-2001.1

McKim, J.M. and D.A. Benoit. 1971. Effects of long-term exposure to copper on survival, growth and reproduction of brook trout (*Salvelinus fontinalis*). *Journal of the Fisheries Research Board of Canada*. 28(5): 655-662

McKim, J.M. and D.A. Benoit. 1974. Duration of toxicity tests for establishing "no effect" concentrations for copper with brook trout (*Salvelinus fontinalis*). *Journal of the Fisheries Research Board of Canada*. 31: 449-452

McKim, J.M., G.F. Olson, G.W. Holcombe, and E.P. Hunt. 1976. Long-term effects of methylmercuric chloride on three generations of brook trout (*Salvelinus fontinalis*): toxicity, accumulation, distribution, and elimination. *Journal of the Fisheries Research Board of Canada*. 33(12): 2726-2739

McKim J.M., P.K. Schmieder, and R.J. Erickson. 1986. Toxicokinetic modeling of [^{14}C] pentachlorophenol in the rainbow trout (*Salmo gairdneri*). Aquatic Toxicology 9:59-80.

McLoughlin, N; Yin, D; Maltby, L; Wood, RM; Yu, H. 2000. Evaluation of sensitivity and specificity of two crustacean biochemical biomarkers. Environ. Toxicol. Chem. 19: 2085-2092.

Meador, J.P. 1991. The interaction of pH, dissolved organic carbon, and total copper in the determination of ionic copper and toxicity. *Aquatic Toxicology*. 19(1): 13-32

Meador, J.P. 2006. Rationale and procedures for using the tissue-residue approach for toxicity assessment and determination of tissue, water, and sediment quality guidelines for aquatic organisms. *Human and Ecological Risk Assessment*. 12(6): 1018-1073

Meador, J.P., T.K. Collier, and J.E. Stein. 2002 Use of tissue and sediment based threshold concentrations of polychlorinated biphenyls (PCBs) to protect juvenile salmonids listed under the Endangered Species Act. Aquatic Conservation: Marine and Freshwater Ecosystems. 12:493-516

Mebane, C.A. 1994. Preliminary Natural Resource Survey - Blackbird Mine, Lemhi County, Idaho. U.S. National Oceanic and Atmospheric Administration, Hazardous Materials Assessment and Response Division, Seattle, WA. 130 pp. *http://profile.usgs.gov/cmebane* [Accessed November 2009].

Mebane, C.A. 2000. Evaluation of proposed new point source discharges to a special resource water and mixing zone determinations: Thompson Creek Mine, upper Salmon River subbasin, Idaho. Idaho Department of Environmental Quality, Boise. 126 pp. *http://deq.idaho.gov/water/data_reports/surface_water/monitoring/mixing_zones.cfm*.

Mebane, C.A. 2001. Testing bioassessment metrics: macroinvertebrate, sculpin, and salmonid responses to stream habitat, sediment, and metals. *Environmental Monitoring and Assessment*. 67(3): 292-322. *http://dx.doi.org/10.1023/A:1006306013724*

Mebane, C.A. 2002a. Effects of metals on freshwater macroinvertebrates: a review and case study of the correspondence between a multimetric index, toxicity testing, and copper concentrations in sediment and water. Pages 281-306 *in* T. P. Simon, editor. *Biological Response Signatures: Indicator Patterns using Aquatic Communities*. CRC Press, Boca Raton, FL.

Mebane, C.A. 2002b. Stream Fish Indexes. Pages 4-1 to 4-65 *in* C. S. Grafe, editor. *Idaho Small Stream Ecological Assessment Framework: an Integrated Approach*. Idaho Department of Environmental Quality, Boise, *http://www.deq.idaho.gov/water-quality/surface-water/monitoring-assessment.aspx*.

Mebane, C.A. 2006. Cadmium risks to freshwater life: derivation and validation of low-effect criteria values using laboratory and field studies. U.S. Geological Survey Scientific Investigation Report 2006-5245 (2010 rev.). 130 pp. *http://pubs.water.usgs.gov/sir20065245/*.

Mebane, C.A. 2010. Relevance of risk predictions derived from a chronic species-sensitivity distribution with cadmium to aquatic populations and ecosystems. *Risk Analysis*. 30(2): 203-223. *http://dx.doi.org/10.1111/j.1539-6924.2009.01275.x*

Mebane, C.A. and D.L. Arthaud. 2010. Extrapolating growth reductions in fish to changes in population extinction risks: copper and Chinook salmon. *Human and Ecological Risk Assessment*. 16(5): 1026-1065. *http://dx.doi.org/10.1080/10807039.2010.512243*

Mebane, C.A., T.R. Maret, and R.M. Hughes. 2003. An index of biological integrity (IBI) for Pacific Northwest rivers. *Transactions of the American Fisheries Society*. 132(2): 239-261. *http://dx.doi.org/10.1577/1548-8659(2003)132<0239:AIOBII>2.0.CO;2*

Mebane, C.A., D.P. Hennessy, and F.S. Dillon. 2008. Developing acute-to-chronic toxicity ratios for lead, cadmium, and zinc using rainbow trout, a mayfly, and a midge. *Water, Air, and Soil Pollution*. 188(1-4): 41-66. *http://dx.doi.org/10.1007/s11270-007-9524-8*

Mebane, C.A., D.P. Hennessy, and F.S. Dillon. 2010. Incubating rainbow trout in soft water increased their later sensitivity to cadmium and zinc. *Water, Air, and Soil Pollution*. 205(1-4): 245-250. *http://dx.doi.org/10.1007/s11270-009-0070-4*

Mebane, C.A., F.S. Dillon, and D.P. Hennessy. 2012. Acute toxicity of cadmium, lead, zinc, and their mixtures to stream-resident fish and invertebrates. *Environmental Toxicology and Chemistry*. 31(6): 1334–1348. *http://dx.doi.org/10.1002/etc.1820*

Meeings, C.C. and R.T. Lackey. 2005. Estimating the size of historic Oregon salmon runs. Reviews in Fisheries Science 13:51-66.

Mehrle, P.M., and R.A Bloomfield. 1974. Ammonia detoxifying mechanisms of rainbow trout altered by dietary dieldrin. Toxicol. Appl Pharmacol. 27:355-365.

Mehrle, P.M., D.L. Stalling, R.A. Bloomfield. 1971. Serum amino acids in rainbow trout (Salmo gairdneri) as affected by DDT and dieldrin. Comp. Biochem. Physiol. 38B:373.

Mendiola, P., F. Mataix, M. Illera, and G. Varela. 1981. [Effect of lindane on protein nutritive utilization in trout (Salmo gairdneri)(author's transl)]. *Revista espanola de fisiologia*. 37(2): 141-146

Mestitzová, M. 1967. On reproduction studies and the occurrence of cataracts in rats after long-term feeding of the insecticide hepatchlor. *Cellular and Molecular Life Sciences*. 23(1): 42-43

Metcalf, R.L., I.P. Kapoor, P-Y. Lu, C.K. Schuth, and P. Sherman. 1973. Model Ecosystem Studies of the Environmental Fate of Six Organochlorine Pesticides. Environ. Health. Perspect. 4:35-44.

Metcalfe, T.L., C.H. Metcalfe, Y. Kiparissis, A.J. Niimi, C. M. Foram, and W.H. Benson. 2000. Gonadal development and endocrine responses in Japanese medaka (Oryzias latipes) exposed to o,p=-DDT in water of through maternal transfer. Environ. Toxicol. Chem. 19:1893-1900.

Meyer, J.S. 1999. A mechanistic explanation for the ln(LC50) vs ln(Hardness) adjustment equation for metals. *Environmental Science and Technology*. 33(6): 908-912. *http://dx.doi.org/10.1021/es980714y*

Meyer, J.S. and W.J. Adams. 2010. Relationship between biotic ligand model-based water quality criteria and avoidance and olfactory responses. *Environmental Toxicology and Chemistry*. 29(9): 2096–2103. *http://dx.doi.org/10.1002/etc.254*

Meyer, J.S., C.J. Boese, and J.M. Morris. 2007a. Use of the biotic ligand model to predict pulse-exposure toxicity of copper to fathead minnows (*Pimephales promelas*). *Aquatic Toxicology*. 84(2): 268-278. *http://dx.doi.org/10.1016/j.aquatox.2006.12.022* |

Meyer, J.S., S.J. Clearwater, T.A. Doser, M.J. Rogaczewski, and J.A. Hansen. 2007b. Effects of Water Chemistry on Bioavailability and Toxicity of Waterborne Cadmium, Copper, Nickel, Lead, and Zinc to Freshwater Organisms. SETAC, Pensacola, Fla. 352 pp

Miles, J. R. W., and C. R. Harris. 1973. Organochlorine insecticide residues in streams draining agricultural, urban-agricultural, and resort areas of Ontario, Canada B 1971. Pestic. Monitor. Jour. 6:363.

Miller, T.G. and W.C. Mackay. 1980. The effects of hardness, alkalinity and pH of test water on the toxicity of copper to rainbow trout (*Salmo gairdneri*). *Water Research*. 14(2): 129-133

Miller, G.W., M.L. Kirby, A.I. Levey, and J.R. Bloomquist. 1999. Heptachlor alters expression and function of dopamine transporters. *NeuroToxicology*. 20(4): 631-637

Miller, L.L., F. Wang, V.P. Palace, and A. Hontela. 2007. Effects of acute and subchronic exposures to waterborne selenite on the physiological stress response and oxidative stress indicators in juvenile rainbow trout. *Aquatic Toxicology*. 83(4): 263-271. *http://dx.doi.org/10.1016/j.aquatox.2007.05.001*

Miyagi, T., K.M. Lam, L.F. Chuang, and R.Y. Chuang. 1998. Suppression of chemokine-induced chemotaxis of monkey neutrophils and monocytes by chlorinated hydrocarbon insecticides. *In Vivo*. 12(5): 441-445

Mok, W.M. and C.N. Wai. 1989. Distribution and mobilization of arsenic species in the creeks around the Blackbird mining district, Idaho. *Water Research*. 23(1): 7-13

Monosson, E. 2000. Reproductive and developmental effects of PCBs in fish: a synthesis of laboratory and field studies. Rev. Toxicol. 3:25-75.

Moore, M.T., D.B. Huggett, W.B. Gillespie Jr, J.H. Rodgers Jr., and C.M. Cooper. 1998, Comparative toxicity of chlordane, chlorpyrifos, and aldicarb to four aquatic testing organisms. Archives of Environmental Contamination and Toxicology 34:152-157.

Moran, P.W., N. Aluru, R.W. Black, and M.M. Vijayan. 2007. Tissue contaminants and associated transcriptional response in trout liver from high elevation lakes of Washington. *Environmental Science and Technology*. 41(18): 6591–6597. *http://dx.doi.org/10.1021/es070550y*

Morgan, T.P., C.M. Guadagnolo, M. Grosell, and C.M. Wood. 2005. Effects of water hardness on toxicological responses to chronic waterborne silver exposure in early life stages of rainbow trout (*Oncorhynchus mykiss*). *Environmental Toxicology and Chemistry*. 24(7): 1642–1647

Moser, V.C., B.M. Cheek, and R.C. MacPhail. 1995. A multidisciplinary approach to toxicological screening: III. Neurobehavioral toxicity. *Journal of Toxicology and Environmental Health*. 45(2): 173-210

Mount, D.I. 1968. Chronic toxicity of copper to fathead minnows (*Pimephales promelas*, rafinesque) *Water Research*. 2(3): 215-223

Mount, D.R. and J.R. Hockett. 2000. Use of toxicity identification evaluation methods to characterize, identify, and confirm hexavalent chromium toxicity in an industrial effluent. *Water Research*. 34(4): 1379-1385

Mount, D.I. and T.J. Norberg-King. 1983. A seven-day life cycle cladoceran toxicity test. *Environmental Toxicology and Chemistr*. 3(3): 425–434

Mount, D.I. and C.E. Stephan. 1969. Chronic toxicity of copper to the fathead minnow (*Pimephales promelas*) in soft water. *Journal of the Fisheries Research Board of Canada*. 26(9): 2449-2457

Mount, D.R., A.K. Barth, T.D. Garrison, K.A. Barten, and J.R. Hockett. 1994. Dietary and water-borne exposure of rainbow trout (*Oncorhynchus mykiss*) to copper, cadmium, lead and zinc using a live diet. Environ. Toxicol. Chem. 13:2031-2041.

Muir, W.D. and T.C. Coley. 1996. Diet of yearling Chinook salmon and feeding success during downstream migration in the Snake and Columbia Rivers. *Northwest Science*. 70(4): 298-305

Muir, D. C. G., B. R. Hobden, and M. R. Servos, MR. 1994. Bioconcentration of pyrethroid insecticides and DDT by rainbow trout: Uptake, depuration, and effect of dissolved organic carbon. Aquat. Toxicol. 29:223-240.

Mullan, J.W., K.R. Williams, G. Rhodus, T.W. Hillman, and J.D. McIntyre. 1992. Production and habitat of salmonids in mid-Columbia River tributary streams. U.S. Fish and Wildlife Service, Monograph I. U.S. Government Printing Office, Washington, D.C. 489 pp.

Munn, M.D., and S.J. Gruber. 1997. The relationship between land use and organochlorine compounds in streambed sediment and fish in the Central Columbia Plateau, Washington and Idaho, USA. Environmental Toxicology and Chemistry 16:1877-1887.

Murphy, P. G. 1970. Effects of salinity on uptake of DDT, DDE, and DDD by fish. Bull. Environ. Contam. Toxicol. 5:404-407.

Murphy, P. G. 1971. The effect of size on the uptake of DDT from water by fish. Bull. Environ. Contam. Toxicol. 6:20-23.

Murty, A. S., and A. P. Devi. 1982. The Effect of Endosulfan and its Isomers on Tissue Protein, Glycogen, and Lipids in the Fish Channa punctata. Pestic. Biochem. Physiol. 17:280-286.

Muscatello, J.R., P.M. Bennett, K.T. Himbeault, A.M. Belknap, and D.M. Janz. 2006. Larval deformities associated with selenium accumulation in northern pike (*Esox lucius*) exposed to metal mining effluent. *Environmental Science and Technology*. 40(20): 6506-6512

Myers, J. M., R. G. Kope, G. J. Bryant, D. Teel, L. J. Lierheimer, T. C. Wainwright, W. S. Grant, F. W. Waknitz, K. Neely, S. T. Lindley, and R. S. Waples. 1998. Status Review of Chinook Salmon From Washington, Idaho, Oregon, and California. NMFS-NWFSC-35. Available from National Marine Fisheries Service, Northwest Fisheries Science Center, Coastal Zone and Estuaries Studies Division, 2725 Montlake Blvd. E., Seattle, Washington 98112-2097. 443 p. *http://www.nwfsc.noaa.gov/publications/techmemos/tm35/index.htm*

Naddy, R.B., W.A. Stubblefield, J.R. May, S.A. Tucker, and J.R. Hockett. 2002. The effect of calcium and magnesium ratios on the toxicity of copper to five aquatic species in freshwater. *Environmental Toxicology and Chemistry*. 21(2): 347–352. *http://dx.doi.org/10.1002/etc.5620210217*

Nagler J.J., P. Aysola, and S.M. Ruby. 1986. Effect of sublethal pentachlorophenol on early oogenesis in maturing female rainbow trout (*Salmo gairdneri*). Archives of Environmental Contamination and Toxicology 15:549-555.

Naik, S., S. Kondekar, and G. Kulkarni. 1997. Heptochlor induced changes in the rate of oxygen consumption and acetylcholinesterase activity in the central nervous system of a freshwater leech Poecilobdella viridis. *Recent advances in biosciences and oceanography.*: 67-74

Naqvi S.M., and C. Vaishnavi. 1993. Bioaccumulative potential and toxicity of Endosulfan insecticide to non-target animals. Comp. Biochem. Physiol. C105:347-361

Narotsky, M.G. and R.J. Kavlock. 1995. A multidisciplinary approach to toxicological screening: II. Developmental toxicity. *Journal of Toxicology and Environmental Health, Part A Current Issues*. 45(2): 145-171

NCASI (National Council of the Paper Industry for Air and Stream Improvement). 1989. Effects of biologically treated bleached kraft mill effluent on cold water stream productivity in experimental stream channels - fifth progress report. National Council of the Paper Industry for Air and Stream Improvement, Inc (NCASI), Technical Bulletin No. 566, Research Triangle Park, N.C. 163 pp.

Nebeker, A.V., and F.A. Puglisi. 1974. Effect of polychlorinated biphenyls (PCB's) on survival and reproduction of *Daphnia, Gammarus,* and *Tanytarsus*. Trans. Amer. Fish. Soc. 103:722-728.

Nebeker, A. V., J. K. McCrady, R. Mshar, and C. K. McAuliffe. 1983. Relative sensitivity of Daphnia magna, rainbow trout and fathead minnows to endosulfan. Environ. Toxicol. Chem. 2:69-72.

Nebeker, A.V., C. Savonen, R.J. Baker, and J.K. McCrady. 1984. Effects of copper, nickel and zinc on the life cycle of the caddisfly *Clistoronia magnifica* (Limnephilidae). *Environmental Toxicology and Chemistry*. 3(4): 645-649. *http://dx.doi.org/10.1002/etc.5620030415*

Nebeker, A.V., C. Savonen, and D.G. Stevens. 1985. Sensitivity of rainbow trout early life stages to nickel chloride *Environmental Toxicology and Chemistry*. 4(2): 233-239. *http://dx.doi.org/10.1002/etc.5620040214*

Nelson, R.L., M. McHenry, and W.S. Platts. 1991. Mining. Pages 425-458 *in* W. R. Meehan, editor. *Influences of forest and rangeland management on salmonid fishes and their habitats, American Fisheries Society Special Publication 19*, Bethesda, MD.

Newman, M.C., D.R. Ownby, L.C.A. Mézin, D.C. Powell, T.R.L. Christensen, S.B. Lerberg, and B.-A. Anderson. 2000. Applying species-sensitivity distributions in ecological risk assessment: assumptions of distribution type and sufficient numbers of species. *Environmental Toxicology and Chemistry*. 19(2): 508-515. *http://dx.doi.org/10.1002/etc.5620190233*

Nicholas, D.R., S. Ramamoorthy, V.P. Palace, S. Spring, J.N. Moore, and R.F. Rosenzweig. 2003. Biogeochemical transformations of arsenic in circumneutral freshwater sediments. *Biodegradation*. 14(1): 123-137

Nichols, J, Bradbury S, and J. Swartout. 1999. Derivation of wildlife values for mercury. Journal of Toxicology and Environmental Health, Part B, 2:325-355.

Niimi, A. J. 1996. PCBs in aquatic organisms. p.117-152 in: W.N. Beyer, G.H. Heinz, and A.W. Redmon-Norwood, [Eds.], Environmental contaminants in wildlife: interpreting tissue concentrations. Lewis Publishers, Boca Raton, FL.

Niimi, A. J., and G.P. Kissoon. 1994. Evaluation of the Critical Body Burden Concept Based on Inorganic and Organic mercury Toxicity to Rainbow Trout (*Oncorhynchus mykiss*). Arch. Environ. Contam. Toxicol. 26:169-178.

Nimick, D.A., D.D. Harper, A.M. Farag, T.E. Cleasby, E. MacConnell, and D. Skaar. 2007. Influence of in-stream diel concentration cycles of dissolved trace metals on acute toxicity to one-year-old cutthroat trout (*Oncorhynchus clarki lewisi*). *Environmental Toxicology and Chemistry*. 26(12): 2667–2678. *http://dx.doi.org/10.1897/07-265.1*

Nimmo, D.R. 1985. Pesticides. Pages 335-373 *in* G. M. Rand, and S. R. Petrocelli, editors. *Fundamentals of Aquatic Toxicology: Methods and Applications*. Hemisphere Publishing, New York, NY.

NIOSH (National Institute for Occupational Safety and Health). 1986. Registry of toxic effects of chemical substances. 1981-1986. Cincinnati, OH.

Niyogi, S. and C.M. Wood. 2004. Biotic Ligand Model, a flexible tool for developing site-specific water quality guidelines for metals. *Environmental Science and Technology*. 38(23): 6177 -6192. *http://dx.doi.org/10.1021/es0496524*

NMFS (National Marine Fisheries Service). 1991. Factors for Decline: A Supplement to the Notice of Determination for Snake River Spring/Summer Chinook Salmon Under The Endangered Species Act. National Marine Fisheries Service, Portland, OR. 75 pp.

NMFS. 2004. Critical habitat for Snake River sockeye salmon, Snake River fall chinook salmon, and Snake River spring/summer chinook salmon. 50 CFR 226.205. *http://ecfr.gpoaccess.gov/cgi/t/text/text-idx?c=ecfr&tpl=/ecfrbrowse/Title50/50cfr226_main_02.tpl* [Accessed November 2011].

NMFS. 2005. Designation of critical habitat for 12 evolutionarily significant units of West Coast salmon and steelhead in Washington, Oregon, and Idaho: Final rule. *Federal Register*. 70(170): 52630-52678

NMFS. 2007. Technical Memorandum: Evaluation of the protectiveness of a site-specific criteria for cobalt in the Panther Creek watershed, Idaho for Chinook Salmon and Steelhead. National Marine Fisheries Service, (*in* Endangered Species Act Section 7 Informal Consultation and Magnuson-Stevens Fishery Conservation and Management Act Essential Fish Habitat Consultation for Remedial Activities for the Blackbird Mine HUC #17060203. March 1, 2007), National Marine Fisheries Service, NMFS Tracking No: 2007/00953, Boise, ID. 28 pp.

NMFS. 2008. Redfish Lake Adult Sockeye Salmon Returns for 2008. Northwest Fisheries Science Center, National Marine Fisheries Service. Available: *http://www.nwfsc.noaa.gov/features/redfish_lake/RedFishLake-sockeye-summary.pdf*. (October 2011).

NMFS. 2010 a. Draft Snake River salmon and steelhead recovery plan – Chapter 8. Idaho State Salmon Recovery Division, Boise, ID.

NMFS. 2010 b. Draft Biological Opinion and Conference Opinion on EPA's Proposed Program of Continuing Approval or Promulgation of New Cyanide Criteria in State and Tribal Water Quality Standards. National Marine Fisheries Service, Office of Protected Resources, Endangered Species Division, Silver Springs, MD. 329 pp.

NMFS. 2011. Draft Snake River Salmon and Steelhead Recovery Plan . Idaho State Salmon Recovery Division, Boise, ID.

NMFS. 2011a. Endangered Species Act Section 7 Informal Consultation and Magnuson-Stevens Fishery Conservation and Management Act Essential Fish Habitat Consultation for the proposed approval of Idaho's Water Quality Criteria for Cadmium. National Marine Fisheries Service, NMFS Tracking No: 2010/04306 (31 January 2011), Boise, ID. 43 pp.

NMFS. 2014. Consultation on Remand for Operation of the Columbia River Power System Northwest Region. Seattle WA, 98115. http://westcoast.fisheries.noaa.gov/publications/hydropower/fcrps/2014_supplemental_fcrps_biop_final.pdf

Norberg-King, T.J. 1989. An evaluation of the fathead minnow seven-day subchronic test for estimating chronic toxicity. *Environmental Toxicology and Chemistry*. 8(11): 1075-1089

Norwood, W.P., U. Borgmann, D.G. Dixon, and A. Wallace. 2003. Effects of metal mixtures on aquatic biota: a review of observations and methods. *Human and Ecological Risk Assessment*. 9(4): 795-811. *http://dx.doi.org/10.1080/713610010*

Norwood, W.P., U. Borgmann, and D.G. Dixon. 2006. Saturation models of arsenic, cobalt, chromium and manganese bioaccumulation by *Hyalella azteca*. *Environmental Pollution*. 143(3): 519-528. *http://dx.doi.org/10.1016/j.envpol.2005.11.041*

Norwood, W.P., U. Borgmann, and D.G. Dixon. 2007. Chronic toxicity of arsenic, cobalt, chromium and manganese to *Hyalella azteca* in relation to exposure and bioaccumulation. *Environmental Pollution*. 147(1): 262-272. *http://dx.doi.org/10.1016/j.envpol.2006.07.017*

Nowak, B. 1992. Histological changes in gills induced by residues of endosulfan. Aquat. Toxicol. 23:65-84.

Nowak, B. 1996. Relationship between endosulfan residue level and ultrastructural changes in the liver of catfish, Tandanus tandanus. Arch. Environ. Contam. Toxicol. 30:195-202.

NRC (National Research Council). 1996. Upstream Salmon and Society in the Pacific Northwest. National Academy Press, Washington, D.C.

NTP (National Toxicology Program). 2001. Ninth Report on Carcinogens. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program.

Nunez, O, J. D. Hendricks, and G. D. Bailey. 1988. Enhancement of aflatoxin B sub(1) and N methyl-N'-nitro-N-nitrosoguanidine hepatocarcinogenesis in rainbow trout (Salmo gairdneri) by 17 beta -estradiol and other organic chemicals. Diseases of Aquatic Organisms 5:185-196.

ODFW (Oregon Department of Fish and Wildlife). 1991. Grande Ronde River subbasin salmon and steelhead plan. Portland, Oregon.

Oduma, J., E. Wango, D. Makawiti, N. Einer-Jensen, and D. Oduor-Okelo. 1995a. Effects of graded doses of the pesticide heptachlor on body weight, mating success, oestrous cycle, gestation length and litter size in laboratory rats. *Comparative Biochemistry and Physiology Part C: Pharmacology, Toxicology and Endocrinology*. 110(2): 221-227

Oduma, J., E. Wango, D. Oduor-Okelo, D. Makawiti, and H. Odongo. 1995b. *In vivo* and i*n vitro* effects of graded doses of the pesticide heptachlor on female sex steroid hormone production in rats. *Comparative Biochemistry and Physiology Part C: Pharmacology, Toxicology and Endocrinology*. 111(2): 191-196

Oeser H., S. Gorbach, and W. Knauf. 1971. Endosulfan and the environment. Giornate Fitopathologiche (Udine) 17 pp.

Oliver, B.G. and A.J. Niimi. 1985. Bioconcentration factors of some halogenated organics for rainbow trout: Limitations in their use for prediction of environmental residues. *Environmental science & technology*. 19(9): 842-849

Oliver, B.G., and A.J. Niimi. 1988. Trophodynamic analysis of polychlorinated biphenyl congeners and other chlorinated hydrocarbons in the Lake Ontario ecosystem. Environ. Sci. Technol. 22:388-397.

Opperhuizen, A., and S.M. Schrap. 1988. Uptake efficiencies of two polychorobiphenyls in fish after dietary exposure to five different concentrations. Chemosphere 17: 253-262.

Orr, P.L., K.R. Guiguer, and C.K. Russel. 2006. Food chain transfer of selenium in lentic and lotic habitats of a western Canadian watershed. *Ecotoxicology and Environmental Safety*. 63(2): 175-188

Ott, D.S. 1997. Selected organic compounds and trace elements in water, bed sediment, and aquatic organisms, Upper Snake River Basin, Idaho and western Wyoming, 1992-94: U.S. Geological Survey Water Open-File Report, OFR 97-18, 100p. (Report online) Open-File Report, OFR 97-18. 100 pp. *http://id.water.usgs.gov/nawqa/reports/ott97/index.html* [Accessed September 9, 2008].

Owen, D.R. 2012. Critical habitat and the challenge of regulating small harms. *Florida Law Review*. 64(1): 141

Palace, V.P., C. Baron, R.E. Evans, J. Holm, S. Kollar, K. Wautier, J. Werner, P. Siwik, G. Sterling, and C.F. Johnson. 2004. An assessment of the potential for selenium to impair reproduction in bull trout, *Salvelinus confluentus*, from an area of active coal mining. *Environmental Biology of Fishes*. 70(2): 169-174.

Palace, V.P., N.M. Halden, P. Yang, R.E. Evans, and G.L. Sterling. 2007. Determining residence patterns of rainbow trout using laser ablation inductively coupled plasma mass spectrometry (LA-ICP-MS) analysis of selenium in otoliths. *Environmental Science and Technology*. 41(10): 3679–3683

Pandey, A. K., K. C. George, and M. P. Mohamed. 1996. Histopathological alterations induced in the liver of an estuarine mullet, Liza parsia by mercuric chloride and DDT. Indian J. Fish. 43:277-284,

Paquin, P.R., J.W. Gorsuch, S. Apte, G.E. Batley, K.C. Bowles, P.G.C. Campbell, C.G. Delos, D.M. Di Toro, F.J. Dwyer, F. Galvez, R.W. Gensemer, G.G. Goss, C. Hogstrand, C.R. Janssen, J.C. McGeer, R.B. Naddy, R.C. Playle, R.C. Santore, U. Schneider, W.A. Stubblefield, C.M. Wood, and K.B. Wu. 2002. The biotic ligand model: a historical overview *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology*. 133(1-2): 3-35. *http://dx.doi.org/10.1016/S1532-0456(02)00112-6*

Parametrix. 2010. Chronic toxicity of lead to the cladoceran, *Ceriodaphnia dubia*, under varying calcium and pH water quality conditions. Report to: International Lead Zinc Research Organization, Durham NC. Parametrix, Corvallis, OR. 30 pp.

Parrish, P.R. et al. 1976. Chlordane: Effects on several estuarine organisms.Journal of Toxicology and Environmental Health. 1: 485-494

Patra, R.W., J.C. Chapman, R.P. Limm, P.C. Gehrke, and R.M. Sunderam. 2009. Effects of temperature on ventilatory behavior of fish exposed to sublethal concentrations of endosulfan and chlorpyrifos. *Environmental Toxicology and Chemistry*. 28(10): 2182-2190. *http://dx.doi.org/10.1897/08-532.1*

Patton, G.W., D.D. Dauble, and C.A. McKinstry. 2007. Evaluation of Early Life Stage Fall Chinook Salmon Exposed to Hexavalent Chromium from a Contaminated Groundwater Source. *Environmental Monitoring and Assessment*. 133(1-3): 285-294

Pauley, G. B., B. M. Bortz, and M. F. Shepard. 1986. Species profiles: life histories and environmental requirements of coastal fishes and invertebrates (Pacific Northwest) – steelhead trout. U.S. Fish and Wildlife Service Biological Report 82(11.62). U.S. Army Corps of Engineers, TR EL-82-4. Available: *http://www.nwrc.usgs.gov/wdb/pub/species_profiles/82_11-062.pdf*. (August 2011).

Pavlou, S., R. Kadeg, A. Turner, and M. Marchlik. 1987. Sediment quality criteria methodology validation: Uncertainty analysis of sediment normalization theory for nonpolar organic contaminants. Work Assignment 45, Task 3, Battelle, Washington, DC.

Pedlar, R.M., M.D. Ptashynski, R. Evans, and J.F. Klaverkamp. 2002. Toxicological effects of dietary arsenic exposure in lake whitefish (*Coregonus clupeaformis*). *Aquatic Toxicology*. 57(3): 167-189

Peither, A., I. Juettner, A. Kettrup, and J-P. Lay. 1996. A pond mesocosm study to determine direct and indirect effects of lindane on a natural zooplankton community. Environ. Pollut. 93:49-56.

Peterson, R.H. 1973. Temperature Selection of Atlantic Salmon (Salmo salar) and Brook Trout (Salvelinus fontinalis) as Influenced by Various Chlorinated Hydrocarbons. J. Fish. Res. Board Can. 30:1091-1097.

Peterson S. M., and G.E. Batley. 1993. The Fate of Endosulfan in Aquatic Ecosystems. Environ. Poll. 82:143-152.

Peterson, S.A., J. Van Sickle, A.T. Herlihy, and R.M. Hughes. 2007. Mercury concentration in fish from streams and rivers throughout the western United States. *Environmental Science and Technology*. 41(1): 58-65

Peterson, S.A., N.V.C. Ralston, D.V. Peck, J. Van Sickle, J.D. Robertson, V.L. Spate, and J.S. Morris. 2009. How might selenium moderate the toxic effects of mercury in stream fish of the western U.S.? *Environmental Science and Technology*. 43(10): 10.1021/es803203g. *http://dx.doi.org/10.1021/es803203g*

PFMC (Pacific Fishery Management Council). 1999. Amendment 14 to the Pacific Coast Salmon Plan. Appendix A: description and identification of essential fish habitat, adverse impacts and recommended conservation measures for salmon. Pacific Fishery Management Council, Portland, Oregon. Available: *http://www.pcouncil.org/wp-content/uploads/99efh1.pdf*. (August 2011).

Phillips, G.R., and D.R. Buhler. 1979. Influences of Dieldrin on the Growth and Body Composition of Fingerling Rainbow Trout (Salmo gairdneri) Fed Oregon Moist Pellets or Tubificid Worms (Tubifex sp.). J. Fish. Res. Board Can. 36:77-80.

Phillips, K. 2003. Cadmium hits trout in the snout. *Journal of Experimental Biology*. 206(11): 1765-1766

Phipps, G.L., V.R. Mattson, and G.T. Ankley. 1995. Relative Sensitivity of Three Freshwater Benthic Macroinvertebrates to Ten Contaminants. Arch. Environ. Contam. Toxicol. 28:281-286.

Pickering, Q.H. and M.H. Gast. 1972. Acute and chronic toxicity of cadmium to the fathead minnow (*Pimephales promelas*). *Journal of the Fisheries Research Board of Canada*. 29(8): 1099-1106

Pilliod, D.S., R.B. Bury, E.J. Hyde, C.A. Pearl, and P.S. Corn. 2003. Fire and amphibians in North America. *Forest ecology and management*. 178(1–2): 163-181. *http://dx.doi.org/10.1016/s0378-1127(03)00060-4*

Pinza, MR; Karle, LM; Mayhew, HL; Word, JQ. 1992. Sampling and analysis of sediments in dredged material from Wilma Uplands Disposal Site. Battelle Pacific Northwest Labs, Richland, WA (USA). Report Number PNL-8300, 25 pp.

Plant, J.A., D.G. Kinniburgh, P.L. Smedley, F.M. Fordyce, and B.A. Klinck. 2007. Arsenic and Selenium. Pages 17-66 *in Treatise on Geochemistry*, volume 9. Elsevier, *http://dx.doi.org/10.1016/B0-08-043751-6/09047-2*.

Playle, R.C. 1998. Modelling metal interactions at fish gills. *Science of the Total Environment*. 219(2-3): 147-163

Playle, R.C. 2004. Using multiple metal–gill binding models and the toxic unit concept to help reconcile multiple-metal toxicity results. *Aquatic Toxicology*. 67(4): 359-370. *http://dx.doi.org/10.1016/j.aquatox.2004.01.017*

Playle, R.C. and C.M. Wood. 1989. Water chemistry changes in the gill micro-environment of rainbow trout: experimental observations and theory. *Journal of Comparative Physiology B: Biochemical, Systemic, and Environmental Physiology*. 159(5): 527-537

Playle, R.C., R.W. Gensemer, and D.G. Dixon. 1992. Copper accumulation on gills of fathead minnows: influence of water hardness, complexation and pH of the gill micro-environment. . *Environmental Toxicology and Chemistry*. 11(3): 381-391

Playle, R.C., D.G. Dixon, and B.K. Burnison. 1993. Copper and cadmium binding to fish gills: modification by dissolved organic carbon and synthetic ligands. *Canadian Journal of Fisheries and Aquatic Sciences*. 50(12): 2667-2677.

Poels, C.L.M., M.A. van Der Gaag, and J.F. J. van de Kerkhoff. 1980. An investigation into the long-term effect of Rhine water on rainbow trout. Water Res. 14:1029-1033.

Ponce R.A., and N. S. Bloom. 1991. Effect of pH on the Bioaccumulation of Low Level, Dissolved Methylmercury by Rainbow Trout (*Oncorhynchus mykiss*). Water, Air, and Soil Pollution. 56:631-640.

Poole, G.C., J.B. Dunham, M.P. Hicks, D.M. Keenan, J.C. Lockwood, E.J. Materna, D.A. McCullough, C.A. Mebane, J.C. Risley, S.T. Sauter, S.A. Spalding, and D.J. Sturdevant. 2001. Technical Synthesis: Scientific Issues Related to Temperature Criteria for Salmon, Trout, and Char Native to the Pacific Northwest. US Environmental Protection Agency., EPA 910-R-01-007, Seattle, WA. 24 pp. *http://yosemite.epa.gov/r10/water.nsf/Water+Quality+Standards/WQS+Temperature+Guidance*.

Porcella, D.B., J.W. Huckabee, and B. Wheatley. 1995. Mercury as a Global Pollutant: Proceedings of the Third International Conference held in Whistler, BC, July 10-14, 1994. Kluwer Academic Publishers, Boston. 1336 pp.

Post, G., and T.R. Schroder. 1971. The toxicity of four insecticides to four salmonid species. Bull. Environ. Contam. Toxicol. 6:144.

Poteat, M.D. and D.B. Buchwalter. 2014. Four reasons why traditional metal toxicity testing with aquatic insects is irrelevant. *Environmental Science & Technology*. 48(2): 887–888

Power, M. and L.S. McCarty. 1997. Fallacies in ecological risk assessment practices. *Environmental Science and Technology*. 31(8): A370-A375 *http://dx.doi.org/10.1007/BF02471945*

Presser, T.S. 1994. The Kesterson Effect. Environmental Management, 18(3):437-454.

Presser, T.S. and S.N. Luoma. 2010. A methodology for ecosystem-scale modeling of selenium. *Integrated Environmental Assessment and Management*. 4(4): 685-710. *http://dx.doi.org/10.1002/ieam.101*

Prothro, M.G. 1993. Office of Water policy and technical guidance on interpretation and implementation of aquatic life metals criteria. U.S. Environmental Protection Agency, Washington, D.C. 52 pp. Accessed July 2006 at *http://dspace.udel.edu:8080/dspace/handle/19716/1556*.

Pyysalo, H., K. Wickstroem, and R. Litmanen. 1981. Contents of Chlordane-, PCB- and DDT Compounds and the Biotransformation Capacity of Fishes in the Lake Area of Eastern Finland. Chemosphere 10:865-876.

Quinn, T.P. 2005. The Behavior and Ecology of Pacific Salmon and Trout. American Fisheries Society and University of Washington, Seattle, Washington, USA. 328 pp

Rader, R.B. 1997. A functional classification of the drift: traits that influence invertebrate availability to salmonids. *Canadian Journal of Fisheries and Aquatic Sciences*. 54(6): 1211-1234. *http://dx.doi.org/10.1139/f97-025*

Radhiah, V., M. Girija, G. Kumari, and K. Rao. 1986. Effects of heptachlor on hydration levels and histosomatic indices in kidney of the fish Tilapia mossambica. *Environment and Ecology*. 4(2): 274-276

Radhaiah, V., M. Girija, and K. Jayantha Rao. 1987. Changes in selected biochemical parameters in the kidney and blood of the fish, Tilapia mossambica (Peters), exposed to heptachlor. *Bulletin of environmental contamination and toxicology*. 39(6): 1006-1011

Rai, P.U., and P. K. Mandal. 1993. Effects of seasonal ambient temperature variations on acute toxicity of chlordane to an air-breathing Indian catfish, Heteropneustes fossilis (Bloch). Bull. Environ. Contam. Toxicol. 51:453-459.

Ralston, N.V.C., J.L. Blackwell, and L.J. Raymond. 2007. Importance of molar ratios in selenium-dependent protection against methylmercury toxicity. *Biological Trace Element Research*. 119(3): 255-268

Ramaneswari, K., and L. M. Rao. 2000. Bioconcentration of endosulfan and monocrotophos by Labeo rohita and Channa punctata. Bull. Environ. Contam. Toxicol. 65:618-22.

Rand, G.M., P.G. Wells, and L.S. McCarty. 1995. Introduction to aquatic toxicology. Pages 3-67 *in* G. M. Rand, editor. *Fundamentals of aquatic toxicology: effects, environmental fate, and risk assessment, second edition*. Taylor and Francis, Washington, D.C.

Rankin, M. G., and D. G. Dixon. 1994. Acute and chronic toxicity of water-borne arsenite to rainbow trout (*Oncorhynchus mykiss*). Can. J. Fish. Aquat. Sci. 51:372-380.

Rao, D. M. 1989. Studies on the relative toxicity and metabolism of Endosulfan to the Indian major Carp Catla catla with special reference to some biochemical changes induced by the pesticide. Pestic. Biochem. Physiol. 33:220-229.

Rao, D. M., and A. S. Murty. 1980. Toxicity, biotransformation and elimination of endosulfan in Anabas testudineus (Bloch). Ind. J. Exp. Biol. 18:664-666.

Rao, D. M. R., A. P. Devi, and A. S. Murty. 1980. Relative toxicity of endosulfan, its isomers, and formulated products to the freshwater fish Labeo rohita. J. Toxicol. Environ. Health 6:825-834.

Rathbun R.E., D.W. Stephens, D.J. Shultz, and D.Y Tai. 1982. Fate of acetone in water. Chemosphere 11:1097-1114.

Ratte, H.T. 1999. Bioaccumulation and toxicity of silver compounds: A review. Environ. Toxicol. Chem. 18:89-108.

Reed-Judkins, D.K., J.L. Farris, D.S. Cherry, A.G. Heath, and J. Cairns, Jr. 1997. Functional responses in *Leptoxis praerosa* to increasing metal concentration and exposure duration. *Environmental Toxicology and Chemistry*. 18(8): 1666-1676

Regetz, J. 2003. Landscape-Level Restraints on Recruitment of Chinook Salmon (*Oncorhynchus tshawytscha*) in the Columbia River Basin, USA. Aquatic Conservation: Marine and Freshwater Ecosystems. 13:35-49.

Reid, S.D. 2011. Molybdenum and Chromium. *Fish Physiology: Homeostasis and Toxicology of Essential Metals*. 31(PART A): 376-415. *http://dx.doi.org/10.1016/S1546-5098(11)31006-0*

Reid, S.D. and D.G. McDonald. 1991. Metal binding activity of the gills of rainbow trout (*Oncorhynchus mykiss*). *Canadian Journal of Fisheries and Aquatic Sciences*. 45(6): 1061–1068.

Reinert, R.E. 1970. Pesticide concentrations in Great Lakes fish. Pestic. Monitor. Jour. 3:233.

Reinert, R.E. 1972. Accumulation of dieldrin in an alga (*Scenedesmus obliquus*), Daphnia magna, and the guppy (*Poecilia reticulata*). J. Fish. Res. Bd. Can. 29:1413-1418.

Reinert, R.E., and L. J. Stone. 1974. Dieldrin and DDT: Accumulation from water and food by lake trout (Salvelinus namaycush) in the laboratory. Proc. 17th Conf. Great. Lakes Res. P. 52.

Reinert, R. E., L. J. Stone, and W. A. Willford. 1974. Effect of temperature on accumulation of methylmercuric chloride and p,p=-DDT by rainbow trout (Salmo gairdneri). J. Fish Res.Board Can. 31:1649.

Reiser, D.W. 1986. Habitat Rehabilitation - Panther Creek, Idaho. Bonneville Power Administration, Division of Fish and Wildlife, BPA Pro. No. 84-29. Report No: DOE/BP/17449-1 NTIS No: DE86015222/HDM, Portland, Oregon. 446 pp.

Rhea, D., A. Farag, D. Harper, E. McConnell, and W. Brumbaugh. 2013. Mercury and Selenium Concentrations in Biofilm, Macroinvertebrates, and Fish Collected in the Yankee Fork of the Salmon River, Idaho, USA, and Their Potential Effects on Fish Health. *Archives of Environmental Contamination and Toxicology*. 64(1): 130-139. *http://dx.doi.org/10.1007/s00244-012-9816-x*

Rhodes, J.J., D.A. McCullough, and F.A. Espinosa, Jr. 1994. A Coarse Screening Process for Potential Application in ESA Consultations. Columbia River Intertribal Fish Commission. Prepared under NMFS/BIA Inter-Agency Agreement 40ABNF3. December.

Riddell, D.J., J.M. Culp, and D.J. Baird. 2005. Sublethal effects of cadmium on prey choice and capture efficiency in juvenile brook trout (*Salvelinus fontinalis*). *Environmental Toxicology and Chemistry*. 24(7): 1751-1758

Rondorf, D.W., G.A. Gray, and R.B. Fairley. 1990. Feeding ecology of subyearling Chinook salmon in riverine and reservoir habitats of the Columbia River. *Transactions of the American Fisheries Society*. 119(1): 16-24

Rozados, M.V., M.D. Andres, and M.A. Aldegunde. 1991. Preliminary studies on the acute effect of lindane (γ-HCH) on brain serotoninergic system in rainbow trout Oncorhynchus mykiss. *Aquatic Toxicology*. 19(1): 33-40. http://dx.doi.org/10.1016/0166-445X(91)90026-6

Rudolph, B.-L., I. Andreller, and C.J. Kennedy. 2008. Reproductive success, early life stage development and survival of westslope cutthroat trout (*Oncorhynchus clarki lewisi*) exposed to elevated selenium in an area of active coal mining. *Environmental Science and Technology*. 42(8): 3109-3114

Sabaliunas, D., J. Lazutka, I. Sabaliuniene, and A. Soedergren. 1998. Use of semipermeable membrane devices for studying effects of organic pollutants: Comparison of pesticide uptake by semipermeable membrane devices and mussels. Environ. Toxicol. Chem. 17:1815-1824.

Safe, S.H. 1994. Polychlorinated biphenyls (PCBs): environmental impact, biochemical and toxic responses, and implications for risk assessment.Crit Rev Toxicol 24:87-149.

Sagar, P.M. and G.J. Glova. 1987. Prey preferences of a riverine population of juvenile Chinook salmon, *Oncorhynchus tshawytscha*. *Journal of Fish Biology*. 31(4): 661-673

Sagar, P.M. and G.J. Glova. 1988. Diel feeding periodicity, daily ration and prey selection of a riverine population of juvenile chinook salmon, *Oncorhynchus tshawytscha* (Walbaum). *Journal of Fish Biology*. 33(4): 643-653

Saiki, M.K., D.T. Castleberry, T.W. May, B.A. Martin, and F.N. Bullard. 1995. Copper, cadmium, and zinc concentrations in aquatic food chains from the upper Sacramento River (California) and selected tributaries. Archives of Environmental Contamination and Toxicology 29:484-491.

Sandahl, J.F., D.H. Baldwin, J.J. Jenkins, and N.L. Scholz. 2007. A sensory system at the interface between urban stormwater runoff and salmon survival. *Environmental Science and Technology*. 41(8): 2998–3004. *http://dx.doi.org/10.1021/es062287r*

Sanders, H. O. 1969. Toxicity of pesticides to the crustacean Gammarus lacustris. U.S. Bur. Sport Fish. Wildl Tech. Pap. 25.

Sanders, H.O. 1972. Toxicity of some insecticides to four species of malacostracan crustaceans. Bur. Sport Fish. Wildl. Tech. Pap. No. 66.

Sanders, H.O. 1980. Sub-Lethal Effects of Toxaphene on Daphnids, Scuds, and Midges. EPA-600/3-80-006, Fish-Pesticide Res. Lab., Fish Wildl. Serv., U.S.D.I., Columbia, MO

Sanders, H.O., and O.B. Cope. 1966. Toxicities of several pesticides to two species of cladocerans. Trans. Am. Fish Soc. 95:165.

Sanders, H.O., and O.B. Cope. 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnol. and Oceanog. 13: 112-117.

Sandheinrich, M.B. and K.M. Miller. 2006. Effects of dietary methylmercury on reproductive behavior of fathead minnows (*Pimephales promelas*). *Environmental Toxicology and Chemistry*. 25(11): 3053–3057

Sandheinrich, M.B. and J.G. Wiener. 2010. Methylmercury in Freshwater Fish: Recent Advances in Assessing Toxicity of Environmentally Relevant Exposures. *in* W. N. Beyer, and J. P. Meador, editors. *Environmental Contaminants in Biota: Interpreting Tissue Concentrations, 2nd edition*. CRC Press, Boca Raton, FL.

Santharam, K.R., B. Thayumanavan, and S. Krishnaswamy. 1976. Toxicity of some insecticides to *Daphnia carinata* King, an important link in the food chain in the freshwater ecosystems. Ind. Jour. Ecol. 3:70-73.

Santore, R.C., P.R. Paquin, D.M. Di Toro, H.E. Allen, and J.S. Meyer. 2001. Biotic ligand model of the acute toxicity of metals. 2. Application to acute copper toxicity in freshwater fish and *Daphnia*. *Environmental Toxicology and Chemistry*. 20(10): 2397-2402. *http://dx.doi.org/10.1002/etc.5620201035*

Sastry, R. V., and A. A. Siddiqui. 1982. Effect of Endosulfan and Quinalphos on Intestinal Absorption of Glucose in the Freshwater Murrel, Channa punctatus. Toxicol. Lett. 12:289 293.

Saunders, R.L. and J.B. Sprague. 1967. Effects of copper-zinc mining pollution on a spawning migration of Atlantic salmon. *Water Research*. 1(6): 419-432. *http://dx.doi.org/10.1016/0043-1354(67)90051-6*

Sauter, S., K.S. Buxton, K.J. Macek, and S.R. Petrocelli. 1976. Effects of exposure to heavy metals on selected freshwater fish: toxicity of copper, cadmium, chromium and lead to eggs and fry of seven fish species. U.S. Environmental Protection Agency, EPA-600/3-76-105, Duluth, Minnesota. 85 pp.

Schaumberg, F.D. et al. 1967. A method to evaluate the effects of water pollutants on fish respiration Water Res. 1:731.(Co-authors needed from Science Center)

Scheuerell, M.D., and J.G. Williams. 2005. Forecasting climate-induced changes in the survival of Snake River spring/summer Chinook salmon (*Oncorhynchus tshawytscha*). Fisheries Oceanography 14:448-457.

Scheuhammer A.M. 1987. The chronic toxicity of mercury, cadmium, and lead in birds: a review. Environ. Pollut., 46, 263-95.

Scheuhammer, A.M., M.W. Meyer, M.B. Sandheinrich, and M.W. Murray. 2007. Effects of environmental methylmercury on the health of wild birds, mammals, and fish. *Ambio*. 36(1): 12–19

Schimmel, S.C., J.M. Patrick, Jr., and J. Forester. 1976. Heptachlor: toxicity to and uptake by several estuarine organisms. Jour. Toxicol. Environ. Health 1:955-965.

Schimmel, S.C., J.M. Patrick, Jr., and J. Forester. 1977. Uptake and toxicity of toxaphene in several estuarine organisms. Arch. Environ. Contam. Toxicol. 5:353-367.

Schlekat, C.E., K.A. Kidd, W.J. Adams, D.J. Baird, A.M. Farag, L. Maltby, and A.R. Stewart. 2005. Toxicity of dietborne metals: field studies. Pages 113-152 *in* J. S. Meyer, W. J. Adams, K. V. Brix, S. N. Luoma, D. R. Mount, W. A. Stubblefield, and C. M. Wood, editors. *Toxicity of dietborne metals to aquatic organisms*. Society of Environmental Toxicology and Chemisty (SETAC), Pensacola, Fla.

Schnoor, J.L. 1981. Fate and Transport of Dieldrin in Coralville Reservoir: Residues in Fish and Water Following a Pesticide Ban. Science 211:840-842.

Schoettger, R.A. 1970. Fish-Pesticide Research Laboratory. U.S. Dept. Interior, Bur. Sport Fish. Wildl. Res., Publ. 106:2-40

Scholz, N.L., N.K. Truelove, J.S. Labenia, D.H. Baldwin, and T.K. Collier. 2006. Dose-additive inhibition of chinook salmon acetylcholinesterase activity by mixtures of organophosphate and carbamate insecticides. *Environmental Toxicology and Chemistry*. 25(5): 1200-1207

Schultz, R. and R. Hermanutz. 1990. Transfer of toxic concentrations of selenium from parent to progeny in the fathead minnow (*Pimephales promelas*). *Bulletin of Environmental Contamination and Toxicology*. 45(4): 568-573

Schulz, R. and Liess M. 1995. Chronic effects of low insecticide concentrations on freshwater caddisfly larvae. Hydrobiologia 299:101-113

Sciera, K.L., J.J. Isely, J.R. Tomasso, Jr., and S.J. Klaine. 2004. Influence of multiple water-quality characteristics on copper toxicity to fathead minnows (*Pimephales promelas*). *Environmental Toxicology and Chemistry*. 23(12): 2900–2905. *http://dx.doi.org/10.1897/03-574.1*

Scott, G.R., K.A. Sloman, C. Rouleau, and C.M. Wood. 2003. Cadmium disrupts behavioural and physiological responses to alarm substance in juvenile rainbow trout (*Oncorhynchus mykiss*). *Journal of Experimental Biology*. 206(11): 1779-1790

Scudder, B.C., L.C. Chasar, D.A. Wentz, N.J. Bauch, M.E. Brigham, P.W. Moran, and D.P. Krabbenhoft. 2009. Mercury in fish, bed sediment, and water from streams across the United States, 1998–2005. U.S. Geological Survey, Scientific Investigations Report 2009–5109. 86 pp. *http://pubs.usgs.gov/sir/2009/5109/* [Accessed 19 August 2009].

Sedell, J.R., Froggatt, J.L. 2000. Importance of Streamside Forests to Large Rivers: The Isolation of the Willamette River, Oregon, U.S.A., from its Floodplain by Snagging and Streamside Forest Removal. Verhandlung Internationale Vereinigung Limnologie Vol. 22, No. 3, p 1828-1834, December, 1984. 2 Fig, 1 Tab, 19 Ref. NSF grant DEB-8112455.

Seim, W.K., L.R. Curtis, S.W. Glenn, and G.A. Chapman. 1984. Growth and survival of developing steelhead trout (*Salmo gairdneri*) continuously or intermittently exposed to copper. *Canadian Journal of Fisheries and Aquatic Sciences*. 41(3): 433-438. *http://dx.doi.org/10.1139/f84-051*

Shannon, L.R. 1977a. Accumulation and Elimination of Dieldrin in Muscle Tissue of Channel Catfish. Bull. Environ. Contam. Toxicol. 17:637-644.

Shannon, L.R. 1977b. Equilibrium Between Uptake and Elimination of Dieldrin by Channel Catfish, Ictalurus punctatus. Bull. Environ. Contamin. Toxicol. 17:278-284.

Shiller, A.M. and E.A. Boyle. 1987. Variability of dissolved trace metals in the Mississippi River. *Geochimica et Cosmochimica Acta*. 51(12): 3273-3277

Shubat, P.J., and L.R. Curtis. 1986. Ration and toxicant preexposure influence dieldrin accumulation by rainbow trout (Salmo gairdneri). Environ. Toxicol. Chem. 5:69-77.

Singh, H., and T.P. Singh. 1980. Short-term effect of two pesticides on the survival, ovarian 32P uptake and gonadotrophic potency in a freshwater catfish, Heteropneustes fossilis (Bloch) J. Endocrinol., 85:193-199.

Singh, N.N., and A.K. Srivastava. 1992. Effect of aldrin on some biochemical parameters of Indian catfish, Heteropneustes fossilis. J. Freshwat. Biol. 4:289-293.

Singh, N.N., V.K. Das, S. Singh. 1996. Effect of Aldrin on Carbohydrate, Protein, and Ionic metabolism of a Freshwater Catfish, Heteropneustes fossilis. Bull. Environ. Contam. Toxicol. 57:204-210.

Skorupa, J.P., T.S. Presser, S.J. Hamilton, A.D. Lemly, and B.E. Sample. 2004. EPA's Draft Tissue-Based Selenium Criterion: A Technical Review. U.S. Fish and Wildlife Service, U.S. Geological Survey, U.S. Forest Service and CH2M Hill, "White paper". 35 pp. *http://wwwrcamnl.wr.usgs.gov/Selenium/Library_articles/joewhite.pdf*.

Smith, A. G. 1991. Chlorinated Hydrocarbon Insecticides. In Handbook of Pesticide Toxicology. Hayes, W. J., Jr.and Laws, E. R., Jr., Eds. Academic Press Inc., New York, NY, 1991.

Smith, E.P. and J. Cairns, Jr. 1993. Extrapolation methods for setting ecological standards for water quality: statistical and ecological concerns. *Ecotoxicology*. 2(3): 203-219

Smith, L.L., S.J. Broderius, D.M. Oseid, G.L. Kimball, and W.M. Koenst. 1978. Acute toxicity of hydrogen cyanide to freshwater fishes. *Archives of Environmental Contamination and Toxicology*. 7(1): 325-337

Smith, M., D. Hawkins, L. Denny, and K. Tardy. 2011. Genetic analysis of the origin of Chinook salmon in Panther Creek, Idaho. U.S. Fish and Wildlife Service, Abernathy Fish Technology Center and Shoshone-Bannock Tribes, Fish and Wildlife Department, Longview, WA and Fort Hall, ID. 26 pp.

Soengas, J.L., E.F. Strong, M. Aldegunde, and M.D. Andrés. 1997. Effects of an acute exposure to lindane (γ-hexachlorocyclohexane) on brain and liver carbohydrate metabolism of rainbow trout. *Ecotoxicology and Environmental Safety*. 38(2): 99-107. http://dx.doi.org/10.1006/eesa.1997.1559

Sorensen, E.M.B. 1991. Metal poisoning in fish. CRC Press, Boca Raton, Florida. 374 pp

Spehar, R.L. and J.T. Fiandt. 1986. Acute and chronic effects of water quality criteria-based metal mixtures on three aquatic species. *Environmental Toxicology and Chemistry*. 5(10): 917-931

Spehar, R.L., E.N. Leonard, and D.L. DeFoe. 1978. Chronic effects of cadmium and zinc mixtures on flagfish (*Jordanella floridae*). *Transactions of the American Fisheries Society*. 107(2): 354-360

Spehar R.L., H.P. Nelson, M.J. Swanson, and J.W. Renoos. 1985. Pentachlorophenol toxicity to amphipods and fathead minnows at different test pH values. Environmental Toxicology and Chemistry 4:389-397.

Spence, B.C, G.A. Lomnicky, R.M. Hughes, R.P. Novitzki. 1996. An Ecosystem Approach to Salmonid Conservation. TR-4501-96-6057. ManTech Environmental Research Services Corp., Corvallis, Oregon. (December 1996). http://www.nwr.noaa.gov/1habcon/habweb/habguide/ManTech/front.htm

Sprague, J. B. 1968. Avoidance reactions of rainbow trout to zinc sulphate solutions. Water Res. 2:367-372.

Sprague, J.B. 1970. Measurement of pollutant toxicity to fish. II. Utilizing and applying bioassay results. *Water Research*. 4(1): 3-32. *http://dx.doi.org/10.1016/0043-1354(70)90018-7*

Sprague, J.B. 1985. Factors that modify toxicity. Pages 124-163 *in* G. M. Rand, and S. R. Petrocelli, editors. *Fundamentals of Aquatic Toxicology: Methods and Applications*. Hemisphere Publishing, New York, NY.

Sprague, J.B., P.F. Elson, and R.L. Saunders. 1965. Sublethal copper-zinc pollution in a salmon river - A field and laboratory study. *International Journal of Air and Water Pollution*. 9: 531-543

Spromberg, J.A. and N.L. Scholz. 2011. Estimating the future decline of wild coho salmon populations resulting from early spawner die-offs in urbanizing watersheds of the Pacific Northwest, USA. *Integrated Environmental Assessment and Management*. 24(4): 648–656. *http://dx.doi.org/10.1002/ieam.219*

SRI (Stanford Research Institute). 1997. Directory of chemical producers, United States, 1997. Stanford Research Institute, Menlo Park, CA.

Stantec. 2004. Biomonitoring study, Panther Creek watershed, September 2003. Prepared by Stantec Consulting, Ltd for the Blackbird Mine Site Group, Salmon, Idaho, Brampton, Ontario. 425 pp.

Statham, C.N., and J.J. Lech. 1975. Potentiation of the Acute Toxicity of Several Pesticides and Herbicides in Trout by Carbaryl. Toxicol. Appl. Pharmacol. 34:83-87.

Steinhart, G.B. and W.A. Wurtsbaugh. 2003. Winter ecology of kokanee: implications for salmon management. *Transactions of the American Fisheries Society*. 132(6): 1076–1088

Stelzer A, Chan HM. 1999. The relative estrogenic activity of technical toxaphene mixture and two individual congeners. Toxicology 138(2):69-80

Stephan, C.E. 1985. Are the 'Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Life and its Uses' based on sound judgments? Pages 515-526 *in* R. C. Bahner, and D. J. Hansen, editors. *Aquatic Toxicology and Hazard Assessment: Seventh Symposium: ASTM STP 891*. American Society for Testing and Materials (ASTM), Philadelphia, PA.

Stephan, C.E. 1986. Proposed goal of applied aquatic toxicology. Pages 3-10 *in Aquatic Toxicology and Hazard Assessment: Ninth Volume, ASTM Special Technical Publication 921*. American Society for Testing and Materials (ASTM), Philadelphia, PA.

Stephan, C.E. 1995. Derivation of conversion factors for the calculation of dissolved freshwater aquatic life criteria for metals. U.S. Environmental Protection Agency, Environmental Research Laboratory - Duluth.

Stephan, C.E. 2002. Use of species sensitivity distributions in the derivation of water quality criteria for aquatic life by the U.S. Environmental Protection Agency. Pages 211-220 *in* L. Posthuma, G. W. Suter, II, and T. P. Traas, editors. *Species sensitivity distributions in ecotoxicology*. CRC Press, Boca Raton, Florida.

Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W.A. Brungs. 1985. Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses. U.S. Environmental Protection Agency, EPA 822-R-85-100, NTIS PB85 227049, Duluth, Narragansett, and Corvallis. 98 pp. *http://epa.gov/waterscience/criteria/aqlife.html*.

Stephan, C.E., W.H. Peltier, D.J. Hansen, C.G. Delos, and G.A. Chapman. 1994a. Guidance concerning the use of "clean techniques" and QA/QC when measuring trace metals Pages 98-108 (Appendix C) *in Interim guidance on determination and use of water-effect ratios for metals*. U.S. Environmental Protection Agency, Washington, DC.

Stephan, C.E., W.H. Peltier, D.J. Hansen, C.G. Delos, and G.A. Chapman. 1994b. Interim guidance on determination and use of water-effect ratios for metals. U.S. Environmental Protection Agency, EPA-823-B-94-001, Washington, DC.

Stephenson, RR. 1983. Effects of water hardness, water temperature, and size of the test organism on the susceptibility of the freshwater shrimp, *Gammarus pulex* (L.) to toxicants. Bull. Environ. Contam. Toxicol. 31:459B466.

Stevens, D.G. 1977. Survival and immune response of coho salmon exposed to copper. U.S. EPA Environmental Research Laboratory, EPA 600/3-77-031, Corvallis.

Stevens, D.G. and G.A. Chapman. 1984. Toxicity of trivalent chromium to early life stages of steelhead trout. *Environmental Toxicology and Chemistry*. 3(1): 125-133

Stewart, A.R., M. Grosell, D.B. Buchwalter, N.S. Fisher, S.N. Luoma, T. Mathews, P.L. Orr, and W.-X. Wang. 2010. Bioaccumulation and trophic transfer of selenium. Pages 93-139 *in* P. M. Chapman, W. J. Adams, M. L. Brooks, C. G. Delos, S. N. Luoma, W. A. Maher, H. M. Ohlendorf, T. S. Presser, and D. P. Shaw, editors. *Ecological Assessment of Selenium in the Aquatic Environment*. CRC Press, Pensacola, Florida.

Stratus. 1996. Preliminary toxicological evaluation, U.S. v. Iron Mountain Mines, Inc. Stratus Consulting, Inc. (formerly Hagler Bailly Services), Boulder, Colo.

Stratus. 1998. Data report: Acute copper toxicity to salmonids in surface waters in the vicinity of the Iron Mountain Mine, California. Stratus Consulting, Inc. (formerly Hagler Bailly Services), Boulder, Colo.

Stratus. 1999. Sensitivity of bull trout *(Salvelinus confluentus)* to cadmium and zinc in water characteristic of the Coeur d'Alene River Basin. Report to US EPA Region 10, Seattle, WA. Stratus Consulting, Boulder, CO. 94 pp.

Stubblefield, W.A., B.L. Steadman, T.W. La Point, and H.L. Bergman. 1999. Acclimation-induced changes in the toxicity of zinc and cadmium to rainbow trout. *Environmental Toxicology and Chemistry*. 18(12): 2875–2881. *http://dx.doi.org/10.1002/etc.5620181231*

Suchanek, T.H., C.A. Eagles-Smith, D.G. Slotton, E.J. Harner, and D.P. Adam. 2008. Mercury in abiotic matrices of Clear Lake, California: human health and ecotoxicological implications. *Ecological Applications*. 18(sp8): A128-A157. *http://dx.doi.org/doi:10.1890/06-1477.1*

Suedel BC, Boraczek JA, Peddicord RK, Clifford PA, Dillon TM. 1994. Trophic transfer and biomagnification potential of contaminants in aquatic ecosystems. Rev Environ Contam Toxicol 136:21-89

Sunderam, R.I.M, D.M.H. Cheng, and G.B. Thompson. 1992. Toxicity of endosulfan to native and introduced fish in Australia. Environ. Toxicol. Chem. 11:1469-1476.

Sunderam, R.I.M., G.B. Thompson, J.C. Chapman, and D.M.H. Cheng. 1994 Acute and chronic toxicity of endosulfan to two Australian cladocerans and their applicability in deriving water quality criteria. Arch. Environ. Contam. Toxicol. 27:541-545.

Supamataya, M. 1988. The study of diseases in sand goby (Oxyeleotris marmoratus Bleeker) in cage culture and some environmental factors related to infection. Abstracts of Master of Science Theses (Fisheries Science)., 1988, P. 3, Notes Fac. Fish. Kasetsart Univ., Bangkok., No. 13.

Suter, G.W., II. 1990. Seven day tests and chronic tests. *Environmental Toxicology and Chemistry*. 9(12): 1435-1436

Suter, G.W., II, A.E. Rosen, E. Linder, and D.F. Parkhurst. 1987. Endpoints for responses of fish to chronic toxic exposures. *Environmental Toxicology and Chemistry*. 6(10): 793-809. *http://dx.doi.org/*

Suter, G.W., II, T.P. Traas, and L. Posthuma. 2002. Issues and practices in the derivation and use of species sensitivity distributions. Pages 437-474 *in* L. Posthuma, G. W. Suter, II, and T. P. Traas, editors. *Species Sensitivity Distributions in Ecotoxicology*. CRC Press, Boca Raton, Florida.

