

Appendix C:

Summary of Toxic Effects on Fish

Summary of Toxic Effects on Fish

(Condensed from EPA 1999)¹

¹ U.S. Environmental Protection Agency. 1999b. Biological assessment of the Idaho water quality standards for numeric water quality criteria for toxic pollutants. U.S. EPA, Region 10, Seattle Wa.

Table of Contents

1.0	Introduction.....	4
1.1	Effect of Abiotic Conditions on Toxicity.....	7
1.2	Uncertainty Analysis	8
1.3	Organization of Toxic Pollutant Determinations for Invertebrates, Fish and Birds.	9
2.0	Arsenic	10
2.1	Fish.....	11
3.0	Cadmium.....	12
3.1	Bioconcentration and Biomagnification.....	13
3.2	Fish.....	14
4.0	Copper.....	15
4.1	Bioconcentration and biomagnification	16
4.2	Fish.....	16
5.0	Lead.....	18
5.1	Bioconcentration and Biomagnification.....	19
5.2	Fish.....	19
6.0	Mercury.....	21
6.1	Bioconcentration and Biomagnification.....	21
6.2	Fish.....	22
7.0	Selenium.....	23
7.1	Bioconcentration and Biomagnification.....	23
7.2	Fish.....	24
8.0	Zinc	26
8.1	Bioconcentration and Biomagnification.....	27
8.2	Fish.....	28
9.0	Analysis of Effects of Numeric Criteria for Toxic Pollutants to White Sturgeon.....	29
9.1	Arsenic	30
9.2	Cadmium	30
9.3	Copper	31
9.4	Lead.....	32
9.5	Mercury.....	32
9.6	Selenium.....	33
9.7	Zinc.....	33

1.0 Introduction

EPA's Water Quality Standards regulations require states to adopt water quality criteria that will protect the designated uses of a water body. These criteria must be based on sound scientific rationale and must contain sufficient parameters or constituents to protect the designated uses. Since 1980, EPA has been publishing criteria development guidelines and national criteria for numerous pollutants. EPA's criteria documents provide a toxicological evaluation of the chemical, tabulate the relevant acute and chronic toxicity information and derive the acute and chronic criteria that EPA recommends for the protection of aquatic life resources. States may choose to adopt EPA's recommended criteria or modify these criteria to account for site-specific or other scientifically defensible factors.

Water quality criteria for aquatic life contain two expressions of allowable magnitude: a criterion maximum concentration (CMC) to protect against acute (short-term) effects; and a criterion continuous concentration (CCC) to protect against chronic (long-term) effects. EPA derives acute criteria from 48- and 96-hour tests of lethality or immobilization. EPA derives chronic criteria from longer term (often greater than 28-day) tests that measure survival, growth, or reproduction.

The quality of an ambient water body typically varies in response to variations of effluent quality, stream flow, and other factors. Organisms in the water body are not typically receiving constant, steady exposure but rather are experiencing fluctuating exposures, including periods of high concentrations, which may have adverse effects. Thus, EPA's criteria indicate a time period over which exposure is to be averaged, as well as an upper limit on the average concentration, thereby limiting the duration of exposure to elevated concentrations. For acute criteria, EPA recommends an averaging period of 1 hour. That is, to protect against acute effects, the 1-hour average exposure should not exceed the CMC. For chronic criteria, EPA recommends an averaging period of 4 days. That is, the 4-day average exposure should not exceed the CCC.

To predict or ascertain the attainment of criteria, it is necessary to specify the allowable frequency for exceeding the criteria. This is because it is statistically impossible to project that criteria will never be exceeded. As ecological communities are naturally subjected to a series of stresses, the allowable frequency of pollutant stress may be set at a value that does not significantly increase the frequency or severity of all stresses combined.

EPA recommends an average frequency for excursions of both acute and chronic criteria not to exceed once in 3 years. In all cases, the recommended frequency applies to actual ambient concentrations, and excludes the influence of measurement imprecision. EPA selected a 3-year average frequency of criteria exceedence with the intent of providing for ecological recovery from a variety of severe stresses. This return interval is roughly equivalent to a 7Q10 design flow condition. Because of the nature of the ecological recovery studies available, the severity of criteria excursions could not be rigorously related to the resulting ecological impacts. Nevertheless, EPA derives its criteria intending that a single marginal criteria excursion (i.e., a slight excursion over a 1-hour period for acute or over a 4-day period for chronic) would require little or no time for recovery. If the frequency of marginal criteria excursions is not high, it can be shown that the frequency of severe stresses, requiring

measurable recovery periods, would be extremely small. EPA thus expects the 3-year return interval to provide a very high degree of protection (EPA, 1994).

Section 303(c)(2)(E) of the Clean Water Act requires that all states adopt chemical-specific, numeric criteria for priority toxic pollutants. In 1992, the State of Idaho had not yet adopted such criteria. Therefore, on December 22, 1992 EPA promulgated such criteria for all waters in the State of Idaho as part of the National Toxics Rule (EPA, 1992). Idaho has since revised the Idaho Water Quality Standards to include the same criteria as EPA promulgated under the National Toxics Rule. Following completion of this consultation, EPA is proposing to recommend a federal action which would remove the State of Idaho from the National Toxics Rule, thus providing for the State's criteria to become effective.

The National Toxics Rule originally promulgated criteria for metals as total recoverable metals. Following EPA's promulgation of this rule, EPA issued a new policy for setting water quality criteria for metals. Therefore, on May 4, 1995 EPA issued a stay on the effectiveness of the metals criteria promulgated in the National Toxics Rule and promulgated revised criteria expressed in terms of dissolved metals (EPA, 1995). At this time, EPA also promulgated conversion factors for converting between dissolved and total recoverable criteria. States, when adopting criteria, may choose to adopt metals criteria measured as either dissolved or total recoverable. The metals criteria in the Idaho Water Quality Standards are expressed as dissolved metals.

In Idaho, both the aquatic life criteria and human health criteria apply to all surface waters of the State. Idaho's water quality standards contain a provision which states that when multiple criteria apply to a water body, the most stringent criterion is the applicable criterion. With regard to the numeric toxic criteria, all but several have more stringent aquatic life criteria than human health criteria. Therefore, with regard to the majority of the toxic criteria, the aquatic life criteria are the applicable criteria for surface waters. An example of an exception to this is arsenic, where the human health criterion is several orders of magnitude lower than the aquatic life criteria. Therefore, in all surface waters in Idaho, the applicable criteria for arsenic is the human health criteria.

All criteria in the Idaho Water Quality Standards, with the exception of the human health criterion for arsenic, are identical to the criteria promulgated by EPA under the National Toxics Rule. These criteria were adopted by reference in IDAPA 16.01.02.250.07. The aquatic life criteria evaluated as part of this assessment are summarized in Table 250.07.a.1. For comparison purposes, this table provides metals criteria expressed as both dissolved and total recoverable.

Idaho's criteria for pentachlorophenol (PCP) is expressed as an equation dependent on pH and seven of the criteria for metals are expressed as a function of water hardness. The PCP criteria in Table 250.07.a.1 were calculated at a pH of 7.8. In the following table, rather than present the equation for the hardness-dependent metals, EPA used a hardness of 100 mg/L CaCO₃ in order to present a value for the metals criteria. Therefore, although the criteria value would be dependent of the particular hardness value for a waterbody, in the following table, criteria were calculated at a hardness of 100 mg/L CaCO₃.

In the NTR, EPA described and required minimum and maximum hardness values (25 mg/L and 400 mg/L as CaCO₃, respectively) to be used when calculating hardness dependent freshwater metals criteria. Most of the data EPA used to develop the hardness formulas were

in the hardness range of 25 to 400 mg/L. Therefore, EPA stated that the formulas were most accurate in that range. Using a hardness of 25 mg/L for calculating criteria, when the actual ambient hardness is less than 25 mg/L, could result in criteria that are underprotective of aquatic life. Because the State of Idaho is still under the NTR, the lower and upper hardness cap values are applicable. Therefore until EPA withdraws the NTR from applying to Idaho the State is unable to use ambient hardness values outside this range for calculating hardness dependent metals criteria. When the NTR is withdrawn from applying to Idaho, the State will then have the option of using ambient hardness values outside the 25-400 mg/L range when calculating criteria for hardness dependent metals.

For reference, average, minimum, and maximum hardness measurements recorded in waters throughout the State of Idaho are presented in Appendix F. Hardness values observed throughout the State range from 14.07 mg/L in the Upper Selway River to 404 mg/L in the Lower Bear River, with an average of 103.8 mg/L. Literature describing the experiments referenced in this section did not always provide hardness values along with data. In cases where hardness values are lacking, comparisons of criteria to research results may not be reliable. For those metals which are hardness dependent, EPA Region 10 calculates NPDES permits limits and load allocations for TMDLs using the fifth percentile of the ambient and or effluent hardness values which are most often calculated from instantaneous data.

Chemical Name	Criteria (µg/L)		Total Recoverable Criteria (µg/L)		Conversion Factor ^a	
	Acute	Chronic	Acute	Chronic	Acute	Chronic
Arsenic	360	190	360	190	1.000	1.000
Cadmium	3.7 ^b	1.0 ^b	3.9 ^c	1.1 ^c	0.944 ^d	0.909 ^c
Copper	17 ^b	11 ^b	18 ^c	12 ^c	0.960	0.960
Cyanide	22 ^e	5.2 ^e		N/A		N/A
Endosulfan (ÿ & ÿ)	0.22	0.056		N/A		N/A
Lead	65 ^b	2.5 ^b	82 ^c	3.2 ^c	0.791 ^d	0.791 ^c
Mercury	2.1	0.012	2.4	0.012	0.85	N/A
Selenium	20	5.0		N/A		N/A
Zinc	110 ^b	100 ^b	120 ^c	110 ^c	0.978	0.986
Aldrin	3	N/A		N/A		N/A
Chlordane	2.4	0.0043		N/A		N/A
Chromium (III)	550 ^c	180 ^c	1,700 ^c	210 ^c	0.316	0.860
Chromium (VI)	15	10	16	11	0.982	0.962
4,4'-DDT	1.1	0.001		N/A		
Dieldrin	2.5	0.0019		N/A		
Endrin	0.18	0.0023		N/A		
Heptachlor	0.52	0.0038		N/A		N/A
Lindane (gamma-BHC)	2	0.08		N/A		N/A
Nickel	1,400 ^b	160 ^b	1,400 ^c	160 ^c	0.998	0.997
PCBs	N/A	0.014		N/A		N/A
Pentachlorophenol	209	139		N/A		N/A

Silver	3.4 ^b	N/A	4.1	N/A	0.85	N/A
Toxaphene	0.73	0.0002		N/A		N/A

N/A - no applicable criteria

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

b - Criteria for these metals are expressed as a function of total hardness (mg/L as CaCO₃), and the following formula:

$$\text{Acute Criteria} = \text{WER} \exp\{m_A[\ln(\text{hardness})] + b_A\} \times \text{Acute Conversion Factor}$$

$$\text{Chronic Criteria} = \text{WER} \exp\{m_C[\ln(\text{hardness})] + b_C\} \times \text{Chronic Conversion Factor}$$

where:

