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FINAL REPORT ON

APPENDIX TO SMOKY CANYON MINE, PANELS F AND G ENVIRONMENTAL IMPACT STATEMENT (EIS): SELENIUM AND FISH

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EXECUTIVE SUMMARY

This Appendix reviews published information on the toxicity of selenium to fish, selenium behaviour and mode of action, and selenium water and tissue guidelines. Background and mining-related data on selenium in water and fish (particularly Yellowstone Cutthroat Trout, YCT) tissues for the Smoky Canyon Mine project area are compared to published water and tissue threshold values (i.e., values above which effects may occur and below which effects are not expected to occur). The purpose of this Appendix is to provide stakeholders with factual information regarding the above issues.

Selenium is unlike other metals/metalloids in that it has two separate modes of toxic action: acute toxicity via water exposure, and chronic (including reproductive) toxicity via dietary exposure. The former occurs at relatively high water column concentrations of inorganic selenium. The latter occurs due to accumulation and maternal transfer of organic selenium into eggs where, during embryonic development, the selenium substitutes for sulphur in the production of proteins resulting in reproductive abnormalities and/or failures. Transfer of inorganic selenium into dietary organo-selenium occurs via a complex series of interconnected hydrological, biogeochemical and biological pathways that vary over time, among sites, and among receptor taxa.

There are two primary forms of inorganic selenium in water bodies: selenite (Se IV) and selenate (Se VI). A wide range of acute waterborne toxicity values exists for each of these forms, spanning several orders of magnitude. There is overlap in the responses of cold- and warm-water fish species; however, the lowest acute toxicity is associated with warm-water species. Chronic waterborne toxicity values are summarized but are too few for meaningful comparisons between cold and warm-water fish species.

Winter stress syndrome (WSS) can develop when exposure to a stressor such as selenium requires metabolic energy at a time when energy reserves are depleted, for instance over winter when fish survive primarily based on energy (lipid, fat) stores rather than by active feeding. The single 1993 study with a warm-water fish species that links WSS and selenium is examined in detail. The available data do not allow for a definitive conclusion regarding the likely magnitude of WSS in cold-water fish, in part, due to insufficient information regarding the magnitude of stress responses to Se in cold-water fish compared to warm-water fish, as well as the potential for differential energy budgets and critical lipid contents in cold-water fish compared to warm-water fish.

A variety of selenium guidelines have been established based on both water and tissue concentrations. These are reviewed in detail with particular focus on the 2004 draft USEPA whole body fish selenium criterion. There is a dichotomy between published studies: one group of studies predicts adverse effects at lower tissue selenium concentrations than the other group. This dichotomy and the reasons behind it, including data and study-specific differences, are examined in detail.

There appears to be a consensus from reproductive effects studies conducted over the last few years (i.e., fertilized fish eggs with different selenium concentrations reared in the laboratory) that cold-water fish species can tolerate higher concentrations of selenium in their tissues than warm-water fish, without adverse reproductive effects. The possibility of growth effects to larval cold-water fish at lower selenium concentrations is suggested by one recent study, and requires further research.

All available site-specific selenium data for water, fish, sediment, benthic invertebrates and aquatic vegetation were reviewed. Many of the fish samples, including both YCT and brown trout, had whole-body tissue selenium concentrations above those predicted to result in reproductive failures. YCT from background stations had higher whole-body selenium concentrations than fish from mining-impacted stations. Factors that may influence expression of selenium effects from individual fish to populations of fish are reviewed and discussed.

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ACR	Acute-to-Chronic Ratio
BCMOE	British Columbia Ministry of Environment
BK	Background
BLM	Bureau of Land Management
CCC	Criteria Continuous Concentration
CCME	Canadian Council of Ministers of the Environment
CCREM	Canadian Council of Resource and Environment Ministers
CMC	Criteria Maximum Concentration
COPC	Contaminant of Potential Concern
DEIS	Draft Environmental Impact Statement
dw	Dry Weight
EC*x*	Effective Concentration (at a specified percentage effect '*x*', such as EC20 or EC50)
FAV	Final Acute Value
FCV	Final Chronic Value
FEIS	Final Environmental Impact Statement
GMAV	Genus Mean Acute Value
GMCV	Genus Mean Chronic Value
IC*x*	Inhibitory Concentration (at a specified percentage effect '*x*', such as IC20 or IC50)
IDEQ	Idaho Department of Environmental Quality
LC*x*	Lethal Concentration (at a specified percentage effect '*x*', such as LC20 or LC50)
LOEC	Lowest Observed Effect Concentration
MATC	Maximum Acceptable Toxicity Concentration
MI	Mine Impacted
NM	Not Measured
NOEC	No Observed Effect Concentration
SLD	San Luis Drain
SMAV	Species Mean Acute Value
SMCV	Species Mean Chronic Value
TRG	Tissue Residue Guideline
USDOI	United States Department of the Interior
USEPA	United States Environmental Protection Agency
USFS	United States Forest Service
WQC	Water Quality Criterion
WQG	Water Quality Guideline
WSS	Winter Stress Syndrome
ww	Wet Weight
YCT	Yellowstone Cutthroat Trout

1.0 INTRODUCTION

1.1 Background

The J.R. Simplot Company (Simplot) conducts open pit phosphate mining operations at its Smoky Canyon Mine in southeast Idaho (Caribou County, ID). Simplot proposes extending these operations into two federal phosphate leases: Manning Creek No. I-27512 (referred to hereafter as Panel F) and Deer Creek No. I-01441 (referred to hereafter as Panel G). A draft Environmental Impact Statement (DEIS) was prepared by the Bureau of Land Management (BLM), the US Forest Service (USFS), and the Idaho Department of Environmental Quality (IDEQ) (BLM, 2005). The DEIS was made available for public comment (through the BLM website) until mid-March 2006.

One specific issue identified through review comments on the DEIS was the potential adverse impact of selenium on fish populations in the project area, particularly Yellowstone cutthroat trout (YCT), *Oncorhynchus clarki bouveri*. Golder Associates Ltd. (Golder) was retained by JBR Environmental Consultants Inc. (JBR) to prepare this Appendix to the final EIS (FEIS) that provides an objective (i.e., factual) synthesis of differing opinions and literature regarding selenium bioaccumulation in fish, effects thresholds, threats and hazards, and existing baseline conditions within the project area.

1.2 Objectives

The specific objectives of this project were to:

1. Provide a review of relevant studies on selenium effects to fish (Chapter 2.0 - acute and chronic water exposures, dietary exposures, maternal transfer, field and mesocosm studies), as background for discussions regarding chapters addressing subsequent objectives (as noted below).

2. Provide a review of selenium behaviour and mode of action (Chapter 3.0) including processes leading to selenium exposure in fish (hydrology, biogeochemistry, biology), toxic action, interactions with other elements or contaminants, winter stress syndrome (experimental evidence and implications).

3. Provide a review of selenium guidelines based on water concentrations (Chapter 4.0) including the derivation of USEPA's water quality criteria (WQC) and other approaches to deriving water quality guidelines (WQGs) for selenium.

4. Provide a review of selenium guidelines based on tissue residue concentrations (Chapter 5.0) including the USEPA 2004 draft criterion (rationale and derivation method), other tissue residue guidelines (a detailed review of published studies), and cold-water selenium studies.

5. Present empirical data on selenium concentrations in water and in fish tissues in the project area (Chapter 6.0), both for areas subject to mining and areas that have not been disturbed by mining, and compare these to the range of water and tissue threshold values previously discussed.

2.0 REVIEW OF STUDIES ON SELENIUM EFFECTS ON FISH

This chapter provides a summary of key studies that have been conducted to investigate the toxicity of selenium to freshwater fish (invertebrates, plants and other aquatic organisms were specifically excluded from consideration). This information is provided as background to the discussions in subsequent chapters regarding the approaches used to establish criteria for selenium for the protection of aquatic life, based on both water and fish tissue residue concentrations, and their potential application to site conditions within the Salt River Watershed in southeast Idaho. The following sections present information on the acute and chronic toxicity of selenium associated with waterborne exposures, dietary exposures, maternal transfer (related to dietary exposure), and field studies. Potential differences in selenium toxicity between warm-water and cold-water fish species are also considered.

2.1 Acute Waterborne Selenium Exposure Studies

As part of the process of developing new aquatic life criteria for selenium, USEPA (2004a) compiled and evaluated available data from acute studies conducted using waterborne selenium exposures, and determined whether they met data quality requirements for use in deriving acute WQC for selenium (see Chapter 4). This compilation included studies published up to 2001. Most freshwater fish are relatively insensitive to selenium in water-only exposures, particularly those of short duration, as compared to adverse effects associated with dietary exposure to selenium. Acute 96-h LC50s[1] are typically on the order of "mg/L" for most species, whereas significant adverse impacts of selenium exposure (i.e., reproductive failure and collapse of warm-water centrarchid fish populations in Belews Lake, NC) have been reported to occur when the water concentration was 10 µg/L but diet was presumed to be a significant exposure pathway. This section provides a brief summary of the range of acute LC50 or EC50 values reported for each freshwater fish species by USEPA (2004a) for use in acute criteria derivation, and from recent studies not included in that compilation. Acute toxicity data are provided for two forms of selenium (selenite [Se IV] and selenate [Se VI]), with selenate toxicity data normalized to a sulphate concentration of 100 mg/L[2].

Coho Salmon (*Oncorhynchus kisutch*): Four acute values for alevin and juvenile coho salmon were reported for selenite, ranging from 3,578 to 35,560 µg/L; four acute values for selenate ranged from 21,000 to 266,000 µg/L (Hamilton and Buhl 1990; Buhl and Hamilton 1991).

[1] The "LC50" is the concentration of test material that is estimated to be lethal to 50% of test organisms in a toxicity test. The "EC50" is the concentration of test material that is estimated to cause a specified effect, other than lethality, in 50% of test organisms in a toxicity test.

[2] As discussed in subsequent chapters, selenate toxicity decreases as sulphate concentration increases and USEPA (2004) has taken this into consideration with their new draft acute criterion for selenate.

Chinook Salmon (*Oncorhynchus tshawytscha*): Eight acute values for eyed egg, alevin and juvenile chinook salmon were reported for selenite, ranging from 8,150 to >348,300 µg/L; eyed eggs were the least sensitive life-stage in these acute exposures (Hamilton and Buhl 1990). In the same study, seven acute values were reported for the same life stages for selenate, ranging from 72,000 to >856,000 µg/L.

Rainbow Trout (*Oncorhynchus mykiss*): Seven acute values for alevin and juvenile rainbow trout were reported for selenite, ranging from 1,800 to 118,000 µg/L (Adams 1976; Hunn et al. 1987; Buhl and Hamilton 1991; Goettl and Davies 1976; Hodson et al. 1980). Four acute values for alevins and juveniles tested with selenate ranged from 22,700 to 330,000 µg/L (Brooke et al. 1985; Buhl and Hamilton 1991; Spehar 1986).

Brook Trout (*Salvelinus fontinalis*): One acute value (10,200 µg/L) was reported for adult brook trout for selenite (Cardwell et al. 1976). No acute values were reported for selenate.

Arctic Grayling (*Thymallus arcticus*): Two acute values for alevin and juvenile Arctic grayling were reported for selenite, ranging from 15,700 to 34,700 µg/L (Buhl and Hamilton 1991). The same study provided two acute values for selenate, ranging from 70,200 to 126,300 µg/L.

White Sucker (*Catastomus commersoni*): Two acute values for white suckers were reported for selenite, ranging from 29,000 to 31,400 µg/L (Klaverkamp et al. 1983; Duncan and Klaverkamp 1983). No acute values were reported for selenate.

Yellow Perch (*Perca flavescens*): One acute value (11,700 µg/L) for yellow perch was reported for selenite (Klaverkamp et al. 1983). No acute values were reported for selenate.

Fathead Minnow (*Pimephales promelas*): Sixteen acute values for larval and juvenile fathead minnow were reported for selenite, ranging from 620 to 11,300 µg/L; nine acute values for selenate ranged from 7,300 to 18,900 µg/L (Adams 1976; Brooke et al. 1985; Mayer and Ellersieck 1986; Cardwell et al. 1976; GLEC 1998; Kimball MS; Spehar 1986). Adams (1976) conducted fathead minnow testing at a range of temperatures and found that there was approximately a four-fold decrease in selenite toxicity at 13ºC (LC50s of 10,500 and 11,300 µg/L) as compared to testing performed at 25ºC (LC50s of 2,200 and 3,400 µg/L).

Bluegill (*Lepomis macrochirus*): Two acute values for juvenile bluegills were reported for selenite, ranging from 12,000 to 28,500 µg/L, and one acute value (216,000 µg/L) was reported for selenate (Brooke et al. 1985; Cardwell et al. 1976).

Razorback sucker (*Xyrauchen texanus*): Six acute values for larvae, fry and juveniles of razorback sucker were reported for selenite, ranging from 4,100 to 11,300 µg/L; six acute values for selenate ranged from 7,800 to 16,200 µg/L (Hamilton 1995; Buhl and Hamilton 1996; Hamilton and Buhl 1997a).

Colorado Squawfish (*Ptychocheilus lucius*): Six acute values for larvae, fry and juveniles of Colorado squawfish were reported for selenite, ranging from 6,400 to 20,700 µg/L; six acute values for selenate ranged from 9,800 to 103,800 µg/L (Hamilton 1995; Buhl and Hamilton 1996; Hamilton and Buhl 1997a).

Bonytail (*Gila elegans*): Five acute values for larvae, fry and juveniles of bonytail were reported for selenite, ranging from 6,900 to 14,500 µg/L; five acute values for selenate ranged from 10,560 to 77,100 µg/L (Hamilton 1995; Buhl and Hamilton 1996).

Flannelmouth Sucker (*Catastomus latipinnis*): One acute value (19,100 µg/L) was reported for larval flannelmouth suckers for selenite; one acute value (27,400 µg/L) was reported for selenate (Hamilton and Buhl 1997b).

Goldfish (*Carassius auratus*): One acute value (26,100 µg/L) was reported for goldfish for selenite (Cardwell et al. 1976). No acute values were reported for selenate.

Striped Bass (*Morone saxilitis*): Two acute values were reported for striped bass for selenite, ranging from 1,325 to 2,400 µg/L (Palawski et al. 1985). No acute values were reported for selenate.

Channel Catfish (*Ictalurus punctatus*): Three acute values were reported for channel catfish for selenite, ranging from 4,110 to 16,000 µg/L; one acute value (226,320 µg/L) was reported for selenate (Brooke et al. 1985; Cardwell et al. 1976; Mayer and Ellersieck 1986).

Flagfish (*Jordanella floridae*): One acute value (6,500 µg/L) was reported for flagfish for selenite (Cardwell et al. 1976). No acute values were reported for selenate.

Western Mosquitofish (*Gambusia affinis*): One acute value (12,600 µg/L) was reported for western mosquitofish for selenite (Reading 1979). No acute values were reported for selenate.

Common Carp (*Cyprinus carpio*): One acute value (35,000 µg/L) was reported for the common carp for selenite (Sato et al. 1980). No acute values were reported for selenate.

Golden Shiner (*Notemigonus crysoleucas*): One acute value (11,200 µg/L) was reported for golden shiner for selenite (Hartwell et al. 1989). No acute values were reported for selenate.

There was no clear relationship between temperature regime and relative sensitivity of different fish species to selenium in acute exposures, although Adams (1976) did report that selenium was more toxic to fathead minnow at higher temperatures than in cooler water. The five salmonid species (coho and Chinook salmon, rainbow trout, brook trout, and Arctic grayling), white sucker, and yellow perch were considered to be cold-water fish, based on their general geographic distribution and the fact that the acute toxicity tests were conducted at temperatures of approximately 15ºC or less. The other 13 fish species (including fathead minnow, bluegills, and razorback sucker) were considered to be warm-water fish, again based on their generally geographic distribution and the fact that the acute toxicity tests were conducted at temperatures of approximately 20ºC or higher (usually 25ºC). Figures 1 and 2 illustrate the ranges of acute toxicity values (plotted on a logarithmic scale) for each fish species for selenite and selenate, respectively. There was considerable overlap in responses to both forms of selenium for cold-water and warm-water fish, but the lowest acute values were associated with warm-water species and the highest acute values were associated with cold-water species.

2.2 Chronic Waterborne Selenium Exposure Studies

A number of studies have been conducted to investigate the effects of chronic water-only selenium exposures on fish, although it has become more common in recent years to incorporate dietary exposure into experimental designs, which is more representative of real-world exposure conditions.

Bluegill (*Lepomis macrochirus*)

Cleveland et al. (1993) conducted a 60-d water-only exposure of juvenile (5-months old) bluegills to a 6:1 ratio of selenate:selenite (a 90-d dietary exposure was also conducted; see Section 2.3). Testing was conducted at 25ºC in a flow-through system, with uncontaminated food provided daily. Measured selenium concentrations in the test solutions ranged from 160 to 2,800 µg/L. Fish were measured to determine condition factor (K), and behavioural surveys were conducted during the first 30 days of the exposure. After 60 d, there were significant differences in mortality in the higher exposure concentrations, including almost complete mortality at the highest concentration (2,800 µg/L). In terms of mortality, the 60-d NOEC[3] was 330 µg/L (22.5% mortality)

[3] The NOEC (no observed effect concentration) is the highest concentration of material tested that was not significantly different from the negative (clean) control in terms of mean response. The LOEC (lowest observed effect concentration) is the lowest concentration of material tested that was significantly different from the negative (clean) control) in terms of mean response.

and the 60-d LOEC was 640 µg/L (52.5% mortality). Among surviving fish on Day 60, there were no significant differences in K at concentrations up to 1,120 µg/L (i.e., the NOEC). Abnormal swimming behaviour (i.e., lethargy, abnormal posture, fish remaining at the bottom of the aquaria) was observed during the first week of exposure, but no other behavioural changes (e.g., aggression, feeding, colouring, response to stimuli) were observed. Measurement of swimming activity on Day 18 showed that swimming movement was reduced in all treatments except 640 µg/L. After 30 d of exposure, the tissue selenium concentration in fish exposed to 2,800 µg/L was approximately 15 µg/g dw; this treatment had 97.5% mortality by Day 60. After 60 d, the tissue selenium concentration in fish exposed to 330 µg/L in water (the NOEC for mortality effects) was approximately 4 µg/g dw.

Goldfish (*Carassius auratus*)

Ellis et al. (1937; cf. Cardwell et al. 1976) exposed goldfish (*Carassius auratus*; 80-mm length) to 2,000 µg/L Se for 46 days (with test solution renewal and feeding provided every 48 h), and observed that feeding stopped within 8 days, mortality began to occur after 18 days, and the highest mortality occurred after 25 to 27 days of exposure. The authors also noted that exposure of goldfish to 5,000 µg/L resulted in mortalities occurring within 4 to 10 days.

Razorback Suckers *(Xyrauchen texanus)*

Hamilton et al. (2005) conducted a study to investigate the effects of dietary selenium on larval razorback suckers. Studies were conducted using different aged larvae, ranging from 5-day old to 28-day old at test initiation. Larval fish were fed zooplankton collected from six sites near the Green River (UT); zooplankton selenium concentrations ranged from 2.1 to 91 µg/g dw.

Beyers and Sodergren (2002) conducted a 28-d study with larval razorback suckers exposed to different site waters from the Colorado River, chosen from locations representing uncontaminated, moderately and highly contaminated conditions (dietary exposures were also conducted; see Section 2.3). Testing was conducted at 20ºC in a static-renewal system, and the fish were fed rotifers (containing <0.702 µg/g dw Se) daily. Selenium concentrations in the site waters ranged from <1 to 20.3 µg/L (<1 µg/L in the negative control[4]), and after the 28-d exposure whole-body fish tissue concentrations[5] ranged from 3.04 to 14.4 µg/g dw (2.34 µg/g dw in the negative control). Mean survival was 97.5 to 100% in all treatments; dry weight was significantly higher (approximately

[4] In a laboratory toxicity test, the negative (clean) control is a treatment that duplicates all conditions of the exposure treatment(s) except the presence of the test substance (i.e., clean laboratory water is typically the negative control for a water-column toxicity test).

[5] Mean moisture content of these razorback sucker larvae was 83%.

13 to 21%) in all treatments relative to the negative control (length was also higher but the relative increases were smaller). These results indicated that exposure to selenium water concentrations up to 20 µg/L had no adverse effects on larval razorback suckers. The authors attributed the increased growth in the site waters to the presence of additional trace elements not present in the laboratory-formulation negative control water. The authors also noted the limited ecological relevance of drawing conclusions about potential selenium-related effects based on water-only exposure (i.e., if selenium concentrations are elevated in the field, then it is likely that dietary items will also contain elevated selenium).

Chinook Salmon (*Oncorhynchus tshawytscha*)

Hamilton et al. (1986) conducted two chronic toxicity studies with fall Chinook salmon to investigate the effects of waterborne exposure to mixtures of selenium, molybdenum and boron on mortality and growth. One study was conducted with well water, and exposed eyed eggs from two weeks pre-hatch until 90 days post-hatch[6]. The second study was conducted with blended water, and exposed juvenile fish (0.3 g wet weight) for 90 days. The blended water was intended to simulate San Luis Drain (SLD) water diluted 10-fold by the San Joaquin River. For both studies (well water and SLD/San Joaquin River water), a dilution series of 0.25X, 0.5X, 1X, 2X and 4X was used, with the 1X treatment consisting of 35 µg/L Se (6:1 ratio of selenate:selenite), 49 µg/L Mo, and 1,400 µg/L B. Testing was conducted at 12ºC under flow-through conditions.

- In the well water study, there were no effects on hatchability, time to hatching, or survival to the swim-up stage (i.e., yolk sac absorbed). There were no effects on mortality at 30 days, but there were significant effects on both mortality and growth (length and weight) in the 2X and 4X treatments at 60 days and 90 days. Mortality in the 4X treatment (140 µg/L Se) was 99.1% at 60 days and 100% at 90 days. Although the effects on growth in the 2X treatment (70 µg/L) were statistically significant, they represented a <10% decrease in length and an approximately 20% decrease in weight relative to the negative control. Based on nominal selenium concentrations (i.e., ignoring the Mo and B), the NOEC for mortality and growth was 35 µg/L and the LOEC for these endpoints was 70 µg/L. Hamilton et al. (1986) did not report point estimates (e.g., LC50, IC25[7]), so we have calculated those endpoints. The 60-d and 90-d LC50s were estimated to be 85 and 74 µg/L, respectively, and the IC25s for growth (length and weight) were >140 µg/L.

[6] The authors referred to this as a 90-d study, and reported results at 30-, 60- and 90-d increments, but the actual exposure period appears to be 104 d.

[7] The "IC25" is the concentration of test material that is estimated to cause a 25% inhibitory effect in the test organisms in a toxicity test.

- In the blended water study, there were statistically significant effects on mortality at 30 days in the 0.5X to 4X treatments (17 to 140 µg/L Se), but this represented mortalities in the range of 11.2 to 13.7%, as compared to 2.5% in the negative control. At 60 and 90 days, there were significant effects on mortality in the 2X and 4X treatments, but significant effects on growth only occurred in the 4X treatment. Unlike the well water study, which had 100% mortality in the 4X treatment, the blended water study had <40% mortality in the 4X treatment. The statistically significant effects on growth in the 4X treatment (140 µg/L) were of similar magnitude to those observed in the well water study (approximately 10% decrease in length and 25% decrease in weight relative to the negative control). Based on nominal selenium concentrations (i.e., ignoring the Mo and B), the NOEC and LOEC for mortality would be 35 and 70 µg/L, respectively. Point estimates (not reported by Hamilton et al. 1986), would be >140 µg/L for both the 90-d LC50 and the 90-d IC25s for growth (length and weight).

2.3 Dietary Selenium Exposure Studies

Fathead Minnow (*Pimephales promelas*)

Bennett et al. (1986) investigated the uptake of selenium in an aquatic food web and its effect on larval fathead minnow. The fish were fed rotifers (*Branchionys calycifloris*) that had been allowed to feed on phytoplankton (*Chlorella pyrenoidosa*) cultured in selenium-enriched media. Newly hatched larval fish were initially fed uncontaminated rotifers, but were then switched to the selenium-contaminated rotifers either 2, 4 or 8-d post-hatch, fed that diet for 7 to 9 d, and switched back to the uncontaminated diet until 28-d post-hatch. Selenium concentrations in the rotifers ranged from 46 to 91 µg/g dw, and mean tissue selenium concentrations in the larval fathead minnows ranged from 43 to 61 µg/g dw when feeding of the selenium-contaminated rotifers was stopped. No selenium-related mortalities were reported, but larval fish growth (i.e., dry weight) was reduced in all three feeding trials relative to negative control fish (reduced feeding by fish offered diets containing elevated selenium has also been reported by other researchers, e.g., Coughlan and Velte 1989; Hilton et al. 1980; Hamilton et al. 1986); this reduction was statistically significant for the trials that began 4- and 8-d post-hatch.

Bluegill (*Lepomis macrochirus*)

Lemly (1993a) conducted a 180-d study that exposed juvenile bluegill to selenium through both diet and water, in addition to simulating winter conditions (i.e., low temperatures). This study is described in detail in Section 3.4.1, in conjunction with discussion regarding the potential implications of winter stress syndrome (WSS) on the ability of various fish species to tolerate seasonal exposure to contaminants such as selenium.

Cleveland et al. (1993) conducted a 90-d dietary study feeding seleno-L-methionine to juvenile (5-months old) bluegills (a 60-d water-only exposure was also conducted; see Section 2.2). Testing was conducted at 25ºC in a flow-through system, using a diet consisting of Oregon Moist Pellet (OMP) feed mixed with seleno-L-methionine to provide nominal dietary concentrations ranging from 1.63 to 26.0 µg/g ww. Fish were measured to determine condition factor (K), and behavioural surveys were conducted after the first 30 days of the exposure. After 90 d, mortality was significantly different in the 6.5 µg/g ww treatment but not in the other treatments (22.5% mortality, as compared to 7.5 to 17.5% mortality in the other treatments and 5% in the negative control). Although K was significantly different at 13 and 26 µg/g ww, values for K were 1.2 as compared to 1.3 in the other treatments. After 60 d, the tissue residue concentration in the 26 µg/g ww diet treatment was approximately 12 µg/g dw, with only 17.5% mortality (this was in contrast to the water-only study where fish with a mean tissue concentration of approximately 15 µg/g dw had 87.5% mortality after only 30 d of exposure). Fish did not accumulate tissue selenium to concentrations higher than in their diet; BCFs were 0.5 to 1.0.

Coyle et al. (1993) conducted a 140-d study with adult bluegills that incorporated both waterborne and dietary selenium exposure and assessed effects on reproductive success. The first 60 d of the study involved exposure of the adult fish, and the final 80 d of the study incorporated a maternal transfer component in that the endpoints exampled were spawning frequency, fecundity, percentage hatch, and survival of fry until 30-d post-hatch. All treatments (except the negative control) included a selenium water concentration of 10 µg/L, as well as an OMP diet amended with seleno-L-methionine to provide dietary selenium concentrations ranging from 4.6 to 33.3 µg/g dw. The study was conducted in a flowthrough system; the initial temperature was 22ºC but this was gradually increased to 28ºC (along with lengthening photoperiod) over the first 60 d to bring the fish into spawning condition. After 60 d, the number of fish in each treatment was reduced, spawning trays were placed in each aquarium, and the increased temperature and photoperiod were maintained for the next 70 d. A total of 135 spawns occurred across all treatments. To assess hatching success, 80 developing eggs from each spawn were placed in four replicate mesh-bottomed containers and suspended in their parental aquaria for 24 h. After hatching success was determined, 20 fry from each spawn were transferred to separate growth chambers and monitored for survival and growth for 30 d. For adult fish, there were no differences in fish size, condition factor (K), or gonadosomatic index (GSI) after 60 d or 140 d; there were differences between mature male and female fish unrelated to the selenium exposure. Reduced feeding was not observed during the study. There were also no significant differences in spawning frequency, fecundity, or percent hatching success. Larval survival was affected by selenium exposure of the adults, although larval growth was not affected. Fry from adult fish that were fed 33.3 µg/g dw Se had only 7% survival, whereas the other treatments had 75 to 90% survival within a few days of hatching. The mean whole-body selenium

concentration in adult fish fed the 33.3 µg/g dw diet was approximately 19 µg/g dw; as with the study by Cleveland et at. (1993), fish did not accumulate selenium to concentrations higher than was present in their diet. After 30 d, fry had tissue selenium concentrations between 3.3 and 6.0 µg/g dw but this was not concentration-related (the brine shrimp used for fry feeding contained 2.7 µg/g Se dw).

Striped Bass (*Morone saxatilis*)

Coughlan and Velte (1989) conducted an 80-d laboratory study in which they fed selenium-contaminated and uncontaminated fish to striped bass. Red shiners (*Notropis lutrensis*), collected from Belews Lake, NC and having a tissue selenium concentration of 9.6 µg/g ww, were used as the contaminated food supply, and golden shiners (*Notemigonus crysoleucus*) from a bait supplier were used as the control (an uncontaminated supply of red shiners was not available). The tissue selenium concentration of the golden shiners was 0.3 µg/g ww. The protein content of the two fish diets was similar, but the fat and ash content of the golden shiners was slightly lower than that of the red shiners; concentrations of metals including arsenic, cadmium, copper, mercury and zinc were similar in the two fish species. The striped bass that were fed the red shiner diet had reduced feeding, lethargy, histological damage, little weight gain, and high mortality, as compared to the control fish (fed golden shiners) that did not demonstrate adverse effects. There were no mortalities among the control fish after 80 d, whereas the exposed fish were all dead or moribund within 78 d. The final tissue selenium concentration in the exposed striped bass was 3.8 µg/g ww, as compared to 1.1 µg/g ww in the control fish. Applying the 75% moisture content value used by the authors, these tissue concentrations would correspond to 15.2 and 4.4 µg/g dw, respectively. Given the study design that was used, with a single exposure treatment, this study provides information about a tissue-effects concentration but does not provide information about where the effects threshold might occur.

Razorback Suckers (*Xyrauchen texanus*)

Beyers and Sodergren (2002) conducted a 28-d study with larval razorback suckers exposed to different site waters (and fed organisms reared in those site waters) from the Colorado River, chosen from locations to represent uncontaminated, moderately and highly contaminated conditions (water-only exposures were also conducted; see Section 2.2). Testing was conducted at 20ºC in a static-renewal system, and the fish were fed rotifers containing selenium concentrations ranging from 2.10 to 21.8 µg/g dw (<0.702 µg/g dw in the control) daily. Selenium concentrations in the site waters ranged from <1 to 20.3 µg/L (<1 µg/L in the control). Following the 28-d exposures to both site water and selenium-contaminated rotifers reared in the corresponding site waters,

whole-body fish tissue concentrations[8] ranged from 5.45 to 42 µg/g dw (2.34 µg/g dw in the control). Mean survival was 97.5 to 100% in all treatments, and there was little difference in either dry weight or length of larvae relative to the control. This growth response was different from that observed for the water-only exposures, in which dry weight and length both increased in the selenium treatments. Therefore, while the greater exposure to selenium (through both water and diet) resulted in lower growth than the water-only exposure, the responses were still consistent with those observed in the control. This study indicates that there should be no adverse effects associated with exposure to 20 µg/L in water or 21.8 µg/g dw in the diet, and associated whole-body tissue residues up to 42 µg/g dw.

Chinook Salmon *(Oncorhynchus tshawytscha)*

Hamilton et al. (1986) conducted a 6-wk study with fall Chinook salmon parr in which the fish were fed a diet amended with organoselenium, and effects on survival, growth, predator avoidance, and parr-smolt transformation were measured. The experimental diet consisted of Oregon Moist Pellet (OMP) combined with selenium-contaminated mosquitofish (*Gambusia affinis*) from the SLD and Kesterson National Wildlife Refuge. The dietary selenium exposures were 6.5, 13 and 26 µg/g [it is unclear whether these were ww or dw]; controls consisting of OMP only and OMP plus uncontaminated mosquitofish were also included. The study was conducted at 10ºC. After 30 days, growth was reduced by up to 20% (reduced feeding was observed in the 13 and 26 µg/g treatments) but the differences were not statistically significant (i.e., NOEC was 26 µg/g). Fish fed the 26 µg/g treatment for 43 to 50 days showed no significant effects on predator avoidance when offered to adult cutthroat trout. Effects on parr-smolt transformation (i.e., seawater challenge) and migratory behaviour were reported for the 26 µg/g treatment. Gill N^+-K^+-ATPase activity was reduced in fish fed the 26 µg/g diet, but not significantly; ATPase activity levels in other treatments suggested the possibility of a nutritional deficiency associated with the mosquitofish, but all three diets had similar compositions. Whole-body selenium tissue concentrations were determined following 33 days of exposure; control fish contained 0.2 to 0.3 µg/g, whereas fish fed diets containing 6.5, 13 or 26 µg/g Se had corresponding whole-body concentrations of 2.1, 2.9 and 4.9 µg/g[9], respectively.

[8] Mean moisture content of these razorback sucker larvae was 83%.

[9] Although the paper includes an equation and a factor (presumably moisture content of each treatment) for converting wet weight to dry weight, it is not clear how the tissue residue concentrations were reported.

Cutthroat Trout (*Oncorhynchus clarki*)

Hardy (2005) conducted a study that was designed to look at the effects of dietary selenium on feeding, growth and reproductive performance of cutthroat trout obtained from a hatchery and also collected from the Blackfoot River in southeast Idaho.

- Eyed eggs were obtained from the hatchery, reared in Heath trays until they hatched, transferred to larger tanks and fed a commercial trout feed until they were approximately 1 g (ww). At that point, fish were transferred to experimental tanks and fed one of six diet treatments incorporating selenomethionine into the fish food; concentrations ranged from 3.8 to 12 µg/g Se dw. Fish were transferred to larger tanks after six months, and moved to outdoor tanks after 80 weeks of feeding so that increasing photoperiod could trigger maturation and spawning; fish were left undisturbed until late spring and then checked periodically for maturation status. Spring spawning was unsuccessful, but gametes were obtained from these fish in fall, fertilized and reared through hatching. Numbers of eggs and fry (normal and abnormal) were determined, and deformed fry were preserved for later microscopic examination of cranio-facial deformities. Fish were sampled for tissue Se analysis after the first 12 weeks of the feeding trial and at the time of spawning. After 44 weeks of feeding, some fish were removed from each treatment, fed the control diet for 32 weeks, and then sacrificed for tissue Se analysis. Fish exposed to the highest selenium diet treatment had whole-body tissue concentrations of 12.5 µg/g dw after 44 weeks; at the time of spawning (after 124 weeks of the selenium diet), fish from this highest treatment had mean whole-body selenium concentrations of 5.61 to 6.4 µg/g dw and mean egg selenium concentrations of 16.04 to 18.0 µg/g dw. After 32 weeks depuration, tissue selenium concentrations in fish from the highest selenium diet treatment were close to baseline levels. Eggs from fish exposed to the highest selenium diet treatment had 85% hatching success, 6.8% incidence of total deformities, and 1.95% incidence of cranio-facial deformities[10]. Hardy (2005) concluded that selenium exposure did not represent a threat to cutthroat trout in the Blackfoot River; the incidence of deformities was low and juvenile fish moving from upstream nursery areas (where selenium concentrations are elevated) to downstream rearing areas (where selenium concentrations are lower) would undergo depuration of selenium.

