Ministry of Environment
Province of British Columbia

Ambient Water Quality Guidelines for Selenium

Technical Report

Update

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Glossary

Alert Concentration – is a concentration below the guideline but above which there may be a risk to some environments and/or species that are particularly sensitive to Se bioaccumulation. If *alert concentrations* are exceeded, a series of actions may be triggered to evaluate whether impacts may be occurring and if necessary, mitigate the effects of Se.

Bioaccumulation – general term describing a process by which chemical substances are accumulated by aquatic organisms from water directly or through consumption of fine suspended particles, sediment and food containing the chemicals (CCME 1999). Bioaccumulation factors (BAFs) are typically expressed as the ratio of the concentration of a chemical in an organism (or tissue) to the concentration of that chemical in diet or from sediment in which the organism resides (sometimes called a biota-sediment accumulation factor (BSAF).

Bioconcentration – a process by which there is a net accumulation of a chemical directly from water alone into aquatic organisms resulting from simultaneous uptake (e.g., by gill or epithelial tissue) and elimination (CCME 1999). Bioconcentration factors (BCFs) are expressed as the ratio of a chemical in an organism (or tissue) to the concentration of that chemical in water. Bioconcentration is sometimes used interchangeably with bioaccumulation where water is the medium being compared to tissue.

Biofilm – refers to the layer of microscopic organisms (yeasts, bacteria, and algae) as well as organic and inorganic particles, typically found living on rocks or sediment surfaces. Biofilm is a term used in biology and engineering fields, and has become synonymous with periphyton. These microbiota assemblages can be further differentiated by the strata on which they live (rocks, mud, or sand) (Wetzel 2001).

Biogenic – produced by living organisms or biological processes.

Biomagnification – result of the processes of bioconcentration and bioaccumulation by which tissue concentrations of bioaccumulated chemicals increase as the chemical passes up through two or more trophic levels. The term implies an efficient transfer of chemicals from food to consumer so that residue concentrations increase systematically from one trophic level to the next (CCME 1999).

EC50 – a concentration of a pollutant or effluent at which 50 percent of the test organisms display non-lethal effects.

ECX (or ICX) – A concentration of a pollutant or effluent at which x percentage of the test organisms display non-lethal effects such as growth or reproduction (or inhibition) after a specific exposure period.

Epilithon – epilithic periphyton or biofilm (the aggregate of attached algae, bacteria, yeast, and fine particles) found on the surface of rocks or stone sediments (lotic environments) (Wetzel 2001).

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Epipelon – epipelic periphyton or biofilm (the aggregate of algae, bacteria, yeast, and fine particles) found on the surface of bottom sediments made up of finer organic matter (lentic environments) (Wetzel 2001).

Euphotic zone –refers to the depth of water to which light penetrates sufficiently for photosynthesis to occur (also called the photic zone).

Genus mean acute values (GMAVs) – US EPA's method for estimating a mean acute (LC50) value for a genus used in deriving water quality criteria.

Genus mean chronic values (GMCVs) – US EPA's method for estimating a mean chronic value for a genus used in deriving water quality criteria.

Herps/herptiles/herpetofauna – a term sometimes used for the classes of organisms that fall under the general heading of amphibians and reptiles.

LC50 – a concentration of a pollutant or effluent at which 50 percent of the test organisms die, commonly used to measure acute toxicity.

LCx – a concentration of a pollutant or effluent at which x percentage of the test organisms display lethal effects after a specific exposure period.

Lentic – refers to waters that have relatively slow flushing rates (still water), such as lakes, ponds, and wetlands.

Lowest observable effect concentration (LOEC) – the lowest tested concentration of a substance that has been reported to cause harmful (adverse) effects on organisms tested.

Lotic – refers to flowing water that has high flushing rates, such as creeks, streams, and rivers.

Maximum acceptable toxicant concentration (MATC) – the MATC is the geometric mean of the NOEC and the LOEC for a chronic level exposure.

No observable effect concentration (NOEC) – the highest tested concentration of a substance that has been reported to have no harmful (adverse) effects on organisms tested.

Thiols – a class of sulphur-containing compounds having the general formula RSH, also called mercaptan.

Uncertainty factor (safety factor, application factor) – a mathematical adjustment made to guideline values to account for incomplete knowledge.

Water Quality Guideline (WQG) – A maximum and/or minimum value for a physical, chemical or biological characteristic of water, sediment or biota, applicable in British Columbia, which should not be exceeded to prevent detrimental effects from occurring to a water use. WQGs may be derived for the protection of six designated water uses, including:

- drinking, public water supply, and food processing;
- aquatic life;

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- wildlife;
- agricultural (irrigation and livestock watering);
- recreation and aesthetics; and,
- industrial water supplies.

Water Quality Objective (WQO) – A WQO is best defined as a site-specific WQG. A water quality objective may be derived in situations where the natural background concentrations of a given variable exceed the BC-approved WQG, where there are ameliorating circumstances affecting the toxicity of the contaminant in question, or the data used to derive the BC approved WQG includes taxonomic groupings not present at the site in question.

Wet Weight – Analysis of tissue selenium (or other compounds) may be expressed as wet weight or dry weight. Tissue analyses are reported on a wet-weight or "as-is" basis following acid digestion of the sample by inductively coupled plasma spectrometry / mass spectrometry (ICP/MS) or cold vapor atomic absorption (CVAA) techniques. Wet Weight data may be converted to dry weight with a correct factor for moisture content, directly from moisture analysis or by estimating tissue moisture from literature values.

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1.0 Executive Summary

Selenium is a relatively rare trace element, but may be elevated in areas with soils that originate from marine sedimentary deposits. The movement of Se from land to water occurs from both natural processes (e.g., erosion) and human activities (e.g., mining) either indirectly through overland runoff or directly from industrial discharges. In the aquatic environment, Se accumulates in sediments and biota, and can continue to cycle and persist for many years.