Metal	m_A^f	b_A^f	m_C^f	b_C^f
Cadmium	1.128	- 3.828	0.7852	- 3.490
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.

c - For comparison purposes, the values displayed in this table correspond to a total hardness of 100 mg/l CaCO₃ and a WER of 1.0.

d - The conversion factors for cadmium and lead are hardness dependent. The values shown in the table correspond to a hardness of 100 mg/L CaCO₃. Conversion factors for any hardness may be calculated using the following equations:

Cadmium:

$$\text{Acute- CF} = 1.136672 - [(\ln(\text{hardness})) \times (0.041838)]$$

$$\text{Chronic- CF} = 1.101672 - [(\ln(\text{hardness})) \times (0.041838)]$$

Lead:

$$\text{Acute and Chronic- CF} = 1.46203 - [(\ln(\text{hardness})) \times (0.145712)]$$

e - Criteria expressed as Weak Acid Dissociable

f - 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

g - Criteria for pentachlorophenol is expressed as a function of pH and calculated as follows:

$$\text{Acute Criteria} = \exp(1.005 (\text{pH}) - 4.830)$$

$$\text{Chronic Criteria} = \exp(1.005 (\text{pH}) - 5.290)$$

1.1 Effect of Abiotic Conditions on Toxicity

pH

The toxicity of several pollutants vary depending upon environmental conditions such as water hardness and pH. pH activity has a significant impact on the availability and toxicity of

metals. The following is summarized from Elder (1988) and Baker et al. (1990) IN ODEQ (1995). Metal-hydroxide complexes tend to precipitate (i.e., reduced ability to remain suspended) and are quite insoluble under natural water pH conditions; thus, the metal is not able to exert a toxic effect. However, the solubility of these complexes increases sharply as pH decreases. pH activity also impacts the sensitivity of organisms to a given amount of metal. There are two types of metals: type I metals (e.g., cadmium, copper, and zinc), that are less toxic as the pH decreases; and type II metals (e.g., lead), that are more toxic at lower pH values. Each metal has its own range where pH and site-specific conditions become factors in the metal's bioavailability. Aluminum is the metal of greatest concern at low pH values. No adverse effects to listed species due to pH-driven changes in metal toxicity (where the metals comply with the respective metals criteria) would occur in the range of Idaho's pH criteria. Both the direct toxicity of pH and that of aluminum result in osmoregulatory failure. The effects of low pH are also more pronounced at low concentrations of calcium. In summary, reductions in pH below natural levels will tend to increase metal availability and toxicity.

Temperature

No single pattern exists for the effects of temperature on the toxicity of pollutants on aquatic organisms. Temperature change in a given direction may increase, decrease, or cause no change in toxicity depending on many factors including the toxicant, species or the experiment. Sprague (1985) demonstrates that the effects of temperature on acute toxicity are diverse, but for the most part are only small or moderate. Some evidence suggests that temperature may not have much effect at all on the chronic "no-effect" thresholds of pollutants. One study described that temperature may either increase or decrease the EC₅₀, but no general pattern was evident. The researchers concluded that temperature had no effect on the EC₅₀ (Sprague, 1985).

Dissolved Oxygen

Reductions in dissolved oxygen may increase the toxicity of aquatic pollutants, but are often not the major factors affecting toxicity. Most evidence suggests that tests conducted at low and high levels of dissolved oxygen may change toxicity by only a factor of 2 or less (low dissolved oxygen being generally in the range of 20% saturation). In studies where low dissolved oxygen significantly modified LC₅₀s, the same effect did not hold true for sublethal toxicity (i.e. growth). Low oxygen appears to be less important than might be expected as a modifier of sublethal toxicity. Sprague suggests that while the picture of the influence of dissolved oxygen on toxicity is incomplete, "the effects may be as small as, or even smaller, than the modest effects on acute lethality" (Sprague, 1985).

1.2 Uncertainty Analysis

Concentrations of metals in the water column may be measured as either total recoverable or dissolved. The Idaho Water Quality Standards express metals concentrations as dissolved metals. Total recoverable analysis of metals allows an estimation of metal content of both the water and particulate matter. Since total recoverable methods take into account both dissolved and bound metal fractions, this method can sometimes overestimate the toxicity of an aquatic system. Dissolved analysis of metals estimates only the metal actually dissolved in the water column. This method may represent more closely the fraction of the metal that is bioavailable to aquatic organisms. However, it does not address metals bound to particulate matter and may underestimate the toxicity of an aquatic system by excluding ingestion of

particulates or ingestion of prey that consume particulates as a pathway for toxic chemical exposure. In addition, in the laboratory, total recoverable methods are often used to determine metal concentrations.

Toxicity of several pollutants for which criteria are included in the Idaho Water Quality Standards are either pH or hardness dependent. In these cases, the State's criteria are expressed as a function of pH or hardness. However, in many cases the literature does not report the environmental conditions under which toxicology experiments have been performed, including pH and hardness. Where relevant, EPA's analysis took into account whether pH and hardness values were provided. Where pH and hardness values were not reported in the literature and the criteria are expressed as a function of pH or hardness, the results should be interpreted with caution.

Other factors may also limit the accuracy of the determinations on the effects of the Idaho Water Quality Standards aquatic life criteria. First, the analysis of the criteria did not address the effects of the criteria on prey items of individual species or on their habitat beyond the water column. Toxic chemicals may affect aquatic organisms via ingestion (of contaminated prey or sediment particles) or through absorption (from water or from sediment). Furthermore, prey populations may decrease as a result of chemical contamination, thus depriving a species of food sources. The development of the criteria included effects for many prey species and should adequately protect prey of the listed species examined in this document. Second, the Idaho Water Quality Standards aquatic life criteria do not take into account the interactions between two or more chemicals which could be present in a water body. Some chemicals may interact resulting in more or less toxicity of one or more of the chemicals involved. Some metals such as cadmium and selenium exhibit antagonistic relationships with respect to toxicity. The literature did not provide any evidence to indicate synergistic interactions between metals (Furness and Rainbow, 1990). Synergism is defined as the interaction of toxicants resulting in greater toxicity than that predicted by the sum of the toxicities of each chemical. Finally, the analysis of the potential effects of toxic pollutants on threatened and endangered species included the examination of research conducted primarily with surrogate species. The surrogate species were selected as the closest related organism for which information was available. For example, little research exists describing the effects of toxic chemicals on chinook and sockeye salmon, but a wealth of information exists describing the effects of toxic chemicals on rainbow trout. Therefore, rainbow trout often served as a surrogate species to determine the effects of toxic pollutants on chinook and sockeye salmon.

1.3 *Organization of Toxic Pollutant Determinations for Invertebrates, Fish and Birds.*

For each of the chemicals receiving a high level of analysis, the determination section is organized as described here: a preliminary description of the chemical and criterion followed by an evaluation of recent research on each of the species of concern or their surrogates. The species are considered together in phylogenetic groups such as invertebrates, fish, birds, mammals, and plants. Within the evaluation for invertebrates and fish, sublethal, and lethal effects are evaluated separately. Determinations for the chemicals that received a minimal level of analysis are grouped together at the end of this section. For each of these chemicals, some background information is provided along with an effects determination.

Chemical Analysis for Metals

Three methods for partitioning metals in surface waters have historically been important in the development of water quality criteria. These are total recoverable, dissolved, and acid soluble. For total recoverable metals, a procedure using nitric and hydrochloric acids is given in section 4.1.4 of "Methods for Chemical Analysis of Water and Wastes, 1979 and 1983" (EPA, 1983). Analysts should be cautioned, however, that this digestion may not be adequate for all samples. For dissolved metals, samples are passed through a 0.45 micron membrane filter prior to acid preservation, digestion, and analysis. For acid soluble metals, the procedure is given in method 200.1 of "Methods for the Determination of Metals in Environmental Samples, 1991" (EPA, 1991a). This method requires the sample pH to be adjusted to 1.75 ± 0.1 , held for 16 hours and filtered through a 0.45 micron filter membrane prior to analysis. The acid soluble procedure is applicable for the determination of arsenic, cadmium, chromium, copper, and lead. Idaho's Water Quality Standards are measured as dissolved metals.

Where appropriate, the first paragraph of each subsection states the dissolved criterion (as adopted under Idaho's Water Quality Standards) and the corresponding total recoverable criterion. Laboratory testing most often uses total recoverable methods of determination for metals. Total recoverable criteria and dissolved criteria are related by a conversion factor promulgated by EPA on May 4, 1995 (EPA, 1995). These factors are listed in Table 250.07.a.1. To obtain a dissolved criterion from a total recoverable criterion, multiply the total recoverable criterion by the appropriate conversion factor.

2.0 Arsenic

The current Idaho Water Quality Standards specify criteria for dissolved acute and chronic arsenite, also known as trivalent arsenic (As), of 360 µg/L and 190 µg/L as acute and chronic criteria, respectively. The corresponding total recoverable criteria for arsenic are the same as the conversion factor for arsenic is 1.0. For pentavalent arsenic (arsenate), insufficient data is available to develop criteria, however the lowest-observed-effect levels (LOEL) measured as total recoverable for freshwater environments are 850 µg/L for acute exposures and 48 µg/L for long-term exposures (EPA, 1986b).

Arsenic occurs naturally in aquatic environments in trace amounts. Typical concentrations for background freshwater streams and rivers are less than 1 µg/L As (Moore and Ramamoorthy, 1984). The toxicity of arsenic can be altered by a number of factors including pH, Eh (redox potential), organic matter, phosphate content, suspended solids, presence of other toxicants, speciation of the chemical itself, and the duration of exposure to arsenic. Temperature has been shown to alter the toxicity of arsenic. In fish, tolerance of arsenic appears to increase with temperature; (McGeachy and Dixon, 1990) whereas in invertebrates the opposite is true (Bryant et al., 1985). Inorganic forms of arsenic are typically more toxic to aquatic species, particularly the more sensitive early life stages (Eisler, 1988a). While evidence does suggest that toxicity of arsenic can be altered by both temperature and phosphorus (two concerns for the mid-Snake River in Idaho), enough information to clearly characterize the relationship between arsenic toxicity and these two factors does not exist.

Arsenic does not readily bioconcentrate (an increase in concentration of a substance in relation to the concentration in the ambient environment) in aquatic species. It is typically water

soluble and does not combine with proteins. Planktivorous fish are more likely to concentrate arsenic than omnivorous or piscivorous fishes (Hunter, 1981). In 1995, Robinson et al. found no evidence of arsenic uptake or accumulation from water in both rainbow and brown trout.

Eisler (1988a) also found no evidence that biomagnification (a progressive increase in concentration from one trophic level to the next higher level) occurs in aquatic food chains. Aquatic invertebrates have been noted to accumulate arsenic more readily than fish, also an indication that biomagnification is unlikely (Spehar et al., 1980).