Suttle, K.B., M.E. Power, J.M. Levine, and C. McNeely. 2004. How fine sediment in riverbeds impairs growth and survival of juvenile salmonids. *Ecological Applications*. 14(4): 969–974. *http://dx.doi.org/10.1890/03-5190*

Swackhamer, D. L., and R. A. Hites. 1988. Occurrence and bioaccumulation of organochlorine compounds in fishes from Siskiwit Lake, Isle Royale, Lake Superior. Environmental Science and Technology 22:543-548.

Swift, M.C. 2002. Stream ecosystem response to, and recovery from, experimental exposure to selenium. *Journal of Aquatic Ecosystem Stress and Recovery*. 9(3): 159 - 184. *http://dx.doi.org/10.1023/A:1021299003516*

Syrjänen, J., K. Korsu, P. Louhi, R. Paavola, and T. Muotka. 2011. Stream salmonids as opportunistic foragers: the importance of terrestrial invertebrates along a stream-size gradient. *Canadian Journal of Fisheries and Aquatic Sciences*. 68(12): 2146-2156. *http://dx.doi.org/10.1139/f2011-118*

Taylor, EJ, Maund, SJ, and Pascoe, D. 1991. Toxicity of four common pollutants to freshwater macroinvertebrates *Chironomus ripartus* Meigen (Insecta:Diptera) and *Gammarus pulex* L. (Crustacea:Amphipoda). Arch. Environ. Contam. Toxicol. 21:371-376.

Taylor, E.J., K.M. Underhill, S.J. Blockwell, and D. Pascoe. 1998. Haem biosynthesis in the freshwater macroinvertebrate Gammarus pulex (L.): effects of copper and lindane. Water Res. 32:2202-2204.

Taylor, L.N., J.C. McGeer, C.M. Wood, and D.G. McDonald. 2000. Physiological effects of chronic copper exposure to rainbow trout (*Oncorhynchus mykiss*) in hard and soft water: evaluation of chronic indicators. *Environmental Toxicology and Chemistry*. 19(9): 2298–2308. *http://dx.doi.org/10.1002/etc.5620190920*

Taylor, L.N., C.M. Wood, and D.G. McDonald. 2003. An evaluation of sodium loss and gill metal binding properties in rainbow trout and yellow perch to explain species differences in copper tolerance. *Environmental Toxicology and Chemistry*. 23(9): 2159–2166. *http://dx.doi.org/10.1897/02-256*

Terriere, L.C., U. Kiigermagi, A.R. Gerlach, and R.L. Borovicka. 1966. The persistence of toxaphene in lake water and its uptake by aquatic plants and animals. J. Agric. Food Chem. 14: 66-69.

Teuscher, D.M. 2004. Review of potential interactions between stocked rainbow trout and listed Snake River sockeye salmon in Pettit Lake, Idaho. Pages 24-36 *in Snake River sockeye salmon habitat and limnological research, Annual Report 1995*. Prepared by the Shoshone Bannock Tribes for the U.S. Department of Energy, Bonneville Power Administration., Portland, Oregon, *http://www.efw.bpa.gov/publications/H22548-4.pdf*.

Thomas, S.A., T.V. Royer, G.W. Minshall, and E. Snyder. 2003. Assessing the historic contribution of marine-derived nutrients to Idaho streams. Pages 41-55 *in* J. G. Stockner, editor. *Nutrients in Salmonid Ecosystems: Sustaining Production and biodiversity. American Fisheries Society Symposium 34*, volume 34, Bethesda, MD USA.

Thompson, R. N., J. B. Haas, L. M. Woodall, and E. K. Holmberg. 1958. Results of tagging program to enumerate the numbers and to determine the seasonal occurrence of anadromous fish in the Snake River and its tributaries. Final report. Contract DA-35-026-eng-20609, Fish Commission of Oregon, Clackamas, Oregon.

Thurow, R. 1987. Evaluation of the South Fork Salmon River steelhead trout fishery restoration program. Lower Snake River Fish and Wildlife Compensation Plan. Job Completion Report, Contract No. 14-16-0001-86505, Idaho Department of Fish and Game, Boise, Idaho.

Tippets, W.E. and P.B. Moyle. 1978. Epibenthic Feeding by Rainbow Trout (Salmo gairdneri) in the McCloud River, California *Journal of Animal Ecology*. 47(2): 549-559

Todd, S.L. 2008. Trophic Biomass and Abundance Relationships in Streams of the Panther Creek Watershed, Idaho: Implications for Drift-Feeding Fishes. M.Sc. Idaho State University, Pocatello.

Tooby, T.E. et al. 1975. The acute toxicity of 102 pesticides and miscellaneous substances to fish. Chem. Ind. 12:523. (Co-authors needed from Science Center)

Tracey, G.A., and D.J. Hansen. 1996. Use of biota-sediment accumulation factors to assess similarity of nonionic organic chemical exposure to benthically-coupled organisms of differing trophic mode. Arch. Environ. Contam. Toxicol. 30:467-475.

USDHHS (U.S. Department of Health and Human Services). 1993a. Registry of Toxic Effects of Chemical Substances (RTECS, online database). National Toxicology Information Program, National Library of Medicine, Bethesda, MD. 1993.

USDHHS. 1993b. Hazardous Substances Databank (HSDB, online database). National Toxicology Information Program, National Library of Medicine, Bethesda, MD. 1993.

Underwood, A.J. 1995. Toxicological testing in laboratories is not ecological testing of toxicology. *Human and Ecological Risk Assessment*. 1(3): 178-182

USFWS (U.S. Fish and Wildlife). 2003. Evaluation of the Clean Water Act Section 304(a) human health criterion for methylmercury: protectiveness for threatened and endangered wildlife in California. U.S. Fish and Wildlife Service, Sacramento Fish and Wildlife Office, Environmental Contaminants Division. Sacramento, California. U.S. Fish and Wildlife Service, Sacramento, CA. 123 pp. *http://sacramento.fws.gov/ec/Methylmercury%20Criterion%20Evaluation%20Final%20Report%20October%202003.pdf*.

USFWS. 2010. Draft Biological Opinion on EPA's Proposed Program of Continuing Approval or Promulgation of New Cyanide Criteria in State and Tribal Water Quality Standards. U.S. Fish and Wildlife Service, Arlington, VA. 329 pp.

USFWS/NMFS. 1998. Endangered Species Act consultation handbook: procedures for conducting section 7 consultations and conferences. U.S. Fish and Wildlife Service and National Marine Fisheries Service, ISBN 0-16-049596-2, U.S. Government Printing Office, Washington, D.C. 190 pp. *http://www.nmfs.noaa.gov/pr/laws/esa/policies.htm#consultation* [Accessed July 2012].

USGS (U.S. Geological Survey). 2012. The StreamStats program: U.S. Geological Survey database, Available from *http://streamstats.usgs.gov* [Accessed March 5, 2014].

Van den Berg, and 22 others. 1998. Toxic equivalency factors (TEFs) for PCBs, PCDDS, PCDFs for humans and wildlife. Environmental Health Perspectives 106: 775-792.

Van den Heuvel, L.S. McCarty, R.P. Lanno, B.E. Hickie, and D.G. Hixon. 1991. Effect of total body lipid on the toxicity and toxicokinetics of pentachlorophenol in rainbow trout (*Oncorhynchus mykiss*). Aquatic Toxicology 20:235-252.

van der Oost, R., A. Opperhuizen, K. Satumalay, H. Heida, and N.P. Vermeulen. 1996. Biomonitoring aquatic pollution with feral eel (< i> Anguilla anguilla</i>) I. Bioaccumulation: biota-sediment ratios of PCBs, OCPs, PCDDs and PCDFs. *Aquatic toxicology*. 35(1): 21-46

Van Genderen, E.J., A.C. Ryan, J.R. Tomasso, and S.J. Klaine. 2005. Evaluation of acute copper toxicity to larval fathead minnows (*Pimephales promelas*) in soft surface waters. *Environmental Toxicology and Chemistry*. 24(2): 408–414. *http://dx.doi.org/10.1897/03-494.1*

Van Genderen, E.J., J.R. Tomasso, and S.J. Klaine. 2008. Influence of copper exposure on whole-body sodium levels in larval fathead minnows (*Pimephales promelas*). *Environmental Toxicology and Chemistry*. 27(3): 1442–1449. *http://dx.doi.org/10.1897/07-467.1*

Van Kirk, R.W. and S.L. Hill. 2007. Demographic model predicts trout population response to selenium based on individual-level toxicity. *Ecological Modelling*. 206(3-4): 407-420. *http://dx.doi.org/10.1016/j.ecolmodel.2007.04.003*

Van Leeuwen C.J., P.S. Griffioen, W.H.A. Vergouw and J.L. Maas-Diepeveen. 1985. Difference in susceptibility of early life stages of rainbow trout (*Salmo gairdneri*) to environmental pollutants. Aquatic Toxicology 7:59-78.

Van Sprang, P., Ph. Léger, and P. Sorgeloos. 1991.A new test system for the evaluation of toxic levels of liposoluble products in the aquatic food chain using Artemia and Mysidopsis bahia as experimental animals. Aquat. Toxicol. 19:319-328.

Van Sprang, P.A., F.A.M. Verdonck, P.A. Vanrolleghem, M.L. Vangheluwe, and C.R. Janssen. 2004. Probabalistic environmental risk assessment of zinc in Dutch surface waters. *Environmental Toxicology and Chemistry*. 23(12): 2993-3002Vanderford, M. and J. Hamelink. 1977. Influence of environmental factors on pesticide levels in sport fish. *Pesticides monitoring journal*. 11(3): 138-145

Veith, G.D., D.W. Kuehl, E.N. Leonard, F.A. Puglisi, and A.E. Lemke. 1979. Polychlorinated biphenyls and other organic chemical residues in fish from major watersheds of the United States, 1976. Pestic. Monit. J., 13(1): 1-11.

Verma, S.R., S.K. Bansal, A.K. Gupta, and R.C. Dalela. 1978. In vivo effect on ATPase in certain tissues of Labeo rohita and Saccobranchus fossilis, following chronic chlordane intoxication. Bull. Environ. Contam. Toxicol., 20(6), 769-777

Verma, S.R., S. Rani, and R. C. Dalela. 1981. Pesticide-induced physiological alterations in certain tissues of a fish, Mystus vittatus. Toxicol. Lett. 9:327-32.

Versteeg, D.J., S.E. Belanger, and G.J. Carr. 1999. Understanding single-species and model ecosystem sensitivity: Data-based comparison. *Environmental Toxicology and Chemistry*. 18(6): 1329-1346

Vidal, V., S.M. Bay, and D. Schlenk. 2005. Effects of dietary selenomethionine on larval rainbow trout (*Oncorhynchus mykiss*). *Archives of Environmental Contamination and Toxicology*. 49(1): 71-75

Vijver, M.G., W.J.G.M. Peijnenburg, and G.R. De Snoo. 2010. Toxicological mixture models are based on inadequate assumptions. *Environmental Science and Technology*. 44(13): 4841–4842. *http://dx.doi.org/10.1021/es1001659*

Vijver, M.G., E.G. Elliott, W.J.G.M. Peijnenburg, and G.R. de Snoo. 2011. Response predictions for organisms water-exposed to metal mixtures: A meta-analysis. *Environmental Toxicology and Chemistry*. 30(7): 1482–1487. *http://dx.doi.org/10.1002/etc.499*

Vuorinen, P. J., J. Paasivirta, M. Keinaenen, J. Koistinen, T. Rantio, T. Hyoetylaeinen, and L. Welling. 1997. The M74 syndrome of Baltic salmon (Salmo salar) and organochlorine concentrations in the muscle of female salmon. Chemosphere 34:1151-1166.

Wai, C.M. and W.M. Mok. 1986. Chemical Speciation Approach to Evaluate Water Quality Problems in the Blackbird Mining District ldaho. Idaho Water Resources Research Institute, University of Idaho. Report to the U.S. Geological Survey, USGS/G-1014-04; NTIS No: PB88- 12811 l/HDM, Moscow, ID. 76 pp. *www.webedit.uidaho.edu/default.aspx?pid=115595* [Accessed 15 October 2009].

Waiwood, K.G. and F.W.H. Beamish. 1978. The effect of copper, hardness and pH on the growth of rainbow trout, *Salmo gairdneri*. *Journal of Fish Biology*. 13(5): 591-598

Walker, M.K., and R.E. Peterson. 1991. Potencies of polychlorinated dibenzo-p-dioxin, dibenzofuran, and biphenyl congeners, relative to 2,3,7,8 tetrachlorodibenzo-p-dioxin, for producing early life stage mortality in rainbow trout (*Oncorhynchus mykiss*). Aquat. Toxicol. 21:219-238.

Wallace, R.R. and H.B.N. Hynes. 1975. The catastrophic drift of stream insects after treatments with methoxychlor (1,1,1-trichloro-2,2-bis(p-methoxyphenyl) ethane). *Environmental Pollution (1970)*. 8(4): 255-268. *http://dx.doi.org/10.1016/0013-9327(75)90081-6*

Wallace, J.B., D.S. Vogel, and T.F. Cuffney. 1986. Recovery of a Headwater Stream from an Insecticide-Induced Community Disturbance. *Journal of the North American Benthological Society*. 5(2): 115-126

Wang, N., C.A. Mebane, J.L. Kunz, C.G. Ingersoll, T.W. May, W.R. Arnold, R.C. Santore, T. Augspurger, F.J. Dwyer, and M.C. Barnhart. 2009. Evaluation of acute copper toxicity to juvenile freshwater mussels (fatmucket, *Lampsilis siliquoidea*) in natural and reconstituted waters. *Environmental Toxicology and Chemistry*. 28(11): 2367–2377. *http://dx.doi.org/10.1897/08-655.1*

Waples, R.S., O.W. Johnson, and R.P. Jones. 1991a. Status Review for Snake River Sockeye Salmon. U.S. Department of Commerce, National Marine Fisheries Service, Northwest Fisheries Science Center, NOAA Technical Memorandum NMFS-F/NWC-195. *http://www.nwfsc.noaa.gov/publications/techmemos/tm195/tm195.htm*

Waples, R. S., R. P. Jones, Jr., B. R. Beckman, and G. A. Swan. 1991b. Status review for Snake River fall Chinook salmon. U.S. Department of Commerce, National Marine Fisheries Service, Northwest Fisheries Science Center, NOAA Technical Memorandum NMFS-F/NWC-201. Available: *http://www.nwr.noaa.gov/Publications/Biological-Status-Reviews/loader.cfm?csModule=security/getfile&pageid=21480*. (November 2011).

Watras, C.J., R.C. Back, S. Halvorsen, K.A. Morrison, R.J.M. Hudson, and S.P. Wente. 1998. Bioaccumulation of mercury in pelagic freshwater food webs. *Science of the Total Environment*. 219(2-3): 183-208

Wattras C.J., and N.S. Bloom. 1992. Mercury and methylmercury in individual zooplankton: Implications for bioaccumulation Limnol. Oceanogr., 37(6),1313-1318.

Watts, M.M., and D. Pascoe. 2000. Comparison of Chironomus riparius Meigen and Chironomus tentans Fabricius (Diptera: Chironomidae) for assessing the toxicity of sediments. Environ. Toxicol. Chem. 19:1885-1892.

Wauchope, R. D., Buttler, T. M., Hornsby A. G., Augustijn Beckers, P. W. M., and Burt, J. P. 1992. SCS/ARS/CES Pesticide properties database for environmental decision making. Rev. Environ. Contam. Toxicol. 123: 1-157, 1992.6-15

Webb P.W., and J .R. Brett. 1973. Effects of sublethal concentration of sodium pentachlorophenate on growth rate, food conversion efficiency, and swimming performance in underyearling sockeye salmon (*Oncorhynchus nerka*). Journal Fisheries Research Board of Canada 30(4): 499-507.

Webb, N.A., and C.M. Wood. 1998. Physiological analysis of the stress response associated with acute silver nitrate exposure in freshwater rainbow trout (*Oncorhynchus mykiss*). Environ. Toxicol. Chem. 17:579-588.

Webb, M.A.H., G.W. Feist, M.S. Fitzpatrick, E.P. Foster, C.B. Schreck, M. Plumlee, C. Wong, and D.T. Gunderson. 2006. Mercury concentrations in gonad, liver, and muscle of white sturgeon *Acipenser transmontanus* in the lower Columbia River. *Archives of Environmental Contamination and Toxicology*. 50(3): 443 - 451. *http://dx.doi.org/DOI:* 10.1007/s00244-004-0159-0

Webber, H.M. and T.A. Haines. 2003. Mercury effects on predator avoidance behavior of a forage fish, golden shiner (*Notemigonus crysoleuca*s). *Environmental Toxicology and Chemistry*. 22(7): 1556-1561

Wedemeyer, G.A., R.L. Saunders, and W.G. Clarke. 1980. Environmental factors affecting smoltification and early marine survival of anadromous salmonids. Mar. Fish. Rev. 42:1-14.

Wegner, S. J., and L. J. Campbell. 1991. Radionuclides, chemical constituents, and organic compounds in water from designated wells and springs from the southern boundary of the Idaho National Engineering Laboratory to the Hagerman area, Idaho, 1989. Geological Surv., Idaho Falls, ID (USA). US Geol. Surv. Report, 53 pp.

Weis, J.S. 2009. Reproductive, developmental, and neurobehavioral effects of methylmercury in fishes. *Journal of Environmental Science and Health, Part C*. 24(4): 212-225

Weis, P., and J. S. Weis. 1974. DDT causes changes in activity and schooling behavior in goldfish. Environ. Res. 7:68.

Welsh, T.L. 1991. Stanley Basin sockeye salmon lakes, upper Salmon River drainage, Idaho. Unpublished report to University of Idaho Aquaculture Institute.

Welsh, P.G., J.F. Skidmore, D.J. Spry, D.G. Dixon, P.V. Hodson, N.J. Hutchinson, and B.E. Hickie. 1993. Effect of pH and dissolved organic carbon on the toxicity of copper to larval fathead minnow (*Pimephales promelas*) in natural lake waters of low alkalinity. *Canadian Journal of Fisheries and Aquatic Sciences*. 50(7): 1356–1362. *http://dx.doi.org/10.1139/f93-155*

Welsh, P.G., J. Lipton, and G.A. Chapman. 2000a. Evaluation of water-effect ratio methodology for establishing site-specific water quality criteria. *Environmental Toxicology and Chemistry*. 19(6): 1616–1623. *http://dx.doi.org/10.1002/etc.5620190619*

Welsh, P.G., J. Lipton, G.A. Chapman, and T.L. Podrabsky. 2000*b*. Relative importance of calcium and magnesium in hardness-based modification of copper toxicity. Env. Tox. Chem. 19:1624-1631.

Welsh, P.G., J. Lipton, C.A. Mebane, and J.C.A. Marr. 2008. Influence of flow-through and renewal exposures on the toxicity of copper to rainbow trout. *Ecotoxicology and Environmental Safety*. 69(2): 199-208. *http://dx.doi.org/10.1016/j.ecoenv.2007.04.003*

Wendelaar Bonga, S.E. and R.A.C. Lock. 2008. The osmoregulatory system. Pages 401-416 *in* R. T. Di Giulio, and D. E. Hinton, editors. *Toxicology of Fishes*. CRC Press, Boca Raton, Florida.

Wente, S.P. 2004. A Statistical Model and National Data Set for Partitioning Fish-Tissue Mercury Concentration Variation Between Spatiotemporal and Sample Characteristic Effects. U.S. Geological Survey, Scientific Investigations Report 2004-5199. 15 pp. *http://emmma.usgs.gov/fishHgAbout2.aspx*.

White, J.L. and G.W. Harvey. 2007. Winter Feeding Success of Stream Trout under Different Streamflow and Turbidity Conditions. *Transactions of the American Fisheries Society*. 136(5): 1187–1192

Whittier, T.R. and J.K. Aitkin. 2008. Can soft water limit bighead carp and silver carp (*Hypophthalmichthys* spp.) invasions? *Fisheries*. 133(3): 122-128

WHO (World Health Organization). 1984. Environmental Health Criteria 38, Heptachlor--Environmental Effects. World Health Organization, Geneva.

WHO. 1989. Environmental Health Criteria 83, DDT and its Derivatives--Environmental Effects. World Health Organization, Geneva.

WHO. 1991. Environmental Health Criteria 124, Lindane--Environmental Effects. World Health Organization, Geneva

Wiener, J.G. 1995. Bioaccumulation of mercury in fish. National forum on mercury in fish. EPA, Office of Water. June 1995. EPA 823-R-95-002

Wiener, J.G. and D.J. Spry. 1996. Toxicological significance of mercury in freshwater fish. Pages 297-339 *in* W. N. Beyer, G. H. Heinz, and A. W. Redmon-Norwood, editors. *Environmental Contaminants in Wildlife: Interpreting tissue concentrations*. CRC Press, Boca Raton, Florida.

Wiener, J.G., D.P. Krabbenhoft, G.H. Heinz, and A.M. Scheuhammer. 2003. Ecotoxicology of mercury. Pages 409–463 *in* D. J. Hoffman, B. A. Rattner, G. A. Burton, and J. Cairns, Jr., editors. *Handbook of ecotoxicology*, 2nd edition. CRC Press, Boca Raton, Florida.

Wiener, J.G., B.C. Knights, M.B. Sandheinrich, J.D. Jeremiason, M.E. Brigham, D.R. Engstrom, L.G. Woodruff, W.F. Cannon, and S.J. Balogh. 2006. Mercury in Soils, Lakes, and Fish in Voyageurs National Park (Minnesota): Importance of Atmospheric Deposition and Ecosystem Factors. *Environmental Science and Technology*. 40(20): 6261 -6268

Wissmar, R.C., J.E. Smith, B.A. McIntosh, H.W. Li, G.H. Reeves, and J.R. Sedell. 1994. Ecological Health of River Basins in Forested Regions of Eastern Washington and Oregon. Gen. Tech. Rep. PNW-GTR-326. U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station. Portland, OR. 65 p.

Williams, G., J.M. West, and E.T. Snow. 2008. Total arsenic accumulation in yabbies (*Cherax destructor clark*) exposed to elevated arsenic levels in Victorian gold mining areas, Australia. *Environmental Toxicology and Chemistry*. 27(6): 1332–1342. *http://dx.doi.org/DOI:* 10.1897/07-407.1

Williamson, A.K., and five others. 1998. Water quality in the Central Columbia Plateau, Washington and Idaho, 1992-95. U.S. Geological Survey Circ. 1144.

Willson, M.F. 1997. Variation in salmonid life histories: patterns and perspectives. U.S. Forest Service, Pacific Northwest Research Station, PNW-RP-498, Portland, OR. 50 pp.

Windom, H.L., J.T. Byrd, R.G. Smith, and F. Huan. 1991. Inadequacy of NASQAN data for assessing metal trends in the nation's rivers. *Environmental Science and Technology*. 25(6): 1137-1142

Winner, R.W., H.A. Owen, and M.V. Moore. 1990. Seasonal variability in the sensitivity of freshwater lentic communities to a chronic copper stress. *Aquatic Toxicology*. 17(1): 75-92

Wolfe, M.F., and J.N. Seiber. 1993. Environmental Activation of Pesticides. Occup. Med. 8:561 573.

Wong, P.T.S., Y.K. Chau, O. Kramar, and G.A. Bengert. 1981. Accumulation and depuration of tetramethyllead bt rainbow trout. Wat. Res. 15:621-625.

Wood, C.M. 1991. Acid–base and ion balance, metabolism, and their interactions, after exhaustive exercise in fish. *Journal of Experimental Biology*. 160(1): 285–308

Wood, C.M. 2011. Silver. *Fish Physiology: Homeostasis and Toxicology of Non-Essential Metals*. 31(PART B): 1-65. *http://dx.doi.org/10.1016/S1546-5098(11)31001-1*

Wood, C.C., and C.J. Foote. 1990. Genetic differences in the early development and growth of sympatric sockeye salmon and kokanee (*Oncorhynchus nerka*) and their hybrids. Canadian Journal of Fisheries and Aquatic Science 47:2250-2260.

Wood, C.M., J.D. Turner, and M.S. Graham. 1983. Why do fish die after severe exercise? *Journal of Fish Biology*. 22(2): 189-201. *http://dx.doi.org/10.1111/j.1095-8649.1983.tb04739.x*

Wood, C.M., W.J. Adams, G.T. Ankley, D.R. DiBona, S.N. Luoma, R.C. Playle, W.A. Stubblefield, H.L. Bergman, R.J. Erickson, J.S. Mattice, and C.E. Schlekat. 1997. Environmental toxicology of metals. Pages 31-56 *in* H. L. Bergman, and E. J. Dorward-King, editors. *Reassessment of metals criteria for aquatic life protection: priorities for research and implementation. SETAC Pellston Workshop on Reassessment of Metals Criteria for Aquatic Life Protection.* SETAC Press, Pensacola, FL.

Wood, C.M., R.C. Playle, and C. Hogstrand. 1999. Physiology and modeling of mechanisms of silver uptake and toxicity in fish. Env. Tox. Chem. 18:71-83.

Woodward, D. F., W. G. Brumbaugh, A. L. DeLonay, E. E. Little, C. E. Smith. 1994. Effects on rainbow trout fry of a metals-contaminated diet of benthic invertebrates from the Clark Fork River, Montana. Transactions of the American Fisheries Society. 123:51-62.

Woodward, D. F., J. A. Hansen, H. L. Bergman, E. E. Little, and A. J. DeLonay. 1995. Brown trout avoidance of metals in water characteristic of the Clark Fork River, Montana. Can. J. of Fish. Aquatic Sci. 52:2031-2037.

Woodward, D. F., J. N. Goldstein, and A. M. Garag. 1997. Cutthroat trout avoidance of metals and conditions characteristic of a mining waste site: Coeur d=Alene River, Idaho. Trans. Amer. Fish. Society 126:699-706.

Woodward-Clyde. 2000. Stibnite Area Site Characterization Report. Woodward-Clyde International-Americas, Report to the Idaho Department of Environmental Quality. Revised June 23, 2000., Denver, CO. 606 pp.

Wren, C.D., and MacCrimmon, H.R. 1986. Comparative Bioaccumulation of Mercury in Two Adjacent Freshwater Ecosystems. Water Resources. Vol 20. No. 6. pp:763-769.

Yang C, and Chen S. 1999. Two organochlorine pesticides, toxaphene and chlordane, are antagonists for estrogen-related receptor alpha-1 orphan receptor. Cancer Res 59(18):4519-4524.

Yount, J.D. and G.J. Niemi. 1990. Recovery of lotic communities and ecosytems from disturbance - a narrative review of case studies. *Environmental Management*. 14(5): 571-587

Zabel, R.W., M.D. Scheuerell, M. M. McClure, and J.G. Williams. 2006. The interplay between climate variability and density dependence in the population viability of Chinook salmon. Conservation Biology 20:190-200.

Zaroogian, G., M. Johnson, and J. Heltshe. 1985. Estimation of bioconcentration in marine species using structure-activity models. *Environmental toxicology and chemistry*. 4(1): 3-12

Zhao, Y. and M.C. Newman. 2004. Shortcomings of the laboratory-derived median lethal concentration for predicting mortality in field populations: exposure duration and latent mortality. *Environmental Toxicology and Chemistry*. 23(9): 2147-2153. *http://dx.doi.org/1897/05-341R.1*

Zhao, Y. and M.C. Newman. 2005. Effects of exposure duration and recovery time during pulsed exposures. *Environmental Toxicology and Chemistry*. 25(5): 1298–1304. *http://dx.doi.org/10.1897/05-341R.1*

Zia, S. and D.G. McDonald. 1994. Role of the gills and gill chloride cells in metal uptake in the freshwater-adapted rainbow trout, *Oncorhynchus mykiss*. *Canadian Journal of Fisheries and Aquatic Sciences*. 51(11): 2482–2492. *http://dx.doi.org/10.1139/f94-247*

Appendix A

A Review of Water Hardness Data for Idaho — 1979-2004

Introduction

Water hardness is an important water-quality parameter, not only because it affects the quality of domestic water, but also because it affects the toxicity of metals to fish. Hardness mitigates metals toxicity, because Ca^{2+} and Mg^{2+} help keep fish from absorbing metals such as lead, arsenic, and cadmium into their bloodstream through their gills. The greater the hardness, the harder it is for toxic metals to be absorbed through the gills. NMFS retrieved hardness data collected in Idaho during the last 25 years from the USGS database in order to assess the relevant data, identify potential water quality problems, and locate regions where further investigation may be warranted.

The USGS National Water Quality Laboratory generally provides hardness analyses in terms of an equivalent concentration of calcium carbonate ($CaCO_3$). Approximately 3600 water samples from 324 sites on Idaho rivers and streams have been analyzed for hardness, defined as equivalent quantities of CaCO3, in milligrams per liter (mg/L), since 1979. Most of these samples were analyzed as part of the Statewide Water Quality Network; other samples were analyzed in the course of other water-quality investigations by the USGS Idaho District office. NMFS used these data to construct a general overview of water hardness over the last 25 years in the State of Idaho. A summary of the results of this preliminary analysis are given below.

A list of the 324 water-quality sampling sites, with USGS station identification numbers (STAID), descriptive names, and the number of samples from 1979-2004, is given in Table 1. A map showing locations of sites is given in figure 1a. The sites are located in 10 different hydrologic units (6-code HUCs). Approximately 75 percent (246) of the sites are within HUCs 170103 (Spokane, 128 sites) and 170402 (Upper Snake, 118 sites). The Middle Snake-Boise (170501), Salmon (170602), and Clearwater (170603) are represented by 33, 20, and 16 sites, respectively. The remaining HUCs have 11 or fewer sites.

The size and color of the symbols in Figure 1b represent the number of samples collected at each site since 1979. The number of samples per site ranges from 1 to 179; approximately 75 percent have been sampled fewer than 10 times. One site had no reported hardness value because calcium was below reporting limits on the single occasion it was sampled (station 12413850, Evans Creek near St. Maries, ID).

Forty sites were sampled intermittently over the course of 20 or more years, but only a few sites were regularly monitored over that period of time.

Overview of Water Hardness in Idaho

Regional variation in maximum water hardness (defined here as variation in the highest value measured at each site between 1979 and 2004) is displayed symbolically in the map in Figure 2. (Recall that some of the sites are represented by only one sample). The color of the symbol indicates the maximum hardness value. Sites where the maximum hardness exceeded 140 mg/L are shown in shades of green. In general, the highest hardness values were found in

southern and southeastern Idaho. Many of these sites are located in areas where carbonate rocks are present (Figure 2). This relation is predictable because rocks containing calcium carbonate are an obvious source of water-soluble Ca^{+} ions, which contribute to hardness.

For domestic water use, water with hardness over 120 mg/L generally is considered "hard" (many different hardness classifications exist), and over 180 is "very hard". "Soft" water has hardness less than 60 mg/L. According to this classification, 56 of the sites would be considered to have soft water and 72 sites would be considered to have very hard water.

Maximum and minimum hardness values data for the 324 sites in Idaho are shown in Figure 2 and 5. A cumulative distribution plot of the data is given in Figure 3.

In addition to important effects of hardness on metals toxicity, the different major ions that contribute to hardness may affect toxicity differently (Naddy *et al.* 2002). Therefore it is useful to examine the Ca:Mg ratio. The Ca:Mg ratio in these data ranges over two orders of magnitude, from 0.9 to 90. The average ratio is 4.9; the median ratio is 3.9. About 53% of the sites have a maximum Ca:Mg ratio less than 4. The sites having ratios greater than 4 are mainly in central Idaho and in the Boise River Basin. Some of the lowest ratios are found in the Coeur d'Alene region and in south-central and southeastern Idaho (figure 4).

Metals

Because low, not high, water hardness directly contributes to metals toxicity for aquatic biota, the minimum hardness value measured at each site is shown on the map in figure 5. This map clearly indicates that potential metals toxicity problems exist in northern, western, and central Idaho, where minimum hardness measurements generally were less than approximately 50 mg/L. Of particular concern are sites in HUC 170103 (Spokane), a region where high metal concentrations in rivers and streams are known to exist.

The EPA has established national recommended water quality criteria (EPA, 2002) to help States and Tribes to establish water quality standards under the Clean Water Act. EPA lists the water quality criteria for 158 pollutants. Because the toxicity of certain dissolved metals, including cadmium, lead, nickel, and zinc, is hardness-dependent, the EPA recommended criteria for these metals, in µg/L are calculated by using equations of the form

$$EXP^{(m_{a,c}(\ln H)+b_{a,c})},$$

where $m_{a,c}$ and $b_{a,c}$ are empirically-determined constants (a = acute, c = chronic), different for each metal, and H is the hardness of the water. A conversion factor for fresh water is also applied. Two criteria are commonly used: the CMC (criterion maximum concentration; for acute toxicity) and the CCC (criterion continuous concentration; for chronic toxicity).

The EPA does not recommend using a low-end hardness "cap" for calculating CMC and CCC in cases where hardness is unusually low, asserting that doing so may provide less protection for aquatic organisms than intended by Guidelines given in EPA 822/R-85-100. Nevertheless, some agencies have used a low-end floor (e.g. 25 mg/L) for establishing standards, substituting 25 mg/L for measured values less than 25 mg/L.

As a demonstration of how hardness is used to establish water quality criteria, we retrieved available data for Cd for the 324 sites. In all cases, the water sample analyzed for Cd was taken at the same date and time as the hardness sample.

Among the 324 sites for which we compiled hardness data, 167 sites had at least one sample that was analyzed for Cd (1,287 total samples analyzed for Cd). The minimum reporting limit for Cd by the National Water Quality Lab has varied throughout the last 25 years; 1 µg/L and 0.04 µg/L were the two reporting limits encountered in the data set. Cadmium was detected at 90 of these sites, in a total of 758 samples. The Cd values for these sites are shown on the map in Figure 6a.

NMFS calculated CMCs for Cd for each of the 90 sites using the EPA equation and parameters given by EPA (2002). In cases where more than one sample was available at a site, we used the minimum hardness value measured at the site with the corresponding Cd value (not necessarily the maximum measured Cd value), assuming this represented the potentially most toxic "instantaneous" situation.

To assess the effect of using different lower floor for hardness, NMFS calculated CMCs for these sites three ways: 1) Using actual measured values for samples having H < or equal to 25 mg/L; 2) substituting H = 10 mg/L for actual values < or = 10 mg/L; and 3) substituting H = 25 mg/L for actual values < or = 25 mg/L.

In the first case (no cap, using measured hardness values to calculate the CMC), 70 of the 90 sites exceeded the criterion. The criterion values ranged from 0.09 µg/L to 36 µg/L (fig. 6b; table 1).

Using a low-end floor of H =10 mg/L (that is, changing all hardness values less than 10 mg/L to 10 mg/L) affected the CMC of 9 sites, and resulted in 69 of the samples exceeding the criterion. In other words, one site that had previously exceeded the criterion now met the criterion. The measured Cd value at this site was 0.11 µg/L; the unadjusted minimum hardness was 5 mg/L. Setting the cap at H = 25 mg/L affected the CMC of 48 out of 90 sites. In this case, 67 samples exceeded the criterion (74%).

When there was no lower hardness floor, the criterion was exceeded in 70 of 90 of the samples (78%) (Figure 6c). This cursory analysis suggests that setting low-end floor for hardness when calculating the CMC and CCC for Cd could make a difference in whether or not a site met the CMC, albeit in a small number of cases.

At 5 of the sites, measured Cd was less than or equal to 0.6 µg/L, but low hardness resulted in potentially toxic situations for aquatic biota. All these samples are in the Spokane-Coeur d'Alene region. On the other hand, a sample from Bannock County contained 20 µg/L Cd but had a minimum hardness of 280 mg/L. The CMC for this site was approximately 5 µg/L Cd, ordinarily considered a high concentration of Cd; the water's high hardness mitigated the toxicity of Cd to some degree, but not sufficiently to meet the criterion.

One site in the Spokane/Coeur d'Alene region contained 2.42 µg/L of Cd, but met the CMC of 36.6 µg/L because the hardness was 2100 mg/L. Clearly this demonstrates a need for an upper cap for hardness as well as a lower floor. EPA provides guidance for hardness > 400 mg/L by recommending two options: 1) calculate the criterion using a Water Effect Ratio (WER) of 1.0 and use a hardness of 400 mg/L in the equation, or 2) calculate the criterion using a WER and the actual hardness of the water. If this sample had been calculated using H = 400 mg/L, the CMC would have been 7.44 µg/L and the sample still would have met the criterion.

A "quick lookup" graph showing the CMC for Cd (EPA, 2002), in µg/L, as a function of water hardness is shown in Figure 7. This graph permits an estimate of the Cd CMC to be easily

determined if the hardness of the water is known. Graphs such as these are easily constructed for other metals by entering the appropriate equations in a spreadsheet.

Trends in water hardness

For this analysis, NMFS selected sites with long-term hardness records (10 or more readings over at least 13 years) to test for temporal trends in hardness. The maximum hardness measured within each year in the period of record was chosen in an effort to compensate for changes in hardness related to seasonal discharge. The Mann-Kendall trend test was integrated into an Excel spreadsheet, which performed the test by comparing each measurement with all the other previous measurements, one at a time, and assigning a "+1" or "-1", depending on whether that measurement is larger or smaller. We compared the sum of all the pluses and minuses (the "S-statistic") with values in a table to determine if a statistically significant (in this case, $p \leq 0.05$) trend was present.

The results of the trend analysis are given in Table 1. Of the 38 tested sites, 3 showed statistically significant positive trends (increasing hardness), 7 showed negative trends, and 28 showed no significant trends. Note that even though some significant trends were found, the magnitude of the changes relative to the total water hardness does not appear to be relevant from a water quality management point of view.

Summary

NMFS analyzed data from 324 water quality sampling sites from 1979 to 2004 to gain insight to water hardness in Idaho. Sites for which water hardness data were collected in the last 25 years are clustered in northern Idaho (Spokane/Coeur d'Alene region), the Snake River and its tributaries in south-central Idaho, Big Lost River basin, the Portneuf River and its tributaries, and the Lower Boise River. A wide range of hardness values was found; some of the statewide variation is apparently related to the bedrock geology. Approximately 38% of the maximum hardness values are classified as "hard" (>120 mg/L). High hardness values were found mainly in south-central and southeastern Idaho. Very low hardness waters were predominant in northern Idaho and were commonly associated with high metal concentrations.

Cd was detected at 90 of the sites for which hardness data were compiled (approximately 27%). Concentration Maximum Criteria for Cd were determined for these 90 sites, using concurrent hardness values; 70 sites failed to meet the EPA-recommended criterion. Most of the noncompliant sites are located in the Spokane/Coeur d'Alene region, but one noncompliant site occurs in each of the following counties: Ada, Bannock, Canyon, Idaho, Nez Perce, Owyhee, and Valley.

The above discussion suggests that the hardness level of receiving waters is an important water quality parameter that needs to be considered in the development of water quality criteria for some metals. There are areas in Idaho with hardness lower that the current floor used in calculating discharge limits of 25mg/L and that results increasing risk and harm to listed salmon and steelhead.

References for Appendix A

Naddy, R.B., Stubblefield, W.A., May, J.R., Tucker, S.A., and Hockett, J.R., 2002, The effect of calcium and magnesium ratios on the toxicity of cotter to five aquatic species in freshwater: Environ. Toxicol. Chem., v. 21 (2), 347-352.

United States Environmental Protection Agency, 2002, National Recommended Water Quality Criteria: 2002, 33 p.

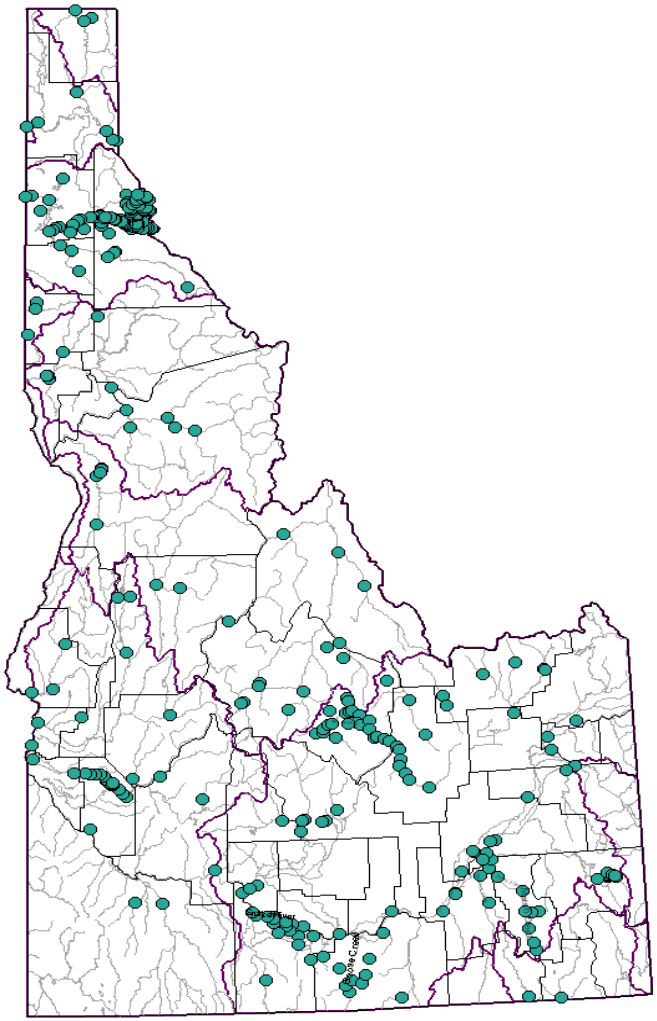


Figure 1a. USGS surface water sampling sites where at least one sample was analyzed for water hardness from 1979 to 2004.

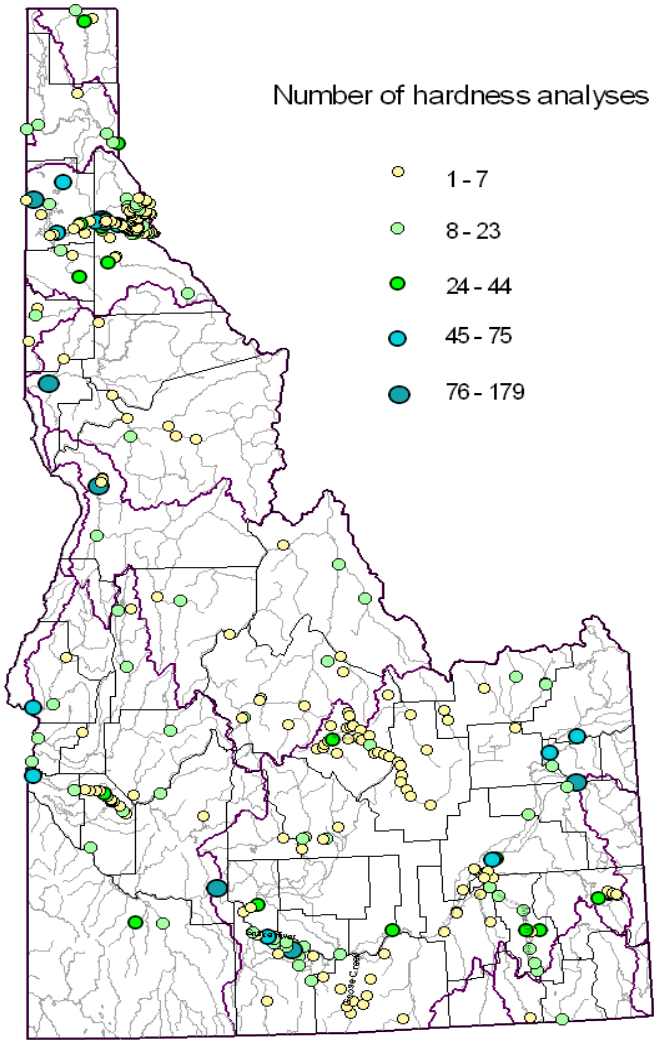


Figure 1b. Number of hardness analyses performed from 1979 to 2004.

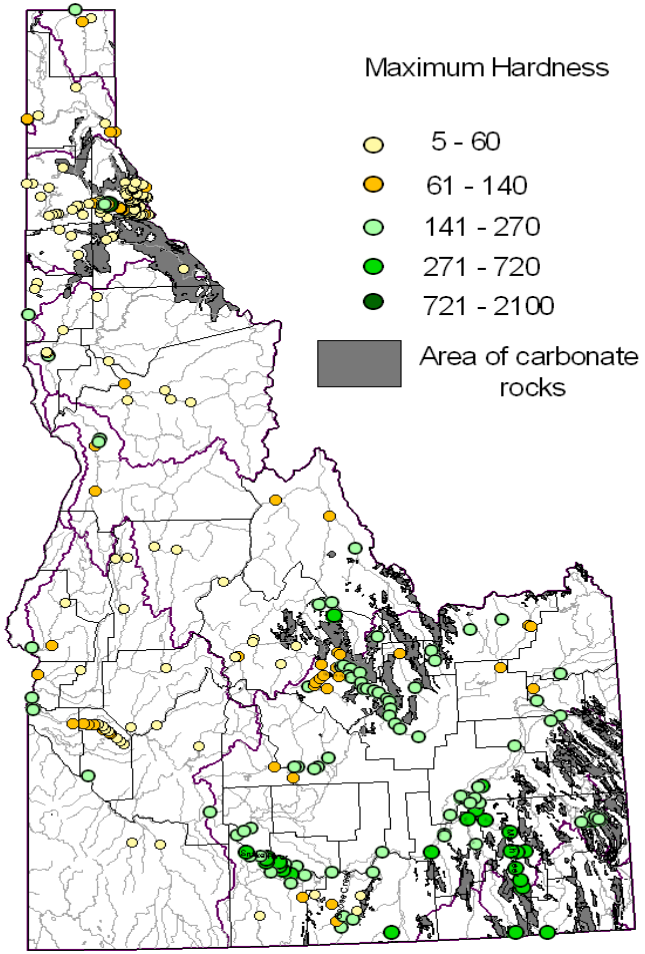


Figure 2. Map showing maximum hardness measured from 1979 to 2004 and areas of carbonate rock in Idaho.

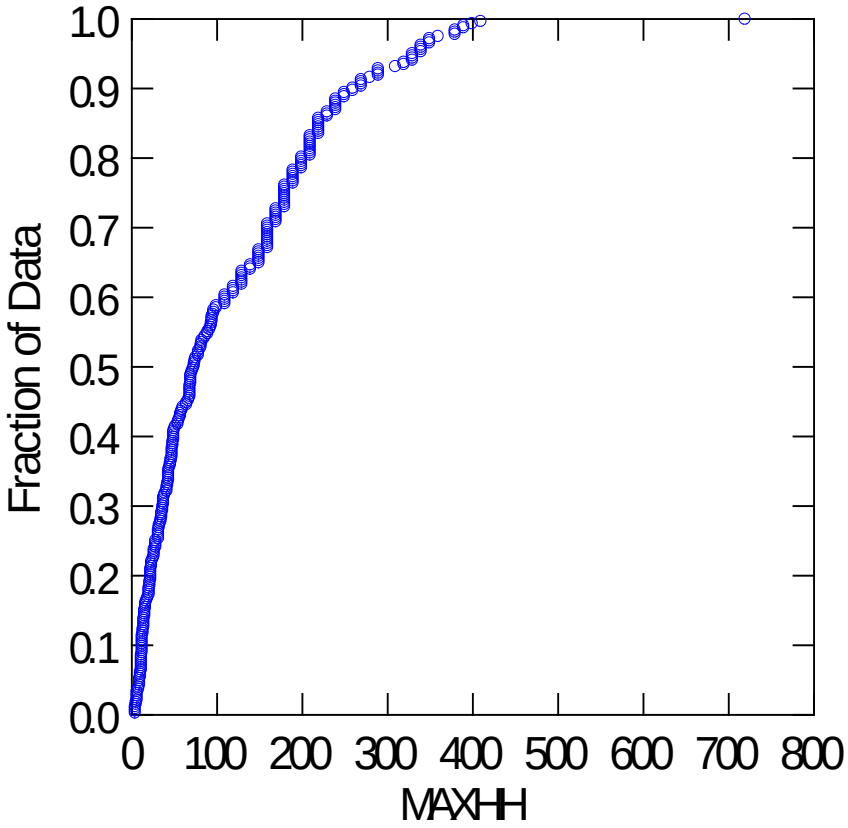


Figure 3. Cumulative distribution plot of maximum hardness (n = 323). Plot does not include one sample of maximum hardness equal to 2100 mg/L.

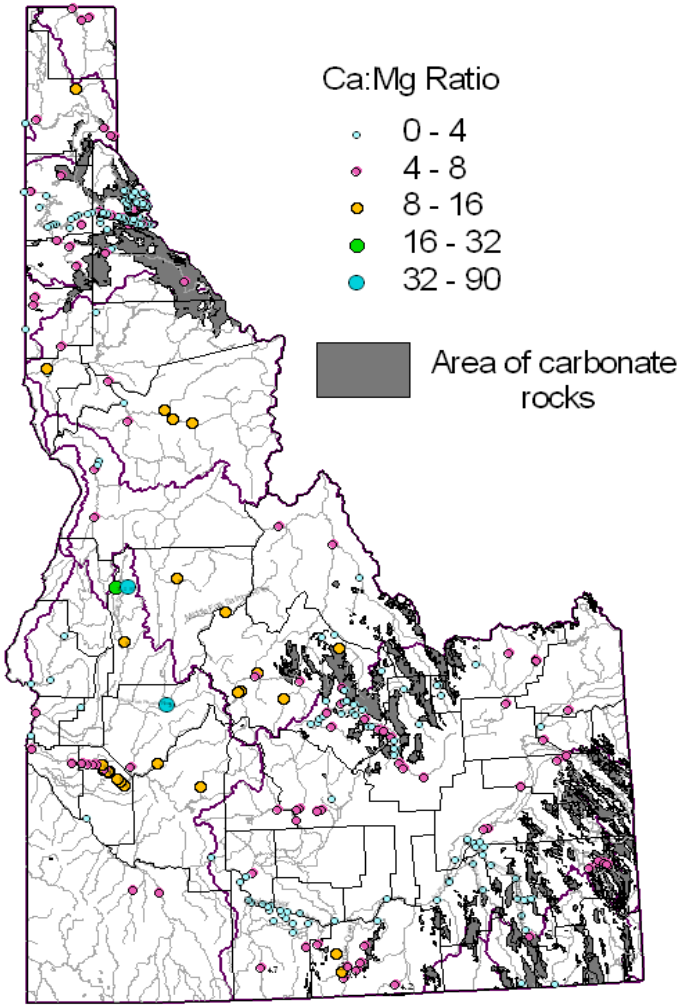


Figure 4. Map showing maximum Ca:Mg ratio at 324 water quality sites in Idaho.

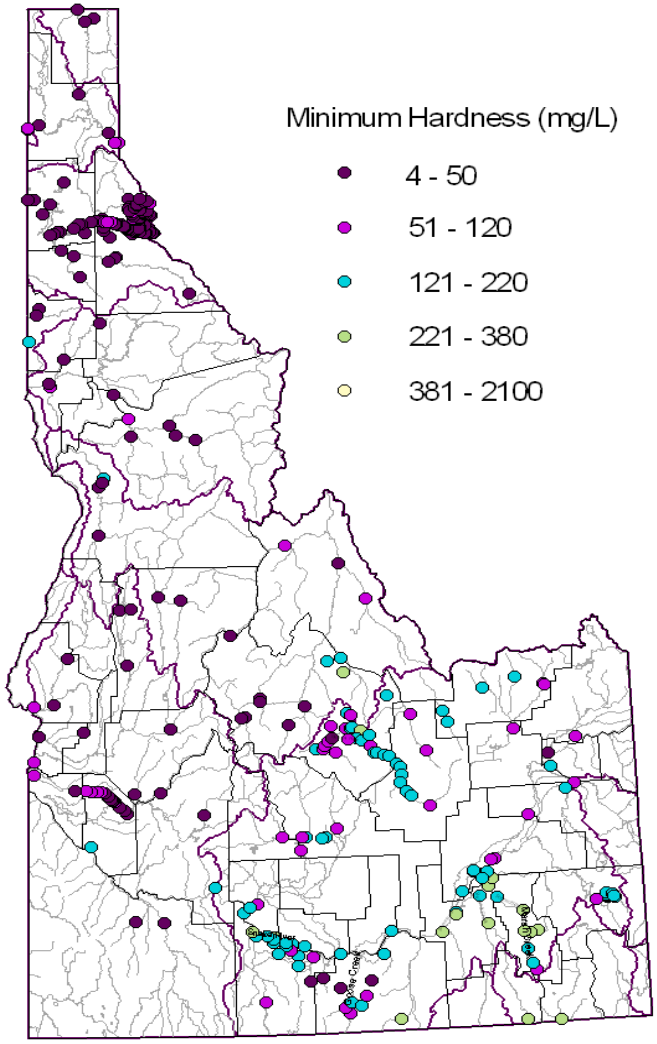


Figure 5. Minimum hardness measured at sampling sites from 1979 to 2004.

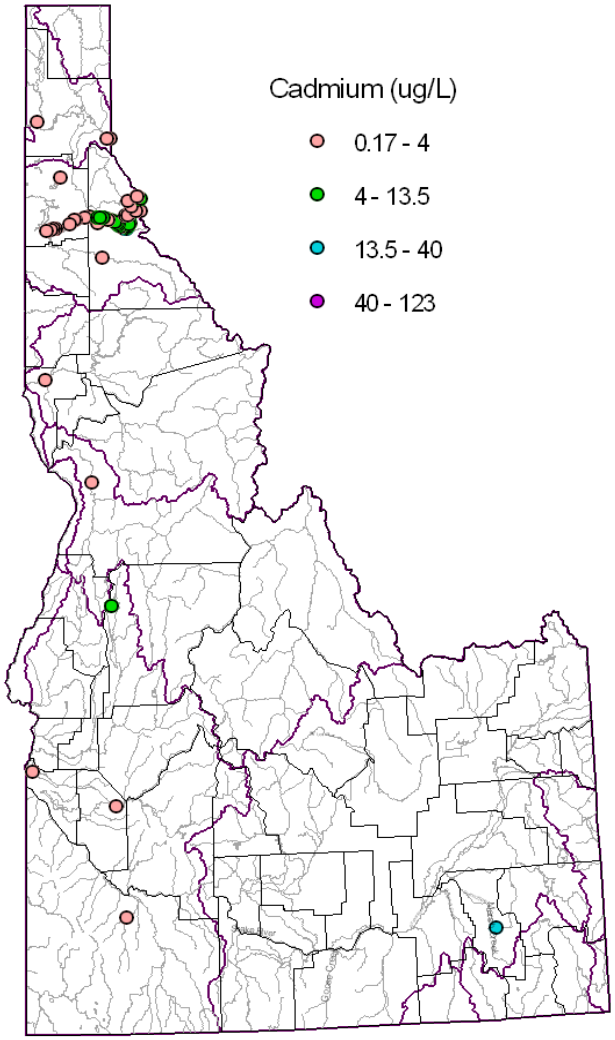


Figure 6a. Cadmium value associated with minimum hardness value (n = 90)

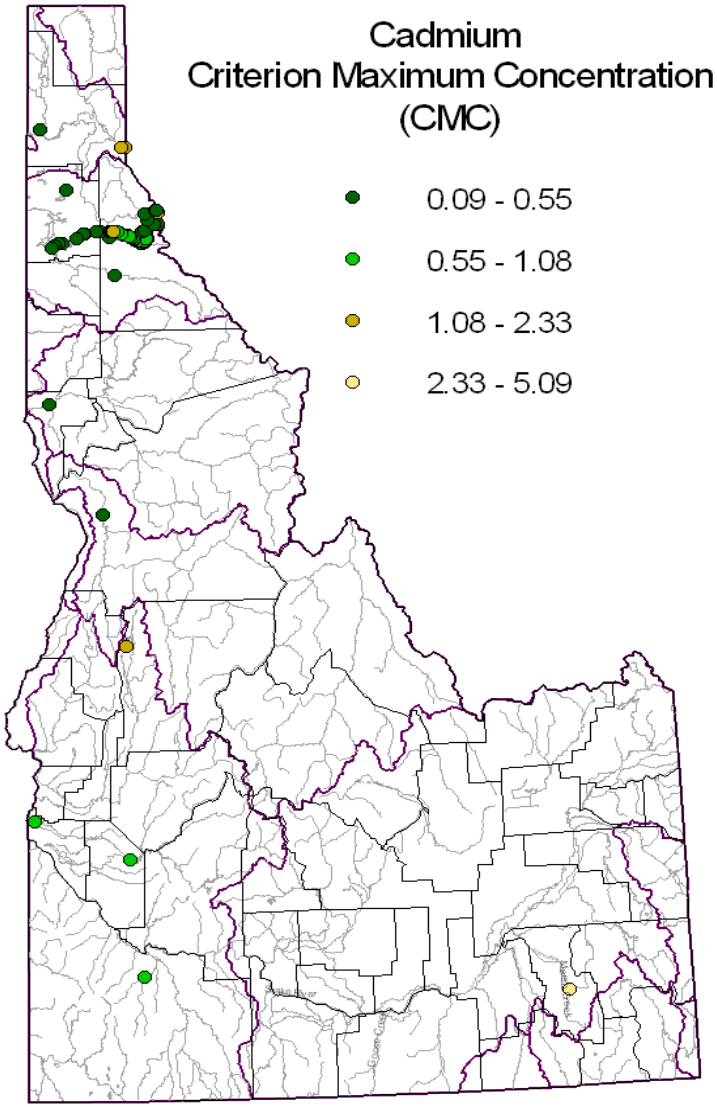


Figure 6b. Criterion Maximum Concentration (CMC) for cadmium calculated with no lower floor on hardness (n = 90).

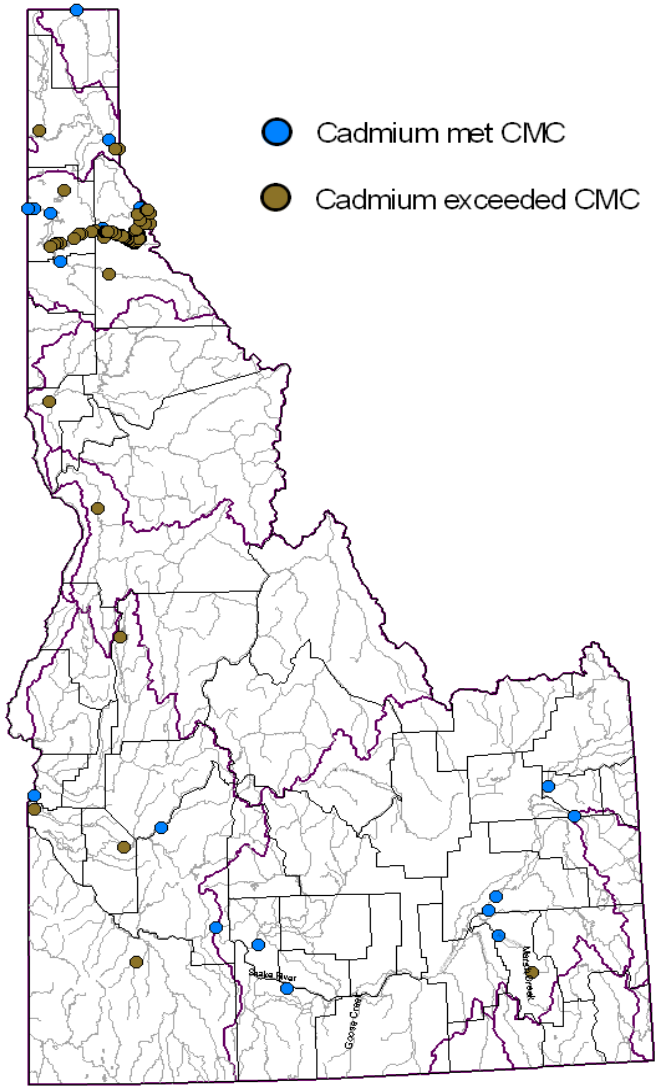


Figure 6c. Sites where Cd CMC was met (n = 20) and exceeded (n = 70).

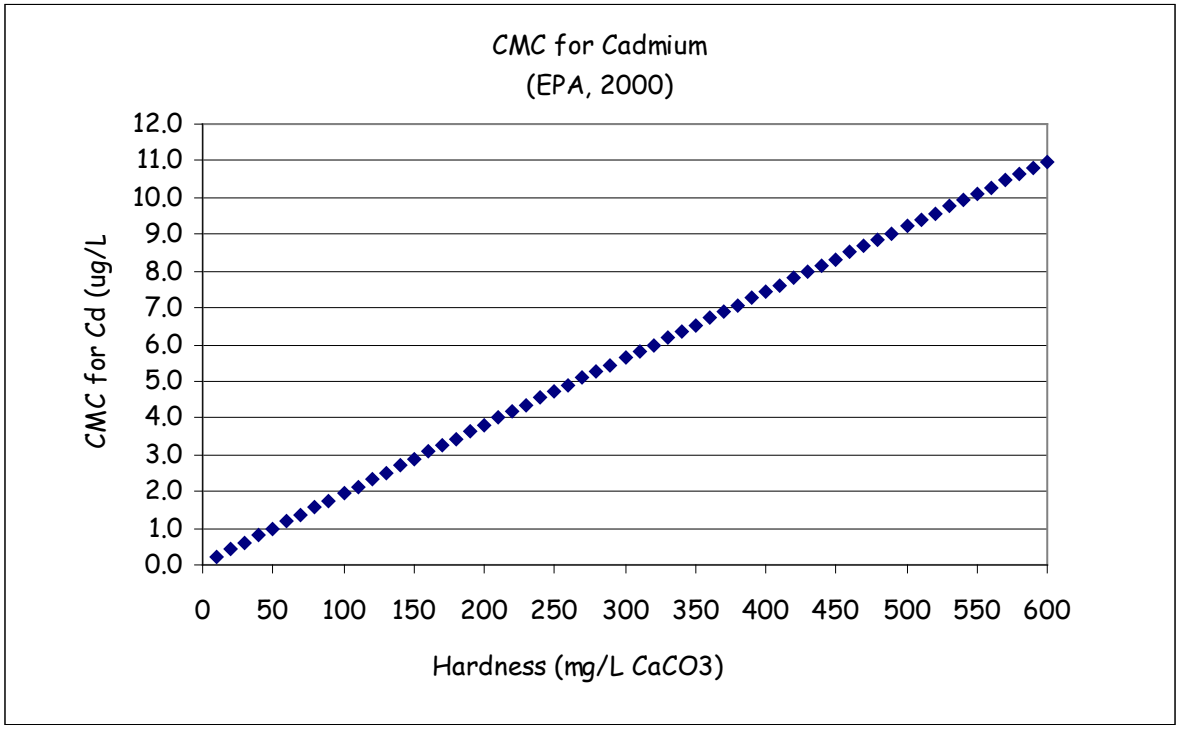


Figure 7. "Quick lookup" graph for the Cd CMC. If water hardness is known, the CMC can be visually estimated. Similar graphs for other metals can easily be constructed.