Selenium is an essential trace element necessary for cellular function in many organisms; however excessive amounts may result in toxic effects. Selenium toxicity in fish results in many adverse effects including: reductions in growth; behavioural changes; increased deformity; and increased mortality in early life stages. For birds that feed in aquatic environments, the most sensitive toxicity endpoint is reduced egg hatchability followed by deformity in offspring. As is often the case in Se toxicity, the adult organism may appear unaffected; however, overall reproductive success and productivity may be negatively impacted.

In humans, Se deficiency and toxicity is rare in North America because food is generally obtained from different geographic areas and food choices are plentiful. Health Canada developed dietary reference intakes recognizing the margins between desirable and undesirable intakes and associated health benefits and adverse affects. The tolerable upper level intake is based on observations of selenosis in China, a condition characterized by symptoms such as hair loss, skin lesions, tooth decay, and abnormalities of the nervous system.

The British Columbia Ministry of Environment (MoE) develops ambient water quality guidelines (WQGs) to assess and manage the health, safety and sustainability of BC's aquatic resources. Guidelines are developed to protect: aquatic life, wildlife, agriculture (irrigation and livestock watering), drinking water sources, and recreation and aesthetics.

In BC, the development of WQGs for aquatic life is directed by the following guiding principles:

- WQGs are science-based and intended for generic provincial application;
- WQGs do not account for site-specific conditions or socio-economic factors;
- all components of the aquatic ecosystem (e.g., algae, macrophytes, invertebrates, amphibians, and fish) are considered when data are available;
- where data are available but limited, interim WQGs may be developed; and,
- all forms of aquatic life and all aquatic stages of their life cycle are to be protected during indefinite exposures.

Guidelines intended for the protection of human health are determined through consultation with the BC Ministry of Health (MoH).

This technical report provides numerical ambient water quality guidelines for selenium (Se). Updated ambient water quality guidelines are provided for aquatic life, wildlife, drinking water sources, and health-based tissue guidelines (see Table 1). Guidelines for irrigation water and livestock watering published by Nagpal and Howell (2001) are unchanged and remain Ministry policy.

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Health Canada's drinking water guideline for Se has been established to prevent toxic effects at excessive levels and was adopted by the MoE for use as an ambient source water quality guideline to reduce adverse risks to drinking water sources, and therefore indirectly to human health. A risk-based approach was used to develop health-based tissue guidelines for Se to protect the human consumer.

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Table 1.1 List of updated and previous WQGs for selenium recommended for use in British Columbia. Water concentrations are measured as total selenium. Details on guideline derivation may be found in Section 8.

Water Use	Updated 2012 BC Se WQG	2001 Approved BC Se WQG	Guideline Derivation Method/Approach
Source Drinking Water	10 µg/L	10 µg/L	*Source Drinking Water:* Adopted from Health Canada; a maximum acceptable concentration of 10 µg/L to protect against adverse effects in humans from excessive exposure.
Human Consumption Screening Values			
High fish intake (0.22 kg/day)	1.8 µg/g (ww), 7.3 (dw)[1]	None proposed	*Tissue Consumption*: Values were derived using Health Canada's recommended equation for ingestion of Se-contaminated fish and the dietary reference value's tolerable upper intake.
Moderate fish intake (0.11 kg/day)	3.6 µg/g (ww), 14.5 (dw)	None proposed	
Low fish intake (0.03 kg/day)	18.7 µg/g (ww), 75.0 (dw)	None proposed	
Aquatic Life			*Water column*: Review of previous WQG (uncertainty factor (UF) applied to toxicity threshold); weight of evidence including food web modelling and reported relationships between impacts and Se concentrations in water.
Water column freshwater & marine			
Alert concentration	1 µg/L	None proposed	
Guideline	2 µg/L	2 µg/L	
Sediment - Alert concentration	2 µg/g (dw)	None proposed	*Sediment*: Weight of evidence; lowest published toxicity thresholds, no UF applied; insufficient data for full guidelines at this time.
Dietary			*Dietary:* Weight of evidence; lowest published toxicity thresholds, no UF applied; insufficient data for full guidelines at this time. Invertebrate tissue as surrogate for aquatic dietary tissue.
Invertebrate tissue (interim)	4 µg/g (dw)	2 µg/g (dw)	
Tissue (fish)			
Egg/ovary	11 µg/g (dw)	None proposed	*Egg/ovary:* Combination weight of evidence and mean of published effects data with an UF of 2 applied; *Whole-body*: previous WB guideline compared with published literature, mean of published effects data with UF (2) applied and weight of evidence; *Muscle*: WB translation to derive muscle WQG, no additional UF applied to muscle guideline.
Whole-body (WB)	4 µg/g (dw)	4 µg/g (dw)	
Muscle/muscle plug (interim)	4 µg/g (dw)	None proposed	
Wildlife			The *water column* guideline for aquatic life (fish) is adopted for wildlife since dietary accumulation is most critical. *Bird eggs* were used as surrogate for all wildlife; weight of evidence; egg Se most direct/sensitive measure; mallard EC10 with UF of 2 applied.
Water column	2 µg/L	4 µg/L (maximum)	
Bird egg	6 µg/g (dw)	7 µg/g (dw)	
Recreation and Aesthetics	None proposed	None proposed	No data
Irrigation Water			Not updated at this time
2001 guideline not updated	10 µg/L	10 µg/L	
Livestock Watering			Not updated at this time
2001 guideline not updated	30 µg/L	30 µg/L	
Industrial Water	None proposed	None proposed	No data

[1] Guideline based on edible portions of tissue. Wet weight to dry weight conversion based on 75% moisture content.