2.1 Fish

Sublethal effects

Sublethal effects including anemia, gallbladder inflammation, and liver degeneration, were observed at aquatic concentrations of 9.64 mg/L and dietary concentrations of 43.1-60 µg/g (Cockell et al., 1992; Woodward et al., 1994; and Rankin and Dixon, 1994). Studies of the effects of long-term arsenate exposures (11 weeks) found that rainbow trout were more tolerant of arsenic concentrations ranging from 5-36 mg/L at higher temperatures, in this case, 15°C versus 5°C (McGeachy and Dixon, 1990). However, a previous study conducted by the same investigators found rainbow trout to be more tolerant of acute arsenate exposures (1.5-18 mg/L for 144 hours) at lower temperatures, again, 15°C versus 5°C (McGeachy and Dixon, 1989). Arsenate is the most stable inorganic form of arsenic in aquatic systems (Eisler, 1988a). Oladimeji et al. (1984) found that arsenic in dietary concentrations of 10-30 mg/kg impaired rainbow trout growth in a dose-dependent manner and caused an inversely related decreases in hemoglobin levels. Pre-exposed fish are more tolerant of arsenic unlike the decreased tolerance seen in invertebrates (Dixon and Sprague, 1981). In other studies of sublethal effects of arsenic, adult and juvenile coho salmon were exposed to 300 µg/L As for 5 and 6 months. The adult coho salmon were exposed for 5 months and experienced some physiological alterations (EPA, 1985a). Parr-smolt coho exposed to the same arsenic concentration for 6 months experienced delayed onset of plasma thyroxine, transient reduction of gill sodium and potassium ATPase activity, and reduced successful seaward migration (Nichols et al., 1984). Therefore, EPA has determined that the acute and chronic arsenic criteria established by the Idaho Water Quality Standards are not likely to adversely affect the health and behavior of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Lethal effects

Various studies estimate LC50 values for salmonids to be between 3,000 and 167,000 µg/L As (Hamilton and Buhl, 1990; EPA, 1985a). Estimates of LC50's for arctic grayling were above 8940 µg/L As (Hamilton and Buhl, 1990). For rainbow trout embryos, an LC50 as low as 550 µg/L As after a month long exposure and an LC10 (concentration at which 10% of test organisms are killed) of 134 µg/L As after a 28-day exposure, indicate that salmonids may be more sensitive in early life stages (Birge et al., 1980). Even though the LC10 concentration is below Idaho's chronic arsenite criteria, EPA previously evaluated this study when it determined the current aquatic life criteria for arsenic. Because of issues with the procedures used in this study, EPA did not consider the results of this study as an acceptable basis for lowering the chronic arsenic criteria (C. Stephan, pers. comm., 1999). Therefore, EPA has determined that the acute and chronic arsenic criteria established by the Idaho Water Quality Standards is not likely to adversely affect the survival of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Summary

From the studies that investigated the effects of arsenic exposures on salmonids, it was determined that rainbow trout embryos may experience some mortality at arsenic concentrations less than those established by the chronic arsenic criteria established by the Idaho Water Quality Standards. Due to a lack of explanation of experimental procedures in the research reports, it is impossible to determine the quality of the results.

While EPA has determined that the approval of the acute and chronic aquatic life arsenic criteria established by the Idaho Water Quality Standards may have the potential to adversely affect the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout. An added level of protection is offered by the following:

The human health arsenic criterion of 50 ug/l is the applicable arsenic criteria in all waters of Idaho. This criterion is significantly lower and more conservative than the acute and chronic aquatic life criteria.

If a recreational use is modified or removed from a waterbody and the criteria become less stringent than 50 ug/l, Idaho is required to submit this revision to EPA for approval/disapproval action. If EPA proposes to approve this revision, the agency will then reinitiate consultation on that approval action .

In light of these currently effective measures, EPA has determined that the approval of the acute and chronic arsenic criteria (360 µg/L=acute, 190 µg/L=chronic) established by the Idaho Water Quality Standards is not likely to adversely affect the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

3.0 Cadmium

The current Idaho Water Quality Standards establish cadmium (Cd) criteria which are hardness dependent. At a hardness of 100 mg/L CaCO₃, the criteria are 3.7 µg/L and 1.0 µg/L for short-term and long-term exposures, respectively (See Table 250.07.a.3). Table 250.07.a.4 lists the criteria for hardness values used in the studies referenced in this report. The corresponding total recoverable criteria are 3.9 µg/L and 1.1 µg/L.

Hardness	Acute Criteria		Chronic Criteria	
	Total	Dissolved	Total	Dissolved
100 mg/L CaCO ₃	3.9 µg/L	3.7 µg/L	1.1 µg/L	1.0 µg/L

Hardness	Acute Criteria (total recoverable)	Chronic Criteria (total recoverable)
----------	------------------------------------	--------------------------------------

9.2 mg/L CaCO ₃	0.27 µg Cd/L	0.17 µg Cd/L
19.5 mg/L CaCO ₃	0.62 µg Cd/L	0.31 µg Cd/L
23 mg/L CaCO ₃	0.75 µg Cd/L	0.36 µg Cd/L
120 mg/L CaCO ₃	4.8 µg Cd/L	1.3 µg Cd/L
165 mg/L CaCO ₃	6.9 µg Cd/L	1.7 µg Cd/L

Cadmium naturally occurs in the aquatic environment, but is of no known biological use and is considered one of the most toxic metals. Concentrations of cadmium associated with background freshwater systems are estimated to range between 0.05-0.2 µg/L (Korte, 1983). While cadmium is released through natural processes, anthropogenic cadmium emissions have greatly increased its presence in the environment. In aquatic systems, cadmium quickly partitions to sediment, but is readily remobilized through a variety of chemical and biological processes (Currie et al., 1997). Toxicity of cadmium to aquatic organisms varies with the type and life stage of organisms, presence of other toxicants and the duration of exposure. Hardness affects cadmium toxicity as well. Møller et al. (1994) discovered that the toxicity of cadmium increases with increasing temperature (5-20°C) for one freshwater snail species at concentrations of 1-4 mg/L Cd. Currie et al. (1997) also found that cadmium can be transported from aquatic to terrestrial food webs by emerging insects. Cadmium removal from aquatic systems by aquatic insects has been shown to be significant: 1.3-3.9 g Cd/ year removed by insects out of a total 0.26-19.5 g Cd/ year removed. Pip (1992) found that cadmium concentration is negatively correlated with percent organic matter in natural environments. The presence of zinc and selenium have been shown to antagonize the toxic effects of cadmium. Other metals do not appear to compete with cadmium for receptors in aquatic organisms nor is there evidence for synergistic toxicity (Furness and Rainbow, 1990).

3.1 Bioconcentration and Biomagnification

Cadmium does not bioconcentrate (an increase in concentration of a substance in relation to the concentration in the ambient environment) significantly in fish species, but does tend to accumulate more readily in invertebrates. Pip (1992) found that snails accumulated a significantly higher level of cadmium when compared with the surrounding habitat. In a study of the uptake and depuration of cadmium in *Lymnea stagnalis*, Presing and Salanki (1993) found that the shells did not uptake cadmium, but the soft body tissues were saturated at concentrations of 200 µg Cd/g. The study exposed the snails to 0.1 mg Cd/L for four weeks then allowed an 8 week depuration period in clean freshwater. Even after 8 weeks in freshwater, Presing et al. still found significant cadmium concentrations in tissues. The average hardness for this experiment was 19.5 mg/L CaCO₃. Omnivorous and insectivorous predators tend to accumulate cadmium in their tissues more than piscivorous predators (Scheuhammer, 1991). After 7, 15, and 30 day exposures to the Po River in Italy, rainbow trout were found to have cadmium residues in the kidney, spleen, gills, muscle, and bone tissues ranging from 0.01-0.38 µg/g. The cadmium concentrations in the tissues increased with longer exposure durations. Cadmium concentrations characteristic of the Po River range from 0.07-0.26 µg/L (Camusso and Balestrini, 1995). Concentrations of cadmium in fish tissues reflect the bioavailability of cadmium in the water. It is unknown what effects may be associated with high tissue cadmium concentrations. No hardness information was reported for the Po River. The nature of this study as a field sample also prohibits the ability to determine whether

accumulation of cadmium resulted from exposure to the waters sampled during the study. Saiki et al. (1995) found no evidence of biomagnification (a progressive increase in concentration from one trophic level to the next higher level) in steelhead on the Upper Sacramento River. Eisler (1985a) also maintains that evidence for cadmium biomagnification suggests that only the lower trophic levels exhibit biomagnification.

Cadmium tends to form stable complexes with metallothionein (a sulfhydryl-rich protein). The resulting cadmium complexes have long half-lives and a tendency to accumulate with age in exposed organisms. As such, long lived species tend to be at a higher risk from chronic low-level dietary cadmium exposure.

3.2 Fish

Sublethal effects

Hontela et al. (1996) exposed juvenile steelhead to cadmium concentrations of 400-2,400 µg/L for durations ranging from 2 hours to 1 week. After only 2-4 hour exposures, the fish experienced a significant increase in plasma thyroxine levels. Plasma cortisol levels significantly increased after 96 hours. Both plasma thyroxine and glucose, as well as liver glucose were significantly lower after one week. The hardness for this experiment was 110 mg/L.

While the obviously adverse sublethal effects recorded by the previously described studies occurred at concentrations higher than the acute and chronic cadmium criteria established by the Idaho Water Quality Standards.

Based on the above information, EPA has determined that the acute and chronic cadmium criteria established by the Idaho Water Quality Standards are not likely to adversely affect the general health and behavior of Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Lethal effects

In chinook salmon, EPA (1985c) reported LC₅₀ values for 96 hour exposures ranged from 1.1-3.5 µg Cd/L dependent on the life stage tested (hardness=23). Sastry and Shukla (1994) found an LC₅₀ value of 11.2 mg/L for the freshwater fish, *Channa punctatus*, when exposed to this concentration for 96 hours at a hardness of 165 mg/L.

The reported lethal cadmium concentrations are above the cadmium criteria established by the Idaho Water Quality Standards. Therefore, EPA has determined that the acute and chronic cadmium criteria established by the Idaho Water Quality Standards are not likely to adversely affect the survival of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Summary

Some reviewers of this document have expressed concern for the accumulation of cadmium in salmonid prey. However, cadmium has not been shown to accumulate significantly in benthic invertebrates in field studies (Frag et al., 1998; Woodward et al., 1994). From the available information, EPA has determined that the approval of the acute and chronic cadmium

criteria (3.7 µg/L=acute, 1.0 µg/L= chronic, with hardness of 100 mg/L CaCO₃) established by the Idaho Water Quality Standards is not likely to adversely affect the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

4.0 Copper

The current Idaho Water Quality Standards establish hardness dependent criteria. At a water hardness of 100 mg/L CaCO₃, the copper (Cu) criteria are 17 µg/L for short-term exposures and 11 µg/L for long-term exposures (See Table 250.07.a.6). Corresponding total recoverable criteria are 18.0 µg/L and 12.0 µg/L for short-term and long-term exposures, respectively. Table 250.07.a.7 lists the criteria calculated for hardness values used in the studies referenced in this report.