- A similar experimental design was also used for the Blackfoot River cutthroat trout, but problems during the feeding trial may have confounded the results of this study. The feeding trial was eventually re-started successfully and the study continued

[10] Egg hatching success, percent total deformities, and percent craniofacial deformities for fish from the two highest diet treatments were similar to control performance, and better than in the lower selenium diet treatments. Across all diet treatments, hatching success was 70 to 93%, total deformities were 6.8 to 20.2%, and cranio-facial deformities were 1.17 to 9.21%.

through to the spawning stage, although no viable eggs were obtained from any treatments. Analysis of selenium concentrations was in progress, and no results from this portion of the study were reported by Hardy (2005).

Rainbow Trout

Vidal et al. (2005) conducted a 90-d dietary exposure in which larval trout were fed fish food spiked with selenomethionine. A review of Vidal et al. (2005) was subsequently conducted by DeForest et al. (2006). Fish weight, length, and selenium tissue concentrations were measured on Days 0, 30, 60 and 90; in addition, the ratio of reduced glutathione (GSH) to oxidized gluthathione (GSSG) was measured at Days 60 and 90 as a biomarker for oxidative stress. Vidal et al. (2005) determined a 90-d LOEC for growth of 1.2 µg/g ww (whole-body Se concentration) and concluded that this value was below the 4 µg/g dw whole-body Se threshold proposed by Maier and Knight (1994), Hamilton (2002) and others; however, this conclusion failed to consider the influence of moisture content (i.e., the LOEC of 1.2 µg/g ww would be 4.8 µg/g dw assuming a moisture content of 75%). DeForest et al. (2006) noted that Vidal et al. (2005)'s selection of 1.2 µg/g ww (whole-body) as the LOEC was based on the fact that it was the lowest average tissue concentration to demonstrate a statistical difference to the negative control; they commented that it would be more appropriate to select the lowest exposure concentration with a statistically significant reduction in growth. The 90-d growth LOEC is actually 4.6 µg/g dw in food, which corresponds to 0.58 µg/g ww in whole-body fish, which corresponds to 2.3 µg/g dw in whole-body fish. As a result, the conclusion that the LOEC was less than 4 µg/g dw whole-body was correct, although the selected LOEC in Vidal et al. (2005) was incorrect. DeForest et al. (2006) noted that whole-body fish Se concentrations peak at Day 60, but decline by Day 90 in all treatments including the negative control; a dose-response relationship was not demonstrated. Vidal et al. (2005) hypothesized that this decline was due to growth dilution, or changes in the uptake or elimination of Se as the fish matured; they noted that a similar decrease in Se tissue concentrations occurred at 90 days in chinook salmon (Hamilton et al. 1990). DeForest et al. (2006) commented that the results from Vidal et al. (2006) are surprising, since the whole-body LOEC of 2.3 µg/g dw falls within the range of reported background concentrations. Vidal et al. (2005) concluded that their results demonstrated the difficulty of using body-burden residues as toxicity thresholds for essential elements in developmental immature stages; DeForest et al. (2006) concluded that this area requires further research, and therefore, applying a threshold value from larval fish to adult fish was not warranted at this time.

2.4 Maternal Transfer Studies

This section describes studies where the experimental design was intended to determine the effects of maternal transfer of selenium from female fish to offspring through the egg.

In these studies, eggs from selenium-exposed fish were collected and reared through hatching and beyond. No selenium was added to the experimental system, either through water or diet, so the only opportunity for selenium exposure was through the eggs.

Gillespie and Baumann (1986) – Bluegill

Gillespie and Baumann (1986) used adult bluegills (*L. macrochirus*) containing high and low tissue selenium concentrations in a breeding study to determine whether parental body burdens affected gamete viability and embryo-larval mortality. This study was conducted in 1982 and 1983 to investigate declines in fish populations that had been observed in the Hyco Reservoir (the cooling reservoir for a coal-fired electric power plant in North Carolina), similar to those observed in Belews Lake. Although selenium water concentrations in the reservoir were approximately 9 to 12 µg/L Se (not high enough to be expected to cause toxicity), adult fish densities had declined by 38 to 75% in one year (1979 to 1980), and larval fish densities had declined by an even larger factor. Adult bluegills were collected from Hyco Reservoir for use as the high-Se treatment, and from Roxboro City Lake (a local municipal water supply) for use as the low-Se treatment. The mean selenium concentration in gonads and carcass of the Hyco Reservoir fish was 7.94 µg/g ww, as compared to 0.38 µg/g ww in the Roxboro Lake fish. Eighteen parental crosses were made, to account for all possible combinations of males and females and selenium concentrations. There were no significant differences in percent fertilization or hatching success for any treatments. However, larvae from crosses of Hyco Reservoir (high-Se) females had external abnormalities in the form of edema, which were also associated with lower mobility abnormal swimming and ultimately death prior to reaching the swim-up stage. This was only associated with the Hyco Reservoir females, which supports the theory that selenium is transferred to offspring through the egg.

Kennedy et al. (2000) – Cutthroat Trout

Kennedy et al. (2000) collected gametes from cutthroat trout (*Oncorhynchus clarki*) from one selenium-impacted and one reference location. Laboratory-based reproductive toxicity tests were conducted with eggs fertilized in the field: percent fertilization, larval survival, time-to-hatch, and deformities in fry surviving to swim-up stage were assessed. Fish from the exposure site had significantly higher concentrations of selenium in their tissues (muscle, liver and eggs) relative to the reference site fish. There were no statistically significant effects on fertilization, time to hatch, percent hatch, survival or incidence of deformities between the exposure and reference treatments, despite mean selenium egg concentrations of 21.2 ug/g dw for exposed fish. The overall incidence of deformities was low (0 to 2.4% among all females) and was actually slightly higher among the reference site fish. Although there were high egg mortalities (up to 100%) for a few females in both the reference and exposure treatments, Kennedy et al. (2000) concluded that this was unrelated to selenium concentrations. Hamilton and Palace (2001) identified multiple concerns regarding this study's methodology and conclusions, which were refuted by McDonald and Kennedy (2002).

Holm (2002); Holm et al. (2003; 2005) – Rainbow Trout and Brook Trout

The data from Holm and coauthors consists of a M.Sc. thesis (Holm 2002) and two peer-reviewed articles (Holm et al. 2003; 2005)[11]. Holm and coauthors collected gametes from rainbow and brook trout from a total of five different streams with varying selenium concentrations (including reference sites) for laboratory-based reproductive toxicity testing. Condition factor and muscle Se concentrations in adult trout were determined. Egg Se concentration, percent fertilization, percent survival to the swim-up stage, larval length and weight, and the frequency of deformity (skeletal, craniofacial, finfold and edema) in surviving fry were determined. Holm et al. (2005) concluded that 15% of a fish population would be affected by a deformity at egg concentrations between 8.8 and 10.5 ug/g ww, and concluded that this relationship was similar to that described by Lemly (1993c) for warm-water centrarchids. Holm et al. (2005) found no relationship between egg tissue residues and the frequency of deformity for brook trout, and commented that the lack of a dose-response for brook trout may indicate a difference in the sensitivity of these closely related species to selenium.

The Holm et al. (2005) conclusions with respect to threshold levels for rainbow trout relative to work by Lemly were not consistent with all aspects of their data. For example, the rainbow trout muscle threshold value was based on a study-specific regression equation needed to convert the selenium egg threshold (10 µg/g ww) established in the reproductive toxicity testing to a corresponding selenium muscle threshold (1.8 µg/g ww). This selenium muscle threshold was compared to a whole-body tissue threshold of 10 to 12.5 µg/g (ww) reported by Lemly (1993c) specific to warm-water centrarchid fish from Belews Lake, to support their conclusion that establishment of a cold-water salmonid tissue residue guideline (TRG) was not warranted. This comparison involved muscle to whole-body selenium concentrations (which are not directly comparable). Additionally, Holm et al. (2005) did not compare their own rainbow trout egg effects threshold to the Lemly (1993b) threshold for eggs/ovaries or other relevant literature. The overall conclusion of Holm et al. (2005) that their data do not support a cold-water salmonid TRG is not supported if their egg data are compared to Lemly's egg threshold values, once the Holm et al. (2005) wet weight egg threshold had been converted to a dry weight number.

[11] This discussion focuses on Holm et al. (2005) since it presents data for all three years in which fish were sampled; also, figures presented in Holm (2002) and Holm et al. (2003) were confirmed by the authors to contain errors which were subsequently corrected in Holm et al. (2005).

De Rosemond et al. (2005) – White Sucker

De Rosemond and colleagues collected white sucker (*Catostomus commersoni*) gametes from a lake in northern Saskatchewan with elevated concentrations of selenium. Embryos were fertilized in the field, water-hardened, and transported to a toxicology laboratory for reproductive toxicity testing. Testing was conducted with water collected from a lake near the site of fish collection which did not have elevated selenium concentrations. Larval survival, macroscopic and microscopic deformities and egg Se concentrations were determined. Total developmental deformities were calculated (combined macroscopic and microscopic deformities). De Rosemond et al. (2005) found that the mean (± standard deviation) percent total developmental deformities was 12.8 ± 4.7%; the majority of macroscopic deformities involved a slight curvature of the spine. Mean egg Se concentrations were 25.9 ± 19.9 µg/g dw[12], which De Rosemond et al. (2005) commented were not substantially different than the concentrations observed by Kennedy et al. (2000). No correlation between Se concentration in white sucker eggs and frequency of deformity was observed.

Muscatello et al. (in review) – Northern Pike

Muscatello et al. (in review) collected northern pike gametes from four locations (one reference location and three locations with varying concentrations of selenium) for laboratory-based reproductive toxicity testing. Muscle and organ Se concentrations (female fish only), egg Se concentrations, percent fertilization, embryo mortality, fry condition factor, and the frequency of deformity (skeletal, craniofacial, finfold and edema) in surviving fry were determined. Reproductive toxicity testing was conducted using water from both the reference and the exposure sites (e.g., eggs from each site were incubated in both reference water and exposure water). Selenium concentrations were significantly higher in fish eggs from the medium and high exposure sites relative to the reference sites (but not the low exposure site). However, there were no significant differences in percent fertilization, embryo mortality, or fry condition factor between any of the sites investigated.

There were no differences in the frequency of deformity in larval fish between the reference and low Se exposure sites (mean egg Se concentrations were 3.19 and 3.80 µg/g dw, respectively). There were significant increases in the frequency of deformity between the reference and medium exposure sites (mean egg Se concentrations were 3.19 and 31.28 µg/g dw, respectively), as well as increases in the frequency of deformity between the reference and the high exposure site (mean egg Se concentrations

[12] Percent moisture was measured at 92.8 ± 0.4% by De Rosemond et al. (2005), which is substantially higher than the assumed moisture content of 75% by other authors.

were 3.19 and 48.23 µg/g dw[13], respectively). Muscatello et al. (in review) determined the threshold egg and female muscle tissue Se concentrations associated with a 20% increase in total deformities relative to the reference site, and calculated threshold values of 32.25 µg/g dw and 14.90 µg/g dw for eggs and muscle tissue, respectively. Muscatello et al. (in review) converted these values into a whole-body fish tissue threshold ranging from 12.77 to 14.96 µg/g dw using the tissue to whole body regression equations provided in USEPA (2004).

2.5 Field and Mesocosm Studies

Skorupa (1998) documented 12 examples where adverse effects to fish (and wildlife) were attributed to selenium poisoning. These included Belews Lake and Hyco Reservoir, two North Carolina waterbodies where selenium was associated with fly ash from coal-fired electric power plants, as well as the Kesterson Reservoir in California, which received selenium-contaminated drainage water from the San Luis Drain (SLD).

Crane et al. (1992) investigated the effects of selenium on fish reproduction and biological community richness using experimental ponds (containing sediment, plants and invertebrates) that received waterborne selenium concentrations of 2 to 25 µg/L. Three fish species (perch *Perca fluviatilis*, grass carp *Ctenopharyngodon idella*, and stickleback *Gasterosteus aculeatus*) were added to the ponds in early summer and left until the next spring. Benthic invertebrates and zooplankton were sampled monthly; there were no major differences in benthic community structure during the study. Perch egg ropes were removed from the ponds and incubated in the laboratory (in control water or with selenium) to assess hatching success. Approximately 50% of the perch and all of the grass carp died (or were not recovered) in the 25 µg/L Se treatment, but there were no deaths in the other treatments. No stickleback were recovered from any ponds, but this was attributed to predation. Although fertilization success was >90% in all but one treatment (unrelated to selenium concentration), none of the perch eggs from the 25 µg/L Se pond hatched. However, perch eggs taken from the control pond and reared in 25 µg/L Se in the laboratory showed no adverse effect on hatching success; this result indicated that water Se concentrations were not causative and implicated the role of maternal transfer of selenium from adult females to their offspring through the eggs. Selenium tissue concentrations in adult perch exposed to 25 µg/L Se were 5.63 µg/g ww in muscle and 6.96 µg/g ww in the gonads; tissue concentrations in the 10 µg/L Se treatment (which did not demonstrate clear reproductive effects) were 3.52 µg/g ww (muscle) and 4.51 µg/g ww (gonads). The authors concluded that exposure to 25 µg/L Se did not affect the benthic invertebrate community but did have significant effects on perch reproduction via dietary uptake.

[13] Only one ripe female pike was available from the high exposure site. Sample sizes ranged from 3 – 5 female for the other sites.

3.0 SELENIUM BEHAVIOUR AND MODE OF ACTION

3.1 Summary of Processes Leading to Dietary Selenium Exposure in Fish

Selenium is released by human activities largely or entirely as inorganic chemical species (i.e., selenite and selenate), and is transported into aquatic habitats predominantly in the dissolved form (Presser and Ohlendorf 1987; Zhang and Moore 1996, 1997; Bond 2000). The greatest risk to fish and birds is widely considered to be from chronic exposure to dietary organoselenium (Finley 1985; Ohlendorf et al. 1986, 1989; Saiki and Lowe 1987; Presser and Ohlendorf 1987; Lemly 1985; Luoma et al. 1992; Presser et al. 1994). The ecological risk posed by selenium releases is therefore largely a function of the degree to which dissolved, inorganic selenium is transformed into dietary organoselenium in receiving environments. This transformation occurs via a complex series of interconnected hydrological, biogeochemical and biological pathways that vary over time, among sites, and among receptor taxa.

3.1.1 Hydrology

Spiralling: When selenium is released to a lotic (flowing water) environment, its fate is determined by a combination of cycling (movement between the various abiotic and biotic matrices) and downstream transport. Cycles are elongated downstream into 'spirals', in a manner analogous to that described for nutrients (Newbold et al. 1982a,b). Selenium differs from nutrients in some important ways (e.g., volatilization may be important, producing what might be termed 'leaky spirals') but the general patterns that have been observed for nutrients apply to all biologically-active materials, including selenium.

The basic unit of material spiralling is spiral length, which is the average distance downstream that a selenium atom travels between one cycle and the next. Long spirals are equivalent to strong flushing; systems with long spirals do not accumulate a large reservoir of selenium in biotic or abiotic matrices. In contrast, systems with short spirals (e.g., slow-flowing reaches or side channels, impoundments, marshes, wetlands) tend to accumulate selenium (Luoma et al. 1992; Skorupa 1998; Lemly 1997b). Spiral length will therefore have a strong influence on spatial patterns of selenium accumulation and thus on the exposure of resident biota.

Spiral length is controlled by a combination of hydrological, geophysical and biological characteristics. In general, spiral length is a function of transport rate (stream flow) and material retention (physical retention and biological uptake). Slow-flowing waters with rapid turnover of organic matter produce short spirals, while fast waters with slow turnover produce long spirals. Spiral length usually increases from stream headwater to mouth due to decreased retention (increased flow, reduced productivity) and decreases in sediment particle size. Spiral length can also change abruptly, such as when a stream enters a wetland or empties into a larger river.

3.1.2 Biogeochemistry

In addition to determining the spatial distribution of selenium, the hydrological characteristics of the receiving system also set the stage for the biogeochemistry of selenium (i.e., environmental partitioning, geochemical speciation and biological transformation). These are highly interdependent processes (e.g., partitioning is influenced by speciation, which may in turn be a function of microbial activity). The resulting distribution of selenium among its various forms determines its physical retention within a system (discussed above), and its bioavailability for uptake by organisms (discussed below).

Partitioning to Particulate Phases: Selenium is most often released into receiving environments in the dissolved phase, but it quickly adsorbs to particulate phases, including detritus, suspended inorganic material, sediments, bacteria and primary producers. Bacteria and primary producers can also absorb selenium, and it is the combination of absorption and adsorption that determines the dietary exposure of consumers. The degree to which selenium partitions to particulate phases depends on the speciation of dissolved selenium, the surface characteristics of the particulate matter, and the geochemical conditions that govern dissolved-particulate reactions. The importance of partitioning to particulate phases is twofold:

1. Particle-bound selenium is more strongly retained in an ecosystem (i.e., short spirals); and,

2. The pathway for nearly all selenium transfer to higher trophic levels is via particulate forms, as animals bioaccumulate dietary selenium to a much greater extent than waterborne selenium (Luoma et al. 1992; Rosetta and Knight 1995; Thomas et al. 1999).

Geochemical Speciation: Selenium partitioning is strongly influenced by geochemical speciation in the dissolved phase. Selenium speciation is determined by factors including pH and redox conditions, the solubility of selenium salts, biological transformation, and chemical reaction kinetics (McNeal and Balistrieri 1989; Masscheleyn et al. 1993).

Selenium has four stable oxidation states:

- Elemental selenium (Se 0);
- Selenide (Se -II), occuring either as organoselenium (substituting for S^{-2} in proteins) or as inorganic selenide salts;
- Selenite (Se IV) as the oxyanion SeO_3^{-2}, an analog to sulfite; and,
- Selenate (Se VI) as the oxyanion SeO_4^{-2}, an analog to sulfate.

Dissolved selenium in aerobic waters is usually dominated by selenate and selenite (Presser and Ohlendorf 1987; Zhang and Moore 1996, 1997), although organoselenium has been found to predominate in highly productive systems where selenium is strongly recycled (Takayanagi and Wong 1984). Selenate is the thermodynamically-predicted stable form in neutral to alkaline, oxic waters, but due to its slow oxidation rate in natural waters, selenite can be an important species. Chemical or microbial reduction of selenate produces selenite, and selenite can be further reduced to insoluble elemental selenium. The reduction of selenate to elemental selenium can dominate the transport of selenium into anoxic wetland sediments (Zhang and Moore 1997), although this elemental pool can be rapidly regenerated into selenate if sediments are re-oxygenated, such as during lake drawdown (Zawislanski and Zavarin 1996). Selenides can also be formed in reducing environments such as anaerobically-active sediments. Inorganic selenides most often occur as insoluble metal selenide salts.

Biological Transformation: Organic selenides are produced when selenate and selenite are absorbed by bacteria, animals, algae and higher plants and reduced intracellularly to biochemical analogues of sulfur. Organic selenides include soluble selenoamino acids and volatile selenides such as dimethyl selenide, dimethyl diselenide and diethyl diselenide (Carvalho et al. 2001). Selenoamino acids are incorporated into the organism's tissues, and ultimately are either consumed by higher trophic levels or released back into the environment (to either the dissolved phase or the detrital pool) when the organism dies (Zhang and Moore 1997). Biogeochemical volatilization can be a substantial loss term in selenium budgets for highly productive systems such as wetlands (Cooke and Bruland 1987; Thompson-Eagle and Frankenburger 1992) and has been applied to the phytoremediation of selenium-contaminated soil and water (Hansen et al. 1998; Terry and Zayed 1998).

3.1.3 Biology

Selenium enters the aquatic food web when it is taken up from the dissolved phase by basal resources (mainly algae and detritus) and passed along feeding links to invertebrates and higher trophic level organisms. Uptake via food is widely considered to be the most important route of selenium transfer to invertebrates and higher trophic level organisms (Ohlendorf et al. 1986; Saiki and Lowe 1987; Presser and Ohlendorf 1987; Lemly 1985; Presser et al. 1994; Luoma et al. 1992; Rosetta and Knight 1995; Thomas et al. 1999). Selenium concentrations in zoobenthos and fish in the field have been shown to correlate poorly with measured aqueous concentrations (Skorupa and Ohlendorf 1991; Luoma et al. 1992; Fan et al. 2002; Wayland and Crosley 2006), and to respond very slowly (with lag times on the scale of years) to declines in aqueous concentrations (Lemly 1997b; Swift 2002). Some authors have gone so far as to conclude that "[t]he accumulation of waterborne selenium by aquatic invertebrates has been shown to be relatively unimportant in the ecotoxicology of selenium" (Malchow et al. 1995). The exposure of fish is therefore a function of the degree to which selenium is bioaccumulated by lower trophic levels, the pattern of feeding links between fish and potential prey items, and the degree to which dietary selenium is then accumulated by fish.

Bioavailability to Lower Trophic Levels: Environmental partitioning, geochemical speciation and biological transformation interact to determine the bioavailability of selenium to lower trophic level organisms (Luoma et al. 1992). Elemental selenium, the primary form found in sediments (Van Derveer and Canton 1997), has little toxicological significance to most organisms (Combs et al. 1996; Schlekat et al. 2000). Selenite is both more bioavailable and more toxic than selenate (Lemly et al. 1993; Maier et al. 1993; Skorupa 1998; Amweg et al. 2003), leading Skorupa (1998) to conclude that "selenite-dominated aquatic systems have 'supercharged' food chains compared to selenate-dominated systems". Organoselenium is the most bioavailable form, and is taken up by algae much more readily than inorganic forms (Lemly et al. 1993; Maier et al. 1993; Amweg et al. 2003).

Fish Diet Composition: There can be large differences in selenium bioaccumulation among potential prey items within a system, due to interspecific (Linville et al. 2002; Schlekat et al. 2004) or intraspecific (Malloy et al. 1999; Besser et al. 1996) variation in factors such as diet, physiology, and microhabitat. The dietary selenium exposure of a fish can therefore be strongly influenced by the availability of various prey species and the fish's choice of food (Linville et al. 2002; Schlekat et al. 2004; Stewart et al. 2004). This is a highly site-specific issue.

Bioaccumulation of Dietary Selenium: Fish ingest predominantly the highly bioavailable (Wang and Lovell 1997) organic forms of selenium, because this is the form that predominates in their prey (e.g., Fan et al. 2002; Vickerman et al. 2004). The factors controlling whether selenium occurs at progressively higher concentrations with each step in a food chain (i.e., biomagnifies), however, are not well understood. Some studies report increasing concentrations of selenium in aquatic food chains (Liu et al. 1987; Barwick and Maher 2003; Lemly 1985; Lemly and Smith 1987; Orr et al. 2006), whereas others report no biomagnification (Saiki et al. 1993; Besser et al. 1993).

3.2 Toxic Action

Cardwell et al. (1976) reported the occurrence of external haemorrhaging within 48 h of exposure to acutely lethal selenium concentrations in water for juvenile fathead minnow, channel catfish and goldfish, whereas adult brook trout displayed more severe tissue damage including discolouration, lesions, and epithelial tissue necrosis and sloughing. Ellis et al. (1937; cf. Cardwell et al. 1976) reported that injecting 3.0 µg/g Se intraperitoneally into channel catfish resulted in death within a few hours, and that daily injections of 0.2 µg/g Se for five days resulted in exophthalmia (eyeball protrusion), anemia, leukopenia (decreased leukocytes), and tissue degeneration in the liver, kidney and spleen.

Selenium is chemically similar to sulphur, and substitution of selenium for sulphur in protein formation is one potential mode of toxic action for selenium (Palace et al. 2004). This can lead to the incorporation of organoselenium (e.g., selenomethionine) into eggs, which is then available to developing embryos during yolk consumption. Palace et al. (2004) investigated the potential of oxidative stress as another mechanism for selenium toxicity; this was based on observations of edema in rainbow trout larvae similar to edema in fish exposed to contaminants known to cause oxidative stress. The authors used rainbow trout eggs or embryos to measure superoxide radical production in the presence of selenomethionine, and found that there was generally little change in response except for a significant relatively short-lived peak corresponding to the eleuthroembryo stage of rainbow trout embryo development (the point at which the heart, circulatory system and liver are functioning). The authors noted that pericardial edema associated with selenium exposure tended to occur at approximately the same time that liver function was established in rainbow trout. Palace et al. (2004) suggested that enzyme activity (methioninase) may be causing deformities in offspring of adult fish exposed to selenomethionine.

3.3 Interactions With Other Elements or Contaminants

Arsenic has been shown to act antagonistically with selenium, reducing adverse effects from dietary uptake including reproductive abnormalities and failures (Hoffman et al. 1992; Stanley et al. 1994). The presence of elevated selenium concentrations can reduce mercury bioaccumulation in freshwater environments (Belzile et al. 2006). For example, Southworth et al. (2000) reported that bioaccumulation of mercury in largemouth bass increased considerably (from 0.02 to 0.61 µg/g over an 8-yr period) following elimination of selenium-contaminated fly ash discharge from a power plant into a Tennessee quarry. However, the previously elevated selenium concentrations in the quarry (20 – 30 µg/L Se) were associated with high incidence of gross abnormalities in fish, although their incidence declined and eventually disappeared within five years of the selenium input being eliminated. Southworth et al. (2000) postulated that selenium inhibited methylmercury formation, rather than blocking its accumulation by fish.

Increasing sulphate concentrations appear to modify selenate toxicity, but not other forms of selenium. Maier et al. (1993) investigated the influence of sulphate on the acute toxicity of various forms of selenium (selenite, selenate, seleno-DL-methionine, and seleno-DL-cystine) to *Daphnia magna*. Daphnids were exposed to each form of selenium at its 48-h LC50 concentration, with sulphate concentrations ranging from 10 to 163 mg/L. Increasing sulphate concentration had the greatest influence on selenate toxicity, reducing it by as much as 93% at the highest sulphate concentration tested. Selenite concentrations were reduced at sulphate concentrations up to 81 mg/L, but not at higher concentrations. Increasing sulphate concentrations did not affect seleno-DL-methionine toxicity. Brix et al. (2001) also investigated the influence of sulphate concentrations on the acute toxicity of selenate to three freshwater invertebrates and to fathead minnow, and found that selenate toxicity generally decreased as sulphate concentrations increased. Brix et al. (2001) compiled similar data from other studies in order to develop a relationship between selenate and sulphate concentrations that could be incorporated into an acute water quality criterion (CMC) for selenate, similarly to the way that hardness is applied for other methods; this led to the development of a proposed sulphate-dependent equation for setting a selenate CMC that is similar to the equation included in USEPA (2004a).

3.4 Implications of Winter Stress Syndrome (WSS) on Selenium Toxicity

Winter stress syndrome (WSS) may develop when a fish requires metabolic energy due to exposure to a stressor (i.e., elevated Se concentrations) at a time that the fish lacks sufficient energy reserves to deal with the increased metabolic requirements. This situation can occur in winter when fish species reduce their feeding activity in favour of utilizing stored lipids (i.e., fat). Lipid depletion for over-wintering fish has been documented for multiple species. However, exposure to selenium results in a stress

response, which increases fish respiration rates (i.e., oxygen consumption) and therefore metabolic rates. Fish consume their stored lipids at an increased rate, which can lead to mortality if stored lipids are inadequate to last until feeding rates increase as water temperature rises.

3.4.1 Experimental Evidence of Se-Induced WSS

Lemly (1993a) described the combined effect of simulated winter conditions and elevated Se concentrations in a 180-d study using juvenile bluegill sunfish (*Lepomis macrochirus)* and coined the term WSS. Fish were exposed to 5.1 µg/g dw Se in food and 4.8 µg/L Se in water under warm (20°C) and cold (4°C; achieved by decreasing water temperature at the rate of 2°C per week during the first eight weeks of the study) conditions. Negative controls for both water temperature treatments were included (0.82 µg/g dw Se in food and 0.16 µg/L Se in water). Photoperiods were also adjusted for the cold water treatments from 16:10 hours light:dark to 10:16 hours light:dark during the first 60 days of the experiment to further simulate winter conditions. Fish were fed at a rate of 3% body weight per day.

Lemly (1993a) found that fish in the warm water treatments continued to feed, and did not demonstrate decreased lipid contents despite elevated oxygen consumption rates in the warm water Se treatment. Fish in the cold water treatments demonstrated increased oxygen consumption and decreased condition factors and lipid contents relative to the warm water treatments. Lemly (1993a) found that bluegill sunfish exposed to simulated winter conditions and selenium resulted in a significant decrease in blood-related biochemical parameters[14] and an increase in oxygen consumption of between 15 – 30% relative to the negative control. Lipid content in these fish dropped from 12 - 14% (at test initiation) to 6 - 11% (by Day 60) and to 3-8% (by Day 120 and beyond). Cumulative mortality during the 180 day exposure was less than 6% for both negative control treatments as well as the warm water Se treatment, but was 33.8% for the cold water Se treatment.

Additional laboratory-based investigations of WSS by the USEPA are currently in the planning phase (C. Delos, USEPA, personal communication). The study is intended to repeat the original Lemly (1993a) study design with the addition of a range of temperature regimes and more realistic dietary selenium exposure scenarios (tentatively, fish food organisms exposed to elevated selenium, rather than fish food dosed with selenomethionine).

[14] Significant decreases were observed in hematocrit (i.e., the percentage by volume of packed red blood cells in a given sample of blood after centrifugation), mean hemoglobin concentrations, and percentage of erythroblasts (i.e., nucleated cells which form red blood cells).

3.4.2 Cited Implications of WSS for Selenium Management

Lemly (1993a) has been cited as the rationale for considering seasonal effects by other papers addressing selenium (and other aquatic hazards) management issues, including two subsequent papers by the same author:

Lemly (1996a)

Lemly (1996a) summarized the findings of Lemly (1993a), and further described the components of WSS (i.e., increased respiratory demands and lipid depletion). Lemly (1996a) concluded that the critical lipid content for survival of juvenile centrarchids was 5%, and noted that three conditions are necessary to induce WSS:

- A significant metabolic stressor must be present;
- Cold water temperatures must be present (defined by the author as <10°C); and,
- Fish must respond to cold water by reducing activity and feeding.

As a result of these conditions, the probability of a fish population developing WSS was defined by Lemly (1996a) as a function of: a) the potential for a given stressor to increase fish metabolic activity; and, b) time of year. Lemly (1996a) concluded that young centrarchids in North America were at high risk of developing WSS (especially young-of-the-year and yearling fish) and suggested that young cyprinids and percids were also susceptible to WSS based on evidence that certain species also demonstrated decreased lipids during overwintering. Lemly (1996a) concluded that "the occurrence of WSS for a few weeks can eliminate 30% or more of a year class of fish".

Lemly (1997a)

Lemly (1997a) summarized the findings of Lemly (1993a) and repeated the discussion provided in Lemly (1996a). Lemly (1997a) noted that "many species of fish in temperate regions of North America and Europe undergo a normal seasonal cycle of reduced feeding and activity" and concluded that "fishes other than centrarchids are likely to experience WSS if they reduce feeding and activity during cold weather". Lemly (1997a) also concluded that the occurrence of WSS for a few weeks can eliminate 30% or more of a year class of fish.

3.4.3 Extrapolation of WSS to Yellowstone Cutthroat Trout

Lemly (1993a) provides the primary experimental evidence that Se-induced WSS can impact fish survival. This section outlines the convergence of factors examined by Lemly (1993a) and discussed in Lemly (1996a) relative to the state of knowledge regarding the potential implications of those factors for inducing WSS in cold-water salmonid populations such as Yellowstone cutthroat trout (YCT; *Oncorhynchus clarkii bouvieri*) Data from other fish species are provided for context where appropriate.

The potential for Se-induced WSS to occur for a particular fish species is a function of:

- The relationship of the stress response (e.g., increased oxygen consumption leading to increased metabolic energy requirements) induced in the species as a result of exposure to elevated concentrations of Se;

- The energy budget of the fish at a given time of year—consideration of lipid loss (i.e., the amount of lipids that are burned to satisfy total metabolic requirements) versus lipid gain (i.e., the degree of winter feeding, if any, and the resulting energy obtained); and,

- The critical lipid content below which fish mortality is likely to occur.

Each of the above topics is discussed further below.

Relationship of Stress Response to Se Exposure

There are minimal data regarding the relationship between Se exposure and the induction of a stress response for fish.

For warmwater fish:

- Lohner et al. (2001) found significant decreases in plasma protein levels and other blood parameters for sunfish (a pattern similar to Lemly 1993a) from a coal-ash impacted site; however, these decreases in blood parameters did not translate to changes in condition factor, liver-somatic indices or fish length and weight.

- Conversely, Staub et al. (2004) found no evidence of increased oxygen consumption in eastern mosquitofish (*Gambusia holbrooki*) inhabiting a coal ash settling basin relative to a reference location, despite evidence of significant maternal transfer of Se to larval offspring. This finding is not consistent with Lemly 1999a's observation of an increase in oxygen consumption of 15 – 30% for bluegill sunfish exposed to Se (relative to negative controls).

For cold-water salmonids:

- Miller et al. (2006) found that the stress responses of two salmonid fishes (brook trout and rainbow trout) sampled from a creek in proximity to a coal mine were different - for example, plasma cortisol levels were elevated in brook trout, but not rainbow trout. No differences in plasma glucose levels were observed for either species as a result of exposure to selenium. Further investigation by Miller and coauthors is underway. Oxygen consumption rates or other measures of metabolic energy expenditure directly comparable to Lemly (1993a) were not measured.

- Hodson et al. (1980) found that condition factor, plasma glucose levels and hemocrit values in rainbow trout were not reduced when post-hatch fry were exposed to selenium concentrations as high as 50 µg/L for 23 weeks. Slight (but not statistically significant) decreases in plasma glucose were noted for fish exposed to selenium for 44 weeks, but not for hemocrit or condition factor. Conversely, Lemly (1993a) found significant decreases in hemocrit values for bluegill sunfish exposed to selenium[15].

Overall, the evidence suggests that a stress response may be induced in cold-water salmonids as a result of exposure to selenium; however, the data are insufficient to conclude that the magnitude of the stress response will be the same for all species as was observed by Lemly (1993a) for bluegill sunfish.

Energy Budgets of Fish

Lemly (1996a; 1997a) concluded that fishes other than centrarchids are likely to experience WSS if they reduce feeding and activity during cold weather, including young cyprinids and percids. Species-specific feeding behaviour during winter influences the ability of a fish to maintain an adequate energy budget. For example, the significant decrease in lipid content of bluegill sunfish (and thus mortality) observed by Lemly (1993a) follows from the fact that feeding rates in fish exposed to simulated winter conditions were decreased by as much as 90% relative to warm-water controls. However, not all cold water salmonids demonstrate a similar change in feeding behaviour in response to water temperature as bluegill sunfish.

- **Yellowstone Cutthroat Trout:** YCT are adapted to cold water, and have been found to be actively feeding under 1 meter of ice cover on Yellowstone Lake at water temperatures between 0 and 4°C. YCT populations exist in streams within Yellowstone National Park with summer maximum water temperatures of between 5 and 8°C (Varley and Gresswell 1988).

[15] Lemly (1993a) did not measure plasma glucose levels, and therefore, no other comparison to Hodson et al. (1980) can be made.

- **Rainbow Trout:** Biro et al. (2004) found that rainbow trout (*Oncorhynchus mykiss*) held during simulated winter conditions[16] did not reduce their feeding activity and concluded that starvation of young trout during overwintering under field conditions was not the result of an inability to feed or metabolize food at low temperatures. Rather, Biro et al. (2004) concluded that starvation in rainbow trout reflects the relationship of prey densities to the severity of winter conditions. Connolly and Peterson (2003) found no evidence that rainbow trout feeding was reduced during a 111-d experiment that simulated three different winter water temperatures (3, 6 and 9°C) representing the distribution of rainbow trout in the wild (southern California to southern Alaska).