Table A.1. Summary of hardness values in Idaho, downloaded from the USGS National Water Information System 1979-2004 (http://nwis.waterdata.usgs.gov) [H – water hardness in mg/L as $CaCO_3$; Ca – calcium; Mg – magnesium, Ave-average. All concentrations in mg/L

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
10092700	Bear River at Idaho-Utah State Line	42.013	-111.919	11	300	330	390	48	63.2	76	33	41.6	52	0.9	1.6	2.2
10125500	Malad River at Woodruff ID	42.03	-112.229	4	370	562.5	720	68	127.0	160	49	59.0	83	1.4	2.2	3.2
12316800	Mission Creek nr Copeland ID	48.932	-116.333	5	8	8.6	10	2.3	2.5	2.7	0.33	0.5	0.73	3.7	5.2	7.9
12318500	Kootenai River nr Copeland ID	48.912	-116.416	34	36	97.6	140	10	27.0	40	2.7	7.2	10	3.5	3.8	4.1
12322000	Kootenai River at Porthill ID	48.996	-116.508	23	46	99.3	150	13	27.7	40	3.3	7.3	11	3.5	3.8	4.1
12391950	Clark Fork River Below Cabinet Gorge Dam ID	48.088	-116.073	39	64	86.2	97	17.7	24.0	27.5	4.8	6.3	7.54	3.4	3.8	4.2
12392000	Clark Fork at Whitehorse Rapids nr Cabinet ID	48.093	-116.118	16	70	88.3	96	20	24.8	27	4.8	6.4	7	3.6	3.9	4.2
12392155	Lightning Creek at Clark Fork ID	48.151	-116.182	16	4	9.3	15	1.29	2.6	3.94	0.272	0.7	1.18	3.0	4.1	4.8
12392300	Pack River nr Colburn ID	48.42	-116.501	6	5	10.2	17	1.5	3.3	5.6	0.004	0.4	0.7	6.0	8.3	12.0
12395000	Priest River nr Priest River ID	48.209	-116.914	18	18	25.3	36	5	7.2	10.7	1.4	1.8	2.32	3.3	3.9	4.7
12395500	Pend Oreille River at Newport Wa	48.182	-117.033	6	71	78.7	83	20	22.0	23	5.1	5.8	6.2	3.7	3.8	3.9
12395502	Pend Oreille River at Us Hwy No.2 at Newport Wa	48.185	-117.033	9	62	78	87	17	21.4	24	4.8	5.9	6.6	3.4	3.7	3.9
12411000	Nf Coeur D Alene R ab Shoshone Ck nr Prichard ID	47.707	-115.977	17	13	21.2	27	2.99	4.8	6.26	1.35	2.2	2.89	2.0	2.1	2.2
12411935	Prichard Creek at Mouth at Prichard ID	47.657	-115.968	18	6	10.8	14	1.68	2.8	3.77	0.519	0.9	1.2	3.0	3.1	3.2
12411950	Beaver Cr ab Carpenter Gulch nr Prichard, ID	47.633	-115.979	2	21	27	33	5.49	6.9	8.26	1.67	2.3	2.93	2.8	3.1	3.3
12413000	Nf Coeur D Alene River at Enaville ID	47.569	-116.252	102	10	18.6	25	2.48	4.5	6.2	0.937	1.7	2.3	2.3	2.6	3.2
12413025	Little Nf Sf Coeur D Alene Riv Abv Mouth nr Mullan	47.465	-115.722	1	7	7	7	1.68	1.7	1.68	0.677	0.7	0.677	2.5	2.5	2.5
12413030	Sf Coeur D Alene R Bl Obrien Gulch nr Larson, ID	47.467	-115.733	2	8	10.5	13	2.29	2.9	3.43	0.672	0.9	1.05	3.3	3.3	3.4
12413040	Sf Coeur D Alene R Abv Deadman Gulch nr Mullan ID	47.473	-115.766	20	12	39.7	69	3.25	10.7	19.2	0.963	3.2	5.11	2.8	3.3	3.9
12413100	Boulder Creek at Mullan ID	47.469	-115.796	1	20	20	20	6.2	6.2	6.2	1	1.0	1	6.2	6.2	6.2
12413103	Sf Coeur D Alene R ab Slaughterhse Gulch at Mullan	47.466	-115.813	1	18	18	18	5.16	5.2	5.16	1.32	1.3	1.32	3.9	3.9	3.9
12413104	Sf Coeur D Alene R Bl Trowbridge Gulch nr Wallace	47.474	-115.869	1	23	23	23	6.32	6.3	6.32	1.8	1.8	1.8	3.5	3.5	3.5
12413118	Canyon Creek at Burke, ID	47.521	-115.818	16	4	9.2	12	1.19	2.4	3.31	0.338	0.7	1	3.1	3.3	3.6
12413120	Canyon Creek at Gem ID	47.508	-115.867	2	10	13	16	2.71	3.5	4.38	0.683	0.9	1.21	3.6	3.8	4.0

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
12413123	Canyon Creek at Woodland Park ID	47.489	-115.889	18	9	31	45	2.49	8.7	12.7	0.623	2.2	3.2	3.8	3.9	4.0
12413125	Canyon Creek ab Mouth at Wallace, ID	47.473	-115.914	43	10	36	58	2.77	10.2	16.2	0.692	2.6	4.25	3.5	3.9	4.2
12413126	Ninemile Cr ab Mouth Of Ef Ninemile Cr nr Blackcld	47.514	-115.898	1	95	95	95	22.6	22.6	22.6	9.43	9.4	9.43	2.4	2.4	2.4
12413127	Ef Ninemile Creek Abv Mouth nr Blackcloud ID	47.513	-115.893	17	8	24.8	42	2.56	8.0	13.8	0.396	1.2	1.9	6.0	6.9	7.3
12413130	Ninemile Creek ab Mouth at Wallace, ID	47.479	-115.919	44	16	50.2	75	4.42	14.0	21.4	1.05	3.7	5.89	3.1	3.8	4.3
12413131	Sf Coeur D Alene R Abv Placer Cr at Wallace ID	47.475	-115.928	1	21	21	21	5.77	5.8	5.77	1.63	1.6	1.63	3.5	3.5	3.5
12413140	Placer Creek at Wallace ID	47.463	-115.937	18	19	36	48	5.85	10.8	14.1	1.07	2.2	3.12	4.5	4.9	5.6
12413150	Sf Coeur D Alene River at Silverton ID	47.492	-115.954	19	18	46.1	69	4.96	12.5	18.9	1.3	3.6	5.53	3.2	3.5	3.8
12413151	Lake Creek ab Mouth nr Silverton, ID	47.49	-115.952	1	27	27	27	7	7.0	7	2.29	2.3	2.29	3.1	3.1	3.1
12413168	Twomile Creek ab Mouth at Osburn, ID	47.51	-115.995	2	23	31.5	40	6.87	9.0	11.1	1.42	2.2	3.01	3.7	4.3	4.8
12413169	Sf Coeur D Alene R Blw Twomile Cr nr Osburn ID	47.51	-115.996	10	19	39.1	70	5.25	10.6	18.6	1.38	3.1	5.63	3.3	3.5	3.8
12413174	Terror Gulch Creek ab Mouth nr Osburn, ID	47.514	-116.021	2	35	41.5	48	7.44	9.0	10.6	3.88	4.6	5.22	1.9	2.0	2.0
12413175	Sf Coeur D Alene R at Terror Gulch at Osburn ID	47.514	-116.022	1	22	22	22	6.24	6.2	6.24	1.66	1.7	1.66	3.8	3.8	3.8
12413179	Sf Coeur D Alene R ab Big Creek nr Big Creek, ID	47.527	-116.049	1	23	23	23	6.29	6.3	6.29	1.69	1.7	1.69	3.7	3.7	3.7
12413185	Big Creek ab Mouth nr Big Creek, ID	47.529	-116.051	1	12	12	12	3.2	3.2	3.2	0.946	0.9	0.946	3.4	3.4	3.4
12413190	Moon Creek Abv Mouth at Elk Creek ID	47.533	-116.058	16	15	27.4	38	3.56	6.2	8.49	1.49	2.9	4.07	2.0	2.2	2.4
12413204	Montgomery Creek ab Mouth nr Elizabeth Park, ID	47.531	-116.088	1	13	13	13	3.21	3.2	3.21	1.16	1.2	1.16	2.8	2.8	2.8
12413209	Elk Creek ab Mouth at Elizabeth Park, ID	47.53	-116.09	1	16	16	16	4.3	4.3	4.3	1.36	1.4	1.36	3.2	3.2	3.2
12413210	Sf Coeur D Alene at Elizabeth Park nr Kellogg ID	47.531	-116.092	64	18	50	79	5.12	13.4	20.9	1.38	4.0	6.5	3.1	3.3	3.7
12413250	Sf Coeur D Alene T at Kellogg, ID	47.545	-116.134	10	19	54.3	71	5.32	14.4	19	1.43	4.5	6	3.0	3.3	3.8
12413290	Government Gulch nr Mouth at Smelterville ID	47.545	-116.166	16	11	35.1	61	2.96	10.1	17.7	0.783	2.4	4.03	3.8	4.1	4.4
12413300	Sf Coeur D Alene River at Smelterville ID	47.549	-116.174	23	22	74	180	6	19.3	45.2	1.65	6.3	16.7	2.7	3.2	3.8
12413360	Ef Pine Creek Abv Gilbert Cr Near Pinehurst ID	47.44	-116.174	1	5	5	5	1.31	1.3	1.31	0.361	0.4	0.361	3.6	3.6	3.6
12413440	Pine Creek ab Mouth Of Ef Pine Cr at Pine, ID	47.487	-116.241	1	6	6	6	1.5	1.5	1.5	0.441	0.4	0.441	3.4	3.4	3.4
12413445	Pine Creek Blw Amy Gulch nr Pinehurst ID	47.516	-116.24	44	4	10.5	16	1.14	2.7	4.26	0.348	0.9	1.46	2.8	3.0	3.3
12413460	Pine Creek ab Mouth nr Pinehurst, ID	47.547	-116.227	1	5	5	5	1.41	1.4	1.41	0.427	0.4	0.427	3.3	3.3	3.3
12413470	Sf Coeur D Alene River nr Pinehurst ID	47.552	-116.237	115	17	70.2	190	4.59	18.4	50	1.34	5.9	18	2.3	3.2	4.7

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
12413490	Sf Coeur D Alene River at Enaville ID	47.56	-116.251	1	42	42	42	12	12.0	12	2.9	2.9	2.9	4.1	4.1	4.1
12413500	Coeur D Alene River nr Cataldo ID	47.555	-116.323	75	12	35.4	65	3.02	9.1	16	1.12	3.1	6.1	2.6	3.0	3.7
12413700	Latour Creek Abv Baldy Creek nr Cataldo ID	47.469	-116.439	3	4	4.7	5	1.2	1.2	1.3	0.27	0.4	0.5	2.4	3.7	4.4
12413755	Coeur D Alene River Blw Latour Creek nr Cataldo ID	47.551	-116.367	3	18	38.7	50	4.56	9.8	12.7	1.57	3.4	4.35	2.8	2.9	2.9
12413810	Coeur D Alene River at Rose Lake ID	47.537	-116.472	31	12	28.9	48	3.06	7.3	12.1	1.07	2.6	4.2	2.5	2.8	3.2
12413815	Coeur D Alene River Blw Rose Creek nr Rose Lake ID	47.535	-116.499	4	19	36	51	5.05	9.1	13	1.62	3.2	4.53	2.6	2.9	3.1
12413825	Coeurdalene Riv ab Kilarney Lk Outlet nr Rose Lake	47.506	-116.554	4	18	34	48	4.65	8.5	12.1	1.57	3.1	4.22	2.6	2.8	3.0
12413850	Evans Creek nr St Maries ID	47.449	-116.567	0				0.02	0.02	0.02	0.004	0.004	0.004	5.0	5.0	5.0
12413858	Coeur D Alene River Blw Blue Lake nr Harrison ID	47.48	-116.699	7	17	26.1	42	4.3	6.5	10.8	1.51	2.4	3.77	2.5	2.7	2.9
12413860	Coeur D Alene River nr Harrison ID	47.479	-116.732	61	12	29.7	50	2.88	7.4	12.7	1.16	2.7	4.71	2.4	2.7	3.2
12413862	Coeur D Alene River at Harrison Bridge nr Harrison	47.465	-116.765	4	17	32.3	44	4.35	8.1	11	1.5	3.0	4.1	2.5	2.7	2.9
12413875	St. Joe River at Red Ives Ranger Station ID	47.056	-115.352	14	13	18.9	24	3.75	5.6	7.16	0.831	1.2	1.52	4.5	4.7	4.9
12414350	Big Creek ab East Fork nr Calder ID	47.306	-116.116	7	12	16.3	21	3	4.3	5.6	1	1.3	1.8	2.7	3.2	3.8
12414400	Ef Big Creek nr Calder ID	47.302	-116.118	2	17	19	21	4.3	4.9	5.4	1.4	1.6	1.7	3.1	3.1	3.2
12414500	St Joe River at Calder ID	47.275	-116.188	26	14	23.5	32	4.14	6.9	9.62	0.898	1.5	1.93	4.3	4.6	5.2
12414900	St Maries River nr Santa ID	47.176	-116.492	26	10	16.4	23	2.79	4.8	6.8	0.686	1.1	1.5	3.5	4.4	6.0
12415075	St Joe River at St Maries ID	47.317	-116.561	1	19	19	19	6	6.0	6	0.86	0.9	0.86	7.0	7.0	7.0
12415140	St Joe River Near Chatcolet ID	47.36	-116.691	13	14	20.5	29	4.03	6.0	8.42	0.888	1.4	1.9	3.8	4.3	4.8
12415300	Mica Creek nr Coeur D Alene ID	47.6	-116.883	2	13	14	15	3.3	3.6	3.9	1.2	1.2	1.2	2.8	3.0	3.3
12416000	Hayden Creek Bl North Fork nr Hayden Lake ID	47.823	-116.654	66	17	30.1	49	4.1	8.0	13	1.4	2.5	4	2.3	3.2	4.1
12417598	Spokane River at Lake Outlet at Coeur D Alene ID	47.676	-116.801	20	17	20.6	27	4.49	5.6	7.4	1.39	1.6	2	3.1	3.5	3.9
12419000	Spokane River nr Post Falls ID	47.703	-116.977	97	16	20.6	29	4.21	5.5	7.9	1.23	1.6	2.6	2.9	3.4	4.4
12419495	Spokane River at Stateline Br nr Greenacres, Wa	47.699	-117.044	5	18	19.6	21	4.68	5.3	5.78	1.46	1.6	1.74	3.2	3.3	3.4
13037500	Snake River nr Heise ID	43.613	-111.659	112	120	188	270	35	53.3	76	8.2	13.4	20	3.4	4.0	4.8
13038500	Snake River at Lorenzo ID	43.735	-111.876	12	140	192.5	240	40	54.9	68	9.7	13.6	17	3.8	4.1	4.4
13055000	Teton River nr St Anthony ID	43.927	-111.615	53	67	132.4	180	19	35.8	50	4.7	10.3	15	2.7	3.5	4.2
13056500	Henrys Fork nr Rexburg ID	43.826	-	52	34	64.9	95	9.7	17.6	26	2.4	5.1	7.4	3.0	3.5	4.0

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
13058000	Willow Creek nr Ririe ID	43.583	-111.904 - 111.746	10	190	197	210	51	52.6	55	13	16.3	19	2.8	3.3	4.2
13060000	Snake River nr Shelley ID	43.414	- 112.135	17	69	133.4	160	20	37.5	47	4.5	9.6	11	3.7	4.0	4.4
13062690	Blackfoot River at Bridge Abv Angus Cr nr Henry,ID	42.824	- 111.323	1	180	180	180	53	53.0	53	10.7	10.7	10.7	5.0	5.0	5.0
13062692	Angus Creek Bl Angus Creek Reservoir	42.827	-111.4	1	290	290	290	78.8	78.8	78.8	22.1	22.1	22.1	3.6	3.6	3.6
13062693	Angus Creek 1.3 Mi Bl Angus Creek Reservoir	42.843	- 111.414	1	220	220	220	64.2	64.2	64.2	15.2	15.2	15.2	4.2	4.2	4.2
13062695	Angus Creek Near Henry ID	42.854	- 111.411	1	200	200	200	57.4	57.4	57.4	13	13.0	13	4.4	4.4	4.4
13062698	Angus Creek at Road 121 Xing nr Henry ID	42.842	- 111.359	1	170	170	170	53.3	53.3	53.3	9.53	9.5	9.53	5.6	5.6	5.6
13062700	Angus Creek nr Henry ID	42.828	- 111.338	1	160	160	160	49.7	49.7	49.7	9.34	9.3	9.34	5.3	5.3	5.3
13063000	Blackfoot River ab Reservoir nr Henry ID	42.817	-111.51	30	120	168.7	210	25.3	50.6	62	7.35	10.2	14.1	1.9	5.1	6.0
13068500	Blackfoot River nr Blackfoot ID	43.131	- 112.476	36	120	203.1	340	34.2	52.3	82	8.3	17.7	34	2.1	3.1	4.3
13069500	Snake River nr Blackfoot ID	43.125	- 112.518	53	81	145.1	170	23	40.5	50	5.7	10.6	14	3.3	3.8	4.5
13069515	Mctucker Creek nr Pingree ID	43.034	- 112.626	5	230	290	320	57	66.0	73	22	30.4	35	2.0	2.2	2.6
13069532	Crystal Waste nr Springfield ID	43.052	- 112.686	3	320	326.7	330	73	74.0	75	32	34.0	35	2.1	2.2	2.3
13069540	Danielson Creek nr Springfield ID	43.059	-112.69	6	210	225	250	51	55.3	61	20	21.0	23	2.5	2.6	2.9
13069565	Aberdeen Waste nr Aberdeen ID	42.924	- 112.811	3	190	213.3	230	49	53.7	57	16	19.3	21	2.6	2.8	3.1
13073000	Portneuf River at Topaz ID	42.625	- 112.088	42	230	336	410	59	80.2	97	18	32.8	43	1.9	2.5	3.4
13073120	Portneuf/Marsh Valley Canal nr Mccammon ID	42.615	- 112.166	5	280	302	340	67	74.2	81	21	28.8	37	2.1	2.7	3.9
13073743	Marsh Creek at Red Rock Pass nr Downey ID	42.356	- 112.126	9	98	206.4	290	30	62.9	95	5.6	12.0	15	4.1	5.3	7.9
13073750	Marsh Creek at Hwy 191 Xing nr Downey ID	42.408	- 112.156	10	150	267	330	43	70.2	90	11	22.4	29	2.5	3.2	3.9
13074810	Marsh Creek ab Hawkins Creek nr Virginia ID	42.506	- 112.192	9	150	276.7	400	39	67.2	100	13	26.6	37	1.9	2.6	3.1
13075000	Marsh Creek nr Mccammon ID	42.63	- 112.225	27	250	321.1	350	59	74.7	85	22	32.7	38	2.0	2.3	3.0
13075050	Marsh Creek ab Mouth nr Inkom ID	42.767	- 112.232	9	250	287.8	330	60	68.4	79	24	28.3	35	2.1	2.4	2.8
13075500	Portneuf River at Pocatello ID	42.872	- 112.468	20	170	280	340	45	64.4	80	13	28.7	35	1.8	2.3	3.5
13075910	Portneuf River nr Tyhee ID	42.945	- 112.544	15	260	282	310	64	68.1	75	23	27.1	30	2.3	2.5	2.8
13075960	Ross Fork nr Fort Hall ID	43.001	- 112.516	3	230	243.3	270	57	60.3	67	22	23.3	26	2.6	2.6	2.6
13075983	Spring Creek at Sheepskin Rd nr Fort Hall ID	43.043	- 112.555	3	210	213.3	220	58	59.7	62	16	16.0	16	3.6	3.7	3.9

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
13075985	Spring Creek nr Fort Hall ID	43.003	-112.6	5	200	210	220	56	57.8	60	15	15.8	17	3.5	3.7	4.0
13076100	Rattlesnake Creek nr Pocatello ID	42.7	-112.561	1	240	240	240	60	60.0	60	23	23.0	23	2.6	2.6	2.6
13076200	Bannock Creek nr Pocatello ID	42.886	-112.642	4	200	275	320	52	66.5	79	17	26.8	31	2.3	2.5	3.1
13076500	American Falls Res at American Falls ID	42.779	-112.879	2	200	210	220	47	53.5	60	18	18.5	19	2.5	2.9	3.3
13076600	Reuger Springs nr American Falls ID	42.767	-112.882	5	250	252	260	63	64.4	68	22	22.4	23	2.8	2.9	3.1
13077650	Rock Creek nr American Falls ID	42.652	-113.014	1	360	360	360	78	78.0	78	41	41.0	41	1.9	1.9	1.9
13078205	Raft River Bl Onemile Creek nr Malta ID	42.07	-113.444	1	380	380	380	110	110.0	110	26	26.0	26	4.2	4.2	4.2
13081500	Snake R nr Minidoka ID (at Howells Ferry)	42.673	-113.5	43	150	186.3	210	42	47.9	55	12	16.3	20	2.5	3.0	3.5
13082500	Goose Creek ab Trapper Creek nr Oakley ID	42.125	-113.939	5	89	161.8	210	27	48.4	62	5.2	10.0	13	4.6	4.9	5.2
13083000	Trapper Creek nr Oakley ID	42.169	-113.972	5	58	107.6	130	19	36.0	43	2.5	4.1	4.8	7.6	8.7	9.1
13084000	Goose Creek nr Oakley ID	42.203	-113.911	1	160	160	160	48.2	48.2	48.2	9.86	9.9	9.86	4.9	4.9	4.9
13084400	Birch Creek ab Feeder Canal nr Oakley ID	42.178	-113.819	1	150	150	150	45	45.0	45	8.7	8.7	8.7	5.2	5.2	5.2
13084590	Mill Creek 14s 23e 04	42.237	-113.777	1	55	55	55	17	17.0	17	3.1	3.1	3.1	5.5	5.5	5.5
13084650	Willow Creek nr Burley ID	42.348	-113.729	1	37	37	37	12	12.0	12	1.6	1.6	1.6	7.5	7.5	7.5
13087995	Snake River Gaging Station at Milner ID	42.528	-114.018	7	140	171.4	220	29	42.8	57	11.5	15.8	19	1.6	2.8	3.4
13088000	Snake River at Milner ID (Total Flow)	42.528	-114.018	9	170	195.6	230	46	50.2	59	13	17.1	21	2.6	3.0	3.5
13088020	Wrong No - Twin Falls Main Canal - See 13087800	42.518	-114.275	7	100	168.6	190	26	42.6	48	9	15.1	18	2.3	2.8	3.2
13088400	Dry Creek nr Artesian City ID	42.372	-114.186	3	24	25	26	7	7.2	7.3	1.6	1.7	1.8	4.1	4.2	4.4
13088510	Cottonwood Creek nr Oakley ID	42.294	-114.022	4	19	34	69	6.1	11.2	23	0.91	1.5	2.7	6.5	7.3	8.5
13090000	Snake River nr Kimberly ID	42.591	-114.36	20	150	203	240	39.5	49.1	58	11.9	19.4	24	2.0	2.6	3.3
13091500	Blue Lakes Outlet nr Twin Falls ID Mv 15	42.608	-114.476	5	210	230	240	53	58.4	62	18.6	19.9	21	2.8	2.9	3.1
13092000	Rock Creek nr Rock Creek ID	42.356	-114.303	9	29	61.7	94	8.7	19.1	29	1.7	3.4	5.3	5.1	5.6	6.7
13092710	Rock Creek Near 3200 East nr Twin Falls ID	42.523	-114.42	9	150	255.6	290	41	68.3	80	12	20.4	24	3.0	3.4	3.8
13092747	Rock Creek ab Hwy 30/93 Xing at Twin Falls ID	42.563	-114.494	107	91	247.5	340	25	61.4	83	6.82	22.8	50.7	1.1	2.8	3.7
13093000	Rock Creek Below Poleline Road nr Twin Falls ID	42.594	-114.529	12	130	272.5	350	33	66.4	83	11	25.6	34	2.4	2.7	3.0
13093095	Rock Creek nr Mouth nr Twin Falls ID	42.624	-114.533	10	210	286	350	53	70.1	86	20	27.3	34	2.5	2.6	2.7
13093394	Crystal Spring at Head nr Buhl ID	42.659	-114.642	1	280	280	280	64	64.0	64	28	28.0	28	2.3	2.3	2.3

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
13093470	Cedar Draw ab Low Line Canal nr Filer ID	42.52	-113.595	2	180	185	190	46	46.5	47	17	17.0	17	2.7	2.7	2.8
13093475	Cedar Draw Bl Low Line Canal nr Filer ID	42.544	-114.613	7	170	195.7	270	43	49.0	62	15	17.9	27	2.3	2.8	3.1
13093500	Cedar Draw nr Filer (Old Station)	42.623	-114.654	9	210	286.7	390	52	66.3	86	20	29.1	42	2.0	2.3	2.6
13093530	Cedar Draw ab Mouth nr Filer ID	42.649	-114.659	9	220	280	380	52	65.2	85	21	28.6	40	2.0	2.3	2.6
13094000	Snake River nr Buhl ID	42.666	-114.711	49	170	223.7	260	43	54.5	63	14.4	21.4	26	2.3	2.6	3.0
13095200	Briggs Creek nr Buhl ID	42.672	-114.817	1	190	190	190	43	43.0	43	19	19.0	19	2.3	2.3	2.3
13106000	Salmon River Canal Co Canal nr Rogerson ID	42.221	-114.738	1	58	58	58	17.2	17.2	17.2	3.67	3.7	3.67	4.7	4.7	4.7
13108150	Salmon Falls Creek nr Hagerman ID	42.696	-114.854	21	240	282.9	330	60	70.9	82	21.3	25.7	30	2.5	2.8	3.0
13108500	Camas Creek at 18mi Shearing Corral nr Kilgore ID	44.3	-111.905	2	66	67.5	69	19	19.5	20	4.4	4.6	4.7	4.3	4.3	4.3
13108900	Camas Creek at Red Road nr Kilgore ID	44.289	-111.894	9	57	72	82	16	20.6	23	4.2	5.0	6	3.8	4.1	4.4
13112000	Camas Creek at Camas ID	44.003	-112.22	3	59	67.3	72	17	19.3	21	3.9	4.6	5	4.0	4.2	4.4
13113000	Beaver Creek at Spencer ID 12n-36e-23a	44.355	-112.18	13	180	213.8	230	53	61.6	67	12	14.5	16	3.6	4.3	4.8
13116000	Medicine Lodge Creek at Ellis Ranch nr Angora ID	44.291	-112.503	1	220	220	220	59	59.0	59	18	18.0	18	3.3	3.3	3.3
13117020	Birch Creek at Blue Dome Inn nr Reno ID	44.153	-112.909	2	180	180	180	44	44.5	45	16	16.0	16	2.8	2.8	2.8
13117030	Birch Creek at Eight-Mile Canyon Rd nr Reno ID	44.08	-112.876	2	170	170	170	40	41.5	43	15	15.5	16	2.5	2.7	2.9
13117390	Summit Cr ab Barney H Sp nr Goldburg 11n 25e 22aaa	44.276	-113.456	1	160	160	160	36	36.0	36	16	16.0	16	2.3	2.3	2.3
13118700	Little Lost River Bl Wet Creek nr Howe ID	44.139	-113.244	5	64	110.8	130	15	26.2	34	6.4	10.9	13	1.9	2.4	3.1
13119000	Little Lost River nr Howe ID	43.886	-113.1	7	110	158.6	200	28	38.4	47	9.2	14.9	19	2.3	2.6	3.0
13119800	Nf Big Lost River nr Chilly ID	43.926	-114.183	1	170	170	170	41	41.0	41	17	17.0	17	2.4	2.4	2.4
13120000	Nf Big Lost River at Wild Horse nr Chilly ID	43.934	-114.113	2	110	110	110	29	29.0	29	8.4	8.7	8.9	3.3	3.4	3.5
13120240	Ef Big Lost R at Rosenkance Rch nr Chilly ID	43.896	-113.983	1	65	65	65	20	20.0	20	3.6	3.6	3.6	5.6	5.6	5.6
13120420	Twin Bridges Creek nr Chilly ID 07n 20e 9b	43.953	-114.103	1	90	90	90	24	24.0	24	7.4	7.4	7.4	3.2	3.2	3.2
13120450	Garden Creek nr Chilly ID 08n 20e 35d	43.979	-114.06	1	110	110	110	31	31.0	31	8.9	8.9	8.9	3.5	3.5	3.5
13120500	Big Lost River at Howell Ranch nr Chilly ID	43.998	-114.021	39	44	81.9	110	13	24.1	31	2.6	5.3	6.9	4.2	4.6	5.4
13121500	Big Lost River at Chilly Bridge nr Chilly ID	44.059	-113.878	2	88	90.5	93	26	26.5	27	5.6	6.0	6.3	4.3	4.5	4.6
13121580	Sage Creek ab Div nr Mackay ID 09n 20e 25ad	44.083	-114.033	1	110	110	110	24	24.0	24	12	12.0	12	2.0	2.0	2.0
13121700	Willow Creek Bl Freighter Spring 10n 22e 28	44.168	-113.861	1	130	130	130	37	37.0	37	8.6	8.6	8.6	4.3	4.3	4.3

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
13121900	Cedar Creek ab Div nr Dickey ID 10n 22e 34cc	44.149	-113.839	2	120	125	130	32	33.0	34	9.6	9.8	10	3.3	3.4	3.4
13122000	Thousand Springs Creek nr Chilly ID	44.067	-113.84	3	210	233.3	270	48	54.7	60	20	23.0	28	2.1	2.4	2.8
13122100	Elkhorn Creek nr Chilly ID 09n 22e 26c	44.075	-113.819	1	160	160	160	26	26.0	26	22	22.0	22	1.2	1.2	1.2
13122400	Long Cedar Creek ab Div nr Chilly ID	44.044	-113.744	1	250	250	250	62	62.0	62	23	23.0	23	2.7	2.7	2.7
13122500	Big Lost River Bl Chilly Sinks nr Chilly ID	43.996	-113.771	2	130	145	160	34	36.0	38	12	14.0	16	2.4	2.6	2.8
13123400	Big Lost River at Goddard Bridge 07n 23e 33cca1	43.977	-113.739	1	160	160	160	38	38.0	38	15	15.0	15	2.5	2.5	2.5
13124030	Hamilton Springs nr Mackay ID	43.991	-113.865	3	110	116.7	120	32	32.3	33	8.3	8.5	8.6	3.7	3.8	3.9
13125800	Upper Cedar Creek ab Div nr Mackay 08n 24e 19cb	44.008	-113.658	1	220	220	220	57	57.0	57	19	19.0	19	3.0	3.0	3.0
13127000	Big Lost River Bl Mackay Res nr Mackay ID	43.939	-113.648	16	120	140	160	34	39.6	44	8.6	9.9	11	3.6	4.0	4.3
13127700	Big Lost River at Mackay ID	43.886	-113.616	1	150	150	150	42	42.0	42	11	11.0	11	3.8	3.8	3.8
13127780	Big Lost River at Alder Cr Rd Brdg nr Mackay ID	43.887	-113.578	1	160	160	160	44	44.0	44	11	11.0	11	4.0	4.0	4.0
13130200	Big Lost River Bl Alder Creek nr Mackay ID	43.871	-113.511	1	180	180	180	53	53.0	53	12	12.0	12	4.4	4.4	4.4
13130300	Big Lost River nr Leslie ID	43.859	-113.466	1	180	180	180	51	51.0	51	12	12.0	12	4.3	4.3	4.3
13130847	Big Lost R at Darlington Rd Xing nr Darlington ID	43.813	-113.392	1	170	170	170	50	50.0	50	12	12.0	12	4.2	4.2	4.2
13132050	Big Lost River ab Moore Div nr Moore ID	43.787	-113.358	1	190	190	190	54	54.0	54	14	14.0	14	3.9	3.9	3.9
13132150	Big Lost River at Moore ID	43.729	-113.359	1	200	200	200	55	55.0	55	14	14.0	14	3.9	3.9	3.9
13132310	Big Lost River ab Arco ID	43.682	-113.366	1	200	200	200	55	55.0	55	15	15.0	15	3.7	3.7	3.7
13132375	Big Lost River at Arco-Minidoka Rd Xing at Arco ID	43.624	-113.311	1	210	210	210	61	61.0	61	14	14.0	14	4.4	4.4	4.4
13132500	Big Lost River nr Arco ID	43.582	-113.271	7	170	210	240	48	59.9	71	12	14.4	16	3.7	4.1	4.5
13132520	Big Lost River Bl Ineel Div nr Arco ID	43.516	-113.082	3	110	136.7	180	33	40.1	52.4	7.1	9.2	12.5	4.2	4.4	4.6
13140800	Big Wood River at Stanton Crossing nr Bellevue ID	43.329	-114.319	2	160	160	160	47.4	47.7	48	9.4	9.5	9.55	5.0	5.0	5.0
13141000	Big Wood River nr Bellevue ID	43.328	-114.342	17	100	162.9	190	30	49.3	56	6.5	9.5	11	4.6	5.2	5.6
13141500	Camas Creek nr Blaine ID	43.333	-114.541	4	59	77.8	94	18	23.8	29	3.3	4.4	5.3	5.3	5.4	5.6
13142500	Big Wood River Bl Magic Dam nr Richfield ID	43.248	-114.356	1	110	110	110	33	33.0	33	6.8	6.8	6.8	4.9	4.9	4.9
13148500	Little Wood River nr Carey ID	43.389	-114	4	77	106.8	150	21	28.8	41	6	8.2	11	3.3	3.5	3.7
13150430	Silver Creek at Sportsman Access nr Picabo ID	43.323	-114.108	17	190	195.9	210	53	55.9	60	13	13.7	17	3.4	4.1	4.4
13152500	Malad River nr Gooding ID	42.887	-114.802	44	71	163.5	210	20	42.8	60	5	13.8	18	2.2	3.2	4.3

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
13152850	Big Wood River at Upper Malad Dam nr Hagerman ID	42.866	-114.868	4	140	145	150	31	33.5	36	14	14.8	15	2.1	2.3	2.6
13152900	Cove Creek nr Hagerman ID	42.867	-114.868	2	150	155	160	35	36.0	37	15	15.5	16	2.3	2.3	2.3
13154500	Snake River at King Hill ID	43.002	-115.202	179	140	188.5	220	36	45.2	57	11	18.3	22	2.1	2.5	3.4
13168500	Bruneau River nr Hot Spring ID	42.771	-115.719	15	25	42.2	59	8	13.5	19	1.2	2.0	2.9	5.6	6.6	7.5
13169500	Big Jacks Creek nr Bruneau ID	42.785	-115.983	41	22	42.4	57	6.2	12.0	16	1.6	3.0	4.2	3.6	4.0	4.7
13172500	Snake River nr Murphy ID	43.292	-116.42	13	170	193.1	210	41	45.4	49	16	19.4	21	2.2	2.3	2.7
13185000	Boise River nr Twin Springs ID	43.659	-115.726	13	18	29.5	34	6.3	10.5	12	0.48	0.8	1	11.1	12.7	15.7
13186000	Sf Boise River nr Featherville ID	43.496	-115.308	4	32	38.3	47	11	13.3	16	1	1.3	1.8	8.9	10.4	12.0
13200000	Mores Creek ab Robie Creek nr Arrowrock Dam ID	43.648	-115.989	4	23	35.5	44	7.2	11.3	14	1.1	1.7	2.1	6.5	6.6	6.7
13202000	Boise River nr Boise ID	43.519	-116.059	3	25	30.7	37	8.3	10.1	12	1	1.2	1.6	7.5	8.3	9.1
13203510	Boise R Bl Diversion Dam nr Boise ID	43.54	-116.094	10	22	32.1	39	7.57	10.8	13	0.838	1.2	1.5	8.1	8.9	10.0
13203760	Boise River at Eckert Rd nr Boise ID	43.566	-116.131	1	32	32	32	11	11.0	11	1.1	1.1	1.1	10.0	10.0	10.0
13204400	51n Storm Drain at Walnut Street at Boise ID	43.601	-116.187	3	91	103.7	120	29	32.3	37	4.5	5.6	6.7	5.4	5.8	6.4
13205300	44s Storm Drain @ Boise State U. at Boise ID	43.605	-116.203	3	16	51.3	100	5.2	16.7	32	0.75	2.4	5	6.4	7.5	9.3
13205505	39n Storm Drain at 9th Street at Boise ID	43.611	-116.208	3	34	76	130	10	24.7	42	2.1	3.1	5	4.8	7.7	10.0
13205518	43 St. Storm Drain at Garden City ID	43.631	-116.251	3	28	54.7	100	9.5	18.8	35	1	2.1	3.8	8.6	9.1	9.5
13205524	31n Storm Drain at Americana Blvd at Boise ID	43.616	-116.221	5	19	34	60	6.4	10.4	15	0.75	2.0	5.5	2.7	7.2	8.7
13205642	Boise R at Veterans Memorial Parkway at Boise ID	43.639	-116.246	2	46	46	46	15	15.0	15	2	2.1	2.1	7.1	7.3	7.5
13206000	Boise River at Glenwood Bridge nr Boise ID	43.66	-116.278	31	25	39.6	55	8.48	13.1	18	0.941	1.7	2.5	6.8	8.0	9.3
13206200	Boise River nr Eagle ID	43.675	-116.317	2	55	55.5	56	18	18.0	18	2.5	2.6	2.6	6.9	7.1	7.2
13206305	Boise River South Channel at Eagle ID	43.675	-116.354	2	69	74	79	22	23.5	25	3.5	3.8	4.1	6.1	6.2	6.3
13209500	Boise River South Channel at Linder Rd nr Eagle ID	43.674	-116.411	3	88	91	95	27	28.0	29	4.9	5.1	5.4	5.4	5.5	5.7
13209800	Boise R at Sundance Ranch nr Star ID	43.683	-116.461	2	66	78	90	21	24.5	28	3.2	4.1	4.9	5.7	6.1	6.6
13210050	Boise River nr Middleton ID	43.684	-116.573	9	34	66.2	86	11	21.1	27	1.5	3.3	4.6	5.9	6.4	7.3
13213000	Boise River nr Parma ID	43.782	-116.971	49	52	143.6	180	16	39.6	48	3	10.7	14	3.3	3.8	5.3
13213100	Snake River at Nyssa Or	43.877	-116.984	11	110	171.8	210	29	41.3	50	10	16.7	20	2.2	2.5	3.0
13235000	Sf Payette River at Lowman ID	44.085	-115.622	14	21	33.7	44	7.5	12.2	16	0.4	0.8	1.03	13.3	15.8	32.5

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
13239000	Nf Payette River at Mccall ID	44.908	-116.118	14	6	8.4	32	1.9	2.9	12	0.26	0.3	0.4	6.1	8.5	30.0
13240000	Lake Fork Payette River ab Jumbo Cr nr Mccall ID	44.914	-115.996	1	5	5	5	1.8	1.8	1.8	0.02	0.0	0.02	90.0	90.0	90.0
13245000	Nf Payette River at Cascade ID	44.525	-116.046	18	10	11.9	14	3	3.7	4.5	0.5	0.7	0.79	4.5	5.4	9.0
13250600	Big Willow Creek nr Emmett ID	44.074	-116.485	1	47	47	47	11	11.0	11	4.6	4.6	4.6	2.4	2.4	2.4
13251000	Payette River nr Payette ID	44.043	-116.924	18	19	48.1	81	6.2	14.2	23	0.95	3.1	5.7	3.9	5.0	6.5
13258500	Weiser River nr Cambridge ID	44.579	-116.643	6	23	36.8	48	5.9	9.3	12	2	3.3	4.4	2.6	2.8	3.3
13266000	Weiser River nr Weiser ID	44.268	-116.771	17	24	46.9	73	6	11.1	17	2.2	4.6	7.4	2.3	2.4	2.7
13269000	Snake River at Weiser ID	44.246	-116.98	57	78	148.5	210	15	35.9	49	6.8	14.0	20	1.1	2.6	3.2
13293800	Salmon River @ Hwy 93 Abv Redfish Cr nr Stanley ID	44.164	-114.886	10	39	54.7	68	14	19.4	24	1	1.5	1.9	11.7	12.7	14.0
13293900	Redfish Lake Creek Bl Lake nr Stanley ID	44.156	-114.911	2	11	11.5	12	3.6	3.9	4.2	0.4	0.4	0.4	9.0	9.8	10.5
13296000	Yankee Fork Salmon River nr Clayton ID	44.288	-114.72	2	22	27	32	7.4	9.2	11	0.9	1.0	1.1	8.2	9.1	10.0
13296500	Salmon River Bl Yankee Fork nr Clayton ID	44.268	-114.733	1	23	23	23	7.5	7.5	7.5	1.1	1.1	1.1	6.8	6.8	6.8
13297450	Little Boulder Creek nr Clayton ID	44.099	-114.447	1	23	23	23	8.2	8.2	8.2	0.57	0.6	0.57	14.4	14.4	14.4
13298000	Ef Salmon River nr Clayton ID	44.224	-114.286	1	46	46	46	15	15.0	15	2	2.0	2	7.5	7.5	7.5
13301510	Grouse Creek at Road Crossing nr May ID	44.447	-113.887	1	290	290	290	97	97.0	97	12	12.0	12	8.1	8.1	8.1
13301535	Sulphur Creek at Road Xing nr May 14n 21e 13aac1	44.549	-113.915	1	180	180	180	43	43.0	43	18	18.0	18	2.4	2.4	2.4
13302005	Pahsimeroi River at Ellis ID	44.525	-114.047	10	160	178	200	42	46.1	51	14	15.2	17	2.9	3.0	3.3
13302500	Salmon River at Salmon ID	45.184	-113.895	16	42	100.3	140	13	28.7	40	2.4	6.7	9.6	3.8	4.4	5.4
13305000	Lemhi River nr Lemhi ID	44.94	-113.639	16	120	186.9	240	30	46.8	62	11	17.0	21	2.1	2.7	3.1
13307000	Salmon River nr Shoup ID	45.322	-114.441	4	69	106.3	140	20	30.0	39	4.7	8.0	11	3.4	3.9	4.4
13309220	Mf Salmon River at Mf Lodge nr Yellow Pine ID	44.722	-115.016	2	34	36	38	12	12.5	13	0.94	1.1	1.3	10.0	11.4	12.8
13310700	Sf Salmon River nr Krassel Ranger Station ID	44.987	-115.724	5	7	10.2	13	2.4	3.8	4.9	0	0.2	0.34			
13313000	Johnson Creek at Yellow Pine ID	44.962	-115.499	14	12	33.1	44	3.9	10.5	14	0.45	1.6	2.2	5.9	6.7	8.7
13316500	Little Salmon River at Riggins ID	45.413	-116.325	13	18	46.9	74	5.6	14.3	23.1	0.9	2.7	3.95	4.0	5.3	6.3
13317000	Salmon River at White Bird ID	45.75	-116.324	99	22	57.4	92	6.9	16.8	26	1.1	3.7	6.5	3.3	4.8	7.5
13317046	Spring Abv Swartz Pond Near White Bird ID	45.805	-116.271	1	35	35	35	8.63	8.6	8.63	3.24	3.2	3.24	2.7	2.7	2.7
13317048	White Bird Cr at Bridge Abv Price Cr nr White Bird	45.778	-116.279	2	19	99.5	180	4.46	22.5	40.6	1.79	10.2	18.7	2.2	2.3	2.5

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
13336300	Gedney Creek nr Selway Falls Guard Station ID	46.058	-115.314	7	6	7.7	12	1.8	2.5	4.1	0.2	0.4	0.48	5.2	6.8	10.0
13336500	Selway River nr Lowell ID	46.087	-115.514	3	5	8	11	1.8	2.7	3.7	0.2	0.3	0.5	7.4	8.4	9.0
13337000	Lochsa River nr Lowell ID	46.151	-115.587	3	7	10	13	2.5	3.3	4.2	0.3	0.5	0.7	5.3	6.6	8.3
13338500	Sf Clearwater River at Stites ID	46.086	-115.977	15	9	19.4	33	2.7	5.3	8.5	0.6	1.5	2.8	3.0	3.6	4.5
13338650	Unnamed Spring Blw Nikesa Creek nr East Kamiah ID	46.21	-116.004	2	92	94.5	97	22	22.5	23	8.94	9.3	9.62	2.4	2.4	2.5
13339500	Lolo Creek nr Greer ID	46.372	-116.163	5	9	10	11	2.6	2.8	3	0.6	0.7	1	2.8	3.9	4.4
13341300	Bloom Creek nr Bovill ID	46.858	-116.291	1	24	24	24	6.8	6.8	6.8	1.7	1.7	1.7	4.0	4.0	4.0
13341500	Potlatch River at Kendrick ID	46.612	-116.658	1	35	35	35	10	10.0	10	2.3	2.3	2.3	4.3	4.3	4.3
13342450	Lapwai Creek nr Lapwai ID	46.427	-116.804	18	54	100.9	150	14	25.7	38.9	4.5	9.1	13.3	2.6	2.9	3.1
13342490	Lapwai Creek at Spalding ID	46.448	-116.816	3	49	99.7	130	0.02	18.7	31.1	0.002	6.9	11.6	2.7	2.7	2.9
13342500	Clearwater River at Spalding ID	46.449	-116.826	98	7	14.3	29	2.2	4.2	7.8	0.3	0.9	2.2	3.4	4.7	9.3
13344800	Deep Creek nr Potlatch ID	46.961	-116.934	1	49	49	49	14	14.0	14	3.4	3.4	3.4	4.1	4.1	4.1
13345000	Palouse River nr Potlatch ID	46.915	-116.95	18	14	26.2	37	3.8	7.3	11	1.2	1.9	2.5	3.2	3.8	4.8
13346800	Paradise Cr at University Of Idaho at Moscow ID	46.732	-117.023	1	160	160	160	44	44.0	44	11	11.0	11	4.0	4.0	4.0
124131265	Ef Ninemile Creek Abv Success Mine nr Blackcloud	47.53	-115.874	1	24	24	24	7.66	7.7	7.66	1.21	1.2	1.21	6.3	6.3	6.3
124131267	Ef Ninemile Creek nr Blackcloud, ID	47.524	-115.88	1	10	10	10	3.23	3.2	3.23	0.526	0.5	0.526	6.1	6.1	6.1
130626914	Angus Creek Reservoir	42.827	-111.4	2	290	335	380	78.6	92.3	106	22.4	25.3	28.2	3.5	3.6	3.8
130626924	Angus Creek 0.7 Miles Blw Angus Cr Res nr Henry ID	42.835	-111.407	1	250	250	250	70.3	70.3	70.3	18.4	18.4	18.4	3.8	3.8	3.8
133170462	Outflow From Swartz Pond nr White Bird ID	45.801	-116.271	1	150	150	150	23.8	23.8	23.8	21.9	21.9	21.9	1.1	1.1	1.1
1313457010	Bell Rapids Mutual Irr Co Pumping Plnt nr Hagerman	42.83	-114.937	1	200	200	200	48	48.0	48	20	20.0	20	2.4	2.4	2.4
422750114251201	High Line Canal Near Twin Falls Airport	42.464	-114.42	11	150	158.2	170	35.4	41.2	44.5	11.2	13.1	14.6	2.5	3.2	3.6
431854114091200	Silver Creek at The Nature Conservancy Preserve	43.315	-114.153	1	190	190	190	57.2	57.2	57.2	12.2	12.2	12.2	4.7	4.7	4.7
472721116480100	Coeur D Alene Lake Btwn Harrison And Harlow Point	47.456	-116.8	4	17	28.3	38	4.35	7.1	9.49	1.48	2.6	3.53	2.5	2.8	2.9
472839115545001	Canyon Creek Seepage Site No. A-7	47.478	-115.914	2	47	48.5	50	13.2	13.5	13.8	3.42	3.5	3.65	3.8	3.8	3.9
472852115541401	Canyon Creek Seepage Site No. A-6	47.481	-115.904	2	47	48	49	13.3	13.5	13.7	3.48	3.5	3.54	3.8	3.8	3.9
472905115534301	Canyon Creek Seepage Site No. A-4	47.485	-115.895	1	43	43	43	12	12.0	12	3.08	3.1	3.08	3.9	3.9	3.9
472931115531501	Canyon Creek Seepage Site No. A-1.2	47.492	-115.888	1	43	43	43	12	12.0	12	3.06	3.1	3.06	3.9	3.9	3.9

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
472931115581201	Sf Coeur D Alene River Seepage Site No. B-1	47.492	-115.97	3	46	60.3	69	12.5	16.2	18.4	3.6	4.9	5.59	3.3	3.3	3.5
473005115593201	Sf Coeur D Alene River Seepage Site No. B-1.1	47.501	-115.992	1	67	67	67	17.8	17.8	17.8	5.44	5.4	5.44	3.3	3.3	3.3
473007115585601	Sf Coeur D Alene River Inflow Pipe Ds Of B-1.1	47.502	-115.982	1	170	170	170	33.5	33.5	33.5	20	20.0	20	1.7	1.7	1.7
473019115523501	Canyon Creek Seepage Site No. A-1	47.505	-115.876	2	41	42.5	44	11.6	11.9	12.2	2.98	3.1	3.17	3.8	3.9	3.9
473022115592001	Sf Coeur D Alene River Seepage Site No. B-2	47.506	-115.989	3	46	61	70	12.2	16.2	18.6	3.65	4.9	5.62	3.3	3.3	3.3
473037116004101	Sf Coeur D Alene River Seepage Site No. B-5	47.51	-116.011	3	48	62	70	12.9	16.5	18.6	3.84	5.0	5.67	3.2	3.3	3.4
473059116013901	Sf Coeur D Alene River Seepage Site No. B-7	47.516	-116.028	3	45	60	69	12	16.1	18.5	3.58	4.9	5.61	3.3	3.3	3.4
473107116020901	Sf Coeur D Alene River Seepage Site, Rosebud Gulch	47.519	-116.036	1	74	74	74	18.7	18.7	18.7	6.55	6.6	6.55	2.9	2.9	2.9
473107116021301	Sf Coeur D Alene River Seepage Site No.B-8	47.519	-116.037	3	49	62.3	70	13.2	16.7	18.7	3.94	5.1	5.7	3.2	3.3	3.4
473208116064501	Sf Coeur D Alene River Seepage Site No. C-1	47.536	-116.113	2	48	59	70	13	15.7	18.3	3.85	4.8	5.8	3.2	3.3	3.4
473210116070601	Sf Coeur D Alene River Seepage Site, Milo Creek	47.536	-116.118	2	35	35.5	36	9.59	9.9	10.3	2.22	2.6	2.88	3.3	4.0	4.6
473251116101701	Sf Coeur D Alene River Seepage Inflow, Govt Gulch	47.548	-116.171	1	27	27	27	7.55	7.6	7.55	1.86	1.9	1.86	4.1	4.1	4.1
473252116095301	Bunker Cr at Mouth Of Culvert at Kellogg, ID	47.548	-116.165	1	2100	2100	2100	594	594.0	594	145	145.0	145	4.1	4.1	4.1
473252116101101	Sf Coeur D Alene River Seepage Site No. C-6	47.548	-116.17	3	65	98.3	120	17.3	26.4	32.6	5.19	8.2	9.96	3.1	3.2	3.3
473253116094001	Sf Coeur D Alene River Seepage Site No. C-5	47.548	-116.161	2	54	66.5	79	14.3	17.5	20.7	4.44	5.6	6.7	3.1	3.2	3.2
473253116130901	Sf Coeur D Alene River Seepage Site No. C-10	47.548	-116.219	3	76	105.3	120	20.4	27.8	31.6	6.17	8.9	10.2	3.1	3.2	3.3
473259116122301	Sf Coeur D Alene River Seepage Site No. C-9	47.55	-116.206	1	75	75	75	20.1	20.1	20.1	6.03	6.0	6.03	3.3	3.3	3.3
473302116115901	Sf Coeur D Alene River Seepage Site No. C-8	47.551	-116.2	3	74	124.7	180	19.8	33.9	49.8	5.87	10.1	14.4	3.2	3.3	3.5
473328115545601	Beaver Cr. ab Ferguson Cr nr Delta, ID	47.558	-115.916	16	13	27.6	50	3.68	8.3	15.2	0.72	1.7	3.07	4.3	5.1	7.6
473329115541800	Dobson Creek	47.558	-115.905	1	22	22	22	6.9	6.9	6.9	1.18	1.2	1.18	5.8	5.8	5.8
473330115541500	Bc12-Old	47.558	-115.904	2	33	34.5	36	10.5	11.4	12.2	1.39	1.5	1.52	6.9	7.8	8.8
473344115525600	Cc4-Adit	47.562	-115.882	2	120	125	130	39.5	42.5	45.5	3.61	3.6	3.68	10.7	11.7	12.6
473344115531400	Cc6-Mid	47.562	-115.887	2	16	24	32	5.3	8.0	10.7	0.73	0.9	1.15	7.3	8.3	9.3
473345115524500	Cc2-Above	47.563	-115.879	2	10	13.5	17	3	4.1	5.1	0.63	0.9	1.07	4.8	4.8	4.8
473347115534600	Bc10-Mid	47.563	-115.896	2	10	13.5	17	3.2	4.4	5.6	0.58	0.7	0.77	5.5	6.4	7.3
473348115533600	Pioneer Creek	47.563	-115.893	1	12	12	12	3.7	3.7	3.7	0.72	0.7	0.72	5.1	5.1	5.1
473349115532201	Carbon Cr ab Mouth nr Delta, ID	47.564	-115.889	7	16	55.6	82	5.2	18.2	27.1	0.72	2.5	3.52	7.1	7.6	9.4

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
473350115532201	Beaver Cr ab Carbon Cr nr Delta, ID	47.564	-115.889	3	7	11	16	2.1	3.2	4.5	0.49	0.8	1.23	3.7	4.4	5.3
473356115515201	Carbon Cr Bl Headwaters nr Delta, ID	47.566	-115.864	1	23	23	23	6.14	6.1	6.14	1.76	1.8	1.76	3.5	3.5	3.5
473404115554801	Beaver Cr ab No Name Gulch nr Delta, ID	47.568	-115.93	1	52	52	52	14.7	14.7	14.7	3.77	3.8	3.77	3.9	3.9	3.9
473421115522200	Ubc5-Mid	47.573	-115.873	2	7	8	9	2	2.4	2.7	0.46	0.5	0.57	4.3	4.5	4.7
473423115520300	Ubc3-Above	47.573	-115.868	2	7	8	9	2	2.4	2.8	0.45	0.5	0.57	4.4	4.7	4.9
473505115555601	Pony Gulch Cr ab Mouth nr Delta, ID	47.585	-115.932	1	50	50	50	11.3	11.3	11.3	5.36	5.4	5.36	2.1	2.1	2.1
473525115440301	Prichard Cr ab Jo Gulch nr Murray, ID	47.59	-115.734	1	7	7	7	1.98	2.0	1.98	0.556	0.6	0.556	3.6	3.6	3.6
473532115475301	Granite Gulch Cr Bl Moonshine Gulch nr Murray, ID	47.592	-115.798	1	12	12	12	3.45	3.5	3.45	0.77	0.8	0.77	4.5	4.5	4.5
473541115453201	Prichard Cr ab Monarch Gulch nr Murray, ID	47.595	-115.759	1	7	7	7	2.01	2.0	2.01	0.484	0.5	0.484	4.2	4.2	4.2
473545115451201	Paragon Gulch Creek Abv. Mouth nr Murray, ID	47.596	-115.753	2	13	13	13	3.45	3.5	3.53	0.952	1.0	0.979	3.6	3.6	3.6
473551115474201	Granite Gulch Cr ab Mouth nr Murray, ID	47.598	-115.795	1	10	10	10	3.06	3.1	3.06	0.686	0.7	0.686	4.5	4.5	4.5
473553115473901	Prichard Cr Abv Confluence Of Granite Cr nr Raven	47.598	-115.794	1	12	12	12	3.48	3.5	3.48	0.877	0.9	0.877	4.0	4.0	4.0
473554115473601	Prichard Cr ab Granite Gulch Cr nr Delta, ID	47.598	-115.793	1	11	11	11	3.2	3.2	3.2	0.812	0.8	0.812	3.9	3.9	3.9
473555115561701	Beaver Cr Bl Gleveland Gulch nr Delta, ID	47.599	-115.938	1	38	38	38	9.88	9.9	9.88	3.27	3.3	3.27	3.0	3.0	3.0
473605115475401	Bear Gulch Cr ab Mouth nr Murray, ID	47.601	-115.798	1	7	7	7	1.95	2.0	1.95	0.506	0.5	0.506	3.9	3.9	3.9
473630115562901	Trail Cr ab Mouth nr Delta, ID	47.608	-115.941	1	76	76	76	19	19.0	19	6.86	6.9	6.86	2.8	2.8	2.8
473641115492701	Prichard Cr Bl ID Gulch nr Murray, ID	47.611	-115.824	1	10	10	10	2.74	2.7	2.74	0.715	0.7	0.715	3.8	3.8	3.8
473648115493501	Butte Gulch Cr nr Muuray, ID	47.613	-115.826	1	15	15	15	4.13	4.1	4.13	1.05	1.1	1.05	3.9	3.9	3.9
473655115470201	Bear Gulch Cr nr Round Top Mtn nr Murray, ID	47.615	-115.784	1	7	7	7	1.83	1.8	1.83	0.531	0.5	0.531	3.4	3.4	3.4
473702115572501	Beaver Cr Bl Prospect Gulch nr Delta, ID	47.617	-115.957	1	44	44	44	10.8	10.8	10.8	4.02	4.0	4.02	2.7	2.7	2.7
473732115513001	Prichard Cr at Murray, ID	47.626	-115.858	12	5	9.2	13	1.27	2.5	3.5	0.361	0.7	0.945	3.5	3.6	3.8
473840115551701	Prichard Cr Abv Eagle Cr at Eagle, ID	47.644	-115.921	1	13	13	13	3.4	3.4	3.4	1.08	1.1	1.08	3.1	3.1	3.1
473841115551601	Eagle Cr Abv Mouth at Eagle, ID	47.645	-115.921	1	15	15	15	4.04	4.0	4.04	1.29	1.3	1.29	3.1	3.1	3.1
473925115530200	Ef Eagle Creek nr Mouth nr Prichard, ID	47.657	-115.884	1	12	12	12	2.92	2.9	2.92	1.04	1.0	1.04	2.8	2.8	2.8
473930115530101	Ef Eagle Cr Abv Fancy Gulch nr Eagle, ID	47.658	-115.884	12	5	9.8	15	1.23	2.5	3.72	0.429	0.9	1.31	2.8	2.9	3.0
474011115450401	Tributary Cr Bl Headwaters nr Jack Waite Forks, ID	47.67	-115.751	1	83	83	83	17.4	17.4	17.4	9.5	9.5	9.5	1.8	1.8	1.8
474017115530601	Wf Eagle Cr Abv Nocelly Gulch nr Eagle, ID	47.671	-115.885	12	6	9.7	13	1.54	2.6	3.61	0.509	0.8	1.08	3.0	3.2	3.3

Station ID	Descriptive Name	latdd	longdd	Count	Min. H	Ave. H	Max H	MinCa	AveCa	MaxCa	MinMg	AveMg	MaxMg	MinCa/Mg	AveCa/Mg	MaxCa/Mg
474041115484401	Ef Eagle Cr Bl Toboggan Cr nr Jack Waite Forks, ID	47.678	-115.812	1	14	14	14	3.36	3.4	3.36	1.34	1.3	1.34	2.5	2.5	2.5
474111115465201	Upper Ef Eagle Cr Blw Trib Cr nr Jack Waite Fork	47.686	-115.781	1	22	22	22	5.27	5.3	5.27	2.13	2.1	2.13	2.5	2.5	2.5
474118115463101	Prichard Creek Blw Paragon Creek nr Murray, ID	47.688	-115.775	2	6	6	6	1.8	1.8	1.8	0.406	0.4	0.407	4.4	4.4	4.4
474118115463201	Tributary Cr ab Mouth at Jack Waite Forks, ID	47.688	-115.776	5	23	24.6	29	5.27	5.7	6.65	2.31	2.5	2.97	2.2	2.2	2.3
474212115513501	West Fork Eagle Creek Abv Bobtail Cr nr Eagle, ID	47.703	-115.86	4	12	12.3	13	3.18	3.2	3.32	0.988	1.0	1.05	3.2	3.2	3.2
	Total observations			3594												
		Minimum			4	4.7										
		1st-percentile		4.23	4.7											
		5th percentile		6.15	4.7											

Appendix B

How to measure insignificance? Comparisons between NOECs, EC1s, and EC0s and the lower confidence limit of EC10s to estimate "insignificant effects"

Summary

To assist in our analysis, NMFS considered what toxicity test statistic best approximated a "true" no-effect concentration for evaluating risks to ESA listed species, We made a comparison of "no-observed effect concentrations" (NOECs) versus regression or distribution based methods for estimating no- or very low effects concentrations. The alternative statistics were regression- or distribution based estimates of the EC1 or EC0 (i.e., concentrations causing adverse effects to 1% or 0% of a test population), and the lower 95th percentile confidence limit of the concentration affecting 10% of the test population (LCL- EC10), which is a statistic used in human health risk assessment for determining benchmark doses of materials that present low increased risk (EPA, 2000a), Our conclusion was that if the data sets had a gradient of effects that would allow calculation of an EC0, the EC0 would be the preferred, best estimate of no-effect value from a toxicity test. If data were insufficient to calculate an EC0, the NOEC may be the best appropriate statistic.

The problem

In evaluations of the risks of chemicals to aquatic species listed as threatened or endangered, the statistical interpretation of toxicity testing has become an issue. Classically, the interpretation of chronic or sublethal tests has involved the use of statistical hypothesis testing, the results of which are commonly reported as "no-observed effect concentration" (NOEC) or "lowest-observed effect concentration" (LOEC). Definitions vary, but for this analysis the LOEC will be considered the lowest concentration for which there is a 95% probability that the biological response of interest (survival, growth, fecundity, etc.) is different from the control response. Similarly, the NOEC is considered the next lowest treatment. It has been assumed that somewhere between the NOEC and LOEC lies a maximum acceptable toxicant concentration (MATC) that represents a "true" but unknown threshold for unacceptable effects. In practice, the MATC concentration is estimated as a simple geometric mean between the NOEC and LOEC (Gelber *et al.*1995). This is the value usually used in EPA criteria documents to estimate "safe" concentrations from a chronic toxicity test, although the term "MATC" is avoided in the Guidelines and instead the statistic is called a "chronic value" for a test. MATCs in turn are averaged to obtain species mean chronic values, and ultimately to set chronic criteria values.

The EPA criteria approach seems to conflict with concepts for evaluating risk to listed species because the EPA approach of averaging NOECs and LOECs assumes that aquatic communities are resilient to, or can recover from, some low-level of adverse effects. In contrast, if a species was listed as threatened or endangered, it is assumed to have substantially less resiliency than general aquatic communities. Therefore, in interpreting

toxicity test data, a statistic that by definition includes some uncertain but probably low level of adverse effect such as the EPA "chronic value" is inappropriate as a statistic of effects on listed species that are expected to be discountable or insignificant. In the ESA Consultation Handbook for evaluating effects of actions to listed species, states that" *' insignificant effects' relate to the size of the impact and should never reach the scale where take occurs. Discountable effects are those extremely unlikely to occur. Based on best judgment, a person would not: (1) be able to meaningfully measure, detect, or evaluate insignificant effects; or (2) expect discountable effects to occur." (USFWS and NMFS 1998).* Thus a meaningful measurement of low-effects from a toxicity test such as an EC10 or EC5 is inherently in conflict with a definition that requires "insignificant" effects to be unmeasurable.

An obvious substitute for use in ESA consultations is the NOEC, and indeed that is the default statistic selected in EPA's methodology for conducting biological evaluations of aquatic life criteria (EPA 2003). However, in recent years the concept of the NOEC has been battered in the ecotoxicology literature. The three complaints relate to the common design of toxicity experiments which usually involve a series of about five treatment concentrations plus a control, each replicated about three times. Complaint #1 is that a NOEC has to be one of the concentrations tested, so its precision is dependent on the number and spacing of treatment concentrations. So for example, if the unknown "true" no-effect concentration is 1.8 µg/L a test series of 1.0, 1.2, 1.6, 2.0, …. will give a more precise NOEC estimate than a series of 1, 2, 4, 8, 16, ….(1.6 vs. 1.0 µg/L). Complaint #2 is that for the low levels of replication used (often 3), the minimum statistically detectable effect level can vary widely, easily from 5 to about 40% for endpoints with low or high variability (e.g. growth in fish (low) or fecundity in invertebrates (highly variable). The NOEC statistic by itself gives no insight into whether a "significant" effect is biologically trivial or whether an effect is biologically serious but too variable to be significant at the arbitrary limit that no more than a 5% risk of being wrong is acceptable (acceptable to the evaluators, not whether it is acceptable to the organism). Complaint #3 is related in that the NOEC-LOEC approach is solely focused on the "Type I" error, or the risk of declaring an adverse effect when the observed effects occurred solely by chance, with no or little regard for Type II error, the risk of failing to detect an adverse effect that was really present but the test had insufficient power to detect it. Type II error rates may be quite high in ecotoxicological studies that fail to detect effects as "significant" at the 5% Type I error rate (Stephan and Rogers, 1985; Laskowski, 1995; Moore and Caux, 1997; Crane and Newman, 2000; McGarvey, 2007; Newman, 2008; Brosi and Bilber, 2009).

An alternative often put forth to the NOEC-LOEC approach is regression or distribution based techniques that fit an effects curve to the observed data, and then any point along that curve can be used to estimate effects at a given concentration. This regression or distribution based approach is the most common technique for defining LC_{50}s in acute data but obviously other effect concentrations percentiles (ECp) besides the 50^{th} percentile could be of interest. The catch in this approach is that it is up to the assessor to independently determine what level of effect is "important." Choices of what level of effect is "important" have either been made subjectively or by comparisons of ECp values back to NOECs and LOECs. For example, in the interpretation of EPA's chronic

whole-effluent toxicity (WET) tests, NOECs are assumed to be equivalent to an EC25. The conclusion that a 25% adverse effect in a biologically important endpoint therefore represents a no-observed effect concentration was supported by a citation to an analysis of 23 pooled chronic WET test results for red algae, sheepshead minnows, sea urchins, *Ceriodaphnia*, and fathead minnows in which NOECs were more frequently similar to EC20s (EPA, 1991, p. 27). No reason was given why the EC25 was endorsed over the EC20, since the analysis supported the use of the EC20, but regardless the EC25 is often the trigger statistic in WET tests.

Subsequent analyses have also shown that NOECs are usually higher than point estimates of low toxic effects such as the EC10 (Moore and Caux, 1997; Crane and Newman, 2000). In an analysis limited to the effects of cadmium, NMFS found that the typical expected adverse effect associated with MATC was often about 20-30% with invertebrates and about 10-15% for fish (Mebane, 2006). However, using ECx values that correspond with a NOEC or MATC to select "x" as a suitable replacement for the unsuitable NOEC falls into circular reasoning. A counterpoint could be made that comparisons of ECps and NOECs to support an ECp value to replace NOECs is a tautology. Instead of matching statistics, biological arguments could be made for assuming different "acceptable" ECp values based upon patterns of variability of the same endpoints in natural populations, life history strategies, projecting effects in population models, and field studies relating year class survival to size differences. No comprehensive analysis along these lines is known to have been published.

Mebane and Arthaud (2010) gave an example of what effect-statistics could be related to population extinction risks or recovery trajectories for a headwaters threatened Chinook salmon population. In this population, Marsh Creek in the upper Salmon River, Idaho, survival of juvenile migrants is strongly related to the size of the fish. A size reduction of 4% as length, i.e., an EC04, was associated with survival reductions ranging from 12 – 38% for different migrant groups from a trap near the headwaters to the first dam encountered downstream. In the toxicity tests with Chinook salmon and rainbow trout that were analyzed for the study, a 4% reduction in length corresponded with about a 12% reduction in weight. When the survival reductions associated with a length EC04 were extrapolated through a population model to changes in extinction risk or recovery time, little difference in extinction risk was projected but an appreciable delay in recovery was projected. This indicates that at least for the length endpoint in chronic fish toxicity tests, the statistical threshold for important adverse effects may not be much higher than statistics such as an EC0 or EC01. Yet for the commonly used weight endpoint in chronic fish toxicity tests, the statistical threshold for important adverse effects would be higher, around the EC10. Presumably, if endpoints are more variable, such as the number of eggs produced per female (fecundity), then a higher ECp value (e.g. EC20) might be appropriate. While the relevance of this example to other species or even different populations of Chinook salmon is not known, it does at least serve as one example of a basis to judge the importance of an ECp value without relying on circular comparisons back to other statistics.

Comparisons between statistics

For this exercise, NMFS evaluated data from a variety of available toxicity tests results that were available in the syntax required by the statistics models. While such data are not comprehensive or necessarily definitive, they are preferable to many journal articles because the latter are sometimes too summarized to make any subsequent analysis of. We selected the examples to illustrate a variety of response patterns ranging from classic, concentration-responses to test results that are difficult to interpret.

NMFS used either reported NOECs or those that could be estimated using Dunnett's test. ECp values were estimated for growth and reproduction using a distribution analysis for survival data (respondents are either alive or dead) or nonlinear regression for more or less continuous data (growth or fecundity measurements). For each type of analysis, a choice of underlying distributions of the populations must be assumed.

(1) Gaussian (Normal) Distribution: This is based on the familiar "bell curve" or gaussian distribution. This produces a sigmoidal toxicity relationship with infinite tails, and is equivalent to prohibit analysis.

(2) Triangular Distribution: This produces a sigmoidal toxicity relationship similar to the gaussian distribution, but with a finite threshold exposure below which responses are zero and a finite exposure above which all organisms are affected. It is also referred to as a "sigmoid threshold" (Erickson 2008).