Hardness	Acute Criteria		Chronic Criteria	
	Total	Dissolved	Total	Dissolved
100 mg/L CaCO ₃	18.0 µg Cu/L	17.28 µg/L	12.0 µg Cu/L	11.52 µg/L

Hardness	Acute Criteria (Total Recoverable)	Chronic Criteria (Total Recoverable)
12 mg/L CaCO ₃	2.4 µg/L	1.9 µg/L
13-13.3 mg/L CaCO ₃	2.6 µg/L	2.1 µg/L
20 mg/L CaCO ₃	3.9 µg/L	3.0 µg/L
25-30 mg/L CaCO ₃	4.8-5.7 µg/L	3.6-4.2 µg/L
28.4 mg/L CaCO ₃	5.4 µg/L	4.0 µg/L
29.69-32.72 mg/L CaCO ₃	5.6-6.2 µg/L	4.2-4.6 µg/L
35-55 mg/L CaCO ₃	6.6-10.0 µg/L	4.8-7.1 µg/L
41.3 mg/L CaCO ₃	7.7 µg/L	5.6 µg/L
46-47 mg/L CaCO ₃	8.5-8.7 µg/L	6.1-6.2 µg/L
50 mg/L CaCO ₃	9.2 µg/L	6.5 µg/L
100 mg/L CaCO ₃	18.0 µg/L	12.0 µg/L

Copper occurs naturally in the environment and is an essential element for most organisms as a component of some oxidative enzymes. Concentrations of copper associated with background freshwater systems are estimated to range between 0.5-1.0 µg/L (Moore and Ramamoorthy, 1984; Groth, 1971). While copper may form complexes with suspended organic matter, it will ultimately settle out of the water column and be deposited in the sediment (EPA,

1984). The toxicity of copper to aquatic organisms is dependent on the speciation of the chemical itself, water hardness and the type and life stage of the exposed organisms. Total organic content in the aquatic system may also decrease copper toxicity, while temperature may affect copper toxicity, although the relationship has yet to be clearly defined. The distinction between deficiency and toxicity for copper is small for organisms that do not have effective mechanisms to control the absorption of copper (e.g. fungi, algae, and invertebrates).

4.1 Bioconcentration and biomagnification

Copper is not strongly bioconcentrated (an increase in concentration of a substance in relation to the concentration in the ambient environment) in vertebrates, but is more strongly bioconcentrated in invertebrates. Bioconcentration factors (BCF's) reported in the EPA water quality criteria for copper (EPA, 1984) ranged from zero in bluegill (*Lepomis macrochirus*) to 22,600 in asiatic clams (*Corbicula fluminea*). The concentration of copper in the tissues of aquatic invertebrates is well-documented. In the Mediterranean snail, *Murex trunculus*, Nott and Nicolaidou (1994) found copper to be progressively accumulated with age in the visceral mass of the snail. Copper is bound strongly to sulfur complexes within the snail tissues and is thus less bioavailable to snail predators. Ying et al. (1993) found that the concentration of copper in the tissues of snails (*Polinices sordidus*) differed between organisms of the same species exposed to the same spiked sediment. The total body burden of copper increases with size and weight, and copper is capable of concentrating in the shell of the snail, *Lymnea stagnalis* (Pip, 1992).

In salmonids the accumulation of copper in muscle, kidney, and spleen tissues occurred at copper concentrations ranging from 0.52-3 µg/L in both seawater and freshwater (freshwater hardness=46-47 mg/L; Camusso and Balestrini, 1995; Peterson et al., 1991; Saiki et al., 1995). The concentrations of copper in fish tissues reflect the amount of bioavailable copper in the environment.

There is little information available concerning biomagnification (a progressive increase in concentration from one trophic level to the next higher level) of copper in aquatic food chains. Also, since the literature describing the effects of copper on birds or mammals are minimal, there is little information from which to quantify the biomagnification of copper. Baudo (1983), Wren et al. (1983) and Mance (1987) have all concluded that copper, along with zinc and cadmium do not biomagnify in the aquatic environment.

4.2 Fish

Sublethal effects

In fish, the toxicity of copper appears to be inversely related to the tendency of the metal to bind with the external gill surface via ionic interactions. In other words, a lower affinity of the gill surface to copper leads to a greater likelihood of disruption of intracellular processes, which may lead to gill dysfunction (Reid and McDonald, 1991). Some studies have examined the

disruption of gill processes by copper. For example, gill Na⁺, K⁺ ATPase activity in chinook parr was unaffected after an 18 hour exposure to stream water with elevated copper levels of 48 µg/L (hardness=13.3). With the same exposure, significant inhibition of gill Na⁺, K⁺ ATPase activity was observed in smolts. Significant increases in hematocrit and plasma glucose were also observed in both parr and smolts resulting from the same 18 hour exposure (Beckman and Zaugg, 1988). Divalent copper (Cu²⁺) totally suppressed gill Na⁺, K⁺ ATPase activity and produced significant cell damage, edema, mucus production, smoothing of apical membranes, swelling of tubular system and destruction of mitochondria in rainbow trout at concentrations of 0.1 and 1 mM CuCl₂, also 13.5 and 134.5 mg/L (Sola et al., 1995). A hardness value was not included in the description of this study. The investigators concluded from this study that bioavailable copper, such as divalent copper, immediately damages the hydromineral balance of rainbow trout and causes morphological modifications that are irreversible.

Carbello et al. (1995) also found rainbow trout to be more susceptible to the microbial parasite, *Saprolegnia parasitica*, at copper levels of 0.25 mg/L (hardness= 28.4 mg/L). Rainbow trout growth was significantly reduced and whole body copper concentrations elevated in fry after 20 days of exposure to copper levels of 4.6 µg/L; whereas 90 µg Cu/L caused a 45% reduction in mean weight after 40 days which was sustained through the end of the experiment at day 60 (hardness=25-30 mg/L; Marr et al., 1996). In another rainbow trout study, Munoz et al. (1991) observed rapid elevations of plasma cortisol, an indicator of stress, after a one hour exposure to 185 ng Cu/L (hardness=12 mg/L). The elevated plasma cortisol levels were maintained throughout the experiment's duration of 21 days.

It has been shown that copper concentrations at or below the established criteria of the Idaho Water Quality Standards may elevate plasma cortisol in rainbow trout. However, elevated cortisol levels are only an indicator of physiological stress. No corresponding adverse physiological effects were observed along with the elevated cortisol levels. Therefore, EPA has determined that the acute and chronic copper criteria established by the Idaho Water Quality Standards is not likely to adversely affect the general health and behavior of Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Lethal effects

For adult chinook, an LC50 value was determined as 10 µg Cu/L at a hardness of 13 mg/L (EPA, 1984). In steelhead smolts, Chapman (1978) found an LC10 of 7 µg Cu/L (hardness=22-25 mg/L). Buhl and Hamilton (1990) also examined copper effects on rainbow trout and calculated a 96-hour LC50 of 13.8 µg/L (average hardness=41.3 mg/L). Brook trout were exposed to copper for 24 hours by Drummond et al. (1973), resulting in an LC50 calculation of 9 µg/L (hardness = 44-46 mg/L).

From this evidence, EPA has determined that the acute and chronic copper criteria established by the Idaho Water Quality Standards are not likely to adversely affect the survival of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Summary

Elevated plasma cortisol was observed in salmonids and sturgeon after exposure to copper concentrations below the criteria established by the Idaho Water Quality Standards. However, it is important to note that adverse physiological effects were not observed along with these results. Therefore, EPA has determined that the approval of the acute and chronic copper criteria (17 µg/L=acute, 11 µg/L=chronic, hardness of 100 mg/L CaCO₃) established by

the Idaho Water Quality Standards is not likely to adversely affect the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

5.0 Lead

The current Idaho Water Quality Standards establish hardness dependent lead criteria. At a water hardness of 100 mg/L CaCO₃, the dissolved lead (Pb) criteria are 65 µg/L and 2.5 µg/L for short-term and long-term exposures, respectively (See Table 250.07.a.10). Corresponding total recoverable lead criteria are 62 µg/L and 3.2 µg/L for short-term and long-term exposures, respectively. Table 250.07.a.11 lists criteria calculated for the hardness values used in the studies referenced in this report.

Hardness	Acute Criteria		Chronic Criteria	
	Total	Dissolved	Total	Dissolved
100 mg/L CaCO ₃	65 µg/L	49.04 µg/L	3.2 µg/L	2.53 µg/L

Hardness	Acute Criteria	Chronic Criteria
8 mg/L CaCO ₃	3.28 µg/L	0.13 µg/L
10-20 mg/L CaCO ₃	4.35-10.52 µg/L	0.17-0.40 µg/L
26-31 mg/L CaCO ₃	14.70-18.38 µg/L	0.56-0.70 µg/L
28 mg/L CaCO ₃	16.15 µg/L	0.62 µg/L
35 mg/L CaCO ₃	21.46 µg/L	0.82 µg/L
42 mg/L CaCO ₃	27.06 µg/L	1.03 µg/L
46 mg/L CaCO ₃	30.38 µg/L	1.15 µg/L
61 mg/L CaCO ₃	43.52 µg/L	1.65 µg/L
100-106 mg/L CaCO ₃	81.65-87.93 µg/L	3.08-3.32 µg/L
101 mg/L CaCO ₃	82.69 µg/L	3.12 µg/L
128 mg/L CaCO ₃	111.79 µg/L	4.21 µg/L
135 mg/L CaCO ₃	119.63 µg/L	4.51 µg/L
139 mg/L CaCO ₃	124.16 µg/L	4.67 µg/L
290 mg/L CaCO ₃	316.64 µg/L	11.86 µg/L
353 mg/L CaCO ₃	406.67 µg/L	15.21 µg/L

Lead is a naturally occurring, ubiquitous compound that can be found in rocks, soils, water, plants, animals, and air. Concentrations of lead associated with background freshwater

systems are estimated to be <3.0 µg/L (Moore and Ramamoorthy, 1984). It is soluble in water and its bioavailability increases in environments with low pH, low organic content, and low metal salt content (Eisler, 1988b). Lead is most often precipitated to sediments in aqueous environments. The toxicity of lead varies with water hardness. As hardness increases, lead precipitates, and becomes less bioavailable to aquatic organisms. Adsorption of lead by aquatic animals is affected by the age, gender and diet of the organism, as well as the particle size, chemical species and presence of other compounds in the water (Eisler, 1988b; Hamir et al., 1982). Aquatic organisms are sensitive to lead are affected more strongly by dissolved rather than total lead. Likewise, the toxicity of lead is increased when it forms organolead compounds and when environmental conditions consist of high temperature and low pH. Animals are also more sensitive at younger life stages and when exposure durations are greater.