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2.0 Introduction

In 2001, the BC MoE published water quality guidelines for selenium (Se), which included numerical concentrations for the protection of drinking water, aquatic life, wildlife, irrigation and livestock watering uses (Nagpal and Howell 2001). Since that publication was released, many studies have been conducted evaluating the effects of Se on human health and ecological receptors, some of which are specific to BC. Because Se is a bioaccumulative substance, derivation of guidelines must account for the accumulation from water, suspended particles, sediment and diet, to the tissues of exposed aquatic organisms, their progeny, and/or their consumers. This document provides a brief summary of the physical and chemical properties, various sources, background concentrations, fate and persistence of Se in the environment. It also summarises the bioaccumulation and toxicity literature used to update the ambient water quality guidelines (Table 1.1).

2.1 BC Approved Ambient Water Quality Guidelines

The BC Ministry of Environment (MoE) develops province-wide ambient water quality guidelines for substances or physical attributes that are important for managing both fresh and marine surface waters. This work has the following goals:

- provide protection of the most sensitive aquatic life form and most sensitive life stage indefinitely;
- provide a basis for the evaluation of data on water, sediment, and biota for water quality and environmental impact assessments;
- provide a basis for the establishment of site-specific ambient water quality objectives
- help to identify areas with degraded conditions that need remediation;
- provide a basis for establishing wastewater discharge limits; and,
- report to the public on the state of water quality and promote water stewardship.

Ambient water quality guidelines are developed for the following water uses:

- aquatic life and wildlife,
- agriculture (irrigation and livestock watering),

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- drinking water sources, and
- recreation and aesthetics.

A water quality guideline in BC is defined as:

"A maximum and/or minimum value for a physical, chemical or biological characteristic of water, sediment or biota, applicable province-wide, which should not be exceeded to prevent specified detrimental effects from occurring to a water use, including aquatic life, under specified environmental conditions." (BC Ministry of Environment Policy 6.10.03.02, signed August 1991).

The following principles guide the development of water quality guidelines for aquatic life in BC (BC MoE 2012). BC's aquatic water quality guidelines (WQGs) are science-based, intended for generic provincial application; they do not account for site-specific conditions or socio-economic factors. All components of the aquatic ecosystem (e.g., algae, macrophytes, invertebrates, amphibians, and fish) are considered if the data are available. Where data are available but limited, interim guidelines may be developed. All forms of aquatic life and all aquatic stages of their life cycle are to be protected during indefinite exposure.

The BC MoE, outlines specific data requirements for derivation of an aquatic life guideline (BC MoE 2012a). It is essential that, at a minimum, data for fish, invertebrates, and plants be included in the guidelines derivation process. Data for amphibians are also highly desirable but often limited or simply not available. Guidelines or interim guidelines may also include studies involving species not required in the minimum data set (e.g., protozoa, bacteria) when reasonable justification exists.

It should be noted that there are several sources of uncertainty when it comes to developing water quality guidelines and therefore it is necessary to apply uncertainty factors. Sources of uncertainty include:

- laboratory to field differences;
- single to multiple contaminants (additive, synergistic, antagonistic effects);

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- toxicity of metabolites;
- intra- and inter-species differences (limited species to conduct tests on, which may not include the most sensitive species);
- indirect effects (e.g., foodweb dynamics);
- whole life-cycle vs. partial life-cycle (many toxicity studies are only conducted on partial life-cycles and it can be difficult to determine the most sensitive life stage);
- delayed effects;
- impacts of climate change (species may be more vulnerable with additional stressors); and
- other stressors including cumulative effects.

The appropriate uncertainty factor is determined on a case-by-case basis, according to the evaluation of data quality and quantity, toxicity of the contaminant, severity of toxic effects, and bioaccumulation potential (BC MoE 2012). Scientific judgement is important in maintaining some flexibility in the derivation process.

Presently, water quality guidelines do not have any direct legal standing. They are intended as a tool to provide the scientific basis and policy direction for decisions affecting water quality. Water quality guidelines can be used to establish the allowable limits in waste discharges. These limits are set out in waste management permits, approvals, plans, or operating certificates which do have legal standing.

2.2 Derivation of Ambient Water Quality Guidelines for Selenium

2.2.1 Human Health Guidelines for Selenium

Guidelines intended for the protection of human health are determined through consultation with the BC Ministry of Health. Health Canada's Guidelines for Canadian Drinking Water Quality for chemical parameters are typically adopted by the BC Ministry of Environment for use as ambient water quality guidelines to reduce adverse risks to drinking water sources, and therefore indirectly to human health.

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For substances that accumulate in aquatic food sources such as fish and wildlife, health-based tissue guidelines may be developed, again in consultation with the Ministry of Health. It is important to note that for the purposes of this document, health-based guidelines do not provide advice regarding consumption limits or advisories. Rather, if health-based guideline levels are exceeded, it may indicate that further assessments or investigations are required to evaluate possible risks to human health. Decisions regarding health investigations, fish consumption limits or consumption advisories are under the purview of the regional Health Authorities and the Ministry of Health.

A risk-based approach was used to develop health-based tissue guidelines for Se; in this document, these are referred to as *screening values*. This document also provides a brief background of selenium pharmacokinetics in humans, essentiality and toxicity, possible exposure routes, background levels in the Canadian population, and recommended health-based guidelines such as dietary reference intakes.

It is beyond the scope and intention of this document to assess the derivation of Health Canada's health-based guidelines such as drinking water guidelines or dietary reference intake values.

2.2.2 Guidelines for the Protection of Aquatic Life

In aquatic environments Se has proven to be a unique element with a complex mode of toxicity related to its uptake and bioaccumulation primarily through the food chain. In developing the guidelines, this complexity and the current state of scientific understanding regarding Se, was conducive to development of a series of values as *guidelines* and *interim guidelines* specific to a particular media that should not be exceeded. *Alert concentrations* for water and sediment have also been incorporated for environmental managers. Although aquatic guidelines represent safe concentration of Se for most ecosystems, in some environments, like wetlands, ponds and lakes, Se can bioaccumulate to very high levels in aquatic life at water concentrations lower than the guideline. Hence, an *alert concentration* was added to the suite of guidelines to address those situations where environments and/or species sensitive to Se bioaccumulation, may be at risk when Se levels are below the guideline. If *alert concentrations* are exceeded, a series of actions may be triggered to evaluate and if necessary, mitigate the effects of Se. In the case of sediment,

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there was insufficient primary or secondary scientific literature to support a full or interim guideline. However, since sediment is an important sink for Se and elevated concentrations could indicate significant risks of bioaccumulation, an *alert concentration* for sediment was established.