- **Kokanee Salmon:** Steinhart and Wartsbaugh (2003) found that landlocked sockeye salmon (*Oncorhynchus nerka*) continued to actively feed in high-mountain lakes (Sawtooth Valley, Idaho) during over-wintering conditions.

There is also conflicting evidence regarding the tendency of perch to reduce feeding during winter. Perch (*Perca fluviatilis*) were found to feed during a 153-d winter in a ice-free lake with an average water temperature of <6°C; minimal over-wintering mortality was observed (Eckmann 2004). Conversely, Post and Evans (1989) found significant mortality in yellow perch (*Perca flacescens*) overwintering in enclosures in a Canadian lake under similar duration and water temperature as Eckmann (2004), but with ice coverage. Eckmann (2004) noted that perch are visual predators, and therefore, differences in light penetration (i.e., the presence or absence of ice coverage) may cause differences in both lake ecology and perch feeding behaviour. Site-specific conditions are likely significant factors influencing fish feeding behaviour.

Critical Lipid Content

Lemly (1996a) concluded that the critical lipid content for survival of juvenile centrarchids was 5%; however, this value may not be applicable to cold-water salmonids.

- **Rainbow Trout:** Biro et al. (2004) conducted stocking experiments with hatchery-raised rainbow trout (*O. mykiss*) placed into small lakes in south central British Columbia. Mortality rates of young-of-the-year rainbow trout as a result of overwinter starvation in these lakes was found to be strongly correlated with fish size; further, fish size was strongly correlated with lipid content. These field experiments were combined with laboratory starvation studies to determine a critical lipid content of 1% for young-of-the-year rainbow trout. Connolly and Peterson (2003) found no mortality in rainbow trout with lipid contents ranging from approximately 4 to 5% during a 111-d simulated winter exposure at 3°C.

[16] Water was maintained at 3°C for 100 days during winter in outdoor artificial ponds with translucent Styrofoam covers to simulate ice coverage.

- **Kokanee:** Steinhart and Wartsbaugh (2003) conducted a starvation experiment with six field-collected kokanee and found that the average lipid content below which fish mortality was observed was 2.3%.

- **Brown Trout:** Berg and Bremset (1998) noted that the lower limit of fat content necessary for survival of small salmonid fry was not known; they found that brown trout (*Salmo trutta*) fry that survived overwintering conditions in a river in Norway had a mean fat content as low as <1%[17].

Implications for Yellowstone Cutthroat Trout

The potential for WSS-induced mortality to occur for a particular fish species is a function of the rate at which elevated Se causes increased metabolic energy requirements, the overall energy budget of the fish, and the critical lipid content below which fish mortality is likely to occur. These three factors vary between different fish species; we cannot conclude that all species will demonstrate WSS to the same degree as reported by Lemly (1993a) for bluegill sunfish. Extrapolation of Lemly (1993a) to cold-water salmonids is especially problematic due to the fact that cold-water fish exhibit different feeding strategies that may limit the likelihood of WSS-induced mortality.

[17] Berg and Bremset (1998) did not report a range of fat content values for brown trout fry.

4.0 SELENIUM GUIDELINES BASED ON WATER CONCENTRATIONS

This chapter presents a review of selenium guidelines based on water concentrations, including the derivation of USEPA's water quality criteria (WQC) and other approaches to deriving water quality guidelines (WQGs) for selenium. A brief clarification regarding terminology is also provided, as this can vary between jurisdictions and regulatory agencies.

USEPA uses the term "criterion" to represent a constituent concentration associated with a degree of environmental effect upon which scientific judgement may be based (i.e., a designated concentration or level that when not exceeded should not result in unacceptable effects to aquatic organisms). Criteria are usually expressed in terms of numerical concentrations (in water, sediment or tissue), but they can also be expressed as narrative statements. Other countries such as Canada and Australia use the term "guideline" in the same context that USEPA uses "criterion", although the derivation methods and levels of protection are different (e.g., in Canada, WQG are intended to protect all species at all times, whereas the USEPA approach is intended to provide 95% protection).

WQC differ from "standards" in that standards are legal entities used in regulation or enforcement for protection of environmental quality for a particular waterbody or designated usage. State or tribal regulatory agencies may adopt USEPA WQC as standards without modification, or they may consider site-specific conditions and other factors (e.g., legal, social, economic or technological) when setting standards.

4.1 Derivation of the USEPA's Water Quality Criteria (WQC) for Selenium

The US Environmental Protection Agency (USEPA) published ambient WQC for the protection of aquatic life for selenium in 1980 (USEPA, 1980). These WQC, which were applicable to inorganic selenite, were 260 µg/L as a maximum concentration that was not to be exceeded at any time and 35 µg/L as a 24-h average concentration.

The USEPA subsequently revised its ambient WQC for selenium in 1987 (USEPA, 1987). Data were presented separately for selenite (Se IV) and selenate (Se VI):

- **Selenite (Se IV):** Freshwater acute toxicity data were available from studies conducted with 12 fish and 11 invertebrate species; overall, the Species Mean Acute Values (SMAVs)[18] ranged from 203,000 to 340 µg/L (35,000 to 1,600 µg/L when only fish were considered). Freshwater chronic toxicity data were available for two fish and two invertebrate species; chronic values (geometric mean of the no observed effect concentration [NOEC][19] and lowest observed effect concentration [LOEC]) ranged from >47[20] to 693 µg/L.

- **Selenate (SeVI):** Freshwater acute toxicity data were available for 4 fish and 8 invertebrate species; SMAVs ranged from 442,000 to 65.38 µg/L (66,000 to 5,500 µg/L for fish only). Freshwater chronic toxicity data were available for two fish and one invertebrate species; chronic values ranged from 1,999 to 565.5 µg/L.

The methodology described by USEPA (1985) for deriving WQC for the protection of aquatic life is briefly summarized here. This two-number approach provides for derivation of acute and chronic criteria, the Criterion Maximum Concentration (CMC)[21] and the Criterion Continuous Concentration (CCC), respectively, for a particular substance.

- To calculate the CMC, acute toxicity data for fish and invertebrates from acceptable studies are compiled, SMAVs and Genus Mean Acute Values (GMAVs; geometric mean of SMAVs for each genus) are calculated, GMAVs are ranked in order of increasing toxicity and assigned cumulative probabilities, and the four GMAVs[22] with cumulative probabilities closest to 0.05 are used in an equation to determine a Final Acute Value (FAV), which is then divided by two to obtain the CMC.

- The CCC is the lowest of the Final Chronic Value (FCV), Final Plant Value (FPV), or Final Residue Value (FRV). The FCV is calculated using chronic toxicity data for aquatic fish and invertebrates; it can be calculated in the same manner as the FAV (if there are enough data), or by dividing the FAV by a Final Acute-to-Chronic Ratio. Alternatively, the FPV is obtained from aquatic plant toxicity data, and the FRV is obtained by dividing a maximum permissible tissue concentration by a bioaccumulation or bioconcentration factor (to express it as a water concentration).

[18] The SMAV is the geometric mean of all the acceptable acute toxicity test results for a particular species.
[19] The geometric mean of the NOEC and LOEC is the chronic value endpoint used by USEPA (1985) for expressing chronic toxicity results for criteria derivation.
[20] The chronic value of >47 µg/L was from Hodson et al. (1980); 47 µg/L Se was the highest selenium concentration tested. The authors reported a reduction in percent hatch (but only 3.5% lower than the negative control), and no effect on fry mortality.
[21] The CMC is a 1-h average concentration that is not to be exceeded more than once every three years on average, and the CCCis a 4-d average concentration that is not to be exceeded more than once every three years on average.
[22] When there are fewer than 59 GMAVs, this will always be the lowest four GMAVs.

USEPA (1987) documents how application of the USEPA (1985) methodology for WQC derivation would yield the following FAVs and FCVs (and corresponding CMCs and CCCs) for selenium in freshwater:

- Selenite (Se IV): The FAV was 372 µg/L, which when divided by two gives a CMC of 186 µg/L. There were not enough chronic toxicity data to allow the FCV to be calculated in the same manner as the FAV. Therefore the FCV, calculated by dividing the FAV by a final acute-to-chronic ratio (ACR) of 8.314, would be 44.7 µg/L. This final ACR was calculated as the geometric mean of five ACR values derived for freshwater and marine species ranging from 5.586 to 13.31 (the marine ACRs were included because they were within the range of the freshwater ACRs). However, one chronic study reported a chronic value of 55.2 µg/L for rainbow trout (a commercially and/or recreationally important species), which was considered too close to the calculated FCV of 44.7 µg/L to be protective. Therefore, the chronic value of 55.2 µg/L was substituted for the calculated FCV and divided by two (to provide a reasonable measure of protection) to obtain a CCC of 27.6 µg/L.

- Selenate (Se VI): The FAV was 25.65 µg/L, which when divided by two gives a CMC of 12.82 µg/L. The low FAV is surprising given that selenium IV is considered to be the more toxic form of selenium. Most of the species listed in USEPA (1987) were tested with both forms of selenium and in all but one case selenium IV was more toxic; however, the toxicity data set was not large and one gammarid amphipod species was much more sensitive to selenium VI than to selenium IV and this influenced the FAV. The FCV, calculated by dividing the FAV by a final ACR of 2.651, was 9.67 µg/L. There were three individual ACRs available (for rainbow trout, fathead minnow and *Daphnia magna*), ranging from 2.651 to 16.26. However, rather than use the geometric mean of the three values, as described in USEPA (1985), only the lowest ACR was used (on the basis that it was for a species with the lowest acute toxicity for which chronic data were also available).

However, while the calculated FAVs and FCVs described above were reported in USEPA (1987), they were not recommended as the national WQC for selenium in that document. Instead, freshwater criteria were determined based on the findings of field studies in Belews Lake, NC (and by studies showing that dietary exposure to selenium resulted in adverse effects), which indicated that fish were adversely affected by chronic exposures at selenium water concentrations of 10 µg/L but not at concentrations of 5 µg/L. Therefore, USEPA (1987) adopted 5 µg/L as the CCC; because there were no field studies on acute effects, a final acute-to-chronic ratio of 7.993 was used to back-calculate an FAV of 39.96 µg/L and generate a CMC of 20 µg/L. These criteria values were established for selenium, and did not distinguish between its different forms.

Some researchers have argued that the USEPA (1987) WQC of 5 µg/L is not sufficiently protective and should be lowered. For example, Lemly (1993b) recommended that selenium concentrations of 2 µg/L or more could be hazardous to sensitive species (but did acknowledge that all species would not necessarily be affected) and that even concentrations <1 µg/L could be of concern in terms of bioaccumulation under certain conditions. Hamilton and Lemly (1999) subsequently recommended a WQC of 2 µg/L, and Skorupa (1998) also recommended that the WQC be <5 µg/L.

The Federal Register (1996) described a proposed rule for a new CMC for selenium in the Great Lakes (the CCC would remain unchanged). This CMC, proposed by the USEPA, was intended to consider the relative toxicities of different forms of selenium (i.e., selenite and selenate) to aquatic organisms previously reported in USEPA (1987), as well as the potential for the toxicity of these different forms to be additive. The equation for this proposed CMC was:

$$CMC_{Se} = 1 / [(f_1/CMC_1) + (f_2/CMC_2)]$$

Where: f_1 = the fraction of total selenium treated as selenite[23];
f_2 = the fraction of total selenium treated as selenate;
CMC_1 = 185.9 µg/L (the CMC for selenite); and,
CMC_2 = 12.82 µg/L (the CMC for selenate; differs from USEPA [1987]).

Using this proposed equation, if all selenium was present as selenite then the CMC would be 185.9 µg/L, if all selenium was present as selenate then the CMC would be 12.82 µg/L, and if the concentrations of selenite and selenate were equal then the CMC would be 23.99 µg/L. In its current list of national recommended WQC, USEPA (2004b) lists 5 µg/L as the CCC for selenium and the above equation for calculating a CMC for selenium (with a caveat that USEPA is working on this criterion and that it could change substantially in the near future).

Canton (1999) also recommended that separate acute WQC be developed for the selenite and selenate forms of selenium, and used the USEPA's updated (at that time) toxicity database to propose re-calculated CMCs for each form of selenium. Using the USEPA (1985) criteria derivation methodology, the revised FAV for selenite increases to 437 µg/L and the CMC is 219 µg/L, whereas the FAV and CMC for selenate are now 826 and 413 µg/L, respectively. While the selenite CMC increased approximately 18%, the increase in the CMC for selenate was considerably higher. This change was due to the increased number of toxicity studies available for a larger number of genera, and the fact that additional studies with gammarid amphipods resulted in a higher GMAV than the value published in USEPA (1987), such that one very low toxicity result was no longer driving the selenate CMC calculation. This higher selenate CMC is also consistent with the fact that selenate is less toxic than selenite, and should therefore have a higher CMC.

[23] For the purpose of this equation, USEPA proposed that forms of selenium other than selenite and selenate be assumed to be half as toxic as selenite and half as toxic as selenate.

The most recent draft of USEPA's revised WQC document for selenium (USEPA, 2004a) proposes a fish tissue residue concentration as the CCC for selenium, which has sparked considerable debate (see Chapter 5 for a discussion of the rationale regarding tissue residue thresholds). However, in terms of a revised acute criterion, USEPA (2004a) has proposed that there be separate criteria for selenite and selenate, and that they be derived from studies conducted with each form of selenium rather than using the value from USEPA (1987) that was back-calculated from Belews Lake field data. The proposed acute CMCs are 258 µg/L for selenite, and would be a sulphate-dependent equation for selenate:

$$\text{CMC (selenate)} = \exp(0.5812\ [\ln(\text{sulphate concentration})] + 3.357).$$

At a sulphate concentration of 100 mg/L, the CMC for selenate would be 417 µg/L. These CMCs are 24-h average concentrations that are not to be exceeded more than once in three years.

4.2 Other Approaches to Deriving Water Quality Guidelines (WQGs) for Selenium

The Idaho Department of Environmental Quality has water quality standards for various water uses within the state (IDEQ 2006). For selenium, the CMC and CCC for protection of aquatic life are 20 and 5 µg/L, respectively. IDEQ has separate water quality designations based on water temperature (i.e., Cold, Salmonid Spawning, Seasonally Cold, and Warm). For example, the temperature requirement under a Cold designation[24] is ≤22ºC with a maximum daily average of ≤19ºC, and the temperature requirement under a Warm designation is ≤33ºC with a maximum daily average of ≤29ºC. Waters designated as Cold are considered "appropriate for the protection and maintenance of a viable aquatic life community for cold water species", and because of the potential for spring and fall spawning fish populations to be present during much of the year, CCC values are the applicable water quality standards for toxic substances (i.e., 5 µg/L for selenium).

The Canadian WQG for protection of aquatic life for selenium is 1 µg/L (CCME 2005), a value that was originally published by CCREM (1987). CCREM (1987) summarized ranges of acute and chronic toxicity data for freshwater organisms that had been compiled for the now-outdated USEPA (1980) selenium criteria document. CCREM (1987) also reported existing WQGs, ranging from 1 µg/L (IJC 1981) to 35 µg/L (USEPA 1980). According to CCREM (1987), the IJC (1981) value of 1 µg/L was intended to protect aquatic life in the Great Lakes because field studies had indicated that

[24] There is an additional bull trout temperature criterion that applies to certain designated geographical areas (≤13ºC maximum weekly temperature June to August for juvenile rearing, and 9ºC daily average temperature September to October for spawning).

selenium concentrations in the range of 5 to 10 µg/L were acutely lethal to fish through food web contamination. Rather than independently deriving a Canadian selenium guideline value for water using available aquatic toxicity data, CCREM (1987) adopted 1 µg/L as the Canadian WQG for protection of aquatic life for selenium.

British Columbia (Canada) has a WQG for protection of freshwater aquatic life of 2 µg/L for selenium, which represents a maximum concentration that should not be exceeded (BCMOE 2001). In addition, BCMOE also has an interim sediment guideline (2 µg/g dw) and an interim whole-body fish tissue guideline (1 µg/g ww); these numbers were developed to provide a basis for the water quality guideline.

- **Water Quality Guideline:** BCMOE (2001) summarized available toxicity data for freshwater fish and reported that effects in acute studies (96-h exposure, lethal or sublethal endpoints) occurred at concentrations ranging from 5 to 126,600 µg/L (96-h LC50s ranged from 620 to 96,800 µg/L), and that effects in chronic studies (i.e., generally defined by BCMOE as exposures >96h) occurred at concentrations between 5 and 40,000 µg/L. BCMOE (2001) identified several studies[25] that reported adverse effects to fish occurring at 10 µg/L, including the Belews Lake reproductive and population failures (e.g., Hermanutz 1992; Schultz and Hermanutz 1990; Hermanutz et al. 1992; Gillespie and Baumann 1986; Cumbie and VanHorn 1978). It was also noted that those studies involved lentic (still water) systems where selenium would be expected to accumulate in organically enriched sediments and cycle through the benthic food web, and that selenium exposures in those studies likely involved both water and diet. BCMOE (2001) used an LOEC of 10 µg/L and a safety factor of 5 to derive a WQG of 2 µg/L. The safety factor was reduced from 10 to 5 because the ratio between selenium effect concentrations and background concentrations was low, because using the higher safety factor would have resulted in a guideline equivalent to background concentrations, and because of the observation in Belews Lake that adverse effects to fish were not observed at 5 µg/L (also noted by USEPA 1987 as the rationale for selecting their CCC for selenium).

- **Sediment Guideline (Interim):** BCMOE (2001) used data from VanDerveer and Canton (1997) that showed that the upper limit for selenium concentrations in uncontaminated sediments and soil was 2 µg/g dw and that the 10th percentile for predicted adverse effects in sediment was 2.5 µg/g dw, and chose 2 µg/g dw as an interim sediment guideline. They also used the work of Canton and VanDerveer (1997) on correlations between sediment and water selenium concentrations and total organic carbon (TOC) to estimate that at a sediment selenium concentration of 2 µg/g dw and 5% TOC, the corresponding water selenium concentration would be 2.2 µg/L.

[25] These studies were all conducted with warm-water fish species.

- **Tissue Guideline (Interim):** BCMOE (2001) took the whole-body fish tissue threshold of 6 µg/g dw proposed by DeForest et al. (1999) and Brix et al. (2000) for cold-water fish (see Section 5.0 for additional discussion of this proposed threshold), and applied an assumed 80% moisture content to obtain a wet weight "safe level" concentration of 1.2 µg/g ww, which was rounded to 1 µg/g ww as the interim whole-body tissue guideline. The 1 µg/g ww guideline is below background fish tissue concentrations in some areas (Carmichael and Chapman 2006). BCMOE (2001) then used a bioaccumulation factor (BAF) of 500 (Hermanutz et al. 1992) to determine that a selenium water concentration of 2.4 µg/L should be protective in terms of selenium accumulation in fish tissue.

Australia derived its WQGs for selenium based on concentrations in water rather than in tissue (ANZACC/ARMCANZ 2000). The freshwater guideline for total selenium was derived based on 12 acute and chronic data points from studies with fish, crustaceans, aquatic insects and phytoplankton. The freshwater "high reliability" trigger value for total selenium is 11 µg/L, which was determined using a statistical distribution method at 95% protection and did not specifically consider bioaccumulation. The 99% protection level for total selenium is 5 µg/L; this protection level is recommended for slightly to moderately disturbed systems where there are no site-specific data to adjust for bioaccumulation. ANZECC/ARMCANZ (2000) also has a WQG for Se IV, which was derived from acute toxicity data for fish and aquatic insects. The freshwater "low reliability" trigger value for Se IV is 11 µg/L, and does not consider bioaccumulation. In most cases, using the total selenium trigger value is preferred due to its higher reliability.

Adams et al. (2000) recommended an approach for developing site-specific water quality guidelines for selenium. In particular, they considered differences between lotic (flowing water) and lentic (still water) environments with respect to selenium biogeochemistry and its bioaccumulation in tissues. The authors found that there was an approximately 10-fold difference in selenium BAFs for fish from lotic and lentic environments. In addition, there was little change in tissue residue concentrations in fish from lotic environments until water selenium concentrations reached approximately 13 µg/L, whereas tissue residue concentrations in fish from lentic environments started to increase at water selenium concentrations closer to 1 µg/L. This difference supports the need for consideration of site-specific conditions with respect to selenium thresholds. Toll et al. (2005) and Brix et al. (2005) describe a methodology for setting site-specific water quality guidelines based on a tissue residue guideline, and provide an example application using selenium. Briefly, site-specific water and tissue residue data are compiled; if the tissue data are below the appropriate tissue residue guideline chosen for comparison, then the water concentration is considered protection. If the tissue residue guideline is exceeded, then modelling and Monte Carlo analysis are used to determine a suitable site-specific water quality guideline based on the site-specific tissue and water chemistry data.

5.0 SELENIUM GUIDELINES BASED ON TISSUE RESIDUE CONCENTRATIONS

This section describes the ongoing debate regarding the derivation of a whole-body fish tissue threshold value, and is organized as follows:

- The draft USEPA tissue criterion of 7.91 µg/g dw (USEPA 2004a) is described in Section 5.1.

- Proposed tissue residue guidelines (TRGs) debated in the literature are also described, with emphasis on two primary citations that capture the current range of proposed TRGs. Section 5.2.1 describes Lemly (1993b) and subsequent published proposals for a TRG of 4 µg/g dw for all fish, while Section 5.2.2 describes DeForest et al. (1999)'s proposal for TRGs of 6 and 9 µg/g dw for cold-water anadromous and warmwater fish, respectively. Other TRGs in the literature are also discussed, including Maier and Knight (1994) (Section 5.2.3), USDOI (1998) (Section 5.2.4) and Hamilton (2002) (Section 5.2.5).

- Additional investigations into tissue thresholds for cold-water salmonids are described in Section 5.3.

5.1 USEPA Draft (2004a) Tissue Criterion

5.1.1 Rationale

In 1998, the USEPA sponsored a peer consultation workshop on selenium toxicity and bioaccumulation (USEPA 1998), for the purpose of reviewing the state of the science with respect to issues that could affect how an aquatic life criterion for selenium could be derived. This workshop took into consideration such issues as the fact that selenium is an essential nutrient at trace concentrations, but that it is toxic to fish and birds at concentrations that are not much higher than those needed to maintain nutrition. Issues associated with basing the aquatic life criterion on water, tissue or sediment concentrations were considered.

USEPA (2004a) proposed a chronic criterion for selenium based on tissue concentrations rather than water concentrations. This approach was adopted due to the finding that the primary route of exposure controlling chronic toxicity to fish is via dietary ingestion. Water-only exposures are less realistic: in order to elicit a chronic effect, water concentrations of greater than 300 µg/L have been required for some studies—chronic effects have been observed under field conditions at approximately one-tenth of that water concentration.

USEPA (2004a) selected whole-body tissue concentrations over several other potential media. Expressing the chronic criterion in terms of dietary concentration was considered inappropriate since: a) dietary concentrations reflect an indirect rather than direct measure of effect; b) dietary preferences vary according to species; and c) selection of a specific organism or taxonomic group for measurement was problematic. Expressing the chronic criterion in terms of sediment concentrations was proposed (Canton and Van Derveer 1997) but rejected due to: a) patchiness of selenium distribution in sediment; and b) insufficient data to determine a causal link between sediment and chronic effects in fish. USEPA (2004a) opted to express the tissue criterion in terms of whole-body fish concentrations (dry weight) due to the availability of sufficient whole-body tissue concentrations, as well as practical considerations regarding the difficulty in collecting organ-specific tissue data (e.g., eggs are only available seasonally).

5.1.2 Derivation Method

USEPA (2004a) assembled chronic toxicity data with corresponding selenium tissue concentrations. In order to increase the number of studies considered, linear regression equations were developed to convert organ-specific selenium concentrations (i.e., muscle, ovary or liver) to a whole-body equivalent. These linear regression equations (which were statistically significant; p <0.01) were based on data from 12 different studies primarily involving rainbow trout, bluegill sunfish or largemouth bass. Wet weight selenium data were converted to dry weight equivalents where necessary assuming a moisture content of 80%.

The data set considered by USEPA (2004a) consisted of a total of 21 studies, comprising eight fish species (plus a mixture of centrarchid species) and one aquatic invertebrate species. Each study was reviewed in terms of extracting a chronic tissue concentration. Chronic values included a variety of toxicological endpoints (i.e., EC20s, MATCs)[26] as well as interpretation of the available data where study designs did not lend themselves to a dose-response relationship (e.g., field studies). Species mean chronic values (SMCVs) and genus mean chronic values (GMCVs) were calculated based on geometric means. When all species were considered, SMCVs ranged from 9.32 to 42.4 µg/g dw and GMCVs ranged from 9.5 to 42.4 µg/g dw (this range is approximate as it included a number of intermediate values that were reported as "<" or ">"). The highest SMCV/GMCV (42.4 µg/g dw) was for the single invertebrate study. Among the fish studies, the highest SMCV/GMCV was >23.28 µg/g dw for razorback sucker (*Xyrauchen texanus*). Among the studies conducted with salmonids (which included Chinook salmon, rainbow trout, cutthroat trout, and brook trout), the SMCVs ranged from 9.32 to 12.84 µg/g dw and the GMCVs ranged from 10.66 to 12.8 µg/g dw. Note that USEPA (2004a) included some but not all of the cold-water fish studies described in Section 5.3.

[26] EC20 = the concentration corresponding to a 20% reduction relative to the negative control; MATC = the geometric mean of the NOEC and LOEC values (also referred to as the "chronic value").

The final chronic value (FCV) in USEPA (2004a) was based on the lowest available GMCV (9.50 µg/g dw for bluegills), but subsequently lowered to 7.91 µg/g dw to acknowledge the findings from Lemly (1993a) (see discussion of Winter Stress Syndrome; Section 3.4). A lower monitoring threshold of 5.85 µg/g dw based on Lemly (1993a) was also included. Fish sampling during winter is recommended if tissue concentrations measured in summer or fall approach the monitoring threshold value.

5.2 Other Tissue Residue Guidelines

There is general agreement that environmental protection for selenium-contaminated systems requires consideration of fish tissue concentrations rather than water concentrations (Hamilton 2002; Sappington 2002; USEPA 2004a) although consensus on a specific threshold limit has not yet been achieved. USEPA (2004a) proposed a draft criterion of 7.91 µg/g dw (see Section 5.1); however, other values have also been proposed as tissue residue guidelines (TRGs) in the literature. This section provides an overview of the literature with respect to the ongoing debate regarding the derivation of whole-body fish TRGs. Recommendations and data regarding organ-specific or dietary TRGs as described in the literature are not summarized since USEPA has opted to pursue a whole-body TRG only.

These other TRGs tend to be presented as "threshold" concentrations (e.g., Lemly 1993b), which may have a different connotation than the term "criterion" as defined by USEPA. A threshold represents a concentration above or below which certainty is attained (e.g., effects will not occur below a certain threshold concentration, but may or may not occur above that concentration). By definition, USEPA criteria are not intended to protect all species at all times. As a result, since thresholds are intended to provide certainty about whether effects will (or will not) occur, then they may be lower than criteria.

5.2.1 Lemly (1993b; 1996b; 2002)

Lemly (1993b, 1996b, 2002) recommends a whole-body "level of concern" of 4 µg/g dw for fish, based on a review of studies with whole-body tissue data. A summary of the interpretation of each study as presented by Lemly (1993b) is provided in Table 1. Although a detailed rationale for the 4 µg/g dw whole-body TRG was not specified, Lemly (1993b) commented that the available data indicated that "tissue damage in major organs, reproductive impairment and mortality begin to occur when levels reach 4 to 16 µg/g dw.

The contents of Lemly (1993b) were subsequently republished as a book chapter (Lemly 1996b). The data summary and supporting text were modified for the majority of studies cited; however, the recommended level of concern of 4 µg/g dw remained. The whole-

body TRG of 4 µg/g dw as presented in Lemly (1996b) was republished in a second book chapter (Lemly 2002) with the addition of one new study (Saiki et al. 1992). Lemly (2002) contains the tabular summary of the re-interpreted data as presented in Lemly (1996b), but the majority of the supporting text is from Lemly (1993b)—the tables and text of Lemly (2002) with respect to whole-body fish tissue data are not consistent.

5.2.2 DeForest et al. (1999) with Hamilton (2003) Critical Review

DeForest et al. (1999)

DeForest et al. (1999) proposed whole-body TRGs of 6 and 9 µg/g dw for cold-water anadromous and warmwater fish, respectively. Results from DeForest et al. (1999) were also presented in Brix et al. (2000). Point-estimates (e.g., EC_{10} values) of the underlying dose-response were determined where possible; otherwise, the geometric mean of the NOEC and LOEC values were used as the effect level. Data from multiple studies using the same species were pooled where possible (see Figure 8 in DeForest et al. 1999).

Studies reviewed by DeForest et al. (1999) differ somewhat from those reviewed by Lemly (1993b, 1996a, 2002). DeForest et al. (1999) discussed the results from studies using a water-only exposure but did not include them in calculations. DeForest et al. (1999) hypothesized that exposure to inorganic selenium in water will result in observed adverse effects at lower tissue concentrations than exposure to organic selenium in diets since inorganic selenium is rapidly depurated from fish. DeForest et al. (1999) cited Cleveland et al. (1993) and Besser et al. (1993) to support their hypothesis. As a result, several studies[27] reviewed by Lemly (1993b, 1996a, 2002) were not included in the statistical analyses (but were summarized) by DeForest et al. (1999). Additionally, four studies[28] not reviewed by Lemly (1993b, 1996a, 2002) were included by DeForest et al. (1999). DeForest et al. (1999) also differ in their interpretation of data from Lemly (1993b, 1996b, 2002) in many instances, including:

- DeForest et al. (1999) lists two studies (Hilton et al. 1980[29]; Schultz and Hermanutz 1990) where Lemly (1993b; 1996b; 2002) reports whole-body tissue data while DeForest et al. (1999) concludes that such data are not available.

- DeForest et al. (1999) excluded the chinook salmon data reported by Hamilton et al. (1986) since forage fish with elevated selenium (fed to chinook salmon) also

[27] Studies include Hodson et al. (1980); Hunn et al. (1987); Saiki et al. (1992).
[28] Bennett et al. (1986); Bertram and Brooks (1986); Dobbs et al. (1996); Lemly (1993a).
[29] DeForest et al. (1999) notes that Hilton et al. (1980) reported carcass, kidney and liver concentrations of approximately 5, 50 and 100 mg/kg dw; he comments that: a) Lemly (1993b; 1996b; 2002) adopted the value of 5 mg/kg dw as a whole-body concentration; and b) that it is uncertain how representative this value would be given the high concentrations of Se in the kidney and liver (which were excluded from the carcass measurement).

contained elevated concentrations of boron, chromium, and strontium, and also likely pesticides such as DDT. DeForest et al. (1999) excluded Hamilton et al. (1986) from consideration due to this potential confounding factor.

- DeForest et al. (1999) used the 60-day results for Hamilton et al. (1990) rather than the 90-day results since negative control survival decreased from 99% on Day 60 to 67% on Day 90. DeForest et al. (1999) noted that the acceptable level of control mortality (30%) for salmon early life-stage tests was exceeded, and recommended that: a) the test be repeated; and b) the 90-d results not be used for developing selenium thresholds.

A comparison of the effects levels for different studies as interpreted by DeForest et al. (1999) and Lemly (1993b, 1996a, 2002) is provided in Table 1.

Hamilton (2003)

Hamilton (2003) criticized DeForest et al. (1999) for focusing on the errors in Lemly (1993b) without considering the corrected values provided by Lemly (1996b), as well as failing to cite additional articles not cited by Lemly (1993b, 1996b) or published later that support the TRG of 4 µg/g dw. Hamilton (2003) disagreed with DeForest et al. (1999)'s decision to exclude water-only exposure studies since selenium residues in fish are the result of all exposures (dietary, water-borne and sedimentary); Hamilton (2003) conclude that the convergence of adverse effects from water-borne and dietary exposure suggest that adverse effects will occur when tissue concentrations reach a threshold level, irrespective of the route of exposure.

Hamilton (2003) also disagreed with DeForest et al. (1999)'s conclusion that pesticide residues in forage fish used as a dietary item were a significant confounding factor in previous work by Hamilton et al. (1986; 1990) based on the following response:

- The diet consisting of forage fish with elevated selenium (SLD) and the diet consisting of fish food spiked with selenomethionine (SEM) both resulted in reduced survival at a dietary Se concentration of 9.6 µg/g, and whole-body residues at this dietary concentration were similar (6.5 and 5.4 µg/g in fish fed the SLD and SEM diets, respectively). Hamilton disagreed with DeForest and coauthors' decision to exclude the SLD diet from consideration[30], presumably since the SLD results were similar to the SEM results.

[30] Note that DeForest et al. (1999) included the SEM results.

- Hamilton (2003) further noted that the confounding effects from pesticides or other contaminants were explored in other Kesterson publications, but no confounding effects were reported. Hamilton (2003) also noted that several other selenium investigations elsewhere expressed concerns about potential interactions from other compounds, but no interactions have been confirmed. The observation that confounding effects have not been reported in other investigations does not preclude the possibility that pesticide residues in the forage fish diet were a confounding effect in Hamilton et al. (1986, 1990).

- Hamilton (2003) noted that the water from where the SLD forage fish were collected demonstrated toxicity to fish, which was linked to high concentrations of major ions and sulphates. The meaning of this observation is not clear given that it could be interpreted as evidence that the SLD forage fish diet did contain abnormal concentrations of other compounds which could also represent a confounding effect on the results of Hamilton et al. (1986, 1990).

Hamilton (2003) did not address the DeForest et al. (1999) decision to exclude the Day 90 data from Hamilton et al. (1990) based on poor negative control survival. Hamilton (2003) concluded that the TRG proposed by DeForest et al. (1999) "does not stand on equal footing with reviews of more extensive datasets by USDOI (1998), Lemly (1996b), Maier and Knight (1994), and Hamilton (2002)".

5.2.3 Maier and Knight (1994)

Hamilton (2003) describes Maier and Knight (1994) as an independent proposal of threshold concentrations for selenium effects similar to those proposed by Lemly (1993b; 1996b). Maier and Knight (1994) proposed that tissue concentrations of less than 3 µg/g Se dw be considered safe for freshwater fish, while tissue concentrations greater than 4.5 µg/g Se dw be considered toxic. However, the whole-body fish tissue data reviewed by Maier and Knight (1994) are limited to a qualitative review of two papers. One paper reviewed was the chinook salmon study conducted by Hamilton et al. (1990)[31]. The second paper was a literature summary (Lemly and Smith 1987) of other studies. The remaining papers discussed by Maier and Knight focus on concentrations of Se in fish dietary items, or report on adverse effects to waterfowl. Maier and Knight (1994) is therefore not considered an independent analyses of the available selenium data to the extent documented by Lemly (1993b, 1996b, 2002) and DeForest (1999).

[31] See Table 1; also see criticism by DeForest et al. (1999) and response by Hamilton (2003).

5.2.4 USDOI (1998)

Hamilton (2003) lists USDOI (1998)[32] as a review of more extensive datasets that support the TRG of 4 µg/g Se dw proposed by Lemly (1993b, 1996b, 2002). USDOI (1998) provides a whole-body "estimated true threshold range (=IC_{10}) for reproductive impairment in sensitive species (perch, bluegill, salmon)" as 4-6 mg/kg dw, and also summarize a toxicity threshold of greater than 4 µg/g Se dw for warm- and cold-water species. Lemly (1996b) is cited as the support for the threshold value, and is described as an excellent review of selenium toxicity thresholds for fish. No other basis for the derivation of the proposed TRG of 4 µg/g Se dw is provided, nor are data regarding the relationship of whole-body tissue concentrations and adverse effects in fish discussed in a comprehensive fashion[33]. USDOI (1998) is therefore not considered an independent analysis of the available selenium data to the extent documented by Lemly (1993b, 1996b, 2002) and DeForest (1999).