5.1 Bioconcentration and Biomagnification

Lead has been shown to bioconcentrate (an increase in concentration in relation to the ambient concentration) in aquatic species. Invertebrates tend to have higher bioconcentration factors (BCF) than vertebrates. For example, the BCF for the freshwater snail, *Lymnaea palustris*, is 1,700 and the BCF for the blue mussel, *Mytilus edulis*, is 2,570. In the freshwater snail, *Physa integra*, tissue concentration changes were correlated with changes in dissolved lead in the water column, but not with changes in the amount of lead found in substrate. Similarly, *Campeloma decisum* (sub-tropical freshwater snail) had lower tissue concentrations than the substrate even though the organism was associated closely with contaminated sediments. Lead was found to accumulate in the ganglia of freshwater snails (*Lymnaea stagnalis*). In vertebrates, such as brook trout embryos, the BCF is 42 (Eisler, 1988b). Inorganic lead is poorly accumulated in fish. Organic lead compounds such as tetraalkyllead are more toxic than smaller compounds such as trialkyllead. This may be due to the rapid accumulation of tetraalkyllead by fish (Hodson et al., 1984). BCFs decrease as waterborne lead concentrations increase, thus suggesting accelerated depuration or saturation of uptake mechanisms (Hodson et al., 1984). Exposures of rainbow trout to 3.5-51 µg/L (hardness = 135) tetramethyllead from 7 days to two weeks resulted in rapid accumulation of lead. However, once the fish were removed to clean water, lead was initially removed rapidly from organs followed by a slower release until base levels were reached. An increase in dietary calcium of 0-8.4 mg/kg (hardness=8 mg/L) reduced the uptake of waterborne lead in coho salmon, possibly due to interactions with gill membrane permeability (Hodson et al., 1984).

In vertebrates, lead concentrations tend to increase with age and localize in hard tissues such as bone or teeth. Lead residues have been shown to be greater in older birds, sexually mature females, and in birds that have ingested lead shot pellets. While lead has been shown to concentrate in aquatic species, there is little evidence for biomagnification (a progressive increase in concentration from one trophic level to the next higher level; Eisler, 1988b).

5.2 Fish

Sublethal effects

Adult trout exposed to lead as part of their diet (0.86-1.77 µg/g) for 21 days experienced increased scale loss and accumulation of lead in their guts. When exposed to lead for the same length of time through the water column (4.3-6.4 µg/L, hardness=100-106), trout experienced scale loss, reduced survival, and accumulation in gill and kidney tissues. A combination of dietary and water-borne lead exposure at the same concentrations resulted in lipid peroxidation in kidneys of adults and a decrease in the whole body potassium of juveniles (Faraq et al., 1994). Other documented sublethal responses include hematological, neurological, teratogenic, growth, and histological effects at lead concentrations of 8-119 µg/L and >1000 µg/L (hardness=42-353) during exposures from 3-16 weeks (Hodson et al., 1984).

Concentrations of lead >10 µg/L (hardness=135) caused long-term effects such as: spinal curvature; anemia; caudal chromatophore degeneration (black tail); caudal fin degeneration; destruction of spinal neurons; ALAD inhibition in blood cells, spleen, liver, and renal tissues; reduced swimming ability; destruction of respiratory epithelium; elevated lead in blood, bone and kidney; muscular atrophy and paralysis; inhibition of growth; retardation of maturity; changes in blood chemistry; testicular and ovarian histopathology; and even death (EPA, 1985d). The effects of lead increase under rapid growth conditions as illustrated by the increase of the rate of intoxication by lead increased with growth rate, but not fish size (Hodson et al., 1982). In sexually maturing male rainbow trout exposed to 10 µg/L (hardness=128) for 12 days during spermatogenesis, spermatogonial cysts increased, spermatocytes declined, and the sensitivity of the reproductive cycle was expressed as the transformation of spermatogonia to spermatocytes decreased (Ruby et al., 1993a). In whitefish (*Coregonus* sp.) from contaminated lakes (0.5-4.5 µg Pb/L, hardness=10-20) γ -aminolevulinic acid (ALAD) activity was inhibited up to 88% when compared to fish from uncontaminated lakes. Inhibition of ALAD activity leads to problems with hemoglobin synthesis that can result in anemia. Higher blood glucose levels and lower plasma sodium content were also found in fish taken from lead contaminated lakes (Haux et al., 1986).

Spinal deformities in rainbow trout resulted from exposure to lead concentrations of 18.9 and 101.8 µg/L (hardness=28 and 35, respectively). In juvenile rainbow trout, ALAD activity was inhibited. Red blood cells and blood iron content were also affected after 28 days exposed to lead levels of 13 µg/L (hardness=135). At 120 µg Pb/L (hardness=135) for 32 weeks, 30% of juvenile rainbow trout exposed had black tails caused by degeneration of caudal chromatophores (EPA, 1985d).

Based on the information presented on this report, EPA has determined that the acute and chronic lead criteria established by the Idaho Water Quality Standards are not likely to adversely affect the general health and behavior of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Lethal effects

At lethal concentrations (543 µg/L; H=135 mg/L), lead can cause increased mucus formation in rainbow trout where the excess mucus coagulates over the fish's entire body, most prominently the gills. The mucus interferes with respiratory function and results in the death of the fish by anoxia (Hodson et al., 1982).

Many studies have determined LC50 values for various life stages of rainbow trout. The 32-week LC50 value for embryo/larval stages was 220 µg/L (hardness=101). Two month old fry had an LC50 of 8,000 µg/L (hardness=82-132 mg/L). For juvenile rainbow trout, the 21-day LC50 value was calculated as 2,400 µg/L (hardness=135). Finally, in adults, LC50 values

ranged from 1,170 µg/L to 471,000 µg/L to 542,000 µg/L depending upon the hardness values: 28, 353, 290, respectively (EPA, 1985d).

All of the above studies found that survival of rainbow trout was not affected at levels allowable under the lead criteria established by the Idaho Water Quality Standards. Therefore, from the information available, EPA has determined that the acute and chronic lead criteria established by the Idaho Water Quality Standards are not likely to adversely affect the survival of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Summary

From the available information, EPA has determined that the approval of the acute and chronic lead criteria (65 µg/L=acute, 2.5 µg/L=chronic, hardness of 100 mg/L CaCO₃) established by the Idaho Water Quality Standards is not likely to adversely affect the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

6.0 Mercury

The current Idaho Water Quality Standards establish an acute criterion for dissolved mercury as 2.1 µg/L. The chronic criterion established for dissolved mercury is 0.012 µg/L. The total recoverable acute and chronic total mercury criteria are 2.4 µg/L and 0.012 µg/L, respectively.

Mercury is cycled through the environment through an atmospheric-oceanic exchange. This cycling is facilitated by the volatility of the metallic form of mercury. Natural bacterial transformation of mercury results in stable, lipid soluble alkylated compounds such as methylmercury (Beijer and Jennelov, 1979). Methylmercury is highly toxic to mammals and can interfere with thiol metabolism resulting in mitotic disturbances. This compound can also irreversibly destroy the neurons of the central nervous system (Clarkson et al., 1984). While mercury does occur naturally in small amounts in aquatic environments, the cycling of mercury prolongs the influence of man-made mercury compounds (Hudson et al., 1995). In sediments, mercury is usually found in its inorganic forms, but aquatic environments are a major source of methylmercury (EPA, 1985e). In background freshwater systems, mercury occurs naturally at concentrations of 0.02-0.1 µg/L (Moore and Ramamoorthy, 1984).

6.1 Bioconcentration and Biomagnification

Mercury has been shown to bioconcentrate (an increase in the concentration of a substance in relation to the concentration in the ambient environment) in a variety of aquatic organisms. Fish have been shown to concentrate mercury as methylmercury even when they are exposed to inorganic mercury. Aquatic predators face the greatest danger of bioconcentrating mercury, and thus their tissue concentrations best reflect the amount of mercury available to aquatic organisms in the environment. Fish, such as rainbow trout, have been found to accumulate mercury in the form of methylmercury at aquatic concentrations as low as 1.38 ng/L (Ponce and Bloom, 1991). Temperature has been shown to affect the magnitude of bioconcentration factors (BCF) in aquatic snails. In the freshwater gastropod, *Viviparus georgianus*, BCFs were observed to increase with temperature in snails from three different age classes. Similar effects were also observed for the medium sized pelecypods,

Elliptia complanata. For animals between 74-86 mm in length, BCF increased with increasing temperature (Tessier et al., 1994).

Some evidence supports the biomagnification (a progressive increase in concentration from one trophic level to the next higher level) of mercury in aquatic food chains. In a comparison of benthic feeding fish and fish that feed on plankton, invertebrates and vertebrates, the greatest mercury concentrations were found in piscivorous fishes. The authors of this study concluded that mercury content in fish increased with higher trophic levels (Wren and MacCrimmon, 1986).

6.2 Fish

The effects of exposure to mercury have been studied extensively in fish. The uptake of mercury is proportional to the concentration of mercury in water. However, the uptake of methylmercury in fish increases with increased water temperature, exposure concentration, 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).

Sublethal effects

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. Dietary exposures of 16-48 µg/g over a period of 84-270 days adversely affected growth, skin color, weight, and behavior in rainbow trout. As little as 7.9 µg/g affected the survival and behavior of walleye. Long-term exposures to waterborne concentrations of mercury ranging from 0.1-0.2 µg/L also affected behavior, reproduction and survival of fish, specifically fathead minnows (Weiner and Spry, 1996).

EPA has determined that the acute and chronic mercury criteria established by the Idaho Water Quality Standards is not likely to adversely affect the general health and behavior of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Lethal effects

EPA (1985g) reported LC50 values for fish exposed to inorganic mercury which ranged between 150-420 µg/L. For organic mercury, LC50s range from 24-84 µg/L. In both cases, the LC50s reported by EPA (1985g) were determined under flow-through conditions. In a study of the chronic toxicity of mercury chloride (HgCl₂) to rainbow trout, Niimi and Kissoon (1994) exposed subadults to 64 µg/L HgCl₂ until the fish died. The average time to death was 58 days at this concentration. At 426 µg/L, the mean time to death was 1 day. Niimi and Kissoon (1994) also conducted a similar experiment using methyl mercury chloride exposures. The investigators found that fish lived more than 100 days when exposed to 4 µg/L, but lived an average of only 2 days when exposed to 34 µg/L methylmercury chloride. The toxicity of methylmercury was also examined by Devlin and Mottet (1992). Coho salmon embryos were exposed to methylmercury at concentrations of 6, 13, 29, 62, and 139 µg/L methylmercury at 10°C for 48 days. The resulting LC50 values ranged from 54-71 µg/L.

All of the mercury concentrations found to affect the survival of fish are well above the mercury criteria established by the Idaho Water Quality Standards. Therefore, EPA has

determined that the acute mercury criterion established by the Idaho Water Quality Standards is not likely to adversely affect the survival of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Summary

From the information available regarding the effects of mercury exposure on both the health and survival of fish species, EPA has determined that the approval of the acute and chronic mercury criteria (2.1 µg/L=acute, 0.012 µg/L=chronic) established by the Idaho Water Quality Standards is not likely to adversely affect the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

7.0 Selenium

The current Idaho Water Quality Standards establish an acute criterion of 20 µg/L and a chronic criterion of 5.0 µg/L for total recoverable selenium. Selenium is not measured as dissolved under the Idaho Water Quality Standards.