Aquatic life guidelines have incorporated values for several water uses and environmental media providing practitioners and resource managers with greater flexibility in detecting and assessing the potential effects and the risks associated with Se in the environment. In some locations, background Se concentrations in water or sediment may be slightly higher, or some species may have naturally higher Se levels in tissues than generic guidelines. If this is the case, site-specific water quality objectives may need to be considered for these aquatic environments (BC MoE 2013). For more information and guidance on how, and under what circumstances, to develop water quality objectives, please contact the BC Ministry of Environment and access the on-line guidance document on the web: http://www.env.gov.bc.ca/wat/wq/pdf/wqo_2013.pdf.

2.3 History

Selenium (Se) was discovered in 1817 by a Swedish chemist, Jöns Jakob Berzelius, who identified it as an impurity resulting from the production of sulphuric acid (Ihnat and Wolf 1989). Selenium is a relatively rare trace element, ranking 68th among the elements in the Earth's crust (Adriano 2001). Although it is distributed widely, soil concentrations are inconsistent, and depend on the origin of parent rock. Selenium is not a metal, but is considered a metalloid or semi-metal, having properties of both metal and non-metal elements (Haygarth 1994). Selenium is essential for the health of organisms like fish, birds and mammals, yet it can also be toxic (Mayland 1994). One of the important characteristics of Se is the very narrow range between sufficient and toxic concentrations. For humans and livestock, the factor between optimal and toxic doses of Se is in the range of 10 to 100 times. For fish the range is as low as 7 to 10 times optimal dietary requirements for fish (Eisler 1985; McNeal and Balistrieri 1989; Lemly 1998). The duality of Se is critically important, as both a nutrient, having a role in protecting against free-radical damage, and as a potential toxicant at higher doses (Ihnat and Wolf 1989; Haygarth 1994; Adriano 2001). The toxicity of Se depends on several factors; therefore, it is important to

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know not only the concentrations of Se, but also understand the mechanisms controlling its distribution and fate in the environment (McNeal and Balistrieri 1989; Haygarth 1994).

Since its discovery, Se has been the subject of much research. The initial impetus for research was diseases related to poultry and livestock grazing in Se-rich areas, or conversely, diseases associated with deficiency in areas of low Se concentrations (Mayland 1994; Ohlendorf 2003). The concentration of Se in forage crops throughout the continental US and Canada, has been used as an index of its geographic distribution and risk of disease associated with deficient or excess Se (Marier and Jaworski 1983).

2.3.1 Observations of Deficiency and Toxicity in Livestock

Marco Polo is credited with recording the first observations of Se toxicity in animals during his travels in western China and Turkestan around 1295 (Barceloux 1999; Vinceti *et al.* 2001; Quinn *et al.* 2007). He described "hoof rot" in horses, a condition where the hooves fell off after grazing on poisonous plants. General Custer's defeat at the battle of Little Big Horn in Montana in 1876 is theorized as being a result of Se poisoning of army horses (Quinn *et al.* 2007).

In 1957, Klaus Schwarz identified the first selenium–responsive disease which led to the recognition of Se as a trace mineral nutrient (Mayland 1994; Oldfield 2002). Since that time, Se deficiency has been found to be responsible for several metabolic diseases in livestock and poultry. Symptoms of Se deficiency in livestock include muscular weakness, skeletal degeneration, lameness, cataracts, hepatic necrosis and reductions in growth, production, and fertility (Eisler 1985; Fan *et al.* 1988).

During the 1930s, Se poisoning of livestock was evident in the western US states of South Dakota, Wyoming, and Nebraska. Horses and cattle were affected with symptoms known as "blind staggers", characterized by an acute and progressive anorexia, emaciation, and impairment of vision. A more subchronic disease was also identified called "alkali disease", resulting in emaciation, stiffness, lameness, loss of hair, and hoof cracking. In both cases; Se was identified as the toxic factor (Eisler 1985; Fan *et al.* 1988; ATSDR 2003, Young *et al.* 2010). Also in the early 1930s in western South Dakota, there were reports of losses of poultry due to

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low survival of hatchlings and congenital malformations related to Se poisoning (Kilness *et al.* 1977).

2.3.2 Observations of Deficiency and Toxicity in Humans

Selenium deficiency in humans was first identified in northeast China in the mid 1930s. Keshan disease, which affected mostly young women and children, was characterized by cardiomyopathy (heart muscle weakness, enlarged heart, impaired heart function, and possibly heart failure) (Whanger 1989; IOM 2000). Another form of Se deficiency called Kashin-Beck disease, afflicted children in Se–poor regions of northern China, North Korea, and eastern Siberia. The symptoms were very different than those of Keshan disease and included degeneration of joint cartilage (osteoarthritis), and in more severe cases, joint deformities, and dwarfism (Linus Pauling Institute 2007). In the 1950s, Se deficiencies were also observed in humans in areas such as Florida, the Pacific Northwest and north-eastern areas of the US and Canada, with similar symptoms as Se toxicity (Eisler 1985).

It was not until the early 1970s, that Se was found to be a critical component of glutathione peroxidase, an important family of metabolic enzymes that protect organisms from oxidative (free radical) damage (Marier and Jaworski 1983).

In China, Se toxicity was identified in people living in Enshi located in the Hubei Province (Whanger 1989). Selenium toxicity in residents of Hubei Province became prevalent in 1958, about the same time as coal (containing up to 5,000 μg/g Se) became the major fuel source. Investigations showed that three routes of Se exposure were responsible for toxicity in the population (Whanger 1989):

- food was cooked or dried over open-pit coal fires in the centre of the room;
- inhaling large amounts of smoke containing volatilised Se from the burning coal; and,
- coal burning in crop fields to fertilise soils.