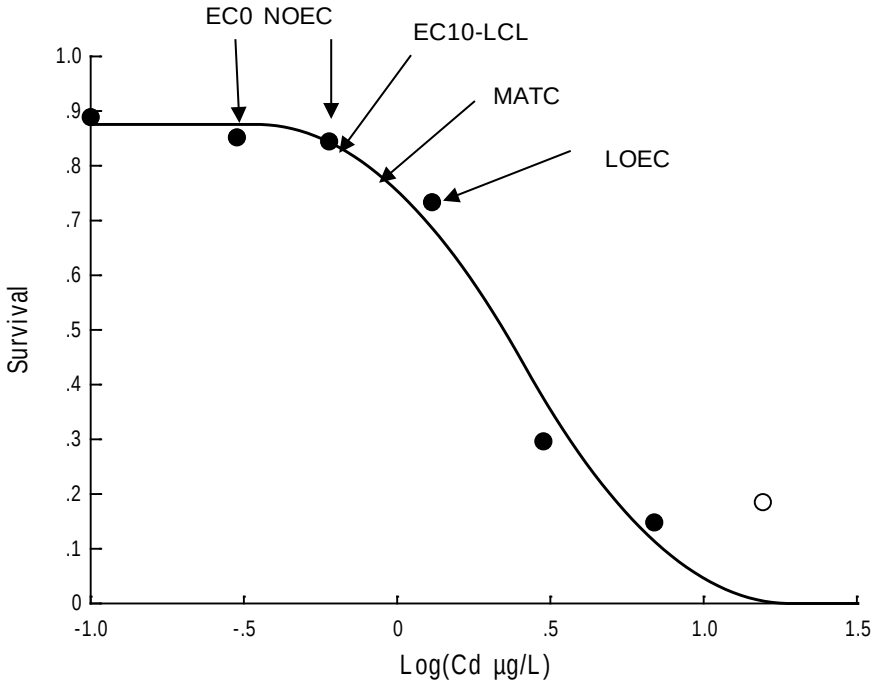
(3) Uniform (Rectangular) Distribution: This produces a piecewise-linear toxicity relationship, for which there is a finite lower and upper exposure limit like the triangular distribution, but for which the decline in response between these limits is linear rather than sigmoidal. Similar analyses have been called "jackknife distributions" in the literature because of its shape.

The assumed statistical distribution and behavior of the data in the tails of the distribution are usually of little consequence when one is trying to estimate the middle of the distribution (LC_{50}). However, when one is trying to estimate no-effects data, these estimates are at the extreme tails of the distribution, and the shape of the tails and the behavior of the models become more important. In the Gaussian, normal distribution, an EC0 can never be achieved because the tails are infinite; in other words some rare organisms are assumed to be infinitely resistant and some sensitive to infinitesimal exposures. Because that assumption is not plausible for ecotoxicology data, methods have been developed using discrete distributions with definite ends, i.e. no organism is infinitesimally sensitive, and an EC0 can be calculated.

NMFS calculations used a beta version of the Toxicity Response Analysis Program, under development EPA's National Health and Environmental Research Laboratory, Mid-Continent Ecological Division (Erickson 2008).

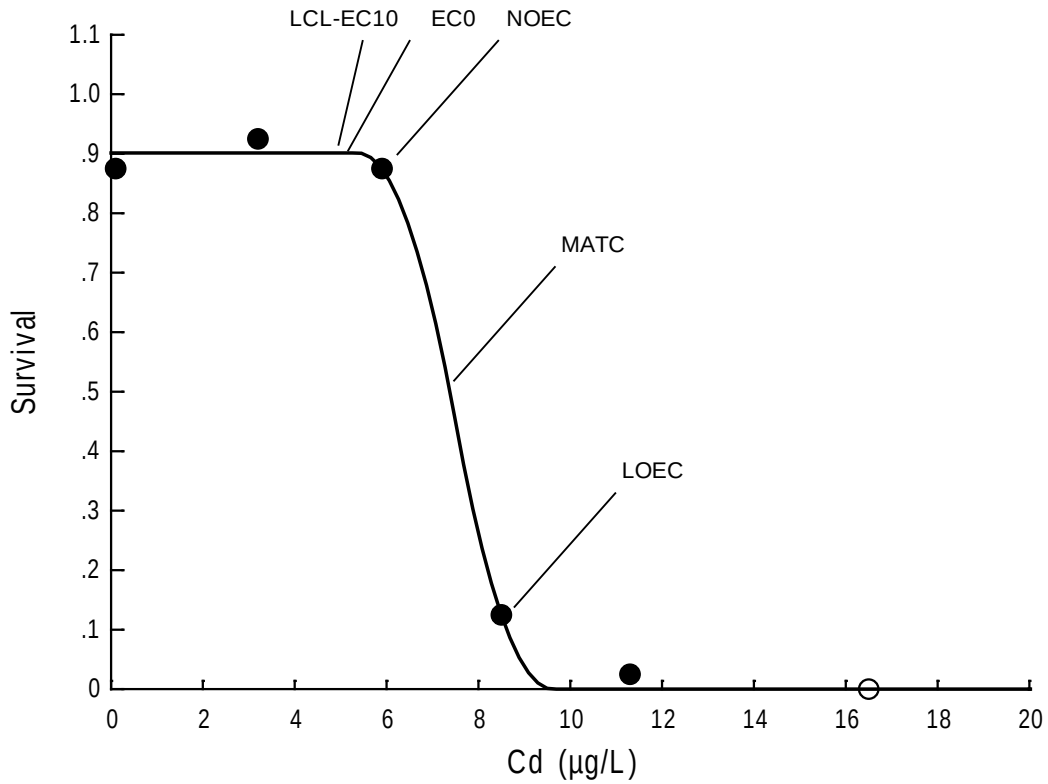
Examples:

Example 1. Rainbow trout 53-day survival with cadmium, using the sigmoid threshold model based upon an assumed triangular distribution. Open circles indicate data points that were excluded from the regression



ECp	ECp est	95 LCL	95% UCL
10	0.85	0.62	1.17
0	0.35	0.21	0.59

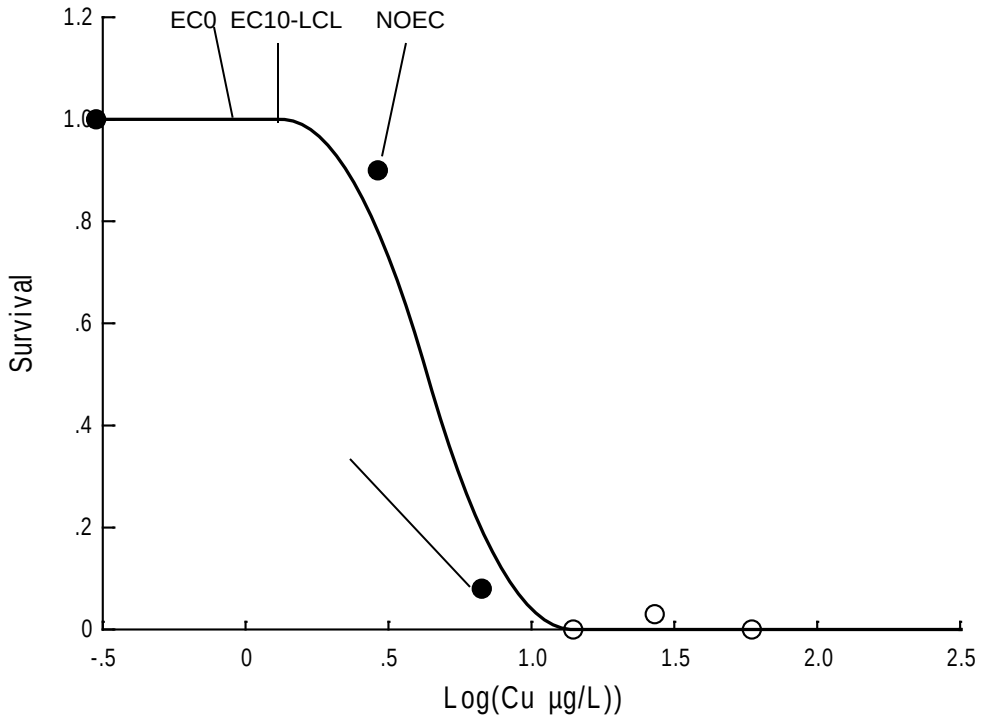
Example 1 was selected to illustrate the classic ski jump curve shape, where the initial part of the curve from the control out to the 2nd treatment shows a slight decline, followed by a steep drop in the center region of the curve where intermediate effects occur, followed by a flattening out of the slope at the bottom as almost all animals are predicted to be killed (Mebane *et al.*, 2008).



Example 2. Fountain darter, 7-day survival with Cd, sigmoid threshold, showing a very steep curve that results with (nearly) all-or-nothing responses. In this case, all of the "nearly-no-effect" estimators give similar values.

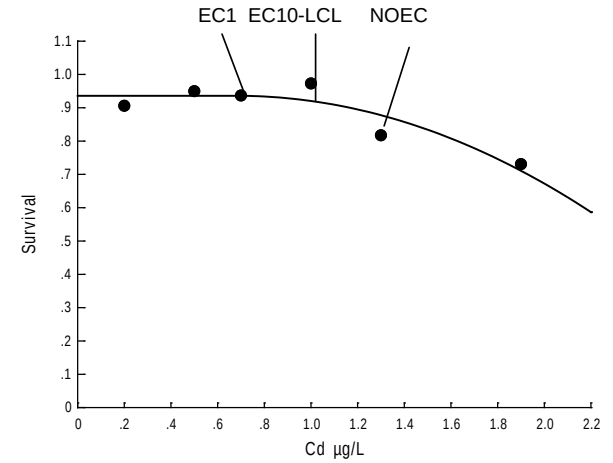
ECp	ECp est	95 LCL	95% UCL
10	6.33	5.06	7.59
0	5.38	2.84	7.92

(Castillo and Longley, 2001)

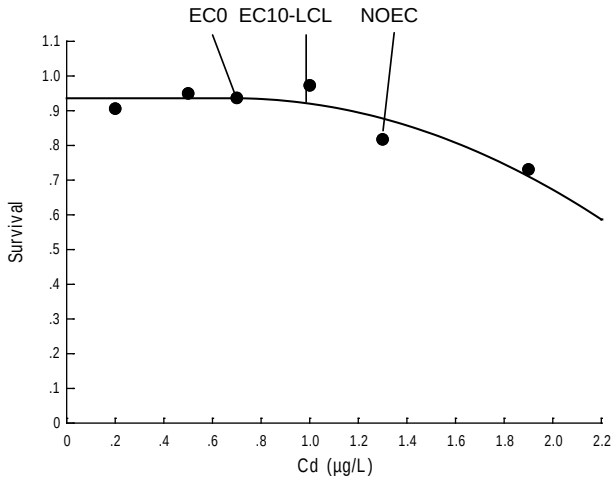


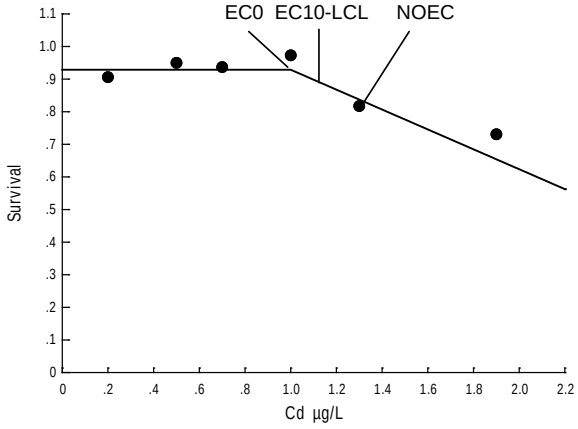
Example 3. Mottled sculpin, 14-day survival with copper (Besser and others, 2007). As with example 2, these data had inadequate partial responses resulting in an uncertain fit between the control and treatment 1, the NOEC. Even so, ECp estimates are reasonable and confidence limits are not large. These type of data are often encountered working with listed species or other poorly tested species for which investigators have little idea in advance what exposure change to test.

ECp	ECp est	95 LCL	95% UCL
10	2.255	1.841	2.762
5	1.934	1.516	2.466
0	1.334	0.924	1.925



LOEC





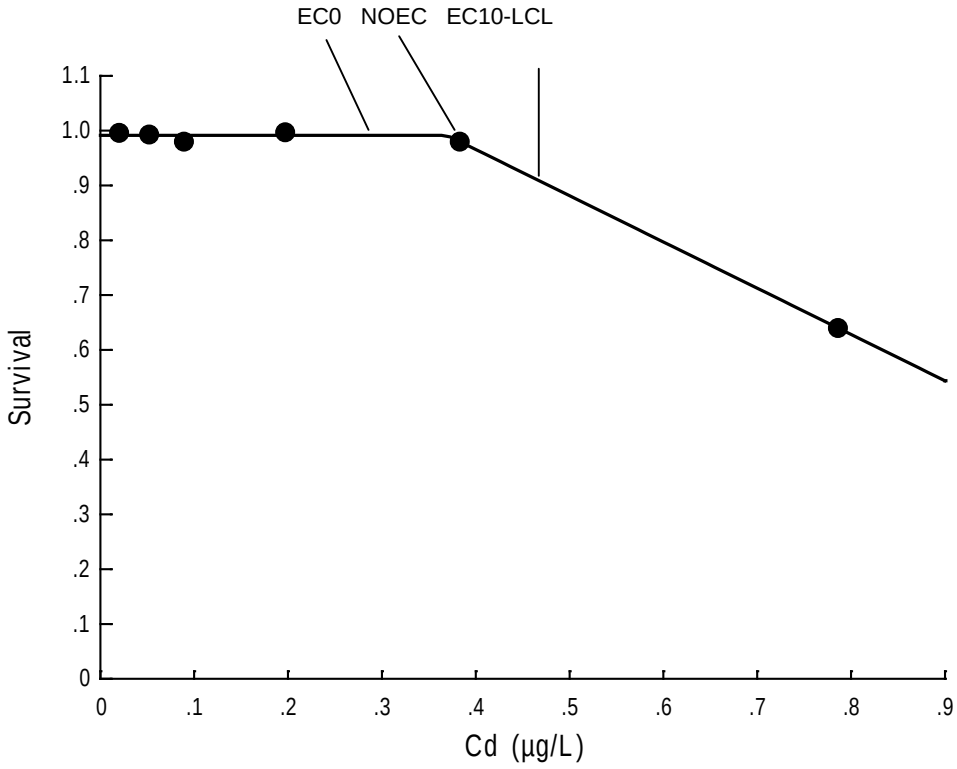
Example 4. Chinook salmon 120 day survival with Cd (Chapman, 1982), illustrating differing ECp estimates resulting from different statistical models. Note that EC0s are conceptually impossible using the normal distribution, but the EC1 in the top figure is close to the EC0 in the middle figure using the triangular distribution. In this example, the linear model (bottom) does the best job of finding the no-effect estimate (visually, treatment 3, the 4th point from the left). Despite the very different underlying models, all ECp estimates were similar in this example.

Chinook and Cd ECp values

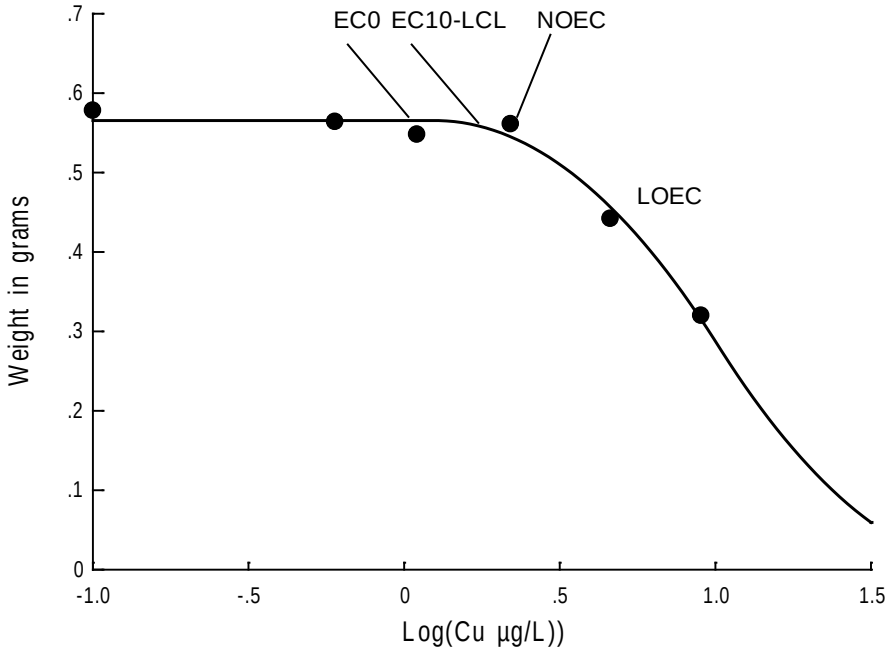
Gaussian	ECp est	95 LCL	95% UCL
20	1.802	1.541	2.063
10	1.480	1.042	1.919
5	1.215	0.503	1.927
1	0.717	-0.789	2.223
Triangular			
20	1.792	1.529	2.056
10	1.466	1.144	1.788
5	1.236	0.764	1.707
0	0.679	-0.454	1.811
Rectagular			
20	1.609	1.366	1.852
10	1.304	1.114	1.495
5	1.152	0.953	1.352
0	1.000	0.763	1.237

Bull trout and Cd ECp values:

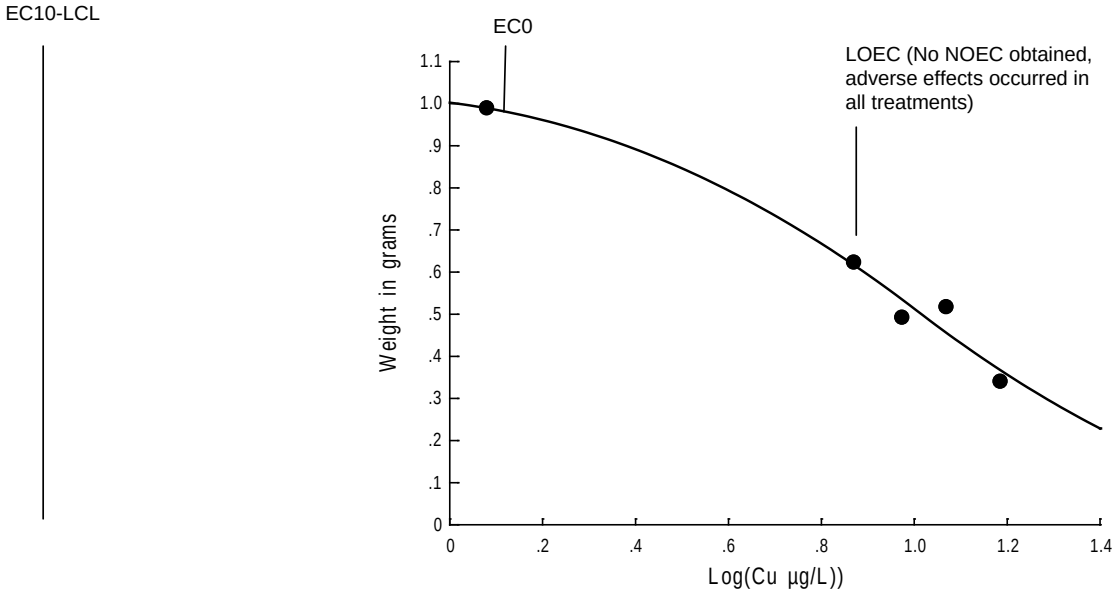
ECp	ECp est	95 LCL	95% UCL
10	0.555	0.477	0.633
5	0.479	0.379	0.578
0	0.294	0.117	0.471
20	0.60496	0.58075	
10	0.48715	0.46026	
5	0.42824	0.39815	
0	0.36933	0.33522	



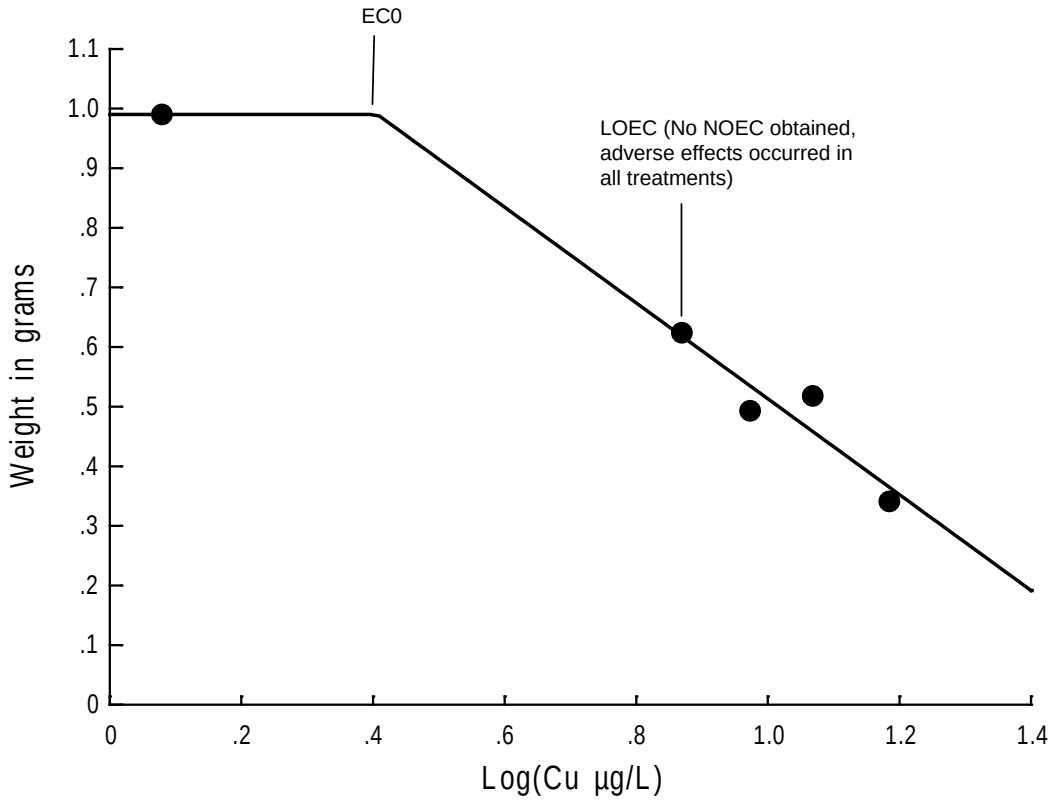
Example 5. Bull trout, 55-day survival with Cd (Hansen and others, 2002c). Here the NOEC is lower than the LCL-EC10. Similar to the Chinook salmon and Cd example, these data would give an inadequate and highly unreliable response for an LC50. However, with chronic testing the interest is in the low-effect part of the curve.



Example 6. Growth of rainbow trout after 60-days Cu exposure (Marr and others, 1996). This data set is nicely balanced with 3 nearly no-effect treatments and 2 treatments above a clearly defined effects threshold.



Example 7. Growth of Chinook salmon after 120-days Cu exposure, sigmoid threshold model (Chapman, 1982). This data set presents uncertain EC0 values because adverse effects occurred in all tested treatments. The LCL-EC10 is less than zero which is clearly impossible and using the sigmoid model, the EC0 falls close to the control. There is no NOEC, although in some data compilations the "less than" for this treatment was lost in translation and the NOEC or chronic value has been treated as 7.4 µg/L rather than < 7.4 µg/L. This mistake results in a 40% reduction in growth being treated as a low- or no-effect.



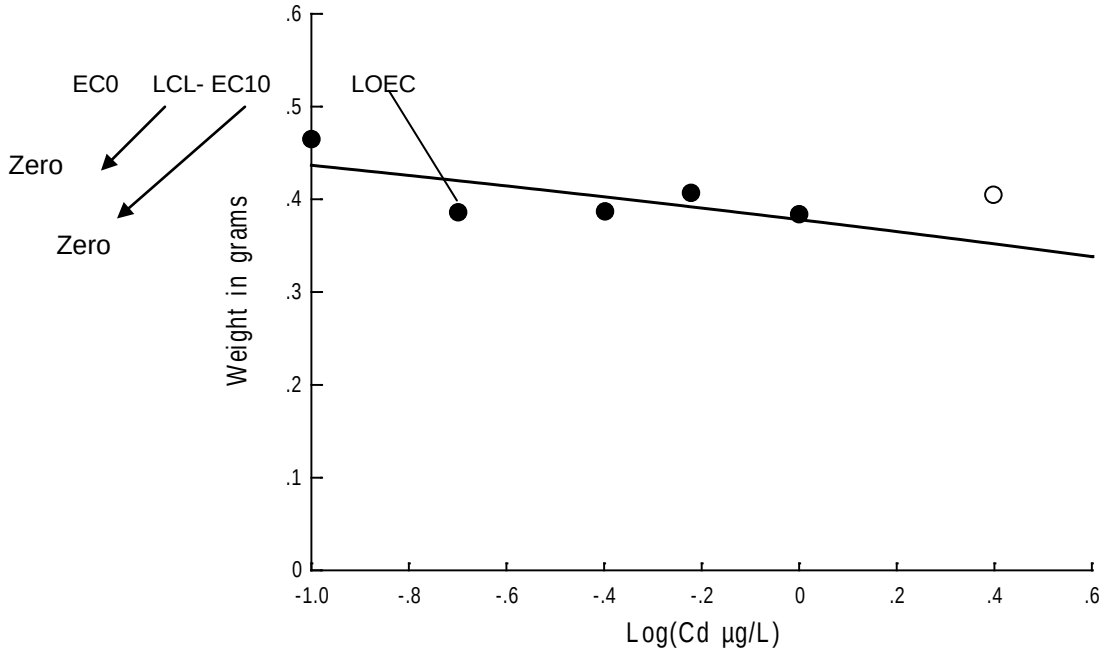
Example 8. Growth of Chinook salmon after 120-days Cu exposure, piecewise linear response (Chapman, 1982). Curves do not always give better fits; here it is more plausible that the onset of adverse effects occurs at a higher copper concentration than the controls. However, in data sets such as this, the interpolation between the control and first treatment data set is so large that the shape of the curve and thus the response is less a statistical question than a professional judgment about what seems most plausible.

Chinook growth (sigmoid threshold)

ECp	ECp est	95 LCL	95% UCL
10	2.215	0.026	185.270
1	0.954	0.000	12238.000
0	0.646	0.000	652500.000

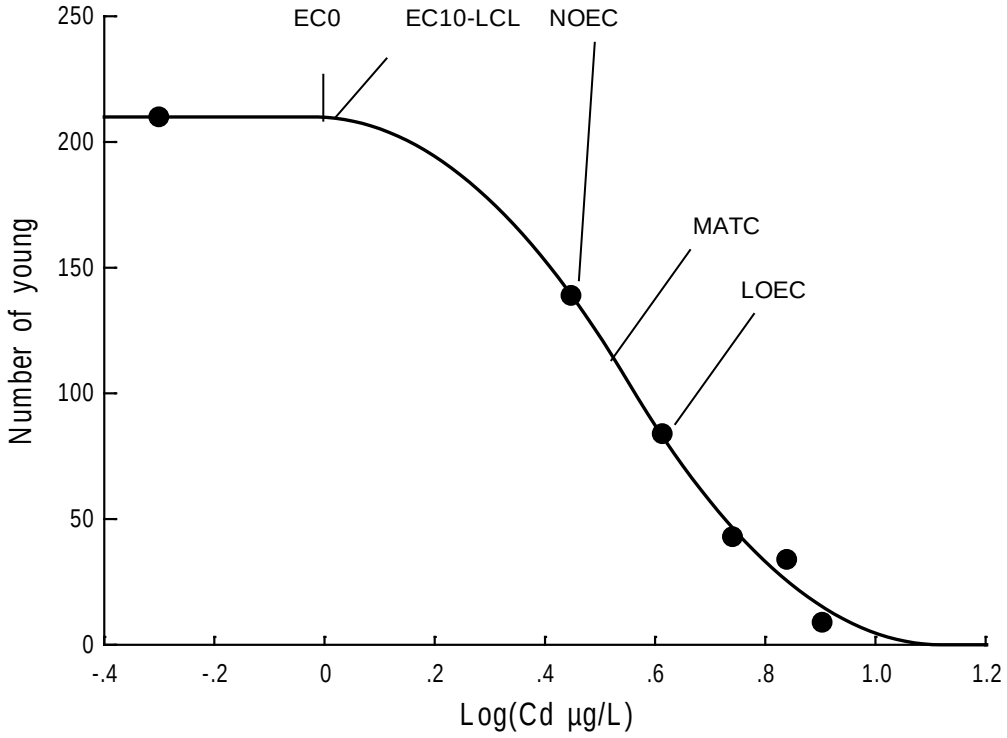
Chinook growth (piecewise linear)

10	3.386	0.699	16.399
1	2.623	0.396	17.354
0	2.550	0.372	17.468



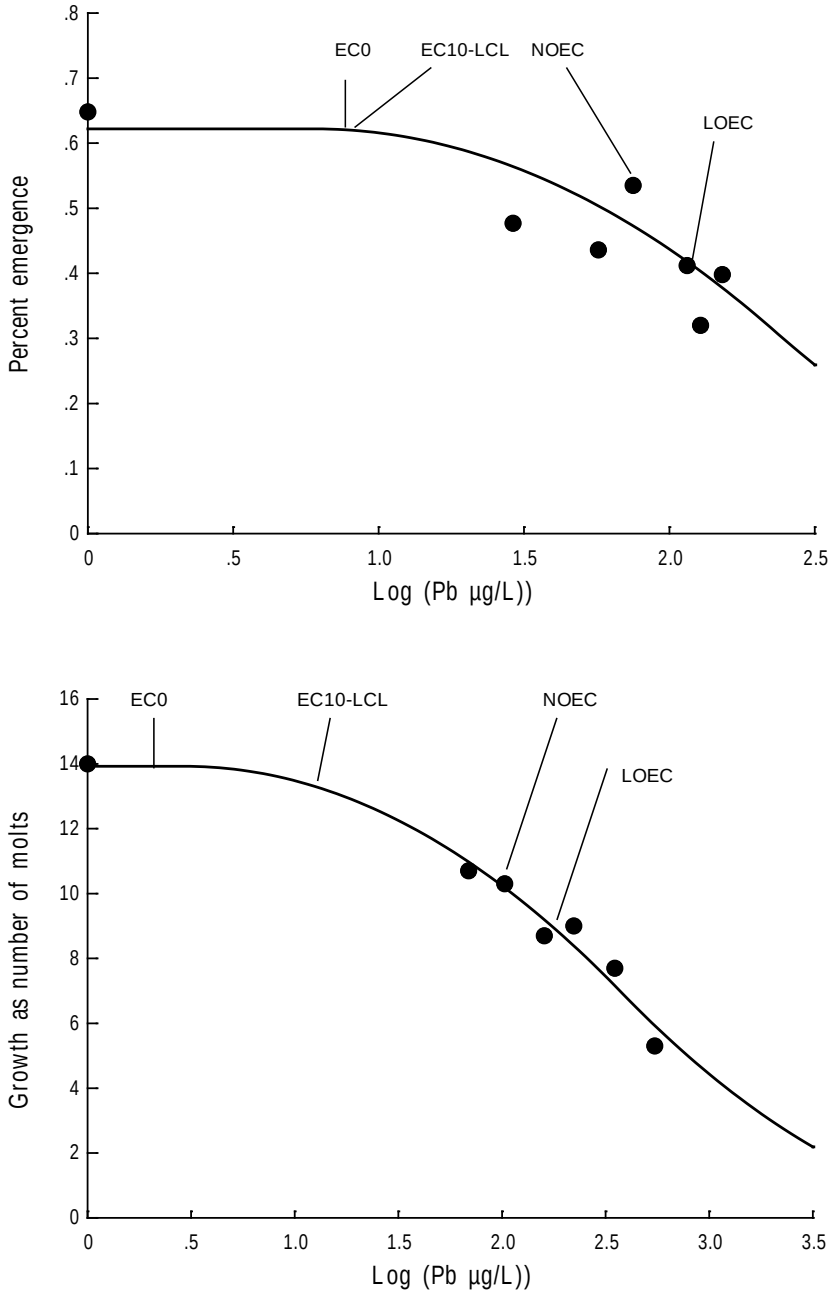
Example 9. Rainbow trout growth after 62-d exposure to Cd (Mebane *et al.* 2008). This example is similar to the Chinook salmon and Cu example in that statistically significant effects were observed in all treatments and no NOEC could be obtained. Further, because no monotonically decreasing concentration response was observed, the curve was almost flat and ECp values are meaningless (numerous errors and warnings were overridden to create this example). In this example, statistics of any type offer little help in interpreting the data.

ECp	ECp est	95 LCL	95% UCL
50	16.61600	0	Infinity
20	0.02234	0	Infinity
10	0.00080	0	Infinity
5	0.00008	0	Infinity
0	0.00000	0	Infinity

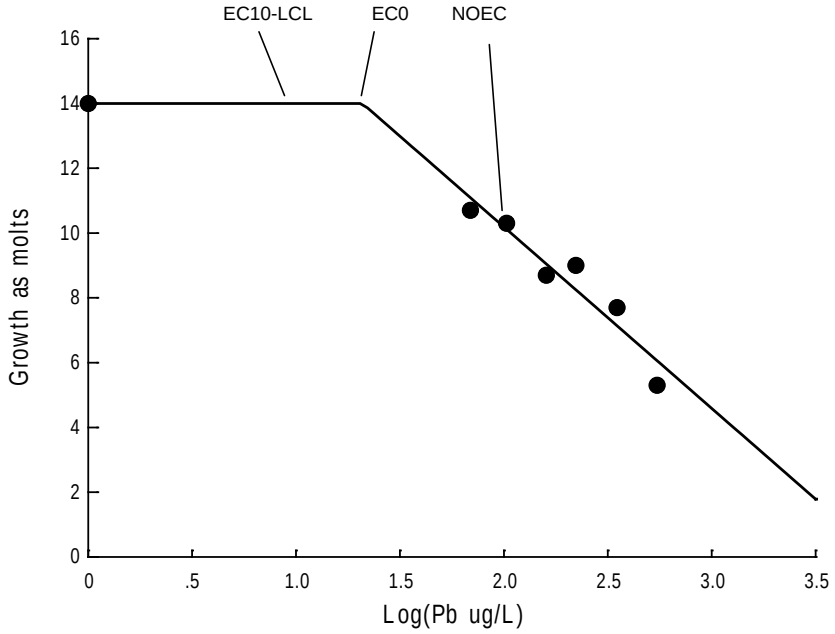


Example 10. Reproduction of *Ceriodaphnia dubia* after 7-d exposure to Cd (Castillo and Longley, 2001). In this test, the NOEC reported by the authors corresponded to about a 35% reduction in reproduction, and greater than a 50% reduction for the MATC .

ECp	ECp est	95 LCL	95% UCL
Sigmoid	2.800		
25	2.414	1.970	2.957
10	1.716	1.239	2.375
1	1.148	0.577	2.282
0	0.953	0.482	1.887



Example 11. Emergence of midge (*Chironomus tentans*) larvae following 21-days exposure to Pb (Top); Mayfly (*Baetis tricaudatus*) molting during 10-days exposure to Pb (Mebane *et al.* 2008). Examples of less than ideal datasets that can arise from testing of non-standard organisms or tests conducted in environmentally realistic but noisy experiments (these were streamside tests). The shape of the curves in both datasets suggest an onset of effects below the lowest concentration tested. This suggests both that NOECs may not be conservative and that low ECp values are uncertain.



Example 12. (Continued) Same mayfly (*Baetis tricaudatus*) as above, but using a piecewise linear or jackknife distribution. As with the case of copper and Chinook salmon growth, assuming a curved distribution would cause the EC0 estimates to be near the control. If that were to be considered implausible, the jackknife "curve" provides a higher "no-effect" value that statistically is equally valid.

C. tentans, Pb Emergence, logistic			
ECp	ECp est	95 LCL	95% UCL
10	30.697	5.793	162.670
5	19.039	1.962	184.720
0	6.009	0.067	540.460

Mayfly, Baetis tricaudatus - logistic				**Mayfly, Baetis tricaudatus – "Jackknife**		
ECp	ECp est	95 LCL	95% UCL	ECp est	95% UCL	95% UCL
20	63.159	25.394	157.090	65.972	28.888	150.66
10	25.713	6.721	98.375	37.103	13.112	104.99
5	13.620	2.514	73.806	27.825	8.7638	88.342
0	2.937	0.206	41.873	20.867	5.8379	74.585

Conclusions

In most of these comparisons, the rank order of the "effects" concentrations were EC0< LCL-EC10 <NOEC. Of the statistics examined, the LCL-EC10s seems particularly suspect. Generally, LCL-EC10 estimates were close to EC0 or EC1 values, however, in all cases where reasonable LCL-EC10 estimates could be obtained, so could EC1 or EC0 values. Confidence intervals on very low effect estimates are large, but at least for EC1 or EC0 values, confidence intervals can be calculated. No confidence limits can be calculated on a confidence limits, and there is no logical reason why the LCL-EC10 is a better estimate of an EC1 or EC0 than would be the EC1 or EC0 themselves. In sum, no empirical or theoretical reason for using LCL-EC10 statistic could be envisioned.

In most instances, the differences between the NOECs, LCL- EC10s, and EC0s were small. This suggests that given the magnitude of uncertainty involved in other aspects of evaluating risks to listed species such as extrapolating effects between species, and extrapolating acute-to-chronic effects, the choice of which statistic used to estimate "no-effect" for a given test response may be of less importance. Some datasets were less than ideal for the statistical models. For most datasets, estimates of these extreme statistics seemed reasonable, based on the datasets from which they were derived. Confidence limits were very large, but the estimates themselves seemed reasonable. Some ECp analyses were uncertain, most commonly because of inadequate partial effects resulting in uncertainty in the shape of the response curve. In other tests, adverse effects resulted in all treatments, so no NOEC could be determined. Differences in results obtained using different assumed statistical distributions (normal, triangular, rectangular) were small.

The results of NMFS' analysis suggest that for initial screening of large databases for chronic effects concentrations to compare with criteria values, any of the NOEC, LCL-EC10, EC1, or EC0 statistics could be useful, and the choice of which statistic to use will probably depend on which is most available. However, in instances where the test is influential in the assessment, a more careful review of the original research might enable the assessor to make a more informed judgment of whether the test indicates reassurance of the lack of effects or indicates that adverse effects are likely. These judgments cannot always follow rote statistical analyses.

References for Appendix B

Besser, J.M., Mebane, C.A., Mount, D.R., Ivey, C.D., Kunz, J.L., Greer, E.I., May, T.W., and Ingersoll, C.G., 2007, Relative sensitivity of mottled sculpins (*Cottus bairdi*) and rainbow trout (*Oncorhynchus mykiss*) to toxicity of metals associated with mining activities: Environmental Toxicology and Chemistry, v. 26, no. 8, p. 1657–1665 http://dx.doi.org/10.1897/06-571R.1

Brosi, B.J., and Bilber, E.G., 2009, Statistical inference, type II error, and decision making under the US Endangered Species Act: Frontiers in Ecology and the Environment, v. 7, no. 9, p. 487-494. http://dx.doi.org/doi: 10.1890/080003

Castillo, V., III, and Longley, G., 2001, Comparison of EPA target toxicity aquatic test organisms to the fountain darter: 7 day chronic toxicity test using cadmium chloride, performed 11/12/99 - 3/6/00 (5 parts): San Marcos, Tex., Edwards Aquifer Research and Data Center (EARDC), Southwest Texas State University, Fed.Assist.Agree.No.X-986345-01 (5 parts). 179 p.

Chapman, G.A., 1982, [Chinook salmon early life stage tests with cadmium, copper, and zinc]: Corvallis, Oregon U.S. Environmental Protection Agency, Environmental Research Laboratory.

Crane, M., and Newman, M.C., 2000, What level of effect is a no observed effect?: Environmental Toxicology and Chemistry, v. 19, no. 2, p. 516-519

EPA, 1991, Technical support document for water quality-based toxics control.: Washington, D.C, Office of Water, U.S. Environmental Protection Agency, EPA 505/2-90-001. 143 p, http://www.epa.gov/npdes/pubs/owm0264.pdf.

EPA, 2000, Benchmark Dose Technical Guidance Document: Washington, D.C., Risk Assessment Forum, U.S. Environmental Protection Agency. 96 p, URL.

EPA, 2003, Final draft methodology for conducting biological evaluations of aquatic life criteria: methods manual: Washington, DC, U.S. Environmental Protection Agency, Office of Science and Technology, EPA-823-B-03-002. 85 p.

Erickson, R.J. 2008. Toxicity Response Analysis Program, version 1.2. U.S. Environmental Protection Agency, National Health and Environmental Research Laboratory, Mid-Continent Ecological Division, Duluth, Minnesota. http://www.epa.gov/med/prods_pubs.htm [Accessed December 2010].

Gelber, R.D., Lavin, P.T., Mehta, C.R., and Schoenfeld, D.A., 1995, Statistical analysis, *in* Rand, G.M., ed., Fundamentals of aquatic toxicology: effects, environmental fate, and risk assessment, second edition: Washington, D.C., Taylor and Francis, p. 111-123.

Hansen, J.A., Welsh, P.G., Lipton, J., and Suedkamp, M.J., 2002, The effects of long-term cadmium exposure on the growth and survival of bull trout (*Salvelinus confluentus*): Aquatic Toxicology, v. 58, no. 3-4, p. 165-174

Laskowski, R., 1995, Some good reasons to ban the use of NOEC, LOEC and related concepts in ecotoxicology: Oikos, v. 73, no. 1, p. 140-144

Marr, J.C.A., Lipton, J., Cacela, D., Hansen, J.A., Bergman, H.L., Meyer, J.S., and Hogstrand, C., 1996, Relationship between copper exposure duration, tissue copper concentration, and rainbow trout growth: Aquatic Toxicology, v. 36, no. 1, p. 17-30

McGarvey, D.J., 2007, Merging precaution with sound science under the Endangered Species Act: BioScience, v. 57, no. 1, p. 65–70

Mebane, C.A., 2006, Cadmium risks to freshwater life: derivation and validation of low-effect criteria values using laboratory and field studies (2010 rev.), U.S. Geological Survey, Sci Inv Rep 2006-5245 (v.1.2). 130 p, http://pubs.water.usgs.gov/sir20065245/.

Mebane, C.A., and Arthaud, D.L., 2010, Extrapolating growth reductions in fish to changes in population extinction risks: copper and Chinook salmon: Human and Ecological Risk Assessment, v. 16, no. 5, p. 1026-1065. http://dx.doi.org/10.1080/10807039.2010.512243

Mebane, C.A., Hennessy, D.P., and Dillon, F.S., 2008, Developing acute-to-chronic toxicity ratios for lead, cadmium, and zinc using rainbow trout, a mayfly, and a midge: Water, Air, and Soil Pollution, v. 188, no. 1-4, p. 41-66. http://dx.doi.org/10.1007/s11270-007-9524-8

Moore, D.R.J., and Caux, P.-Y., 1997, Estimating low toxic effects: Environmental Toxicology and Chemistry, v. 16, no. 4, p. 794–801

Newman, M.C., 2008, “What exactly are you inferring?" A closer look at hypothesis testing: Environmental Toxicology and Chemistry, v. 27, no. 5, p. 1013–1019. http://dx.doi.org/DOI: 10.1897/07-373.1

Stephan, C.E., and Rogers, J.W., 1985, Advantages of using regression analysis to calculate results of chronic toxicity tests, *in* Bahner, R.C., and Hansen, D.J., eds., ASTM STP 891: Philadelphia, PA, American Society for Testing and Materials (ASTM), p. 328-338.

USFWS, and NMFS, 1998, Endangered Species Act consultation handbook: procedures for conducting section 7 consultations and conferences: U.S. Government Printing Office, Washington, D.C., U.S. Fish and Wildlife Service and National Marine Fisheries Service, ISBN 0-16-049596-2. 190 p,

http://www.nmfs.noaa.gov/pr/laws/esa/policies.htm#consultation.

Appendix C

An evaluation of the accuracy and protectiveness of EPA's 2007 biotic ligand model (BLM)-based copper criteria for copper

Contents *1*
Summary *1*
Introduction *3*
Development of the BLM *5*
Analyses of the accuracy of the copper-BLM and criteria for predicting toxic or non-toxic conditions *7*
Acute toxicity predictions for fish *8*
Acute and chronic toxicity predictions with invertebrates *24*
Chronic toxicity predictions with fish *32*
Chemosensory or behavioral effects *37*
Field and experimental ecosystem studies *43*
Conclusions and Recommendations *49*
Short-term implementation considerations *49*
References *60*

Summary

In 2007, EPA revised their national freshwater ambient water quality criteria for copper. The 2007 criteria replaced the longstanding statistical hardness-toxicity site-specific modifiers of copper toxicity with the much more advanced and complex biotic ligand model (BLM). The BLM uses a more mechanistic approach, which combines a geochemical model of copper speciation and binding to dissolved organic carbon (DOC) in the water, and a model of competition between copper and major ions in water for binding sites on the gills of fish or other biological surfaces. The BLM predictions include the concentration of total dissolved copper in water that is predicted to accumulate on the gills of fish, or for small invertebrates that have less defined gill structures, other surface tissues, to non-specific critical accumulation levels that kill the organisms. The version of the model used in the 2007 criteria was supported by a large body of research (Di Toro *et al*. 2001; Santore *et al.* 2001; EPA 2003b; Niyogi and Wood 2004; EPA 2007a). Because the hardness-based copper criteria which date from the 1980s have not been demonstrated to be consistently protective of listed salmonids and their ecosystems, the application of the more recent and scientifically advanced BLM-based criteria is an obvious potential alternative.

However, there are fundamental questions about the BLM's performance and BLM-criteria's protectiveness. These include:

1. The BLM concept is intended to be capable of predicting copper toxicity to any aquatic animal but the performance of the BLM has been principally validated with toxicity data from fathead minnows and daphnids. Does the BLM reliably predict the toxicity or non-toxicity to other aquatic organisms including salmonids?

2. Earlier versions of the BLM were criticized for under predicting toxicity in low hardness waters and over predicting the mitigating effect of dissolved organic carbon (DOC). Is this still the case with the 2007 version? If so, are these concerns important enough to recommend against the use of the 2007 criteria?
3. The BLM that the 2007 criteria are based upon is an acute toxicity model for predicting short-term, lethal (acute) toxicity of copper. However, for the 2007 criteria it was extrapolated to predict for, and protect against chronic effects as well. No analyses of the efficacy of this extrapolation were included or referenced in the 2007 criteria document. Does this BLM also predict long-term, chronic effects?
4. Because the 2007 BLM-based criteria were developed solely from acute lethality data, no consideration of sublethal effects related to chemosensation and behavior such as impaired olfaction, predator avoidance, and prey capture were considered. These types of behaviors are considered fundamental for salmonids and other fish to complete their life cycles in the wild. Does the BLM reasonably predict and prevent against impairment of these types?
5. Laboratory experiments with single-species have an inherent artificiality to them. Field tests or tests in experimental ecosystems can be very different from those in laboratory experiments. Do the BLM-based criteria appear protective in more natural field settings or with experimental ecosystems?

This review addresses these questions through analyses of existing data sets using the BLM. The results of our analysis are mostly favorable toward the performance of the BLMs and also mostly favorable toward the protectiveness of the 2007 criteria values resulting from the BLM outputs. Our review suggested opportunities for refining the BLM. For example, the model appears to be overly sensitive to dissolved organic carbon (DOC) and under sensitive to calcium. That is, in the data sets reviewed, increasing DOC in water predicted a greater protection than appeared to be the case, and increasing calcium predicted a lower protection than appeared to be case. Regardless, in most cases, the 2007 criteria appeared protective from the adverse effects described in the studies. That is, for the water quality characteristics of a particular test, the copper criteria values produced from the BLM were mostly lower than the corresponding, measured adverse effect values. Although the data were thin, some of the field studies indicated risks that adverse shifts in invertebrate communities could occur at copper concentrations lower than those estimated for the 2007 criteria for the situations. However, in the field studies from which adverse effects were inferred, the 2007 copper criteria values were as low as or lower than corresponding hardness-based criteria equations. Appropriately designed and well executed field studies of the effects copper in the context of BLM predictions would be particularly valuable. Still, for the present, the 2007 BLM-based copper criteria appear sufficiently protective for listed salmonids and their ecosystems.

Reviews of seasonal time series data from a variety of streams that were considered representative of conditions within the action area indicate that "critical conditions," i.e., conditions when the BLM predicts that organisms would be most vulnerable to a given concentration of copper, are highly predictable. If the site-specific water chemistry information needed to directly calculate the BLM-based criteria were unavailable, table values are suggested for conservative but realistic critical conditions for waters across the range of anadromous salmon in Idaho that could be used to ensure protective conditions for listed salmon and steelhead.

Introduction

The purpose of this review is to evaluate whether EPA's (2007a) aquatic life criteria for copper would be a protective alternative to apply in lieu of EPA's (1985) hardness-dependant aquatic life criteria for copper, which are the criteria adopted by Idaho and under review in this consultation. Whereas the 1985 copper criteria, along with most other metals criteria developed by EPA prior to 2007, were based upon statistical regressions between water hardness and toxicity, the 2007 copper criteria are based on a fundamentally different approach. The 2007 copper criteria are derived from the biotic ligand model (BLM) which predicts copper toxicity based on copper's expected bioavailability to aquatic organisms, as estimated using a geochemical model (HydroQual 2007). The BLM concept can be generalized to a variety of metals, and a variety of effects measurements such as short-term acute exposures that kill organisms outright, long-term chronic exposures that may not, predicting death or sublethal effects such as sensory impairment or reduced growth. However, for brevity, henceforth "BLM" refers to the version of the BLM for predicting acute toxicity of copper that was incorporated into EPA's 2007 copper criterion.

The EPA's Biological Assessment of for Idaho's toxics criteria proposed the use of the BLM as a "*strategy for reduction in uncertainty of water quality criteria for the protection of threatened and endangered species*." Further, EPA Region 10 committed to "*review the schedule and plan for updating the aquatic life criterion for copper*" and that "*the Services and EPA Region 10 will determine if the plan for updating the criteria will provide protection for salmonids.*" (EPA 2000, p. 24). Consistent with their commitments in the BA, EPA developed and published an updated, BLM-based copper criterion (EPA 2007a).

In 2005, the Idaho Department of Environmental Quality (IDEQ) updated all of its aquatic life criteria for metals, except for copper. The metals criteria in use in Idaho as of 2005 had been developed in the 1980s and were initially promulgated for use in Idaho through EPA's (1992) National Toxics Rule (NTR). EPA completed a series of updates to its metals criteria in 1995; these updates were subsequently published in a 2002 compendium of recommended water quality criteria, which in turn provided the technical basis for most of IDEQ's updates in 2005 (EPA 1996, 2002). IDEQ in 2005 adopted updates to all of their metals criteria except for copper. The explanation given in public meetings held by IDEQ was that although EPA's 2002 copper criteria were more protective than the NTR versions, EPA had also published the 2003 draft BLM update to the copper criteria, and that IDEQ would rather wait until the pending BLM-based criteria was published in final form, rather than revising their copper criteria twice. In February 2007, EPA published their final revised aquatic life criteria for copper (EPA 2007a). Nevertheless, there has been no indication that IDEQ is considering updating their 1992 NTR version of the copper aquatic life criteria.

The EPA's training materials for implementing the copper BLM suggest an incremental implementation as the most feasible and efficient means of implementing the updated criteria. EPA (2010) suggested that this incremental approach "s*hould result in more appropriate criteria more quickly for waters where the hardness-based copper criteria may be potentially overprotective, such as waters with high DOC, or potentially under-protective, such as waters with low pH.*" (emphasis added). Despite the apparent even-handed treatment of risks of either over- or under-protection of the hardness-based criteria in the previous sentence, all the examples given in EPA (2010) of site-specific application were for effluent influenced waters that were

expected to provide considerably higher criteria. Further, the quoted sentence could be misleading, since waters with near neutral pH of 7.5, the hardness-based criteria may be underprotective by more than 6X in sites with moderately-hard waters and low DOC (Table 1).

This review focuses on whether the updated criteria as published will likely provide adequate protection for juvenile salmonids, their invertebrate prey, and other aquatic life. The EPA's (2007a) aquatic life criteria for copper represented a fundamental and ambitious change from earlier statistical regression-models in part because the BLM seeks to actually simulate some of the mechanisms of toxicity. Previous criteria just reflected overall statistical regression models of toxicity using water hardness, which is one of many factors that influence toxicity. In effect, the BLM is expected to be applicable and flexible enough across a variety of water quality conditions that it would produce a site-specific criterion for any specific location. While the 2007 BLM-based copper criteria are the most advanced and complex water quality criteria developed by EPA to date (Di Toro *et al*. 2001; Niyogi and Wood 2004), there are fundamental untested assumptions and unanswered questions relating to their protectiveness for aquatic ecosystems, especially those ecosystems inhabited by threatened or endangered species. These include:

1. Low hardness or soft water streams are common in Idaho. An earlier version of the acute copper BLM severely under predicted toxicity in very soft waters (Sciera *et al*. 2004; Van Genderen *et al*. 2005). Does the 2007 version predict toxicity accurately in soft waters? Regardless, would criteria values be lower than observed toxicity concentrations?

2. Some studies have suggested prediction bias in waters related to dissolved organic carbon (DOC). In tests with *Daphnia magna* and rainbow trout, the default BLM predictions tended to over predict the mitigating effect of elevated DOC on copper toxicity, and better model predictions were obtained by reducing the "metals reactive" portion of DOC by half in the model inputs (e.g., De Schamphelaere *et al*. 2004; Welsh *et al*. 2008). Is this borne out by other studies? If so, is this bias of a magnitude to undermine the protectiveness of the 2007 criteria?

3. The published validation of the reliability of the acute copper BLM for accurately predicting effects from short-term copper exposures was based only on three species, the fathead minnow, *Pimephales promelas*, and the cladocerans *Daphnia magna* and *Daphnia pulex* (Santore *et al*. 2001). Does the model produce reasonably accurate toxicity predictions for salmonids? What about other species that might be representative of prey or other co-occurring taxa?

4. The BLM was developed using short-term data, but has also been used to extrapolate against long-term effects (or the lack thereof) using acute to chronic ratios (ACRs). This approach has been criticized for its implicit assumption that acute and chronic effects are the results of similar internal mechanisms and as violating the mechanistic foundations of the BLM (Niyogi and Wood 2004). For example, the protection of factors such as DOC or calcium from copper accumulation and toxicity might be leaky. For instance, organic carbon as humic acid delayed the loss of sodium in longer-exposures of rainbow trout to copper, but did not ultimately prevent sodium losses (McGeer *et al*. 2002), also adding calcium to soft water protected against the

acute respiratory and osmoregulatory effects of exposure to a combined, relatively high Cd and Cu concentration on trout, but did not protect against the longer term ionoregulatory effects of the Cd and Cu mixture and the longer term accumulation of Cd and Cu by the fish (Richards and Playle 1999). Does the 2007 acute copper BLM also predict chronic toxicity? Does the ACR extrapolation result in criteria that are protective for fish or other aquatic organisms?

5. The sense of smell in fish is tied to critical behaviors including predator evasion, finding mates, and navigation but particularly so with migratory salmonids. Copper interferes with olfactory function in fish, and the olfactory bulb in a fish snout has very different structure and function than do the gills, for which the BLM was developed (Hansen *et al*. 1999a; Hansen *et al*. 1999b; McIntyre *et al*. 2008b). Do the 2007 BLM based criteria sufficiently protect against olfactory impairment?

6. As with almost all EPA's criteria, the 2007 BLM-based copper criteria predicts the absence or presence of adverse effects in the field from extrapolations of mostly short-term laboratory toxicity tests with "standard" laboratory test species that may not represent any real ecosystem. The 2007 criteria represent a fundamental change from previous criteria, and under some conditions can produce criteria values that can allow considerably higher copper concentrations than previous versions, and some field validation of the criteria protectiveness seems prudent. Are there field studies that indicate whether the BLM-based criteria are likely protective?

Development of the BLM

The BLM relies on the concept that metals in water are not toxic to fish and other aquatic organism per se, but rather, only when metals accumulate to critical concentrations in tissues does toxicity result. Thus it is not necessary to specify some metal species such as free Cu^{2+} in water as being bioavailable and other metal species such as CuOH as being less bioavailable. Rather, the presence of the gill causes the chemical equilibria in water to change. Thus it is the degree to which the gill complexation sites are occupied by metal that determines whether toxicity will occur. A fundamental concept and assumption of the BLM is that the fraction of receptor binding sites occupied by a toxic metal would be the same for a given biological response, independent of water chemistry.

With fish, the gill is considered to be the target organism tissue used to predicts toxicity as function of three "C's": 1) complexation of copper with dissolved organic carbon (DOC) and carbonate in the water, 2) concentration of copper forms that can be toxic, which are assumed to be ionic free copper and copper hydroxides; and 3) competition between copper and other ions in water such as calcium, hydrogen, magnesium, and sodium for essential calcium and sodium channels on the surface of fish gills, or in the case of invertebrates that may not have distinct gill surfaces, on the surface of the "biotic ligand."

The development of the BLM can be traced back to the demonstration that the concentrations of cadmium accumulated on the gills of fish were a reliable predictor of cadmium caused deaths (Mount and Stephan 1967), that concentrations of metals accumulated on gills of fish can be predicted as a function of inorganic water chemistry (Pagenkopf 1983), and that organic carbon in water is an important modifier of metal accumulation (Playle *et al.* 1993b). These concepts were further refined and validated to develop what has become known as biotic

ligand models for copper that could be manipulated to make predictions for any taxa (Di Toro *et al.* 2001; Santore *et al.* 2001). The EPA then extended the BLM concept from predicting toxicity for single species to predicting non-toxicity to 95% of the taxa represented in a species sensitivity distribution of available data (EPA 2003b, 2007a). A more detailed history of the BLM is given by Paquin *et al.* (2002) however Mount and Stephan's (1967) original insights do not appear to have previously been credited. More recently, an important practical aspect that has greatly popularized the BLM in recent years was the development of functional personal computer software that has made the ability to make BLM predictions for several taxa accessible to non-specialists.[9]

The data requirements to calculate copper criteria using the BLM are greater than that for the hardness based criteria (i.e., calcium and magnesium or direct titration). The BLM requires data on temperature, pH, DOC, Ca, Mg, Na, K, sulfate, chloride, and alkalinity and the underlined values appear more important, especially DOC and pH.

While the intended level of protection for 95% of the species-sensitivity distribution is unchanged when EPA updates a criterion, as a practical matter, the higher or lower criteria concentrations allowed for the same characteristics of a water body make different criteria more or less protective for species and more or less stringent for dischargers. Comparing the 1992 copper criterion under consultation with the updated 2002 or 2007 criteria values show that there is little difference between the 1992 and 2002 versions. In contrast, the 2007 BLM-based chronic criteria values are strikingly different from the Idaho values under consultation (Table 1). For the moderately hard "BLM-standard" water conditions used in the 2007 criteria derivation that were used to make data more comparable, the BLM based criteria are over 6X lower than the Idaho/NTR criteria . However, the BLM-based criteria are strongly influenced by the concentration of dissolved organic carbon (DOC) in the water, and when the DOC is increased to 8 mg/L but other water characteristics are kept the same, the BLM-based criteria are twice as high as the Idaho/NTR criteria. Therefore, a key question for reviewing the accuracy and protectiveness of the BLM-toxicity predictions is whether DOC is likely to control toxicity to the extent predicted by the BLM.

Table 1. Comparison of chronic copper criteria (CCC) from the 1992 hardness-based NTR, hardness-based 2002 update, and 2007 BLM-based updated criteria.

Water-chemistry condition	NTR CCC µg/L	2002 CCC µg/L	2007 CCC µg/L
Hardness 85 mg/L, pH 7.5, DOC 0.5 mg/L (ASTM/EPA moderately-hard water)	9.9	7.8	1.5
Same except DOC of 1 mg/L	9.9	7.8	2.8
Same except DOC of 2 mg/L	9.9	7.8	5.5
Same except DOC of 4 mg/L	9.9	7.8	11.
Same except DOC of 8 mg/L	9.9	7.8	22.
Same except DOC of 12 mg/L	9.9	7.8	33

The DOC range of 0.5 to 12 mg/L includes the vast majority of DOC measurements in Pacific Northwestern streams although higher values likely occur briefly during runoff or in waters with extensive riparian or littoral marshes).

[9] http://www.hydroqual.com/wr_blm.html

Analyses of the accuracy of the copper-BLM and criteria for predicting toxic or non-toxic conditions

To address the five questions from the introduction, copper effects data from many relevant studies were tracked down and reviewed to see they had sufficient data to analyses in the BLM-context. If so, the water characteristics corresponding with the empirical effects data where run through the HydroQual, Inc. The BLM software using the 2007 model parameter values from Table 2. To evaluate the ability of the model to predict the observed effects across different species, types of effects, and diverse waters, a critical or lethal accumulation value (CA or LA) was estimated at the biotic ligand associated with a given effect as the sum of predicted biotic ligand concentrations of Cu^{+2} and $CuOH^{+}$ for each test value. For example, a copper concentration causing 50% mortality in a test, that is the LC50, would be used with the model to predict a LA50 for each test. When multiple CAs for the same endpoint and species were available, such as with rainbow trout for LC50 concentrations or concentrations causing a 10% growth reduction (EC10) from multiple tests for example, a geometric mean CA was calculated. This mean CA50 was in turn used to predict how well the model could predict LC50s or EC10s for that species across diverse water conditions.

The parameters used in the 2007 criteria were noted to be different differed from those that were described in previous technical evaluations supporting the technical basis of the BLM-based copper criteria. The differences are that binding affinity factors between the biotic ligand magnesium (Mg) and copper hydroxide ($CuOH^{+}$) are included in the computer parameter files but not in the model documentation (Santore *et al.* 2001; EPA 2003b). The bioavailability and toxicity of $CuOH^{+}$ were described in EPA (2003) and its omission from the summary parameter Table 2 was probably simply an oversight. In contrast, Mg was specifically excluded in earlier versions of the gill binding model, including the public review draft of the criteria update, because it did not mitigate toxicity as much as Ca (Santore *et al.* 2001; EPA 2003a). While not in the criteria documentation, discussions with the model and criteria developers indicated that the about face on including Mg in the criteria version of the model was not documented because it occurred shortly before publication. This was because despite earlier evidence of the lack of protectiveness of Mg for fish in "normal" waters (Erickson *et al*. 1996; Welsh *et al*. 2000; Santore *et al.* 2001; Naddy *et al.* 2002), at least in some extremely hardwater effluents in the arid west, Mg did have some protective effects against copper toxicity (Van Genderen *et al*. 2007). Because of the late and informal addition of Mg to the model, the protectiveness of the BLM-criteria in natural waters with differing Mg content is specifically considered here.

In addition to the inorganic species listed in Table 2, a fundamental part of the BLM is its procedure for estimating the amount of copper in the water column (i.e. before the copper ever gets to the gill) that is bound to dissolved organic carbon (DOC). This is implemented in the BLM through an implementation of the Windermere Humic Aqueous Model (WHAM V) originally developed by Tipping and Hurley (1992). As will be shown, the BLM is extremely sensitive to DOC, making the accurate measurement of DOC in the waters of interest highly important to the performance of the BLM.

Table 2. Parameters of the BLM versions used in the criteria technical support document that was prepared for peer review (EPA 2003b) and revised parameters used in the 2007 criteria (EPA 2007a)

log K conditional equilibrium stability constants of binding affinity of the biotic ligand (BL) with inorganic species	**BLM Technical support document** (EPA 2003b)	**2007 BLM versions 2.2.1 (EPA 2007a)and 2.2.3**[10]
log K_{BL-Cu}^{2+}	7.4	7.4
log $K_{BL-CuOH}^{+}$	Not included	6.22
log K_{BL-Ca}^{2+}	3.6	3.6
log K_{BL-Mg}^{2+}	not used	3.6
log K_{BL-Na}^{+}	3.0	3.0
log K_{BL-H}^{+}	5.4	5.4
Fathead minnow critical gill lethal accumulation value (LA50, nmol/gill ww) predicted to cause 50% mortality, on the average	6.2	2.97
Daphnia magna " " "	Not included	0.0483
Ceriodaphnia dubia " " "	Not included	0.052
Rainbow trout " "	Not included	0.4424
Final acute value (FAV) " " "	Not included	0.03395

Acute toxicity predictions for fish

The BLM is expected to be at its strongest for predicting the acute toxicity of copper to fish because the BLM was initially developed with acute toxicity data for fish, and the initial published calibration of and validation of model was with fish (Santore *et al*. 2001). In particular, Santore *et al.* (2001) used an extensive data set by Erickson *et al.* (1996) in which fathead minnows were tested with copper while manipulating natural water from Lake Superior with varying factors that could potentially control toxicity, such as DOC (humic acid), pH, Ca, Mg, Na, temperature, and suspended solids. While a very good fit was obtained for most data, subsequent analyses with fathead minnows suggested that the BLM for fathead minnows was biased high and underpredicted toxicity (that is, over predicted LC50s). Underpredictions were more pronounced in very soft water (Van Genderen *et al*. 2005). However, these analyses were based upon an earlier version of the BLM fathead minnow model than that derived by EPA (2007). The NMFS compiled and analyzed these and many other tests with fathead minnows using the 2007 model to predict toxicity. While the fathead minnow is not of direct interest in the present consultation, the fathead minnow is emphasized because it is a model organism that is extensively used by aquatic toxicologists worldwide to evaluate the relative potency of compounds and factors affecting toxicity (Ankley and Villeneuve 2006). Patterns developed with fathead minnows are thus presumed relevant to other fish species, even though in an absolute sense, fathead minnows are probably less sensitive to copper than are the salmonids that are the focus of the present analysis and consultation (EPA 2007a).

Before one can validate or refute BLM predictions by comparing them to empirical results from toxicity tests, one must evaluate the inherent precision and repeatability of empirical toxicity tests. Santore *et al.* (2001) found that after excluding outlying data, their model was able to predict the toxicity of copper to fatheads across a wide range of water chemistries by about a factor of 2. Santore *et al*. (2001) considered agreement within a factor of 2 for predicted and

[10] http://www.hydroqual.com/wr_blm.html last accessed 12 August 2010

measured LC50s to be "quite good" noting that replicate toxicity tests by Erickson *et al*. (1996) with copper and fatheads in un-manipulated Lake Superior water sometimes varied by up to a factor of 6. Because the inherent limits on the accuracy of the BLM model are a fundamental benchmark for evaluating the model and criteria, these comparisons of replicate variability were reproduced from Erickson's original dataset (Figure 1).