5.2.5 Hamilton (2002)

Hamilton (2002) concluded that a TRG of 4 µg/g Se dw is a conservative value for a national tissue-based criterion, based on a "convergence of data from laboratory and field studies" which is described as the process used by Maier and Knight (1994) and Lemly (1993b, 1996b). No further rationale for the selection of 4 µg/g dw as the TRG is provided. Hamilton (2002) provided a tabular summary of whole-body fish tissue concentrations associated with adverse effects; however, a description of what constitutes an adverse effect was not provided. Hamilton (2002) summarized most papers cited by Lemly 1993b, 1996b, 2002) but not all[34], and also summarized recent work by Hamilton and coauthors on razorback suckers and bonytail (Hamilton et al. 2000a, 2000b). As shown in Table 1, several effects levels reported by Hamilton (2002) do not match those reported by Lemly (2002) or DeForest et al. (1999) for the same studies.

5.3 Consideration of Cold-Water Selenium Studies

Several studies regarding selenium threshold levels in cold-water fish (e.g., salmonids, northern pike) have been published subsequent to the TRGs proposed by Lemly (1993b, 1996b, 2002), Maier and Knight (1994), USDOI (1998), DeForest et al. (1999) and

[32] Joseph Skorupa of the US Fish and Wildlife Service is listed as the primary author for the chapter in USDOI (1998) dealing with selenium. Skorupa et al. (1996) is often cited as a separate compendium of selenium toxicity data but is actually the US Fish and Wildlife submission for the selenium chapter in USDOI (1998) (according to Skorupa 1999).

[33] USDOI (1998) summarizes some studies regarding organ-specific and dietary TRGs. Whole-body studies are limited to Coyle et al. 1993; Hamilton and Wiedmeyer 1990; Hamilton et al. 1990; Cleveland et al. 1993. A fifth paper (Hamilton 1996) is cited but no reference is provided.

[34] Studies not included in Hamilton 2002 include Hodson et al. 1980; Hermanutz et al. 1992; Schultz and Hermanutz 1990; Gillespie and Baumann 1986 and Saiki et al. 1992 (cited by Lemly 2002), as well as Bertram and Brooks (1986) and Dobbs et al 1996 (cited by DeForest et al. 1999).

Hamilton (2002)[35]. USEPA (2004a) includes Kennedy et al. (2000), Holm (2002) and Holm et al. (2003), and a 2002 version of Hardy (2005), but not subsequent studies.

The findings from studies with cold-water fish species (e.g., Vidal et al 2005; Kennedy et al. 2000; Hardy 2005; Holm et al. 2005; Muscatello et al. in review) have implications relative to the current debate regarding selection of a suitable fish tissue residue threshold, because there are clear indications that under certain conditions (particularly in cold-water environments), cold-water fish may not display the same sensitivity to selenium that has been demonstrated in warm-water centrarchid fishes (e.g., bluegill). Therefore, in cold-water environments, site-specific and species-specific conditions may warrant adoption of a higher tissue residue threshold for selenium than for warm water environments (Chapman 2007). Table 2 presents selenium effect thresholds for egg Se concentrations determined by Kennedy et al. (2000), Hardy (2005), Holm et al. (2005).

De Rosemond et al. (2005), and Muscatello et al. (in review) compare these thresholds to Lemly's (1997b) egg Se threshold of 10 µg/g dw for freshwater and anadromous fish. The thresholds derived from these four studies with cold-water fish are close to two to four times higher, depending on the moisture content used to convert between wet and dry weight, than the Lemly (1997b) threshold.

Vidal et al. (2005) determined a 90-d LOEC for growth (not reproduction) of 1.2 µg/g ww (whole-body Se concentration) for rainbow trout. DeForest et al. (2006) note that Vidal and coauthor's selection of 1.2 µg/g ww (whole-body) as the LOEC is based on the fact that it was the lowest average tissue concentration to demonstrate a statistical difference to the negative control; they comment that it would be more appropriate to select the lowest exposure concentration with a statistically significant reduction in growth. The 90-d growth LOEC was actually 4.6 µg/g dw in food, which corresponded to 0.58 µg/g ww in whole-body fish, which corresponded to 2.3 µg/g dw in whole-body fish. As a result, the conclusion that the LOEC was less than 4 µg/g dw whole-body was correct, although the selected LOEC in Vidal et al. (2005) was incorrect. Vidal et al. (2005) concluded that their results demonstrated the difficulty of using body-burden residues as toxicity thresholds for essential elements in developmental immature stages; DeForest et al. (2006) concluded that this area requires further research, and therefore, applying a threshold value from larval fish to adult fish was not warranted at this time.

Kennedy et al. (2000) found no significant adverse effects on cutthroat trout fertilization, hatching, survival or deformities, with a mean selenium egg concentration of 21.2 µg/g dw for the exposure treatments. Using the equation provided in USEPA (2004a) to convert selenium egg concentration to a whole-body concentration, would result in a

[35] Kennedy et al. (2000) was referenced in Hamilton (2002), but excluded from consideration based on Hamilton and Palace (2001). McDonald and Kennedy (2002)'s rebuttal of Hamilton and Palace (2001) was not referenced in Hamilton (2002).

whole-body threshold of >9.8 µg/g dw for cutthroat trout, which is higher than the USEPA (2004a) criterion on 7.91 µg/g dw and Lemly's (1993b) threshold of 4 µg/g dw. Kennedy et al. (2000) proposed that these fish (inhabiting cold, lentic waters in a coal mining area in southeast British Columbia) may have evolved increased tolerance to higher selenium concentrations.

Hardy (2005) reared cutthroat trout (obtained from a hatchery as eyed eggs) on a selenium-amended diet (treatments ranged from 2 to 10 µg/g dw) for 124 weeks and was able to spawn adult fish and assess egg hatchability. When fish were two to three years old, mean whole-body tissue concentrations were up to 5.61 to 6.4 µg/g dw and mean egg concentrations were up to 16.04 to 18.0 µg/g dw. Total deformities ranged from 10.8 to 20.2% in the lower selenium-amended diets (2, 4 and 6 µg/g dw) and from 6.8 to 7.0% in the two highest diets (8 and 10 µg/g dw); total deformities in the negative control were 5.6%. When Hardy (2005) considered only the cranio-facial deformities, their incidence was 6 and 9% in the 2 and 4 µg/g dw diets, as compared to 1 to 4% in the 6 to 10 µg/g dw diets and 1% in the negative control. According to the author, the increased incidence of deformities was usually due to a high percentage of deformed offspring associated with eggs from one or two fish with the rest having lower percentage deformities (Hardy did not present results for individual fish so this could not be evaluated independently). Although there was a slight effect at lower dietary selenium concentrations, there were no adverse effects on offspring of fish exposed to the higher selenium concentrations and no linear dose-response relationship between dietary selenium concentration and the incidence of larval deformities.

Holm et al. (2005) used a study-specific regression equation to convert their rainbow trout egg tissue threshold of 10 µg/g ww to a selenium muscle concentration (1.8 µg/g ww), which they then compared to whole-body values (10 to 12.5 µg/g ww) previously reported by Lemly (1993c) for warm-water centrarchids from Belews Lake, NC to conclude that their data did not support establishment of a higher whole-body TRG for cold-water salmonids. This comparison involved muscle to whole-body selenium concentrations (which are not directly comparable). If Holm et al. (2005) had converted their 10 µg/g ww egg threshold to a dry weight value, they would have obtained values of either 25.6 µg/g dw (assuming 61% moisture used by the authors) or 40 µg/g dw (assuming 75% moisture as used by Lemly 1993b, 1996b, 2002), their egg threshold would have been 2.6 to 4.0 times higher than the 10 µg/g dw threshold recommended by Lemly (1993b, 1996b, 2002). These dry weight egg thresholds are more consistent with the findings of Kennedy et al. (2000) for cutthroat trout (>21.2 µg/g dw for eggs; see Chapman and McPherson 2004) than those of Lemly for centrarchids. The implications of the finding that brook trout accumulated lower Se concentrations in eggs than rainbow trout with respect to establishment of a TRG for cold-water salmonids was not discussed by Holm et al. (2005).

Muscatello et al. (in review) determined the threshold egg and female muscle tissue Se concentrations for northern pike associated with a 20% increase in total deformities relative to the reference site, and calculated threshold values of 32.25 µg/g dw and 14.90 µg/g dw for eggs and muscle tissue, respectively. Muscatello et al. (in review) converted these values into a whole-body fish tissue threshold ranging from 12.77 to 14.96 µg/g dw using the tissue to whole body regression equations provided in USEPA (2004a). Muscatello et al. (in review) concluded that their experimental design demonstrated that adverse effects on larval pike were associated with maternal transfer of Se, rather than water-borne selenium, and that their results suggest that northern pike may be up to three times less sensitive than warm-water fish such as centrarchids and cyprinids.

De Rosemond et al. (2005) concluded that the observed frequency of larval deformity in their study with white suckers (*C. commersoni*) would translate into slight or moderate effects on the white sucker population, based on Lemly (1997c) who noted that less than 5% population mortality as a result of teratogenesis[36] was considered negligible. De Rosemond et al. (2005) noted that the findings from the white sucker study (i.e., 12.8% mean total developmental deformities associated with a mean egg concentration of 25.9 µg/g dw) tended to support those found by Kennedy et al. (2000) for cutthroat trout, and hypothesized that the white sucker population sampled may have developed some level of tolerance since it has been exposed to selenium for more than 20 years.

Kennedy et al. (2000) and de Rosemond et al. (2005) hypothesized that fish populations can develop tolerance to elevated Se concentrations although Hamilton and Palace (2001) disagreed. Saiki et al. (2004) found that western mosquitofish (*Gambusia affinis*) inhabiting selenium-enriched areas had fry survival rates greater than 96%; also, none of the fry demonstrated deformities considered characteristic of selenium effects, despite fry whole-body tissue concentrations as high as 29.2 ug/g dw. Saiki et al. (2004) commented that mosquitofish (*Gambusia* spp.) have demonstrated tolerance to elevated Se at other sites, including Belews Lake and the San Luis Drain (SLD; see Hamilton et al. 1990; Hamilton 2003).

[36] Teratogenesis refers to congenital malformations that occur as a result of exposure of eggs to contaminants during development. In the case of fish and selenium exposure, this can include malformations of the skeleton, fins, eyes and head.

6.0 REVIEW OF SITE-SPECIFIC SELENIUM DATA

In southeast Idaho, there are numerous active and inactive phosphate mines in the Blackfoot River and Bear River Watersheds, as well as the Salt River Watershed (where the Smoky Canyon Mine is located).

The Middle Permian Phosphoria Formation extends over a large portion of the middle Rocky Mountain Basin, and is one of the world's largest phosphate rock resources. Phosphate-bearing sedimentary rock was deposited approximately 265 million years ago, and underwent extensive deformation until approximately 10 to 20 million years ago. Concentrations of phosphorite (which is of commercial interest for mining) are highest in the Meade Peak Member of this formation, which extends over southern Idaho and western Wyoming (BLM 2005; USGS 2002). Areas of the Meade Peak Member also contain selenium and other metals, which can be mobilized into soluble forms when exposed to water and oxygen. Selenium concentrations measured within the formation vary considerably between different areas, as well as within the same areas. Surface outcrops of the Phosphoria Formation occur within the study area (Panels F and G) for the EIS, and outcrops also occur in the upper portions of the Crow Creek and Deer Creek watersheds where there has been no mining activity to date (Hamilton and Buhl 2003b). These outcroppings represent a potential natural source of selenium to Crow and Deer Creeks that has been present on a far longer time-scale than any sources associated with mining activity.

A number of studies have been undertaken in southeast Idaho since 1997, in part to investigate concerns regarding elevated selenium concentrations in water and biota, but also to provide information on overall water quality and other contaminants of potential concern (COPCs). Some of the studies have been site-specific and focussed on the Smoky Canyon Mine, while others were conducted to assess area-wide conditions and therefore encompassed portions of some or all three watersheds. These studies are described briefly in Section 6.1 to provide a summary of the available area-wide information regarding selenium concentrations in the environment.

Section 6.2 provides information regarding the range of selenium concentrations measured in different aquatic matrices at stations within the Salt River Watershed, as well as comparisons to the water and tissue residue guidelines discussed in earlier sections. The focus of this document was on the potential effects of selenium on fish, so these summaries were limited to selenium data for water, fish, sediment, benthic invertebrates, and aquatic vegetation. Data on birds, riparian vegetation and terrestrial wildlife were specifically excluded.

6.1 Summary of Area-Wide and Site-Specific Studies

In 1997, the Idaho Mining Association (IMA) Selenium Committee initiated the Southeast Idaho Phosphate Resource Area Selenium Project. This initiative was triggered in part by the occurrence of selenosis (selenium poisoning) in horses from areas located below phosphate mining operations; as such it included both terrestrial and aquatic components in its activities. As part of this project, various monitoring and assessment activities were undertaken annually between 1997 and 2000. This work was undertaken on an area-wide basis encompassing sampling locations in both the Blackfoot River and Salt River Watersheds. Brief descriptions of the types of activities undertaken each year, and the amount of data relevant to the Salt Watershed and more specifically the Smoky Canyon Mine, are provided below:

- **Fall 1997 Interim Surface Water Survey (Montgomery Watson 1998):** As the first task of this area-wide initiative, an interim surface water survey was conducted in September 1997 to assess water quality at locations where selenium was potentially released as a result of phosphate mining operations, and to determine whether livestock health could be impacted by those selenium releases. Most of the surface water sampling was conducted at locations in the Blackfoot River Watershed. Within the Salt River Watershed, water samples were collected from Pole Creek, North Fork Sage Creek, and Tailings Pond 1 (all locations potentially affected by mining operations). Field measures of water quality were made, and samples were analysed for selenium and total dissolved solids (TDS); qualitative observations of water usage by wildlife, livestock, birds or fish were also made.

- **1998 Regional Investigation (Montgomery Watson 1999):** In 1998, sampling of surface water, sediment, groundwater, soil, vegetation, and fish tissue was performed for analysis of selenium and five metals (cadmium, manganese, nickel, vanadium and zinc). These data were compared to applicable regulatory criteria, and to background conditions, and were used for the development of human health and ecological risk assessments (HHERA). Sample stations were located in the Bear, Blackfoot, Portneuf/Ross Fork, and Salt River Watersheds. Surface water sampling was conducted in May and September 1998, sediments (15-cm cores) and fish tissue (skin-on muscle fillets) were sampled in September 1998, and soil and terrestrial vegetation was sampled in July 1998. The target fish species was cutthroat trout, but brown trout and brook trout were also sampled; fish were submitted as composites, and length/weight data were not collected. Within the Salt River Watershed, surface water and sediments were sampled from Sage Creek, South Fork Sage Creek, North Fork Sage Creek, Roberts Creek, Smoky Creek and Deer Creek (encompassing stations located upstream and downstream of the Smoky Canyon Mine); fish were only sampled from South Fork Sage Creek.

- **1999 Interim Investigation (Montgomery Watson 2000):** Activities undertaken in spring 1999 included continued surface water monitoring as well as initiation of avian and cutthroat trout studies, and investigations with cattle and elk. Surface water monitoring was conducted in May 1999, but only at 12 stations in the Blackfoot River Watershed. The cutthroat trout studies included investigation of egg viability and dietary exposure to selenium (the findings of this work were reported in Hardy 2005), as well as genetic analyses of the two cutthroat trout populations used for those studies.

- **1999 - 2000 Regional Investigation for Surface Water, Sediment and Aquatic Biota Sampling Activities, September 1999 (Montgomery Watson 2001a):** This report describes the results of sampling conducted in September and October 1999 of surface water, sediments, plankton, aquatic vegetation, fish tissue, and riparian vegetation. Within the Salt River Watershed, only surface water and sediment samples were collected from Sage Creek, North Fork Sage Creek, South Fork Sage Creek, and Tygee Creek (these creeks are influenced by mining activities, except for the upper reaches of North Fork and South Fork Sage Creeks.

- **1999 - 2000 Regional Investigation for Surface Water, Sediment and Aquatic Biota Sampling Activities, May – June 2000 (Montgomery Watson 2001b):** This report describes the results of sampling conducted in May and June 2000 of surface water, benthic invertebrates, aquatic vegetation, and fish tissue. Within the Salt River Watershed, only surface water samples were collected from Sage Creek, South Fork Sage Creek, and Tygee Creek.

Hamilton et al. (2002) and Hamilton and Buhl (2003a, 2003b) reported the results of area-wide field sampling programs that were conducted in June 2000, September 2000 and May 2001 at stations within the Blackfoot, Bear and Salt River Watersheds. Concentrations of selenium and other inorganic analytes were measured in samples of surface water, surface sediment, aquatic plants, aquatic invertebrates, and fish at each station. Hamilton and Buhl (2003b) included three sampling stations in the Salt River Watershed (Crow Creek, Deer Creek and Smoky Creek) in May 2001, but no stations from this watershed were included in the 2000 sampling programs. Crow Creek and Deer Creek are located upstream of existing Smoky Canyon Mine operations (and therefore represent background conditions) and Smoky Creek is located downstream of mining operations.

TetraTech EM (TtEMI 2002a) performed an area-wide human health and ecological risk assessment (HHERA) of the Southeast Idaho Phosphate Resource Area; the purpose of this HHERA was to evaluate baseline human health risks and population-level risks to ecological receptors. The HHERA incorporated data from previous studies such as those conducted since 1997 (see above), but also provided data on selenium concentrations in

Sage Creek and Crow Creek. TtEMI (2002b) reported the results of three sampling events conducted in 2001 to collect baseline surface water data in the Blackfoot, Bear and Salt River Watersheds; this report included two results for selenium in surface water samples from different locations in Sage Creek, which were already reported in TtEMI (2002a). TtEMI (2002c, 2004) provided surface water monitoring data collected in 2002 and 2001 to supplement TtEMI (2002b).

Mariah Associates Inc. (Mariah 1980) conducted selenium monitoring in surface waters as part of a baseline aquatic ecology study of the Smoky Canyon phosphate lease in June and October 1979. These data were obtained from the NewFields (2005) Access database; the original report was not provided. Selenium concentrations were undetected (<10 µg/L in June samples, and <1 µg/L in October samples) in all samples collected from Sage Creek (North and South Forks), Pole Creek, Smoky Creek, and Tygee Creek.

Maxim Technologies, Inc. (Maxim 2002) conducted selenium monitoring in surface waters associated with the Smoky Canyon Mine in March, June and September 2000. These data were obtained from the NewFields (2005) Access database; the original report was not provided. Selenium concentrations were measured in samples collected from Sage Creek (North and South Forks), Pole Creek, Smoky Creek, and Tygee Creek. Maxim (2004a, 2004b, 2006) provide the results of baseline sampling that was conducted in Panels F & G of the phosphate lease area (i.e., the subject of the environmental impact statement). These results document the results of sampling for fish and benthic inverebrates conducted in August 2003 and January 2006.

MFG (2003a, 2003b) describe the results of a baseline ecological risk assessment that was performed on Area B of the Smoky Canyon Mine, which consists of the mine's tailings impoundments, and provide data on groundwater and other environmental media that were sampled in order to characterize conditions in the tailings ponds.

NewFields (2005) documented the results of a site investigation conducted in 2003 and 2004 to evaluate environmental conditions associated with Area A of the Smoky Creek Mine (Area A encompasses the mine and related facilities located on National Forest system land under lease and special-use permit to Simplot; as noted above, Area B is the tailings ponds). Fish, benthic invertebrates and aquatic vegetation were sampled as part of this investigation. NewFields also compiled a large Access™ database containing much of the historical monitoring data gathered for the Smoky Canyon Mine and the surrounding area.

Weber (2005a, 2005b) reported the results of selenium analyses performed on water and biota samples collected in 2005 on behalf of the Greater Yellowstone Coalition (GYC) from stations in Crow Creek, Deer Creek, Sage Creek and Smoky Creek.

6.2 Data Quality Assessment

As part of the data compilation process undertaken in preparing this appendix, an assessment of data quality was performed for each of the data sources described in Section 6.1. In most cases the original reports included sections that described the relevant quality assurance/quality control (QA/QC) procedures that were followed with respect to chemical analyses, including analysis of applicable QC samples and evaluation of data for completeness, precision, accuracy and representativeness. One exception was Maxim (2004a, 2006), which did not include discussions of QC data in their reports. We were able to obtain the QC report from the analytical laboratory for the data reported in Maxim (2006), but were not able to obtain the QC report for the data reported in Maxim (2004a).

Selenium analyses appear to have been performed using either hydride generation atomic absorption spectrophotometry, inductively coupled plasma mass spectrometry or graphite furnace atomic absorption spectrophotometry, with either hotplate or microwave digestion as part of the sample preparation process. It appears that appropriate QC samples were included with all analyses, including method blanks, laboratory control samples, laboratory duplicates, matrix spike/matrix spike duplicates, and standard reference materials. Based on evaluation of the available QC results, it appears that the selenium data reported for the various environmental matrices were generally of acceptable quality and acceptable for their intended use. Maxim (2006), NewFields (2005) and TtEMI (2002a) reported that matrix spike recoveries for selenium in some sample batches were below control limits and in some cases data qualifiers were applied. MFG (2003a) reported repeated difficulties with their analytical laboratory not meeting matrix spike recovery control limits due to matrix interferences; analyses were repeated using dilutions to reduce interference, resulting in improved (but not fully compliant) recoveries but also requiring the use of higher detection limits (data from the re-analyses were still considered usable by MFG). Only Montgomery Watson (1999) reported that their analytical data were corrected for both laboratory blanks and recovery of spikes and standards, resulting in "laboratory corrected concentrations" being reported.

The only consistent data quality issue for selenium analyses appears to have been with regard to matrix spike recovery, suggesting that reported selenium concentrations could be biased low in some cases. However, results for laboratory blanks, duplicates, and standard reference materials were all within acceptable limits, which suggests that this is not likely to be a significant data quality concern.

6.3 Selenium Concentrations In the Salt River Watershed

Data on selenium concentrations in environmental matrices in the Salt River Watershed were compiled from the documents listed in the previous section, as well as from data

compilations provided by JBR. In addition to the NewFields (2005) database, data compiled by the Greater Yellowstone Coalition (GYC) and submitted as part of their comments on the DEIS were also considered. Because selenium data have been collected by numerous sources, many station locations have been used and different station names have been used to describe the same sampling location. To simplify presentation of the compiled data, where data were collected from the same station or from stations in close proximity we combined those data to represent one location and assigned a new station identification code to that data set.

6.3.1 Selenium Concentrations in Water

Monitoring of selenium concentrations in water at selected stations within the Salt River Watershed has been ongoing since 1979, although not all stations have been sampled every year. Sampling frequency varied between stations, although a number of stations were monitored in spring (May) and fall (September – October) each year. Selenium water data collected between 1979 and 1983 were excluded from this data set because all values were undetected but the detection limits (DLs) used at that time ranged from 10 to 100 µg/L (as compared to 0.2 to 2 µg/L in recent years). Including these older undetected values in calculations of mean concentrations would have artificially skewed results upward. When calculating mean concentrations for each station, values reported as undetected were assigned a value equal to one-half the DL for that sample.

Selenium water concentrations presented in this report are limited to surface water data collected from streams within the study area. Groundwater data are not included in this review, nor are data on selenium concentrations associated with mine operations (e.g., waste dumps, monitoring wells). Data from selected springs were included if selenium data for fish or other matrices had also been collected. The objective of this data assessment was not to document all potential sources of selenium to the study area, which has been done as part of other site-specific studies (e.g., NewFields 2005), but rather to present information on the distribution of selenium concentrations in waters inhabited by fish within the study area.

Selenium water concentrations are summarized in Table 3; station locations are shown in Figure 3. To facilitate data presentation, the selenium water data have been grouped together by regions within the watershed (e.g., all stations on Crow Creek above Sage Creek grouped together, and all stations on Deer Creek grouped together), and then summary statistics (sampling year range, number of measurements, mean and range of selenium concentrations) are reported for each sampling station.

Selenium was frequently undetected (<0.2 to <1 µg/L) in water from stations located upstream of mining influences (Crow Creek above Sage Creek, Deer Creek, upper Pole Creek, Upper Sage Creek, upper Smoky Creek, the south fork of Sage Creek, and upper Tygee Creek).

- In Crow Creek (above Sage Creek), selenium was monitored at seven stations in the mainstem creek and four stations in adjacent canyons. Mean selenium concentrations ranged from <1 to 3.93 µg/L, and maximum concentrations ranged from <1 to 7.6 µg/L. The highest concentrations were reported in Lane Canyon (Station LC-500; 3 µg/L) and Stewart Canyon (Station STC-500; 7.6 µg/L).

- In Deer Creek, mean selenium concentrations were 1.32 µg/L among mainstem stations, 0.68 µg/L in the North Fork, and 1.03 µg/L in the South Fork. The highest selenium concentration was 2.85 µg/L, at one of the mainstem stations.

- At two stations in Pole Creek, above the waste rock dump, mean selenium concentrations were 0.32 and 0.59 µg/L, and the maximum concentration was 4 µg/L.

- In upper Sage Creek, selenium was undetected (<1 µg/L) at three stations, and ranged from <0.2 to 11 µg/L at Station USC-4 (the most upstream station).

- In Sage Creek South Fork, selenium concentrations were below or near detection limits (<0.02 to <1.0 µg/L) at five of eight sampling stations; mean concentrations at the other three stations were <1.0 to 1.8 µg/L, and the maximum concentration was 4.3 µg/L.

- In upper Smoky Creek, mean selenium concentrations ranged from 0.49 to <2.0 µg/L at three stations, and the maximum concentration was 3 µg/L.

- In upper Tygee Creek (and the East Fork), mean selenium concentrations ranged from 0.65 to 1.02 µg/L at three stations, and the maximum concentration was 5 µg/L.

Stations upstream of mining influences with selenium concentrations above the IDEQ standard of 5 µg/L were in upper Sage Creek (Station USC-4: 11 µg/L) and Stewart Canyon off upper Crow Creek (Station STC-500: 7.6 µg/L).

At stations that were considered to be mine-impacted, mean selenium concentrations for individual stations ranged from <0.3 to 2,350 µg/L. Although some of these sampling stations were located downstream of inputs from mining operations, selenium concentrations were low.

- At four stations in Sage Creek North Fork, mean selenium concentrations ranged from 0.44 to 4.7 µg/L, and maximum concentrations for each station ranged from 1.3 to 41 µg/L. At Station SCNF-1, where the 41 µg/L value was reported, the majority of measurements were near the detection limit (however this station is located below the confluence with Pole Creek, so at least the occasional elevated selenium concentration would not be unexpected).

- Pole Creek stations located at or downstream of the waste rock dump had the highest selenium concentrations; at six stations, mean selenium concentrations ranged from 210 to 2,350 µg/L and maximum concentrations ranged from 248 to 2,400 µg/L.

- Four stations associated with Hoopes Spring (which joins Lower Sage Creek upstream of the South Fork) had mean selenium concentrations ranging from 6.7 to 10.3 µg/L, and maximum concentrations between 10 and 17 µg/L.

- At 12 stations in Lower Sage Creek (below the confluence with the North Fork), mean selenium concentrations ranged from 0.78 to 8.2 µg/L, and maximum concentrations for each station ranged from 1.2 to 9.1 µg/L.

- Three stations in Crow Creek, below Sage Creek, had mean selenium concentrations between 0.73 and 2.81 µg/L. At Station CC-800 (the most downstream station on Crow Creek, several miles below the confluence with Sage Creek), selenium concentrations ranged from <0.2 to 1.7 µg/L.

- Lower Smoky Creek and lower Tygee Creek (which receives inputs from Smoky Creek) had mean selenium concentrations <1 µg/L at a total of 12 stations; maximum concentrations were 4 µg/L for lower Tygee Creek and 10.98 µg/L for lower Smoky Creek.

Stations affected by mining activity with selenium concentrations above the IDEQ standard of 5 µg/L were in Pole Creek (seven stations ranging from 248 to 2,400 µg/L), Hoopes Spring (four stations ranging from 10 to 17 µg/L), Sage Creek North Fork (Station SCNF-1: 41 µg/L), Lower Sage Creek (seven stations ranging from 5.4 to 9.1 µg/L), lower Roberts Creek (Station RCL-1: 6 µg/L), and lower Smoky Creek (Station SMC-6: 10.98 µg/L).

6.3.2 Selenium Concentrations in Fish

The majority of available fish tissue data from the Salt River Watershed was reported in terms of whole-body concentrations; there were only three samples reported as either muscle or as unspecified tissue. No data on selenium concentrations in fish eggs or other organs (e.g., liver) were reported. Most of the data were reported on a dry weight (dw)

basis; where data were reported on a wet weight (ww) basis in the original source, we converted those results to dry weight either by using sample-specific moisture content values reported with the tissue chemistry data or using a value of 75% for moisture content (as recommended by Lemly 1993b).

Data on tissue selenium concentrations in fish from the Salt River Watershed are summarized in Table 4; station locations are shown in Figure 4. No data were available regarding fish ages or sex, but where length and/or weight data were reported, these have also been included in Table 4. Tissue data were available for cutthroat trout, brown trout, dace, suckers, sculpins, and shiners; the largest data set was for cutthroat trout. Tissue selenium concentrations have only been monitored since 2001 (except for one fish sampled in September 1998). Sampling programs were generally conducted between May and August, except for the Maxim (2006) study which involved sampling fish in January 2006. Sample sizes ranged from 1 to 12 fish of a particular species per sampling event; where sample sizes are small, it is not possible to determine whether measured selenium tissue concentrations are representative of conditions at that sampling location and results should be evaluated with caution. The majority of fish sampled were cutthroat trout, brown trout and sculpins. Tissue concentrations were variable between sampling stations, as well as within the same station, which was likely related to fish movement patterns. Many of the fish sampled, including cutthroat and brown trout, had whole-body tissue selenium concentrations that were higher than Lemly's (1993b) recommended threshold of 4 µg/g dw and the draft USEPA (2004a) TRG of 7.91 µg/g dw, even at stations that represented background conditions.

Variability in fish tissue selenium concentrations, particular within a station, may be influenced by a number of factors, including timing of sampling, individual fish characteristics (size, age, sex), and life history. Fish tissue sampling should ideally be conducted at similar times of the year to account for changes in flow and climate conditions, fish residence patterns, and dietary selenium uptake (i.e., fish tissue selenium concentrations may be lower when there is little prey available for consumption such as soon after winter). In terms of YCT life history, three possible strategies (resident, adfluvial and fluvial) can affect fish distribution patterns and therefore their potential exposure to selenium in a particular waterbody. According to USFS (2003), "resident" populations remain in small tributaries year-round and do not migrate to spawn or overwinter; "fluvial" populations spend most of their lives in large streams and rivers and return to small streams for spawning and rearing of young; "adfluvial" populations behave similarly to fluvial ones, except that they spend most of their lives in lentic waterbodies such as lakes. Both migratory and non-migratory populations of YCT are present in the Upper Snake River Basin (Thurow et al. 1988).

Cutthroat trout were sampled at 16 stations classified as being representative of background conditions (i.e., not influenced by mining activity) for the study area; this

included three stations in Crow Creek above Sage Creek, eight stations in Deer Creek, four stations in upper Sage Creek, and one station in upper Smoky Creek. Mean total selenium concentrations at these stations ranged from 0.44 to 18 µg/g dw. Maxim (2004a) reported whole-body selenium concentrations for three Deer Creek stations that were conspicuously low (0.44 to 0.76 µg/g dw) relative to other stations. According to Maxim (2004a), all fish were sampled for whole-body analysis and not for muscle only (which would have most likely yielded lower tissue concentrations). Supporting quality control (QC) results from the analytical laboratory were not available for verification of data quality. Excluding those three stations, mean tissue selenium concentrations at background stations ranged from 2.3 to 18 µg/g dw.

Cutthroat trout were also sampled at two stations in lower Sage Creek, which is influenced by mining-related activities. Mean tissue selenium concentrations were within the same range reported for background stations (3.6 and 11.8 µg/g dw), suggesting that cutthroat trout are migrating throughout the system.

Brown trout were sampled at four background stations (in upper Crow Creek and Sage Creek South Fork) and five mining-influenced stations (in lower Crow Creek, lower Smoky Creek, and the Sage Creek drainage). Mean tissue concentrations for the background stations (5.2 to 9.7 µg/g dw) were lower than mean tissue concentrations measured at mining-influenced stations (5.29 to 21.8 µg/g dw).

Paiute sculpin were sampled at two upper Crow Creek stations and two Deer Creek stations in January 2006 (Maxim 2006). Mean tissue concentrations at these four stations ranged from 6.3 to 8.9 µg/g dw. Other sculpins (not identified to species) were also sampled at background stations in upper Crow Creek and Deer Creek and at mining-influenced stations in Sage Creek, Hoopes Spring, Smoky Creek and Tygee Creek. Mean tissue selenium concentrations ranged from 4.3 to 22 µg/g dw at the background stations, and from 5.95 to 28.8 µg/g dw at the mining-influenced stations, but sample sizes at all stations were small (one to three fish).

Several fish were also sampled from within the Smoky Canyon Mine's tailing ponds, including speckled dace, leatherside chub, and mountain suckers. Mean tissue selenium concentrations for these fish ranged from 22 to 39.2 µg/g dw.

Figure 5 illustrates the distribution of selenium tissue concentrations for brown trout, cutthroat trout, sculpins and dace. Individual data for each species are separated based on whether they are from background (BK) or mining-impacted (MI) stations, and then each result was plotted, along with the various TRGs discussed in the previous chapter. All four fish species had multiple results that exceeded one or more of these TRGs; the range of concentrations was higher for brown trout, sculpin and dace than it was for cutthroat trout. However, cutthroat trout from background stations had higher whole-body selenium concentrations than fish from mining-impacted stations.

Based only on comparison to the TRGs shown in Figure 5, one might conclude that all fish populations in this stream system were at significant risk as a result of selenium exposure. However, as discussed in the previous chapter (and see Table 2 and Chapman 2007) there is evidence that cold-water fish such as cutthroat trout may be able to tolerate exposure to relatively high (i.e., > TRGs) selenium concentrations without adverse effect. Hardy (2005) showed that when cutthroat trout eggs from a hatchery were exposed to dietary selenium at a concentration of 12 µg/g dw (the highest dietary concentration tested) for two years, whole-body tissue concentrations were approximately 6 µg/g dw but this body burden did not result in significant adverse effects to offspring (i.e., total and cranio-facial larval deformities were 6.8 and 1.95%, respectively). Higher deformity rates were observed for lower dietary treatments (maximum total and cranio-facial deformities of 20.2 and 9.2%, respectively), but Hardy attributed this to anomalous high deformities associated with offspring from a limited number of fish. Allowing for some likely reduction to the total deformity results to match the Lemly (1997) definition of teratogenic defects[37], negligible impacts occurred at Hardy's (2005) highest dietary concentrations. Also, the tissue selenium data for benthic invertebrate samples (see Section 6.2.3) reflect a range of concentrations that on average are lower[38] than the 12 µg/g dw dietary exposure concentration that was used by Hardy (2005). With respect to cutthroat trout in the Blackfoot River, Hardy (2005) also noted that juvenile fish that rear in upstream nursery areas where selenium concentrations tend to be elevated move downstream to other adult rearing areas as they grow older, and those downstream areas tend to have lower selenium concentrations. Therefore, fish would not be exposed to the same elevated selenium concentrations throughout their life, and would be able to depurate excess selenium from their tissues over time. Limited deformities to larval fish may occur based on consideration of dietary selenium concentrations in the study area and the range of effects observed by Hardy (2005) relative to the decision criteria provided by Lemly (1997). However, the continued presence of healthy YCT populations in Crow Creek and Deer Creek suggest that a population-level impact associated with selenium is unlikely.