Following observations of toxicity in livestock in the US in the 1930's, public health officials began to investigate the possibility that intoxication may also be occurring in humans residing in the same regions (Smith and Westfall 1936, 1937). Signs and symptoms of human Se toxicity,

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such as "bad teeth", pathologic nails, and lethargy, were observed although no definitive links to Se intoxication were concluded (Smith and Westfall 1937; Yang *et al.* 1983; Longnecker *et al.* 1991).

2.3.3 Ecological Toxicity and Impacts

In the late 1970s and early 1980s, Se became a serious ecological concern with the discovery that Se bioaccumulation could cause severe impacts to fish and other aquatic wildlife (Ohlendorf 2003). Belews Lake, North Carolina, was a man-made reservoir constructed in 1970, to supply cooling water to a coal-fired power plant (Skorupa 1998). In 1974, the reservoir began receiving effluent from the fly ash settling basin resulting in an increase of mean Se concentrations in the main reservoir to about 10µg/L. The increased Se was associated with the subsequent localized extinction of 26 of 29 resident fish species by 1978, only four years after discharges commenced (Skorupa 1998; Young *et al.* 2010). Sorensen *et al.* (1984) studied Belews Lake including an isolated, less contaminated sub-basin (Se concentrations between 3 and 4 µg/L) and documented fish with sublethal effects that included changes in histopathology (ovarian tissue damage in female fish), hematology and generalised edema. This was the same area studied by Cumbie and Van Horn (1987) who found no apparent overt effects on fish at the population level.

In a similar example, Se contamination from agricultural drainage water, directed to a series of interconnected wetlands known as the Kesterson Reservoir, in California, was responsible for the loss of most of the resident fish species (Skorupa 1998; Young *et al.* 2010). However, the most notable impacts at Kesterson were the reproductive effects to birds using the wetlands. High rates of reproductive failure and deformed hatchlings were reported, as well as signs of acute poisoning in adult birds in the most contaminated areas (Ohlendorf *et al.* 1986; Skorupa 1998).

The concerns regarding Se contamination in wildlife soon lead to growing interest about the possible impact on human consumers. In 1987, the California Department of Health issued a consumption advisory for Suisun Bay near San Francisco, specific to surf scoter, and lesser and greater scaup which fed on Se-contaminated clams and molluscs (Fan *et al.* 1988; Barceloux 1999). Advisories were issued when Se concentrations in the flesh of waterfowl and fish reached 2 ppm (Fan *et al.* 1988). The advisory was based on the 5-fold increase of mean Se in the muscle

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of ducks in contaminated areas (2.2 to 3.6 µg/g wet weight) compared with control areas (USDOI 1998). In this case, the source of Se contamination was oil refinery effluents that were discharged into San Francisco Bay.

Although Se is an essential element for organisms, introduction of Se into the environment from natural and anthropogenic sources can lead to increased concentrations in surface water, groundwater, soils, and vegetation. Consequently, Se can bioaccumulate and may become toxic to sensitive aquatic life, birds, and mammals including humans. The margin between essentiality and toxicity of Se is the narrowest of all trace elements, making the risk of negative impacts from environmental contamination extremely high (Luoma and Rainbow 2008).

In Canada, there are also several examples of anthropogenic Se releases where studies have shown adverse effects on aquatic life and birds are occurring. Good examples of these have been documented in the provinces of BC, Alberta, Saskatchewan, Manitoba and Ontario. In BC and Alberta, large-scale open-pit coal mining has resulted in the mobilization of Se from waste rock leachate with high concentrations of Se into surface and groundwater potentially threatening fish and bird populations (McDonald and Strosher 1998; Casey and Siwik 2000; Kennedy *et al.* 2000; Casey 2005; Holm *et al.* 2005; Harding *et al.* 2005; Wayland *et al.* 2007; Rudolph *et al.* 2008; Canton *et al.* 2008; Minnow *et al.* 2011; Nautilus Environmental and Interior Reforestation Co. Ltd. 2011). In Saskatchewan, uranium mining has been associated with elevated Se in receiving waters and deformity in fish (Pyle *et al.* 2001; Muscatello *et al.* 2006; Muscatello and Janz 2009b). Selenium associated with smelter emissions and effluents have also been studied in Ontario and Manitoba (Nriagu and Wong 1983; Manitoba Conservation 2007).

3.0 Physical and Chemical Properties

Selenium is a member of Group 16 on the periodic chart of elements with the atomic number 34. Selenium is situated on the periodic table between the non-metal element sulphur, and the metal tellurium (IUPAC 1988). The unique Chemical Abstracts Service (CAS) registry number for Se is 7782-49-2 (ATSDR 2003). Selenium and sulphur are chemically very similar in their form,

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compounds, and properties. Selenium is commonly found in association with sulphur-containing (pyrite) rock or soils and often substitutes for S, which accounts for the many interactions between Se and sulphur in both geology and biology. Selenium has a relative atomic weight of 78.96 g/mol, a melting point of 217 °C, a boiling point of 684.9 °C, and a specific gravity ranging between 4.28 (vitreous form) and 4.79 (crystalline form). Elemental Se can be amorphous or crystalline in structure and is found in three general forms: the black vitreous form, the red crystalline monoclinic form, or the metallic grey crystalline hexagonal form, which is the most stable (Adriano 2001; BEAK 2002).

Selenium has six naturally occurring stable isotopes with varying degrees of abundance (in brackets): ^{74}Se (0.89%), ^{76}Se (9.37%), ^{77}Se (7.63%), ^{78}Se (23.77%), ^{80}Se (49.61%), and ^{82}Se (8.73%). Several other unstable radioisotopes exist, for example ^{75}Se, which has a half-life of 120 days, used in radiological and biological tracer applications (Rosman and Taylor 1998).