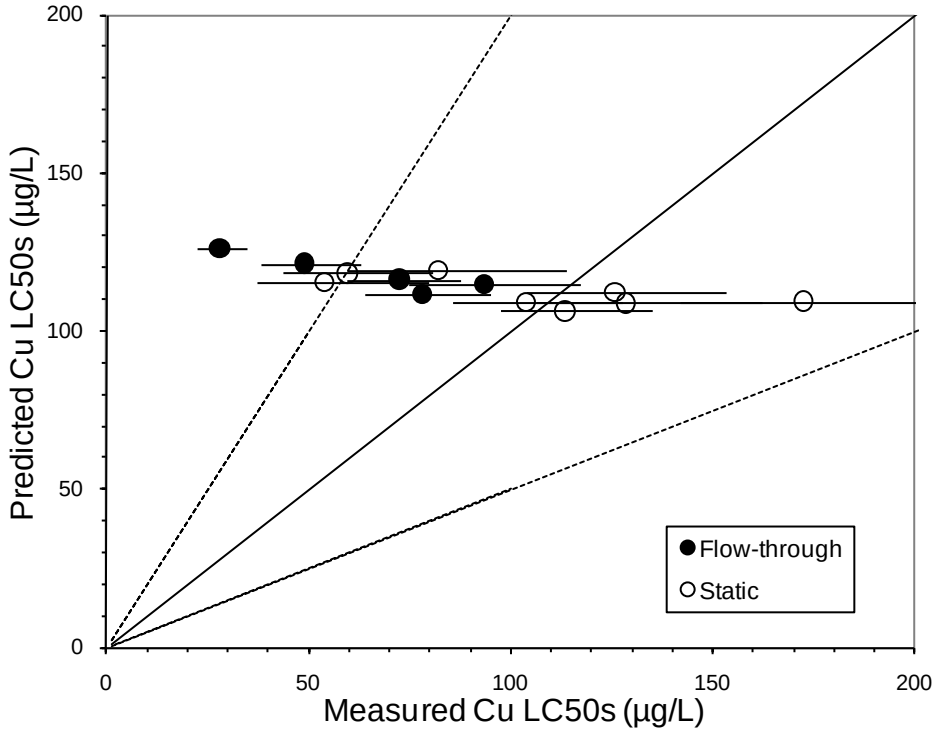


Figure 1. Comparison of measured and BLM predicted copper LC50s for fathead minnows and copper using flow-through or static exposures with unmanipulated Lake Superior dilution water. Unmanipulated Lake Superior water was used as a reference condition as part of a larger study of the effects of water chemistry on the acute toxicity of copper (124 tests total, Erickson *et al*. 1987, 1996). Lake Superior water is commonly used as the dilution water for toxicity testing at the EPA's Duluth laboratory because of its stable characteristics and low background contaminant levels. Closed symbols denote flow-through test results and open symbols denote renewal test results, error bars show 95% confidence intervals on observed LC50s. The solid line indicates the 1:1 line of perfect agreement, dashed lines indicate 1:2 and 2:1 lines, i.e., bounds for predicted values being within 2X of observed values. The same convention is used in following figures.

Lake Superior water may be nearly ideal as a standard reference water for comparing the performance of toxicity tests. The water chemistry near the EPA's Duluth, Minnesota Environmental Research Laboratory appears to be very stable based upon different analyses over time and the water has low background contaminant levels (McKim and Benoit 1971; Maier and Swain 1978; Erickson *et al.* 1996; Cotner *et al.* 2004). Average conditions for the tests in Figure 1 were DOC 1.35 mg/L, alkalinity 42 mg/L, hardness 45 mg/L, pH 7.9, although the differences in the predicted fathead minnow tests (vertical scale) result from variance from these average values. All tests were initiated with <24 hour old fish, so the confounding issue of size or age of

fish should be minimized. The tests are grouped by whether they were conducted as "flow-through" tests where the test solutions are constantly being replaced with an average water residency in the test chambers of about 45 minutes, and as "static" tests where the fish were placed in test solutions at the start of the test and the solution was not refreshed during the 96hr test. Figure 1 clearly shows that copper was more toxic in the flow through tests than in the static tests. Among the flow-through tests, copper LC50s ranged over a factor of about 3.5X, from about 25 to 90 µg/L and among the static tests, copper LC50s ranged over a factor of about 2.5X, from about 50 to 125 µg/L, not including an outlying LC50 at about 170 µg/L (Figure 2).

This analysis illustrates why it is not reasonable to expect the BLM to predict toxicity much more precisely than by about a factor of ± 2, since replicate tests often vary by more than that. This supports the convention started by Santore *et al*. (2001) to use the "within a factor of 2" prediction factor as one guideline for evaluating model performance. In our review, when the predicted/empirical comparisons showed a pattern in their residual errors, we investigated the bias to see if it indicated systematic error, especially underprotection by the model and criteria.

NMFS compiled a large number of toxicity tests with fathead minnows, independent of the Erickson *et al*. (1996) data used to calibrate the model. Test water chemistries were used to predict the toxicity of fathead minnows through the BLM, and we compared these predictions to the empirical LC50s for each test. The 2007 version of the Hydroqual BLM (v. 2.2.3) includes a fathead minnow prediction using a critical accumulation of copper on the gill surface of 5.48 nmol/g gill wet wt (ww) (i.e., the "LA50"). However, the data sources of this fathead minnow prediction file were not described and using the 2007 Hydroqual BLM for fathead minnows using the standard water conditions used by EPA (2007) to normalize data with the BLM produces a fathead minnow LC50 that is 2X higher than the species mean acute value (SMAV) for fathead minnows derived by EPA (2007), 116 µg/L vs. 63 µg/L for the 2007 Hydroqual version 2.2.3 of the BLM and EPA SMAV respectively. Thus to evaluate the performance of the BLM as used in the 2007 criteria, it was necessary to reconstruct the LA50 for fathead minnow and other species in the same manner as done in EPA (2007). A "critical" species mean LA50 for fathead minnow value was estimated as the geometric mean of 141 "LA50" values which in turn had been estimated from LC50 values listed in Table E of EPA (2007). Table E has 150 tests with fathead minnows, but Table 1 says that "Underlined LC50s or EC50s not used to derive SMAV because considered extreme value." No tests in Table 1 were underlined, although in the 2003 draft report, 9 tests with fathead minnows were marked as excluded. Excluding those same tests reproduces the SMAV given in the document, whereas if all tests were used, a critical value of 2.37 would be obtained, which produces a lower SMAV than given in the 2007 document. Thus Table 1 in the March 2, 2007 version is apparently in error, and instead Table 1 of the draft 2003 version is actually the reference for tests used or excluded from the 2007 "final" document. The reconstructed LA50 for fathead minnows produced a SMAV of 69.34 µg/L which is nearly identical to the fathead SMAV of 63.69 µg/L given in Table 1 of EPA (2007). Critical LA50 estimates for *Ceriodaphnia dubia*, *Daphnia magna*, and rainbow trout were similarly reconstructed and also agreed well with their respective 2007 SMAVs. These reproductions of the EPA (2007) values confirmed both the EPA values as well as giving reassurance that the present analyses are indeed comparable.

For species and endpoints that are not based on EPA (2007) values such as critical LA50 values for acute LC50s other species or untested endpoints such as chronic growth reductions or olfactory impairment, we estimated "critical" values in the same manner as used in EPA (2007).

Effects values were determined (i.e., EC50s, EC20s, EC10s), chemistry compiled, and the BLM was run in geochemical speciation mode to predict the copper accumulation on the ligand as Cu^{2+} and $CuOH^{+}$, the sum of which was considered the "critical accumulation" for the test. Where multiple values were available, the geometric mean of test critical accumulation values for a particular endpoint and species was used as the species mean critical value for the endpoint. These critical values were in turn used to predict EC values for the same tests. The scatter or bias of these predictions was used to evaluate the performance of the BLM.

The BLM predictions are compared with empirical or so-called "observed" effects data by plotting scatterplots of the observed and predicted values along with the 1 to 1 line of perfect agreement, bracketed by lines illustrating the factor of 2 test of good agreement between the modeled and observed values. Also, linear regressions are shown, where a slope of 1.0 indicates perfect agreement, and R^2 coefficient of regression values indicate the proportion of variability explained by the regression. Optimal performance would be reflected by a tight scatter of points close to the 1:1 line of perfect agreement; poor model performance would be reflected by a random "shotgun" pattern or distinctly biased patterns that systematically over- or under-predicted toxicity.

The first of these examples is the original calibration data set with fathead minnows that was presented in EPA (2003) and in Santore *et al.* (2001). This modeling used a comprehensive set of toxicity tests with copper and fathead minnows in a variety of artificial and amended natural waters in which the effects of changes in hardness, calcium, magnesium, sodium, alkalinity, humic acid, temperature and other factors were tested (Erickson *et al.* 1987; 1996). (Erickson *et al*. 1987 and 1996 describe the same testing although some data details and tests were not included in the shorter 1996 published version.). As a benchmark, copper toxicity was also tested by Erickson *et al*. (1987; 1996) in un-amended Lake Superior water which varies little in consistency (Figure 1). The model performance for the Erickson dataset was remarkably good, with very little bias in the predictions or scatter, considering that the LC50s ranged from about 10 to 1000 µg/L. Both the highest and lowest LC50s fell very close to the 1:1 line of perfect agreement (Figure 2, top). Because these data and modeling were seminal for the BLM development and the technical support of the subsequent criteria, the methods and sources described in EPA (2003) were repeated here to see if the results were reproducible. For most of the datapoints, the results from Santore *et al.* (2001) and EPA (2003) were successfully reproduced, particularly for the less-toxic samples with measured LC50's $\geq\approx$ 100 µg/L. While the Santore/EPA results were also very good for the more toxic samples, the reconstructed results underpredicted toxicity. The results of the discrepancy are not easy to reconcile, but might be related to uncertainty about whether or not biotic ligand-bound $CuOH^{+}$ was considered toxic in the Santore *et al.* (2001)/EPA (2003) modeling (Table 2).

In Erickson *et al.*'s (1996) data, copper tended to be more toxic in tests that used the flow-through exposure methods rather than static exposures (Figure 2, bottom). In flow through tests, the test solution is intermittently metered into the vessels, replacing the complete water volume several times each day. In static tests, the fish are introduced into the test vessel and maintained in the same volume of water throughout the duration of the tests (e,g., 96-hours). In the Erickson *et al.* (1996) test, the replacement rate resulted in a test residence time of about 45 minutes, or about 32 volume replacements per day. The "renewal" method is a compromise between the flow-through and static methods. Renewal tests are the same as static, except that

the majority of the test solution is siphoned off and replaced midway through the tests (ASTM 1997).

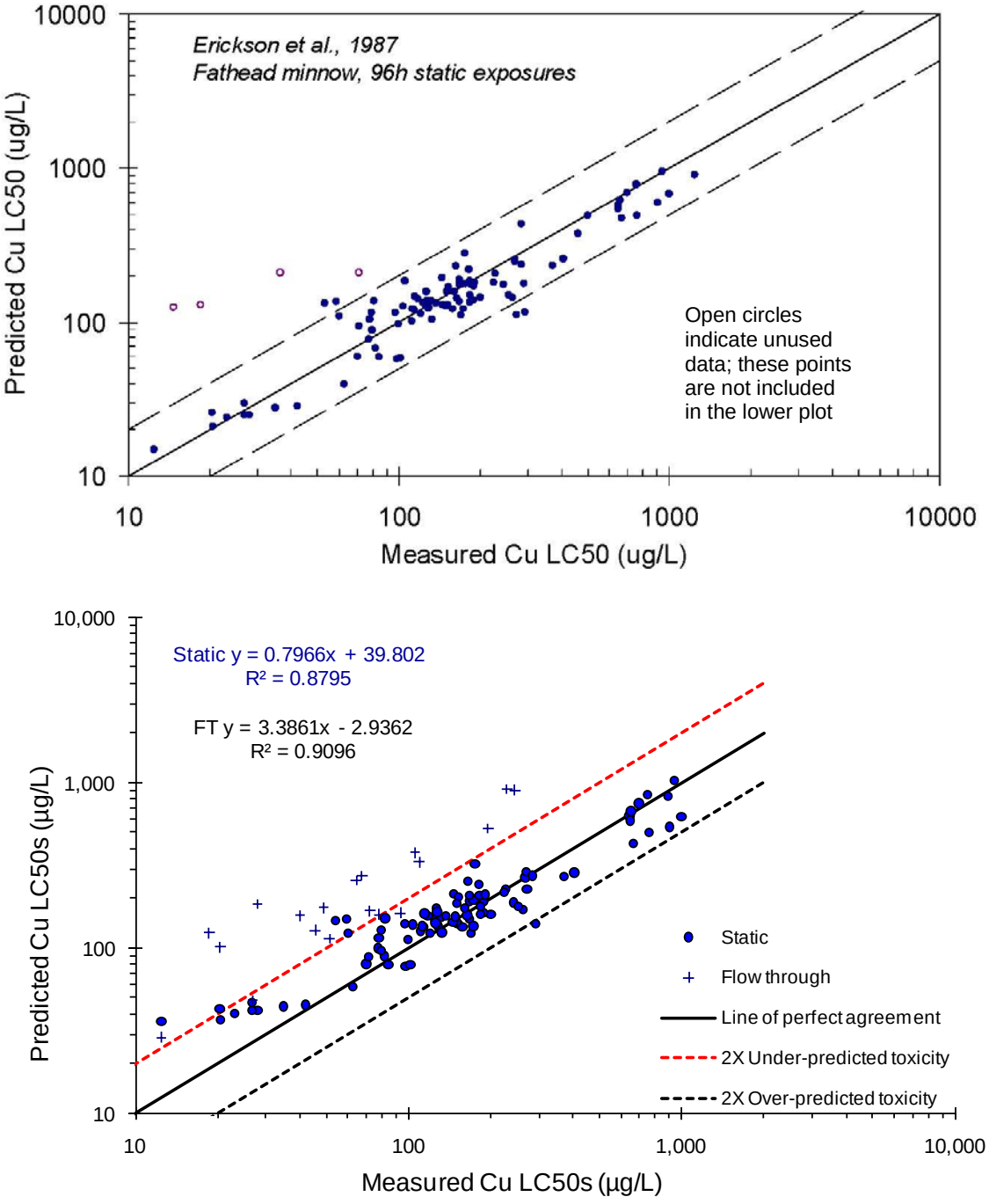


Figure 2. Top - Biotic ligand model predicted versus observed LC50 values for fathead minnows in static toxicity exposures. Figure is from EPA (2003, their Figure 14) in which data from Erickson *et al.* (1987, 1996) were used with the BLM parameters and a fathead minnow LA50 of 6.32 listed in Table 1. The solid line is the 1:1 line of perfect agreement and the dotted lines show the 2:1 and 1:2 lines showing

values 2X more toxic than predicted or 2X less toxic than predicted. Bottom – Reconstruction of EPA's top figure using original data and BLM parameters from EPA (2003) as listed here in Table 2. The modeling very nearly reproduced most values as shown by the nearly identical patterns of points between the two plots. In EPA's 2003 modeling at top, the most toxic measured conditions at the bottom left of the plot with the lowest LC50s were predicted by the model quite accurately. However, in the reconstruction toxicity tended to be under-predicted. This discrepancy is unexplained.

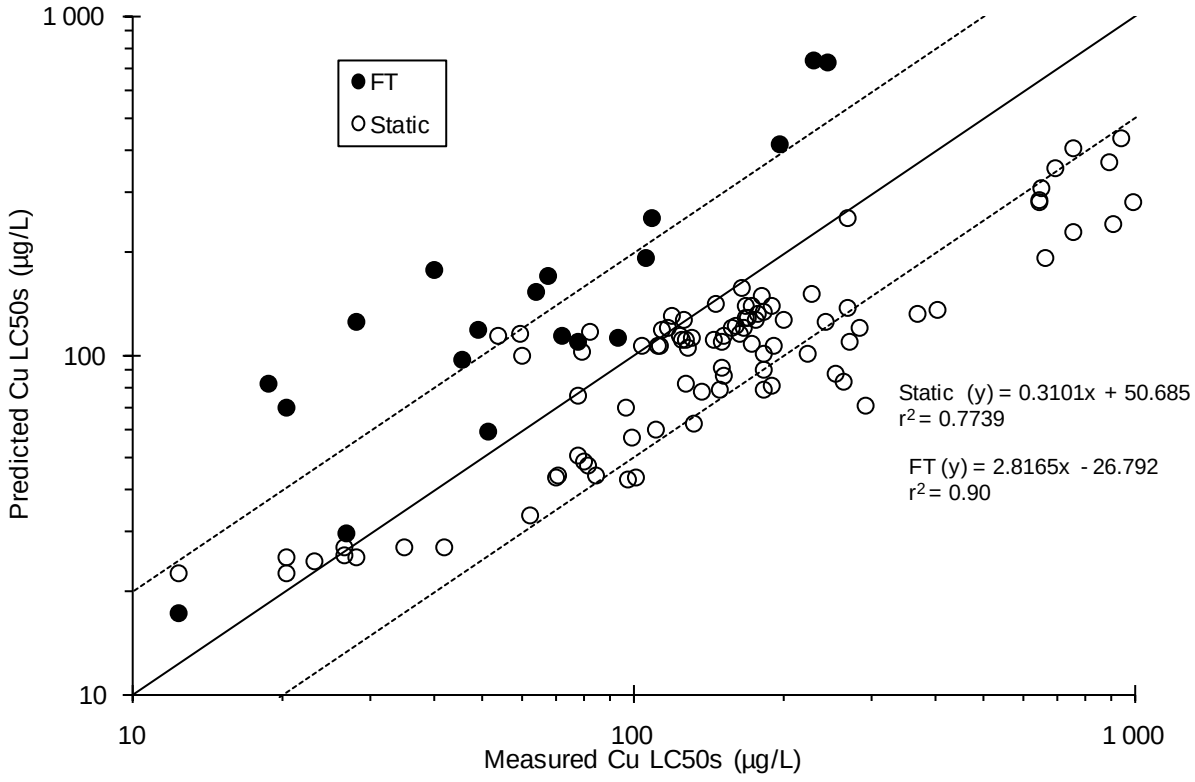


Figure 3. Fathead minnow BLM predicted and measured Cu LC50s, from Erickson's 1996 tests, using EPA's 3-02-2007 LA50 and model parameters from Table 2. "FT" – flow through tests.

Santore *et al*. (2001) interpreted the increased toxicity in the flow-through tests as an indication that the copper had not yet reached equilibrium with DOC and because the metal speciation and complexing equations in the BLM were based on the assumption of equilibrium, the static results were relied upon by EPA in EPA's (2003b) validation (Figure 2, top). In contrast, Welsh *et al.* (2008) also showed that DOC may build up in renewal tests and can explain lessened copper toxicity to rainbow trout. Their flow-through rates were lower than Erickson's (about 4 to 6 hours vs. 45 minutes per volume replacement), suggesting that copper and DOC had more of an opportunity to approach equilibrium than in the Erickson's tests with fathead minnows. The "non-equilibrium" and "increased DOC concentration" explanations for increased copper toxicity in flow-through tests relative to static or renewal tests are not mutually

exclusive. Further, metals in streams may not be at chemical equilibria, which can influence toxicity of metals (Nimick *et al.* 2003; Meyer *et al.* 2007; Nimick *et al.* 2007). Thus, in this review, we examined results from both flow-through and static or renewal tests.

The different patterns of copper toxicity in flow-through or static tests are obvious in the plots of BLM predicted and measured values from Erickson's (1996) data. Initially, the model was calibrated using the static results only, following the belief that the flow-through results greatly exaggerated toxicity (Figure 2). However, EPA (2007a) compiled additional toxicity data on fathead minnow (and other species) toxicity that was analyzed in their (EPA 2003b) technical support document. When we used the 141 test values used by EPA (2007a) to establish the species mean acute value (SMAV) for fathead minnows to estimate a species mean LA50, we obtained considerably more sensitive estimates of copper toxicity to fathead minnows (Table 2). When we predicted the same Erickson *et al.* (1996) data using the BLM parameters used in EPA (2007a), a very different impression resulted. Instead of the static results looking "about right" and the flow-through results looking skewed, both the flow-through and static results roughly straddle the 1 to 1 line of perfect agreement (Figure 3). More interestingly, in the EPA 2003b version, the static data set had a measured to predicted regression slope that was at least 0.9 and a R^2 coefficient of variation that was probably at least 0.9 (described as "at least" because by eye the original 2003 plot had a better fit than the reconstructed plot in Figure 2). However, using the 2007 parameters, the Erickson static data take on a much shallower slope of 0.3 and a lower R^2 value (Figures 2 and 3).

Some previous efforts to validate the BLM-toxicity predictions noted that the model severely underpredicted toxicity in very soft water. Erickson *et al.'s* (1996) data covered a wide hardness range from ~19 to 250 mg/L, although most were conducted at hardnesses of ~40 mg/l and above. Curiously, in Figure 2 bottom, the cluster of solid points at the lower left corner with predictions that drift toward being less protective at lower LC50 values correspond with the lowest hardness levels tested. In tests in very-soft natural waters from the South Carolina plain, the BLM so underpredicted the toxicity of copper to fathead minnows that the LA50 that had been derived primarily from the Erickson data had to be empirically lowered by a factor of 36 to fit the model (Van Genderen *et al.* 2005). Similar results were attained by Sciera *et al*. (2004). These results lead to the question, 'does that the BLM may systematically under predict toxicity in softwater'? This is a significant concern for application in the Pacific Northwest or other areas where softwater is common. Thus NMFS compiled these additional datasets and other softwater toxicity data and compared them to the 2007 version of the BLM. We also analyzed two additional datasets from the Canadian Shield area of central Ontario (Welsh *et al.* 1993; Welsh *et al.* 1996). The Canadian Shield is characterized by thin soils over crystalline bedrock which leads to very low calcium contents in the waters and pH values less than 7 units. DOC may range from as low as 0.5 to over 20 mg/L. While few streams in headwaters regions of the Salmon or Clearwater Rivers in Idaho or most other mountainous regions of the Pacific Northwest have DOC values as high, Canadian Shield waters otherwise appear to have many aquatic chemistry characteristics as waters draining the granitic watersheds in the Idaho Batholith region of central Idaho or the Precambrian metamorphic rocks found further north in much of the Clearwater River watersheds. A fourth important dataset is one in which fathead minnows were tested with copper under uniform hardwater conditions, but with various concentrations of DOC from natural organic material that had been isolated from Nordic reservoirs (Ryan *et al.* 2004).

Comparison of the BLM-predicted and measured EC50s show a systematic bias where the BLM tends to under predict the toxicity of copper in the softwater settings (tests with higher toxicity/lower LC50s) that plot near the bottom left corner (Figure 4.) Across the different datasets, the under prediction bias is diminished as the tests waters become less toxic (higher LC50s) which corresponds with increasing hardness. The tests by Ryan *et al.* (2004) in hardwater with various DOC show no obvious bias. The model performance in softwater was at least an improvement over the magnitude of under prediction in the version used by Van Genderen *et al.* (2005).

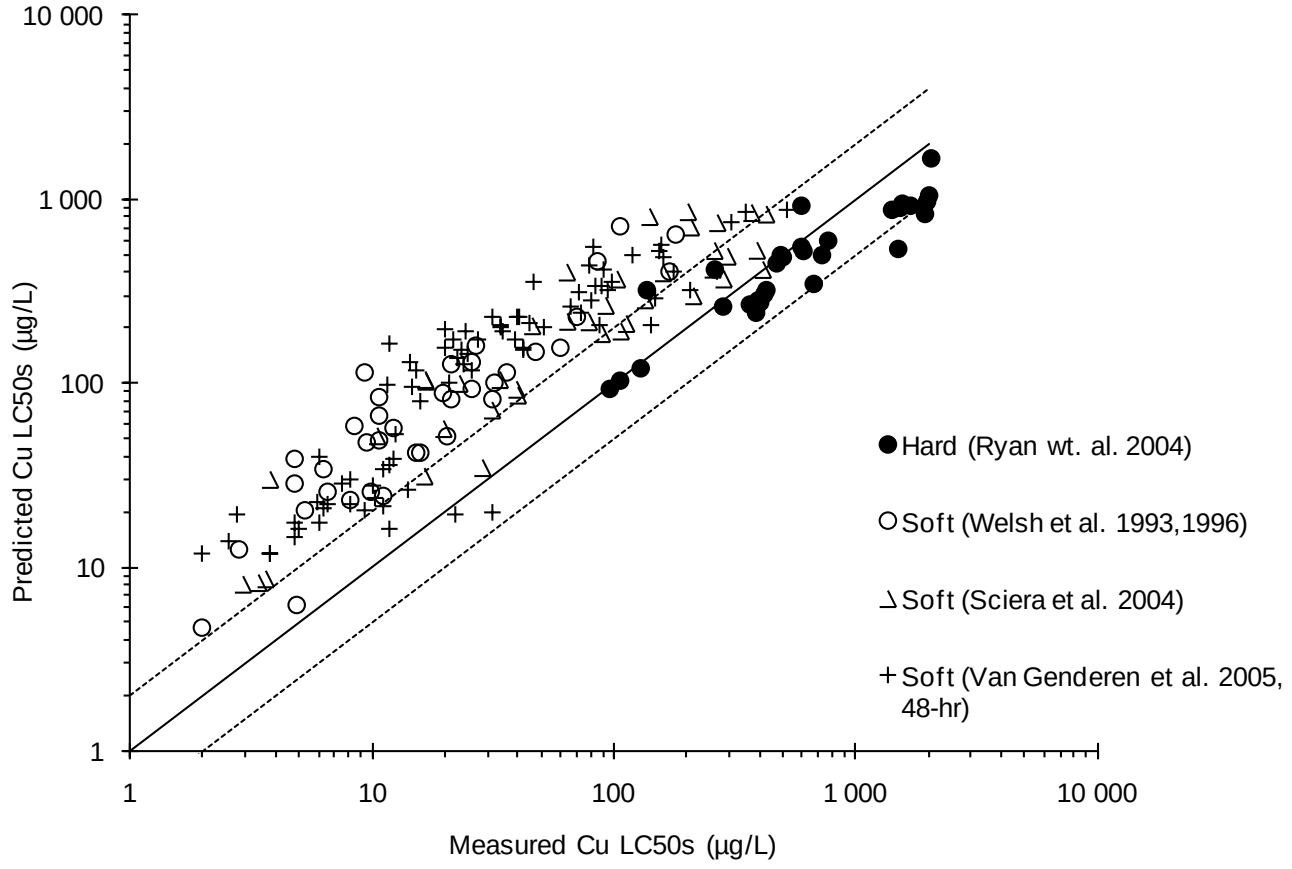


Figure 4. Fathead minnows, BLM predicted and measured Cu LC50s labeled by hard or soft dilution waters, using BLM v 2.2.3 and EPA's 2007 LA50. The comparison shows the BLM generally underpredicted toxicity in the more toxic samples with low measured LC50s.

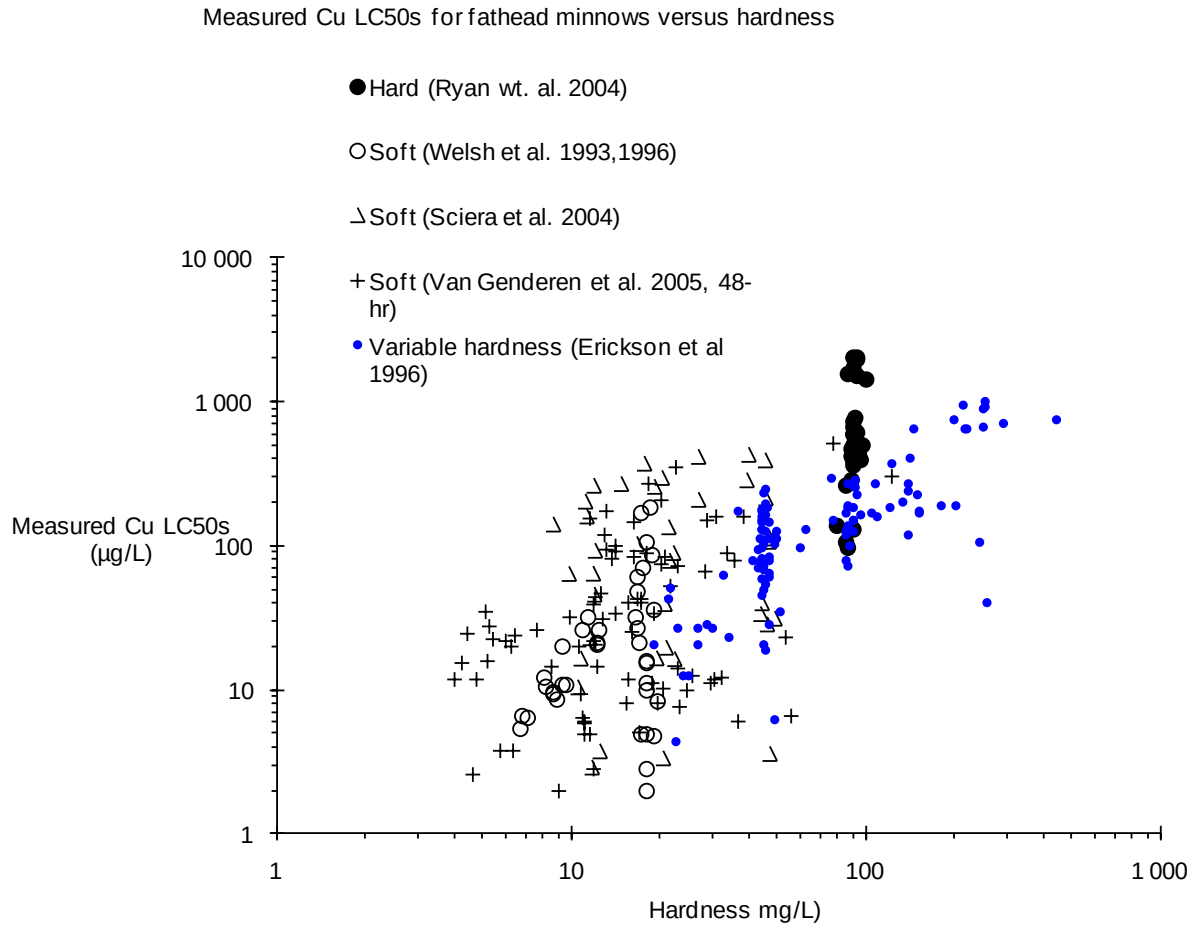


Figure 5. Hardness as a predictor of copper toxicity to fathead minnows: at a hardness of 20 mg/L, fathead minnow LC50s could range from about 2 to 300 µg/L, and at a hardness of about 90 mg/l, LC50s could range from about 100 to over 2000 µg/L.

Because there appeared to be a prediction bias in the BLM that was associated with hardness in these softwater datasets, NMFS compared the LC50s with hardness to see if hardness may be a better predictor of toxicity than the BLM (Figure 5). While there is clearly a pattern of increasing LC50s with increasing hardness (i.e., decreasing toxicity), the variability is so severe as to render a hardness-toxicity relationship dubious for water quality management. For instance, at a hardness of 20 mg/L, fathead minnow LC50s could be anywhere from 2 to over 300 µg/L (factor of 150), and at a uniform hardness of about 90 mg/L, LC50s range over a factor of about 20 (~100 to >2000 µg/L). In contrast, although the BLM-predictions were severely skewed, the predictions seldom varied by more than a factor of five and most of the data varied by much less. This suggests that with additional calibrations, it would be feasible to better tune the model performance in soft waters. In fact, encouraging results with this problem have been recently published (Ryan *et al.* 2009).

The accuracy of BLM predictions and the protectiveness of BLM-based criteria are related but not identical issues. A goal of criteria development is to be able to make useful predictions whether a specific addition of a toxic agent such as copper to a particular aquatic

ecosystem will cause any unacceptable effect on that ecosystem (Stephan 1986). However, from the perspective of protecting listed species, where it is better to err on the side of the species, if exceeding a criterion fails to predict adverse effects, that is not a problem for the species. Rather, what is essential is that the criterion is protective of the listed species and their ecosystems. Thus the suboptimal performance of the BLM in predicting copper toxicity in softwaters, indicates that the BLM-criteria would provide less protection in these waters than intended. Yet it does not necessarily demonstrate that the criteria would be unprotective for the fish tested (fathead minnows). When we calculated the 2007 FAV for each individual test condition, we found that only 4 of 187 or 2% of the FAVs were greater than the empirical LC50s for the same waters. The reason that the FAV was sufficiently protective even though it was biased in softwater is likely because the fathead minnow is sufficiently less sensitive to copper than were the more sensitive Daphnid and mollusc species that defined the FAV.

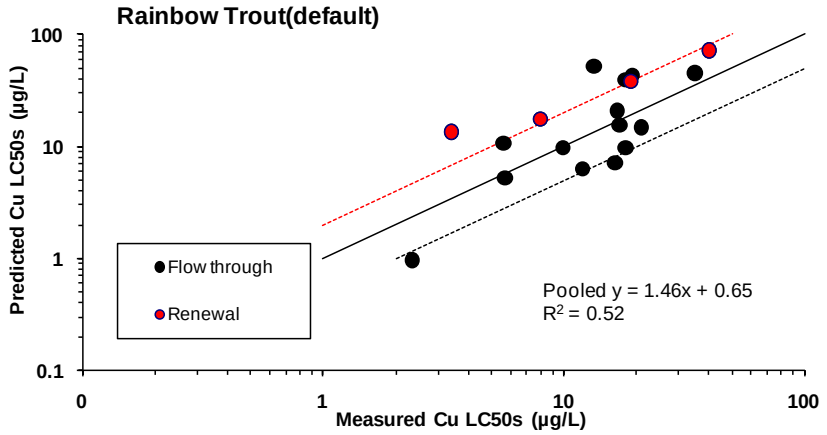
Next, we consider the performance of the BLM with salmonids. While the foundational work to develop the copper BLM used experiments with rainbow trout (Playle *et al*. 1992; Playle *et al.* 1993b, a; MacRae *et al.* 1999), relatively little has been published since then regarding the performance of the copper BLM criteria with rainbow trout our other salmonids (but see Welsh *et al.* 2008). Despite this, we located several very relevant datasets that were well suited for evaluating the protectiveness of the copper BLM for salmonids.

The first dataset with salmonids was from a comprehensive study that tested the comparative sensitivity of rainbow trout and Chinook salmon to copper in natural waters of the upper Sacramento River in northern California. Tests were also conducted in laboratory waters in which calcium, magnesium, and pH were manipulated (Stratus 1996, 1998). All necessary water chemistry parameters were measured and experimental controls were exceptionally tight and well described. With rainbow trout, tests were conducted under both flow-through and renewal designs, but Chinook salmon were only tested with a flow through design. The natural river waters used tended to have soft water, low DOC, and pH in the ranges that are typical of other salmon and steelhead waters in Idaho and the Pacific Northwest. Although the ranges of water chemistry data are fairly narrow, these data are otherwise nearly ideal for the evaluation of the BLM performance under environmentally realistic conditions. The study reports contain a wealth of data and are well supported by data quality control and quality assurance information, and some of the tests were available for incorporation into EPA's (2007) criteria dataset; however, these data have never been further published and have mostly been unavailable to the scientific community.

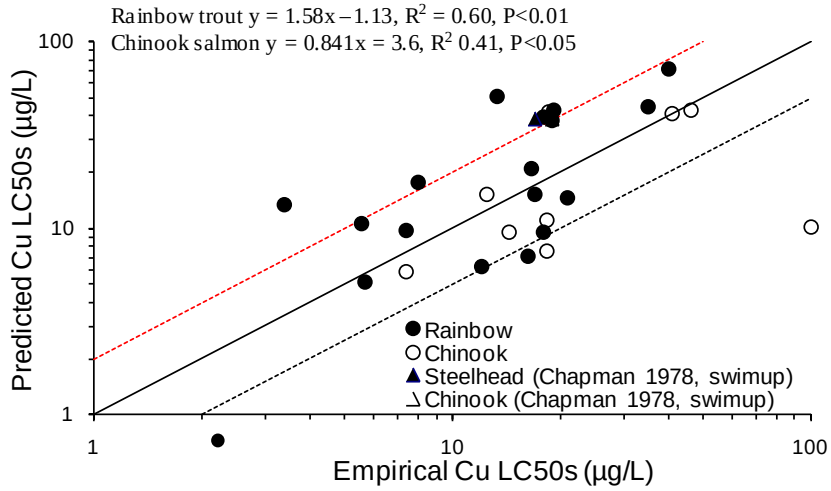
The results of our review of this dataset were reasonably favourable to the BLM's performance, with the regression slopes not greatly different from 1.0. When comparing rainbow trout predictions by whether they used a flow-through or renewal design, the plots do suggest that renewal tests tended to higher LC50s (lower toxicity) for given predicted values than did the flow through tests (Figure 6, top). However, the apparent disparities were not nearly as pronounced as those with fathead minnows discussed earlier (Figures 1 through 3). Thus, it seems that considering renewal or flow-through tests as being more or less appropriate for testing copper toxicity or to use with the BLM is probably not warranted. At least this appears the case for tests with salmonids that were conducted in aquaria with slower water replacement times (longer water residence times) than was used with the fathead minnow mini-diluter study design.

The Chinook salmon predicted and observed toxicity values fell among the rainbow trout values, indicating that at least for the tested stocks, the sensitivity of the two species to copper is

very similar (Figure 6, middle). In fact, the predicted toxicity values were produced using LA50 estimates developed for rainbow trout without any obvious sensitivity bias between the species.

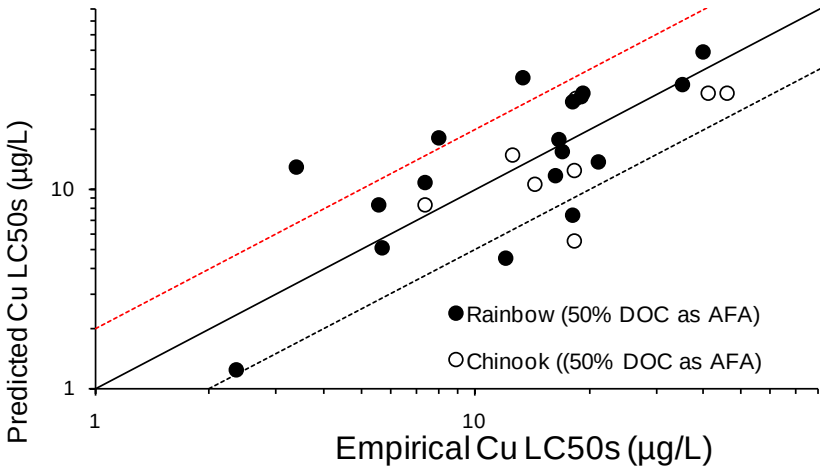


A.



B.

Rainbow trout y = 1.46x +0.65, R^2 = 0.52,
Chinook salmon y = 0.96x + 0.92, R^2 0.61,



C.

Rainbow trout y = 0.94x +4.6, R^2 = 0.52,
Chinook salmon y = 0.56x + 0.92, R^2 0.61,

Figure 6. Rainbow trout and Chinook salmon: empirical and BLM predicted toxicity in 96-hour tests using natural Sacramento River water and lab waters, DOC <0.11 to 2.0 mg/L, pH manipulated from 6-

8, hardness 19-60 mg/L (Stratus 1996, 1998). A. Flow-through vs. renewal tests with rainbow trout; B. rainbow vs. Chinook; and C. rainbow vs. Chinook after reducing DOC availability by 50%.

An issue that has been unresolved in the scientific literature on BLM development is whether some empirical adjustment to the copper reactivity of different DOC sources is beneficial. The argument is that if only 50-65% of DOC in natural waters is reactive with copper (De Schamphelaere *et al.* 2004; Schwartz and Vigneault 2007; Welsh *et al.* 2008), then if 100% of DOC were treated as copper reactive in the BLM it could bias toxicity predictions high when DOC is abundant, and conversely bias predictions low when DOC is scarce. The approximation of metal-binding by a large, complex, and variable group of organic acids making up natural DOC in waters is an extremely difficult problem, and some studies have found that the WHAM model used in the BLM may markedly over-predict organic carbon complexation of copper, resulting in measured free-ion concentrations exceeding predicted values (Boeckman and Bidwell 2006).

The 2007 BLM-based criteria treat 100% of DOC as copper reactive. We evaluated this issue in several of the datasets including the Stratus Sacramento River data with rainbow trout and Chinook salmon by reducing the input DOC by 50% as fulvic acid and generating a new LA50 and predicting toxicity. Welsh *et al*. (2008) give more details on the "50% active fulvic acid (AFA)" adjustment. Curiously, this adjustment slightly improved results with rainbow trout, but slightly worsened predictions with Chinook salmon (Figure 6, bottom). With rainbow trout, the 50% AFA adjustments brought the slope of the empirical vs. predicted line to nearly 1.0, reduced the standard error and reduced the average prediction error slightly from 2.0 to 1.7 (i.e. with a prediction error or "prediction factor" of 2.0, on the average predicted values were within a factor of 2 of the empirical values). However, with Chinook, the prediction factors were little changed with 1.55 to 1.60 for the default and 50% AFA approaches. Thus the 50% AFA "improvement" was not important for the Chinook data.

A second large, comprehensive, and similarly unpublished dataset with rainbow trout and copper is from a "water-effect ratio" (WER) study from the Clark Fork River, Montana (ENSR 1996). The WER approach involves toxicity testing in tandem in dilution waters collected from the site water of interest and in a standard reconstituted laboratory water. The WER is the ratio of the test LC50 in site water divided by the LC50 in laboratory water; the ratio is then multiplied by the aquatic life criteria to obtain a WER-adjusted site-specific criteria. The WER approach is considered here to be a fundamentally limited concept because it is unrealistic to expect any laboratory water to represent the variety of natural and synthetic waters used in testing laboratories. However, in instances such as the Clark Fork testing, WER studies may produce important datasets that are very useful for evaluating BLM performance, because tests are well matched, often conducted across a wide range of DOC and inorganic chemistries, and the better studies measure detailed water characteristics that may influence copper toxicity. In the case of the Clark Fork testing, rainbow trout were tested in laboratory and in natural waters from tributary and river sites during different seasonal "rounds" of testing with measurements of all BLM chemical parameters. This resulted in values ranging from very soft to very hard waters and DOC from less than 1 mg/L to 11 mg/L. Because the "Round 1" and "Round 4" data were collected from the same places in September 1994 and September 1995, but DOC values were much higher in Round 1 and higher than USGS data for similar locations, we considered the DOC data from Round 1 unreliable and excluded it from our evaluation. This still left a very robust censored data set of 73 tests conducted in diverse waters (Figure 7).

Using the censored data, the BLM predictions followed the empirical LC50s reasonably well, with an average prediction error of 1.65 and the worst prediction error of 5.0. When we tried the 50% AFA adjustment as described earlier, the R^2 coefficient of determination value was noticeably improved and the average prediction error was lessened to 1.46 with the worst prediction error lowered to 3.5. The "50% AFA" approach improved the prediction errors in 46 pairs and worsened the errors in 27 pairs. Thus the 50% AFA "improvement" seemed real with this dataset.

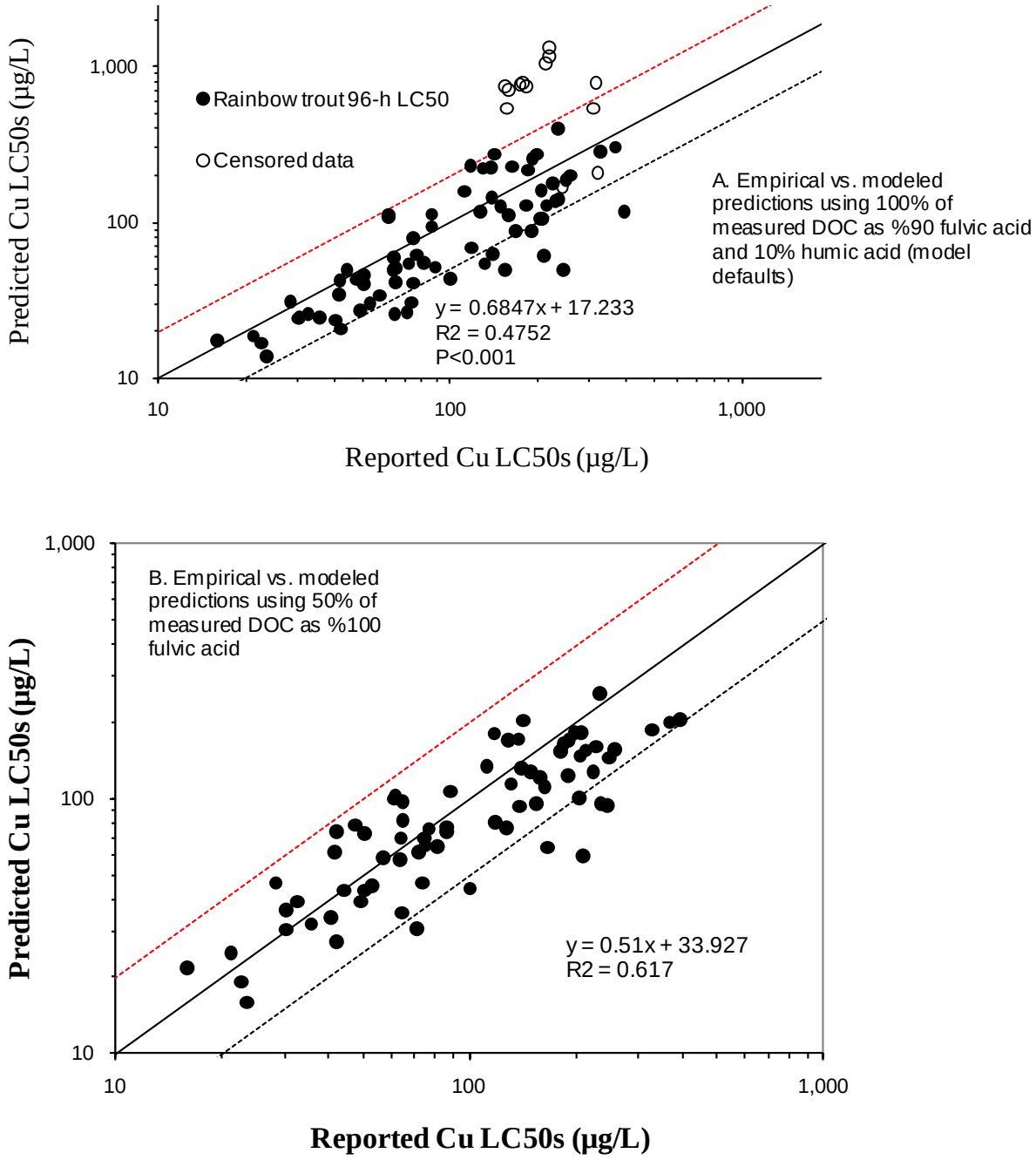


Figure 7. Rainbow trout: predicted vs. empirical toxicity, using the 2007 BLM, in 96-hr renewal tests using lab and site waters, hardness 23-308 mg/L, DOC from <1 to 11 mg/L. Data from ENSR (1996).

In contrast to the studies we described here that evaluated BLM performance in natural waters where DOC and pH were probably the most important factors, the following evaluations consider inorganic factors that affect copper toxicity, such as calcium, magnesium, and alkalinity. These comparisons allow better evaluation of performance of BLM parameters than with natural waters, because in natural waters inorganic chemical factors tend to be correlated with each other. If the model performs well in replicating observed toxicity, then evaluations with natural waters are persuasive. However, if the model performs poorly, if the factors all rise and fall together, there is no way to tease out which factors need adjusting in the model. Thus even though the chemical combinations in such "factors testing" may be contrived in ways that would seldom ever occur in nature, together with testing in natural waters these "factors tests" may provide a thorough examination of model performance.

Welsh *et al.* (2000; 2001) and Naddy *et al.* (2002) tested the sensitivity of rainbow trout to copper in waters in which they tested the relative importance of Ca or Mg in mitigating toxicity by concocting waters which had similar hardnesses, but different Ca and Mg ratios. Both studies found that Mg conferred little protection from copper toxicity to fish, although Naddy *et al.* (2002) found Mg did reduce copper toxicity to Daphnia. The 2003 version of the BLM for copper did not include Mg. However, Mg was included on an equal basis to Ca in the 2007 version (Table 2). The 2007 BLM performed poorly in our review, with the BLM predicting toxicity to decrease with increasing Mg contribution to hardness, when little or no reduction occurred (Figure 8). For example, the Naddy *et al.* (2002) tests were all predicted to have LC50s of about 50 µg/L, when in fact they varied from about 15-70 µg/L. In a pair of tests with different Na content, the BLM accurately predicted the observed pattern.

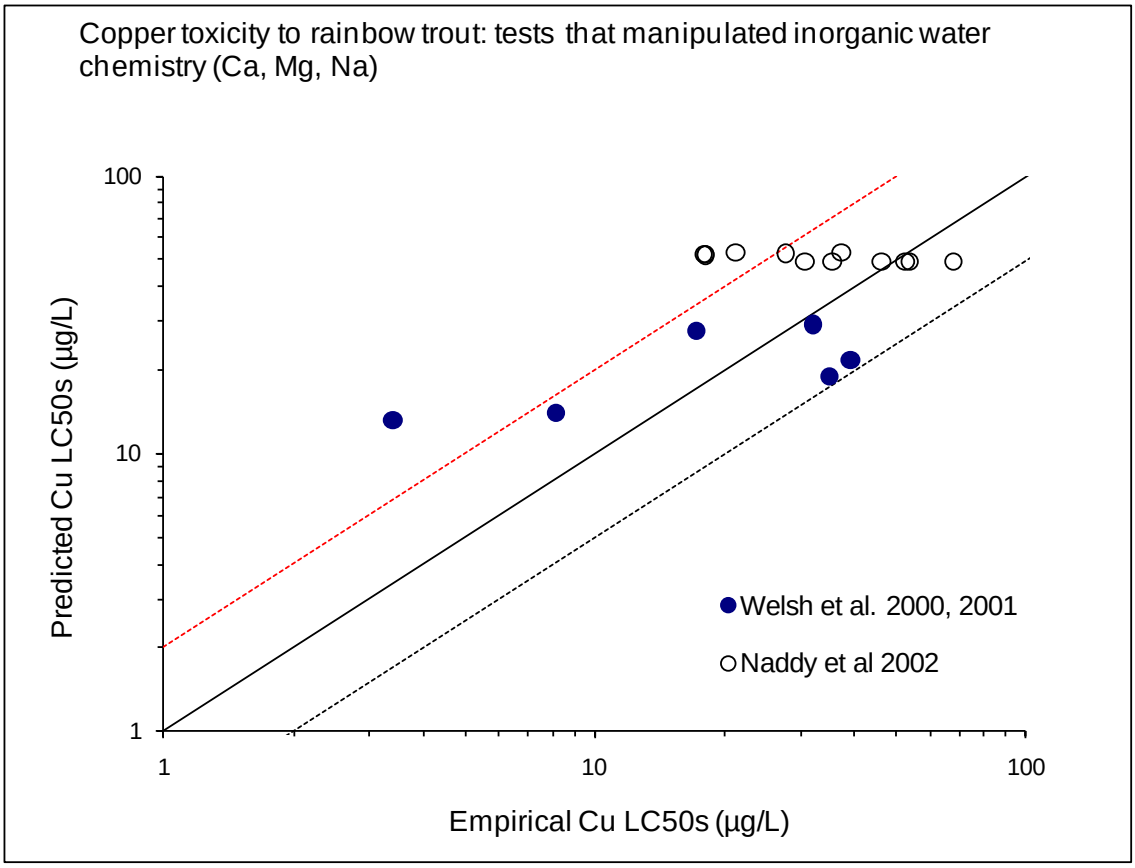


Figure 8. Rainbow trout copper 96h LC50s, with varying Ca and Mg while keeping hardness and alkalinity about the same and with uniform low DOC. Data from Welsh *et al.* (2000; 2001) and Naddy *et al.* (2002), using the 2007 BLM.

Naddy *et al*. (2002) also attempted one test in which magnesium hardness made up all of the total hardness, that is no calcium was added. However, all of the fish died within 48-hours even in the controls with no added copper. This reinforces the critical role of calcium in stream water, that very low calcium waters are stressful independent of metals, and that metals can be exceptionally toxic in low hardness water.

For NMFS' final evaluation of the BLM performance with inorganic factors, we evaluated a series of 9 tests with cutthroat trout and copper that alternately held alkalinity constant and varied hardness or vice versa (Chakoumakos *et al.* 1979). Because the alkalinity manipulations involved different proportions of spring water and amended distilled water, and the spring water contained higher DOC, we grouped the tests by alkalinity, for which DOC was probably about uniform across the tests.

In all, these results suggest that Ca and Mg should not be treated as equally important in the BLM (Table 2), but that Ca should be given a higher binding affinity log K value. While these results suggest that the 2007 BLM modifications tend to lessen the BLM's conceptual improvement over the hardness-equations, because most natural waters tend to have more Ca than Mg (Appendix A), the poor model performance in these datasets probably should not be given more importance than performance with diverse natural waters.

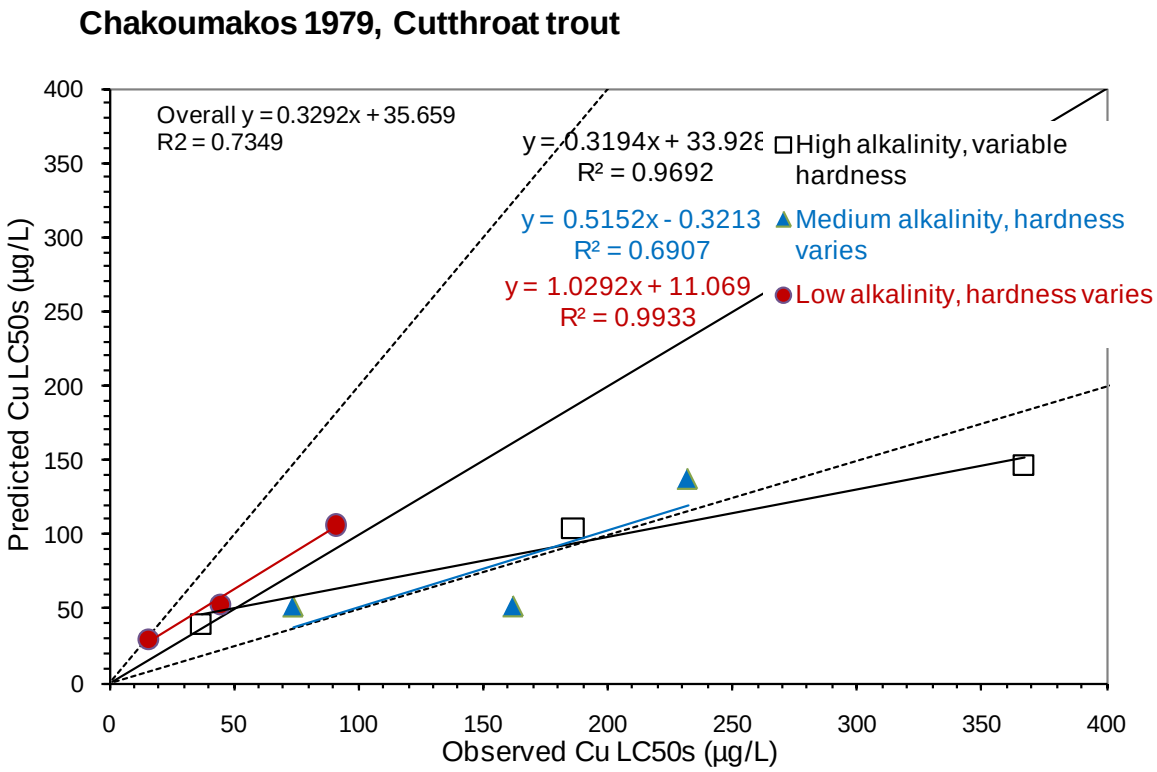


Figure 9. Cutthroat trout modeled and predicted responses to copper under various combinations of low, medium, and high hardness and low, medium, and high alkalinity, in waters with DOC ranging from ~0.9mg/L in low alkalinity water to 3.3 mg/L in their high alkalinity spring water, 2.7 to 9.7g fish (Chakoumakos *et al.* 1979). Because DOC was nearly uniform within alkalinity treatments, data are grouped by alkalinity groups.

The BLM performance seemed mixed in these comparisons (Figure 9). For the low alkalinity series, the BLM and empirical LC50 estimates were nearly perfect with a slope of 1.0 for predicted:empirical best fit line. Yet, for the tests at higher hardness, while the predictions were correlated with the empirical results, the slopes were progressively lower with copper being less toxic than predicted. This pattern is hard to interpret with just these data, but seems to support the idea that the log K value for Ca in the model could be higher.

In summary, this portion of NMFS' review evaluated the ability of the 2007 BLM to accurately predict acute copper toxicity by evaluating hundreds of separate tests with fathead minnows, rainbow trout, Chinook salmon, and cutthroat trout in diverse natural and artificial waters. With one exception, the BLM performed substantially better than did the hardness-toxicity derived criteria (i.e., the NTR and Idaho criteria). In the exception, Naddy *et al.*'s Ca and Mg manipulations shown in Figure 8, the BLM and hardness models were similarly poor; the BLM under predicted toxicity in very soft waters. The DOC influence in the BLM has been suggested to be too strong and a source of bias. This idea was generally supported in our evaluations of BLM performance in natural waters with fathead minnows and rainbow trout, but not of the (much smaller) Chinook salmon dataset. However, the magnitude of this apparent bias was not great. These analyses suggest areas of potential refinement and possible further improvement in the BLM, but do not necessarily indicate that the 2007 BLM is inappropriate to

use as published. However, of the various analyses completed thus far, the evaluations of the overall BLM performance in natural waters is considered more important than "factors testing." In the great majority of tests, the BLM correctly predicted the direction of relations (i.e., more or less toxic) and most predictions of specific LC50 concentrations were reasonably close to empirical estimates.

Acute and chronic toxicity predictions with invertebrates

While our analyses so far evaluated the performance of the BLM with acute copper toxicity in fish, the criteria generated by the BLM apply to all aquatic animals, even invertebrates with gills that are too small or diffuse to directly test. This assumption that the BLM criteria are protective of all aquatic species must largely be true for the BLM to be a valid basis for protecting aquatic communities and, for example, avoiding adverse effects to food chains for ESA listed fish species. Previous versions of the model had good performance predicting toxicity to the zooplankter *Daphnia pulex* (Santore *et al.* 2001). Here, NMFS attempts to validate the performance of the 2007 BLM with other invertebrates. This is easier said than done, for invertebrates are woefully underrepresented in toxicity testing datasets compared to their relative diversity in the wild. For instance, there are at least 10X more aquatic insect species in North America than fish species, but few insects are represented, especially for long-term toxicity datasets (Mount *et al.* 2003; Mebane 2006). A more sensitive and practical approach is to test aquatic insect communities in experimental stream mesocosms, although these sturdies are complicated to interpret. (We discuss this in a later section "Accuracy of copper BLM toxicity predictions in field and experimental ecosystem studies.") We located and evaluated useable acute toxicity datasets with copper for invertebrate taxa: two additional zooplankters, *Daphnia magna* and *Ceriodaphnia dubia*, the freshwater benthic crustacean *Hyalella azteca*, and for two freshwater mussels, fatmucket, *Lampsilis siliquoidea* and rainbow mussel (*Villosa iris*).

We also located sufficient data to evaluate the ability of the BLM to predict chronic toxicity to rainbow mussel and *C. dubia*. This is of particular import because although the 2007 BLM is used to derive chronic criteria, it was developed as an acute model and is not known to have been previously validated for chronic predictions.

The first dataset we evaluated was an acute study with different combinations of pH and DOC tested with acute *Daphnia magna* (Meador 1991). Strengths of this data set include that the test conditions were well controlled and that natural DOC was concentrated from algae exudates, as opposed to some studies that evaluated the role of DOC by adding Aldrich humic acid that is commercially prepared for sale as a gardening soil amendment. The BLM predictions were reasonably favorable in comparison with the empirical results with a slope slightly less than 1.0. The BLM explained a little less than half of the variability in the data, although most values fell within the "factor of 2" rule of thumb for adequate model performance (Figure 10).

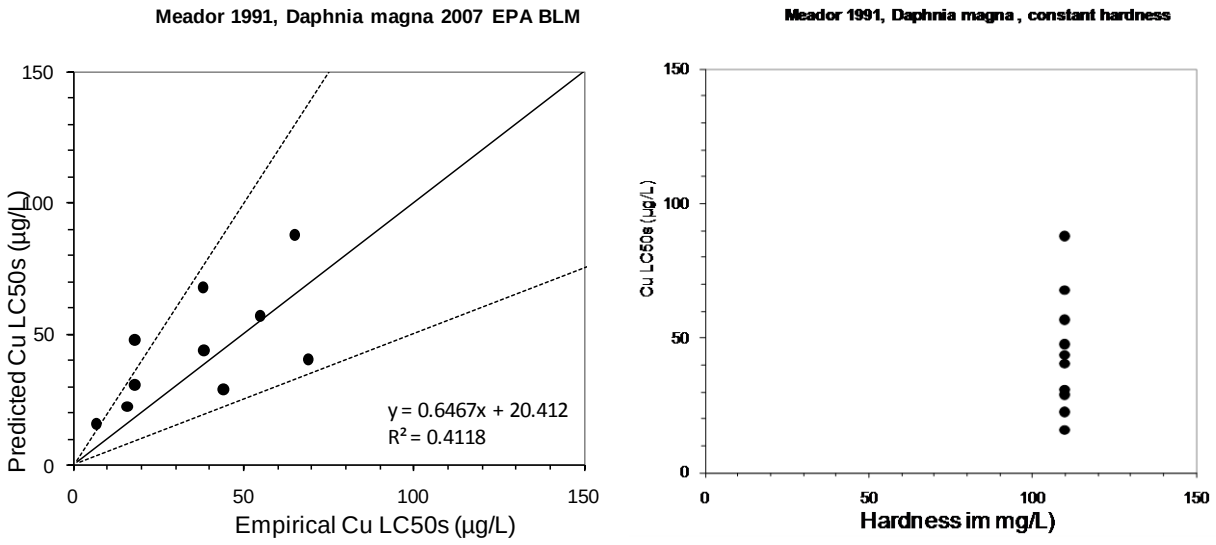


Figure 10. (Left) Daphnia magna, BLM predicted and empirical copper toxicity, pH 6.9 to 7.9, DOC 2.4 – 6 mg/L, and (right) hardness as a predictor of toxicity (Meador 1991).

We located two useful datasets with the amphipod *Hyalella azteca* in which the animals were tested across a gradient of water chemistry conditions within each study (Welsh 1996; Collyard 2002). Thus although one study compared *Hyalella* responses in 96-hour exposures and one in 48-hour exposures, the responses can be compared within the datasets for each study.

We reviewed but excluded two other studies with *Hyalella* and copper for our use comparison. The first was a large study on the effects of major ions (Ca, Mg, Na, and K) and pH on Cu toxicity to *Hyalella azteca* (Borgmann *et al*. 2005). These tests were not as useful for testing the BLM-copper toxicity predictions as the studies shown here because DOC concentrations were variable and uncertain. The data were from static, non-renewal, 1-week exposures in which the animals were fed twice during the tests. The DOC in the artificial media used as a dilution water rose from between <0.1 to 0.2 mg/L before introduction of animals or food to a range of 0.4 to 2.8 mg/L (average 1.72 mg/L) at the end of the test. Modeling the predicted LC50s using either the initial or average end of test DOC values showed that this uncertainty in DOC values alone was carried through to an average additional prediction error of 3X. Using 0.2 mg/L DOC in the model inputs resulted in an average 7-day LC50 prediction of 16 µg/L copper compared to 50 ug/L for the end of test conditions. For tests with low alkalinities, this uncertainty resulted in prediction differences greater than a factor 10. Thus, this dataset was not used to evaluate the BLM performance. The second, a study on the effects of pH on metals toxicity, did not include sufficient water chemistry to re-analyze their data through the BLM (Schubauer-Berigan *et al.* 1993). The EPA (2007a, Appendix E) had estimated ion content for the base dilution waters based on the recipe for very-hard reconstituted water. However, the base water was amended with hydrochloric acid (HCl) to experimentally lower the pH, which would have also lowered the alkalinity and raised chloride content relative to the base water. Differences in chloride can influence *Hyalella* growth and reproduction (Dave Mount, EPA, Duluth, MN, personal communication) so perhaps it is not too great a logical stretch to assume

chloride might influence acute survival as well. Regardless, BLM predictions for copper are sensitive to the alkalinity of the waters, and alkalinity was unmeasured and assumed constant in the amended waters. Thus, the uncertainties regarding this dataset seemed such that they could invalidate validation attempts.