Selenium occurs naturally in aquatic environments in trace amounts. While selenium is ubiquitous in the earth's crust, only trace levels occur in aquatic environments. Selenium enters aquatic habitats from a number of anthropogenic and natural sources. Elevated levels in aquatic systems are found in regions where soil is selenium-rich or where soils are extensively irrigated (Dobbs et al., 1996). As an essential micronutrient, selenium is used by animals for normal cell functions. However, the difference between useful amounts of selenium and toxic amounts is small. The toxic effects of selenium range from physical malformations during embryonic development to sterility and death (Lemly and Smith, 1987). Selenium has also been shown to protect some species from the toxicity of other chemicals. For example, the toxicity of cadmium in freshwater snails is inhibited by selenium and antagonizes mercury toxicity in rainbow trout (Eisler, 1985b).

The behavior of selenium in biological systems is complex. Selenium is a metalloid that exists in three oxidation states in water: selenide (-2), selenite (+4) and selenate (+6). The toxicity of selenium varies with its chemical species. Organic and reduced forms of selenium (e.g. seleno-methionine and selenite) are generally more toxic and will bioaccumulate (Besser et al., 1993; Kiffney and Knight, 1990). Toxicity also varies with the species exposed. Species at higher trophic levels, such as piscivorous fish and birds, are affected by the lowest concentrations of selenium. It appears that long term, low level exposures from water or food have the greatest effect on aquatic organisms (Lemly, 1985).

7.1 Bioconcentration and Biomagnification

Bioconcentration of selenium may be modified by water temperature, age of receptor organism, organ, and tissue specificity and mode of administration (Eisler, 1985b). Fish bioconcentrate selenium in their tissues with particularly high concentrations observed in ovaries when compared to muscle tissues (Lemly, 1985; Hamilton et al., 1990) and milt (Hamilton and Weddall, 1994). Reproductive failure is often associated with bioaccumulation of

selenium in ovaries and offspring (Hamilton et al., 1990). Selenium that is bioconcentrated appears to occur in its most harmful concentrations in predator species such as mallard ducks or chinook salmon (Hamilton et al., 1990). At concentrations greater than 0.002-0.005 mg/L in water, selenium can be bioconcentrated and cause significant toxicity and reproductive failure in fish (Hermanutz et al., 1992). Bioconcentration factors (BCFs) in rainbow trout range from 2-20 after exposure to 220-410 µg/L selenium. The magnitude of the BCFs appeared to be inversely related to exposure concentration (Adams and Johnson, 1981). The transformation of selenium to organoselenium increases the bioconcentration of the compounds in fish ovaries resulting in significant pathology and reproductive failure (Srivastava and Srivastava, 1994; Sorenson and Bauer, 1983; Baumann and Gillespie, 1986).

Biomagnification (a progressive increase in concentration from one trophic level to the next higher level) of selenium has also been well documented. The magnitude of the biomagnification ranges from 2-6 times between producers and lower consumers (Lemly and Smith, 1987). Piscivorous fish accumulate the highest levels of selenium and are generally one of the first organisms affected by selenium exposure, followed by planktivores and omnivores (Lemly, 1985).

7.2 Fish

Studies have shown that selenium negatively affects aquatic organisms at concentrations between 10-100 µg/L (EPA, 1980i). Fish appear to be sensitive to selenium toxicity under conditions of long-term exposure from both water and dietary sources. Waterborne selenium is depurated in fish via a passive excretion pathway, while dietary selenium is excreted more actively. The half-life of selenium is inversely proportional to dietary loading. Inorganic selenium absorbed from water is stored in fish as inorganic selenium. However, inorganic selenium absorbed from the diet is transformed by the liver to an organic form that is more toxic, but can be excreted easily (Hodson et al., 1984b). Selenium taken up from water is absorbed across the gills and taken directly to all tissues except the liver. The liver receives its blood supply via a portal system from the gut. Dietary selenium is taken up through the gut, thus passing through the liver first. The tissue distribution of selenium within fish is a function of the loading rate, but not the source of selenium (Hodson and Hilton, 1983).

Due to the sensitivity of fish to long-term low concentration exposures of selenium, the indications of relative sensitivity to waterborne selenium may become reversed when comparing acute and chronic studies. For this reason, comparisons of acute and chronic sensitivities of fish to selenium should be interpreted with caution (Lemly, 1985). Hermanutz et al. (1992) also suggest that the estimation of effects using studies of waterborne exposure exclusively may underestimate the danger of selenium exposure to fish. The optimum dietary selenium level in rainbow trout is estimated to be between 0.15-0.38 µg/g by Hilton et al. (1980). However, trout appear to be able to accommodate excess dietary selenium in the short term using both behavioral and physiological adaptations.

Sublethal effects

Studies have shown that exposure to selenium can reduce fish growth particularly weight and, to a lesser extent, length (Albers et al., 1996; Green and Albers, 1997; Hamilton et al., 1990). At selenium concentrations of 250 ppb in water, rainbow trout fry growth was

reduced following a 21-day exposure (Eisler, 1985b). Weight was reduced by 29-70% in fall-run chinook fed greater than 18.2 µg/g for 90 days (Hamilton et al., 1990). Concentrations of 35.4 µg/L for 60-days and 9.6 µg/L for 90-days reduced chinook salmon body weight and survival (Hamilton et al., 1986).

Selenium exposures can also reduce red blood cell volumes and cellular blood iron content in rainbow trout juveniles at concentrations greater than or equal to 53 and 16 µg/L, respectively, after 44 weeks. Hatchability of eggs was affected at concentrations as low as 16 µg/L in the same experiment. A slight decrease in the time to hatch was observed at 4.4 µg/L, however the results were not statistically significant when compared to controls (Hodson et al., 1980). Selenium also affects the immune responses of fish by influencing the activity of glutathione peroxidase (GPX). GPX is an antioxidant that protects cellular membranes and organelles from peroxidative damage that may be caused by superoxide radicals (Felton et al., 1990). Selenium concentrations of 13 µg/L for 6 weeks reduced smolting success of chinook salmon (Hamilton et al., 1986).

At concentrations of 47-50 ppb (µg/L) in water, selenium exposures were associated with anemia and reduced hatch of rainbow trout (Eisler, 1985b). At 47 µg/L over 41 days, investigators observed reduced hatch of eyed embryos of rainbow trout (EPA, 1980i). Significant deformities resulted from exposure of rainbow trout eggs to 80 µg/L selenium (Lemly and Smith, 1987).

Due to the ability of fish and invertebrates to bioconcentrate selenium, fish can be exposed to harmful concentrations of selenium via diet even when water concentrations are low. In chinook salmon, specifically, swim-up larvae and fingerlings, 3.2 µg/g selenium in the diet adversely affected growth. Using a bioaccumulation factor of 1,800 for aquatic invertebrates (Pease et al., 1992), it would be possible to obtain a dietary concentration of 3.2 µg/g at a water concentration as low as 1.8 µg/L. Lemly (1996) set forth a limit of 2 µg/L on a chronic basis as hazardous to the health and survival of fish. Selenium concentrations at low levels near this limit would primarily act through bioaccumulation.

The results of research examining the sublethal effects of selenium on trout indicate that fish are adversely affected by selenium concentrations in water that are above both the acute and chronic selenium criteria established by the Idaho Water Quality Standards. It is possible however, that water concentrations lower than the chronic selenium criteria may result in dietary concentrations of selenium that may be harmful to fish species.

Therefore, due to the potential adverse effects due to bioaccumulative exposures to selenium, EPA has determined that the chronic selenium criterion established by the Idaho Water Quality Standards is likely to adversely affect the general health and behavior of the Snake River sockeye and chinook salmon, Snake River steelhead, bull trout, and Kootenai River white sturgeon. From the available information, EPA has determined that the acute selenium criteria established by the Idaho Water Quality Standards are not likely to adversely affect the general health and behavior of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Lethal effects

Dietary concentrations as low as 13 µg/g caused elevated mortality, reduced feeding, slower growth, higher feed-to-weight gain ratios and liver paleness in trout within 4 weeks. Dying fish were reported to swim in uncoordinated spirals and were noted as being oblivious to

physical obstacles (Hilton et al., 1980). In rainbow trout, 96-hour and 9-day LC₅₀s were determined to be 8.1 mg/L and 6.5 mg/L, respectively. After 44 weeks, significant mortality was observed in rainbow trout eyed eggs at concentrations greater than or equal to 25 µg/L (Hodson et al., 1980). In fall-run chinook salmon, reduced survival was observed at 35.4 µg/g dietary selenium for 60 days and greater than 9.6 µg/g dietary selenium for 90 days (Hamilton et al., 1990). Long-term exposures (44 weeks) to 130 µg/L selenium caused elevated mortality rates in rainbow trout along with increased incidence of deformities at concentrations as low as 60 µg/L (Hodson et al., 1984b).

Lethal effects of selenium can vary among and within species. For example, when Puget Sound wild and hatchery reared coho salmon were compared, wild fish survival rates were found to be 1.5-2.0 times higher than those of hatchery reared fish exposed to the same selenium contaminated water. Selenium residues were also higher in wild fish versus hatchery reared fish (Felton et al., 1990). In chinook salmon fry, exposures to 17 µg/L for 30 days caused a significant increase in mortality (Hamilton et al., 1986). The 43-day LC₅₀ for chinook larvae and the 48-day LC₅₀ for chinook fry was 160 µg/L (Eisler, 1985b; Lemly and Smith, 1987). In rainbow trout, the 9-day LC₅₀ was estimated to range between 5,400-7,000 µg/L (EPA, 1980i). The 48-day LC₅₀ for rainbow trout larvae was determined to be 500 µg/L and significant mortality was observed at 80 µg/L over a 12 month exposure (Lemly and Smith, 1987). In bull trout, the LC₅₀ was estimated to be 10,200 µg/L (EPA, 1980i).

From the information presented regarding the lethal effects of selenium on salmon species, EPA has determined that the acute and chronic selenium criteria established by the Idaho Water Quality Standards are not likely to adversely affect survival of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Summary

EPA has determined that the approval of the acute selenium criterion (20 µg/L=acute) established by the Idaho Water Quality Standards is not likely to adversely affect the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout. Furthermore, EPA has determined that the approval of the chronic selenium criterion (5.0 µg/L=chronic) established by the Idaho Water Quality Standards may be likely to adversely affect the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

8.0 Zinc

The current Idaho Water Quality Standards establish hardness dependent zinc criteria . At a hardness of 100 mg/L CaCO₃, the acute and chronic criteria are 110 µg/L and 100 µg/L, respectively. The corresponding total recoverable criteria are 120µg/L and 110 µg/L at a water hardness of 100 mg/L CaCO₃ for short-term and long-term exposures, respectively (Table 250.07.a.15). Table 250.07.a.16 lists criteria calculated at the hardness levels used in the studies referenced in this report.