Selenium exists in four oxidation states in nature, shown in Table 3.1: selenides (-II), elemental Se (0), selenites (+IV), and selenates (+VI). This results in many forms and compounds found in the environment. The concentrations, speciation, and associations of Se depend on pH, redox potential, solubility of the seleno-salts, complexing ability of the aqueous or solid ligands, and biological activity and reaction kinetics (McNeal and Balistrieri 1989). Since the different chemical species of Se have differing biological reactivity and availability, and chemical and geochemical properties, knowledge of Se speciation is important to understand its fate and environmental effects.

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Table 3.1 Examples of the forms of selenium found in the environment (adapted from Haygarth 1994, Terry *et al*. 2000, Simmons and Wallschlager 2005, and Maher *et al*. 2011).

Name	Valence/ Oxidation State	Forms/Se Species	Occurrence
Selenides	-II, Se^{II-}, Se^{2-}	Inorganic selenides, (Se^{2-}, HSe^{-})	Found in reducing environments, sorbed onto soil/mineral particles, e.g., ferroselite ($FeSe_2$), chalcopyrite ($CuFeSe_2$)
		Hydrogen selenide, H_2Se	Unstable highly toxic gas, converts to Se^0 in H_20
		Organic selenides, R_2Se	
		Volatile organic selenides:	
		dimethyl selenide (DMSe), $(CH_3)_2Se$;	Gas, volatilization from soil/sediment bacteria and fungi
		dimethyl diselenide (DMDSe), $(CH_3)_2Se_2$;	Gas, volatilization from soil/sediment plants
		dimethyl selenone $(CH_3)2SeO_2$	Volatile metabolite, intermediate form between DMSe and DMDSe
		Biochemical intermediates, amino acids	Many forms, but most common are the amino acids selenomethionine (SeMet) and selenocysteine (SeCys)
Elemental selenium	0, Se^0		Insoluble, fairly stable, unweathered mineral form of Se, found in water, soil, sediment and biological tissue
Selenium dioxide	+II, Se^{+II}, Se^{+2}	SeO_2	Gas, not a naturally occurring form, product of fossil fuel combustion (coal, oil, gas), and smelting, soluble, forms selenous acid with water
Selenites/selenous acid	+ IV, Se^{+IV}, Se^{+4}	SeO_3^{2-} Hydrogen selenite ($HSeO_3^-$) Selenous acid (H_2SeO_3)	Soluble, found in mildly oxidizing conditions in air, water, soil/sediment, Common form of selenites in soils, easily sorbed onto iron(hydr)oxide minerals $Fe(OH)SeO_3$, or other ions e.g., sodium selenite Na_2SeO_3, highly mobile and available to plants
Selenates/selenic acid	+ VI, Se^{+VI}, Se^{+6}	SeO_4^{2-} Hydrogen selenate $HSeO_4^-$ Selenic acid H_2SeO_4	Common form of Se in surface water and soils, very soluble in water, stable in well-oxygenated water, not easily transformed biologically to more reduced forms, reduction reactions slow. In plants, selenate is actively transported against electrochemical potential gradient.

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Changes in ambient redox potential (Eh) and pH can influence the theromdynamic equilibrium and hence form of Se (Ralston *et al*. 2008). Figure 3.1 is a pourbaix diagram showing the expected speciation of Se as a function of pH and redox potential.