[37] Lemly (1997) defines true teratogenic deformities as those affecting the skeleton, fins, head and mouth., and describes edema, exopthalmus (bulging eyes) and cataracts as being symptoms of selenium poisoning rather than teratogenic defects. Although Hardy (2005) did not report the different types of deformities (other than the cranio-facial category), it is likely that the percentage deformities that would fit the Lemly (1997) classification is lower than the total deformity results that were reported. The Lemly (1997) teratogenic deformity index is based on terata-mortality relationships; <6% terata indicates negligible impact, 6 to 25% terata indicates slight to moderate impact, and >25% terata indicates major impact, but Lemly acknowledged that these relationships were derived for centrarchid and cyprinid fish and may not be directly applicable to cold-water species such as salmonids (i.e., trout).

[38] Hamilton and Buhl (2003a) reported a maximum selenium concentration in benthic invertebrates of 75.2 µg/g dw. However, this value was measured in East Mill Creek (Blackfoot River watershed, downstream of the inactive North Maybe Mine) where selenium concentrations in water, sediment, aquatic plants and benthic invertebrates are considerably higher than corresponding concentrations reported by Hamilton and Buhl (2003b) for Deer Creek and Crow Creek.

A number of population surveys have been undertaken in southeastern Idaho, including background locations such as Crow Creek and Deer Creek, to assess trout populations including YCT. Populations of YCT have decreased substantially over the last century (due to factors such as hybridization, displacement by non-native trout, excessive sportfishing, and habitat alteration), and are now estimated to occupy approximately 43% of their historical range (Meyer et al. 2003; USFS 2003). However, there is also evidence that current YCT populations have remained stable in many areas over the past 20 years, and that populations within the study area are healthy. Data specific to Crow Creek, Deer Creek and portions of Sage Creek as detailed below indicate that, despite the presence of elevated selenium concentrations from natural sources (un-mined phosphoria outcroppings), YCT populations in these areas are healthy[39].

- Meyer et al. (2003) surveyed 77 stream segments in southeastern Idaho between 1980 and 1989, and again in 1999 and 2000, to determine whether there had been changes in abundance and size structure of YCT populations. Consistent sampling locations were used, and sampling was conducted within six weeks of the original sampling date (65% within two weeks), to reduce potential spatial and seasonal variability. There was no overall change in abundance of YCT in the >10-cm size range (average 41 fish/100 m stream) and no change in the proportion of YCT making up the total trout population (82% in the 1980s and 78% in 1999 – 2000). Non-native fish (rainbow trout, and rainbow trout x cutthroat trout hybrids) were present at a greater number of locations, but did not increase in terms of proportion of the total trout catch. A total of four stations in Crow Creek, Deer Creek and Sage Creek were included in this survey; all showed increases in YCT abundance in 1999-2000 (10 to 117 fish/100 m stream). Size distributions patterns were similar in Crow Creek and Deer Creek for the two time periods, whereas Sage Creek showed an increase in the proportion of larger fish in 1999-2000.

- Meyer and Lamansky (2004) conducted electrofishing surveys at 961 sites in the Upper Snake River Basin (Idaho and neighboring states) between 1999 and 2003 (sampling after spring runoff and before winter). Data specific to Crow, Sage or Deer Creeks were not reported, although the Palisades/Salt drainage was sampled. YCT were present at 457 of 961 sites, and had the widest distribution of all trout species. The average total trout abundance for >10-cm fish was 19.6 fish/100 m stream; 47% of all trout were YCT. The overall conclusion was that non-native trout species were a threat to YCT, but that YCT populations were widely distributioned in the Upper Snake River Basin and appeared to be healthy.

[39] A July 24, 2006 letter from IDFG to USFS regarding the Smoky Canyon Mine Engineering Evaluation/Cost Analysis stated that IDFG was unaware of any published information that supports the proposition that resident fish populations of any species in the Crow Creek drainage have elevated tolerances to selenium, and that determining such a relationship could be difficult due to a lack of reference streams (Gamblin 2006 pers comm). This Appendix presents data from a number of sources that support the idea that cold-water fish species such as YCT are able to tolerate elevate selenium concentrations under certain conditions, as reflected in the presence of healthy fish populations in Deer and Crow Creek where selenium concentrations are elevated due to natural sources rather than mining activity.

- USFS (2003) reported that surveys of YCT populations have been conducted in the Caribou National Forest since 1996, and the majority of hydrologic unit subbasins have strong YCT populations. The Palisades/Salt metapopulation of YCT is considered to be robust, and has some of the highest quality habitat. The presence of rainbow trout was reported to be low in Crow Creek and elsewhere, but strong brook trout populations are present in some tributaries of the Upper Salt River (e.g., Smoky Canyon Creek) and are a threat to YCT in terms of competition for habitat. In addition to non-native trout species, roads and trails were also considered a moderate threat to YCT populations; although Crow Creek has high road density (>1.8 miles of road/square mile), its YCT population is considered to be strong.

- Isaak (2001) conducted sampling in Crow, Deer, and Sage Creeks in 1996 and 1997 (sampling in July and September). The total number of fish sampled at four reaches in Crow Creek ranged from 11 to 147, with YCT accounting for 8.2 to 100% of total abundance (mountain whitefish were the other dominant species). The total number of fish sampled at five reaches in Deer Creek ranged from 0 to 26 fish, and the proportion of YCT ranged from 65 to 100%. The total number of fish sampled in four reaches in Sage Creek ranged from 17 to 48 fish; the proportion of YCT ranged from 0 to 100% (brown trout were dominant in the two reaches where YCT abundance was low or absent).

- Cegelski et al. (2006) investigated the genetic structure of YCT populations in the Upper Snake River Basin. The Salt River drainage had the highest genetic diversity and lowest genetic differentiation.

According to Meyer (2006 pers comm.), the IDFG does not have a standard for classifying the health of a fish population based on abundance. Crow Creek and its tributaries have average fish densities that are similar to other Palisades/Salt River tributaries, and which are slightly higher than other drainages in the Upper Snake River Basin.

Selenium effects on fish populations occur as a result of deformities and/or death of developing embryos and alevins. However, other stressors can also affect fish populations. How selenium and other stressors may affect salmonids such as trout is shown in Figure 6. Whether reduced survival of eggs or subsequent life stages due to selenium toxicity ultimately affects the population as a whole will depend on the ability of available habitat to assimilate all of the young produced. Typically, habitat is limited and there are natural die-offs of young as they compete with other fish for available resources. This process is called density-dependent mortality. It is entirely possible in habitat-limited situations (which typically would include freshwater streams in the study area) for selenium toxicity not to have any observable differences in fish abundance between exposed and reference streams because selenium water concentrations are not at levels that would be toxic to young fish from other areas moving into areas where local reproduction may have been reduced.

Population-level effects can also occur due to multiple stressors, for example selenium toxicity combined with fishing pressure, floods, etc. However, in these cases it can be extremely difficult to separate out the contributions of the different stressors (i.e., establish causation) without rigid experimental protocols and a study design that explicitly targets selenium toxicity. Natural factors also come into play. For example, resident fish species may be displaced by other species due to natural (e.g., climatic or other environmental changes) or anthropogenic factors (e.g., selenium toxicity), or a combination of both (e.g., selenium toxicity + competition for limited resources).

Immigration and emigration from streams also makes it difficult to evaluate population trends. Fish from an area with populations that are limited by habitat will tend to move into areas where habitat is less limiting (e.g., where the resident fish populations have decreased). Reproductive failure in one section of a stream may be masked by reproductive success in another stream section, if density-dependent movements occur between the two sections.

Finally, when the numbers of fish are very small and the available habitat is fully utilized, it can be very difficult to determine causation for any population changes. Small numbers of fish (i.e., <50) can be severely impacted by a single event, such as a poacher, a single piscivorous bird or mammal, beaver dam, or a short term drought or flood. The risks of demographic extirpation of these small groups of fish because of random effects is high, thus their reestablishment by new emigrants likely occurs frequently. The relatively high variability of small populations over short time scales can give the false impression of trends caused by anthropogenic influences, which may in fact be caused by random natural events.

6.3.3 Selenium Concentrations in Other Media

Data on selenium concentrations including sediment, benthic invertebrates, and aquatic plants (macrophytes and periphyton) were also available for stations associated with the Smoky Canyon Mine.

Sediment: There were 53 measurements of sediment selenium concentrations made at stations within the Salt River Watershed, ranging from background stations in Deer Creek and Crow Creek (upstream of current mining activity) and upper reachs of Sage Creek to downstream stations in lower Sage Creek, Pole Creek, Hoopes Spring, Roberts Creek, lower Smoky Creek and lower Tygee Creek that could be expected to be impacted by phosphate mining activity. Station locations are shown in Figure 7. The sediment chemistry data are summarized in Table 5; most stations were only sampled once or a few times, although stations sampled under different programs were sometimes in reasonably close proximity to each other and data were pooled where appropriate to allow for calculation of means and ranges for sediment selenium concentrations. Overall,

selenium concentrations in sediments ranged from 0.37 to 4.5 µg/g dw at background stations, and from 0.3 to 6.2 µg/g dw at mining-impacted stations (except for a measurement of 58.1 µg/g dw in lower Pole Creek, a mining-impacted station approximately 0.5 miles east of the toe of the waste rock dump. Selenium concentrations in sediments from Crow Creek and Deer Creek ranged from 0.5 to 4.2 µg/g dw. Apart from the Pole Creek result, there was little difference between background and mining-impacted stations.

Benthic Invertebrates: Data on selenium concentrations in benthic invertebrates are summarized in Table 6; station locations are shown in Figure 8. These data should be interpreted with caution because data from different studies were reported on both a wet and dry weight basis. On a dry weight basis, the highest tissue selenium concentration was 15 µg/g dw (lower Sage Creek). On a wet weight basis, the highest tissue selenium concentration was 16.6 µg/g ww (lower Pole Creek). Apart from lower Pole Creek (which has high selenium concentrations in all media), lower Sage Creek and Smoky Creek downstream of the Smoky Canyon Mine, the upstream background stations on Crow Creek and Deer Creek had the highest tissue selenium concentrations in benthic invertebrates. Surface water selenium concentrations were low at these locations, however these elevated concentrations in benthic invertebrate samples appear to be unrelated to current mining activities at the Smoky Canyon Mine.

Aquatic Vegetation: Data on selenium concentrations in aquatic vegetation (including macrophytes and periphyton) are summarized in Table 7; station locations are shown in Figure 9. With the exception of lower Pole Creek (which had the highest selenium concentrations), there was generally little difference in aquatic vegetation concentrations between background and mining-impacted stations.

6.4 Summary

Selenium concentrations in all media were generally highest at stations in lower Pole Creek (which was not unexpected given the proximity of this creek to a waste rock dump), except that there were no data for fish tissue selenium concentrations in lower Pole Creek. Selenium water concentrations were generally low in Crow Creek and Deer Creek (with a few exceptions) relative to other stations. A wider range of tissue selenium concentrations was reported for YCT at background stations (0.44 to 18 µg/g dw) as compared to mining-impacted stations (3.6 and 11 µg/g dw). There was also overlap in brown trout and sculpin tissue concentrations between background and mining-impacted stations. Whole-body selenium tissue concentrations in YCT and other fish species are elevated above the Lemly (1993b) threshold of 4 µg/g dw and the draft USEPA (2004a) TRG of 7.91 µg/g dw, at both background and mining-impacted stations. Work by Hardy (2005) to investigate effects of dietary selenium exposure on YCT indicates the likelihood of negligible impact to YCT populations based on the incidence of larval

deformities. The presence of fish with elevated selenium tissue concentrations at background stations in Crow Creek and Deer Creek (where there is no mining activity but phosphoria outcroppings occur) indicates that these fish are either exposed to natural sources of selenium or that they are migrating from other locations within the drainage system. The fact that population surveys indicated that YCT populations in these creeks are healthy, including comparisons conducted over the past 10 to 20 years, indicates that fish are not being adversely impacted by selenium.

7.0 STATEMENT OF LIMITATIONS

This report has been prepared for the exclusive use of JBR Environmental Consultants, Inc. and is intended to provide a comprehensive assessment of the available scientific information regarding selenium threshold values in water and fish tissue applicable to the proposed Smoky Canyon Mine expansion. Any use that a third party may make of this report, or any reliance on or decisions made based on it, is the responsibility of the third parties. We disclaim responsibility for consequential financial effects on transactions or property values, or requirements for follow-up actions and costs.

The report is based on data and information collected and/or compiled by Golder Associates Ltd. as described in this report. It is based solely on the conditions as described by this report. In evaluating the available information, we have relied in good faith on information provided by others as noted. We assume that the information provided is factual and accurate. We accept no responsibility for any deficiency, misstatement or inaccuracy contained in this report as a result of omissions, misinterpretations or fraudulent acts of persons interviewed or contacted. Assessment has been made using the results of chemical and biological analyses of discrete samples from a limited number of locations. Site conditions between sampling locations have been inferred based on conditions observed at test locations. Additional study can reduce the inherent uncertainties associated with this type of study. However, it is never possible, even with exhaustive sampling and testing, to dismiss the possibility that part of the project area may be different than the parts sampled.

The services performed as described in this report were conducted in a manner consistent with the level of care and skill normally exercised by other members of the engineering and science professions currently practising under similar conditions, subject to the time limits and financial and physical constraints applicable to the services. The content of this report is based on information collected during our investigation, our present understanding of site conditions, the assumptions stated in this report, and our professional judgement in light of such information at the time of this report. This report provides a professional opinion and, therefore, no warranty is expressed, implied, or made as to the conclusions, advice and recommendations offered in this report. This report does not provide a legal opinion regarding compliance with applicable laws. With respect to regulatory compliance issues, it should be noted that regulatory statutes and the interpretation of regulatory statutes are subject to change. The findings and conclusions of this report are valid only as of the date of the report. If new information is discovered in future work, or if the assumptions stated in this report are not met, Golder Associates Ltd. should be requested to re-evaluate the conclusions of this report, and to provide amendments as required.

8.0 CLOSURE

We trust the information contained in this report is sufficient for your present needs. If you have any questions regarding this report, please do not hesitate to contact the undersigned.

Yours very truly,

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9.0 REFERENCES CITED

Adams WJ. 1976. The toxicity and residue dynamics of selenium in fish and aquatic invertebrates. Ph.D. Dissertation. Michigan State University, East Lansing, MI.

Adams WJ, Toll JE, Brix, KV, Tear LM, DeForest DK. 2000. Site-specific approach for setting water quality criteria for selenium: differences between lotic and lentic systems. Proceedings Mine Reclamation Symposium: Selenium Session. Sponsored by Ministry of Energy and Mines, Williams Lake, BC, Canada. June 21 – 22, 2000.

Amweg EL, Stuart DL, Weston DP. 2003. Comparative bioavailability of selenium to aquatic organisms after biological treatment of agricultural drainage water. Aquat Toxicol 63:13-25.

ANZECC/ARMCANZ (Australian and New Zealand Environment and Conservation Council / Agriculture and Resource Management Council of Australia and New Zealand). 2000. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. October 2000.

Barwick M, Maher W. 2003. Biotransference and biomagnification of selenium copper, cadmium, zinc, arsenic and lead in a temperate seagrass ecosystem from Lake Macquarie Estuary, NSW, Australia. Mar Environ Res 56: 471-502.

BCMOE (British Columbia Ministry of Environment). 2001. Water quality guidelines for selenium. Technical appendix. BC Ministry of Environment, Victoria, BC. September 2001.

Belzile N, Chen Y-W, Gunn JM, Tong J, Alarie Y, Delonchamp T, Lang C-Y. 2006. The effect of selenium on mercury assimilation by freshwater organisms. Can J Fish Aquat Sci 63:1-10.

Bennett WN, Brooks AS, Borass ME. 1986. Selenium uptake and transfer in an aquatic food chain and its effect on fathead minnow larvae. Arch Environ Contam Toxicol 15: 513-517.

Berg OK, Bremset G. 1998. Seasonal changes in the body composition of young riverine salmon and brown trout. J Fish Biol 52: 1272-1288.

Bertram PE, Brooks AS. 1986. Kinetics of accumulation of selenium from food and water by fathead minnows. Arch Environ Contam Toxicol 15: 513-517.

Besser JM, Canfield TJ, La Point TW. 1993. Bioaccumulation of organic and inorganic selenium in a laboratory food chain. Environ Toxicol Chem 12: 57–72.

Besser JM, Giesy JP, Brown RW, Buell JM, Dawson GA. 1996. Selenium bioaccumulation and hazards in a fish community affected by coal fly ash effluent. Ecotoxicol Environ Safety 35:7-15.

Beyers DW, Sodergren. 2002. Assessment of exposure of larval razorback sucker to selenium in natural waters. Arch Environ Contam Toxicol 42:53-59.

Biro PA, Morton AE, Pose JR, Parkinson EA. 2004. Over-winter lipid depletion and mortality of age-0 rainbow trout (*Oncorhynchus mykiss*). Can J Fish Aquat Sci 61: 1513-1519.

BLM (Bureau of Land Management). 2005. Draft Environmental Impact Statement: Smoky Canyon Mine, Panels F and G. Report prepared by the Bureau of Land Management, Pocatello, ID, United States Forest Service, and Idaho Department of Environmental Quality. December 2005.

Bond MM. 2000. Characterization and control of selenium releases from mining in the Idaho phosphate region. M.Sc. thesis, University of Idaho.

Brix KV, DeForest DK, Fairbrother A, Adams WJ. 2000. Critical review of tissue-based toxicity thresholds for fish and birds. Proceedings Mine Reclamation Symposium: Selenium Session. Sponsored by Ministry of Energy and Mines, Williams Lake, BC, Canada. June 21-22, 2000.

Brix KV, Volosin JS, Adams WJ, Reash RJ, Carlton RG, McIntyre DO. 2001. Effects of sulphate on the acute toxicity of selenate to freshwater organisms. Environ Toxicol Chem 20:1037-1045.

Brix KV, Toll JE, Tear LM, DeForest DK, Adams WJ. 2005. Setting site-specific water-quality standards by using tissue residue thresholds and bioaccumulation data. Part 2. Calculating site-specific selenium water-quality standards for protecting fish and birds. Environ Toxicol Chem 24:231-237.

Brooke LT, Call DJ, Harting SL, Lindberg CA, Markee TP, McCauley DJ, Poirier SH. 1985. Acute toxicity of selenium(IV) and selenium(VI) to freshwater organisms. Center for Lake Superior Environmental Studies, University of Wisconsin-Superior, Superior, WI.

Buhl KV, Hamilton SJ. 1991. Relative sensitivity of early life stages of Arctic grayling, coho salmon, and rainbow trout to nine inorganics. Ecotoxicol Environ Safety 22:184-197.

Buhl KV, Hamilton SJ. 1996. Toxicity of inorganic contaminants, individually and in environmental mixtures, to three endangered fishes (Colorado squawfish, bonytail, and razorback sucker). Arch Environ Contam Toxicol 30:84-92.

Canton SP. 1999. Acute aquatic life criteria for selenium. Environ Toxicol Chem 18:1425-1432.

Canton SP, VanDerveer WD. 1997. Selenium toxicity to aquatic life: an argument for sediment-based water quality criteria. Environ Toxicol Chem 16: 1255-1259.

Cardwell RD, Foreman DG, Payne TR, Wilbur DJ. 1976. Acute toxicity of selenium dioxide to freshwater fishes. Arch Environ Contam Toxicol 4:129-144.

Carmichael NB, Chapman PM. 2006. Baseline selenium in sculpins related to the northeast coal zone. In: Proceedings of the 30th Annual British Columbia Mining Reclamation Symposium – "Case Studies of Reclamation and Environmental Protection", British Columbia Technical and Research Committee on Reclamation, Smithers, BC. June 19 – 22, 2006.

Carvalho KM, McGettigan MJ, Martin DF. 2001. GC/MS analysis of volatile organic selenium species produced during phytoremediation. J Environ Science Health Part A 36: 1403-1409.

CCME (Canadian Council of Ministers of the Environment). 2005. Canadian water quality guidelines for the protection of aquatic life. Summary table: update October 2005. Canadian Council of Ministers of the Environment, Winnipeg, MB.

CCREM (Canadian Council of Resource and Environment Ministers). 1987. Canadian Water Quality Guidelines. Prepared by the Task Force on Water Quality Guidelines of the Canadian Council of Resource and Environment Ministers.

Cegelski CC, Campbell MR, Meyer KA, Powell MS. 2006. Multiscale genetic structure of Yellowstone cutthroat trout in the Upper Snake River Basin. Trans Am Fish Soc 135: 711-726.

Chapman PM. 2007. Selenium thresholds for fish from cold freshwaters. Human Ecol Risk Assess (in press: Feb 2007 issue).

Chapman PM, McPherson CA. 2004. Possible selenium thresholds for trout. SETAC Globe 5(6): 22-25

Cleveland L, Little EE, Buckler DR, Wiedmeyer RH. 1993. Toxicity and bioaccumulation of waterborne and dietary selenium in juvenile bluegill (*Lepomis macrochirus*). Aquat Toxicol 27: 265-280.

Combs GF Jr, Garbisu C, Yee BC, Yee A, Carlson DE, Smith NR, Magyarosy AC, Leighton T, Buchanan BB. 1996. Bioavailability of selenium accumulated by selenite-reducing bacteria. Biol Trace Elem Res 52: 209- 225.

Connolly PJ, Petersen JH. 2003. Bigger is not always better for overwintering young of the year steelhead. Trans Amer Fish Soc 132: 262-274.

Cooke TD, Bruland KW. 1987. Aquatic chemistry of selenium: Evidence of biomethylation. Environ Sci Technol 21:1214–1219.

Coughlan, DJ, Velte JS. 1989. Dietary toxicity of selenium-contaminated red shiners to striped bass. Trans Am Fish Soc 118: 400-408.

Coyle JJ, Buckler DR, Ingersoll CG, Fairchild JF, May TW. 1993. Effect of dietary selenium on the reproductive success of bluegill sunfish (*Lepomis macrochirus*). Environ Toxicol Chem 12: 551-565.

Crane M, Flower T, Holmes D, Watson S. 1992. The toxicity of selenium in experimental freshwater ponds. Arch Environ Contam Toxicol 23:440-452.

Cumbie PM, Van Horn SL. 1978. Selenium accumulation associated with fish mortality and reproductive failure. Proc Ann Conf Southeast Assoc Fish Wildl Agencies 32:612-624.

DeForest DK, Brix KV, Adams WJ. 1999. Critical review of proposed residue-based selenium toxicity thresholds for freshwater fish. Human Ecol Risk Assess 5: 1187-1228.

DeForest DK, Adams WJ, Brix KV. 2006. Comments on Vidal et al. (2005) and potential implications for tissue-based guidelines.criteria development for selenium. SETAC Globe 7(2): 26-27.

De Rosemond SC, Liber K, Rosaasen A. 2005. Relationship between embryo selenium concentrations and early life stage development in white sucker (*Catostomus commersoni*) from a northern Canadian lake. Bull Environ Contam Toxicol 74: 1134-1142.

Delos C. E-mail correspondence between Charles Delos (USEPA) and Peter Chapman (Golder Associates Ltd) dated June 20, 2006.

Dobbs MG, Cherry DS, Cairns J Jr. 1996. Toxicity and bioaccumulation of selenium to a three-trophic level food chain. Environ Toxicol Chem 15: 340-347.

Duncan DA, Klaverkamp JF. 1983. Tolerance and resistance to cadmium in white suckers (*Catastomus commersoni*) previously exposed to cadmium, mercury, zinc, or selenium. Can J Fish Aquat Sci 40:128-138.

Eckmann R. 2004. Overwinter changes in mass and lipid content of *Perca fluviatilis* and *Gymnocephalus cernuus*. J Fish Biol 65: 1498-1511.

Ellis MM, Motley HL, Ellis MD, Jones RO. 1937. Selenium poisoning in fishes. Proc Soc Exp Biol Med 36:519-522.

Fan TW, Teh SJ, Hinton DE, Higashi RM. 2002. Selenium biotransformations into proteinaceous forms by foodweb organisms of selenium-laden drainage waters in California. Aquat Toxicol 57: 65–84.

Federal Register. 1996. Proposed selenium criterion maximum concentration for the water quality guidance for the Great Lakes system: proposed rule. Federal Register 40 CFR Part 132, November 14, 1996.

Finley KA. 1985. Observations of bluegills fed selenium-contaminated *Hexagenia* nymphs collected from Belews Lake, North Carolina. Bull Environ Contam Toxicol 35: 816-825.

Gamblin M. 2006. Personal communication. Letter from Mark Gamblin, Southeast Regional Supervisor, Idaho Department of Fish and Game, Pocatello, ID to US Forest Service, Caribou National Forest regarding the Smoky Canyon Mine Engineering Evaluation.Cost Analysis. July 24, 2006.

Gillespie RB, Baumann PC. 1986. Effects of high tissue concentrations of selenium on reproduction by bluegills. Trans Am Fish Soc 115:208-213.

GLEC (Great Lakes Environmental Center). 1998. Effect of sulfate concentration on acute toxicity of selenite and selenate to invertebrates and fish. Final report TR-111878 to the Electric Power Research Institute prepared by Great Lakes Environmental Center, Traverse, MI.

Goettl Jr. JP, Davies PH. 1976. Water pollution studies. Job Progress Report, Federal Aid Project F-33-R-11. Colorado Division of Wildlife, Fort Collins, CO. pp. 31-34.

Hamilton SJ. 1995. Hazard assessment of inorganics to three endangered fish in the Green River, Utah. Ecotoxicol Environ Safety 30:134-142.

Hamilton SJ. 2002. Rationale for a tissue based selenium criterion for aquatic life. Aquat Toxicity 57: 85-100.

Hamilton SJ. 2003. Review of residue-based selenium toxicity thresholds for freshwater fish. Ecotoxicol Environ Safety 56: 201-210.

Hamilton SJ, Buhl KJ. 1990. Acute toxicity of boron, molybdenum, and selenium to fry of Chinook salmon and coho salmon. Arch Environ Contam Toxicol 19:366-373.

Hamilton SJ, Buhl KJ. 1997a. Hazard assessment of inorganics, individually and in mixtures, to two endangered fish in the San Juan River, New Mexico. Environ Toxicol Water Qual 12:195:209.

Hamilton SJ, Buhl KJ. 1997b. Hazard assessment of inorganics, singly and in mixtures to flannelmouth sucker *Catastomus latipinnis* in the San Juan River, New Mexico. Ecotox Environ Safety 38:296-308.

Hamilton SJ, Buhl KJ. 2003a. Selenium and other trace elements in water, sediment, aquatic plants, aquatic invertebrates, and fish from streams in southeastern Idaho near phosphate mining operations: September 2000. Final report as part of the USGS Western US Phosphate Project. United States Geological Survey, Columbia Environmental Research Center, Yankton, SD.

Hamilton SJ, Buhl KJ. 2003b. Selenium and other trace elements in water, sediment, aquatic plants, aquatic invertebrates, and fish from streams in southeastern Idaho near phosphate mining operations: May 2001. Final report as part of the USGS Western US Phosphate Project. United States Geological Survey, Columbia Environmental Research Center, Yankton, SD.

Hamilton SJ, Lemly AD. 1999. Water-sediment controversy in setting environmental standards for selenium. Ecotox Environ Safety 44:227-235.

Hamilton SJ, Palace VP. 2001. Assessment of selenium effects in lotic systems. Ecotox Environ Safety 50:161-166.

Hamilton SJ, Wiedmeyer RH. 1990. Concentrations of boron, molybdenum, and selenium in chinook salmon. Trans Am Fish Soc 119: 500-510.

Hamilton SJ, Palmisano AN, Wedemeyer GW, Yasutake WT. 1986. Impacts of selenium on early life stages on early life stages and smoltification of fall chinook salmon. Trans N Amer Wild Nat Res Conf 51: 343-356.

Hamilton SJ, Buhl KJ, Faerber NL. 1989. Toxicity of selenium in the diet to chinook salmon. In: Howard AQ, ed, Selenium and Agricultural Drainage: Implications for San Francisco Bay and the California Environment, Proceedings of the Fourth Selenium Symposium. The Bay Institute of San Francisco, Tiburon, CA, pp 22 – 30.

Hamilton SJ, Buhl KJ, Faerber NL, Wiedmeyer RH, Bullard FA. 1990. Toxicity of organic selenium in the diet to chinook salmon. Environ Toxicol Chem 9: 347-358.

Hamilton SJ, Buhl KL, Bullard FA, McDonald SF. 1996. Evaluation of toxicity to larval razorback of selenium-laden food organisms from Ouray NWR on the Green River, Utah. Final Report. Recovery Implementation Program for Endangered Fish Species in the Upper Colorado River Basin. National Biological Service, Yankton, SD.

Hamilton SJ, Buhl KJ, Bullard FA, Little EE. 2000a. Chronic toxicity and hazard assessment of an inorganic mixture simulating irrigation drainwater to razorback sucker and bonytail. Environ Toxicol 15: 48-64.

Hamilton SJ, Muth RT, Waddell B, May TW. 2000b. Hazard assessment of selenium and other trace elements in wild larval razorback sucker from the Green River, Utah. Ecotoxicol Environ Safety 45: 132-147.

Hamilton SJ, Holley KM, Buhl KL, Bullard FA, Weston LK, McDonald SF. 2001. The evaluation of contaminant impacts on razorback sucker held in flooded bottomland sites near Grand Junction, Colorado – 1996. US Geological Survey, Yankton, SD. Report to the Upper Colorado River Basin Endangered Fish Recovery Program, Denver, CO.

Hamilton SJ, Buhl KJ, Lamothe, PJ. 2002. Selenium and other trace elements in water, sediment, aquatic plants, aquatic invertebrates, and fish from streams in southeastern Idaho near phosphate mining operations: June 2000. Final report as part of the USGS Western US Phosphate Project. United States Geological Survey, Columbia Environmental Research Center, Yankton, SD.

Hamilton SJ, Buhl KJ, Bullard FA, McDonald SF. 2005. Reduced growth and survival of larval razorback sucker fed selenium-laden zooplankton. Ecotox Environ Safety 61:190-208.

Hansen D, Duda P, Zayed AM, Terry N. 1998. Selenium removal by constructed wetlands: role of biological volatilization. Environ Sci Technol 32: 591–597.

Hardy RW. 2005. Effects of dietary selenium on cutthroat trout (*Oncorhynchus clarki*) growth and reproductive performance. Final report prepared for Montgomery Watson Harza (MWH), Bellevue, WA by University of Idaho, Aquaculture Research Institute, Hagerman, ID. December 14, 2005. 44 pp.

Hartwell SI, Jin JH, Cherry DS, Cairns Jr J. 1989. Toxicity versus avoidance response of golden shiner, *Notemigonus crysoleucus*, to five metals. J Fish Biol 35:447-456.

Hermanutz RO. 1992. Malformation of the fathead minnow (*Pimephales promelas*) in an ecosystem with elevated selenium concentrations. Bull Environ Contam Toxicol 49:290-294.

Hermanutz RO, Allen KN, Roush TH, Hedtke SF. 1992. Effects of elevated selenium concentrations on bluegills (*Lepomis macrochirus*) in outdoor experimental streams. Environ Toxicol Chem 11: 217-224.

Hilton FW, Hodson PV, Sliniger SJ. 1980. The requirements and toxicity of selenium in rainbow trout (*Salmo gairdneri*). J Nutr 110: 2527-2535.

Hodson PV, Spry DJ, Blunt BR. 1980. Effects on rainbow trout (*Salmo gairdneri*) of a chronic exposure to waterborne selenium. Can J Fish Aquat Sci 37: 233-240.

Hoffman DJ, Sanderson CJ, LeCaptain LJ, Cromartie E, Pendleton GW. 1992. Interactive effects of arsenate, selenium and dietary protein on survival, growth and physiology in mallard ducklings. Arch Environm Contam Toxicol 22:55-62.

Holm J. 2002. Sublethal effects of selenium on rainbow trout (*Oncorhynchys mykiss*) and brook trout (*Salvelinus fontinalis*). M.Sc. Thesis. Department of Zoology, University of Manitoba, Winnipeg, MB. November 2002.

Holm J, Palace VP, Wautier K, Evans RE, Baron CL, Podemski C, Siwik P, Sterling G. 2003. An assessment of the development and survival of wild rainbow trout (*Oncorhynchus mykiss*) and brook trout (*Salvelinus fontinalis*) exposed to elevated selenium in an area of active coal mining. In: Browman HI and Skiftesvik AB (eds.). The Big Fish Bang. Proceedings of the 26th Annual Larval Fish Conference. Institute of Marine Research, Bergen, Norway.

Holm J, Palace V, Siwik P, Sterling G, Evans R, Baron C, Werner J, Wautier K. 2005. Developmental effects of bioaccumulated selenium in eggs and larvae of two salmonid species. Environ Toxicol Chem. 24: 2372-2381.

Hunn JB, Hamilton SJ, Buckler DR. 1987. Toxicity of sodium selenite to rainbow trout fry. Water Res 21: 233-238.

IDEQ (Idaho Department of Environmental Quality). 2006. Water Quality Standards. Idaho Department of Environmental Quality. Idaho Administrative Code (IAC), IDAPA 58.01.02. April 2006.

IJC (International Joint Commission). 1981. Report of the Aquatic Ecosystem Objectives Committee. Great Lakes Science Advisory Board, International Joint Commission, Windsor, ON.

Isaak DJ. 2001. A landscape ecological view of trout populations across a Rocky Mountain watershed. PhD Thesis. Department of Zoology and Physiology, University of Wyoming, Laramie, WY.

Jarvinen AW, Ankley GT. 1999. Linkage of effects to tissue residues: development of a comprehensive database for aquatic organisms exposed to inorganic and organic chemicals. SETAC Press, Pensacola, FL, USA. 364 pp.

Kennedy CJ, McDonald LE, Loveridge R, Strosher MM. 2000. The effect of bioaccumulated selenium on mortalities and deformities in the eggs, larvae and fry of a wild population of cutthroat trout (*Oncorhynchus clarki lewisi*). Arch Environ Contam Toxicol 39:46-52.

Kimball G. Manuscript. The effects of lesser known metals and one organic to fathead minnows (*Pimephales promelas*) and *Daphnia magna*. (Available from CE Stephan, USEPA, Duluth, MN).

Klaverkamp JF, Hodgins DA, Lutz A. 1983. Selenite toxicity and mercury-selenium interactions in juvenile fish. Arch Environ Contam Toxicol 12:405-413.

Lemly AD. 1985. Toxicology of selenium in a freshwater reservoir: Implications for environmental hazard evalutaion and safety. Ecotoxicol Environ Safety 10: 314-338.

Lemly AD. 1993a. Metabolic stress during winter increases the toxicity of selenium to fish. Aquat Toxicol 27: 133-158.

Lemly AD. 1993b. Guidelines for evaluating selenium data from aquatic monitoring and assessment studies. Environ Monit Assess 28:83-100.

Lemly AD. 1993c. Teratogenic effects of selenium in natural populations of freshwater fish. Ecotoxicol Environ Safety 26: 181-204.