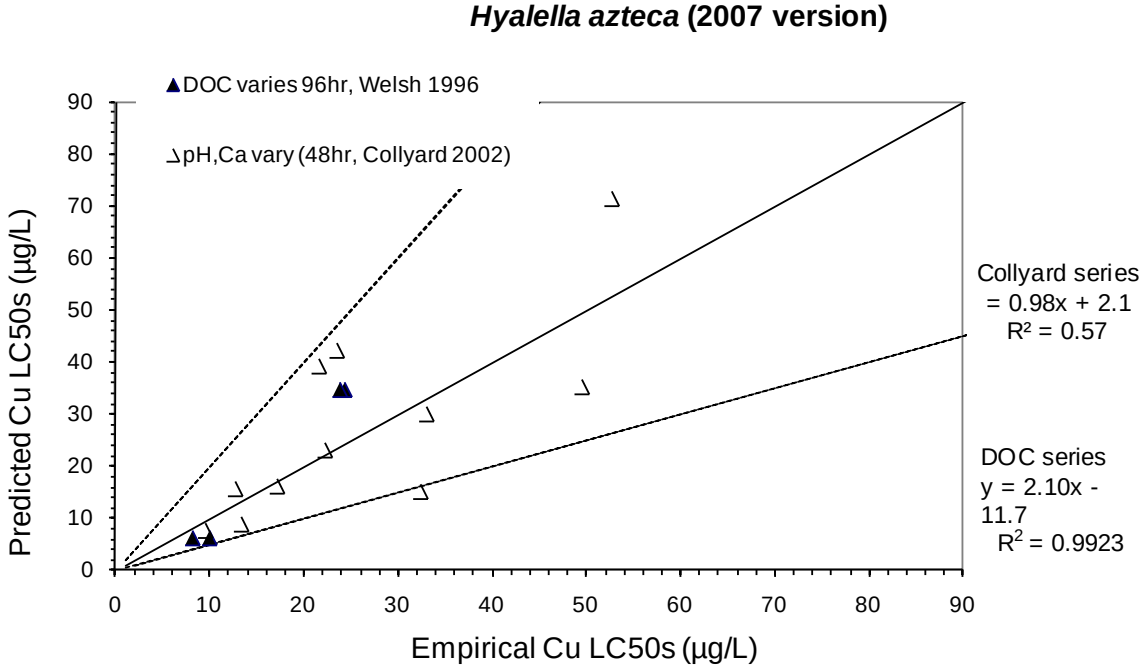


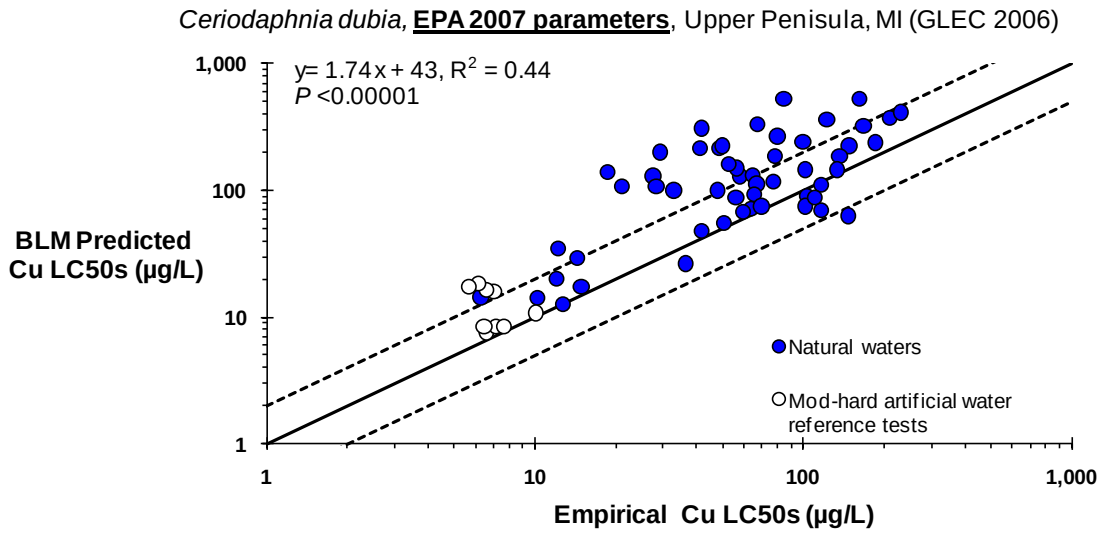
Figure 11. Amphipod *Hyalella azteca*: Predicted and empirical copper toxicity to Hyalella azteca under conditions of varying natural organic matter (NOM), pH, and calcium.

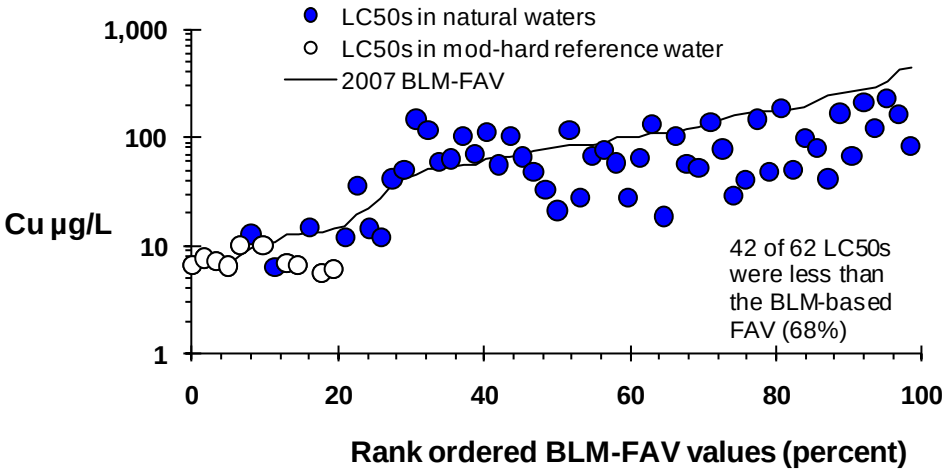
Of the *Hyalella* and copper datasets retained, the results of the BLM predictions were favorable. For the series of four tests using natural lake waters with different DOC (i.e., naturally occurring organic matter or NOM) concentrations, the predictions were highly correlated with observations although the slope of the predicted: empirical toxicity line was about 2, indicating that a stronger DOC effect was predicted than observed for these data (Figure 11). The results of the series with variable calcium and pH in the absence of DOC showed reasonable agreement, with the slope of the empirical to predicted toxicity regression approximately 1.0 and with the model explaining about half the variability in the data (R^2 of 0.56).

We then considered a comprehensive study of relative copper toxicity in natural waters collected from forested streams in Michigan's Upper Peninsula, with a wide range of hardnesses and DOC. The Great Lakes Environmental Center ("GLEC," a private environmental consulting firm) tested the toxicity to the cladoceran zooplankter *Ceriodaphnia dubia* in about 25 natural waters and in a moderately-hard artificial reference water. The hardnesses of the stream and lake waters ranged from about 17-185 mg/L $CaCO_3$ and 0.8 to 30 mg/L DOC. The toxicity in the artificial reference water was repeatedly tested as a benchmark of the inherent variability of (GLEC 2006).

The results showed that the *Ceriodaphnia* copper LC50s were correlated with the BLM predictions, with the regression explaining 44% of the variability in toxicity (R^2 value of 0.44). This is lower than some other datasets examined, and the slope of 1.7 is steeper than optimal (Figure 12, top). Still, the results were reasonably favorable, especially when compared to hardness-toxicity plot, where hardness explained less than 1% of the variability in the data, and the slope of the best fit line actually went the wrong way (decreasing toxicity with increasing hardness, Figure 12, bottom).

Comparing the *Ceriodaphnia* LC50 values with the BLM-based FAV shows that in general *Ceriodaphnia* would not be fully protected by the BLM copper criteria (Figure 12, middle). This is a two edged observation. First, *Ceriodaphnia* arguably never were intended to be protected by the 2007 copper criteria because they fall below the 5th percentile of the species sensitivity distribution (SSD) used to define the criteria and presumably *Ceriodaphnia* provide redundant ecosystem functions and are not "important" species that warrant a downward adjustment of the criteria to afford them protection. This emphasizes how fundamental the assumptions are that protecting 95% of representatives in a dataset is sufficient to protect freshwater ecosystems. With copper, these assumptions could be questionable since of the 27 genera included in the 2007 copper FAV, the lowest two genus mean acute values and lowest three species mean acute values (GMAVs and SMAVs) are for cladocerans. While individually, a cladoceran species may not necessarily be "important" because of the assumed functional redundancy provided by other cladoceran zooplankters, cladocerans as a group probably have keystone ecological functions by their intermediate role in lake food webs between algae (phytoplankton) and planktivorus fish species such as sockeye salmon. In theory, cladocerans might not be fully protected by the acute copper criterion, since of the four species included in the acute toxicity dataset, three (*Ceriodaphnia dubia*, *Daphnia magna* and *Daphnia pulicaria*) have SMAVs near or below the FAV, and the fourth should not have been included because it does not meet data quality guidelines for criteria derivation. This fourth GMAV, *Scapholeberis sp.*, listed 5th most sensitive of 27 genera, was from a single test with an adult (EPA 2007a). Under EPA's Guidelines, tests with daphnids and other cladocerans should be started with organisms that are less than 24 hours old. Tests with older animals could be considered as "other data" (Stephan *et al.* 1985, p. 33). Because the three daphnid species in the criteria dataset cannot be assumed to be the most sensitive of the widely distributed North American daphnids (Koivisto *et al*. 1992; Harmon *et al.* 2003; Shaw *et al*. 2006), this undermines conceptual support for the 5th percentile SSD criteria approach in cases where a major taxonomic group might go under protected together.


Ceriodaphnia dubia, **EPA 2007 parameters**, Upper Penisula, MI (GLEC 2006)
y= 1.74x + 43, R^2 = 0.44
P <0.00001
BLM Predicted Cu LC50s (µg/L)
Natural waters
Mod-hard artificial water reference tests
Empirical Cu LC50s (µg/L)


LC50s in natural waters
LC50s in mod-hard reference water
2007 BLM-FAV
Cu µg/L
42 of 62 LC50s were less than the BLM-based FAV (68%)
Rank ordered BLM-FAV values (percent)

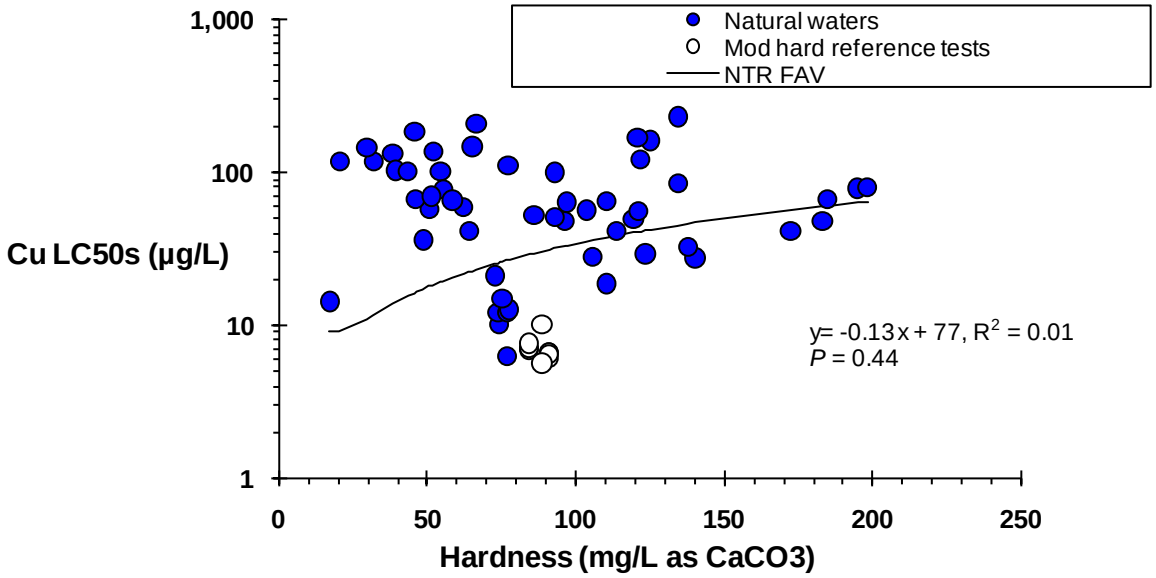

Natural waters
Mod hard reference tests
NTR FAV
Cu LC50s (µg/L)
y= -0.13x + 77, R^2 = 0.01
P = 0.44
Hardness (mg/L as CaCO3)

Figure 12. Cladoceran *Ceriodaphnia dubia:* correspondence between BLM predicted and empirical copper LC50s for in natural and reference waters (top); comparison of copper FAV and LC50s (middle); and lack of correspondence between hardness and Ceriodaphnia LC50s (bottom). Data from (GLEC 2006).

There is a flip side to the concerns that *Ceriodaphnia* and its relatives may not be adequately protected by the 2007 BLM based criteria or the hardness-based criteria. That is, because the *Ceriodaphnia dubia* three brood test is one of the two routinely required for whole effluent toxicity (WET) tests for effluent discharges, this shows that, for copper the *Ceriodaphnia* test is a sensitive tool for evaluating instream toxicity from discharges. Because there are sometimes similarities in relative sensitivity rankings of organisms with different substances, these patterns suggest the *Ceriodaphnia* WET test could be sensitive to other metals and other substances as well.

Chronic toxicity predictions with invertebrates

The mostly favorable evaluations of the performance of the BLM and the protectiveness of the BLM-based criteria for invertebrates thus far considered only acute test data. The 2007 BLM criteria are based upon an acute model of copper toxicity, and the acute BLM predictions are extrapolated to derive chronic criteria through a fixed acute-to-chronic ratio (ACR). This approach has been criticized as counter to knowledge on different mechanisms of acute and chronic toxicity of metals (Niyogi and Wood 2004) and by practical arguments that an acute copper biotic ligand model (BLM) for *D. magna* could not serve as a reliable basis for predicting chronic copper toxicity (De Schamphelaere and Janssen 2004a).

Considering these criticisms, we compiled four well-characterized datasets of chronic copper toxicity to invertebrates in order to evaluate the performance of the acute-BLM to predict chronic toxicity. The datasets evaluated were:

1. cladoceran (*Ceriodaphnia dubia*) in tests with DOC varying from <1 mg/L to 10 mg/L (Wang *et al*. 2011);

2. parallel tests with copper in 28-day exposures with the rainbow mussel (*Villosa iris*) (Wang *et al*. 2011);

3. three tests with Daphnia magna growth, survival, and reproduction in 21-day exposures in water with low DOC and a range of water hardnesses (Chapman *et al*. 1980); and

4. 35 tests with *Daphnia magna* growth, survival, and reproduction in 21-day exposures in water with mostly high DOC (range 2 – 22 mg/L) and a range of water hardnesses and pH (De Schamphelaere and Janssen 2004b).

Our review of the mussel and *Ceriodaphnia* study showed that the BLM performed remarkably well, explaining over 90% over the variability observed with the mussel survival and growth endpoints and the *Ceriodaphnia* acute and chronic survival endpoints. The empirical estimates of *Ceriodaphnia* reproduction endpoint did not vary as much as did the predicted estimates, producing weaker relations between predictions and empirical results with the model explaining only about 33% of the observed variability (Figure 13).

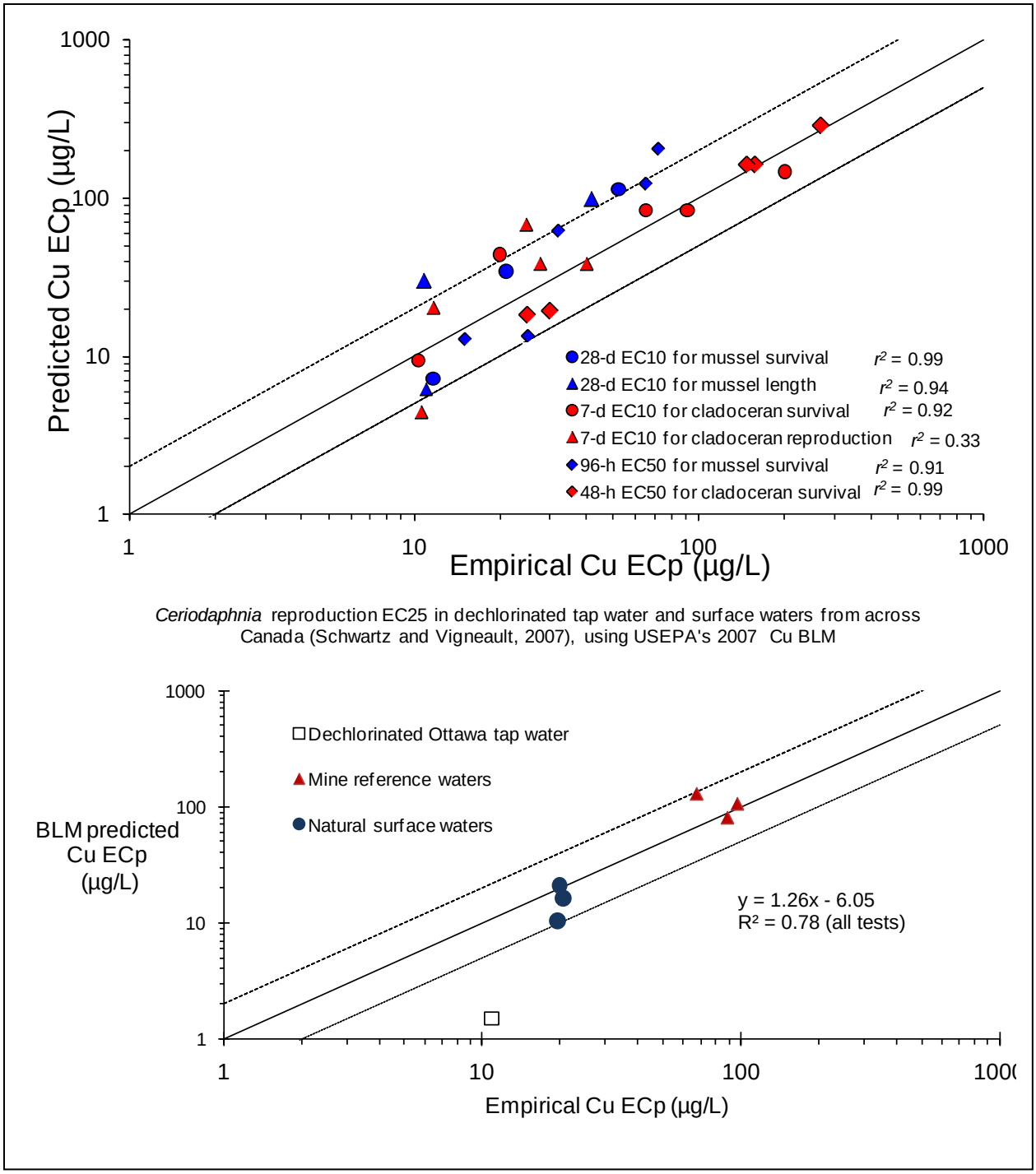


Figure 13. Chronic toxicity of copper to rainbow mussel (*Villosa iris*) and the cladoceran (*Ceriodaphnia dubia*): A. BLM-predicted and empirical acute and chronic toxicity of copper to in tests with DOC varying from <1 mg/L to 10 mg/L (Wang *et al.* 2011), B. reproductive impairment of *C. dubia* in waters with DOC ranging from 0.4 to 33 mg/L and water hardness of 23 to 170 (Schwartz and Vigneault 2007).

The difference in comparisons between endpoints may reflect the inherent variability in biological testing, and reproductive endpoints are generally more variable than growth or mortality endpoints. Using the mean critical accumulation value estimated for the BLM from

Wang *et al*'s (2011) *Ceridaphnia dubia* reproductive EC20s, *Ceriodaphnia dubia* reproductive EC25s determined with diverse surface waters from across Canada by Schwartz and Vigneault (2007) were predicted remarkably well by the BLM (Figure 13, bottom)

The performance of the BLM-based copper criteria with freshwater mussels in chronic exposures is important because many freshwater mussels are in decline in the United States, some species are among the most sensitive taxa reported to date with copper, and traditional hardness-based copper criteria are under-protective of mussels (March *et al*. 2007). Yet with acute and chronic exposures in artificial lab waters with very low DOC and with acute exposures in natural waters with a range of DOC concentrations from different sources, the BLM-based copper criteria appear mostly protective (Wang *et al*. 2007a; Wang *et al*. 2007b; Wang *et al*. 2009). The most recent study with a freshwater mussel in waters with natural DOC added indicates that the BLM-based chronic copper criteria is protective for at least the species tested, and presumably also for closely related, untested taxa.

De Schamphelaere and Janssen (2004b) tested the effects of pH (5.3–8.7), water hardness ($CaCO_3$ at 25–500 mg/L), DOC concentration (1.6–18.4 mg/L), and DOC source on the chronic toxicity of copper to *Daphnia magna* as 21-day survival or reproductive impairment. Chapman *et al*. (1980) similarly tested the chronic toxicity of copper to *Daphnia magna* as 21-day survival or reproductive impairment, in waters with hardness varying from about 50 to 200 mg/L. Reproduction was the most sensitive endpoint in both tests, and the statistical no-observed effect concentrations (NOECs) are used here for comparability between the studies.

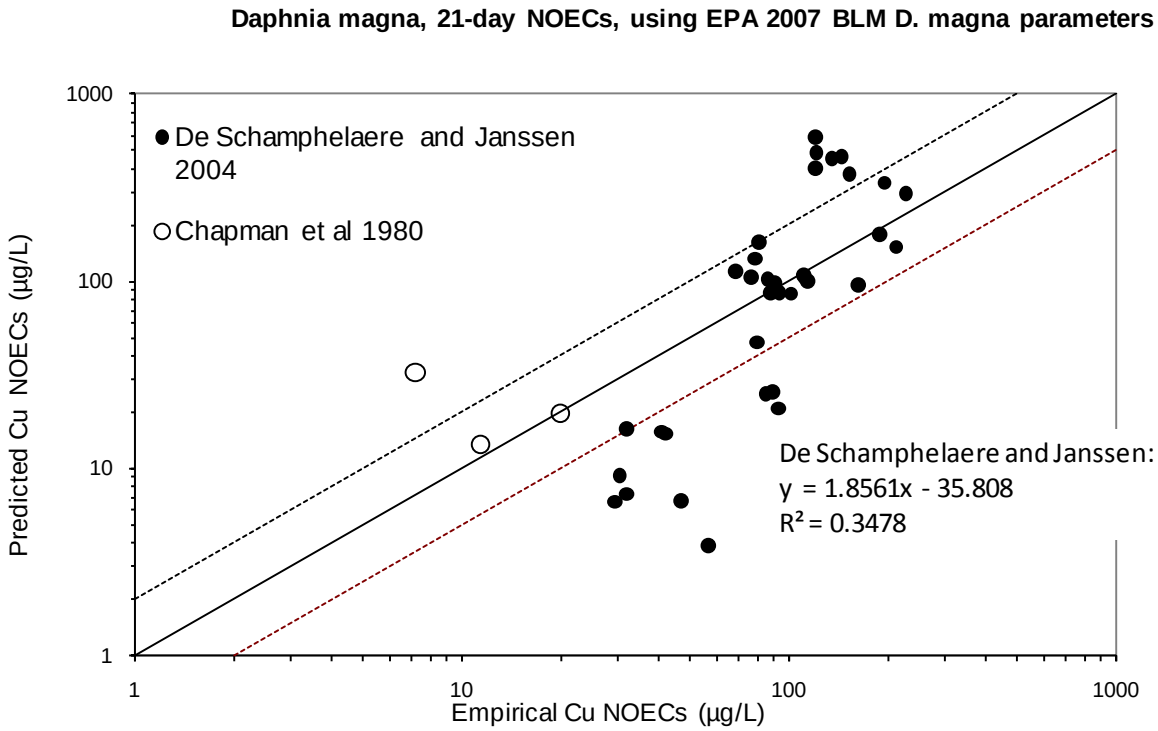


Figure 14. With chronic *Daphnia magna* in the De Schamphelaere and Janssen dataset, the tests with higher DOC tended to underpredict toxicity (NOECs too high) in tests with relatively high DOC and overpredict toxicity in tests with low DOC. In the Chapman dataset, the two points falling on the 1 to 1

line of agreement are with hardness 50 and 100 mg/L. Their test at hardness 200 mg/L was more sensitive to copper than the tests at lower hardness.

With these data, the agreement between the BLM predictions was considerably worse than in most other datasets (Figure 14). This might in part be related to working with the NOEC statistic. The NOEC is not the best statistic for comparing effects between tests because the NOEC has to be one of the concentrations tested, and the actual magnitude of this "no-observed effect" might in fact range from a 0% to 30% or more reproductive impairment, whereas with EC10 or EC50 values, a common magnitude of effect, e.g., 10% or 50%, is estimated from nonlinear curve fits (Appendix C). However, by inspecting the underlying values, this seems unlikely to explain these "noisy" relations. Instead, De Schamphelaere and Janssen (2004b) suggested a bias associated with the BLM predicting more mitigation of copper effects from DOC than indicated in the empirical data and pH having a stronger influence than accounted for in the model. The Chapman data are particularly puzzling because while toxicity was reduced exactly as predicted from a hardness of about 50 to 100 mg/L, in the test at a hardness of 200 mg/L, copper was more toxic than at either lower hardness condition. Review of the raw data from the tests indicated no test performance data quality problems, and this result remains unexplained.

We estimated critical accumulation values for Daphnia magna 21-day reproduction from the De Schamphelaere and Janssen (2004b) study with the BLM in order to make the comparisons between predicted and empirical effects. Curiously, we found, it was nearly identical to the species mean critical accumulation value ("LA50") for 48-hour lethality to Daphnia magna estimated from the EPA (2007) SMAV for Daphnia magna. Generally, 48-hour LC50 values for *Daphnia magna* are expected to be considerably higher than 21-day reproductive no-apparent values for *Daphnia magna*. Such was the case with the Chapman *et al*. (1980) study. The reason for the relative insensitivity of the De Schamphelaere and Janssen (2004b) chronic data is unexplained, although they suggest that their treatment of only considering about 50% of the DOC as having a role in reducing copper toxicity, compared to the EPA (2007) approach of treating 100% of the DOC as having a role in reducing copper toxicity as one factor (De Schamphelaere and Janssen 2004a).

Chronic toxicity predictions with fish

No previous evaluations of the 2007 BLM, developed as an acute model of copper toxicity, are known of for predicting chronic copper toxicity to fish, especially listed salmonids or their surrogates. As with the case of predictions of chronic effects to invertebrates, the extrapolation of the acute model to chronic predictions through a fixed acute-to-chronic ratio (ACR) has been strongly criticized as speculative and counter to knowledge on different mechanisms of acute and chronic toxicity of metals (Niyogi and Wood 2004). However, this is the approach used by EPA (2007). Here the question considered is not whether the ACR extrapolation compromises the mechanistic basis of the model (it does), but as a pragmatic issue, can the 2007 acute BLM produce reasonable estimates of chronic toxicity to fish? If not, are the criteria still protective?

Because of the expense and complexity of chronic toxicity testing, chronic data are much rarer than acute data. Also, with acute data, usually only the 50% mortality endpoint is considered, but with chronic data a variety of endpoints may be tested (e.g., growth as length or weight, fecundity, survival), and statistical endpoints that approach the threshold for the onset of

adverse effects are of more interest than an extreme 50% effect. Test durations of "chronic" tests generally range from 30 days to over 2 years. For instance, with growth reductions, fish will often die before growth reductions on the order of 50% are ever realized. We located several datasets, although the conditions tested were much more limited than with acute data. Few data tested different water chemistry conditions within a study. Exceptions were a pair of 30-day tests with fathead minnows in natural waters with different DOC and pH by Welsh 1996; and a study with nine tests of different hardness and pH, although in that case some assumptions about missing water chemistry had to be made (Waiwood and Beamish 1978).

With salmonids, we located interpretable datasets with rainbow/steelhead trout (Waiwood and Beamish 1978; Seim *et al*. 1984; Marr *et al*. 1996; Hansen *et al*. 2002a; Besser *et al*. 2005), Chinook salmon (Chapman 1982), and brook trout (McKim and Benoit 1971; McKim and Benoit 1974; Sauter *et al*. 1976; Besser *et al*. 2001). With fathead minnows, interpretable data sets were located that used natural waters (Lind *et al*. 1978; Welsh 1996) and laboratory waters (Mount 1968; Besser *et al*. 2005).

Where needed, we estimated necessary major ion chemistry and DOC inputs to the BLM from other studies or regional values, or from data collected at different times from the same water source. For example, the Sauter *et al*. (1976) tests used as a water supply a 400 ft deep well into bedrock that is still in use. Because the water chemistry in the well appears stable (Mark Cafarella, Springborn Smithers Laboratories, personal communication), and hardness, alkalinity, and pH from the well were similar in 2006 and 1976, major ion and DOC data from 2006 were assumed similar as well. Waiwood and Beamish (1978) said their water chemistry was similar to their (1978) study, which still didn't have all the needed information but was assumed to be similar to that reported by Dixon and Sprague (1981) for the same laboratory. No information on DOC in the University of Guelph toxicity laboratory water supply or in the City of Guelph drinking water reports[11] could be found. Because the water source was dechlorinated City of Guelph tap water that originated as well water, a fixed DOC value of 1 mg/L, since that has been considered a reasonable estimate for similar waters (EPA 2007a, Appendix C).

As an estimate of thresholds of adverse effects, EC10s were estimated for the most sensitive endpoint of each test by nonlinear regression using EPA's Toxicity Response Analysis Program (TRAP) (Erickson 2008). In the Waiwood and Beamish (1978) dataset, no raw data were reported, so it was necessary to use their EC25 values for reduced growth. Swimming performance (10% reduction) was proportional to 25% growth reductions over the same pH and hardness combinations, but growth was more sensitive (Waiwood and Beamish 1978). Thus only growth was evaluated here.

[11] e.g., http://guelph.ca/uploads/ET_Group/waterworks/2003_Waterworks_Summary_Report.pdf

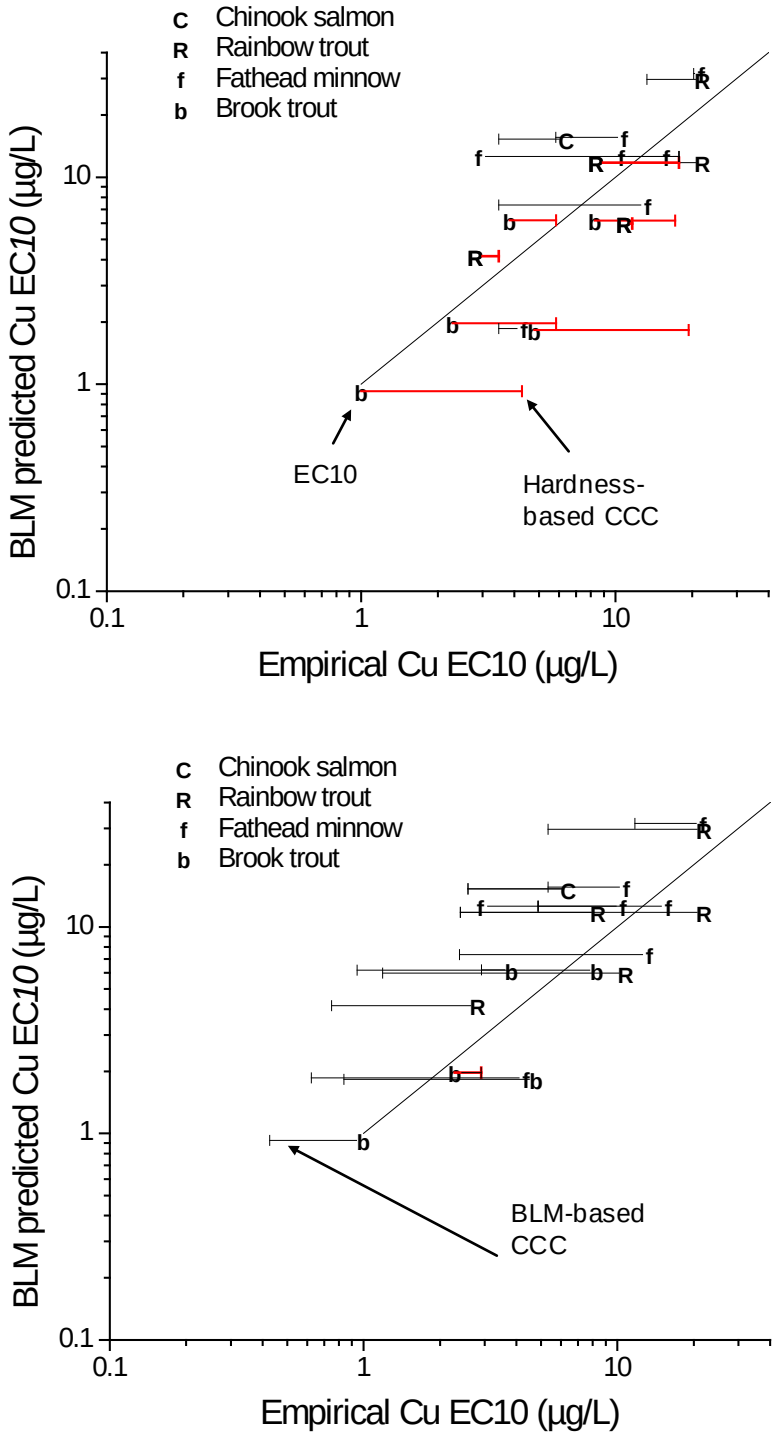


Figure 15. Protectiveness or non-protectiveness of both hardness-based (top) or BLM-based Cu criteria (bottom) and observed vs. BLM-predicted chronic EC10 values for Chinook salmon, rainbow trout, brook trout and fathead minnows. Horizontal error bars leading to the left of the symbols indicate the difference between the EC10 and the CCC or "safety margin" for the occurrence of low adverse effects and the criterion; horizontal error bars, emphasized in red, leading to the right of the symbols indicate

the degree of non-protectiveness of the criterion for that test value. Solid diagonal line is the 1:1 line of perfect agreement. (BLM version 2.2.3, default parameters)

Despite the disparate test methods used across studies and the many estimates needed to come up with the inputs for the BLM, the results were surprising good, as good or better than some of the acute comparisons. The BLM was able to account for 54% to 71% of the variability in the chronic datasets (Figure 15).

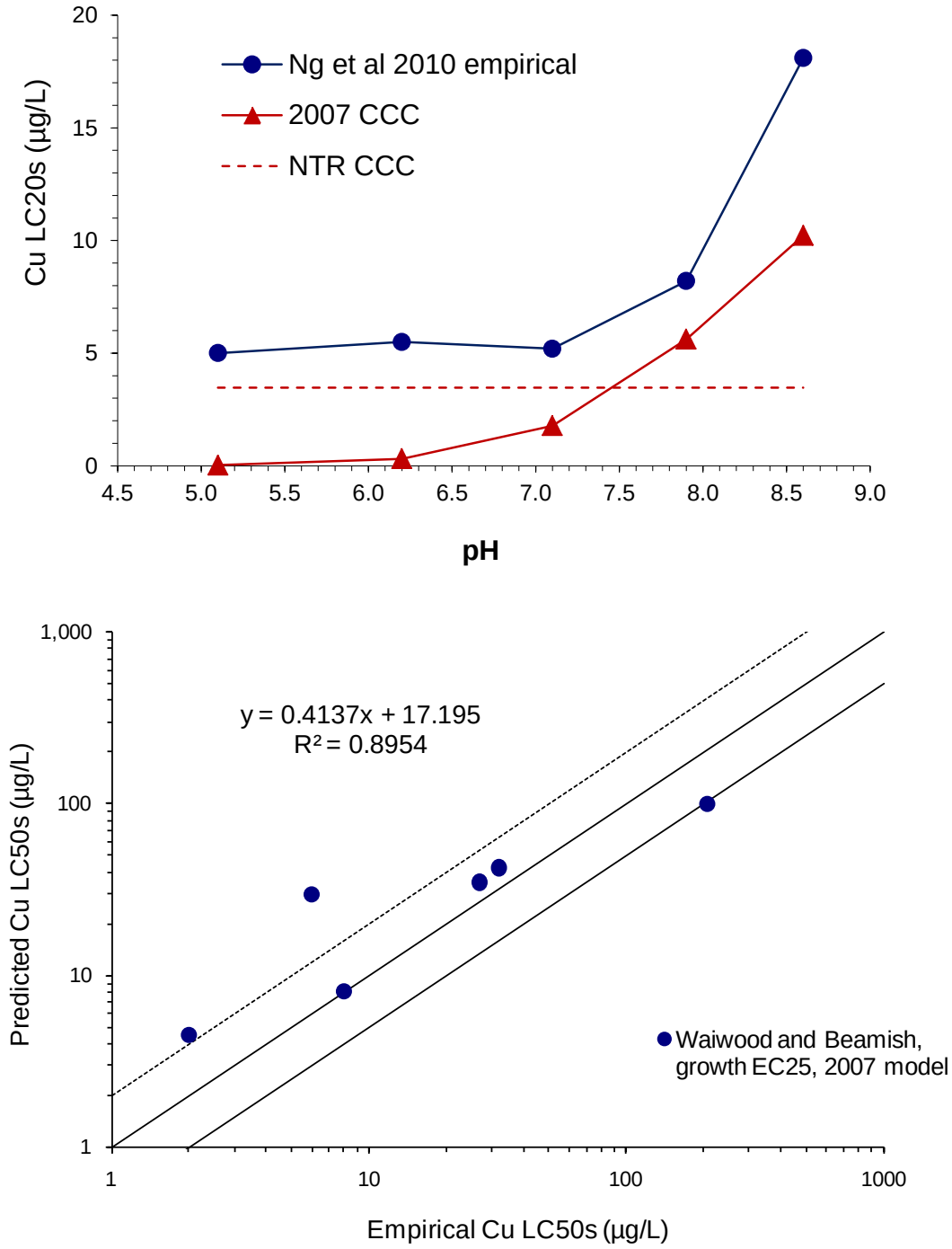


Figure 16. Comparisons of empirical results of juvenile rainbow trout 30 day exposures with BLM predictions. (Top) Rainbow trout copper 30-day LC20s across pH gradient in low hardness (11-22 mg/L), water with DOC ~1.5 mg/L (Ng *et al*. 2010). Both the BLM-based and hardness-based chronic Cu criteria were protective, however, only the BLM-based criteria mimicked the direction of responses. The BLM-based criterion values were very low compared to the empirical results at pH <6.5. (Bottom) BLM compared with empirical EC25 estimates of growth (weight) reductions from 30-day growth tests that manipulated inorganic water chemistry (pH and hardness), using the 2007 BLM parameters (Waiwood and Beamish 1978).

For these chronic tests estimating the thresholds where adverse effects just begin to occur (EC10, discussed in Appendix B), rather than the "factor of 2" prediction bands, the protectiveness or lack thereof of the chronic criterion is shown as a horizontal line extending to the left or right of each EC10 estimate. The ends of the horizontal lines show the chronic criteria for the water chemistry conditions of each test. Thus, a horizontal line extending left from each point is favorable, indicating that the chronic criterion was lower than the test EC10. For 17 of 18 tests analyzed in this way, the chronic criterion was protective for that test. The exception was one of three replicate 30-day fathead minnow tests conducted by Besser *et al.* (2005); this point is located parallel with the legend entry "Fathead minnow") in Figure 15.

The comparisons of the BLM predictions and empirical estimates with Waiwood and Beamish's tests in which they manipulated pH, hardness, and alkalinity were also surprisingly strong, considering the number of estimates needed for the water chemistry model inputs. The empirical effects estimates ranged from 2 to 206 µg/L, and the BLM predictions ranged from about 6 to 100 µg/L, with the BLM accounting for almost 90% of the observed variability (Figure 16).

Chemosensory or behavioral effects

Another type of adverse effect caused by copper is neurotoxicity, which can impair the ability of fish to complete normal migrations and prevent salmonids from migrating downstream or from homing on their natal stream (Hecht *et al*. 2007; McIntyre *et al.* 2008a; Green *et al*. 2010). Copper is neurotoxic to fish and interferes with the function of the peripheral olfactory nervous system, as well as the function of the mechanical sensory cells located on the lateral lines of fish that keep fish oriented to currents, schooling behaviors, and flight responses among other functions. However, the structure of the olfactory epithelium, lateral line epithelium, and gill epithelium all differ, leading to debates whether the BLM which was developed for the gill epithelium, functions adequately for olfactory or lateral line toxicity (Linbo *et al.* 2006; McIntyre *et al*. 2008a; Meyer and Adams 2010).

To evaluate this issue, we re-interpreted three studies: (1) destruction of lateral line hair cells on larval zebrafish following copper exposures under differing inorganic chemistry and with natural organic matter additions (Linbo *et al.* 2009); (2) olfactory inhibition in coho salmon following short-term (30 minute) exposures to copper under differing pH, hardness, alkalinity and DOC conditions (McIntyre *et al.* 2008b, a); and (3) reduced survival of olfactory inhibited copper-exposed coho salmon in staged encounters with a wild predator, cutthroat trout (McIntyre *et al.* 2012).

Our analysis of the lateral line study showed very strong correlations between predicted and empirical EC50 values for mechanical-sensory hair cell destruction. However, the patterns were very different for different water quality parameters (Figure 17). Increasing alkalinity by adding sodium bicarbonate had little effect on empirical EC50s, although predicted EC50s rose steeply with increasing alkalinity and sodium. Predictions also nearly perfected correlated with empirical results from increasing DOC, although predicted values increased more than empirical values, with a slope of 2.7. Calcium, Mg, Ca+Mg, and Na additions resulted in very high R^2 values, with slopes less than 1. No hair cell EC50s were lower than the BLM-based FAV. As with olfaction, these results support the value of further investigations, but suggest that the 2007

BLM acute criterion is probably protective of lateral line damage from copper, assuming that the function and physiology of lateral lines is similar across fish species (e.g. Linbo *et al*. 2009).

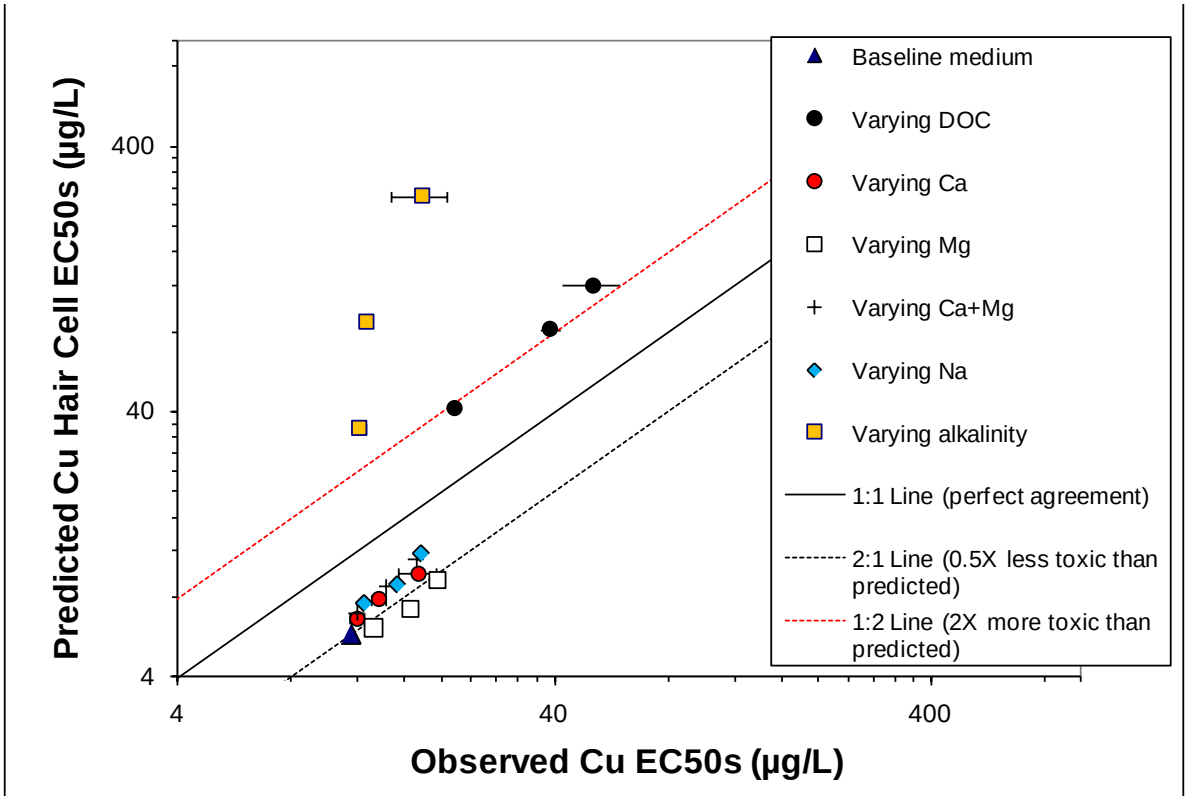
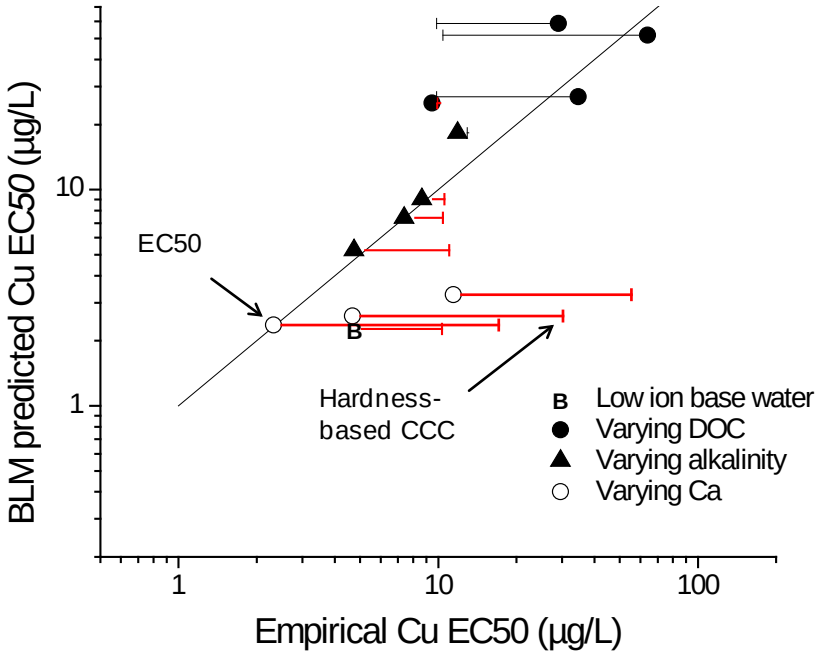


Figure 17. Destruction of lateral line hair cells in zebra fish, empirical and BLM predicted values (data from Linbo *et al.* 2009).

With the inhibition of olfaction in coho salmon from copper (McIntyre *et al*. 2008b, a), the results showed reasonable agreement between the BLM predicted copper concentrations causing 50% olfactory inhibition in coho salmon using the "factor of 2" rule-of-thumb for evaluating model predictions (Figure 18). However, there were differences in how well the BLM handled the different water chemistries. With varying alkalinity, predictions were nearly perfectly correlated with empirical estimates, although the slope was steeper than 1.0. With varying calcium, correlations were similarly very strong, but the slope was much shallower than 1.0. With added DOC, correlations were more scattered and weaker. These patterns suggest further evaluations would be appropriate, yet the overall pattern of model predictions seemed favorable. Further, none of the empirical results were lower than the BLM based FAV, indicating that the acute BLM-based criteria would likely be protective from olfactory inhibition in coho salmon caused by copper.

Other tests from the same laboratory suggest that other salmon species have similar responses to copper, and thus these results with coho salmon are considered to be relevant to other salmonid species including Chinook salmon and steelhead (Baldwin *et al.* 2010).



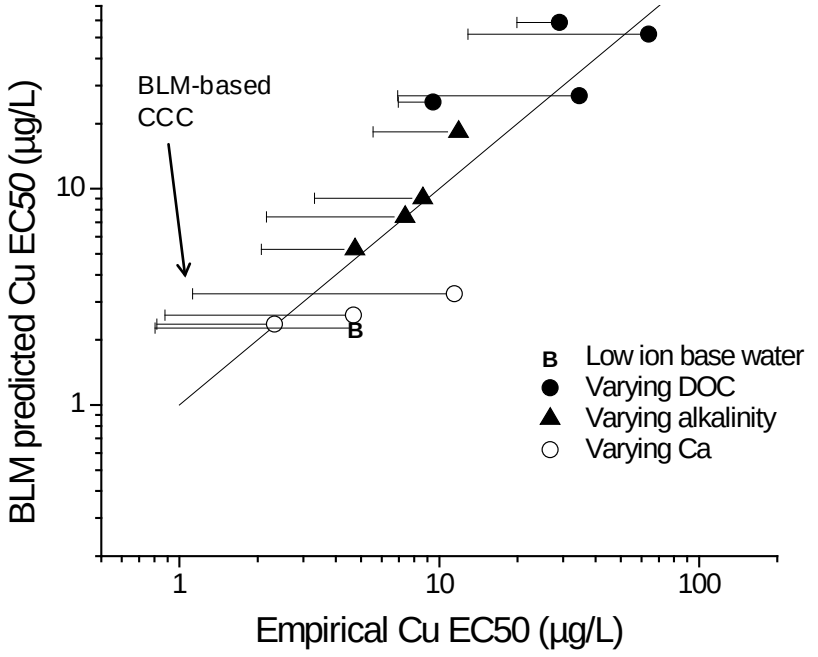


Figure 18. Inhibition of olfaction in coho salmon from copper following 30 minute exposures, observed and BLM predicted values (data from McIntyre *et al.* 2008a,b). Horizontal error bars leading to the left of the symbols indicate the margin of safety between the EC50s and the Final Acute Value (FAV); error bars, emphasized in red, leading to the right of the symbols indicate the degree of non-protectiveness of

the FAV for that test value. Both the EPA (1992) hardness-based criteria (top) and the EPA (2007) BLM-based criteria (bottom) are shown. Solid diagonal line is the 1:1 line of perfect agreement.

Prey fishes have a behavioral alarm response to olfactory predation cues that provides a survival benefit when under attack (Mirza and Chivers 2001, 2003). It logically follows that that survival benefit could be compromised if a pollutant such as copper disrupts the behavioral alarm response (Scott and Sloman 2004; Sandahl *et al.* 2007). This presumed reduction in survival of copper-exposed prey fish in predator-prey encounters was demonstrated by McIntyre *et al*. (McIntyre *et al*. 2012). They found that copper exposure altered prey (juvenile coho salmon) response to olfactory predation cues in the presence of predators (adult cutthroat trout), and that this "info-disruption" reduced prey fish survival. Copper exposure made prey easier for predators to detect and capture. The primary impact of copper on predator-prey dynamics in her study was faster prey detection, shown as faster time to attack and time to capture. Copper-exposed prey were more active than control prey during predation trials. For visual predators of juvenile fishes (e.g. salmonids, birds, otters), prey activity is a critical determinant of detection by predators (McIntyre *et al*. 2012).

McIntyre *et al.* (2012) conducted the predator-prey interactions in relation to copper exposures in two trials. The first trial tested encounters between copper-exposed prey and non-copper exposed predators. The result was a graded decline in prey survival times over copper concentrations ranging from about 0.2 µg/L in controls to 20 µg/L, in a freshwell water with very low organic carbon concentrations (≤ 0.25 mg/L DOC). No threshold of response was found; reduced survival times were observed at the lowest concentration tested, 5 µg/L (Figure 19). In McIntyre *et al*.'s second trial, both the prey and predators were exposed to copper in a subset of the trials, which did not markedly improve the ability of copper-exposed prey to evade the copper-exposed predator (Figure 19). This result was attributed to the fact that cutthroat trout are visual predators and copper exposure is not expected to affect their eyesight.

For the water chemistry conditions of the exposure waters from McIntyre *et al*.'s (2012) study, the 2007 BLM-based acute copper criterion is only slightly higher than the average copper concentrations measured in the control waters. In contrast, the acute hardness-based criterion (the Idaho and NTR criterion, EPA 1992) values for a hardness of 56 mg/L $CaCO_3$ is about 10 µg/L, well into the range of decreased prey survival. In this study, no minimum threshold below which copper-exposures have no or little effect on predator-prey interactions was obtained, so no strong conclusions about "safe" copper concentrations could be made. However, adverse effects at the BLM-based criterion concentration would seem unlikely since it was close to the control concentration.

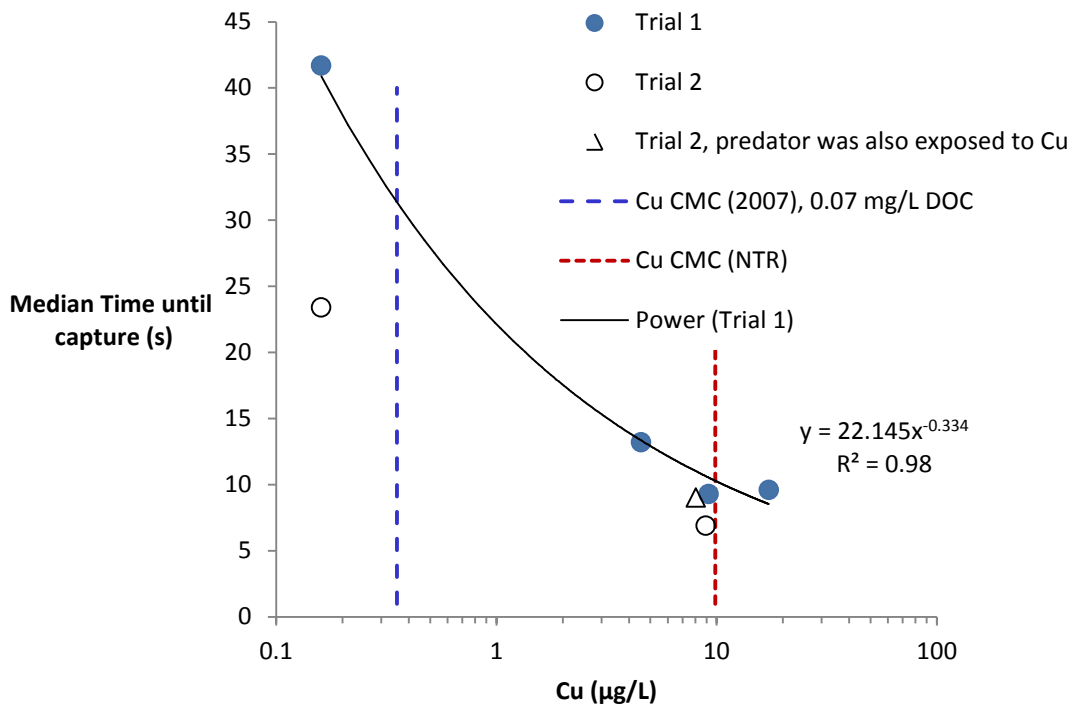


Figure 19. Survival times of juvenile coho salmon prey before being eaten by an adult cutthroat trout predator, as a function of copper exposure concentrations (3-hr durations) prior to the predation experiments. Unlike natural environments, in the circular tank there was no place to hide or escape, so all prey were eventually eaten regardless of copper exposure. In a natural environment with hiding places and escape routes, the prey that evaded capture longest in would presumably have a better chance of ultimate escape. Data from McIntyre *et al*. (2012).

Chinook salmon and rainbow trout have also been shown to be very sensitive to avoidance, and the loss of olfactory responses at copper concentrations less than the hardness-based copper criteria (Hansen *et al*. 1999a). Hansen *et al.* (1999a) found that behavioral avoidance of copper in soft water differed greatly between rainbow trout and chinook salmon. Chinook salmon avoided at least 0.7 µg Cu/L, whereas rainbow trout avoided at least 1.6 µg Cu/L in water with low DOC and a hardness of 25 mg/L in 20-minute exposures. These lowest observed effect concentrations (LOECs) were considerably lower than the hardness-based acute criterion for the test waters, 4.6 µg/L. In contrast, the estimated BLM-based allowable acute criterion concentrations for the test conditions were below or close to these LOECs for behavioral avoidance, about 0.4 to 1.1 µg/L. The BLM-based criterion concentrations are estimated from other studies because Hansen *et al.* (1999a) did not measure all the necessary BLM parameters. The major ion data were taken from Marr *et al.* (1996), a nearly contemporaneous study at the same lab, with the same targeted blend of well and reverse osmosis (RO) deionized water, and most of the same investigators. Hansen *et al*. (2002b) reported DOC near 0.1 mg/L in further tests that used similar softwater blends in the same lab with about the same proportions of well and RO water as did the Hansen *et al.* (1999a) study. This yielded a BLM-based acute criterion concentration of about of 0.4 µg/L for Hansen *et al.*'s (1999a) behavioral tests.

Hansen *et al.*'s (1999a) behavioral avoidance results were also reinterpreted by Meyer and Adams (2010) in the context of whether the BLM-based or hardness-based copper aquatic life criteria would be protective. Meyer and Adams' (2010) reinterpretation differed from that used here in that instead of comparing LOECs, they developed regression based estimates of 20% and 50% increases in behavioral avoidance. This avoided a limitation of using statistics like LOECs when comparing effects across studies. The LOECs simply reflect the lowest concentration with a response that with 95% confidence was statistically different from the controls, but tell nothing about the size of the effect that was different, for instance whether a 5% or 50% response was "different." Using Meyer and Adams' (2010) estimate of 20% avoidance effect as a threshold of appreciable avoidance (EC20) of about 0.84 and 0.91 µg/L would also indicate that the BLM-based acute criterion was close to or below the concentration causing olfactory-related impairment. Meyer and Adams' (2010) argued that while less than 20% olfactory-impairment might be considered important for some species of concern, the variability associated with behavioral testing would make a smaller effect percentile of questionable meaning. As with the LOECs, the EC20s and even EC50s were lower than the hardness-based acute criterion.

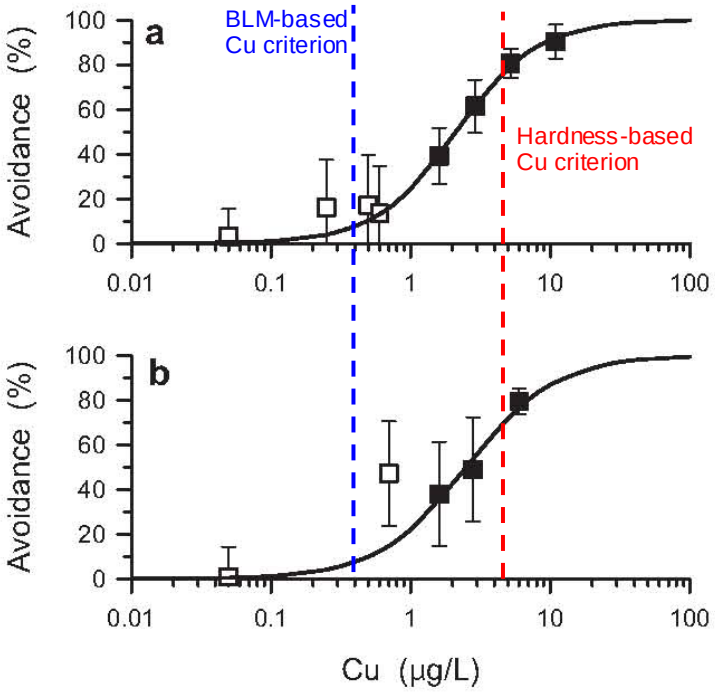


Figure 20. Avoidance of copper by rainbow trout (a) and Chinook salmon (b) in softwater with low DOC in relation to 2007 BLM-based (blue dashed line) or 1992 hardness-based acute copper criteria (red dashed line). Open or closed symbols indicate values lower or above the copper detection limit used. Base figure was taken from Meyer and Adams (2010) using original data from Hansen *et al.* (1999a).

In summary, the available information indicates that the BLM-based copper acute criterion would likely be protective against neurological damage or behavioral impairment

resulting from short-term (<< 1 day) copper exposures. The older hardness-based copper acute criterion (the Idaho criterion under consultation) would be considerably underprotective for chemoreception, behavioral avoidance, predator avoidance, and survival from predators.

Field and experimental ecosystem studies

Our reviews up to this point have relied on carefully controlled laboratory studies. For, our final evaluation of the protectiveness of the BLM-based chronic criterion we consider how the BLM is likely to perform under more realistic field conditions. Field validation of laboratory or mathematical models through field surveys or ecosystem manipulations may represent some ideal for ecotoxicology, but it is an elusive ideal to achieve. This is in part due to the scale of effort needed to conduct a rigorous study and ethical constraints on manipulating natural ecosystems, but also because field studies tend to be specific to the locale, season, etc. studied and may be difficult to extrapolate to other ecosystems. Yet some ambitious experimental manipulations of whole streams by adding copper have been completed that are very relevant, as well as small scale stream tests constructed streamside. NMFS located and re-interpreted three high quality field experiments and a small scale microcosm test relevant to our evaluation of the BLM-based copper criteria.

The most ambitious study we reviewed was an intensive, multi-year study of Shayler Run, an Ohio stream. The study took place prior to and during 33 months of copper additions, and during recovery from the copper additions. The Shayler Run drainage basins is underlain by limestone and other carbonate rocks and received sewage input from a small town upstream of the study area. Thus the hardness, alkalinity, pH, and DOC were all fairly high in Shayler Run. Measured direct effects on fish were death, avoidance, and restricted spawning. Chronic tests were done on-site at Shayler Run with stream species and fathead minnow(Geckler *et al*. 1976). The stream and test waters were well characterized chemically, and all necessary BLM data except for sulfate could be pieced together reasonably well from the study report. Sulfate was well estimated by regression from chloride (R^2 0.94) from USGS data for station 03247400, Shayler Run near Perintown OH.

While well conducted field studies such as the Shalyer Run study may identify adverse effects thresholds with some precision, a difficult question is what stream chemistry conditions should be attributed to the observed effects? The effects probably resulted from long-term exposures to copper, but the stream characteristics such as pH, DOC, and thus the water quality criteria vary over the course of the experiment in a manipulated system like Shayler Run. Presumably adverse effects have resulted from some critical condition where for example, DOC was low and thus copper more toxic, but this is an educated guess that cannot be demonstrated or falsified from the available data.

For this review, we considered the approximate range of apparently "safe" copper concentrations for the stream ecosystem to be about 29-47 µg/L, using ACRs they determined with streamside acute and chronic tests (Geckler *et al*. 1976, p. 170). This upper range could be optimistic, since they noticed that the chronic tests underestimated the instream toxicity by about two times because only the effects of copper on survival, growth, and reproduction were measured; avoidance was not measured, and it was a significant effect in the stream. For instance, bluntnose minnows only spawned where total copper concentrations ranged from 35-77 µg/L (minimum they could access), but still a seven-fold reduction in fry occurred. With these considerations, for an effect benchmark for the overall study, we estimated an approximate

NOEC for the streamside chronic tests, of 29 µg/L. This value is about two times lower than the lower range of clearly adverse effects and might account for the unmeasured avoidance effects on minnow populations.

Figure 21 shows this benchmark in comparison to a monthly time series of BLM-chronic criteria values during the study. Since copper was held nearly constant in the streamside tests, but characteristics affecting toxicity varied, the conditions when copper would have been most toxic are shown as the dips in Figure 21, which occurred when DOC and pH were relatively low. These conditions seem more important than the peaks in graph when conditions were least toxic or some average condition. At these times the BLM-chronic criterion was lower than the selected benchmark. This suggests that the BLM-CCC probably would be protective for the Shayler Run situations.

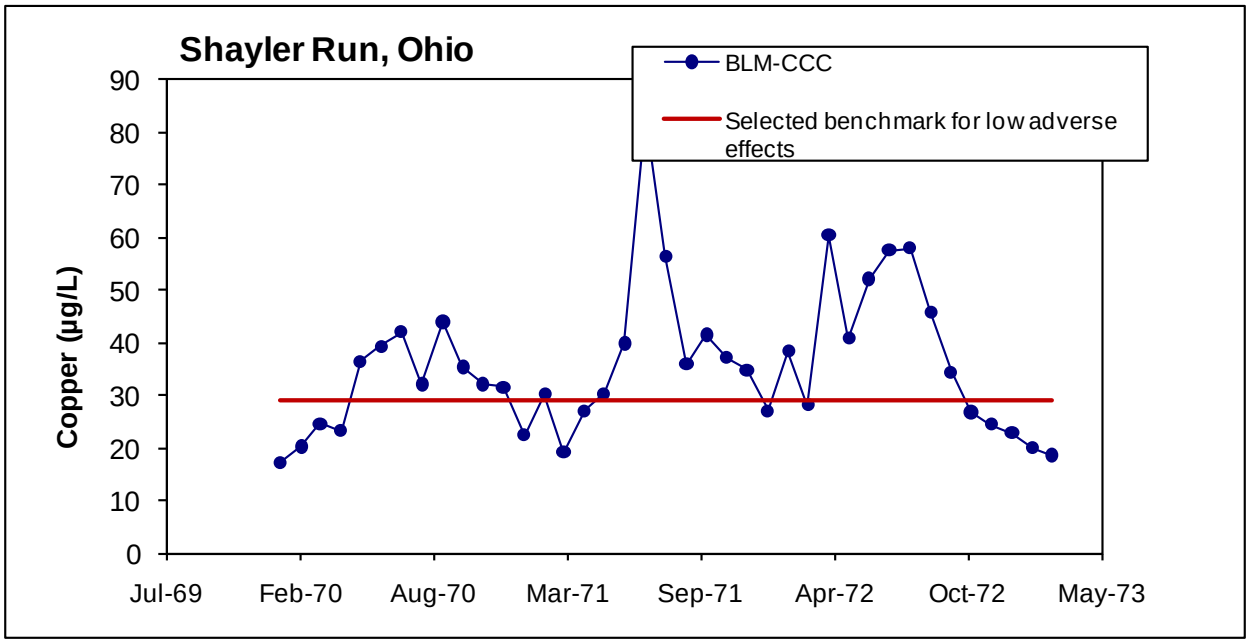


Figure 21. Seasonal patterns in BLM-chronic criterion in Shalyer Run field study, in comparison to an estimated benchmark of adverse effects based on streamside tests. Average hardnesses 180 (126-220), DOC 6 (4-12 mg/L), pH 8,1 (7.75-8.3). Original data from Geckler *et al.* (1976).

The second experimental stream study we examined was smaller in scale but was a western montane stream in the Sierra Nevada with soft water in a granitic drainage basin and thus particularly relevant to Idaho mountain streams. Convict Creek was dosed with copper for one year in different reaches with average copper concentrations of about 2.5, 5, 10, and 15 µg/L. Measured effects included stream ecosystem structural measures such macroinvertebrate community diversity and stream ecosystem functional measures such as stream metabolism energy production. (Kuwabara *et al.* 1984; Leland and Carter 1984, 1985; Leland *et al*. 1989). All needed BLM inputs except DOC were reported. We estimated a range of plausible DOC values for Convict Creek from a study of DOC in high lakes in the Sierra Nevada that were likely similar to that expected for the Convict Creek drainage basin, with a mean (range) of 1.9

(0.9 – 2.5) mg/L (Brooks *et al.* 2005; Daniel Dawson, Sierra Nevada Aquatic Research Laboratory, personal communication).

We interpret these data as follows. The minimum value from the Sierra high lakes seems prudent to use as the baseflow value that lasts most of the year. The maximum measured DOC value would be expected to occur during runoff high flow, which during the study water year appeared to have occurred in early July when calcium dropped. This assumption follows from our review of stream chemistry seasonal patterns discussed in the following sections.