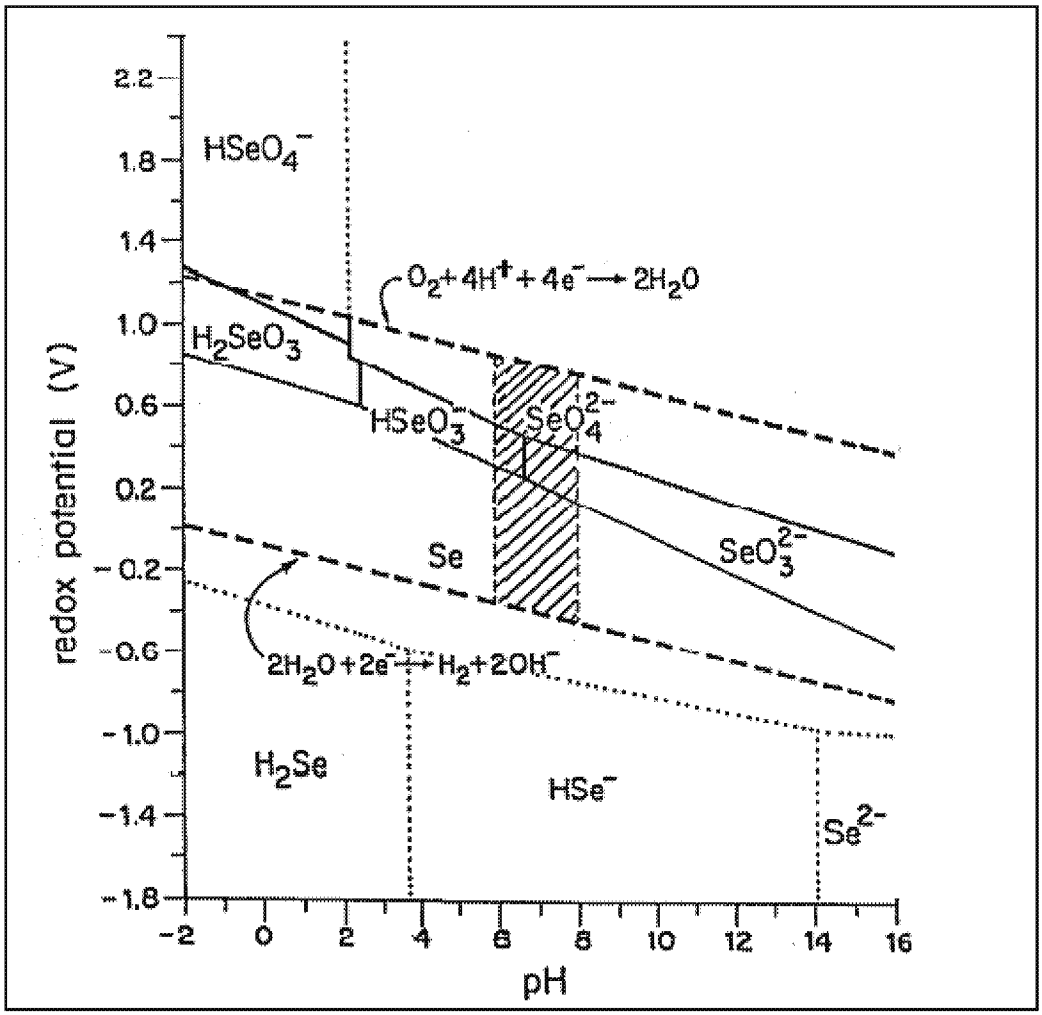

Figure 3.1 Pourbaix diagram: Equilibrium speciation of aqueous inorganic selenium as a function of pH and redox potential (from Milne 1998). The hatched area delineates normal physiological conditions necessary for living cells, and the dashed lines show the equilibrium potentials for water dissociation to hydrogen and oxygen.

While Figure 3.1 in general predicts the stability fields typically found for Se, it is important to recognize that many other factors, like the presence of metals or biological activity, can affect the speciation of Se in natural environments (Luoma and Rainbow 2008).

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In natural waters, selenate (SeO_4) dominates under oxidizing conditions, and is relatively stable even under reducing conditions. Selenides and Se-rich sulphides generally dominate in reducing, acidic, and organic environments. Hydrogen selenide (H_2Se) is a foul-smelling toxic gas which easily oxidizes in the presence of water to elemental Se, (McNeal and Balistrieri 1989). Metal cations react with selenides (Se^{2-}) to form insoluble selenides. Metal selenides, found in metal sulphide ores and Se-sulphide salts are not only insoluble, but also resistant to oxidation. Selenides of mercury, silver, copper, and cadmium are very insoluble (Langmuir *et al.* 2003).

Organic selenides can be found primarily as seleno-amino acids (e.g. selenomethionine, selenocysteine) in biological tissues and in reducing and anoxic environments. Particulate organo-selenides in the water column are highly bioavailable and may be rapidly incorporated into sediments or taken up by organisms (Luoma and Rainbow 2008).

Elemental Se (0) is stable in reducing environments and often found in association with sulphur compounds such as selenium sulphide (Se_2S_2) or polysulphides (McNeal and Balistrieri 1989). Elemental Se also shows some tendency to form catenated (chain) species such as organic diselenides (Milne 1998). Elemental Se has very low solubility with slow oxidation-reduction kinetics but may be transformed (oxidized) by microorganisms to sediment-bound selenites and trace amounts of selenates (McNeal and Balistrieri 1989).

Selenium dioxide (SeO_2) is a yellow to red powder or crystal which is highly toxic if inhaled, swallowed, or absorbed through the skin and dissolves easily in water to form selenous acid (H_2SeO_3) (Eisler 1985; GFS Chemicals 2010). Selenium dioxide does not occur naturally but is economically important to several manufacturing sectors (see Section 4.1.3). It is formed by the combustion of fossil fuels and solid waste, and is a by-product of smelting. Elemental Se is present in petroleum products, in wastes, or metal ores, is converted to SeO_2 during the combustion or smelting process.

Selenite (SeO_3) and selenate (SeO_4) are the dominant selenium oxyanions in soils and surface waters (refer to Table 3.1). Both are very water soluble, with selenate being more soluble than selenite (Maier and Knight 1994; Adriano 2001). Within normal surface water pH and redox

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ranges, only elemental Se (Se^0), selenite ($HSeO_3^-$ or SeO_3^{-2}) and selenate (SeO_4^{-2}), are thermodynamically stable (Milne 1998). Selenite and selenate are both adsorbed strongly by iron (Fe) and aluminum oxyhydroxides and will compete with phosphate and sulphate for sorption sites on Fe-oxides. (Langmuir *et al.* 2003). Microorganisms reduce selenate to elemental Se and selenides (Mayland 1994). Selenate is easily taken up into terrestrial plants through root membranes primarily by high-affinity active transport, against the electrochemical potential gradient (Terry *et al.* 2000). Selenite and organic forms of Se are also taken up by plants but with different mechanisms and in lesser amounts. Microorganisms, plants, and animals have the ability to reduce selenite to selenide, eliminating some Se as respiratory products in the form of volatile organic Se as dimethyl selenide, dimethyl diselenide or dimethyl selenone (Mayland 1994; Terry *et al.* 2000).

Sulphate (SO_4^{-2}) competes directly with selenate (SeO_4^{-2}), affecting its availability to plants, and microorganisms which transform and bioconcentrate Se up through the food web (Simmons and Wallschläger 2005). Fate and transport of Se as it relates to aquatic environments are discussed more fully in Section 5.

4.0 Selenium in the Environment

4.1 Sources

4.1.1 Natural Sources

The primary geologic source of Se is volcanic (Presser 1994a). During the Cretaceous period volcanic activity was extensive, leading to deposition of Se in Cretaceous seas from the gases, ash, and dust associated with volcanic eruptions and the erosion and sedimentation of volcanic rock. Bioaccumulation of Se by microscopic marine organisms then formed the sediments that were deposited during the Cretaceous period, also contributed to the source of Se in soils of marine origin (Presser 1994a). The highest concentrations of Se are found in marine shales, particularly carbon-rich black shale, and phosphate-rich sedimentary rock, formed during the Tertiary and Upper Cretaceous periods (McNeal and Balistrieri 1989; Haygarth 1994; USDOI 1998).The observed distribution of naturally elevated Se concentrations in surficial soils, groundwater and surface water today, is the result of weathering and sedimentary processes acting on these volcanic parent rocks over millions of years.