Lemly AD. 1996a. Winter stress syndrome: an important consideration for hazard assessment of aquatic pollutants. Ecotoxicol Environ Safety 34: 223-227.

Lemly AD. 1996b. Selenium in aquatic organisms. In: Beyer WN, Heinz GH, Redmon-Norwood AW, eds, Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. CRC Press, Boca Raton, FL, pp 427-445.

Lemly AD. 1997a. Role of season in aquatic hazard assessment. Environ Monit Assess 45: 89-98.

Lemly AD. 1997b. Ecosystem recovery following selenium contamination in a freshwater reservoir. Ecotoxicol Environ Safety 36: 275-281.

Lemly AD. 1997c. A teratogenic deformity index for evaluating impacts of selenium on fish populations. Ecotoxicol Environ Safety 36: 259-266.

Lemly AD. 2002. Selenium Assessment in Aquatic Ecosystems: A Guide for Hazard Evaluation and Water Quality Criteria. Springer-Verlag, New York, NY.

Lemly AD, Smith GJ. 1987. Aquatic cycling of selenium: Implications for fish and wildlife. Leaflet No. 12. U.S. Fish and Wildlife Service, Washington, DC.

Lemly AD, Finger SE, Nelson MK. 1993. Sources and impacts of irrigation drainwater contaminants in arid wetlands. Environ Toxicol Chem 12: 2265-2279.

Linville RG, Luoma SN, Cutter L, Cutter GA. 2002. Increased selenium threat as a result of invasion of the exotic bivalve *Potamocorbula amurensis* into the San Francisco Bay-Delta. Aquat Toxicol 57: 51–64.

Liu DL, Yang YP, Hu MH, Harrison PJ, Price NM. 1987. Selenium content of marine food chain organisms from the coast of China. Mar Environ Res 22: 151–165.

Lohner TW, Reash RJ, Willet VE, Rose LA. 2001. Assessment of tolerant sunfish populations (*Lepomis* sp.) inhabiting selenium-laden coal ash effluents. Ecotoxicol Environ Safety 5: 203-216.

Luoma SN, Johns C, Fisher NS, Steinberg NA, Oremland RS, Reinfelder JR. 1992. Determination of selenium bioavailability to a bivalve from particulate and solute pathways. Environ Sci Technol 26: 485–491.

Maier KJ, Knight AW. 1994. Ecotoxicology of selenium in freshwater systems. Rev Environ Contam Toxicol 134: 31-48.

Maier KJ, Foe CG, Knight AW. 1993. Comparative toxicity of selenate, selenite, seleno-DL-methionine and seleno-DL-cystine to *Daphnia magna*. Environ Toxicol Chem 12: 755-763.

Malchow DE, Knight AW, Maier KJ. 1995. Bioaccumulation and toxicity of selenium in *Chironomus decorus* larvae fed a diet of seleniferous *Selenastrum capricornutum*. Arch Environ Contam Toxicol 29: 104-109.

Malloy JC, Meade ML, Olsen EW. 1999. Small-scale spatial variation of selenium concentrations in chironomid larvae. Bull Environ Contam Toxicol 62: 122-129.

Mariah (Mariah Associates Inc.) 1980. Baseline aquatic ecology report for the Smoky Canyon Phosphate Lease, Southeast Idaho. Prepared for Maxim Technologies.

Masscheleyn PH, Delaune RD, Patrick WHJ. 1990. Transformation of selenium as affected by sediment oxidation–reduction potential and pH. Environ Sci Technol 24: 91–96.

Maxim (Maxim Technologies, Inc.). 2002. Revised final water resources baseline report, Smoky Canyon Mine, Caribou County, ID.

Maxim. 2004a. Baseline technical report, fisheries and aquatic resources, Manning Creek and Deer Creek phosphate lease areas (Panels F&G). Smoky Canyon, Caribou County, ID.

Maxim. 2004b. Baseline technical report, water resources, Manning Creek and Deer Creek phosphate lease areas (Panels F&G). Smoky Canyon, Caribou County, ID.

Maxim. 2006. Second addendum to baseline technical report, fisheries and aquatic resources, Manning Creek and Deer Creek phosphate lease areas (Panels F&G). Smoky Canyon, Caribou County, ID. Prepared by Maxim Technologies, Helena, MT for JR Simplot, Pocatello, ID.

Mayer Jr FL, Ellersieck MR. 1986. Manual of acute toxicity: interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resource Publication No. 160. US Fish and Wildlife Service, Washington, DC, 438 pp.

McDonald LE, Kennedy CJ. 2002. Reply to commentary on the effects of selenium bioaccumulation in a wild population of cutthroat trout. SETAC Globe 3(4):36-37.

McNeal JM, Balistrieri LS. 1989, Geochemistry and occurrence of selenium - an overview. In: Jacobs LW, ed, Selenium in Agriculture and the Environment. Madison, WI. American Society of Agronomy and Soil Science Society of America, pp 1-13.

Meyer KA. 2006. Personal communication. E-mail from Kevin Meyer, Idaho Department of Fish and Game [IDFG] to Laura Arneson, JBR Environmental Consultants, August 8, 2006.

Meyer KA, Lamansky JA. 2004. Assessment of native salmonids above Hells Canyon Dam, Idaho. Project Progress Report. 2003 Annual Report. Idaho Department of Fish and Game, Boise, ID. IDFG Report No. 04-26.

Meyer KA, Schill DJ, Elle FS, Schrader WC. 2003. A long-term comparison of Yellowstone cutthroat trout abundance and size structure in their historical range in Idaho. North Am J Fish Man 23:149-162.

MFG (MFG, Inc.). 2003a. Draft Smoky Canyon Mine Area B (tailings ponds area) baseline ecological risk assessment report. Prepared by MFG, Inc., Boulder, CO for JR Simplot Co., Pocatello, ID.

MFG (MFG, Inc.). 2003b. Draft Smoky Canyon Mine Area B (tailings impoundments) groundwater and environmental media investigation report. Prepared by MFG, Inc., Boulder, CO for JR Simplot Co., Pocatello, ID.

Miller LL, Rasmussen JB, Palace V, Hontela A. 2006. The effects of selenium on the physiological stress response of rainbow trout and brook trout from a coal mining area. Metals in the Human Environment (MITHE) Research Symposium, Gatineau, QC, Canada. January 26 – 27, 2006.

Montgomery Watson. 1998. Fall 1997 interim surface wate survey report, Southeast Idaho phosphate resource area selenium project. Idaho Mining Association Selenium Committee.

Montgomery Watson. 1999. Final 1998 regional investigation report, Southeast Idaho phosphate resource area selenium project. Idaho Mining Association Selenium Committee.

Montgomery Watson. 2000. 1999 Interim investigation data report, Southeast Idaho phosphate resource area selenium project. Idaho Mining Association Selenium Committee.

Montgomery Watson. 2001a. Draft 1999-2000 regional investigation data report for surface water, sediment and aquatic biota sampling activities, September 1999. Southeast Idaho phosphate resource area selenium project. Idaho Mining Association Selenium Committee.

Montgomery Watson. 2001b. Draft 1999-2000 regional investigation data report for surface water, sediment and aquatic biota sampling activities, May – June 2000. Southeast Idaho phosphate resource area selenium project. Idaho Mining Association Selenium Committee.

Muscatello JR, Bennett PM, Himbeault KT, Belknap AM, Janz DM. In Review. Larval deformities associated with selenium accumulation in northern pike (*Esox lucius*) exposed to metal mining effluent. Submitted to Environ. Sci. Technol.

Newbold JD, O'Neill RV, Elwood JW, Van Winkle W. 1982a. Nutrient spiralling in streams: implications for nutrient limitation and invertebrate activity. Am Nat 120: 628-652.

Newbold JD, Mulholland PJ, Elwood JW, O'Neill RV. 1982b. Organic spiralling in stream ecosystems. Oikos 38: 266-272.

NewFields. 2005. Final site investigation report, Smoky Canyon Mine, Caribou, County, Idaho. Prepared by NewFields, Boulder, CO for JR Simplot Co., Pocatell, ID.

Ogle RS, Knight AW. 1989. Effects of elevated foodborne selenium on growth and reproduction of the fathead minnow (*Pimephales promelas*). Arch Environ Contam Toxicol 18: 795-803.

Ohlendorf HM, Hoffman DJ, Saiki MK, Aldrich TW. 1986. Embryonic mortality and abnormalities of aquatic birds: Apparent impacts of selenium from irrigation drainwater. Sci Total Environ 52 :49-63.

Ohlendorf HM, Hothem RL, Welsh D. 1989. Nest success, cause-specific nest failure, and hatchability of aquatic birds at selenium contaminated Kesterson Resevoir and a reference site. Condor 91: 787–796.

Orr PL, Guiguer KR, Russel CK. 2006. Food chain transfer of selenium in lentic and lotic habitats of a western Canadian watershed. Ecotoxicol Environ Safety 63: 175-188.

Palace VP, Spallholz JE, Holm J, Wautier K, Evans RE, Baron CE. 2004. Metabolism of selenomethionine by rainbow trout (*Oncorhynchus mykiss*) embryos can generate oxidative stress. Ecotox Environ Safety 58:17-21.

Palawski D, Hunn JB, Dwyer FJ. 1985. Sensitivity of young striped bass to organic and inorganic contaminants in fresh and saline waters. Trans Am Fish Soc 114:748-753.

Post JR, Evans DO. 1989. Size-dependent overwinter mortality of young-of-the-year yellow perch (*Perca flavescens*): laboratory, in situ enclosure and field experiments. Can J Fish Aquat Sci 46: 1958-1968.

Presser TS, Ohlendorf HM. 1987. Biogeochemical cycling of selenium in the San Joaquin Valley, California. Environ Manag 11: 805-821.

Presser TS, Sylvester MA, Low WH. 1994. Bioaccumulation of selenium from natural geologic sources in western states and its potential consequences. Environ Manag 18: 423-436.

Reading JT. 1979. Acute and chronic effects of selenium on *Daphnia pulex*. M.S. Thesis, Virgina Polytechnic Institute and State University, Blacksburg, VA.

Rosetta TN, Knight AW. 1995. Bioaccumulation of selenate, selenite, and seleno-DL-methionine by the brine fly larvae *Ephydra cinerea* Jones. Arch Environ Contam Toxicol 29: 351-357.

Saiki MK, Lowe TP. 1987. Selenium in aquatic organisms from subsurface agricultural drainage water, San Joaquin Valley, California (USA). Arch Environ Contam Toxicol 16: 657-670

Saiki MK, Jennings MR, Wiedmeyer RH. 1992. Toxicity of agricultural subsurface drainwater from the San Joaquin Valley, California, to juvenile chinook salmon and striped bass. Trans Am Fish Soc 121: 78-93.

Saiki MK, Jennings MR, Brumbaugh WG. 1993. Boron, molybdenum and selenium in aquatic food chains from the Lower San Joaquin River and its tributaries California. Arch Environ Contam Toxicol 24: 307-319.

Saiki MK, Martin BA, May TW. 2004. Reproductive status of western mosquitofish inhabiting selenium-contaminated waters in the Grassland Water District, Merced County, California. Arch Environ Contam Toxicol 47: 363-369.

Sappington KG. 2002 Development of aquatic life criteria for selenium: a regulatory perspective of critical issues and research needs. Aquat Toxicol 57: 101-113.

Sato T, Ose Y, Sakai T. 1980. Toxicological effect of selenium on fish. Environ Pollut 21A:217-224.

Schlekat CE, Dowdle PR, Lee B-G, Luoma SN, Oremland RS. 2000. Bioavailability of particle-associated Se to the bivalve *Potamocorbula amurensis*. Environ Sci Technol 34: 4505-4510.

Schlekat CE, Purkerson DG, Luoma SN. 2004. Modeling selenium bioaccumulation through arthropod food webs in San Francisco Bay, California, USA. Environ Toxicol Chem 23: 3003–3010.

Schultz R, Hermanutz R. 1990. Transfer of toxic concentrations of selenium from partent to progeny in the fathead minnow (*Pimephales promelas*). Bull Environ Contam Toxicol 19: 845-853.

Skorupa JP. 1998. Selenium poisoning of fish and wildlife in nature: lessons from twelve real-world examples. In: Frankenberger W, Engberg RA, eds, Environmental Chemistry of Selenium, Marcel Dekker Inc, NY, pp 315-354.

Skorupa JP. 1999. Beware missing data and undernourished statistical models: comment on Fairbrother et al's critical evaluation. Human Ecol Risk Assess 5: 1255-1262.

Skorupa JP, Ohlendorf HM. 1991. Contaminants in drainage water and avian risk thresholds. In: Dinar A, Zilberman D, eds, The economics and management of water and drainage in agriculture. Kluwer, New York, NY, pp 345–368.

Skorupa JP, Mormon SP, Sefchick-Edwards JS. 1996. Guidelines for interpreting selenium exposure of biota associated with non-marine aquatic habitats. US Fish and Wildlife Service, National Irrigation Water Quality Program, Sacramento, CA, USA.

Southworth GR, Peterson MJ, Ryon MG. 2000. Long-term increased bioaccumulation of mercury in largemouth bass follows reduction of waterborne selenium. Chemosphere 41:1101-1105.

Spehar RL. 1986. Memorandum to DJ Call, Center for Lake Superior Environmental Studies, University of Wisconsin-Superior, Superior, WI. US Environmental Protection Agency, Duluth, MN. September 16, 1986.

Stanley TR Jr, Spann JW, Smith GJ, Rosscoe R. 1994. Main and interactive effects of arsenic and selenium on mallard reproduction and duckling growth and survival. Arch Environ Contam Toxicol 26: 444-451.

Staub BP, Hopkins WA, Novak J, Congdon JD. 2004. Respiratory and reproductive characteristics of eastern mosquitofish (*Gambusa holbrooki*) inhabiting a coal ash settling basin. Arch Environ Contam Toxicol 46: 96-101.

Steinhart GB, Wurtsbaugh WA. 2003. Winter ecology of kokanee: implications for salmon management. Trans Amer Fish Soc 132: 1076-1088.

Stewart AR, Luoma SN, Schlekat CE, Doblin MA, Hieb KA. 2004. Food web pathway determines how selenium affects aquatic ecosystems: a San Francisco Bay case study. Environ Sci Technol 38:4519-4526.

Swift MC. 2002. Stream ecosystem response to, and recovery from, experimental exposure to selenium. J Aquat Ecosyst Stress Recovery 9: 159-184.

Takayangi K, Wong GTF. 1984. Total selenium and selenium(IV) in the James River estuary and southern Chesapeake Bay. Estuar Coastal Shelf Sci 18: 113-119

Terry N, Zayed AM. 1994. Selenium volatilization by plants. In: Frankenberger WT Jr, Benson S, eds, Selenium in the Environment. Marcel Dekker, New York, NY, pp 343-367.

Thomas BV, Knight AW, Maier KJ. 1999. Selenium bioaccumulation by the water boatman *Trichocorixa reticulata* (Guerin-Meneville). Arch Environ Contam Toxicol 36: 295-300.

Thompson-Eagle ET, Frankenberger WT Jr. 1990. Volatilization of selenium from agricultural evaporation pond water. J Environ Qual 19:125-131.

Thurow RF, Corsi CE, Moore VK. 1988. Status, ecology and management of Yellowstone cutthroat trout in the Upper Snake River Basin, Idaho. pp. 26 – 36. In: Gresswell RE (ed). Status and Management of Interior Stocks of Cutthroat Trout. American Fisheries Society Symposium 4. Bethesda, MD.

Toll JE, Tear LM, DeForest DK, Brix KV, Adams WJ. 2005. Setting site-specific water-quality standards by using tissue residue criteria and bioaccumulation data. Part 1. Methodology. Environ Toxicol Chem 24:224-230.

TtEMI (Tetra Tech EM Inc.). 2002a. Final area-wide human health and ecological risk assessment, selenium project, Southest Idaho phosphate mining resource area. Prepared for Idaho Department of Environmental Quality (IDEQ), Potatello, ID by Tetra Tech EM Inc, Boise, ID.

TtEMI. 2002b. Final 2001 total maximum daily load baseline monitoring report. Upper Blackfoot, Salt, and Bear River Watersheds. Southern Idaho Phosphate Resource Area. Prepared for Idaho Department of Environmental Quality (IDEQ), Potatello, ID by Tetra Tech EM Inc, Boise, ID.

TtEMI. 2002c. Final 2002 supplement to 2001 total maximum daily load baseline monitoring report. Southern Idaho Phosphate Resource Area. Prepared for Idaho Department of Environmental Quality (IDEQ), Potatello, ID by Tetra Tech EM Inc, Boise, ID.

TtEMI. 2004. Final 2003 supplement to 2001 total maximum daily load baseline monitoring report. Southern Idaho Phosphate Resource Area. Prepared for Idaho Department of Environmental Quality (IDEQ), Potatello, ID by Tetra Tech EM Inc, Boise, ID.

USDOI (US Department of Interior). 1998. Guidelines for interpretation of the biological effects of selected constituents in biota, water and sediment: selenium. National Irrigation Water Quality Program Information Report No. 3.

USEPA (US Environmental Protection Agency). 1980. Ambient water quality criteria for selenium. Criteria and Standards Division, Washington, DC. EPA-440/5-80-070.

USEPA. 1985. Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses. Washington, DC.

USEPA. 1987. Ambient water quality criteria for selenium – 1987. Office of Water, Washington, DC. EPA-440/5-87-008.

USEPA. 1998. report on the peer consultation workshop on selenium aquatic toxicity and bioaccumulation. Washington, DC. EPA-822-R-98-007.

USEPA. 2004a Draft aquatic life water quality criteria for selenium – 2004. Office of Water and Office of Science and Technology, Washington, DC. EPA/822/D-04/001.

USEPA. 2004b. National recommended water quality criteria. Office of Water, Washington, DC.

USFS (US Forest Service). 2003. Final environmental impact statement – Caribou Revised Forest Plan. US Department of Agriculture, Forest Service, Caribou-Targhee National Forest, Idaho Falls, ID.

USFWS (US Fish and Wildlife Service). 1990. Agricultural irrigation drainage studies in support of the San Joaquin Valley Drainage Program – final report. National Fisheries Contaminant Research Center, Columbia MO.

USGS (US Geological Survey). 2002. Mineral Resources Program. Western Phosphate Field, USA: science in support of land management. USGS Fact Sheet FS-100-02.

Van Derveer WD, Canton SP. 1997. Selenium sediment toxicity thresholds and derivation of water quality criteria for freshwater biota of western streams. Environ Toxicol Chem 16: 1260-1268.

Varley JD, Gresswell RE. 1988. Ecology, status, and management of the Yellowstone cutthroat trout. Amer Fish Soc Symp 4: 13-24.

Vickerman DB, Trumble JT, George GN, Pickering II, Nichol H. 2004. Selenium biotransformations in an insect ecosystem: Effects of insects on phytoremediation. Environ Sci Technol 38: 3581-3586.

Vidal D, Bay SM, Schlenk D. 2005. Effects of dietary selenomethionine on larval rainbow trout (*Oncorhynchus mykiss*). Arch Environ Contam Toxicol 49: 71-75.

Wang C, Lovell RT. 1997. Organic selenium sources, selenomethionine and selenoyeast, have higher bioavailability than an inorganic source, sodium selenite, in diets for channel catfish (*Ictalurus punctatus*). Aquaculture 152: 223-234.

Wayland M, Crosley R. 2006. Selenium and other trace elements in aquatic insects in coal mine-affected streams in the Rocky Mountains of Alberta, Canada. Arch Environ Contam Toxicol 50: 511-22.

Weber R. 2005a. Technical report (May 14, 2005). Prepared for Greater Yellowstone Coalition, Idaho Falls, ID by Frank X. Weber, Research Triangle Institute, Research Triangle Park, NC.

Weber F. 2005b. Technical Report (September 19, 2005). Prepared for Greater Yellowstone Coalition, Idaho Falls, ID by Frank X. Weber, Research Triangle Institute, Research Triangle Park, NC.

Zawislanski PT, Zavarin M. 1996. Nature and rates of selenium transformations in Kesterson Reservoir soils: A laboratory study. Soil Sci Soc Am J 60: 791–800.

Zhang YQ, Moore JN. 1996. Selenium speciation and fractionation in a wetland system. Environ Sci Technol 30: 2613–2619.

Zhang YQ, Moore JN. 1997. Interaction of selenate with a wetland sediment. Appl Geochem 12: 685–691.

TABLES

TABLE 1: Summary of Whole-Body Selenium Concentrations (dw) Associated with Adverse Effects as Summarized in Lemly (1993b, 1996a, 2002), DeForest et al. (1999) and Hamilton (2002).

Study	Species and Exposure Route	Table 1 from Lemly (1993b)	Table 1 from Lemly (1996a); Table 2.2 from Lemly (2002)	Table 3 from DeForest et al. (1999)	Table 1 from Hamilton (2002)
Bennett et al. (1986)	Larval fathead minnow exposed to dietary selenium	Not summarized	Not summarized	Reduced growth at the lowest concentration measured (i.e. <43 µg/g dw)	Reduced growth at 43-61 µg/g dw
Bertram and Brooks (1986)	Fathead minnow exposed to waterborne and dietary selenium	Not summarized	Not summarized	No effect on growth at highest concentration tested (i.e., >2.2 µg/g dw)	Not summarized
Coughlan and Velte (1989)	Striped bass exposed to dietary selenium	Mortality at 14 µg/g dw	Mortality at 14 µg/g dw	Not summarized	Mortality at 15 µg/g dw
Coyle et al. (1993)	Adult bluegills were exposed to waterborne and dietary selenium	Offspring from female fish with whole-body tissue concentrations of 16 µg/g dw failed to survive beyond the swim-up stage	Offspring from female fish with whole-body tissue concentrations of 19 µg/g dw failed to survive beyond the swim-up stage	Geometric mean of NOEC and LOEC for larval survival was 10.6 µg/g dw	Reproductive failure at 19 µg/g dw
Dobbs et al. (1996)	Fathead minnow exposed to waterborne and dietary selenium	Not summarized	Not summarized	Reduced growth at concentrations of 47.5 – 76.0 µg/g dw	Not summarized
Finley 1985	Bluegills exposed to dietary selenium	Mortality at 20 µg/g dw (muscle concentration)	Mortality at 20 µg/g dw (muscle concentration)	Study did not report whole-body tissue data	Mortality at 31 µg/g dw (muscle concentration)
Hamilton et al. (1986, 1989, 1990)	Juvenile chinook salmon exposed to waterborne and dietary selenium	Impaired growth at 3 µg/g dw; impaired smolting at 9.5 µg/g dw; mortality at concentrations greater than 10 µg/g dw	Impaired growth at 2 µg/g dw;impaired smolting at 20 µg/g dw; mortality at concentrations greater than 5 µg/g dw	Geometric means of NOECs and LOECs for growth and survival after 60 days were 7.4 and 16 µg/g dw, respectively	Reduced growth at 4.0 and 10.8 µg/g dw (different diets);reduced migration at 8.4 µg/g dw; mortality at 5.4 and 6.5 µg/g dw (different diets)
Hamilton et al. (1996, 2001)	Razorback suckers exposed to dietary selenium	Not summarized	Not summarized	Not summarized	Mortality at 3.6 – 8.7 µg/g dw
Hamilton et al. 2000a	Bonytail exposed to waterborne selenium	Not summarized	Not summarized	Not summarized	Reduced growth at 9.4 µg/g dw

Study	Species and Exposure Route	Table 1 from Lemly (1993b)	Table 1 from Lemly (1996a); Table 2.2 from Lemly (2002)	Table 3 from DeForest et al. (1999)	Table 1 from Hamilton (2002)
Hamilton et al. (2000a)	Razorback suckers exposed to waterborne selenium	Not summarized	Not summarized	Not summarized	Reduced growth at 5.9 µg/g dw
Hermanutz et al. (1992)	Fathead minnow exposed to waterborne selenium in outdoor artificial streams (food source was insects in the artificial streams)	Reproductive failure when adults had concentrations of 12 µg/g dw	Reproductive failure when adults had concentrations of 18 µg/g dw	Significant decreases in hatchability and larval survival at <18.4 µg/g dw	Not summarized
Hodson et al. (1980); Hilton et al. (1980)	Rainbow trout fry exposed to waterborne and dietary selenium	Significant changes in blood chemistry at 3 µg/g dw. Reduced survival at 5 µg/g dw	Significant changes in blood chemistry at 2 µg/g dw. Reduced survival at 5 µg/g dw	Study did not report whole-body tissue data; Hodson et al. (1980) not cited	Mortality and reduced growth at 5.2 µg/g dw (carcass concentration) Hodson et al. (1980) not cited
Hunn et al. (1987)	Rainbow trout fry exposed to waterborne selenium	Significant mortality at a whole-body tissue concentration of 4 µg/g dw	Significant mortality at a whole-body tissue concentration of 1 µg/g dw	Geometric mean of NOEC and LOEC was 3.3 µg/g dw	Mortality and reduced length at 5.2 µg/g dw
Lemly (1993a)	Bluegills exposed to waterborne and dietary selenium	Not summarized	Not summarized	No effects of survival at concentrations > 6.0 µg/g dw; 34% mortality at concentrations of 7.9 µg/g under simulated winter stress	Mortality at 5.5 µg/g dw
Lemly (1993c)	Field populations of centrarchids	Teratogenic defects at 15 µg/g dw	Not summarized	Teratogenic defects at 15 µg/g dw	Not summarized
Ogle and Knight (1989)	Fathead minnow exposed to waterborne and dietary selenium	Impaired growth at 6 µg/g dw	Impaired growth at 5 µg/g dw	No effect on reproduction at highest concentration measured (>7.5 µg/g dw)	Reduced growth at 5.4 µg/g dw
Schultz and Hermanutz (1990)	Fathead minnow exposed to waterborne selenium in outdoor artificial streams	Reduced fry survival when fry had concentrations of 8 µg/g dw	Reduced fry survival when fry had concentrations of 16 µg/g dw	Study did not report whole-body tissue data	Not summarized
USFWS (1990); Cleveland et al. (1993)	Juvenile bluegills exposed to dietary selenium	Mortality was associated with 5 µg/g dw	Mortality was associated with 5 µg/g dw	Geometric mean of NOEC and LOEC for survival was 4.4 µg/g dw (water-only exposure)	Mortality at 4.3 µg/g dw

TABLE 2: Summary of Egg Effects Threshold Levels for Selenium.

Egg Se Concentration (ug/g dw)	Holm et al. (2005)		Kennedy et al. (2000)	Hardy (2005)	De Rosemond et al. (2005)	Muscatello et al. (in review)	Lemly (1993b)
	Rainbow trout	Brook trout	Cutthroat trout	Cutthroat trout	White sucker	Northern pike	Freshwater and Anadromous Fish
'Effects Threshold'	32-40[1]	>26.4-31.2[2]	>21.2[3]	>16.04 – 18.0[4]	25.6[5]	32.2[6]	10

1. Threshold of between 8 and 10 µg/g wet weight; converted to dry weight based on 75% moisture.
2. No increase in larval deformities at 6.6 and 7.8 µg/g wet weight; converted to dry weight based on 75% moisture.
3. Mean value; no effects found at egg Se concentrations as high as 81.3 µg/g dry weight.
4. Mean values; no effects.
5. Mean value; corresponds to a mean frequency of deformity of 12.8%.
6. EC20 value for larval deformities relative to reference.

TABLE 3: Summary of Selenium Concentrations Measured in Surface Water at Stations within the Salt River Watershed.

Golder Station ID[1]	Designation	Date Range	Number of Measurements	Mean Total Se (ug/L)	Min Total Se (ug/L)	Max Total Se (ug/L)
Crow Creek above Sage Creek						
CC	BK	2001	1	<2	<2	<2
CC-100	BK	2003 - 2004	5	0.79	0.28	0.97
CC-2	BK	2003 - 2004	4	0.23	<0.2	<1
CC-3	BK	Not reported	2	1.10	0.99	1.21
CC-300	BK	2004	2	0.56	0.48	0.63
CC-5	BK	2001	3	<1	<1	1.1
CC-50	BK	2004	2	0.4	0.3	0.51
LC-500	BK	2002 - 2004	7	1.2	<0.3	3
STC-500	BK	2003 - 2004	3	3.93	1.2	7.6
STC-700	BK	2004	2	0.68	0.47	0.88
WC-800	BK	2003 - 2004	4	0.7	0.49	0.87
Deer Creek						
Deer Creek	BK	2001 - 2004	23	1.32	0.50	2.85
Deer Creek North Fork	BK	2002 - 2003	16	0.68	<0.2	0.98
Deer Creek South Fork	BK	1998 - 2004	5	1.03	<0.14	2
Pole Creek (above waste dump)						
PC-3	BK	1979 - 2005	48	0.59	<0.2	4
PC-5	BK	2004	2	0.32	<0.3	0.5
Upper Sage Creek						
USC-2	BK	2001	1	<1.0	<1.0	<1.0
USC-3	BK	2000	2	<1.0	<1.0	<1.0
USC-4	BK	1990 - 2005	36	0.83	<0.2	11
USC-5	BK	2002	2	<1.0	<1.0	<1.0
Sage Creek South Fork						

Golder Station ID[1]	Designation	Date Range	Number of Measurements	Mean Total Se (ug/L)	Min Total Se (ug/L)	Max Total Se (ug/L)
SCSF-1	BK	2003	4	1.80	<1.0	4.2
SCSF-2	BK	2001 - 2003	8	0.9	<1.0	2.2
SCSF-200	BK	2003	2	0.3	<0.2	<1.0
SCSF-4	BK	1979 - 2005	44	1.7	<1.0	4.3
SCSF-5	BK	1992 - 2005	20	0.45	<0.2	1
SCSF-500	BK	2002 - 2003	2	0.30	<0.2	<1.0
SCSF-6	BK	1998 - 2000	5	0.35	<0.27	<1.0
SCSF-7	BK	2003	1	<1.0	<1.0	<1.0
Smoky Creek, upper						
SMC-1	BK	1998 - 2001	5	0.49	0.18	<1.0
SMC-2	BK	1979 - 2005	47	0.67	<0.3	3
SMC-3	BK	2001	1	<2.0	<2.0	<2.0
Tygee Creek						
TCEF	BK	1990 - 2001	21	1.02	<1.0	5
TCU-1	BK	1990 - 2005	36	0.65	<0.2	5
TCU-2	BK	1979 - 1987	13	0.77	<1.0	2
Crow Creek below Sage Creek						
CC-1	MI	2002 - 2004	7	1.19	<0.3	3
CC-4	MI	Not reported	4	2.81	2.03	3.64
CC-800	MI	2003 - 2004	4	0.73	<0.2	1.7
Hoopes Spring						
HS	MI	1979 - 2004	48	6.7	<1.0	17
HS-2	MI	2002	2	9.5	9	10
HS-3	MI	2002 - 2003	4	9.85	9	11
HS-4	MI	2003	3	10.3	10	11
Sage Creek North Fork						
SCNF-1	MI	1979 - 2004	12	4.7	0.43	41

Golder Station ID[1]	Designation	Date Range	Number of Measurements	Mean Total Se (ug/L)	Min Total Se (ug/L)	Max Total Se (ug/L)
SCNF-2	MI	1979 - 2004	12	0.44	<0.2	1.3
SCNF-3	MI	2002 - 2004	8	1.19	<0.2	3.7
SCNF-4	MI	1997	4	1.73	<0.112	3.37
Lower Sage Creek						
LSC-1	MI	2003	2	4.7	4	5.4
LSC-2	MI	2001	1	4	4	4
LSC-3	MI	2002 - 2005	9	4.7	<1.0	7
LSC-4	MI	2003	2	8.2	8	8.4
LSC-5	MI	1999 - 2003	5	6.6	3.3	9.1
LSC-6	MI	2002 - 2004	6	1.4	<1.0	3.6
LSC-7	MI	1990 - 2005	43	0.56	<0.07	4
LSC-8	MI	Not reported	2	6.13	4.58	7.68
SCHS-1	MI	2002 - 2004	6	7.5	5.2	8.8
SCHS-2	MI	2003	2	0.9	<1.0	1.3
SCM	MI	2001 - 2004	22	4.3	<0.3	6.1
SCPC	MI	2001 - 2003	8	0.78	<1.0	1.2
Pole Creek						
PC-1	MI	2004	3	210	164	248
PC-2	MI	2004	1	250	250	250
PC-4	MI	1997 - 2004	5	1012	680	1500
PC-6	MI	1979 - 2005	30	432	<1.0	1330
PC-7	MI	1999	2	2350	2300	2400
PC-8	MI	1997	4	588	566	612
Roberts Creek						
RCL-1	MI	1981 - 1987	12	1.4	<1.0	6
RCU-1	MI	2004	1	<0.3	<0.3	<0.3
RCU-2	MI	2002 - 2005	8	0.48	<0.2	1.4

Golder Station ID[1]	Designation	Date Range	Number of Measurements	Mean Total Se (ug/L)	Min Total Se (ug/L)	Max Total Se (ug/L)
RCU-3	MI	1998 - 2000	5	0.44	<0.06	1
Smoky Creek, lower						
SMC-4	MI	2000 - 2005	12	0.53	<0.2	3
SMC-5	MI	1998	2	0.54	<0.16	1
SMC-6	MI	1979 - 2005	56	0.76	<0.2	10.98
SMC-7	MI	2000	2	<1.0	<1.0	<1.0
SMC-8	MI	Not reported	2	0.47	<0.5	0.698
Tygee Creek, lower						
TCL-1	MI	1979 - 1987	13	0.92	<1.0	3
TCL-2	MI	1979	1	<1.0	<1.0	<1.0
TCL-3	MI	2002	1	<1.0	<1.0	<1.0
TCL-4	MI	1986 - 2005	37	0.90	<0.2	4
TCL-5	MI	2000	4	0.62	<1.0	1
TCL-6	MI	2000	2	<1.0	<1.0	<1.0
TCL-7	MI	1999 - 2000	2	0.20	<0.18	<0.65

1. Station locations are shown on Figure 3; Golder assigned station identifications to consolidate data from multiple sources collected at or near similar locations.
BK = Background; MI = Mine Impacted.

TABLE 4: Summary of Selenium Concentrations Measured in Fish Tissue at Stations within the Salt River Watershed.