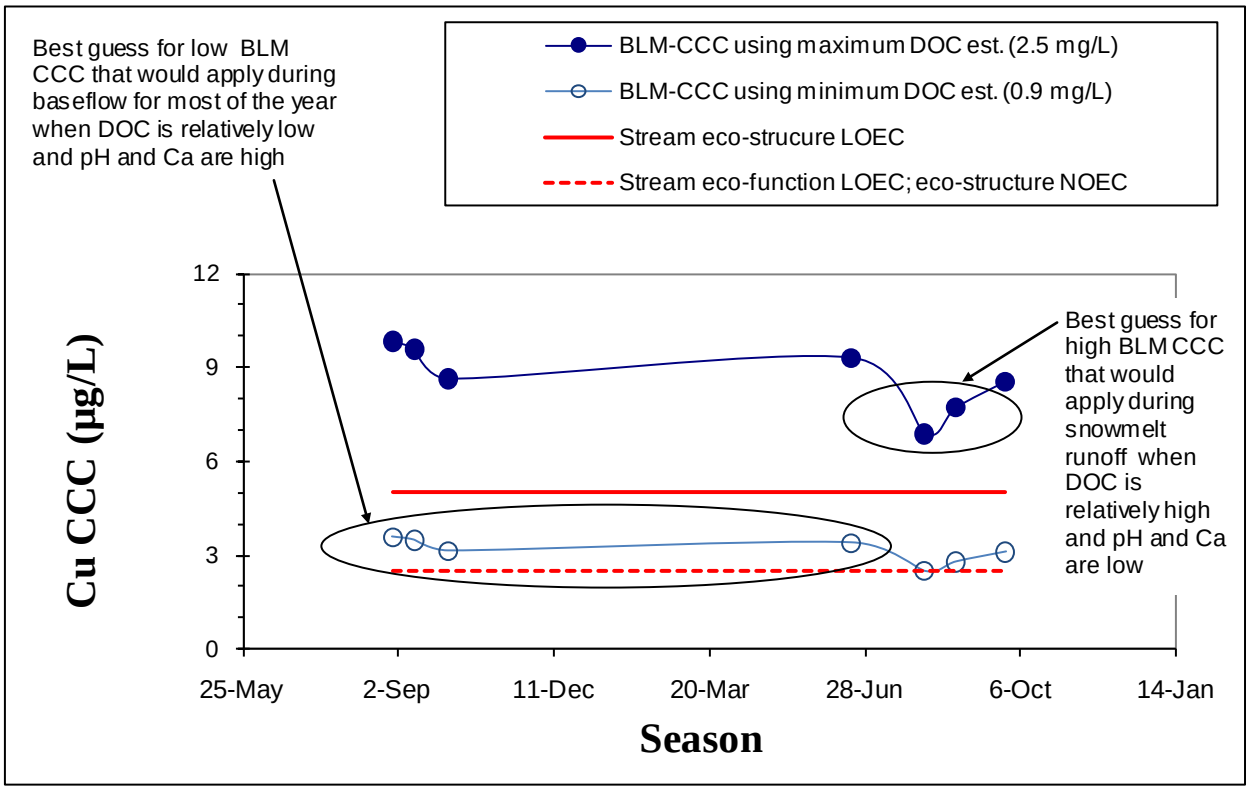


Figure 22. Stream ecosystem alteration following copper additions in Convict Creek (Sierra Nevada), California vs. estimated BLM-based chronic copper criteria. Original data from Kuwabara *et al.* 1984; Leland *et al.* (1985,1989). LOEC for adverse effects to ecological function (energy production) LOEC for adverse effects ecological structure (invertebrate diversity).

Measureable shifts in ecosystem function occurred in even the lowest copper treatment (decreased gross primary productivity and decreased respiration) of 2.5 µg/L. No effects of copper to the macroinvertebrate community were detected at 2.5 µg/L, but declines in population density of species representing all major orders (Ephemeroptera, Plecoptera, Coleoptera, Trichoptera, and Diptera) occurred at 5 µg/L copper and higher (Leland and Carter 1985; Leland *et al.* 1989). The BLM-CCC for the test conditions would probably be low enough to be below the adverse 5 µg/L treatment most of the time, and would be close to the 2.5 µg/L treatment that caused no apparent adverse effects to the macroinvertebrate community. In contrast, the Idaho/NTR-hardness based chronic criterion for these conditions would be between 6 and 8 µg/L. The BLM-CCC would not have been low enough to prevent stream metabolism depression (Figure 22). However, the depressed primary production via algae was not obviously reflected in secondary energy production in the macroinvertebrate community. Thus, the BLM-

CCC does not appear to be low enough to prevent measureable effects, but so long as secondary production from the macroinvertebrate community remained intact, the reduction in primary productivity is unlikely to carry over to salmonids.

Next we considered a rigorous, ecologically relevant streamside study of copper effects on macroinvertebrates. Natural assemblages of aquatic macroinvertebrates were established on substrate-filled trays which were then transferred to outdoor stream mesocosms adjacent to the New River, Virginia. Exposure of these communities to low levels of copper and zinc (target concentration = 12 µg/L) significantly reduced the number of taxa, number of individuals, and abundance of most dominant taxa within 4 days (Clements *et al*. 1988; Clements *et al*. 1989). Zinc concentrations on the order of 12 µg/L were unlikely sufficient to contribute to the reductions (Clements *et al*. 2000). A 42-day exposure almost completely extirpated mayfly communities as well as the sensitive *Tanytarsini* midges. In the second experiment, after 10 days, 6 µg/L copper was sufficient to eliminate 50% of the total macroinvertebrates (i.e, community EC50 of 6 µg/L) and even 2-3 µg/L copper were sufficient to cause a significant decline in macroinvertebrate communities.

As is commonly the case, only hardness, alkalinity, pH, conductivity, and temperature were measured as part of the study, which required NMFS to make estimates of other water chemistry parameters needed to run the BLM. The study was conducted close to a USGS monitoring station with sufficient data to make reasonable estimates of likely BLM-input for the experimental conditions (USGS station 0317500, New River at Glen Lyn, Virginia). During 1987 to 1988 when the biological tests were conducted major ion, conductivity, and pH data were collected by the USGS at the site, but not DOC. DOC data were collected during 1997. The inorganic parameters at the location were similar during the summers of 1987, 1988, and 1997. DOC concentrations during 1997 were not highly variable (1.1 to 2.4 mg/L). Assuming that DOC concentrations in the New River at Glen Lyn in the summer of 1997 were representative of DOC concentrations in the summers of 1987 and 1988, estimated BLM criteria for the macroinvertebrate experiments can be compared to the adverse effects values (Figure 23).

The results of the comparison shows that macroinvertebrate communities are very sensitive to copper with declines in abundance occurring as low as 2 -3 µg/L, which is about the same as the minimum BLM CCC estimate during summer at the study site. Severe effects occurred by 6 µg/L, and a 12 µg/L treatment for 42-days almost completely extirpated mayflies. In 1997, the BLM-CCC rose in late summer because of a half unit pH rise to about 8.3, which was similar to the fairly high initial pH values in the mesocosms. Still, pH variations of half unit or more over the course of a day are not uncommon in streams, even streams that are fairly oligotrophic (e.g., Nimick *et al*. 2011; Balistrieri *et al*. 2012), so perhaps the high BLM CCC values resulting from the high pH values should not get undue emphasis, and the BLM CCC calculated for the lower pH values should also be considered representative of the experimental conditions (Sep. 1996 to June 1997 values in Figure 23). These values are generally below the copper concentrations causing severe adverse effects in the New River experiments.

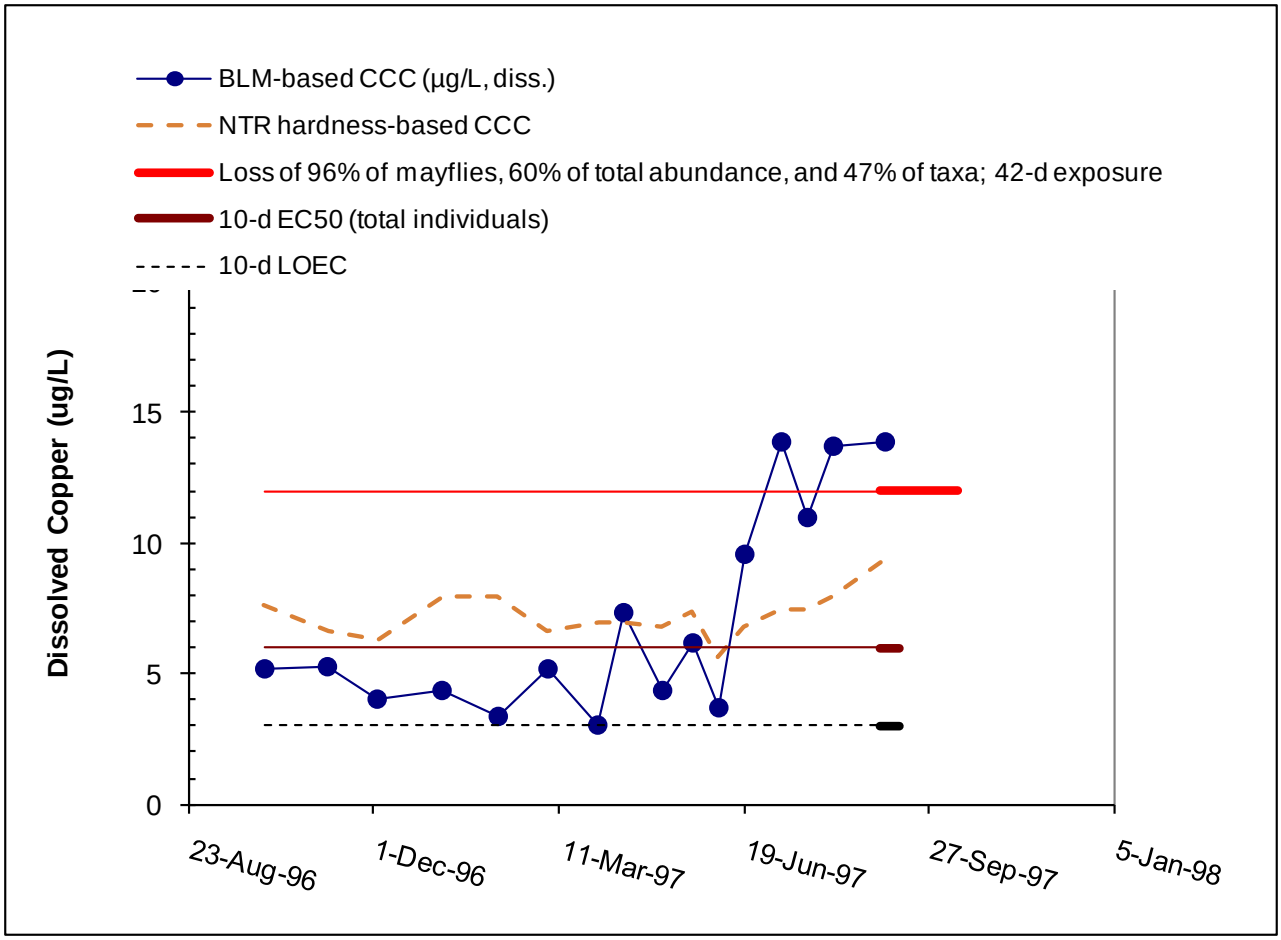


Figure 23. Macroinvertebrate community effects concentrations and the chronic BLM-based copper criteria: New River at Glen Lyn, VA. Effects concentrations from Clements *et al.* (1988, 1989); BLM inputs estimated from USGS station 0317500 (New River at Glyn Lyn, Virginia). Hardness was 48-75 mg/L during the tests and DOC estimated in the ranges of 1.1 to 2.4 mg/L. Horizontal lines indicate different effects concentrations from the tests; the thick "handles" on the right ends of the lines correspond to the time of year that the tests were actually carried out.

The best interpretation of the analyses summarized in Figure 23 may be that macroinvertebrate communities are very sensitive to copper, and that community richness may decline at concentrations lower than the BLM-based CCC. At best, there is a narrow range between allowable chronic criteria concentrations and pronounced adverse effects. This conclusion seems consistent with a recent field study relating mixtures of copper, cadmium, and zinc in Colorado streams to benthic community alterations using a modification of the BLM (Schmidt *et al*. 2010). Declines in diversity and abundance of the Cu-sensitive Heptageniid criteria co-occurred with Cu concentrations less than the BLM-based criteria.

The final experimental ecosystem study considered here used pond microcosms that were dosed with copper ranging from 4 to 420 µg/L (Hedtke 1984). In contrast to the previous ecosystem studies, these microcosms were much smaller, which might make them less realistic

but allowed a more experimental control and replication of treatments. Most required BLM inputs were measured and reported in the article; we estimated pH and K from the water recipe.

Natural pond sediments were collected and allowed to develop in the microcosms for 30 days before the microcosms were dosed with copper for 32 weeks. A variety of ecosystem functional and structural endpoints were measured. The most sensitive results were the loss of most snails and most cladocerans in the 8.8 µg/L LOEC treatment. Additionally, at the 8.8 µg/L LOEC at 30 weeks, gross primary production, DOC production, and the filamentous green alga *Vaucheria* were significantly impacted. More severe effects developed at higher copper treatments.

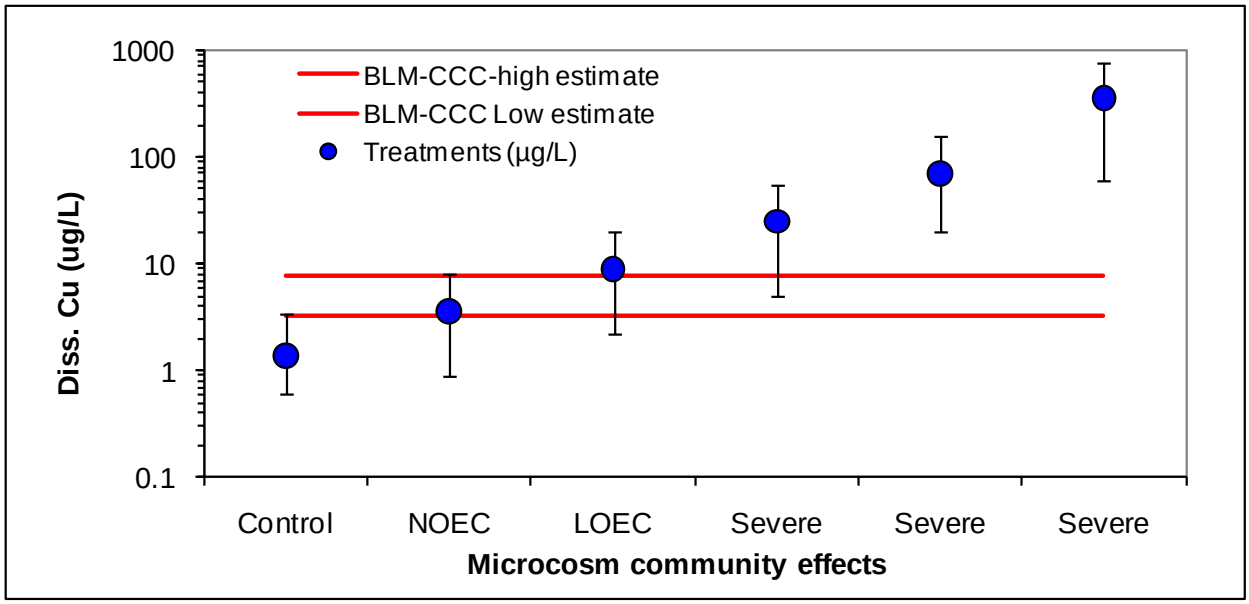


Figure 24. Effects of copper in pond microcosm tests compared with BLM CCC. The values overlapped the no- and low observed effects concentrations, giving equivocal support for the protectiveness of the CCC. Original data from Hedtke (1984).

The comparisons between the no- and lowest-effect concentrations and the BLM-CCC overlapped but leaned toward being favorable to the BLM CCC. The CCC was determined over the DOC range of 0.7 to 1.8 mg/L in the microcosm inflows, which is reflected in the BLM-CCC high and low estimates in Figure 24. The higher estimate of the BLM-CCC approached the LOEC although the lower estimate was below the LOEC and happened to match the NOEC. Presuming that adverse effects likely developed at times when copper was most bioavailable, i.e., when DOC was lowest, then the lower BLM estimate would be given more emphasis. However, as with the Convict Creek and New River experiments, these results emphasize the fine line between probable protectiveness of the BLM-CCC and water conditions in which copper causes much more severe effects to aquatic insects and other benthic macroinvertebrates. Still, the BLM-based 2007 chronic criterion is clearly more appropriate and protective than the Idaho/NTR hardness-based chronic criterion, which was 19 µg/L for the test conditions. In the microcosm treatment that was the closest match to the Idaho/NTR chronic criterion (25 µg/L) all measured ecosystem components except large oligochaetes were significantly impacted.

Conclusions and Recommendations

Overall, NMFS' analyses of the performance of the 2007 BLM-based copper criteria tended to be favorable. With many independent data sets that tested a diverse assortment of aquatic organisms and endpoints across a wide variety of natural and laboratory waters, the 2007 copper BLM toxicity predictions were invariably at least correlated with empirical toxicity observations, which is considerably better than can be said for the Idaho/NTR hardness-based copper criteria. The analyses were most equivocal for the field experiments with aquatic insect and other benthic macroinvertebrates, yet even for these analyses the 2007 BLM-based criteria performance was clearly superior to that of the hardness-based criteria. Because listed juvenile steelhead and salmon are feeding generalists, so long as the overall benthic community remains diverse and abundant, steelhead or salmon populations could likely withstand minor losses of benthic diversity, which was evaluated under the heading "Salmonid Prey Items" in section 2.4.1.12 of the main body of this Opinion. Thus these adverse effects would not rise to the level of jeopardy or adverse modification of critical habitats.

Implementation Considerations

Our mostly favorable evaluation of the 2007 BLM-based criteria leads to the following logical problem: When compared to the old hardness-based criteria, the data requirements of the BLM-based criteria are novel and extensive. Could the 2007 BLM-based criteria be reasonably implemented as an alternative to the Idaho/NTR hardness-based criteria? Can the BLM-based criteria be safely estimated for different water body types even if measurements of all the BLM inputs are not available?

Some efforts have been made to develop regional estimates of BLM input parameters that could be used when measured data are unavailable. For instance, Carleton (2008) describes a proof-of-concept approach where "*one possible way to deal with such missing information is to develop conservative (realistic but protective) default values for these various model inputs… Given that ambient surface water chemistry reflects, among other things, the influences of local soil types and land uses, it may make sense that any such defaults be developed on some kind of regional or local basis*." The EPA (2012) gives further detail of such an approach, and gives potential interim values that could be used on an ecoregional geographic basis. Within the range of anadromous fishes in Idaho, waters and their drainage basins can be grouped according to their expected water chemistry characteristics. We compiled datasets of BLM input parameters for representative waters and examined for seasonal patterns of "critical conditions" which are the annual worst case conditions for that water body (i.e., when copper would be most toxic). Because of the paucity of streams with sufficient high quality chemistry data, some of the waters we used are located outside the range of anadromous fish, but we judged them likely to have characteristics similar to waters occupied by salmonids. Most data were obtained from the USGS National Water Information System database,[12] and were limited to data collected from 1994 or later. The 1994 cutoff was selected because older DOC data were consistently higher than more recent data from the same sites, suggesting there may systematic sample contamination from bottles, filters, or other sources (e.g., Yoro *et al.* 1999).

The first stream we considered was Panther Creek, one of the major tributaries to the Salmon River, Idaho. Since the early 1990s because of copper contamination from mining

[12] http://nwis.waterdata.usgs.gov

activities that led to the loss of Chinook salmon and steelhead populations, Panther Creek has been the focus of many studies, litigation, and restoration efforts (e.g., Mebane 1994, 2002; EcoMetrix 2007; EPA 2008). Water chemistry was measured in detail in Panther Creek during 1993-1994; more recent data are unfortunately inadequate for the BLM.

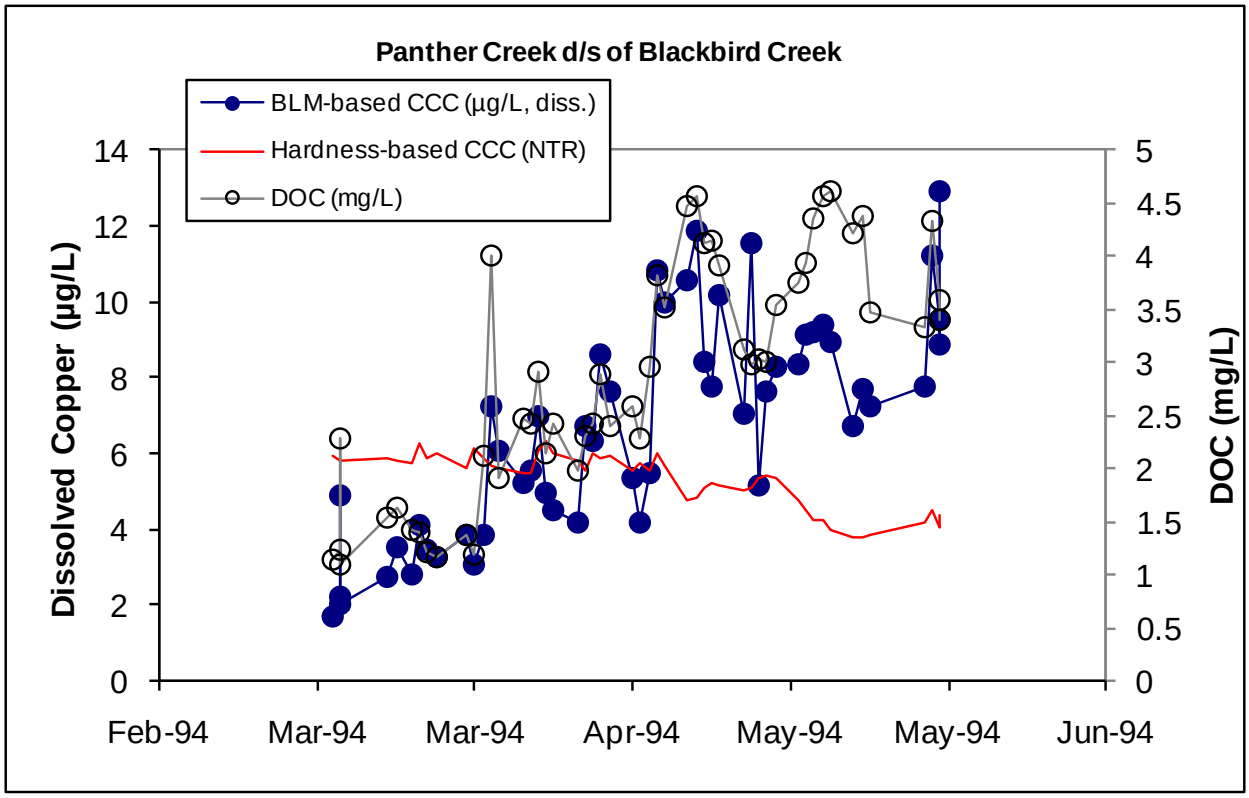


Figure 25. The BLM-CCC in relation to DOC and the hardness-based CCC during the spring snowmelt and runoff in Panther Creek, Idaho. When sampling began in late winter baseflow conditions before the snowmelt began, DOC and the BLM-CCC were at their minimums and steadily rose as the runoff progressed. The hardness-based CCC shifted in the opposite direction. Thus the BLM- and hardness-based criteria give opposite indications of when copper would be most bioavailable and at risk. Original data from (Maest *et al.* 1995).

The BLM-criteria were clearly driven by the DOC concentration of the water, since the two data series shift in nearly perfect unison (Figure 25). In contrast, hardness had little influence on the BLM criteria, with the hardness-based criteria dropping as the BLM-criteria increased. Thus the hardness-based criteria are telling us that the critical conditions for copper toxicity are at the end of the sampling period in late May, and that the lowest risk occurs at base flows. The BLM-criteria tell us just the opposite. Based on our previous review, it appears that the hardness-based criteria are giving misleading information and are completely wrong in their indications of relative risk to aquatic life from elevated copper concentrations.

Very similar patterns of the BLM- and the hardness-based CCC were apparent across datasets that we considered representative of drainage basins occurring within the range of listed salmon and steelhead in Idaho. The Teton River, Idaho (Figure 26, top) has geomorphic and water chemistry similarities to the Lemhi and Pahsimeroi Rivers, tributaries to the Salmon River, Idaho. In the Teton River, DOC clearly drove the BLM-criteria values, since the two data series

track so closely together. (This tendency occurred in all of the data sets analyzed here as well as many other USGS river datasets reviewed but not presented, however the plots get cluttered and so DOC is omitted from some.) Opposite from the misleading information provided by the hardness-criterion, the critical conditions during which the BLM-criteria were near their annual minima again occurred during base flow conditions in fall or winter, and lowest risks for any given copper concentration occurred during spring snowmelt (Figure 26, top).

The Clark Fork River, Montana, has water chemistries that are probably roughly similar to those of the middle Salmon River between the confluences of the Lemhi River in the town of Salmon at river mile 260 and the Middle Fork near river mile 200. The Clark Fork has been the subject of much research and ecological risk assessment regarding copper risks to aquatic life, and so it is surprising that only one water year of BLM-quality water chemistry data was available (Figure 26, middle). Here again, the plots of BLM- and hardness-based criteria look like mirrored opposites, with the BLM indicating that copper would be most toxic during base flow in winter and least toxic in April to June (Figure 26, middle).

The Snake River, as it flows out of Yellowstone National Park in Wyoming near the Idaho border, has moderately low hardness and low DOC. These conditions probably make this location on the Snake River a reasonable surrogate for the upper Salmon River, upstream of the Pahsimeroi confluence near Salmon River mile 305 (Minshall *et al*. 1992). The Snake River at this location has one of the richest water quality datasets in the region, with comprehensive monthly sampling from 1993 until the USGS discontinued monitoring the site in 2004. Through the seasons, the BLM- and hardness-based criteria vary in nearly regular cycles that look almost like two sine waves that are out of phase. Again, the peaks in the BLM-criteria when copper is at its lowest risk correspond to the dips in the hardness-criteria, with their misleading risk indications (Figure 26, bottom).

The next panel of plots shows a very different situation of risk patterns for very soft waters and low organic content or low pH (Figure 25). The North Fork of the Coeur d'Alene River (NFCDA) is a comparatively well monitored stream in northern Idaho with very dilute water chemistries. The NFCDA has some similar characteristics to the upper Clearwater River basin streams and probably some of the Salmon River basin streams that are located in granitic geology with very dilute waters. In the NFCDA, the BLM-criteria are consistently much lower than the hardness-based criteria (Figure 27, top). The hardness-based criteria do not vary in these low hardness waters because the criteria-equations require that when the actual water hardness is less than 25 mg/L, the criteria shall be calculated using a hardness of 25 mg/L rather than the actual water hardness (EPA 1992).

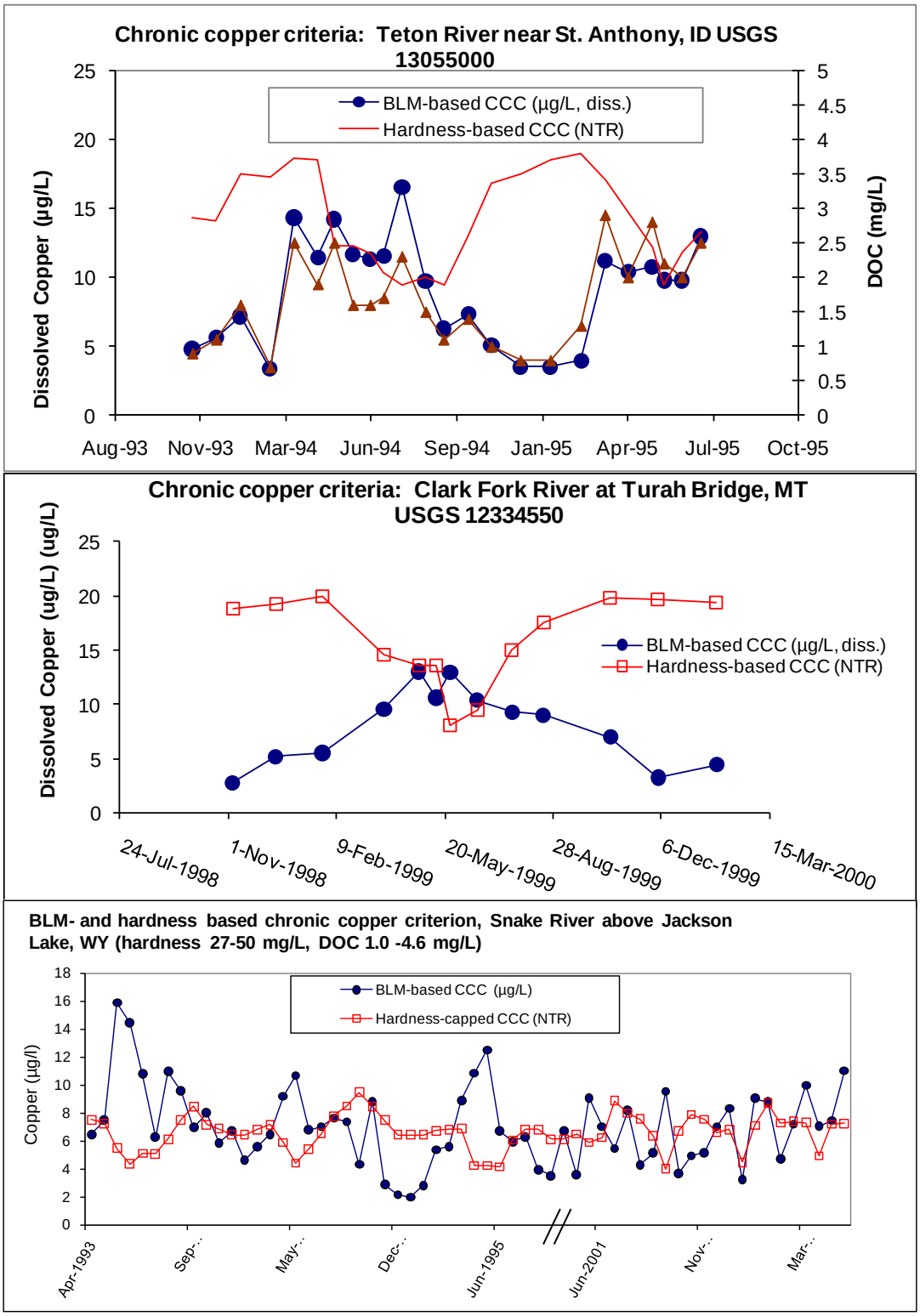


Figure 26. Seasonal patterns in BLM-CCC and hardness-based CCC in streams with a strong spring snowmelt influence, and moderately-hard to softwater chemistry: the Teton River, ID; the Clark Fork River, MT; and the Snake River as it leaves Yellowstone National Park, WY. A consistent asynchronous pattern is apparent where the BLM and hardness-criteria shift in opposition to one another.

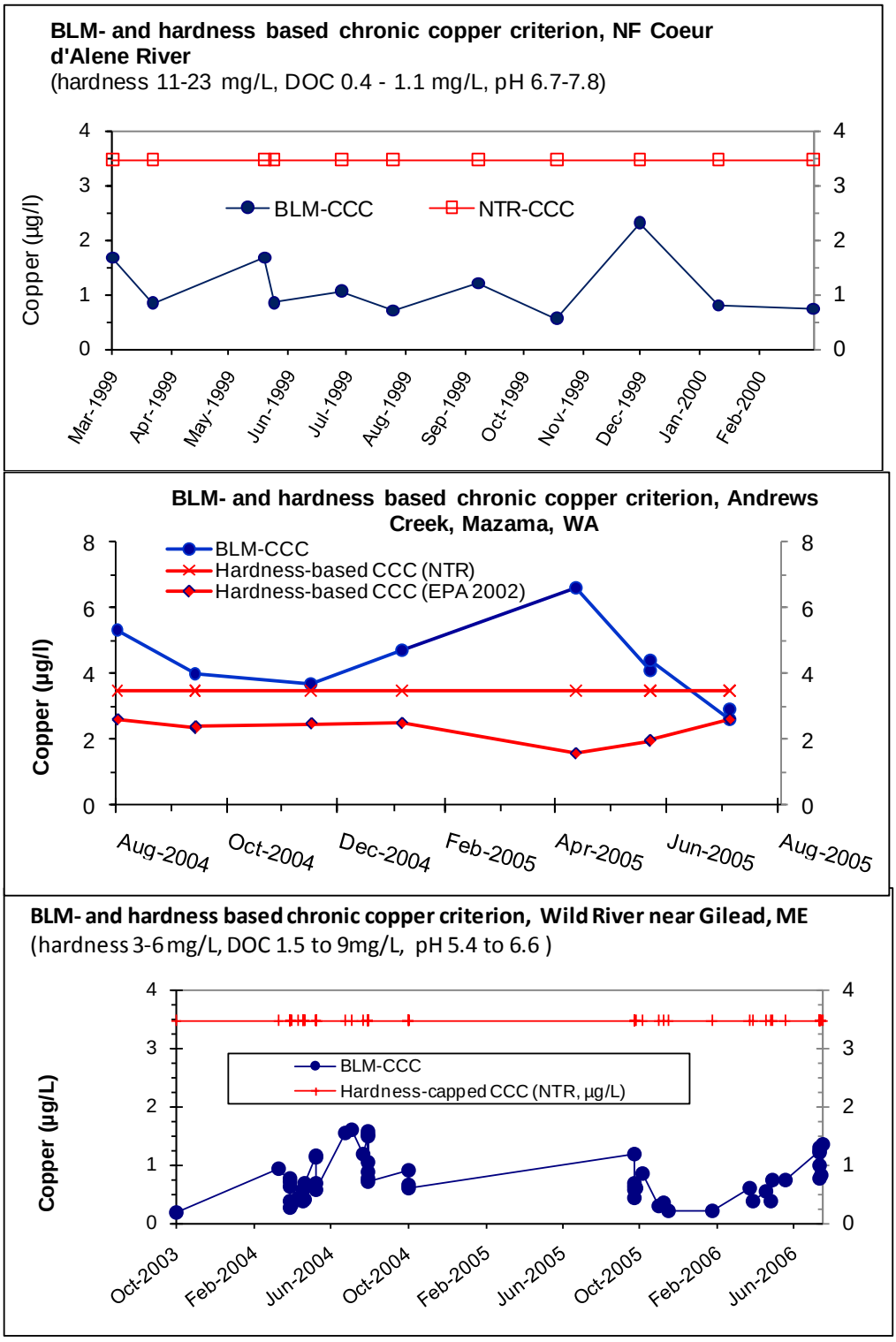


Figure 27. Seasonal patterns in BLM- and hardness-based CCC values for three streams distinguished by snowmelt springflows, softwater, variable DOC, and in the bottom, low pH. Because in Idaho hardness-based criteria equations are "capped" at 25 mg/L, and the hardnesses of these streams never exceeded 25 mg/L, the hardness-based criteria are a constant 3.6 µg/L. In the NF Coeur d'Alene River,

Idaho, DOC is low year round resulting in low CCC values year round. In Andrews Creek, the "uncapped" CCC again mirrors the BLM-CCC with opposite trends.

Andrews Creek is located on the eastside of the North Cascade Mountains near Mazama, Washington. Similar to Convict Creek in the Sierra Nevadas and to streams in the Idaho Batholith geology that underlies a large portion of the Salmon River drainage in Idaho, the Andrews Creek watershed is granitic with thin soils. Andrews Creek has very soft water and low organic matter, although not as extremely low as some waters in northern Idaho such as the NFCDA, Lochsa, or Selway River drainages. If the hardness-floor were ignored, the BLM- and hardness-based criteria again would show the now familiar opposite patterns, with the BLM-based low values occurring mostly in fall and winter (Figure 27, middle).

The Wild River near Gilead, Maine, is included to illustrate conditions that have produced some of the lowest BLM-based criteria time series values for natural waters we located (Figure 27, bottom). While not physically close to Idaho, the Wild River drainage has other geographic similarities to the action area. The Wild River drainage is underlain by erosion resistant bedrock with poorly buffered thin soils which results in extremely soft water. The pH of the Wild River is lower than that in any of the other "BLM-quality" datasets compiled for this review and is probably lower than is typical in softwater areas of Idaho. Still, in streams draining basins with granitic geology from Idaho Batholith or Precambrian metamorphic rocks, pH values are commonly in the low 6s and sometimes less than 6. While these BLM-based CCC values are very low (0.2 µg/L to <2 µg/L), 96-hour fathead minnow LC50s as low as 2 µg/L have been obtained in similarly mildly acidic, low calcium waters (Figure 4), and presumably had effects been obtained from longer exposures, sublethal endpoints, or more sensitive species would have been lower.

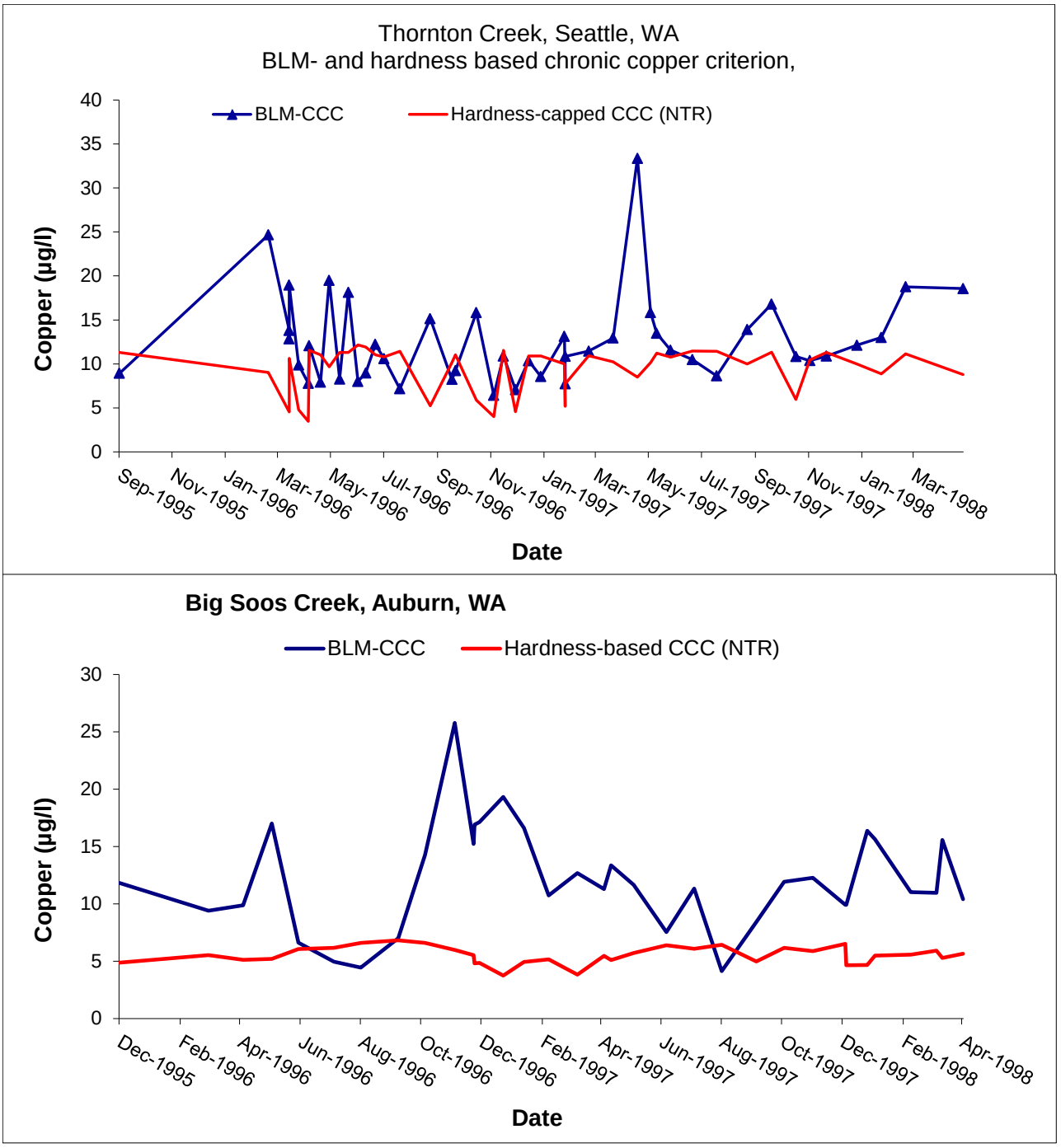


Figure 28. Seasonal patterns in BLM- and hardness-based CCC values for two streams distinguished by high flows from winter rainfall instead of snowmelt. Thornton Creek is an urban stream with moderately hard water and higher DOC than most streams examined; Big Soos Creek is a softwater stream in a mostly rural area with periodic high DOC during the winter rainy season.

DOC concentrations occurring across several streams sampled systematically from April through September 2007, show considerable variability in the timing of peak DOC, but except for the South Fork Coeur d'Alene River (SFCDA), for each stream the lowest DOC

concentrations were measured in the late September samples (Figure 28). For the SFCDA, DOC was low and nearly uniform throughout the period of record. This particular sampling effort did not collect the major ion data needed to calculate BLM time series, but the DOC patterns give further support that critical conditions for vulnerability to copper toxicity are predictable and probably will occur in fall during base flow conditions.

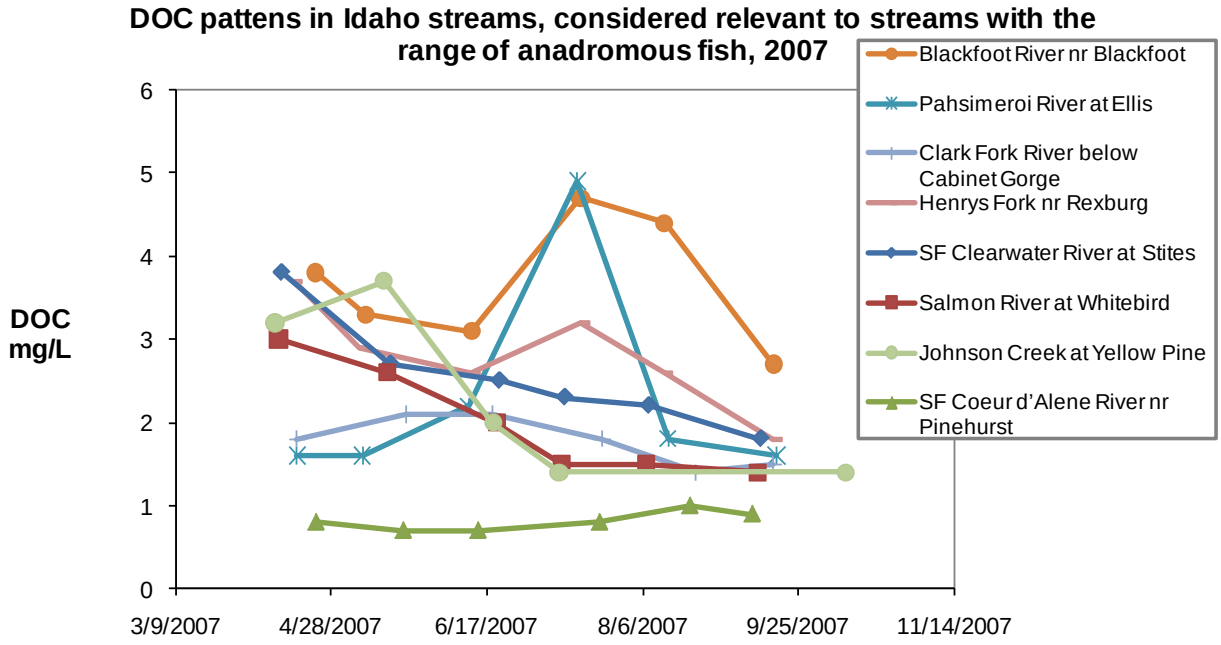


Figure 29. Seasonal DOC patterns in Idaho streams considered relevant to listed salmon and steelhead waters, including four within their critical habitats. In all cases by late September, DOC was at or near its lowest measured value.

Returning to the question posed at the beginning of this section on implementation considerations, are the regional and seasonal water chemistry patterns sufficiently predictable that conservative (realistic but protective) default BLM-criterion table values can be defined? For the annual critical conditions when copper would be at its most toxic, the answer appears to be "Yes." The most critical conditions almost invariably occur in the fall, and over the range of waters with listed anadromous fish in Idaho, data relevant to these conditions were either directly available or could be estimated from watersheds with similar characteristics (Table 3). Conservative high estimates of annual maxima could also be made. For example, if for the upper Salmon River, the Snake River above Jackson Lake is used as a surrogate, the lowest measured dip in the BLM-based criteria plots would be about 2 µg/L and the lowest annual peak would be about 6 µg/L (Figure 26).

The handling of discrete pH data is an important detail note in the BLM calculations to estimate late-summer copper benchmark concentrations in Table 3. None of the pH data in the USGS data for the streams in Table 3 were collected in the early morning hours near dawn when pH would be expected to be at the daily minimum. Some data were collected in the late afternoon, which is when pH would be expected to be near its maximum. In the copper BLM,

pH is an important variable, and copper toxicity is predicted to markedly decrease as pH increases. Daily pH variations in excess of 0.5 units over the course of a day are not uncommon in streams, even streams that are fairly oligotrophic; and in streams with high primary productivity, pH can swing by at least 2 units (e.g., Nimick *et al*. 2011; Balistrieri *et al*. 2012). Accumulations of metals on gills can be rapid, with sufficient accumulation occurring over time periods of 45 minutes to 3 hours to predict later toxicity (Balistrieri and Mebane 2014). Until the importance of time varying pH for metals speciation, accumulation, and toxicity are better investigated, it seems prudent to use daily minimum pH values in BLM calculations. Thus, in the BLM calculations to estimate late-summer copper benchmark concentrations in Table 3, for those sites with pH >7.5, pH was lowered by 0.6 units to adjust for high bias from mid-day water samples.

Table 3. Ranges of chronic copper criterion concentrations estimated for critical late summer/fall baseflow conditions in subbasins within the range of anadromous salmonids in the Snake River basin, Idaho.

Subbasin	Common subbasin geologic characteristics	Critical late-summer Cu benchmark concentration (µg/L)	Based upon EPA's 2007 Cu chronic criterion (CCC) using data collected or estimated using:
Selway, Lochsa, MF Clearwater R	Granitic or intrusive rocks from Idaho Batholith or Precambrian metamorphic rocks	0.6	St Joe River at Red Ives, 9/14/2007; SF Coeur d'Alene R at Pinehurst, 9/10/2007; NFCDA Fig 25
SF Clearwater River	Idaho Batholith	1	SF Clearwater at Stites
MF and SF Salmon and tributaries	Idaho Batholith	1	Extrapolated using low conductivity measured in undisturbed streams in the Salmon R basin (Ott and Maret 2003), ~30 µs/cm, pH 6.9, using DOC of 1 mg/L and then estimating major ions with regression equations from streams in Coeur d'Alene R with similarly low conductivity
Upper Salmon R	Idaho Batholith and Challis volcanics	3	Snake River (Fig. 24); Johnson Creek at Yellow Pine, 10/10/2007
Upper Salmon R tributaries	Challis volcanics	3	Assumed similar to Panther Creek
Panther Creek	Challis volcanics and Idaho Batholith	3	Minimum BLM=CCC calculated for low-flow, low DOC conditions from a 1994 dataset (Maest *et al*. 1995)
Lemhi and Pahsimeroi Rivers	Tertiary sediments from ancient lake bottoms	6	Pahsimeroi at Ellis, 9/18/2007
Lower Salmon (downstream of SF Salmon)	Diverse	3	Salmon River at White Bird, 9/27/2007
Snake River	Diverse	6	Minimum BLM calculated for Snake River at mouth (Burbank, WA)

Data collected in 2007 were for a single data collection. It seemed reasonable to assume that late summer baseflow conditions were probably close the critical condition (i.e., annual minimum) CCC calculated using the BLM-based Cu criteria. However, because the BLM-based criteria is sensitive to pH and these mid-day collected samples probably represented close to the daily high for pH, pH was lowered by 0.6 units for those sites with high pH (>7.5) because pH can vary up to 2 units per day (Balistrieri *et al*. 2012), although in oligotrophic, coldwater streams in summer, pH swings on the order of 0.6 units over the day seemed more likely, with maximum pH occurring near midday. SpC = specific conductivity

This approach would also be consistent with the concept that greater conservatism in environmental management is appropriate when information is uncertain and this conservatism may be relaxed when uncertainties are reduced through better information. Assume for example that a facility manager was concerned that this approach of using conservative estimates of BLM-based criteria for regulating copper in effluents during base flow and that provided no relief from unnecessarily conservative hardness-based copper criteria during spring runoff when hardnesses were low, could result in costly discharge restrictions that might be of little environmental benefit. In such a case, since the BLM parameters probably only add a modest increase in sampling cost, compared to the labor costs of getting samples in the first place, it would be cost-effective for the facility manager to arrange to include the BLM parameters in their ambient monitoring program. The major dischargers operating within the range of anadromous fish in Idaho and that have metals limits are all mining facilities. These operations tend to collect ambient water quality data from their receiving waters four times a year, with one

sampling event during low, base flows and three during the more variable April to June conditions. From the patterns observed from the 16 datasets shown here (Figures 23-27), such a seasonal sampling would be sufficient in at least streams with snowmelt dominated high flows, and over time could probably be backed off to one spring and one base flow sampling effort. A compromise seasonal table-value approach might be useful on a watershed or river reach approach in lieu of ongoing monitoring if risks of exceedences were low (Figure 30).

A more scientifically defensible and efficient approach would be to develop surrogate measures to predict the major ions and DOC in natural waters. In natural waters the inorganic parameters used in the BLM tend to be correlated with each other and with conductivity and water hardness. Similarly, DOC tends to be correlated with water color and with specific absorption (Schwartz *et al.* 2004; Dittman *et al.* 2009; Gheorghiu *et al.* 2010). It should be feasible to develop surrogate models to estimate BLM parameters with sufficient accuracy across diverse waters that would simply require a conductivity meter and a field spectrophotometer. These could be deployed in-situ and set to transmit in real-time, which offers promise as low-cost and data rich surrogate measures for DOC.

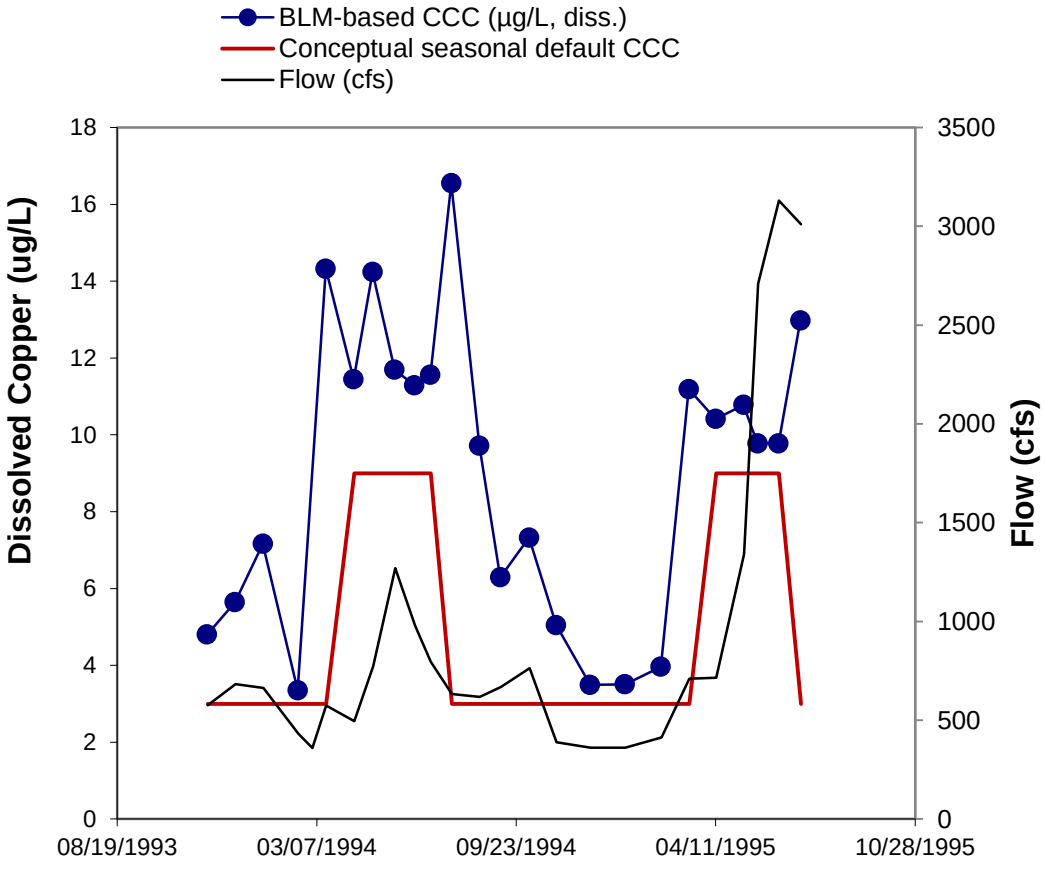


Figure 30. Conceptual example of a simplified, default table-value approach to defining BLM-based copper criteria in lieu of routine monitoring of BLM data requirements. The sample data used are from the Teton River, ID (Figure 26).

From a practical point of view for planning sampling for BLM inputs, targeting critical conditions that persist for several months during the low flow, dry season is considerably easier than trying to plan for sampling near the peak of runoff when hardnesses are at their annual minima, conditions that may develop quickly if an unexpected spring thaw occurs and may only last for a few days, and when access to collect samples may be hampered by rotten snow, high water, and mud.

In summary, NMFS' review has shown that Idaho's hardness-based copper criteria would likely result in instream copper concentrations above levels protective of listed salmonids and their critical habitats. Calcium, the main determinant of water hardness, is one factor affecting the toxicity of copper, but in natural waters it is generally less important than DOC or pH. Overall, EPA's 2007 version of the copper BLM did a credible job of predicting acute and chronic toxicity to taxonomically diverse organisms over a wide variety of waters, and had some predictive power with chemosensory functions in fish. Whether the BLM-based criteria would be fully protective of benthic macroinvertebrate communities is equivocal, but would be more protective than the alternative hardness-based criteria. While not optimal, minor losses of benthic diversity could likely be withstood by listed steelhead or salmon populations because juvenile steelhead and salmon are feeding generalists. Thus these likely adverse effects would not be expected to rise to the level of jeopardy or adverse modification of critical habitats.

The performances of the 2007 BLM based criteria were not ideal, and refinements would be worthwhile to pursue. For example, the BLM performance in very soft waters could be re-evaluated in the light of developments subsequent to the 2007 version (e.g., Ryan *et al.* 2009; Paquin *et al.* 2011). Regardless of this prediction bias, in practice the BLM-based criteria still produced quite low values in natural soft waters relative to toxicity values (Figure 5, Figure 25). So while our analyses suggest areas where the 2007 version copper BLM could be refined (e.g., treatment of DOC, competitive conditional stability constants), its mostly robust performance with a diverse array of organisms with sublethal and lethal endpoints in diverse waters validate earlier testing of the BLM performance (e.g., Santore *et al.* 2001; EPA 2003b). As is, the 2007 criteria represent a huge improvement over the NTR copper criteria and generally represent a major advance in the science of water quality criteria. Its application appears to be protective of listed salmon, steelhead, and their ecosystems.

References for Appendix C

Ankley GT, and Villeneuve DL. 2006. The fathead minnow in aquatic toxicology: Past, present and future. *Aquat Toxicol* **78**: 91-102.

ASTM. 1997. Standard guide for conducting acute toxicity tests on test materials with fishes, macroinvertebrates, and amphibians. Method E729-96. *In* Annual Book of ASTM Standards. American Society for Testing and Materials, West Conshohocken, PA. p. 22.

Baldwin DH, Tatara CP, and Scholz NL. 2010. Copper-induced olfactory toxicity in salmon and steelhead: Extrapolation across species and rearing environments. *Aquat Toxicol* **101**: 295-297.

Balistrieri LS, and Mebane CA. 2014. Predicting the toxicity of metal mixtures. *Sci Total Environ* **466-467**: 788–799.

Balistrieri LS, Nimick DA, and Mebane CA. 2012. Assessing time-integrated dissolved concentrations and predicting toxicity of metals during diel cycling in streams. *Sci Total Environ* **425**: 155–168.

Besser JM, Allert AL, Hardesty DK, Ingersoll CG, May JT, Wang N, and Lieb KJ. 2001. Evaluation of metal toxicity in streams of the upper Animas River watershed, Colorado. U.S. Geological Survey, Biological Science Report 2001–001.

Besser JM, Wang N, Dwyer FJ, Mayer FL, and Ingersoll CG. 2005. Assessing contaminant sensitivity of endangered and threatened fishes: 2. Chronic toxicity of copper and pentachlorophenol to two endangered species and two surrogate species. *Arch Environ Con Tox* **48**: 155-165.

Boeckman CJ, and Bidwell JR. 2006. The effects of temperature, suspended solids, and organic carbon on copper toxicity to two aquatic invertebrates. *Water Air Soil Poll* **171**: 185-202.

Borgmann U, Nowierski M, and Dixon DG. 2005. Effect of major ions on the toxicity of copper to *Hyalella azteca* and implications for the biotic ligand model. *Aquat Toxicol* **73**: 268-287.

Brooks PD, O'Reilly CM, Diamond SA, Campbell DH, Knapp R, Bradford D, Corn PS, Hossack B, and Tonnessen K. 2005. Spatial and temporal variability in the amount and source of dissolved organic carbon: implications for ultraviolet exposure in amphibian habitats. *Ecosystems* **8**: 478 - 487.

Carleton JN. 2008. Spatial trends in water chemistry and the biotic ligand model. *In* American Water Resources Association (AWRA) 2008 Spring Specialty Conference. *Edited by* D.R. Maidment, San Mateo, California. p. 6.

Chakoumakos C, Russo RC, and Thurston RV. 1979. Toxicity of copper to cutthroat trout (*Salmo clarki*) under different conditions of alkalinity, pH, and hardness. *Environ Sci Technol* **13**: 213-219.

Chapman GA. 1982. [Chinook salmon early life stage tests with cadmium, copper, and zinc]. U.S. Environmental Protection Agency, Environmental Research Laboratory, Letter of December 6, 1982 to Charles Stephan, US EPA Environmental Research Laboratory, Duluth, Corvallis, Oregon

Chapman GA, Ota S, and Recht F. 1980. Effects of water hardness on the toxicity of metals to *Daphnia magna*. U.S. EPA, Office of Research and Development, Corvallis, Oreg.

Clements WH, Carlisle DM, Lazorchak JM, and Johnson PC. 2000. Heavy metals structure benthic communities in Colorado mountain streams. *Ecol Appl* **10**: 626-638.

Clements WH, Cherry DS, and Cairns J, Jr. 1988. The impact of heavy metals on macroinvertebrate communities: a comparison of observational and experimental results. *Can J Fish Aquat Sci* **45**: 2017-2025.

Clements WH, Farris JL, Cherry DS, and Cairns J, Jr. 1989. The influence of water quality on macroinvertebrate community responses to copper in outdoor experimental streams. *Aquat Toxicol* **14**: 249-262.

Collyard SA. 2002. Bioavailability of copper to the amphipod *Hyalella azteca*. MSc.thesis, Department of Zoology and Physiology, University of Wyoming, Laramie, Wyo.

Cotner J, Biddanda B, Makino W, and Stets E. 2004. Organic carbon biogeochemistry of Lake Superior. *Aquat Ecosyst Health Manage* **7**: 451 - 464.

De Schamphelaere KAC, and Janssen CR. 2004a. Development and field validation of a biotic ligand model predicting chronic copper toxicity to *Daphnia magna*. *Environ Toxicol Chem* **23**: 1365–1375.

De Schamphelaere KAC, and Janssen CR. 2004b. Effects of dissolved organic carbon concentration and source, pH, and water hardness on chronic toxicity of copper to *Daphnia magna*. *Environ Toxicol Chem* **23**: 1115–1122.

De Schamphelaere KAC, Vasconcelos FM, Tack FMG, Allen HE, and Janssen CR. 2004. Effect of dissolved organic matter source on acute copper toxicity to *Daphnia magna*. *Environ Toxicol Chem* **23**: 1248–1255.

Di Toro DM, Allen HE, Bergman HL, Meyer JS, Paquin PR, and Santore RC. 2001. Biotic ligand model of the acute toxicity of metals. 1. Technical basis. *Environ Toxicol Chem* **20**: 2383-2396.

Dittman JA, Shanley JB, Driscoll CT, Aiken GR, Chalmers AT, and Towse JE. 2009. Ultraviolet absorbance as a proxy for total dissolved mercury in streams. *Environ Pollut* **157**: 1953-1956

Dixon DG, and Sprague JB. 1981. Acclimation to copper by rainbow trout (*Salmo gairdneri*) - a modifying factor in toxicity. *Can J Fish Aquat Sci* **38**: 880-888.

EcoMetrix. 2007. Biomonitoring study, Panther Creek watershed, September 2006. Prepared by EcoMetrix Incorporated, Mississauga, Ontario, for the Blackbird Mine Site Group, Salmon, Idaho.

ENSR. 1996. Development of site-specific water quality criteria for copper in the upper Clark Fork River: Phase III WER Program testing results. ENSR Consulting and Engineering, 0480-277, Fort Collins, Colo.,.

EPA (U.S. Environmental Protection Agency). 1985. Ambient water quality criteria for copper - 1984. U.S. Environmental Protection Agency, EPA 440/5-84-031, Duluth, MN.

EPA. 1992. National Toxics Rule. *Federal Register* **57**: 60848-60910.

EPA. 1996. 1995 updates: water quality criteria documents for the protection of aquatic life in ambient water. U.S. Environmental Protection Agency, EPA 820-B-96-001, Washington, D.C.

EPA. 2000. Biological Assessment of the Idaho Water Quality Standards for Numeric Water Quality Criteria for Toxic Pollutants (final 8-4-2000). U.S. Environmental Protection Agency, Seattle, WA.

EPA. 2002. National recommended water quality criteria: 2002. U.S. Environmental Protection Agency, EPA-822-R-02-047, Washington, DC.

EPA. 2003a. 2003 update of ambient water quality criteria for copper. U.S. Environmental Protection Agency, Office of Water, Office of Science and Technology, EPA 822-R-03-026, Washington, DC.

EPA. 2003b. The Biotic Ligand Model: Technical support document for its application to the evaluation of water quality criteria for copper. EPA 822-R-03-027.

EPA. 2003c. Record of Decision: Blackbird Mine Superfund Site, Lemhi County, Idaho. U.S. Environmental Protection Agency, Seattle, WA.

EPA. 2007a. Aquatic life ambient freshwater quality criteria - copper, 2007 revision. U.S. Environmental Protection Agency, EPA-822-R-07-001 (March 2, 2007), Washington, DC.

EPA. 2007b. Framework for Metals Risk Assessment. U.S. Environmental Protection Agency, EPA 120/R-07/001.

EPA. 2008. First Five-Year Review Report, Blackbird Mine Site, August 2008. U.S. Environmental Protection Agency, Seattle, WA.

EPA. 2010. Training materials on Copper BLM: Implementation.

EPA. 2012. Development of Tools to Estimate Water Quality Parameters for the Biotic Ligand Model. U.S. Environmental Protection Agency, Office of Water, EPA 820/R1/2008 (draft), Washington, D.C.

Erickson RJ. 2008. Toxicity Response Analysis Program, version 1.2. U.S. Environmental Protection Agency, National Health and Environmental Research Laboratory, Mid-Continent Ecological Division, Duluth, Minnesota.

Erickson RJ, Benoit DA, and Mattson VR. 1987. A prototype toxicity factors model for site specific copper water quality criteria (Revised September 5, 1996). U.S. Environmental Protection Agency, Environmental Research Laboratory, Duluth, Minnesota.

Erickson RJ, Benoit DA, Mattson VR, Nelson HP, and Leonard EN. 1996. The effects of water chemistry on the toxicity of copper to fathead minnows. *Environ Toxicol Chem* **15**: 181-193.

Geckler JR, Horning WB, Nieheisel TM, Pickering QH, Robinson EL, and Stephan CE. 1976. Validity of laboratory tests for predicting copper toxicity in streams. U.S. EPA Ecological Research Service, EPA 600/3-76-116, Cincinnati, OH.

Gheorghiu C, Smith DS, Al-Reasi HA, McGeer JC, and Wilkie MP. 2010. Influence of natural organic matter (NOM) quality on Cu–gill binding in the rainbow trout (*Oncorhynchus mykiss*) *Aquat Toxicol*: doi:10.1016/j.aquatox.2010.1001.1003.

GLEC. 2006. Development of a copper criteria adjustment procedure for Michigan's Upper Peninsula waters. Great Lakes Environmental Center, Traverse City, Michigan and Columbia, Ohio, Prepared for the Michigan Department of Environmental Quality.

Green WW, Mirza RS, Wood CM, and Pyle GG. 2010. Copper binding dynamics and olfactory impairment in fathead minnows (*Pimephales promelas*). *Environ Sci Technol* **44**: 1431–1437.

Hansen JA, Lipton J, Welsh PG, Morris J, Cacela D, and Suedkamp MJ. 2002a. Relationship between exposure duration, tissue residues, growth, and mortality in rainbow trout (*Oncorhynchus mykiss*) juveniles sub-chronically exposed to copper. *Aquat Toxicol* **58**: 175-188.

Hansen JA, Marr JCA, Lipton J, and Bergman HL. 1999a. Differences in neurobehavioral responses of chinook salmon *(Oncorhynchus tshawytscha)* and rainbow trout *(Oncorhynchus mykiss)* exposed to copper and cobalt: behavioral avoidance. *Environ Toxicol Chem* **18**: 1972-1978.

Hansen JA, Rose JD, Jenkins RA, Gerow KG, and Bergman HL. 1999b. Chinook salmon (*Oncorhynchus tshawytscha*) and rainbow trout (*Oncorhynchus mykiss*) exposed to copper: neurophysiological and histological effects on the olfactory system. *Environ Toxicol Chem* **18**: 1979-1991.

Hansen JA, Welsh PG, Lipton J, Cacela D, and Dailey AD. 2002b. Relative sensitivity of bull trout *(Salvelinus confluentus)* and rainbow trout *(Oncorhynchus mykiss)* to acute exposures of cadmium and zinc. *Environ Toxicol Chem* **21**: 67–75.