Table 250.07.a.15. Idaho Zinc Water Quality Criteria				
Hardness	Acute Criteria		Chronic Criteria	
	Total	Dissolved	Total	Dissolved

100 mg/L CaCO ₃	120 µg/L	117.36 µg/L	110 µg/L	108.46 µg/L
----------------------------	----------	-------------	----------	-------------

Table 250.07.a.16. Idaho Water Quality Criteria for Zinc Calculated for Referenced Hardness Values and Total Recoverable Analysis		
Hardness	Acute Criteria (Total Recoverable)	Chronic Criteria (Total Recoverable)
2 mg/L CaCO ₃	4.3 µg/L	3.9 µg/L
2.7 mg/L CaCO ₃	5.5 µg/L	5.0 µg/L
15 mg/L CaCO ₃	23 µg/L	21 µg/L
20 mg/L CaCO ₃	30 µg/L	27 µg/L
41.3 mg/L CaCO ₃	55 µg/L	50 µg/L
60 mg/L CaCO ₃	76 µg/L	69 µg/L
100 mg/L CaCO ₃	120 µg/L	110 µg/L
170 mg/L CaCO ₃	180 µg/L	170 µg/L

Zinc is naturally introduced into aquatic systems, usually via leaching from igneous rocks. Concentrations of zinc associated with background freshwater systems are estimated to range between 0.5-15 µg/L (Moore and Ramamoorthy, 1984; Groth, 1971). Most of this naturally introduced zinc is adsorbed to sediments, however a small amount remains in the water, predominantly in the form of the free Zn²⁺ ion. Release of zinc from sediment is enhanced by the combination of high dissolved oxygen, low salinity, and low pH (Eisler, 1993). All life forms require zinc as an essential element, however aquatic animals tend to accumulate excess zinc which can result in growth retardation, hyperchromic anemia, and defective bone mineralization. Zinc primarily affects zinc-dependent enzymes regulating RNA and DNA. Zinc also increases the numbers of metallothioneins, low molecular weight proteins involved in zinc homeostasis. In mammals and birds, the pancreas and bone seem to be the primary targets of zinc toxicity, whereas in fish, it is the gill epithelium (Eisler, 1993). Toxicity of zinc to aquatic organisms is dependent upon the type and life stage of organism as well as the concentrations of other chemicals in the water. Substances such as calcium and magnesium can reduce zinc toxicity. Other compounds such as cadmium, copper, iron, and molybdenum also interact antagonistically with zinc (Hammond and Beliles, 1980). Zinc ions and other toxic species affect aquatic organisms most severely in environments characterized by low pH, low alkalinity, low dissolved oxygen and elevated temperature (Eisler, 1993). However, there is some evidence that fish acclimate to elevated temperature are more tolerant of zinc toxicity. An increase in temperature during exposure to zinc appears to cause increased sensitivity to zinc as a result of temperature stress, while fish that have acclimated to higher temperatures (no temperature stress) are less sensitive to zinc (Hodson and Sprague, 1975).

8.1 *Bioconcentration and Biomagnification*

Because zinc combines with biomolecules in target species and most of these species accumulate more than they need for normal metabolism, data showing bioconcentration factors

for target receptors may be misleading. Bioconcentration (an increase concentration of a substance in relation to the concentration in the ambient environment) is also dependent on the target organism of interest. Bioconcentration factors (BCF's) reported in the EPA water quality criteria for zinc (EPA, 1987b) ranged from 51 in Atlantic salmon (*Salmo salar*) to 1,130 for the mayfly (*Ephemerella grandis*).

Little to no evidence exists indicating the successive biomagnification (a progressive increase in concentration from one trophic level to the next higher level) of zinc in tissues of fish and avian receptors. This assumption is based on several factors. First, existing BCF data (EPA, 1987b) shows that the greatest BCF was seen in mayflies while the least was found in Atlantic salmon. This trend was also seen in Elder and Collins (1991) who showed that molluscs accumulated more zinc than the fish who feed off of these molluscs. Furthermore, the existing zinc toxicity data for birds is predominantly based on force feeding studies of zinc shot or dietary supplements (Eisler, 1993).

8.2 Fish

Sublethal effects

Coho salmon and cutthroat trout fry were observed to avoid water contaminated with zinc at nominal concentrations ranging from 6.54-28 µg/L at hardnesses of 15-100 mg/L CaCO₃ (Rehnberg and Schreck, 1986; Woodward et al., 1997). However, the significance of the zinc avoidance in the Rehnberg and Schreck study may have been due to small sample size as higher zinc concentrations did not deter juvenile coho salmon. In the Woodward study, it should be noted that the measured zinc concentrations in waters avoided by cutthroat trout ranged from 66-74 µg/L, much higher than the nominal concentrations (Hardness = 15-25 mg/L CaCO₃). Therefore, EPA has determined that the acute and chronic zinc criteria established by the Idaho Water Quality Standards are not likely to adversely affect the general health and behavior of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Lethal effects

Sayer et al. (1989) saw 75-95% mortality for brown trout yolk sac fry at exposures to concentrations of 4.9-19.6 µg/L Zn for 30 days. This mortality occurred in waters with a hardness of only 2 mg/L CaCO₃ and low pH, not typical of waters in Idaho. For steelhead, Buhl and Hamilton (1990) observed LC₅₀s of 169-215 µg/L Zn at a hardness of 41.3 mg/L CaCO₃. Similarly, Buhl and Hamilton found LC₅₀ values ranging between 112-168 µg/L Zn at a hardness of 41.3 mg/L CaCO₃ for arctic grayling juveniles, (*Thymallus arcticus*). An LC₅₄ was obtained when rainbow trout larvae and alevins were exposed to 10 µg/L zinc for 28 days (hardness = 2.7 mg/L; Affleck, 1952). When the Idaho Water Quality Standards for zinc recalculated for comparable hardness values (see Table 250.07.a.16), concentrations of zinc that cause lethal effects in salmonid species are above those allowed by the Idaho Water Quality Standards. Therefore, EPA has determined that both the acute and chronic zinc criteria established by the Idaho Water Quality Standards are not likely to adversely affect the survival of the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

Summary

EPA has determined that the approval of the acute and chronic zinc criteria (110 µg/L=acute, 100 µg/L=chronic, hardness of 100 mg/L CaCO₃) established by the Idaho Water Quality Standards is not likely to adversely affect the Snake River sockeye and chinook salmon, Snake River steelhead, and bull trout.

9.0 Analysis of Effects of Numeric Criteria for Toxic Pollutants to White Sturgeon

Kootenai River White Sturgeon

A literature search yielded very limited information on effects of toxicants to white sturgeon. An accepted practice in this situation is to use a species for which there is adequate toxicity information as a surrogate for the species in question (EPA, 1995). It is not difficult to state the likelihood of adverse effects with relatively good certainty if we apply interspecies correlation models (e.g., rainbow trout vs. shortnose sturgeon; see below) as an estimate of toxicity to the sturgeon family. However, the available cold water species surrogate, rainbow trout, is very different from white sturgeon in terms of life history, habitat use, and feeding strategy. For example, the long lives of adult sturgeon may result in bioaccumulation of persistent toxicants that could be passed to offspring (Bennett and Farrell, 1998). However, other species of fish, such as catfish, have more similar life histories, but they occur in warm water. Used in combination, this data can offer a good estimate of toxicity of the criteria to sturgeon.

Limited studies that have been conducted on sturgeon species suggests that they have some resistance to certain toxicological effects that is variable compared to other species. Bennett and Farrell (1998) concluded that juvenile white sturgeon lie within the sensitivity range of other juvenile fish for chlorinated phenols. But, white sturgeon fry appear to have greater sensitivity to didecyldimethylammonium chloride than other fish species. In a study of early growth of coho and Masu salmon and Siberian sturgeon related to toxic conditions, all three species exhibited similar growth impairment disturbance (Glubokov, 1990). Of the three species, Masu salmon were the most sensitive to copper and phenol. Variation in the toxicoresistance among aquatic species and among chemicals, with no single species always being the most sensitive has been well documented (Mayer and Ellersieck, 1986). However, Mayer et al. (1987) found that interspecies correlation models for acute toxicity were highly dependable in estimating toxicity for species with unknown sensitivity to chemicals from acute toxicity values for common test fishes (rainbow trout, fathead minnows, bluegills). Correlations are good among many phyletic families and a variety of chemicals (Doherty, 1983), but with pesticides, correlations are best within families or closely related families (e.g., fishes) as reported by Kenaga (1978), LeBlanc (1984), Mayer et. al. (1987), and Suter and Vaughan (1985).

Rather than taking the default approach and assigning a “likely to adversely affect” determination for white sturgeon, we have chosen to evaluate the proposed standards by examining toxicity data for a variety of fish species, including cold water species (e.g. salmonids) and benthic species (e.g. catfish). If the proposed standards are protective of a variety of fish species, we can assume that the standards will also adequately protect white sturgeon for the following reasons: 1) the proposed standards are below the limits for other fish species and 2) the limited data available show that sturgeon have variable sensitivity compared to other species (i.e. they are not consistently more sensitive than other species). Thus,

standards that protect other fish species will adequately protect white sturgeon. This has recently been supported in research led by F.L. Mayer (Dwyer et al. 1995, Dwyer et al. 1999a, 1999b, Mayer et al. 2000). Acute toxicity tests with five chemicals (carbaryl, copper, 4-nonylphenol, pentachlorophenol, permethrin) and 19 fish species (rainbow trout, fathead minnows, sheepshead minnows, and 16 endangered fishes) indicated that salmonid data are generally protective of sturgeons (shortnose sturgeon). Also, interspecies correlations with rainbow trout or fathead minnows are highly predictive for acute toxicity with the shortnose sturgeon.

9.1 Arsenic

The discussion of arsenic effects on salmonids is discussed in the acute and chronic cadmium criteria section of this Biological Assessment. Based on EPA's review of the literature, the Agency has determined that approval of Idaho's acute arsenic criterion is not likely to adversely affect endangered salmonids. Some lethal effects may occur at arsenic levels permitted by the chronic criterion, however, the human health arsenic criterion of 50 µg/L will apply in all Idaho surface waters. This number has been shown to be protective of endangered salmonid species.

Sublethal effects

In catfish, sublethal effects including impaired growth and altered histopathology occur at arsenic concentrations of 1,500-15,000 µg/L (Clemens and Sneed, 1959; Gupta and Chakrabarti, 1993; Shukla et al., 1985; Shukla et al. 1987). Green sunfish experienced sublethal effects such as bioaccumulation of arsenic and histopathological changes when exposed to 31,700-62,500 µg/L arsenic (Sorensen, 1976).

Lethal effects

Catfish experience increased mortalities at arsenic concentrations between 10,900-100,000 µg/L (Clemens and Sneed, 1959; Gupta and Chakrabarti, 1993; Shukla et al., 1987). Other fish species, such as asiatic knifefish and goldfish, experience lethal effects when arsenic concentrations reach 490-30,930 µg/L (Birge et al., 1979; Ghosh and Chakrabarti, 1990).

9.2 Cadmium

The discussion of cadmium effects on salmonids is discussed in the acute and chronic cadmium criteria section of this Biological Assessment. Based on EPA's review of the literature, the Agency has determined that the acute and chronic cadmium is not likely to adversely affect endangered salmonids. Effects to other fish species are described in the following sections.