Golder Station ID[1]	Stream	Designation	Sampling Date	N	Mean (Range)			Reference
					Total Se (µg/g dw)	Fish Length (cm)	Fish Weight (g)	
Cutthroat Trout								
CC-100	Crow Creek, near mouth of Wells Canyon	BK	January 2006	6	5.9 (4.7 – 8.2)	22 (19.6 – 25.1)	101 (64–154)	Maxim (2006)
CC-3	Crow Creek, above Sage Creek	BK	July 2005	1	7.4	NM	NM	Weber (2005b)
CC-5	Crow Creek, 300 ft downstream of Deer Creek	BK	January 2006	9	5.34 (0.2 – 8.3)	18 (7.6 – 26)	66 (3 – 141)	Maxim (2006)
DC-1	Deer Creek	BK	May 2001	2	10.2 (9.3 – 11)	NM	NM	Hamilton & Buhl (2003b)
DC-100	Deer Creek, uppermost reach	BK	August 2003	1	0.76	24	170	Maxim (2004a)
DC-2	Deer Creek, 0.1 mile west of Riede's fence	BK	July 2005	1	18	NM	NM	Weber (2005b)
DC-200	Deer Creek, d/s of South Fork Sage Creek	BK	August 2003	3	0.44 (0.34 – 0.57)	17 (11.6 – 22)	65 (20 – 115)	Maxim (2004a)
DC-400	Deer Creek, d/s of North Fork Sage Creek	BK	August 2003	3	0.64 (0.48 – 0.8)	16 (12 – 23)	51.7 (15 – 120)	Maxim (2004a)
DC-400	Deer Creek, d/s of North Fork Sage Creek	BK	January 2006	12	5.6 (0.8 – 8.5)	13.2 (5.8 – 20)	27.2 (2 – 80)	Maxim (2006)
DC-600	Deer Creek, 1 mile u/s of Crow Creek	BK	January 2006	10	7.1 (2.1 – 9.7)	18 (14.5 – 20.5)	56 (25 – 82)	Maxim (2006)
DCNF-700	Deer Creek, North Fork, 300 ft u/s of mainstem Deer Creek	BK	August 2003	3	5.2 (3.6 – 7.1)	16 (11.3 – 24)	67 (15 – 170)	Maxim (2004a)
DCNF-700	Deer Creek, North Fork, 300 ft u/s of mainstem Deer Creek	BK	January 2006	5	4.7 (0.1 – 12.4)	14 (6.7 – 19.8)	35 (2 – 78)	Maxim (2006)
DCSF-100	Deer Creek, South Fork, u/s of Wells Canyon	BK	August 2003	3	2.3 (1.9 – 2.3)	13 (10.5 - 16.5)	29 (13 – 51)	Maxim (2004a)
SMC-3	Smoky Creek	BK	May 2001	2	4.2 (3.5 – 5)	NM	NM	Hamilton & Buhl (2003b)
USC-1	Sage Creek, upper, 140 yd u/s of road crossing	BK	July 2004	4	16.2 (3.13 – 5.7)	NM	52 (3.5–118)	NewFields (2005)
USC-2	Sage Creek, upper	BK	July 2001	1	4.6	NM	NM	TtEMI (2002a)

Golder Station ID[1]	Stream	Designation	Sampling Date	N	Mean (Range)			Reference
					Total Se (µg/g dw)	Fish Length (cm)	Fish Weight (g)	
USC-3	Sage Creek, 1.3 miles above NF boundary	BK	July 2004	4	3.9 (3.26 – 4.87)	NM	124 (22–185)	NewFields (2005)
SCSF-3	Sage Creek, South Fork, within mine lease modification area	BK	August 2003	3	2.4 (2.2 – 2.6)	16 (12.6 – 19.1)	57 (20 – 80)	Maxim (2004a)
LSC-2	Sage Creek, lower	MI	July 2001	1	11.8 (tissue type not specified)	NM	NM	TtEMI (2002a)
LSC-7	Sage Creek, at mouth of canyon at SCM haul road crossing and NF boundary	MI	July 2004	4	3.6 (3.2 – 4.2)	NM	38 (31– 53)	NewFields (2005)
Brown Trout								
CC	Crow Creek	BK	January 2006	1	9.7	NM	NM	Hamilton & Buhl (2003b)
CC-100	Crow Creek, near mouth of Wells Canyon	BK	August 2003	2	5.6 (4.6 – 6.7)	34 (32 – 37)	1000	Maxim (2004a)
CC-5	Crow Creek, 300 ft d/s of Deer Creek	BK	August 2003	1	5.4	31.5	360	Maxim (2004a)
SCSF-4	Sage Creek, South Fork, above Sage Creek	BK	September 1998	1	5.2	NM	NM	NewFields (2005)
SCSF-4	Sage Creek, South Fork, above Sage Creek	BK	July 2004	2	5.24 (5.05 – 5.43)	NM	144 (126–161)	NewFields (2005)
CC-4	Crow Creek, on Hartman Ranch	MI	July 2005	1	10.8	NM	NM	Weber (2005b)
HS	Hoopes Springs	MI	July 2004	3	21.8 (14.2 – 31.9)	NM	120 (47–234)	New Fields (2005)
LSC-8	Sage Creek, 150 ft d/s of Crow Creek Road	MI	July 2005	1	18.4	NM	NM	Weber (2005b)
LSC-2	Sage Creek, above Crow Creek	MI	July 2004	4	15.8 (13.5 – 19.2)	NM	347 (83–550)	NewFields (2005)
SMC-8	Smoky Creek, d/s of Smoky Canyon Mine	MI	July 2005	1	5.29	NM	NM	Weber (2005b)
Dace								
CC-3	Crow Creek, above Sage Creek	BK	July 2005	1	7.33	NM	NM	Weber (2005b)
CC-4	Crow Creek, on Hartman Ranch	BK	July 2005	1	21.2	NM	NM	Weber (2005b)

Golder Station ID[1]	Stream	Designation	Sampling Date	N	Mean (Range)			Reference
					Total Se (µg/g dw)	Fish Length (cm)	Fish Weight (g)	
Leatherside Chub								
TP-1	Tailings Pond 1	MI	June 2002	2	37.2 (30.4 – 44)	9.4 (11.5 – 12)	25.5 (24–27)	MFG (2003a)
Longnose Dace								
CC	Crow Creek	BK	May 2001	2	12.1 (10.8 – 13.4)	NM	NM	Hamilton & Buhl (2003b)
RCU-3	Roberts Creek, above Tailings Pond 1	MI	July 2004	1	4.86	NM	3.4	NewFields (2005)
TCL-6	Tygee Creek, below Smoky Creek	MI	July 2004	1	2.1	NM	27.7	NewFields (2005)
Mottled Sculpin								
CC	Crow Creek	BK	May 2001	1	8.2	NM	NM	Hamilton & Buhl (2003b)
DC-1	Deer Creek	BK	May 2001	1	12	NM	NM	Hamilton & Buhl (2003b)
Mountain Sucker								
TP-1	Tailings Pond 1	BK	August 2002	6	22 (23.6 – 36)	6.8 (6.5 – 7)	62 (54 – 89)	MFG (2003a)
Mountain Whitefish								
CC-5	Crow Creek, 300 ft d/s of Deer Creek	BK	August 2003	1	5.0	35.2	500	Maxim (2004a)
CC-4	Crow Creek, on Hartman Ranch	BK	July 2005	1	12.4	NM	NM	Weber (2005b)
Paiute Sculpin								
CC-100	Crow Creek, near Wells Canyon	BK	January 2006	11	6.3 (3.7 – 7.9)	9.9 (7.6 – 12.1)	14.6 (6 – 27)	Maxim (2006)
CC-5	Crow Creek, 300 ft d/s of Deer Creek	BK	January 2006	12	7.5 (4.3 – 10.9)	8.2 (6.1 – 10)	7.7 (4 – 16)	Maxim (2006)
DC-400	Deer Creek, d/s North Fork Deer Creek	BK	January 2006	5	8.9 (5.1 – 13.6)	10 (8.6 – 11.8)	15.4 (7 – 24)	Maxim (2006)
DC-600	Deer Creek, 1 mile u/s of Crow Creek	BK	January 2006	10	6.4 (0.5 – 9.1)	8.9 (7.7 – 11.1)	9.3 (5 – 19)	Maxim (2006)
Redside Shiner								
TP-1	Tailings Pond 1	MI	June 2002	1	27.6	10.5	11	MFG (2003a)

Golder Station ID[1]	Stream	Designation	Sampling Date	N	Mean (Range)			Reference
					Total Se (µg/g dw)	Fish Length (cm)	Fish Weight (g)	
Sculpin								
CC-100	Crow Creek, near Wells Canyon	BK	August 2003	3	5.0 (3.9 – 6.5)	7.5	5.3	Maxim (2004a)
CC-3	Crow Creek, above Sage Creek	BK	July 2005	1	9.23	NM	NM	Weber (2005b)
DC-2	Deer Creek, 0.1 miles west of Riede's fence	BK	July 2005	2	22 (11.6 – 32.3)	NM	NM	Weber (2005b)
DC-400	Deer Creek, d/s of North Fork Deer Creek	BK	August 2003	3	4.3 (0.7 – 5.8)	9.2 (8.5 – 10)	11.2 (10–13)	Maxim (2004a)
HS	Hoopes Springs	MI	July 2004	1	28.8	NM	NM	NewFields (2005)
LSC-8	Sage Creek, 150 ft d/s of Creek Creek Road	MI	July 2005	2	27.8 (20.7 – 34.9)	NM	NM	Weber (2005b)
LSC-2	Sage Creek, above Crow Creek	MI	July 2004	1	17.2	NM	NM	NewFields (2005)
SMC-8	Smoky Creek, d/s Smoky Canyon Mine	MI	July 2005	1	8.11 (6.44 – 9.77)	NM	NM	Weber (2005b)
TCL-6	Tygee Creek, below Smoky Creek	MI	July 2004	2	5.95 (7.6 – 8.12)	NM	NM	NewFields (2005)
Speckled Dace								
TP-1	Tailings Pond 1	MI	June 2002	1	39.2	7.2	10	MFG (2003a)
TP-2	Tailings Pond 2	MI	June 2002	4	26.6 (8.8 – 41.6)	6.9 (6.2 – 7.8)	7.6 (4.5 – 10)	MFG (2003a)

1. Station locations are shown on Figure 4; Golder assigned station identifications to consolidate data from multiple sources collected at or near similar locations.
BK = Background; MI = Mining Impacted; NM = Not Measured.

TABLE 5: Summary of Selenium Concentrations Measured in Sediments at Stations within the Salt River Watershed.

Golder Station ID[1]	Stream	Designation	Date Sampled	Total Se (µg/g (dry)	Reference Citation
CC	Crow Creek	BK	May 2001	2.1	Hamilton and Buhl (2003b)
CC-5	Crow Creek	BK	Aug 14, 2003	0.71	Maxim (2004b)
CC-5	Crow Creek, above Deer Creek	BK	Jun 14, 2001	1.2	TtEMI (2002a)
DC-1	Deer Creek	BK	May 2001	4.5	Hamilton and Buhl (2003b)
DC-1	Deer Creek (mouth)	BK	Sep 18, 2001	4.2	TtEMI (2002a,b)
DC-400	Deer Creek	BK	Aug 13, 2003	0.79	Maxim (2004b)
DC-500	Deer Creek	BK	Aug 12, 2003	1.3	Maxim (2004b)
DCNF-500	Deer Creek, North Fork	BK	Aug 13, 2003	0.5	Maxim (2004b)
DCNF-900	Deer Creek, North Fork	BK	Aug 13, 2003	1.1	Maxim (2004b)
DCSF	Deer Creek, South Fork	BK	Sep 1998	0.95	Montgomery Watson (1999)
DCSF-800	Deer Creek, South Fork	BK	Aug 13, 2003	0.76	Maxim (2004b)
USC-4	Sage Creek, above mining	BK	Jun 14, 2001	1	TtEMI (2002a)
USC-4	Sage Creek, above Smoky Canyon Mine	BK	Sep 1998	0.38	Montgomery Watson (1999)
USC-4	Sage Creek, upper	BK	Jul 23, 2004	0.78	NewFields (2005)
USC-2	Sage Creek, Upper	BK	Jul 24, 2001	1	TtEMI (2002a)
USC-1	Sage Creek, upper, 140 yards upstream from road crossing	BK	Jul 23, 2004	0.68	NewFields (2005)
SCNF-2	Sage Creek, North Fork, above Pole Creek	BK	Jul 22, 2004	0.37	NewFields (2005)
SCNF-2	Sage Creek, North Fork, upstream of Pole Canyon Creek	BK	Sep 15, 1999	0.8	Montgomery Watson (2001a)
SCSF-6	Sage Creek, upstream of permit boundary	BK	Sep 16, 1999	1	Montgomery Watson (2001a)
SCSF-2	Sage Creek, South Fork, below mining	BK	Jun 14, 2001	1.4	Tt EMI (2002a)
SCSF-4	Sage Creek, South Fork	BK	Aug 12, 2003	0.84	Maxim (2004b)
SCSF-4	Sage Creek, South Fork, 75 feet above road crossing, above confluence with Sage Creek	BK	Jul 21, 2004	1.2	NewFields (2005)
SCSF-4	Sage Creek, South Fork, at fish sampling reach	BK	Sep 1998	1.1	Montgomery Watson (1999)

Golder Station ID[1]	Stream	Designation	Date Sampled	Total Se (µg/g (dry)	Reference Citation
SCSF-4	Sage Creek, South Fork, downstream of permit boundary	BK	Sep 16, 1999	1.7	Montgomery Watson (2001a)
SCSF-5	Sage Creek, South Fork, 1.3 miles above National Forest boundary	BK	Jul 22, 2004	0.47	NewFields (2005)
SCSF-6	Sage Creek, South Fork, below Phosphoria Formation outcrop	BK	Sep 1998	1.2	Montgomery Watson (1999)
SMC-1	Smoky Creek, above activity at Smoky Canyon Mine	BK	Sep 1998	2.5	Montgomery Watson (1999)
SMC-2	Smoky Creek, above activity at Smoky Canyon Mine	BK	Jul 24, 2004	0.51	NewFields (2005)
SMC-1	Smoky Creek, above mining	BK	Jun 14, 2001	1.4	TtEMI (2002a, 2002b)
WC-800	Wells Canyon	BK	Aug 14, 2003	0.46	Maxim (2004b)
PC-3	Pole Creek, above Pole Canyon Waste Dump at FS Station S-B & M-8 (SW65)	BK	Jul 24, 2004	0.46	NewFields (2005)
HS	Hoopes Spring	MI	Jul 21, 2004	2.1	NewFields (2005)
MC-500	Manning Creek	MI	Aug 14, 2003	0.65	Maxim (2004b)
NC-100	Nate Canyon	MI	Aug 15, 2003	<0.4	Maxim (2004b)
PC-6	Pole Creek, lower, approximately ½ mile east of toe of waste rock dump	MI	Jul 22, 2004	58.1	NewFields (2005)
RCU-3	Roberts Creek, above Tailing Pond No. 1 and upstream of diversion	MI	Sep 1998	0.69	Montgomery Watson (1999)
RCU-3	Roberts Creek, above Tailing Pond No. 1 and upstream of diversion	MI	Jul 24, 2004	0.3	NewFields (2005)
LSC-2	Sage Creek, above Crow Creek just above bridge for main Crow Creek road	MI	Jul 20, 2004	3.3	NewFields (2005)
LSC-7	Sage Creek, at mouth of canyon at Smoky Canyon Mine haul road crossing and National Forest boundary	MI	Jul 22, 2004	1.8	NewFields (2005)
LSC-7	Sage Creek, below Smoky Canyon Mine	MI	Sep 1998	4.1	Montgomery Watson (1999)
LSC-7	Sage Creek, downstream of permit boundary	MI	Sep 16, 1999	6.2	Montgomery Watson (2001a)
LSC-5	Sage Creek, downstream of South Fork Sage	MI	Sep 17, 1999	1.8	Montgomery Watson (2001a)

Golder Station ID[1]	Stream	Designation	Date Sampled	Total Se (µg/g (dry)	Reference Citation
	Creek				
LSC-2	Sage Creek, Lower	MI	Jul 24, 2001	3.4	Tt EMI (2002a)
LSC-5	Sage Creek, mouth	MI	Jun 14, 2001	2.8	Tt EMI (2002a,b)
SCNF-1	Sage Creek, North Fork, below Pole Creek	MI	Sep 1998	4.1	Montgomery Watson (1999)
SCNF-1	Sage Creek, North Fork, below Pole Creek	MI	Sep 1998	0.48	Montgomery Watson (1999)
SCNF-1	Sage Creek, North Fork, downstream of Pole Canyon Creek	MI	Sep 15, 1999	3.9	Montgomery Watson (2001a)
SCPC	Sage Creek, below Pole Creek	MI	Jun 15, 2001	2.8	Tt EMI (2002a,b)
SMC-3	Smoky Creek	MI	May 2001	1.2	Hamilton and Buhl (2003b)
SMC-5	Smoky Creek, below Smoky Canyon Mine	MI	Sep 1998	1	Montgomery Watson (1999)
SMC-6	Smoky Creek, lower, at cattle guard/gate to Tailings Ponds	MI	Jul 25, 2004	1.8	NewFields (2005)
SMC-4	Smoky Creek, lower, spring located 600 feet u/s of beaver ponds in Smoky Canyon	MI	Jul 25, 2004	1.3	NewFields (2005)
TCL-6	Tygee Creek, below Smoky Creek	MI	Sep 17, 1999	1.1	Montgomery Watson (2001a)

1. Station locations are shown on Figure 7; Golder assigned station identifications to consolidate data from multiple sources collected at or near similar locations.
BK = Background; MI = Mine Impacted.

TABLE 6: Summary of Selenium Concentrations Measured in Benthic Invertebrates at Stations within the Salt River Watershed.

Golder Station ID[1]	Stream	Designation	Date Sampled	Total Se (µg/g)[2]	Reference Citation
CC	Crow Creek	BK	May 2001	6.7 dw	Hamilton and Buhl (2003b)
CC-3	Crow Creek, above Sage Creek apx 200 feet east of Crow Creek road	BK		6.79 dw	Weber (2005b)
DC-1	Deer Creek	BK	May 2001	8.7 dw	Hamilton and Buhl (2003b)
DC-2	Deer Creek, approx 0.10 miles west of fence line between Riede's and FS on FS land	BK		3.59 dw	Weber (2005b)
USC-2	Sage Creek, Upper	BK	Jul 24, 2001	0.29	TtEMI (2002a)
USC-4	Sage Creek, Upper	BK	Jul 23, 2004	0.6 ww	NewFields (2005)
USC-1	Sage Creek, upper, 140 yards upstream from road crossing	BK	Jul 23, 2004	0.63 ww	NewFields (2005)
SCNF-2	Sage Creek, North Fork, above Pole Creek	BK	Jul 22, 2004	1.09 ww	NewFields (2005)
SCSF-4	Sage Creek, South Fork, 75 feet above road crossing, above confluence with Sage Creek	BK	Jul 21, 2004	1.48 ww	NewFields (2005)
SCSF-5	Sage Creek, South Fork, 1.3 miles above National Forest boundary	BK	Jul 22, 2004	3.13 ww	NewFields (2005)
PC-3	Pole Creek, above Pole Canyon Waste Dump at FS Station S-B & M-8 (SW65)	BK	Jul 24, 2004	0.57 ww	NewFields (2005)
SMC-1	Smoky Creek, above activity at Smoky Canyon Mine	BK	Jul 24, 2004	0.64 ww	NewFields (2005)
SMC-3	Smoky Creek	BK	May 2001	4.1 dw	Hamilton and Buhl (2003b)
PC-6	Pole Creek, lower, approximately ½ mile east of toe of waste rock dump	MI	Jul 22, 2004	16.6 ww	NewFields (2005)
HS	Hoopes Spring	MI	Jul 21, 2004	4.51 ww	NewFields (2005)
LSC-2	Sage Creek, Lower	MI	Jul 24, 2001	1.2	TtEMI (2002a)
LSC-7	Sage Creek, at mouth of canyon at Smoky Canyon Mine haul road crossing and National Forest boundary	MI	Jul 22, 2004	0.57 ww	NewFields (2005)

Golder Station ID[1]	Stream	Designation	Date Sampled	Total Se (µg/g)[2]	Reference Citation
LSC-8	Sage Creek, approx 150 feet downstream and east of Crow Creek Road	MI		15 dw	Weber (2005b)
LSC-2	Sage Creek, above Crow Creek just above bridge for main Crow Creek road	MI	Jul 20, 2004	3.66 ww	NewFields (2005)
CC-4	Crow Creek, on Hartman Ranch just off entrance road from Crow Creek Road	MI	Not reported	9.47 dw	Weber (2005b)
SMC-4	Smoky Creek, lower, spring located 600 feet u/s of beaver ponds in Smoky Canyon	MI	Jul 25, 2004	0.52 ww	NewFields (2005)
SMC-6	Smoky Creek, lower, at cattle guard/gate to Tailings Ponds	MI	Jul 25, 2004	0.68 ww	NewFields (2005)
SMC-8	Smoky Creek, downstream of Smoky Canyon Mine approx 240 feet east of FS Road 110 (Smoky Canyon Road)	MI	Not reported	14.4 dw	Weber (2005b)
TCL-6	Tygee Creek, below Smoky Creek	MI	Jul 23, 2004	4.01 ww	NewFields (2005)
RCU-2	Tailings Pond 1	MI	Sep 24, 2002	2.9 ww	MFG (2003a)
TP-1	Tailings Pond 1	MI	Sep 24, 2002	2.8 ww	MFG (2003a)
TP-1	Tailings Pond 1	MI	Sep 24, 2002	4 ww	MFG (2003a)
TP-1	Tailings Pond 1	MI	Sep 24, 2002	3.1 ww	MFG (2003a)
TP-1	Tailings Pond 1	MI	Sep 24, 2002	3.7 ww	MFG (2003a)

1. Station locations are shown on Figure 8; Golder assigned station identifications to consolidate data from multiple sources collected at or near similar locations.
2. Tissue Se data reported as dry weight (dw); except NewFields (2005) reported data as wet weight (ww) and TtEMI (2002a) did not specify whether data were ww or dw.
BK = Background; MI = Mine Impacted.

TABLE 7: Summary of Selenium Concentrations Measured in Aquatic Vegetation at Stations within the Salt River Watershed.

Golder Station ID[1]	Stream	Designation	Date Sampled	Total Se (µg/g dry)[2]	Comments	Reference Citation
Aquatic Plants / Macrophytes						
CC	Crow Creek	BK	May 2001	4.6	Leaves of white-water buttercup (*Ranunculus longirostris)*	Hamilton and Buhl (2003b)
DC-1	Deer Creek	BK	May 2001	4.3	Leaves of white-water buttercup (*Ranunculus longirostris)*	Hamilton and Buhl (2003b)
SMC-3	Smoky Creek	BK	May 2001	2.5	Leaves of white-water buttercup (*Ranunculus longirostris)*	Hamilton and Buhl (2003b)
USC-2	Sage Creek, Upper	BK	Jul 24, 2001	0.53*		TtEMI (2002a)
CC-3	Crow Creek, above Sage Creek apx 200 feet east of Crow Creek road	BK		1.82		Weber (2005b)
DC-2	Deer Creek, approx 0.10 miles west of fence line between Riede's and FS on FS land	BK		3.6		Weber (2005b)
SMC-2	Smoky Creek, above activity at Smoky Canyon Mine	BK	Jul 24, 2004	1.54	bryophyte	NewFields (2005)
SMC-2	Smoky Creek, above activity at Smoky Canyon Mine	BK	Jul 24, 2004	0.45	Equisetum	NewFields (2005)
SMC-2	Smoky Creek, above activity at Smoky Canyon Mine	BK	Jul 24, 2004	1.37	bryophyte	NewFields (2005)
SMC-2	Smoky Creek, above activity at Smoky Canyon Mine	BK	Jul 24, 2004	no data	bullrush	NewFields (2005)
SCNF-2	Sage Creek, North Fork, above Pole Creek	BK	Jul 22, 2004	0.65	duckweed	NewFields (2005)
SCNF-2	Sage Creek, North Fork, above Pole Creek	BK	Jul 22, 2004	0.18	Juncas	NewFields (2005)
SCSF-4	Sage Creek, South Fork, 75 feet above road crossing, above confluence with Sage Creek	BK	Jul 21, 2004	4.95	bryophyte	NewFields (2005)

Golder Station ID[1]	Stream	Designation	Date Sampled	Total Se (µg/g dry)[2]	Comments	Reference Citation
SCSF-4	Sage Creek, South Fork, 75 feet above road crossing, above confluence with Sage Creek	BK	Jul 21, 2004	4.7	water cress	NewFields (2005)
SCSF-5	Sage Creek, South Fork, 1.3 miles above National Forest boundary	BK	Jul 22, 2004	0.83	bryophyte	NewFields (2005)
USC-4	Sage Creek, upper	BK	Jul 23, 2004	2.51	bryophyte	NewFields (2005)
USC-1	Sage Creek, upper, 140 yards upstream from road crossing	BK	Jul 23, 2004	2.11	bryophyte	NewFields (2005)
PC-3	Pole Creek, above Pole Canyon Waste Dump at FS Station S-B & M-8 (SW65)	BK	Jul 24, 2004	0.48	Equisetum	NewFields (2005)
PC-3	Pole Creek, above Pole Canyon Waste Dump at FS Station S-B & M-8 (SW65)	BK	Jul 24, 2004	1.64	Filamentous algae	NewFields (2005)
PC-3	Pole Creek, above Pole Canyon Waste Dump at FS Station S-B & M-8 (SW65)	BK	Jul 24, 2004	1.7	bryophyte	NewFields (2005)
CC-4	Crow Creek, on Hartman Ranch just off entrance road from Crow Creek Road	MI		2.32		Weber (2005b)
LSC-2	Sage Creek, Lower	MI	Jul 24, 2001	6.3*		TtEMI (2002a)
LSC-2	Sage Creek, Lower	MI	Jul 24, 2001	2.8*		TtEMI (2002a)
LSC-2	Sage Creek, Lower	MI	Jul 24, 2001	6.6*		TtEMI (2002a)
LSC-2	Sage Creek, Lower	MI	Jul 24, 2001	5.4*		TtEMI (2002a)
HS	Hoopes Spring	MI	Jul 21, 2004	17.3	water cress	NewFields (2005)
LSC-7	Sage Creek, at mouth of canyon at Smoky Canyon Mine haul road crossing and National Forest boundary	MI	Jul 22, 2004	0.88	milfoil	NewFields (2005)
LSC-8	Sage Creek, approx 150 feet downstream and east of Crow Creek Road	MI		3.85		Weber (2005b)
PC-6	Pole Creek, lower, approximately ½ mile east of toe of waste rock dump	MI	Jul 22, 2004	66.1	water cress	NewFields (2005)
PC-6	Pole Creek, lower, approximately ½ mile east of toe of waste rock dump	MI	Jul 22, 2004	87.7	bryophyte	NewFields (2005)
RCU-3	Roberts Creek, above Tailing Pond No. 1 and upstream of diversion	MI	Jul 24, 2004	2.3	bryophyte	NewFields (2005)
RCU-3	Roberts Creek, above Tailing Pond No. 1 and upstream of diversion	MI	Jul 24, 2004	0.09	bullrush	NewFields (2005)

Golder Station ID[1]	Stream	Designation	Date Sampled	Total Se (µg/g dry)[2]	Comments	Reference Citation
RCU-3	Roberts Creek, above Tailing Pond No. 1 and upstream of diversion	MI	Jul 24, 2004	0.16	Juncas	NewFields (2005)
LSC-2	Sage Creek, above Crow Creek just above bridge for main Crow Creek road	MI	Jul 20, 2004	7.67	bullrush	NewFields (2005)
LSC-2	Sage Creek, above Crow Creek just above bridge for main Crow Creek road	MI	Jul 20, 2004	6.89	water cress	NewFields (2005)
LSC-2	Sage Creek, above Crow Creek just above bridge for main Crow Creek road	MI	Jul 20, 2004	2.29	Milfoil	NewFields (2005)
LSC-2	Sage Creek, above Crow Creek just above bridge for main Crow Creek road	MI	Jul 20, 2004	5.52	unknown submergent vegetation	NewFields (2005)
SMC-4	Smoky Creek, lower, spring located 600 feet u/s of beaver ponds in Smoky Canyon	MI	Jul 25, 2004	0.78	Filamentous algae	NewFields (2005)
SMC-4	Smoky Creek, lower, spring located 600 feet u/s of beaver ponds in Smoky Canyon	MI	Jul 25, 2004	0.22	Juncas	NewFields (2005)
SMC-4	Smoky Creek, lower, spring located 600 feet u/s of beaver ponds in Smoky Canyon	MI	Jul 25, 2004	1.4	Milfoil	NewFields (2005)
SMC-6	Smoky Creek, lower, at cattle guard/gate to Tailings Ponds	MI	Jul 25, 2004	0.09	Juncas	NewFields (2005)
SMC-8	Smoky Creek, downstream of Smoky Canyon Mine approx 240 feet east of FS Road 110 (Smoky Canyon Road)	MI	x	0.79		Weber (2005b)
TCL-6	Tygee Creek, below Smoky Creek	MI	Jul 23, 2004	1.2	Milfoil	NewFields (2005)
TCL-6	Tygee Creek, below Smoky Creek	MI	Jul 23, 2004	0.42	Juncas	NewFields (2005)
TCL-6	Tygee Creek, below Smoky Creek	MI	Jul 23, 2004	1.17	Equisetum	NewFields (2005)
Periphyton						
SMC-1	Smoky Creek, above activity at Smoky Canyon Mine	BK	Jul 24, 2004	2		NewFields (2005)
USC-4	Sage Creek, upper	BK	Jul 23, 2004	1.84		NewFields (2005)
USC-1	Sage Creek, upper, 140 yards upstream from road crossing	BK	Jul 23, 2004	1.45		NewFields (2005)
PC-3	Pole Creek, above Pole Canyon Waste Dump at FS Station S-B & M-8 (SW65)	BK	Jul 24, 2004	3		NewFields (2005)
SCSF-4	Sage Creek, South Fork, 75 feet above road crossing, above confluence with Sage	BK	Jul 21, 2004	1.58		NewFields (2005)

Golder Station ID[1]	Stream	Designation	Date Sampled	Total Se (µg/g dry)[2]	Comments	Reference Citation
	Creek					
SCSF-5	Sage Creek, South Fork, 1.3 miles above National Forest boundary	BK	Jul 22, 2004	1.02		NewFields (2005)
HS	Hoopes Spring	MI	Jul 21, 2004	7.15		NewFields (2005)
LSC-7	Sage Creek, at mouth of canyon at Smoky Canyon Mine haul road crossing and National Forest boundary	MI	Jul 22, 2004	2.14		NewFields (2005)
PC-6	Pole Creek, lower, approximately ½ mile east of toe of waste rock dump	MI	Jul 22, 2004	69.1		NewFields (2005)
RCU-3	Roberts Creek, above Tailing Pond No. 1 and upstream of diversion	MI	Jul 24, 2004	1		NewFields (2005)
LSC-2	Sage Creek, above Crow Creek just above bridge for main Crow Creek road	MI	Jul 20, 2004	4		NewFields (2005)
SMC-4	Smoky Creek, lower, spring located 600 feet u/s of beaver ponds in Smoky Canyon	MI	Jul 25, 2004	2.1		NewFields (2005)
TCL-6	Tygee Creek, below Smoky Creek	MI	Jul 23, 2004	2.42		NewFields (2005)

1. Station locations are shown on Figure 9; Golder assigned station identifications to consolidate data from multiple sources collected at or near similar locations.
2. Data are reported as dry weight, except that samples marked with an asterisk (*) were not identified as being dry weight or wet weight in the original reference.
BK = Background; MI = Mine Impacted.

FIGURES

FIGURE 1: Acute Toxicity of Selenite to Cold-Water and Warm-Water Fish Species.