Harmon SM, Specht WL, and Chandler GT. 2003. A comparison of the daphnids *Ceriodaphnia dubia* and *Daphnia ambigua* for their utilization in routine toxicity testing in the southeastern United States. *Arch Environ Con Tox* **45**: 79-85.

Hecht SA, Baldwin DH, Mebane CA, Hawkes T, Gross SJ, and Scholz NL. 2007. An overview of sensory effects on juvenile salmonids exposed to dissolved copper: Applying a benchmark concentration approach to evaluate sublethal neurobehavioral toxicity. National Marine Fisheries Service, NOAA Technical Memorandum NMFS-NWFSC-83, Seattle, WA.

Hedtke SF. 1984. Structure and function of copper-stressed aquatic microcosms. *Aquat Toxicol* **5**: 227-244.

HydroQual. 2007. The biotic ligand model Windows interface, Version 2.2.3: User's guide and reference manual. HydroQual, Inc., Mahwah, New Jersey.

Koivisto S, Ketola M, and Walls M. 1992. Comparison of five cladoceran species in short- and long-term copper exposure. *Hydrobiol* **248**: 125-136.

Kuwabara JS, Leland HV, and Bencala KE. 1984. Copper transport along a Sierra Nevada stream. *J Environ Eng* **110**: 646-655.

Leland HV, and Carter JL. 1984. Effects of copper on species composition of periphyton in a Sierra Nevada, California, stream. *Freshwater Biol* **14**: 281-296.

Leland HV, and Carter JL. 1985. Effects of copper on production of periphyton, nitrogen fixation and processing of leaf litter in a Sierra Nevada, California, stream. *Freshwater Biol* **15**: 155-173.

Leland HV, Fend SV, Dudley TL, and Carter JL. 1989. Effects of copper on species composition of benthic insects in a Sierra Nevada, California, stream. *Freshwater Biol* **21**: 163-179.

Linbo TL, Baldwin DH, McIntyre JK, and Scholz NL. 2009. Effects of water hardness, alkalinity, and dissolved organic carbon on the toxicity of copper to the lateral line of developing fish. *Environ Toxicol Chem* **28**: 1455–1461.

Linbo TL, Stehr CM, Incardona JP, and Scholz NL. 2006. Dissolved copper triggers cell death in the peripheral mechanosenory system of larval fish. *Environ Toxicol Chem* **25**: 597-603.

Lind D, Alto K, and Chatterton S. 1978. Regional copper-nickel study, aquatic toxicology study. Minnesota Environmental Quality Board, St Paul, MN.

MacRae RK, Smith DE, Swoboda-Colberg N, Meyer JS, and Bergman HL. 1999. Copper binding affinity of rainbow trout (*Oncorhynchus mykiss*) and brook trout (*Salvelinus fontinalis*) gills: implications for assessing bioavailable metal. *Environ Toxicol Chem* **18**: 1180–1189.

Maest A, Beltman DJ, Cacela D, Lipton J, Holmes J, LeJeune K, and Podrabsky T. 1995. Spring 1994 surface water injury assessment report: Blackbird Mine site NRDA. Submitted by: RCG/Hagler Bailly, Boulder, CO. Submitted to: State of Idaho and National Oceanic and Atmospheric Administration.

Maier WJ, and Swain WR. 1978. Organic carbon-a nonspecific water quality indicator for Lake Superior. *Water Res* **12**: 523-529.

March FA, Dwyer FJ, Augspurger T, Ingersoll CG, Wang N, and Mebane CA. 2007. An evaluation of freshwater mussel toxicity data in the derivation of water quality guidance and standards for copper. *Environ Toxicol Chem* **26**: 2066–2074.

Marr JCA, Lipton J, Cacela D, Hansen JA, Bergman HL, Meyer JS, and Hogstrand C. 1996. Relationship between copper exposure duration, tissue copper concentration, and rainbow trout growth. *Aquat Toxicol* **36**: 17-30.

McGeer JC, Szebedinszky C, McDonald DG, and Wood CM. 2002. The role of dissolved organic carbon in moderating the bioavailability and toxicity of Cu to rainbow trout during chronic waterborne exposure. *Comp Biochem Physiol C Toxicol Pharmacol* **133**: 147-160.

McIntyre JK, Baldwin DH, Beauchamp DA, and Scholz NL. 2012. Low-level copper exposures increase visibility and vulnerability of juvenile coho salmon to cutthroat trout predators. *Ecol Appl* **22**: 1460–1471.

McIntyre JK, Baldwin DH, Meador JP, and Scholz NL. 2008a. Chemosensory deprivation in juvenile coho salmon exposed to dissolved copper under varying water chemistry conditions. *Environ Sci Technol* **42**: 1352–1358.

McIntyre JK, Baldwin DH, Meador JP, and Scholz NL. 2008b. Chemosensory deprivation in juvenile coho salmon exposed to dissolved copper under varying water chemistry conditions (correction). *Environ Sci Technol* **47**: 6774–6775.

McKim JM, and Benoit DA. 1971. Effects of long-term exposure to copper on survival, growth and reproduction of brook trout (*Salvelinus fontinalis*). *J Fish Res Board Can* **28**: 655-662.

McKim JM, and Benoit DA. 1974. Duration of toxicity tests for establishing "no effect" concentrations for copper with brook trout (*Salvelinus fontinalis*). *J Fish Res Board Can* **31**: 449-452.

Meador JP. 1991. The interaction of pH, dissolved organic carbon, and total copper in the determination of ionic copper and toxicity. *Aquat Toxicol* **19**: 13-32.

Mebane CA. 1994. Preliminary Natural Resource Survey - Blackbird Mine, Lemhi County, Idaho. U.S. National Oceanic and Atmospheric Administration, Hazardous Materials Assessment and Response Division, Seattle, WA.

Mebane CA. 2002. Effects of metals on freshwater macroinvertebrates: a review and case study of the correspondence between a multimetric index, toxicity testing, and copper concentrations in sediment and water. *In* Biological Response Signatures: Indicator Patterns using Aquatic Communities. *Edited by* T.P. Simon. CRC Press, Boca Raton, FL. pp. 281-306. doi: 10.1201/9781420041453.ch16.

Mebane CA. 2006. Cadmium risks to freshwater life: derivation and validation of low-effect criteria values using laboratory and field studies. U.S. Geological Survey Scientific Investigation Report 2006-5245 (2010 rev.).

Meyer JS, and Adams WJ. 2010. Relationship between biotic ligand model-based water quality criteria and avoidance and olfactory responses. *Environ Toxicol Chem* **29**: 2096–2103.

Meyer JS, Boese CJ, and Morris JM. 2007. Use of the biotic ligand model to predict pulse-exposure toxicity of copper to fathead minnows (*Pimephales promelas*). *Aquat Toxicol* **84**: 268-278.

Minshall GW, Peterson RC, Bott TL, Cushing CE, Cummins KW, Vannote RL, and Sedell JR. 1992. Stream ecosystem dynamics of the Salmon River, Idaho: an 8th-order system. *J N Am Benthol Soc* **11**: 111-137.

Mirza RS, and Chivers DP. 2001. Chemical alarm signals enhance survival of brook charr (*Salvelinus fontinalis*) during encounters with predatory chain pickerel (*Esox niger*). *Ethology* **107**: 989-1005.

Mirza RS, and Chivers DP. 2003. Response of juvenile rainbow trout to varying concentrations of chemical alarm cue: response thresholds and survival during encounters with predators. *Can J Zool* **81**: 88-95.

Mount DI. 1968. Chronic toxicity of copper to fathead minnows (*Pimephales promelas*, rafinesque) *Water Res* **2**: 215-223.

Mount DI, and Stephan CE. 1967. A method for detecting cadmium poisoning in fish. *The Journal of Wildlife Management* **31**: 168-172.

Mount DR, Ankley GT, Brix KV, Clements WH, Dixon DG, Fairbrother A, Hickey CW, Lanno RP, Lee CM, Munns WR, Ringer RK, Staveley JP, Wood CM, Erickson RJ, and Hodson PV. 2003. Effects assessment. *In Reevaluation of the State of the Science for Water-Quality Criteria Development. Edited by* M.C. Reiley, W.A. Stubblefield, W.J. Adams, D.M. Di Toro, P.V. Hodson, R.J. Erickson and F.J. Keating. SETAC Press, Pensacola, FL. pp. 53-118.

Naddy RB, Stubblefield WA, May JR, Tucker SA, and Hockett JR. 2002. The effect of calcium and magnesium ratios on the toxicity of copper to five aquatic species in freshwater. *Environ Toxicol Chem* **21**: 347–352.

Ng TY-T, Chowdhury MJ, and Wood CM. 2010. Can the biotic ligand model predict Cu toxicity across a range of pHs in softwater-acclimated rainbow trout? *Environ Sci Technol* **44**: 6263–6268.

Nimick DA, Gammon JR, and Parker SR. 2011. Diel biogeochemical processes and their effect on the aqueous chemistry of streams: A review. *Chem Geol* **283**: 3-17.

Nimick DA, Gammons CH, Cleasby TE, Madison JP, Skaar D, and Brick CM. 2003. Diel cycles in dissolved metal concentrations in streams: Occurrence and possible causes. *Water Resour Res* **39**: 1247.

Nimick DA, Harper DD, Farag AM, Cleasby TE, MacConnell E, and Skaar D. 2007. Influence of in-stream diel concentration cycles of dissolved trace metals on acute toxicity to one-year-old cutthroat trout (*Oncorhynchus clarki lewisi*). *Environ Toxicol Chem* **26**: 2667–2678.

Niyogi S, and Wood CM. 2004. Biotic Ligand Model, a flexible tool for developing site-specific water quality guidelines for metals. *Environ Sci Technol* **38**: 6177 -6192.

Ott DS, and Maret TR. 2003. Aquatic assemblages and their relation to temperature variables of least-disturbed streams in the Salmon River basin, Idaho, 2001. U.S. Geological Survey, Water-Resources Investigative Report 03-4076, Boise, Idaho.

Pagenkopf GK. 1983. Gill surface interaction model for trace-metal toxicity to fishes: role of complexation, pH, and water hardness. *Environ Sci Technol* **17**: 342-347.

Paquin PR, Gorsuch JW, Apte S, Batley GE, Bowles KC, Campbell PGC, Delos CG, Di Toro DM, Dwyer FJ, Galvez F, Gensemer RW, Goss GG, Hogstrand C, Janssen CR, McGeer JC, Naddy RB, Playle RC, Santore RC, Schneider U, Stubblefield WA, Wood CM, and Wu KB. 2002. The biotic ligand model: a historical overview *Comp Biochem Physiol C Toxicol Pharmacol* **133**: 3-35.

Paquin PR, Redman A, Ryan AC, and Santore RC. 2011. Modeling the physiology and toxicology of metals. *Fish Physiology: Homeostasis and Toxicology of Non-Essential Metals* **31**: 429-484.

Playle RC, Dixon DG, and Burnison BK. 1993a. Copper and cadmium binding to fish gills: estimates of metal-gill stability constants and modelling of metal accumulation. *Can J Fish Aquat Sci* **50**: 2678-2686.

Playle RC, Dixon DG, and Burnison BK. 1993b. Copper and cadmium binding to fish gills: modification by dissolved organic carbon and synthetic ligands. *Can J Fish Aquat Sci* **50**: 2667-2677.

Playle RC, Gensemer RW, and Dixon DG. 1992. Copper accumulation on gills of fathead minnows: influence of water hardness, complexation and pH of the gill micro-environment. . *Environ Toxicol Chem* **11**: 381-391.

Richards JG, and Playle RC. 1999. Protective effects of calcium against the physiological effects of exposure to a combination of cadmium and copper in rainbow trout (*Oncorhynchus mykiss*). *Can J Zool* **77**: 1035–1047.

Ryan AC, Tomasso JR, and Klaine SJ. 2009. Influence of pH, hardness, dissolved organic carbon concentration, and dissolved organic matter source on the acute toxicity of copper to *Daphnia magna* in soft waters: implications for the biotic ligand model. *Environ Toxicol Chem* **28**: 1663–1670.

Ryan AC, Van Genderen EJ, Tomasso JR, and Klaine SJ. 2004. Influence of natural organic matter source on copper toxicity to larval fathead minnows (*Pimephales promelas*): implications for the biotic ligand model. *Environ Toxicol Chem* **23**: 1567–1574.

Sandahl JF, Baldwin DH, Jenkins JJ, and Scholz NL. 2007. A sensory system at the interface between urban stormwater runoff and salmon survival. *Environ Sci Technol* **41**: 2998–3004.

Santore RC, Paquin PR, Di Toro DM, Allen HE, and Meyer JS. 2001. Biotic ligand model of the acute toxicity of metals. 2. Application to acute copper toxicity in freshwater fish and *Daphnia*. *Environ Toxicol Chem* **20**: 2397-2402.

Sauter S, Buxton KS, Macek KJ, and Petrocelli SR. 1976. Effects of exposure to heavy metals on selected freshwater fish: toxicity of copper, cadmium, chromium and lead to eggs and fry of seven fish species. U.S. Environmental Protection Agency, EPA-600/3-76-105, Duluth, Minnesota.

Schmidt TS, Clements WH, Mitchell KA, Church SE, Wanty RB, Fey DL, Verplanck PL, and San Juan CA. 2010. Development of a new toxic-unit model for the bioassessment of metals in streams. *Environ Toxicol Chem* **29**: 2432–2442.

Schubauer-Berigan MK, Dierkes JR, Monson PD, and Ankley GT. 1993. pH-dependent toxicity of Cd, Cu, Ni, Pb, and Zn to *Ceriodaphnia dubia, Pimephales promelas, Hyalella azteca,* and *Lumbriculus variegatus*. *Environ Toxicol Chem* **12**: 1261-1266.

Schwartz ML, Curtis PJ, and Playle RC. 2004. Influence of natural organic matter source on acute copper, lead, and cadmium toxicity to rainbow trout (*Oncorhynchus mykiss*). *Environ Toxicol Chem* **23**: 2889–2899.

Schwartz ML, and Vigneault B. 2007. Development and validation of a chronic copper biotic ligand model for *Ceriodaphnia dubia*. *Aquat Toxicol* **84**: 247-254.

Sciera KL, Isely JJ, Tomasso JR, Jr., and Klaine SJ. 2004. Influence of multiple water-quality characteristics on copper toxicity to fathead minnows (*Pimephales promelas*). *Environ Toxicol Chem* **23**: 2900–2905.

Scott GR, and Sloman KA. 2004. The effects of environmental pollutants on complex fish behaviour: integrating behavioural and physiological indicators of toxicity. *Aquat Toxicol* **68**: 369-392.

Seim WK, Curtis LR, Glenn SW, and Chapman GA. 1984. Growth and survival of developing steelhead trout (*Salmo gairdneri*) continuously or intermittently exposed to copper. *Can J Fish Aquat Sci* **41**: 433-438.

Shaw JR, Dempsey TD, Chen CY, Hamilton JW, and Folt CL. 2006. Comparative toxicity of cadmium, zinc, and mixtures of cadmium and zinc to Daphnids. *Environ Toxicol Chem* **25**: 182-189.

Stephan CE. 1986. Proposed goal of applied aquatic toxicology. *In* Aquatic Toxicology and Hazard Assessment: Ninth Volume, ASTM Special Technical Publication 921. American Society for Testing and Materials (ASTM), Philadelphia, PA. pp. 3-10.

Stephan CE, Mount DI, Hansen DJ, Gentile JH, Chapman GA, and Brungs WA. 1985. Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses. U.S. Environmental Protection Agency, EPA 822-R-85-100, NTIS PB85 227049, Duluth, Narragansett, and Corvallis.

Stratus. 1996. Preliminary toxicological evaluation, U.S. v. Iron Mountain Mines, Inc. Stratus Consulting, Inc. (formerly Hagler Bailly Services), Boulder, Colo.

Stratus. 1998. Data report: Acute copper toxicity to salmonids in surface waters in the vicinity of the Iron Mountain Mine, California. Stratus Consulting, Inc. (formerly Hagler Bailly Services), Boulder, Colo.

Tipping E, and Hurley MA. 1992. A unifying model of cation binding by humic substances. *Geochim Cosmochim Acta* **56**: 3627-3641

Van Genderen EJ, Gensemer RW, Smith C, Santore RC, and Ryan AC. 2007. Evaluation of the Biotic Ligand Model relative to other site-specific criteria derivation methods for copper in surface waters with elevated hardness *Aquat Toxicol* **84**: 279-291.

Van Genderen EJ, Ryan AC, Tomasso JR, and Klaine SJ. 2005. Evaluation of acute copper toxicity to larval fathead minnows (*Pimephales promelas*) in soft surface waters. *Environ Toxicol Chem* **24**: 408–414.

Waiwood KG, and Beamish FWH. 1978a. The effect of copper, hardness and pH on the growth of rainbow trout, *Salmo gairdneri*. *J Fish Biol* **13**: 591-598.

Waiwood KG, and Beamish FWH. 1978b. Effects of copper, pH and hardness on the critical swimming performance of rainbow trout (*Salmo gairdneri*). *Water Res* **12**: 611-619.

Wang N, Ingersoll CG, Greer EI, Hardesty DK, Ivey CD, Kunz JL, Brumbaugh WG, Dwyer FJ, Roberts AD, Augspurger T, Kane CM, and Neves RJ. 2007a. Chronic toxicity of copper and ammonia to freshwater mussels (Unionidae). *Environ Toxicol Chem* **26**: 2048–2056.

Wang N, Ingersoll CG, Hardesty DK, Ivey CD, Kunz JL, May TW, Dwyer FJ, Roberts AD, Augspurger T, Kane CM, Neves RJ, and Barnhart MC. 2007b. Acute toxicity of copper, ammonia, and chlorine to glochidia and juveniles of freshwater mussels (Unionidae). *Environ Toxicol Chem* **26**: 2036-2047.

Wang N, Mebane CA, Kunz JL, Ingersoll CG, Brumbaugh WG, Santore RC, Gorsuch JW, and Arnold WR. 2011. Influence of DOC on toxicity of copper to a unionid mussel (*Villosa iris*) and a cladoceran (*Ceriodaphnia dubia*) in acute and chronic water exposures. *Environ Toxicol Chem* **30**: 2115–2125.

Wang N, Mebane CA, Kunz JL, Ingersoll CG, May TW, Arnold WR, Santore RC, Augspurger T, Dwyer FJ, and Barnhart MC. 2009. Evaluation of acute copper toxicity to juvenile freshwater mussels (fatmucket, *Lampsilis siliquoidea*) in natural and reconstituted waters. *Environ Toxicol Chem* **28**: 2367–2377.

Welsh PG. 1996. Influence of dissolved organic carbon on the speciation, bioavailability and toxicity of metals to aquatic biota in soft water lakes. Ph.D. dissertation, University of Waterloo, Waterloo, Ontario, Canada.

Welsh PG, Chapman GA, Hansen JA, and Lipton J. 2001. Importance of ionic composition of reconstituted laboratory test water in interpreting metal toxicity test results. *In* Environmental Toxicology and Risk Assessment: Science, Policy, and Standardization - Implications for Environment Decisions: Tenth Volume ASTM STP 1403. *Edited by* B.M. Greenberg, R.N. Hull, M.H. Roberts and R.W. Gensemer. American Society for Testing and Materials (ASTM), West Conshohoken, PA. pp. 3-15.

Welsh PG, Lipton J, Mebane CA, and Marr JCA. 2008. Influence of flow-through and renewal exposures on the toxicity of copper to rainbow trout. *Ecotox Environ Safe* **69**: 199-208.

Welsh PG, Lipton J, Podrabsky TL, and Chapman GA. 2000. Relative importance of calcium and magnesium in hardness-based modification of copper toxicity. *Environ Toxicol Chem* **19**: 1624–1631.

Welsh PG, Parrott JL, Dixon DG, Hodson PV, Spry DJ, and Mierle G. 1996. Estimating acute copper toxicity to larval fathead minnow (*Pimephales promelas*) in soft water from

measurements of dissolved organic carbon, calcium, and pH. *Can J Fish Aquat Sci* **53**: 1263-1271.

Welsh PG, Skidmore JF, Spry DJ, Dixon DG, Hodson PV, Hutchinson NJ, and Hickie BE. 1993. Effect of pH and dissolved organic carbon on the toxicity of copper to larval fathead minnow (*Pimephales promelas*) in natural lake waters of low alkalinity. *Can J Fish Aquat Sci* **50**: 1356–1362.

Yoro SC, Panagiotopoulos C, and Sempéré R. 1999. Dissolved organic carbon contamination induced by filters and storage bottles. *Water Res* **33**: 1956-1959.

Appendix D

Conservative assumptions to be used in implementing criteria through effluent limits

The EPA's approach to implementing water quality based effluent limits in Idaho generally includes several conservative assumptions (EPA 2010:pp. 67-69). These conservative assumptions are designed to limit the discharge of pollutants in effluent such that pollutants would seldom be allowed to reach their "face value" criteria concentrations in waters receiving permitted discharges, downstream of mixing zones. Pursuant to Reasonable and Prudent Measure 2, Term and Condition 3.a., EPA will consistently apply three of these conservative assumptions in calculating effluent limits for discharges composed of any of the pollutants subject to this consultation for all NPDES permits in Idaho. The NMFS expects that application of these assumptions will reduce and minimize the take of the listed species subject to this Opinion.

The three conservative assumptions that EPA will implement pursuant to Term and Condition 3.a. are: (1) Assume that Only a Portion of the Low Stream Flow is Available for Mixing to Control Chronic Toxicity (mixing zone allowances); (2) Assume Receiving Stream Flows are Very Low; and (3) Assume the Maximum Permitted Discharge Volume. The NMFS evaluates the expected efficacy of these measures below

To evaluate the likely effectiveness of the three required conservative assumptions quantitatively, we selected the NPDES limits for the Thompson Creek Mine (TCM) as a relevant case study. This facility's permit was chosen because this facility had the necessary information most readily and transparently available to us. This information included:

1. Long-term flow records for the receiving waters were readily available via the internet, with a 37-year period of record (Figure D-2)[13];

2. A written description of the mixing zone allowances was available online (IDEQ 2000); and

3. The effluent limitations were available online and the calculations were described in a transparent and reproducible manner (EPA 2000).

The TCM facility has five permitted outfalls that discharge into three very different stream types:

1. Thompson Creek, a small stream with moderately-hard water (5th percentile hardness of 85 to 93 mg/L calcium carbonate) and very little dilution capacity during low flows with a 7Q10 flow of only 2.1 cfs (the 7Q10 is explained later);

[13] http://waterdata.usgs.gov/nwis.

2. Squaw Creek, a larger, hard water stream (5th percentile hardness of 290 mg/L calcium carbonate) with about double the flows of Thompson Creek and a 7Q10 of about 4.6 cfs; and

3. The upper Salmon River, a much larger, soft water stream (5th percentile hardness of 27 mg/L calcium carbonate) with a 7Q10 of about 323 cfs.

The characteristics of these discharges to these three water bodies are reasonably representative examples of the other facilities in the action area for which less information was readily available online.

D.1 Conservatism of assuming that only a portion of the low stream flow is available for mixing (mixing zone allowances);

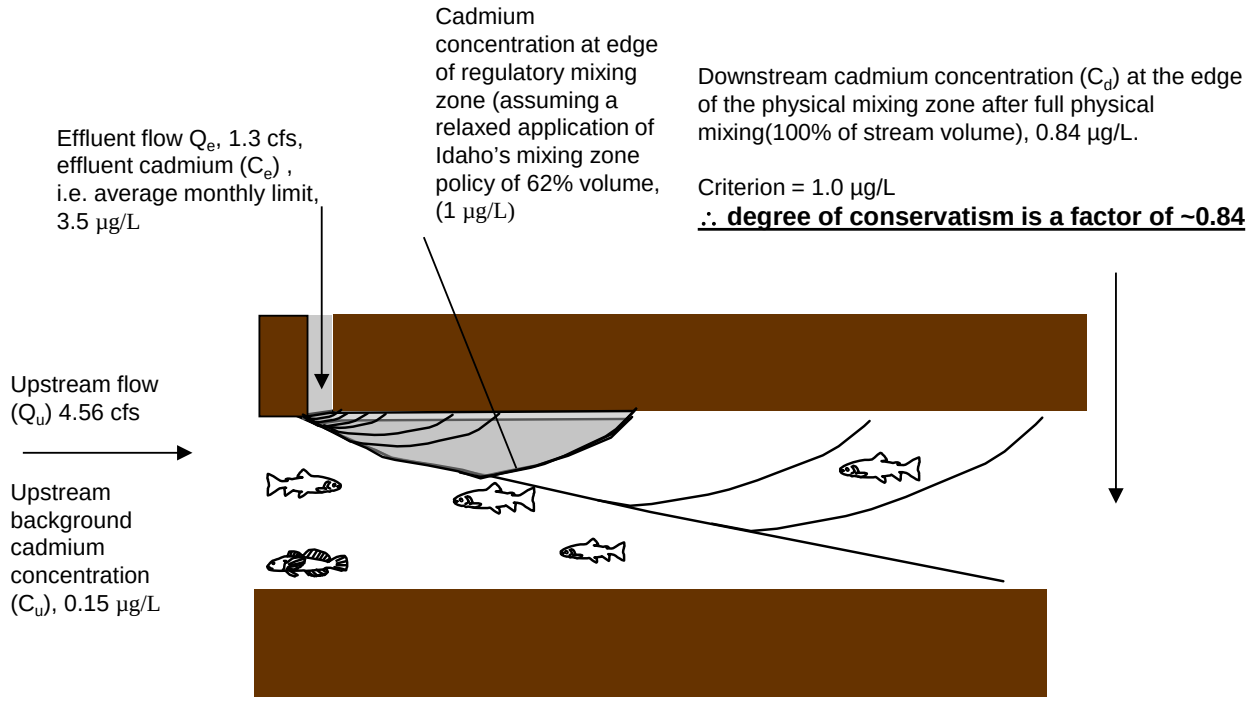
Under the first conservative assumption, EPA uses only a portion of the low receiving waterbody flow for dilution when calculating chronic limits. This is done in order to theoretically allow space in streams for passage of fish and other mobile aquatic species without having to pass through the mixing zone. This procedure further reduces the volume of the receiving stream which is used for permitting purposes, and therefore provides additional protection to aquatic species from chronic effects. The portion of the flow allowed for dilution is presumed to be 25% based on Idaho's Water Quality Standards, but based upon site-specific analyses of physical, biological, and chemical conditions, other fractions may be allowed. This discretion to relax or tighten the mixing zone percentage means that the actual conservative factor resulting from this policy may differ from the presumed limitation to 25% of the low stream flow. The State of Idaho is publishing more rigorous guidance on their mixing zone policies and it is now unlikely that mixing zone determinations would be proposed that would permit greater than 25% of receiving water flows to be used to dilute effluents without supporting technical analyses.[14]

For the TCM facility, some flexibility for both the listed species and the discharger was demonstrated by the state and EPA, with 0% mixing zone allowed for copper under certain flows and up to 62% of the stream volume allowed for cadmium. With cadmium, the allowable portions of receiving waters allowed for mixing range from 5% to 62% of actual stream flow for different streams and flow conditions (IDEQ 2000). The rationales for setting mixing zone fractions included avoiding concentrations likely to cause behavioral avoidance in salmonids, retaining sufficient zone of passage with suitable water velocities and depths for juvenile and adult salmonids, load allocations between outfalls, and limiting the travel time for drifting organism through the "acutely toxic" portion of effluent plumes to 1 minute or less, based upon the calculated instream concentrations and modeled time and distance for plume dilutions (IDEQ 2000, table 21).

Using the calculation methods of EPA (2000), NMFS evaluated the degree of conservatism resulting from various mixing zone limitations. A pessimistic (i.e., least-

[14] http://www.deq.idaho.gov/water/data_reports/surface_water/monitoring/mixing_zones.cfm accessed 01Oct2010.

conservative) example in which 50% of the portion of the receiving water flow was allowed for mixing of effluents is shown in Figure D-1. There, the degree of conservatism resulting from the limitation that only a portion of the receiving water stream flow could be used was a factor of 0.84. Other permitted conditions at the TCM facility were calculated as conservative factors ranging from a minimum of 0.22 for the most restrictive 5% mixing zone authorization; to 0.39 for the quasi-default mixing zone of 25% portion of flow; and to 0.84 for the mixing zone allowing 62% of the stream flow to be used.



$$C_d = \frac{C_u Q_u + C_e Q_e}{Q_e + Q_u} = C_{(downstream)} = \frac{(0.15\ \mu g/L \cdot 4.56\ cfs) + (3.5\ \mu g/L \cdot 0.87\ cfs)}{(0.87+4.56)\ cfs} = 0.84\ \mu g/L$$

Figure D-1. Conservatism resulting from a liberal application of Idaho's mixing zone policy which allowed 62% of the stream flow to be used for diluting effluents.

D.2 Conservatism of assuming receiving stream flows are very low

The second conservative assumption measure is to assume that receiving stream flows are very low, based on EPA's concept of design flows for effluent discharges. Stream flows are variable and a target of effluent limitations is to approximate provisions in the aquatic life criteria that limit the tolerable frequency of excursions above water quality criteria. In the IWQS, for chronic criteria this is defined as the 7-day, once in 10-year low flow or 7Q10 (EPA 1991; IDEQ 2007).

In the Thompson Creek example, the concept of a 7Q10 was interpreted by EPA more liberally than a "7-day, once in 10-year low flow." Rather, EPA defined "seasonal 7Q10s" where there is a conventional 7Q10, and then defined effluents set for a higher flow tier that occurs during spring snowmelt. By effectively having two 7Q10s for the same time period, the allowable frequency of excursions is greater than if a conventional 7Q10 were used. The higher flow tiers during spring runoff were considered appropriate by EPA (2000b) because of the extreme variability in effluent and receiving water flows. To keep comparable levels of protection during the high flow tiers when more effluents could be discharged, EPA (2000) required minimum dilution ratios as part of the permit.

We evaluated the degree to which the assumption that receiving stream flows are very low acts as a conservative measure (as stated in EPA (2010a)) by comparing the assumed low flows to the actual flows in Thompson and Squaw Creeks (Figure D-2). To avoid an optimistic review, we used water year 2007 because it was a year with lower than average flows. Flows in late summer and fall of 2007 (blue line) were considerably lower than the long-term average (brown line). Thompson Creek was in its higher flow tier for about 4 months of the year from March through July. The minimum measured flow in Thompson Creek in 2007 was effectively equal to the 7Q10 flow used in the effluent calculations, 2.1 vs. 2.05 CFS respectively (Figure D-2).

To determine to what extent the actual flows provided a "conservative factor," we compared to the "low flow 7Q10" of 2.05 CFS and the "high flow 7Q10" of 7 CFS and divided the low or high "7Q10" by the actual flow for each day during calendar year 2007, and then calculated summary statistics for the year. The same thing was done with mean daily values for the 37 year period of record (i.e., the mean daily flow for October 1 for all 37 years, October 2 for all 37 years, and so on). These results are summarized in Table D-1.

For the four scenarios we analyzed, 95% of the time, the low-flow assumption resulted in a "conservative factor" of at least 0.84 (range 0.66-0.84). On the average, the "conservative factors" were about 0.4 (Table D-1).

When calculated in this manner, lower proportions are more conservative, and a value of one indicates no conservatism context. It would be equivalent to express the "conservative factors" as reciprocals so that bigger numbers correspond with increasing conservatism. Thus, it would be equivalent to say that 95% of the time, the low-flow assumption resulted in a "conservative factor" of at least 1.2 (range 1.2 to 1.6), and on the average the "conservative factors" were about 2.5.

Table D-1. "Conservative factors" resulting from assumed low flows in two streams receiving mining effluents. Lower factors are more protective and a factor of 1.0 provides no additional conservatism.

Conservative Factor	Thompson Creek 2007	Squaw Cr 2007	Thompson Cr – 37 year average	Squaw Cr – 37 year average
Median	0.44	0.38	0.43	0.41
Average	0.45	0.40	0.41	0.40
90th percentile	0.70	0.56	0.58	0.50
95th percentile	0.84	0.75	0.76	0.66
Least conservative	1.00	1.00	1.00	0.98

Thus, a moderately pessimistic estimate of how much protection the "conservative factors" actually provided by limiting a portion of the low stream flow allowed for mixing is a factor of 0.84 and for assuming low receiving water flows coincidentally is also about 0.84 (i.e., 95% of the time it is more protective). Since these two measures are combined jointly, their product is 0.70.

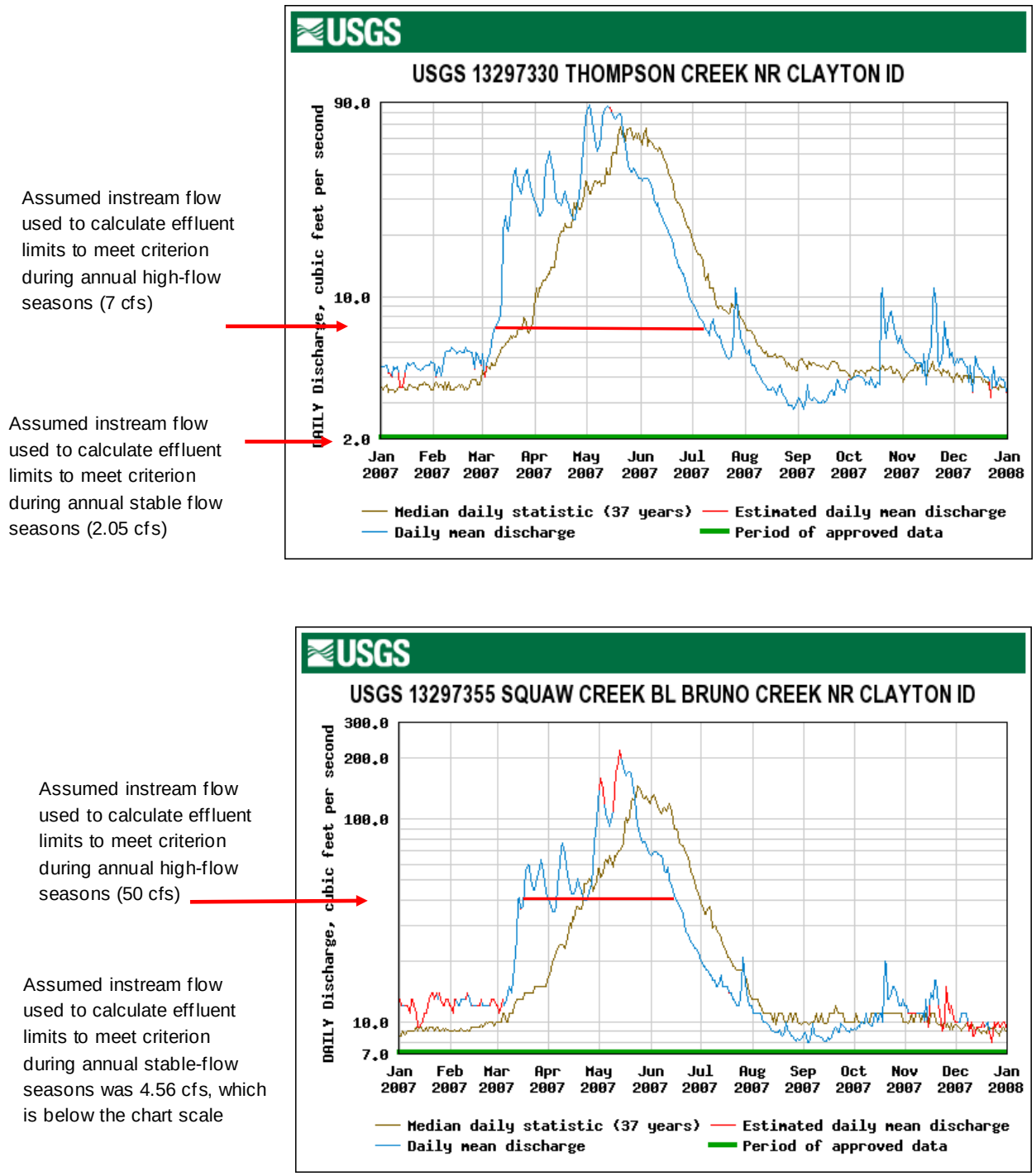


Figure D-2. Examples of actual stream flows versus stream flows that were assumed to calculate seasonally variable wastewater discharge limits for a facility. Actual flows were estimated to be lower than the seasonally adjusted assumed flows about 98% of the time (IDEQ 2000; EPA 2000).

D.3 Conservatism of assuming the maximum permitted discharge volumes

The EPA's (2010a) final conservative measure is to assume the Maximum Permitted Discharge Volume is closely related to the analysis of receiving water stream flows. This assumption is overstated slightly in that EPA assumes a higher than average permitted discharge volume, not the absolute maximum. For example, at Thompson Creek outfall #2, the maximum effluent volume contributed 14% of the flow of Thompson Creek. The NPDES permit assumed that the effluent would contribute about 8% of the flow, which was close to the 99^{th} percentile of flow percentages. The 95^{th} percentile effluent volume contributed about 5% of upstream flows (IDEQ 2000; EPA 2000). This means that for this outfall, about 95% of the time, effluent volumes were less than or equal to about 5/8 of those permitted providing another "conservative factor" of 0.7. The likely compounded conservatism of this aspect of effluent limitations would be 0.7 X 0.84 X 0.84 for at least 0.95^2 of the time which equals 0.5 for at least 90% of the time. This can be restated as follows.

The overall conservatism of the three conservative assumptions evaluated here can be summarized and were estimated as:

Assumption 1: Limiting the portion of stream flow allowed for mixing of effluents. The conservatism factor for this measure was estimated at about 0.84 or less (from Figure D-1), where the conservatism factor is expressed as a proportion and smaller values are more conservative;

Assumption 2: Assuming receiving stream flows are very low. About 95% of the time, the conservatism factor for this measure was also estimated as about 0.84 or less (from D-1); and

Assumption 3: Assuming unusually high permitted discharge volumes. About 95% of the time, the conservatism factor for this measure was estimated at about 0.7 or less (from text following Figure D-2).

The overall conservatism of these factors can be estimated as their product, i.e., $1 \times 2 \times 3 = 0.84 \times 0.84 \times 0.7 \approx 0.5$. The protectiveness of assumptions 2 and 3 vary over time, thus the proportions of time need to be combined. If stream flows and effluent volumes vary independently, then the time proportions should be multiplied together, i.e., $0.95 \times 0.95 = 0.9$. This can be restated that at least 90% of the time, the overall conservatism factor of measures 1, 2, and 3 is a factor of 0.5 or less.

If the effluent and receiving water assumptions made for Thompson Creek are further assumed to not be much more stringent or lenient than is typical, then it could be assumed that these three conservative assumptions will reduce the allowed chemical concentrations from point source discharges to about 50% of the criterion values for the great majority of the time. This

provides a significant reduction in exposure to pollutants from NPDES permit discharges and will minimize take of listed salmon and steelhead.

References for Appendix D

D.4 References

EPA. 1991. Technical support document for water quality-based toxics control. Office of Water, U.S. Environmental Protection Agency, EPA 505/2-90-001, Washington, D.C. 143 pp.

EPA. 2000. [Fact sheet to Reissue a Wastewater Discharge Permit to Thompson Creek Mining, Clayton, ID, NPDES Permit Number: ID-002540-2]. U.S. Environmental Protection Agency, Office of Water, Seattle, WA. 61 pp.

EPA. 2010. Biological evaluation of the Idaho water quality criteria for cadmium with revised hardness cap (September 2, 2010). U.S. Environmental Protection Agency, September 2, 2010, Seattle, WA. 194 pp.

IDEQ. 2000. Evaluation of proposed new point source discharges to a special resource water and mixing zone determinations: Thompson Creek Mine, upper Salmon River subbasin, Idaho. Idaho Department of Environmental Quality, Boise. 126 pp. http://deq.idaho.gov/media/450859-thompson_creek_mixing_zone_report.pdf (accessed April 2014)

IDEQ. 2007. Rules of the Department of Environmental Quality, IDAPA 58.01.02, "Water Quality Standards". revised March 30, 2007.

Appendix E

Biomonitoring of Effects

When Biomonitoring is necessary to implement RPAs or RPMs the following protocols are to be used.

1. At a minimum, samples will be collected upstream (reference) and downstream of the discharge location(s).

2. At a minimum, benthic macroinvertebrates are to be evaluated to make sure effects are not greater that those described in the effects section. Fish communities shall also be monitored, to the extent such monitoring is not otherwise prohibited by regulation or policy. At a minimum, monitoring shall be conducted annually during late summer or fall base flows; annual monitoring is recommended. Because of the need to minimize confounding variability other than the discharge constituents:

    a Reference and comparison sites need to be similar, except for the discharge; e.g. size, gradient, channel type, temperature, substrate, other variables that structure communities;

    b Because some biological variables can be confounded by natural upstream-downstream changes (e.g., temperature, habitat size), paired watershed, or other out-of-watershed reference sites are recommended in addition to within-watershed upstream reference sites;

    c Artificial substrates (rock baskets) may be needed for macroinvertebrate monitoring if comparable habitats cannot be located (e.g., similar sized gravels and cobbles, velocities, depths, and shading).

3. Taxonomic enumeration of macroinvertebrate samples will be sufficient to be comparable with that used in the IDEQ stream ecological assessment program (Grafe 2002). Generally this means that invertebrates must be identified to the lowest practical level, which for insects in the Ephemeroptera, Plecoptera, and Trichoptera orders (EPT, mayflies, stoneflies, and caddisflies) means to the species level; and for non-EPT insects other than chironomids, crustaceans and molluscs usually means at least to the genus level. Other non-insect invertebrates except annelid worms can usually be identified to family, annelids are often only identified to order.

4. Sampling for tissue residues of concern. With arsenic, the focus is evaluating residues in salmonid invertebrate prey. This is because adverse effects of arsenic at environmentally relevant concentrations have been demonstrated from feeding studies with trout (Cockell *et al.* 1991; Hansen *et al.* 2004; Erickson *et al.* 2010). Laboratory analyses of arsenic in invertebrate tissues should include both inorganic and total arsenic, because inorganic forms of arsenic appear to be more toxic to fish than organic forms such as arsenobetaine

and di- or monomethyl arsenic (Erickson *et al.* 2011). Monitoring should target representative, composite invertebrate samples for analysis. It seems reasonable to assume that benthic invertebrates that are vulnerable to capture with disturbance techniques (kick nets, rock scrubs) roughly represent those invertebrates that are likewise vulnerable to being eaten by juvenile salmonids.

With selenium, the focus is evaluating if tissue residues are accumulating to harmful concentrations in the fish themselves. Juvenile fish are recommended for sampling because adverse effects to juveniles are more likely to occur in the first place, or affect population dynamics more than adverse effects to adult fish (Lemly and Skorupa 2007; Van Kirk and Hill 2007). Sculpin may be a useful surrogate species to target in tissue monitoring because they are often abundant in streams, have significant dietary overlap with juvenile salmonids, have a sedentary life style that makes them more likely to have experienced and integrated the exposures at the place they are collected from, and permits to capture and kill sculpin are less likely to be obstructed by regulators than listed salmonids. Further, Rhea *et al.* (2013) found that sculpin were good indicators of selenium exposure and sublethal effects in the Yankee Fork, Idaho. However, based on anecdotes of sculpin being abundant in selenium enriched streams in southeastern Idaho, sculpin are probably not so sensitive that they would eliminated from streams with elevated selenium which would make them a poor choice of a monitoring species for tissue residues.

5. Adverse effects will be gaged in comparison to deviation from upstream or other reference sites using at least the following metrics or indexes (Table E-1).

    a Deviation from reference may be assessed based upon values compared to effects differences listed in table Table E-1 without the need for statistical testing. This is because sufficient replication necessary for statistical hypothesis testing approaches to be sensitive may be precluded by concerns about effects of monitoring or by costs. Further, the magnitudes of difference from expected reference conditions are probably more biologically meaningful than whether a reduction is statistically significant at a given probability. If statistical approaches are used, the following issues are to be considered.

    b Many valid approaches to statistical interpretation of monitoring data have been developed, and the following approaches are not intended to preclude other supportable approaches. However, the appropriateness of alternate approaches must be described.

    c Hypothesis tests, which aim to minimize type I errors (false positive results), are standard procedures in scientific research, but they are often inappropriate in ESA reviews, where the primary objective is to prevent type II errors (false negative results). Recognizing this disparity is particularly important when the best data available are sparse and therefore lack statistical power, because hypothesis tests that use data sets with low statistical power are likely to commit type II errors, thereby denying necessary protection to threatened and endangered species (Johnson 1999; McGarvey 2007).

d Hypothesis tests, if used, to test for statistical difference between sites for metrics that are expected to be sensitive to pollutants, should be interpreted with balanced power for type I and type II errors (Dayton 1998; Di Stefano 2003; Denton *et al.* 2011). That is, for macroinvertebrate data, if retrospective power analysis indicates an 80% probability of detecting a specified effect size (β at 0.8) then the corollary test whether the effect was "statistically significant" is 20% (α of 0.2 or $p<0.2$). No fixed value for α (the probability of making a type I error, for example to incorrectly concluding impairment exists when in fact the apparent effect was only due to chance) is specified. While traditionally 'adequate' power has been settled by adherence to the 'five eighty' convention in which statistical significance (type I error rate, a) is fixed at 5% and statistical power considered adequate if it reaches 80% (type II error rate, b, of 20%) this places the 'burden of proof' disproportionately on those concerned about avoiding type II errors (Field *et al.* 2007). If statistical power analyses are used, the specified effect sizes are given in Table E-1.

Except for tissue residues, for which the bases for the table values are given in the respective sections of this Opinion, the references given in Table E-1 explain the methods and rationales for measuring the different effect metrics, but do not necessarily specify the effect values listed in Table E-1. Rather the magnitude of "critical effect" sizes were selected values are based upon subjective, professional judgments that were, in turn, influenced by two recent reviews (Munkittrick *et al.* 2009; Janz *et al*. 2010). The effect sizes listed in listed in Table E-1 probably are optimistic compared to the minimum differences detectable from statistical hypothesis testing using common replication efforts. This subjectivity and likely conservatism to the detectable differences seems both appropriate and unavoidable based on the information reviewed. For instance Janz *et al*. (2010) suggest that "*the stipulation of an effect size threshold is a judgment about biology, not simply a statistical or procedural decision, and relies on many underlying explicit or implicit judgments about the biological importance of an effect of a nominated magnitude.*"

Table E-1. Biomonitoring metrics to evaluate for effects of toxic discharges

	Effects difference for comparison to reference or table value	Reference/notes
Macroinvertebrates in streams and rivers:		
Idaho Stream Macroinvertebrate Index (SMI)	10%	(Jessup and Gerritsen 2002)
SMI component metrics (9 metrics related to taxa richness, dominance and tolerance)	10% for richness and dominance metrics; 20% for other metrics	(Carlisle and Clements 1999; Jessup and Gerritsen 2002)
Total macroinvertebrate biomass	20%	
Abundance of invertebrates considered vulnerable to predation by juvenile salmonids	20%	(Suttle et al. 2004)
Biomass of invertebrates considered vulnerable to predation by juvenile salmonids	20%	(Suttle et al. 2004)
Similarity between reference and assessment stations (Jaccard similarity or comparable index, e.g. observed/expected (O/E) comparison)	10%	Effects difference assumed to be similar to taxa richness measures
Fish		
Community surveys (IBI)	10%	(Mebane 2002b; Mebane et al. 2003)
Sentinel species (e.g. sculpin abundance or age classes)	20% for abundance; no difference for age classes	(Janz et al. 2010)
Relative abundance (catch per unit effort, CPUE, or snorkel counts)	20%	
Length-frequency analysis or numbers of age classes of salmonids or sculpins	10% difference for median lengths; no difference in age classes	
Mean condition factor of salmonid species		
Jaccard similarity	10%	Minimum detectable difference assumed to be similar to taxa richness measures
Tissue Residues		
Arsenic in benthic invertebrate prey organisms (as a representative composite community sample)	< 20 mg/kg dry weight	This review
Selenium in juvenile salmonids (whole-body)	< 7.6 mg/kg dry weight	This review
Selenium in adult sculpins (whole-body)	< 7.6 mg/kg dry weight	This review

References for Appendix E

Cockell, K.A., J.W. Hilton, and W.J. Bettger. 1991. Chronic toxicity of dietary disodium arsenate heptahydrate to juvenile rainbow trout (*Oncorhynchus mykiss*) *Archives of Environmental Contamination and Toxicology*. 21(4): 518-527

Dayton, P.K. 1998. Reversal of the burden of proof in fisheries management. *Science*. 279(5352): 821-822

Denton, D., J.M. Diamond, and L. Zheng. 2011. Test of significant toxicity: A statistical application for assessing whether an effluent or site water is truly toxic. *Environmental Toxicology and Chemistry*. 30(5): 1117-1126. *http://dx.doi.org/10.1002/etc.493*

Di Stefano, J. 2003. How much power is enough? Against the development of an arbitrary convention for statistical power calculations. *Functional Ecology*. 17(5): 707–709

Erickson, R.J., D.R. Mount, T.L. Highland, J.R. Hockett, E.N. Leonard, V.R. Mattson, T.D. Dawson, and K.G. Lott. 2010. Effects of copper, cadmium, lead, and arsenic in a live diet on juvenile fish growth. *Canadian Journal of Fisheries and Aquatic Sciences*. 67(11): 1816-1826. *http://dx.doi.org/10.1139/F10-098*

Erickson, R.J., D.R. Mount, J.D. Fernandez, T.L. Highland, J.R. Hockett, D.J. Hoff, and C.T. Jenson. 2011. Arsenic Toxicity to Juvenile Fish: Effects of Exposure Route, Arsenic Speciation, and Fish Species [platform presentation]. *in* Abstracts, SETAC North America 32nd Annual Meeting, November 16, 2011, Boston, MA. Society of Environmental Toxicology and Chemistry (SETAC), *http://boston.setac.org/*.

Field, S.A., P.J. O'Connor, A.J. Tyre, and H.P. Possingham. 2007. Making monitoring meaningful. *Austral Ecology*. 32(5): 485-491

Grafe, C.S., (ed.). 2002. Idaho Small Stream Ecological Assessment Framework: an Integrated Approach. Idaho Department of Environmental Quality, Boise. 276 pp. *http://www.deq.state.id.us/water/data_reports/surface_water/monitoring/publications.cfm*.

Hansen, J.A., J. Lipton, P.G. Welsh, D. Cacela, and B. MacConnell. 2004. Reduced growth of rainbow trout (*Oncorhynchus mykiss*) fed a live invertebrate diet pre-exposed to metal-contaminated sediments. *Environmental Toxicology and Chemistry*. 23(8): 1902–1911

Janz, D.M., D.K. DeForest, M.L. Brooks, P.M. Chapman, G. Gilron, D. Hoff, W.A. Hopkins, D.O. McIntyre, C.A. Mebane, V.P. Palace, J.P. Skorupa, and M. Wayland. 2010. Selenium toxicity to aquatic organisms. Pages 139-230 *in* P. M. Chapman, W. J. Adams, M. L. Brooks, C. G. Delos, S. N. Luoma, W. A. Maher, H. M. Ohlendorf, T. S. Presser, and D. P. Shaw, editors. *Ecological Assessment of Selenium in the Aquatic Environment*. Society of Environmental Toxicology and Chemistry (SETAC), Pensacola, Florida, *http://dx.doi.org/10.1201/EBK1439826775-c6*.

Johnson, D.H. 1999. The insignificance of statistical significance testing. *Journal of Wildlife Management*. 63(3): 763–772

Lemly, A.D. and J.P. Skorupa. 2007. Technical issues affecting the implementation of US Environmental Protection Agency's proposed fish tissue–based aquatic criterion for selenium. *Integrated Environmental Assessment and Management*. 3(4): 552–558. *http://dx.doi.org/10.1897/IEAM_2007-024.1*

McGarvey, D.J. 2007. Merging precaution with sound science under the Endangered Species Act. *BioScience*. 57(1): 65–70

Munkittrick, K.R., C.J. Arens, R.B. Lowell, and G.P. Kaminski. 2009. A review of potential

methods for determining critical effect size for designing environmental monitoring programs. *Environmental Toxicology and Chemistry*. 28(7): 1361-1371. *http://dx.doi.org/10.1897/08-376.1*

Rhea, D., A. Farag, D. Harper, E. McConnell, and W. Brumbaugh. 2013. Mercury and Selenium Concentrations in Biofilm, Macroinvertebrates, and Fish Collected in the Yankee Fork of the Salmon River, Idaho, USA, and Their Potential Effects on Fish Health. *Archives of Environmental Contamination and Toxicology*. 64(1): 130-139. *http://dx.doi.org/10.1007/s00244-012-9816-x*

Van Kirk, R.W. and S.L. Hill. 2007. Demographic model predicts trout population response to selenium based on individual-level toxicity. *Ecological Modelling*. 206(3-4): 407-420. *http://dx.doi.org/10.1016/j.ecolmodel.2007.04.003*

Appendix F

Salmonid Zone of Passage Considerations

A zone of passage must be maintained around a mixing zone, sufficient to allow unimpeded passage of adult and juvenile salmonids. Determining what is "sufficient" may require site specific analysis. There is a long established precedent of using published expert opinion or expert consensus if no more than 25% of the cross sectional area was impinged upon, that would be sufficient for a zone of passage (FWPCA 1968; EPA 1994).

Recent examples have used different passage criteria. In a site-specific analysis, Mebane (2000) concluded that if the mixing zone of effluents into a small trout stream did not exceed 50% of the volume and width, then the unaffected portion of the channel was likely be sufficient for unimpeded passage around the mixing zone. That conclusion considered species and life stage requirements for appropriate depths, velocities, and habitat features including instream cover from predation in the unaffected portion of the channel. Other important considerations include situating mixing zones to avoid affecting or creating attractive habitat features in the effluent-exposed portions of the stream channel that could lead to fish congregating in mixing zones and risk disproportionate exposure to effluents. These habitat features to avoid influencing or creating might include locally important pool habitats, spawning areas or thermal refuges (e.g., Harper *et al.* 2009) in the mixing zones. The concept of avoiding spawning areas is necessarily subjective and cannot be defined in absolute terms. This is because fish can spawn in a variety of habitats, including those that experienced fisheries biologists might consider suboptimal, and the absence of spawning can rarely be proven. The intent is to avoid local concentrations of spawning habitat, not to preclude discharges into marginal habitats where spawning could potentially occur.

Instream flow studies for trout and salmon are another source of information for passage criteria. For example, the minimum depth criterion for adult fish passage must be present in greater than 25% of the total stream width in representative transects to allow passage (Maret *et al.* 2006).

The concept of mixing zone limitations are illustrated in Figures 2.9.1 and 2.9.2. Figure F-2 gives illustrates the results of effluent limit calculations with copper for a water body subject to the restriction that the volume of the receiving waters that is used for determining dilution and preserving a zone of passage for migrating fish and other aquatic life is limited to 25% of the stream volume. This example was calculated following the recent practices used by EPA Region 10 staff for determining effluent limits[15] and EPA's technical support manual for water quality based effluent limits (EPA 1991).

In the calculations presented for copper, when the effluents are limited in this manner, the increase in copper concentrations allowed after complete mixing is less than 0.6 µg/L, a concentration increase likely to contribute to impairment of olfaction and predator avoidance in

[15] http://yosemite.epa.gov/r10/WATER.NSF/NPDES+Permits/Permits+Homepage

juvenile salmon (Figure F-2). So long as the approaches described herein are followed, it seems likely that criteria for copper would adequately minimize adverse effects to listed salmonids. While an infinite variety of effluent and receiving water geometries, concentrations and flow conditions could be envisioned, the approach illustrated in Figure F-2 would result in similar results when the same decisions rules are applied in other configurations. Other cationic metals and other substances can cause chemosensory or avoidance behavior, but none were obviously more severe than copper (considered in the individual chemical sections). Thus, this approach would presumably be appropriate and as protective for other chemicals evaluated. A mixing zone demonstration in ESA waters should be rigorous enough to satisfy the information needs listed in Table F-1.

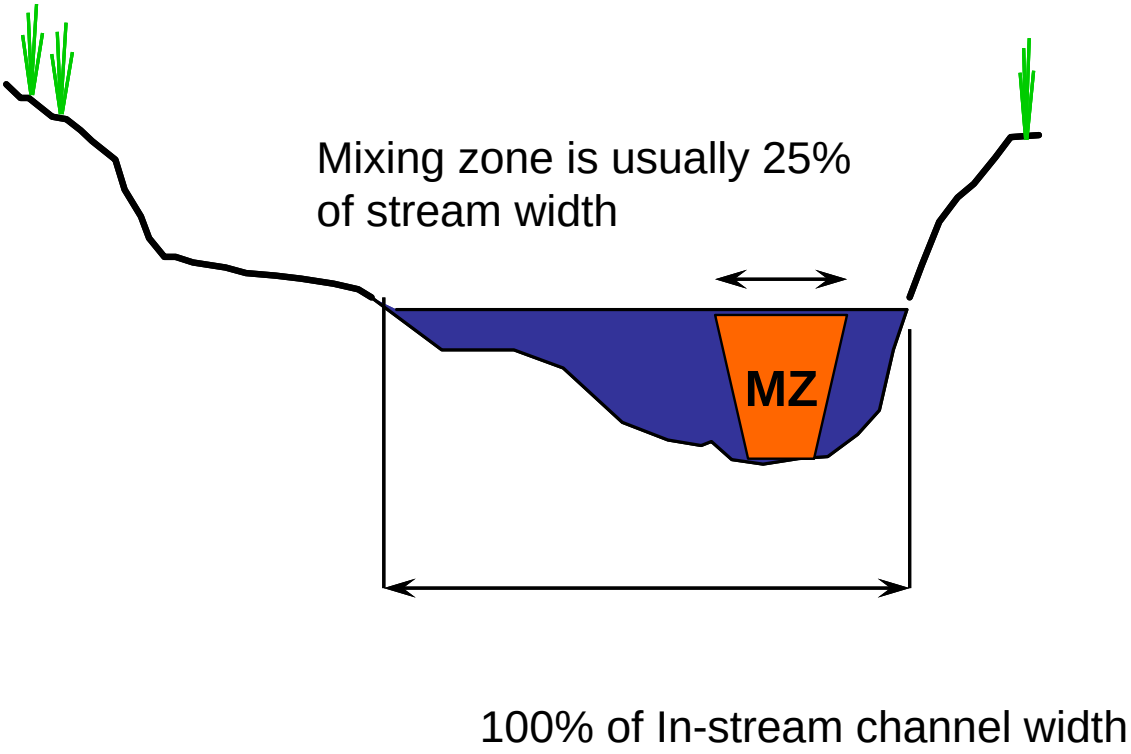
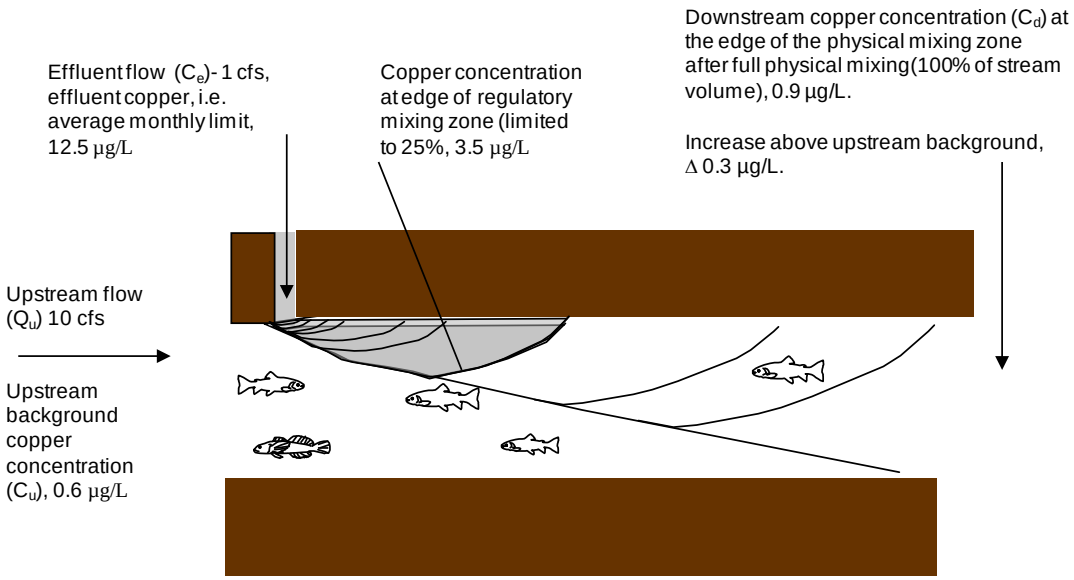


Figure F-1. Illustration of an effluent mixing zone cross section, illustrating how an effluent plume containing copper or other chemicals (trapezoid) would be limited to a fraction of the actual receiving stream width.



Calculated using simple mass balance equation

$$C_d = \frac{C_u Q_u + C_e Q}{Q_e Q_u}$$ where

C_d is the concentration downstream of the effluent discharge, C_u is the upstream concentration, Q_u is the upstream flow, C_e is the effluent concentration and Q_e is the effluent flow.

Figure F-2. Illustration of effluent limit calculations and resulting copper concentrations for a springtime, low-hardness scenario where the volume of the receiving water allowed to be used in calculated the effluent limits was limited to 25% of the actual stream volume.

Table F-1. Mixing zone demonstration in ESA waters which exceed either 25% volume or cross sectional area of a stream would require consideration of following elements:

Definition of location, width, downstream extent (where should compliance be monitored). In open-water (reservoirs, lakes) describe where discharge-induced mixing ends;
Describe stream channel characteristics, including depth and velocity profiles at high and low flows. Present an interpretation of available suitable habitat for juvenile and adult salmon and steelhead either using simple fixed criteria comparisons (e.g., Bjornn and Reiser. 1991; Mebane 2000) or with habitat suitability curves(e.g., Maret et al. 2006).
Map habitat features within the mixing zone, including geomorphic channel units (pools, runs, riffles), presence of fish cover from predation, including overhanging vegetation, instream vegetation, woody debris or boulders. Describe habitat features in the affected reach context of the overall stream segment and any likely limiting habitat features for the area.
Describe measured or projected discharge and receiving water temperatures in the context of whether the effluents would represent an “attractive nuisance” by providing a thermal refuge and leading to disproportionately greater exposure of fish to effluents than would be expected based on spatial proportions. A difference of 3°C warmer in winter or 3°C cooler in summer between the effluent and receiving water respectively is considered sufficient to create a potentially harmful thermal attractant (Poole et al. 2001).
Show that the mixing zone is unlikely interfere with or block passage of fish or aquatic life. If mixing zone impinges on a large fraction of the zone of passage, e.g., more than 50% of the channel cross sectional area, then rigorous demonstration of passage adequacy by techniques such as telemetry may be needed. For copper, zone of passage is sufficient if at least 50% of channel cross sections (under critical conditions) have relative dissolved copper concentrations of <0.6 µg/L greater than background concentrations. Additionally, in at least 50% of channel cross sections, absolute dissolved copper should be no greater than that allowed by EPA’s (2007) biotic ligand model-based criteria.
Does not otherwise interfere with aquatic ecosystems (protect uses), as demonstrated through biomonitoring and WET testing.
Describe background, show that adjacent mixing zones do not overlap, evaluate whether the organisms would be attracted to the MZ.
Evaluate the size of mixing zone in relation to the availability of critical habitat for a species. Describe the extent (i.e., physical and temporal extents, including fraction of total).

References for Appendix F

Bjornn, T.C. and D.W. Reiser. 1991. Habitat requirements of salmonids in streams. Pages 83-138 *in* W. R. Meehan, editor. *Influences of forest and rangeland management on salmonid fishes and their habitats. American Fisheries Society Special Publication 19*, Bethesda, MD.

EPA. 1991. Technical support document for water quality-based toxics control. Office of Water, U.S. Environmental Protection Agency, EPA 505/2-90-001, Washington, D.C. 143 pp. *http://www.epa.gov/npdes/pubs/owm0264.pdf*.

EPA. 1994. Water Quality Standards Handbook. U.S. Environmental Protection Agency, EPA-823-B-94-005a, Washington, D.C. *http://www.epa.gov/ost/standards/handbook/*.

EPA. 2007a. Aquatic life ambient freshwater quality criteria - copper, 2007 revision. U.S. Environmental Protection Agency, EPA-822-R-07-001 (March 2, 2007), Washington, DC. 208 pp. *http://www.epa.gov/waterscience/criteria/copper/* [Accessed 30 March 2008].

FWPCA (Federal Water Pollution Control Administration). 1968. Water Quality Criteria, report of the National Technical Advisory Committee to the Secretary of the Interior. Federal Water Pollution Control Administration, U.S. Department of the Interior, Washington, DC. 234 pp.

Harper, D.D., A.M. Farag, C. Hogstrand, and B. MacConnell. 2009. Trout density and health in a stream with variable water temperatures and trace element concentrations: does a cold-water source attract trout to increased metal exposure? *Environmental Toxicology and Chemistry*. 28(4): 800–808. *http://dx.doi.org/10.1897/08-072R.1*

Maret, T.R., J.E. Hortness, and D.S. Ott. 2006. Instream flow characterization of upper Salmon River basin streams, central Idaho, 2004. U.S. Geological Survey, Scientific Investigations Report 06-5230, Boise, Idaho. *http://id.water.usgs.gov/projects/salmon_streamflow/index.html* and *http://pubs.usgs.gov/sir/2006/5230/*.

Mebane, C.A. 2000. Evaluation of proposed new point source discharges to a special resource water and mixing zone determinations: Thompson Creek Mine, upper Salmon River subbasin, Idaho. Idaho Department of Environmental Quality, Boise. 126 pp. *http://deq.idaho.gov/water/data_reports/surface_water/monitoring/mixing_zones.cfm*.

Poole, G.C., J.B. Dunham, M.P. Hicks, D.M. Keenan, J.C. Lockwood, E.J. Materna, D.A. McCullough, C.A. Mebane, J.C. Risley, S.T. Sauter, S.A. Spalding, and D.J. Sturdevant. 2001. Technical Synthesis: Scientific Issues Related to Temperature Criteria for Salmon, Trout, and Char Native to the Pacific Northwest. US Environmental Protection Agency., EPA 910-R-01-007, Seattle, WA. 24 pp. *http://yosemite.epa.gov/r10/water.nsf/Water+Quality+Standards/WQS+Temperature+Guidance*.