Sublethal effects

Sublethal effects such as altered enzyme activity, physiology and histopathology occur in catfish exposed to cadmium concentrations ranging from 300-400,000 µg/L (Bhattacharya et al., 1987; Bhattacharya et al. 1989; Dalal, 1989; Dalal and Bhattacharya, 1991; Dalal and Bhattacharya, 1994; Dalwani et al., 1985; Ghosh and Bhattacharya, 1992; Ghosh and Jana, 1988; Gupta and Rajbanshi, 1982; Gupta and Rajbanshi, 1988; Jana and Sahana, 1988; Jana and Sahana, 1989; Katti and Sathyanesan, 1984a; Katti and Sathyanesan, 1984b; Katti and

Sathyanesan, 1985; Saksena and Agarwal, 1986; Sastry and Subhadra, 1984; Sastry and Subhadra, 1985; Sastry et al., 1997; Smith et al., 1976). Whitefish appear to preferentially select water with 5µg/L cadmium over control waters in avoidance testing (McNicol and Scherer, 1993). Sublethal hemaetological effects on greenfish occur at concentrations ranging from 300-20,000 µg/L (Kuroshima, 1992), while goldfish experienced similar effects at 445 µg/L (Houston and Keen, 1984).

Lethal effects

For the Siberian sturgeon, Blubokov (1990) found that for early fry, exposure to cadmium concentrations of 5, 50, and 500 µg/L resulted in 11.5%, 6%, and 100% mortality respectively after a 16 day exposure. A hardness value for the test water was not given in this report.

Increased mortality occurs in catfish exposed to cadmium at levels ranging from 338.3-405,000 µg/L (Birge et al. 1985; Dalal and Bhattacharya, 1994; Chakrabarti and Ghosh, 1990; Das and Benerjee, 1980; Ghosh and Chakrabarti, 1993; Gupta, 1988; Gupta and Rajbanshi, 1982; Gupta and Rajbanshi, 1988; Gupta and Rajbanshi, 1991; Mitra, 1991; Phipps and Holcombe, 1985; Rausina et al., 1975; Sastry et al., 1997; Saxena and Parashari, 1983; Saxena et al., 1993; Spehar and Carlson, 1984). Squawfish experience significant mortalities at cadmium concentrations of 78-10,000µg/L (EPA, 1985b; Buhl, 1997). Scientists measured lethal effects in goldfish at 170µg/L (Birge et al., 1979). In a comparison study, researchers found LC₅₀s for bonytail and razorback sucker to be 148-168 µg/L and 139-160 µg/L, respectively, at a hardness of 199 mg/L CaCO₃ (Buhl, 1997). For the Siberian sturgeon, Glubokov (1990) found that for early fry, exposure to cadmium concentrations of 5, 50, and 500µg/L resulted in 11.5%, 6%, and 100% mortality respectively after a 16 day exposure. A hardness value for the test water was not given in this report.

9.3 Copper

The discussion of copper effects on salmonids is discussed in the acute and chronic copper criteria section of this Biological Assessment. Based on EPA's review of the literature, the Agency has determined that the acute and chronic copper is not likely to adversely affect endangered salmonids. Effects to other fish species are described in the following sections.

Sublethal effects

Apperson (1992) found 1.18-3.2 µg Cu/kg in white sturgeon oocytes in the Kootenai River whereas copper levels in the Kootenai River range from 2-12 µg/L. She concluded that the chronic effects of copper on wild sturgeon spawned in polluted waters and reared in contaminated sediments pose a severe threat on reproductive success. The average hardness for the Kootenai River ranges from 29.69-32.72 mg/L CaCO₃. However, it is important to note that not enough information was provided in this study to determine which ambient concentrations resulted in bioaccumulation of copper in sturgeon oocytes.

In catfish, sublethal effects such as altered enzyme levels, hemaetological parameters, histopathology, growth, and physiology occur at copper levels from 50-200,000 µg/L (Ansari, 1987; Asztalos, 1986; Bakshi, 1991; Benerjee and Homechaudhuri, 1990; Bhattacharya and Mukherjee, 1976; El-Domiaty, 1987; EPA, 1984; Ghosh and Jana, 1988; Gupta and Rajbanshi, 1979; James et al., 1995; James and Sampath, 1995; Jana and Sahana, 1988; Jana and

Sahana, 1989; Khangarot et al., 1988; Khangarot, 1992; Mukherjee and Bhattacharya, 1974; Mukherjee and Bhattacharya, 1975; Mukherjee and Bhattacharya, 1977; Nemcsok et al., 1991; Perkins et al., 1997; Rajbanshi and Gupta, 1988; Sastry and Sachdeva, 1994; Sastry et al., 1997; Shaffi, 1978; Shaffi and Jeelani, 1985; Srivastava and Pandey, 1982; Sultana and Devi, 1995; Wurts and Perschbacher, 1994).

Lethal effects

Squawfish mortality increases at copper levels of 363-10,000 µg/L (EPA, 1984; Buhl and Hamilton, 1996). Lethal effects occur when killifish encounter waters with concentrations of copper measuring 330-1,300 µg/L (EPA, 1984).

9.4 Lead

The discussion of lead effects on salmonids is discussed in the acute and chronic lead criteria section of this Biological Assessment. Based on EPA's review of the literature, the Agency has determined that the acute and chronic lead criteria is not likely to adversely affect endangered salmonids. Effects to other fish species are described in the following sections.

Sublethal effects

Lead concentrations ranging from 2,300-145,720 µg/L caused sublethal effects such as bioaccumulation, altered enzyme levels and hemaetology and histopathological effects in catfish (Abdelhamid and El-Ayouty, 1991; Chaurasia et al., 1996; Jana et al., 1986; Jha and Pandey, 1989; Jha, 1991; Katti and Sathyanesan, 1983; Katti and Sathyanesan, 1985; Katti and Sathyanesan, 1986a; Katti and Sathyanesan, 1986b; Katti and Sathyanesan, 1987a; Katti and Sathyanesan, 1987b; Mishra and Singh, 1997; Sastry and Gupta, 1978a; Sastry and Gupta, 1978b; Sastry and Gupta, 1979; Sastry and Gupta, 1980; Shaffi and Jeelani, 1985; Sharma et al., 1985). Hawkfish bioaccumulated lead at levels of 250,000-1,000,000 µg/L (Shakoori et al., 1992). In goldfish, sublethal effects such as cellular, enzyme, histopathological, and other physiological effects occur at lead levels of 400-5,000 µg/L (Bolognani Fantin et al., 1992; Bolognani Fantin et al., 1993; EPA, 1985d; Franchini et al., 1991).

Lethal effects

LC50s for catfish ranged from 16,600-38,000 µg/L (Saxena and Parashari, 1983), while the LC 50 determined for mosquitofish was greater than 56,000,000 µg/L (EPA, 1985d). In goldfish, LC50s ranged from 1660-40,000 µg/L (Birge et al., 1979; Bolognani Fantin et al., 1992).

9.5 Mercury

The discussion of mercury effects on salmonids is discussed in the acute and chronic mercury criteria section of this Biological Assessment. Based on EPA's review of the literature, the Agency has determined that approval of the chronic mercury criterion is likely to adversely affect salmonids. We have determined that there is sufficient data in the Biological Assessment to conclude that the approval of the chronic mercury criterion is likely to adversely affect

Kootenai River white sturgeon as well. Effects of the acute mercury criterion to non-salmonid species are described in the following sections.

Sublethal effects

Catfish experience adverse effects from mercury concentrations ranging from 12-12,000 µg/L. The sublethal effects include bioaccumulation, altered enzyme activity and histopathological and physiological effects (Kendall, 1975; Kendall, 1977).

Lethal effects

The LC₅₀s determined for catfish fall between 340-50,000 µg/L (Clemens and Sneed, 1958; Kirubagaran and Joy, 1988). In killifish, scientists found LC₅₀ values between 110-270 µg/L (EPA, 1985e).

9.6 Selenium

The discussion of selenium effects on salmonids is discussed in the acute and chronic selenium criteria section of this Biological Assessment. Based on EPA's review of the literature, the Agency has determined that approval of the chronic selenium criterion is likely to adversely affect salmonids. We have determined that there is sufficient data in the Biological Assessment to conclude that the approval of the chronic selenium criterion is likely to adversely affect Kootenai River white sturgeon as well. Effects of the acute selenium criterion to non-salmonid species are described in the following sections.

Sublethal effects

In goldfish and flagfish, behavior and growth were affected by selenium concentrations between 250-33,200 µg/L (EPA, 1980i; Weir and Hine, 1970).

Lethal effects

Catfish LC₅₀s ranged from 19,100-46,700 µg/L, while LC₅₀ s for goldfish were determined to fall between 8,800-110,000 µg/L (EPA, 1980i).

9.7 Zinc

The discussion of zinc effects on salmonids is discussed in the acute and chronic zinc criteria section of this Biological Assessment. Based on EPA's review of the literature, the Agency has already determined that approval of Idaho's acute and chronic zinc criteria is not likely to adversely affect endangered salmonids. Effects to other fish species are described in the following sections.

Sublethal effects

Effects on catfish ranging from altered enzyme levels, bioaccumulation, and hemaetological, and histopathological effects occurred at concentrations of zinc ranging from 500-130,000 µg/L (Banerjee, 1993; Banerjee, 1998; Banerjee and Banerjee, 1988; Dalal and Bhattacharya, 1991; Dalal and Bhattacharya, 1994; Hemalatha and Dalal, 1989; Jeelani, 1989;

Khangarot et al., 1981a; Khangarot, 1982b; Khangarot, 1984; Nemcsok and Boross, 1981; Shaffi, 1980; Shandilya and Banerjee, 1989; Shukla and Pandey, 1986a; Shukla and Pandey, 1986b; Sultana and Devi, 1995).

Lethal effects

In killifish, LC50s ranged from 840-22,600 µg/L (EPA, 1987b; Rehwoldt et al., 1971), while LC50s determined for squawfish occurred at 1,660-40,000 µg/L (Andros and Garton, 1980; Buhl and Hamilton, 1996; Hamilton, 1995). Researchers determined LC50s for catfish to be between 1,700-12,000 µg/L (Banerjee, 1998; Hemalatha and Banerjee, 1993; Hilmy et al., 1987; Khangarot et al., 1981a; Khangarot, 1981b; Khangarot, 1982a; Khangarot and Durve, 1982; Reed et al., 1980; Saxena and Parashari, 1983; Saxena et al., 1993).

Summary

With the information regarding the toxicity of these seven chemicals to a variety of fish species, we have determined that EPA's approval of the acute and chronic criteria for arsenic, cadmium, copper, cyanide, endosulfan, lead, and zinc and the acute criteria for mercury and selenium is not likely to adversely affect Kootenai River white sturgeon.

We have also determined that EPA's approval of the chronic criteria for mercury and selenium is likely to adversely affect Kootenai River white sturgeon.

10.0 References

Literature cited in the EPA Biological Assessment document consists of 33 pages of citations and is not included here.

For a copy of the unabridged report or the citations, contact U.S. Environmental Protection Agency, Seattle, Washington, Water Quality Standards section. As of the Year 2000, the water quality standards coordinator for the State of Idaho is Lisa Macchio, (206) 553-1834.