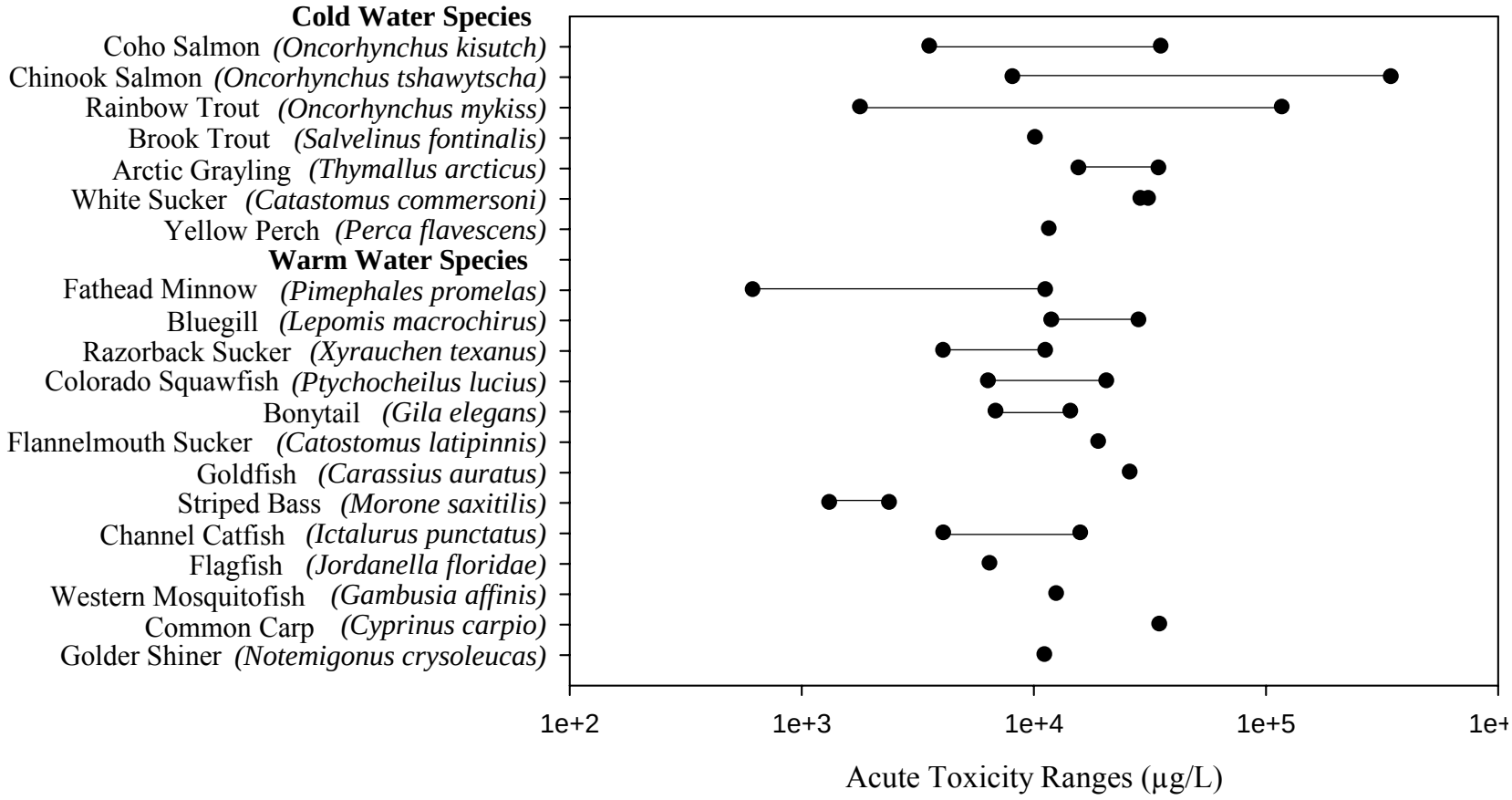
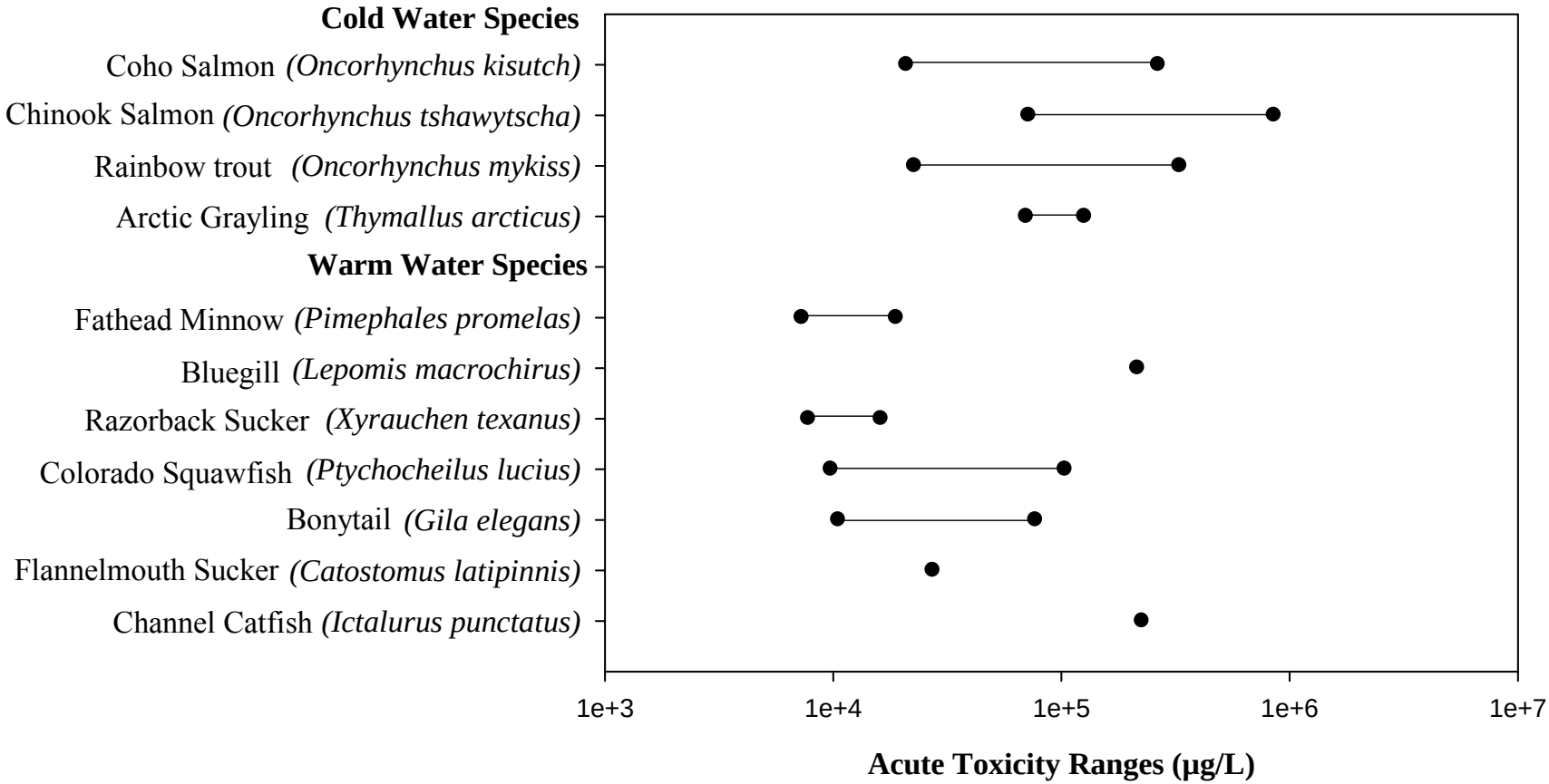
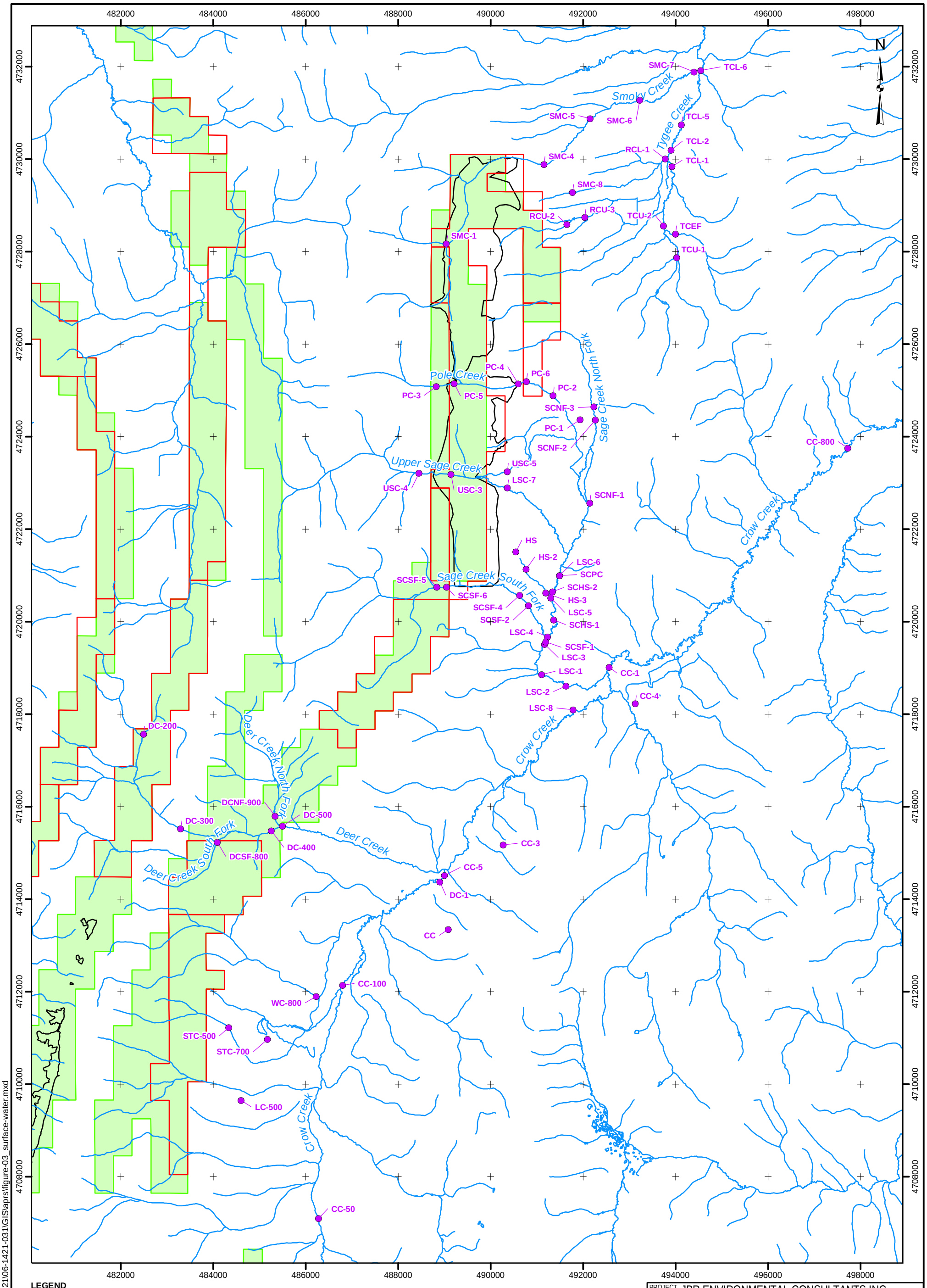


FIGURE 2: Acute Toxicity of Selenate to Cold-Water and Warm-Water Fish Species.





LEGEND

- Sampling Location - Surface Water
- Watercourse
- Mine
- Mine Lease
- Known Phosphorus

REFERENCE

Basedata provided by JBR Environmental Consultants Inc.
Datum: NAD 83 Projection: UTM Zone 12

1,500 750 0 1,500 Meters

Scale - 1:75,000

PROJECT JBR ENVIRONMENTAL CONSULTANTS INC.
ENVIRONMENTAL IMPACT STATEMENT
SMOKY CANYON MINE - PANELS F AND G

TITLE

SURFACE WATER SAMPLING LOCATIONS

Golder Associates
North Vancouver, B.C.

PROJECT No. 06-1421-031			SCALE AS SHOWN	REV. 0
DESIGN	CM	29 Jun. 2006	FIGURE 3	
GIS	CDB	29 Jun. 2006		
CHECK	CM	29 Jun. 2006		
REVIEW				

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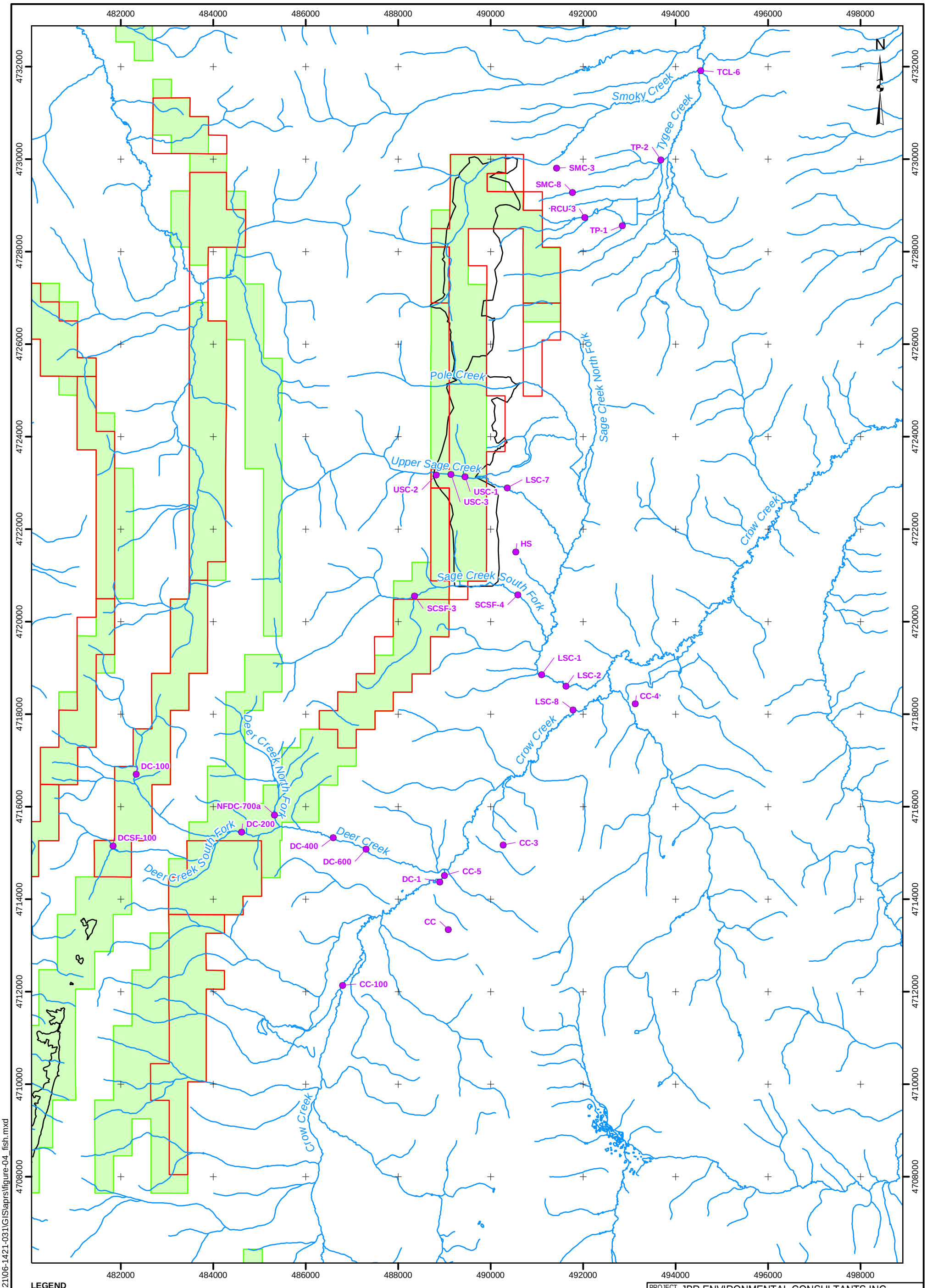


LEGEND

- Sampling Location - Fish
- Watercourse
- Mine
- Mine Lease
- Known Phosphorus

REFERENCE

Basedata provided by JBR Environmental Consultants Inc.
Datum: NAD 83 Projection: UTM Zone 12

1,500 750 0 1,500 Meters

Scale - 1:75,000

PROJECT JBR ENVIRONMENTAL CONSULTANTS INC.
ENVIRONMENTAL IMPACT STATEMENT
SMOKY CANYON MINE - PANELS F AND G

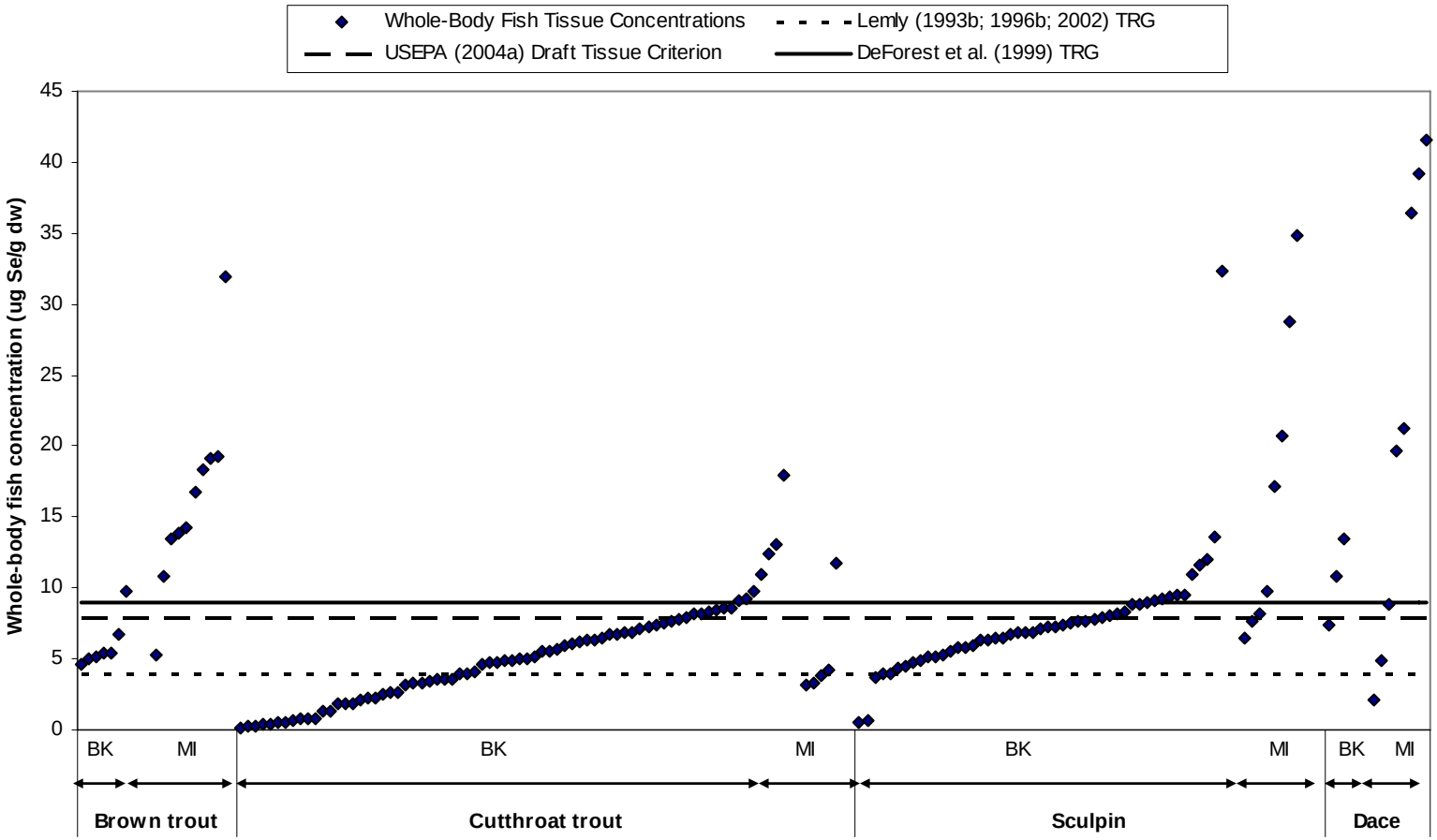
TITLE

FISH SAMPLING LOCATIONS

Golder Associates
North Vancouver, B.C.

PROJECT No. 06-1421-031			SCALE AS SHOWN	REV. 0
DESIGN	CM	29 Jun. 2006	FIGURE 4	
GIS	CDB	29 Jun. 2006		
CHECK	CM	29 Jun. 2006		
REVIEW				

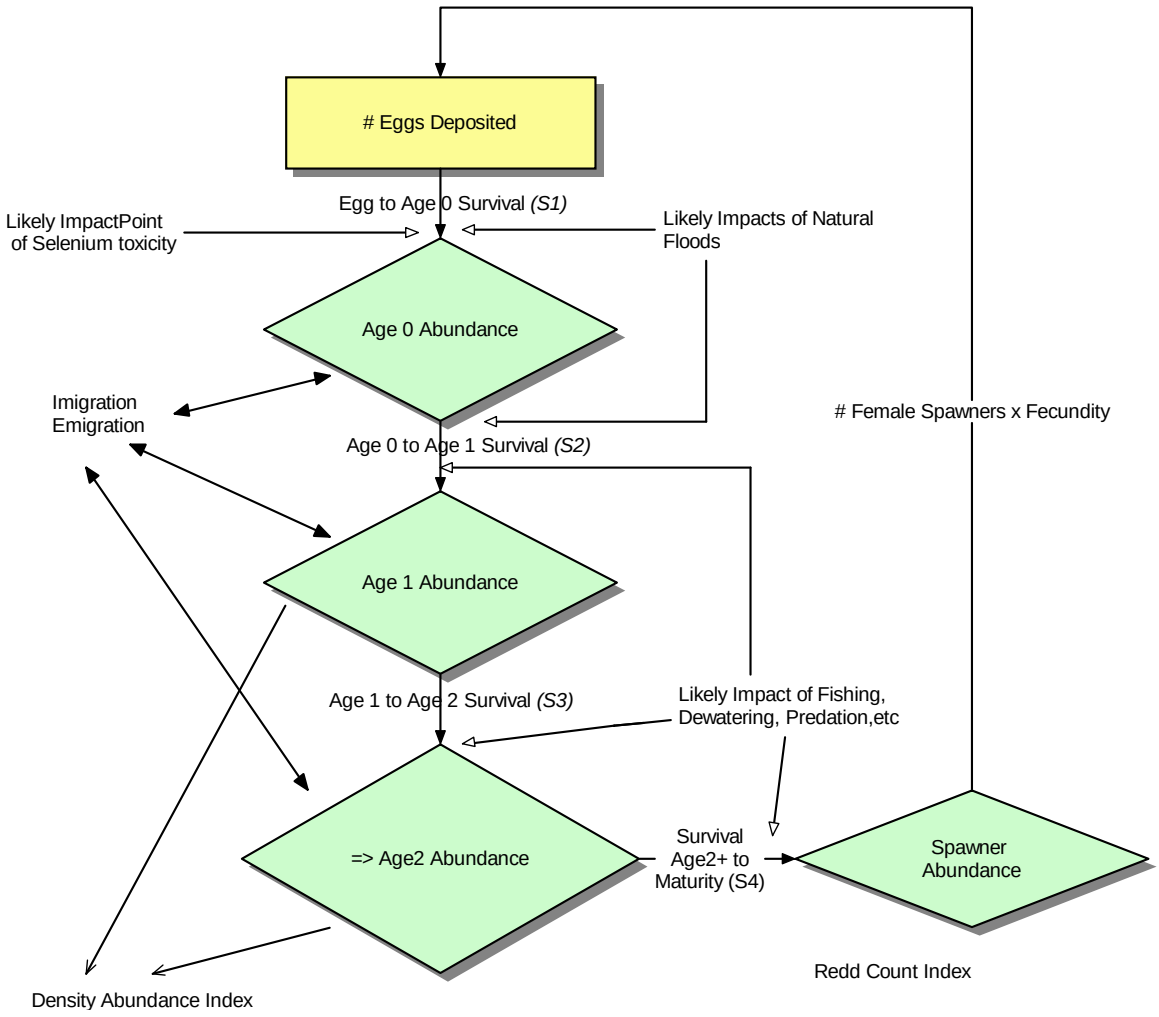
FIGURE 5: Summary of Available Whole-Body Fish Tissue Concentrations from Background (BK) and Mine-Impacted (MI) Locations Relative to USEPA Draft Tissue Criterion and Other Tissue Residue Guidelines from the Literature.

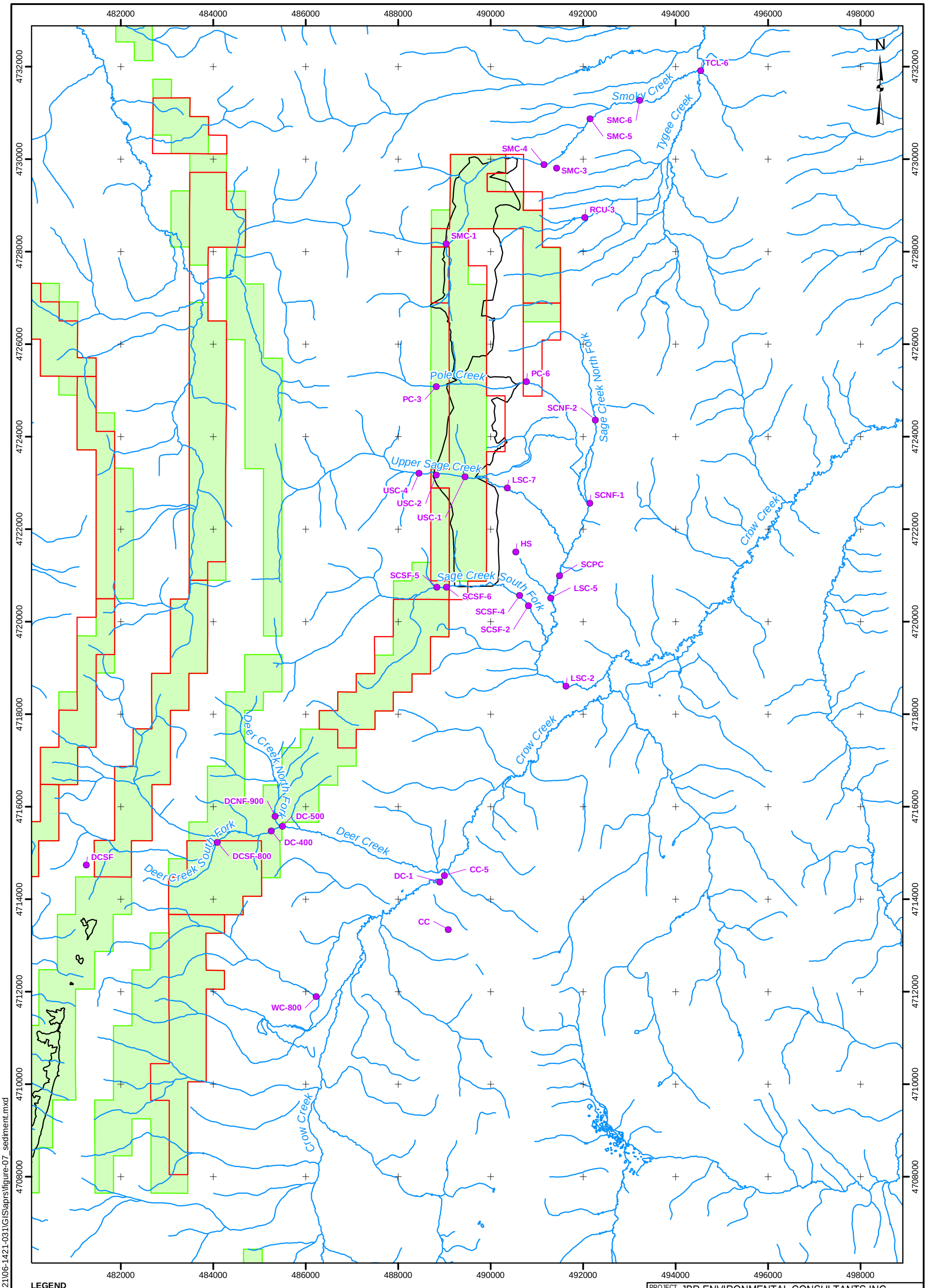


Notes: Data for mountain sucker (n = 5 for MI), redside shiner (n = 1 for MI), mountain whitefish (n = 2 for BK) and leatherside chub (n = 2 for MI) are not included due to lack of sufficient information to make comparisons between BK and MI areas. See Table 4 for these data.

All data are for whole-body fish concentrations except one BK cutthroat trout sample (4.6 µg/g) and one MI cutthroat trout sample (11.8 µg/g) listed as "fish tissue, unspecified" (also not specified as dry or wet weight) and one MI brown trout sample (5.2 µg/g dw) listed as "fish tissue, muscle" in the original data sources.

FIGURE 6: Potential Impacts of Selenium and Other Stressors on the Life History Stages of a Typical Salmonid, Survival and Migration Strategies That May Impact Abundance Estimates Over a Time Series.





LEGEND

- Sampling Location - Sediment
- Watercourse
- Mine
- Mine Lease
- Known Phosphorus

REFERENCE

Basedata provided by JBR Environmental Consultants Inc.
Datum: NAD 83 Projection: UTM Zone 12

1,500 750 0 1,500 Meters

Scale - 1:75,000

PROJECT JBR ENVIRONMENTAL CONSULTANTS INC.
ENVIRONMENTAL IMPACT STATEMENT
SMOKY CANYON MINE - PANELS F AND G

TITLE

SEDIMENT SAMPLING LOCATIONS

Golder Associates
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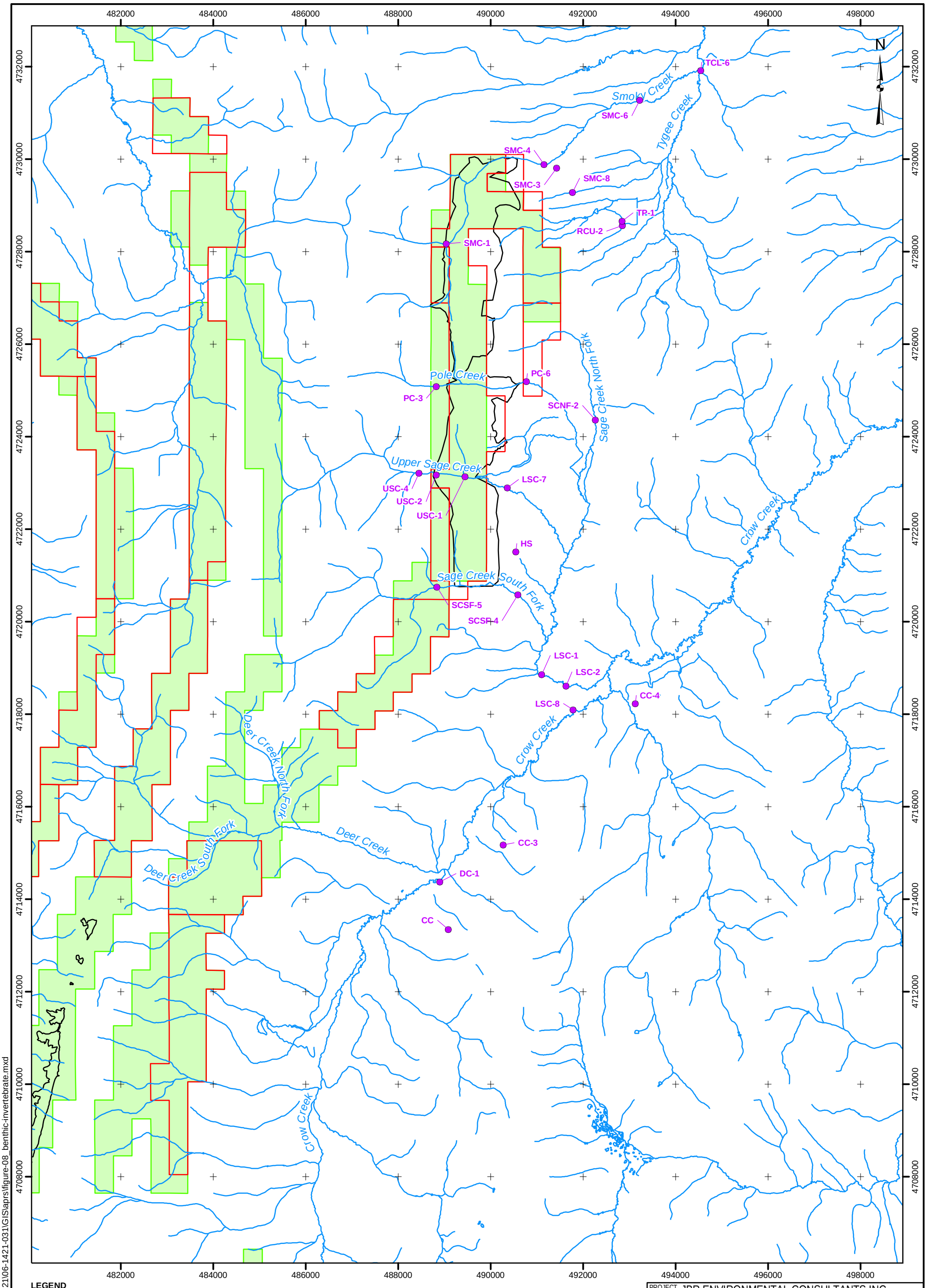
PROJECT No. 06-1421-031			SCALE AS SHOWN	REV. 0
DESIGN	CM	29 Jun. 2006	FIGURE 7	
GIS	CDB	29 Jun. 2006		
CHECK	CM	29 Jun. 2006		
REVIEW				


482000
484000
486000
488000
490000
492000
494000
496000
498000
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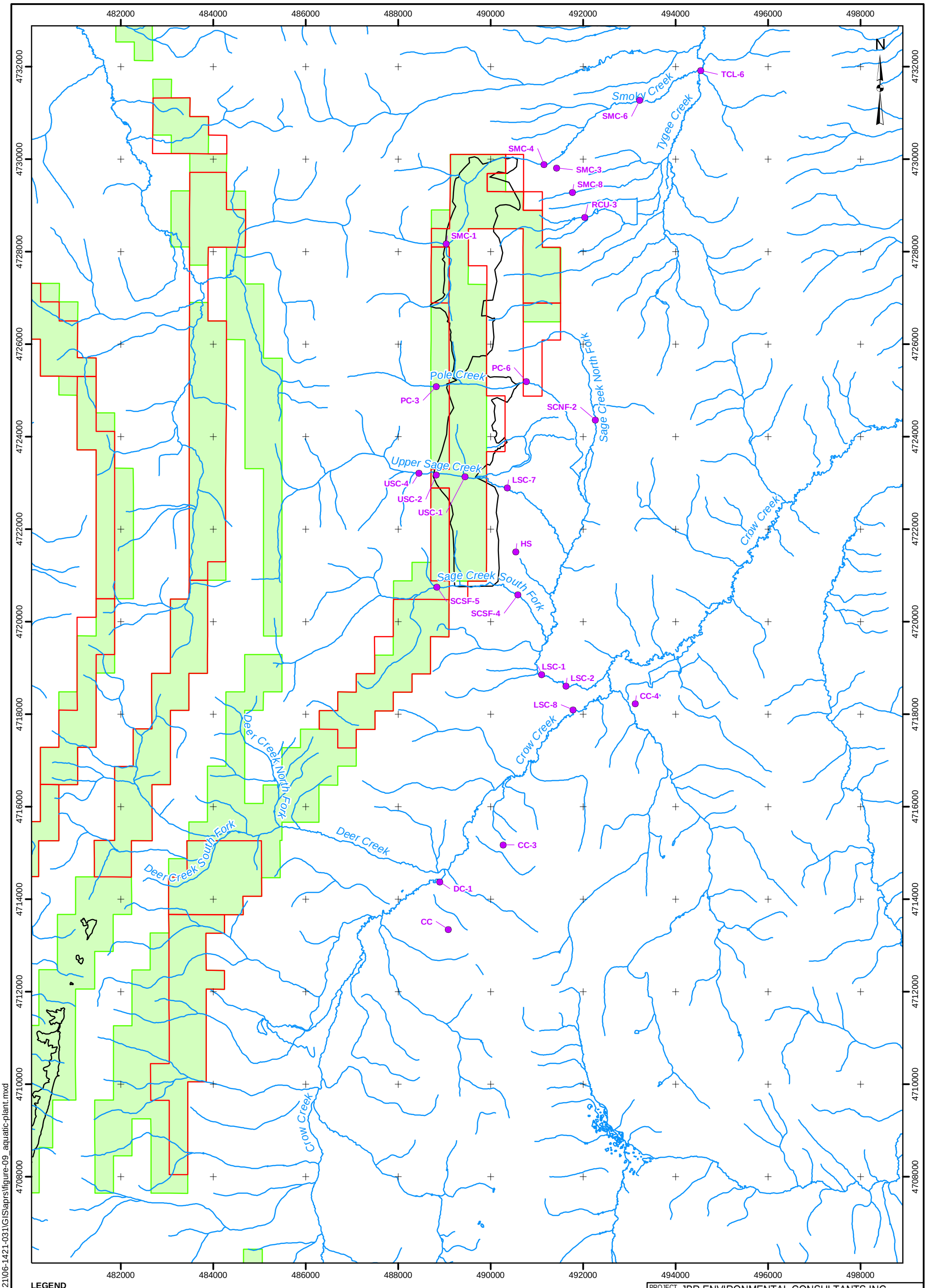
4728000
4726000
4724000
4722000
4720000
4718000
4716000
4714000
4712000
4710000
4708000
N
TCL-6
Smoky Creek
SMC-6
Tygee Creek
SMC-4
SMC-3
SMC-8
TP-1
RCU-2
SMC-1
PC-6
Pole Creek
PC-3
SCNF-2
Sage Creek North Fork
Upper Sage Creek
USC-4
USC-2
USC-1
LSC-7
Crow Creek
HS
Sage Creek South Fork
SCSF-5
SCSF-4
LSC-1
LSC-2
CC-4
LSC-8
Deer Creek North Fork
Deer Creek South Fork
Deer Creek
CC-3
DC-1
CC
Crow Creek
LEGEND
Sampling Location - Benthic Invertebrate
Watercourse
Mine
Mine Lease
Known Phosphorus
1,500 750 0 1,500
Meters
Scale - 1:75,000
REFERENCE
Basedata provided by JBR Environmental Consultants Inc.
Datum: NAD 83 Projection: UTM Zone 12
PROJECT
JBR ENVIRONMENTAL CONSULTANTS INC.
ENVIRONMENTAL IMPACT STATEMENT
SMOKY CANYON MINE - PANELS F AND G
TITLE
BENTHIC INVERTEBRATE
SAMPLING LOCATIONS
Golder Associates
North Vancouver, B.C.
PROJECT No. 06-1421-031
SCALE AS SHOWN
REV. 0
DESIGN CM 29 Jun. 2006
GIS CDB 29 Jun. 2006
CHECK CM 29 Jun. 2006
REVIEW
FIGURE 8
N:\Bur-Graphics\Projects\2006\1421\06-1421-031\GIS\aprs\figure-08_benthic-invertebrate.mxd



LEGEND

- Sampling Location - Aquatic Plant (macrophyte/periphyton)
- Watercourse
- Mine
- Mine Lease
- Known Phosphorus

1,500 750 0 1,500 Meters

Scale - 1:75,000

REFERENCE

Basedata provided by JBR Environmental Consultants Inc.

Datum: NAD 83 Projection: UTM Zone 12

PROJECT: JBR ENVIRONMENTAL CONSULTANTS INC.
ENVIRONMENTAL IMPACT STATEMENT
SMOKY CANYON MINE - PANELS F AND G

TITLE: AQUATIC PLANT (MACROPHYTE/PERIPHYTON) SAMPLING LOCATIONS

Golder Associates
North Vancouver, B.C.

PROJECT No. 06-1421-031			SCALE AS SHOWN	REV. 0
DESIGN	CM	29 Jun. 2006	FIGURE 9	
GIS	CDB	29 Jun. 2006		
CHECK	CM	29 Jun. 2006		
REVIEW				

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