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Secondary natural sources of Se include those of a *biogenic* (produced through biological processes) nature, precipitation of minerals and organic matter, adsorption, chemical or bacterial reduction, oxidation, and metabolic uptake and release by plants and animals (McNeal and Balistieri 1989). Natural atmospheric releases of Se result primarily from plants and microorganisms (terrestrial and oceanic) which transform Se into volatile organoselenides, and from physical processes like volcanic activity (ATSDR 2003). Forest fires can also be a source of Se to the atmosphere and to local soils from deposition of fly ash (Marier and Jaworski 1983). Soils naturally high in Se are typically found in the arid and semi-arid areas of the world where soils are also alkaline, including some areas of the Prairie Provinces (Hu *et al.* 2009) and mid-western United States (Adriano 2001). Problems can result where naturally high seleniferous deposits or Se-poor soils exist, but more recently it has been the anthropogenic sources of Se that have caused a high level of concern.

4.1.2 Anthropogenic Sources

Anthropogenic release of Se to the environment is associated with industrial, agricultural, mining, and petrochemical operations (such as oil and gas refining) as well as wastewater discharges from municipal sewage treatment plants and landfills (Lemly 2004). Selenium is also released to the atmosphere from combustion of coal and other fossil fuels, and through emissions from smelting and manufacturing of pyritic ores. Selenium bound to fly ash from coal-fired power plants can enter the atmosphere and be deposited to water, or contaminate surface waters from effluent discharges from fly ash storage facilities. Selenium concentrations in soils and organisms tend to be significantly higher in areas of high population density, where Se wastes are being introduced, or sub-surface irrigation drainwater is released (Eisler 1985). A well-known example of anthropogenically-caused Se toxicity occurred during the mid-1970s at Belews Lake in North Carolina. Selenium, found predominantly as selenite in fly ash, was associated with effluents being discharged from a coal-fired power plant which caused extirpation of 16 of 20 resident fish species (Lemly 2002a; Huggins *et al.* 2007). Another well documented example of Se toxicity, is the Kesterson Reservoir in California, where subsurface drainage of agricultural irrigation water that reached concentrations as high as 4,200 µg/L (predominantly as selenate), resulted in devastating impacts to fish and wildlife populations dependent on those habitats (Presser and Ohlendorf 1987; Lemly 2004).

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4.1.3 Production and Uses

Even though Se is widely distributed in the Earth's crust, it is relatively rare and usually not found in concentrations sufficient to warrant economical recovery. Selenium is associated with sulphide (pyritic) ores and is recovered as a by-product of copper smelting and to a lesser extent from the production of gold, lead, nickel or zinc. Anode slimes from electrolytic refining of copper can contain as much as 10% Se. Although coal can contain between 0.5 and 12 μg/g Se (80-90 times that of copper), recovery of Se from coal, while technically possible, is not considered to be practical (USGS 2009a).

Canada is among the top five producers of Se, along with the US, Japan, Belgium, and Chile. In the 2008 Canadian Minerals Yearbook, Natural Resources Canada (NRCan) reported annual production of Se in Canada of 106, 144, and 156 tonnes in 2006, 2007 and 2008, respectively (NRCan 2009a). Based on yearly mineral production estimates compiled by NRCan, Canada's production of Se occurs primarily in the provinces of Quebec, Ontario and Manitoba with 29, 66, and 60 metric tonnes produced respectively (NRCan 2009b). The global supply and demand for Se had been relatively stable until the mid-2000s when demand increased largely as a result of China's increased consumption of SeO_2 which is used as a substitute for SO_2 in the refining of manganese. This substitution reduces power consumption and increases manganese yield in the refining process. Selenium dioxide is economically important to countries exporting this product to China due to its increase in demand and price (USGS 2008).

Selenium has a wide variety of uses. In the early 1900s, Se as sodium selenate, was used in control of plant pests like mites and spiders. Although Se is still used sparingly in some limited pest control applications (greenhouse-grown chrysanthemums, carnations, and cotton plants), its use has largely been discontinued since it was expensive, highly stable, and bioaccumulative resulting in the contamination of crops and imparting negative impacts on birds and mammals (Eisler 1985; ATSDR 2003). In Canada, there has never been a registered pesticide product with Se identified as the active ingredient (Y. Herbison, pers. comm., Pest Management Regulatory Agency, December 2010).

Selenium is used in the manufacture of glass, metal alloys, textiles, petroleum products, medical therapeutic formulas, and photographic emulsions (Eisler 1985; USGS 2008). In the early to

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mid-1900s, SeO_2 was used as an oxidizing agent for many organic compounds, in the production of many new chemical compounds previously unobtainable or produced with much greater difficulty (Waitkins and Clark 1945). SeO_2 is also used as a glass or plastic colourant, as a component of photographic developing and as the main ingredient in gun blueing (Haygarth 1994; USGS 2008).

After studies revealed that lead in ceramics, plumbing fixtures, fluxes, and solder could elevate lead in drinking water or food coming into contact with those materials, Se, along with bismuth, was added to brass as a substitute for lead in these products. Selenium has also been used as a lead substitute in other metal alloys of steel and copper. Owing to selenium's intrinsic properties to convert light energy directly into electricity (and vice versa), other new uses and demands for Se have emerged including photovoltaic cells (e.g., in light meters and security alarms) photocopiers, and other photoconductive technologies (USGS 2009c). Selenium is incorporated in many electronic and technical applications, such as rectifiers (to convert alternating to direct electric current), and semiconductors. There are also diverse industrial and military applications, for example Se is incorporated into eye-shields to protect the vision of workers from laser beams (USGS 2009c).

Most recently, Se has been used as a component in nanotechnology, making Se of greater commercial and medical interest in this emerging field. Engineered CdSe nanoparticles are used in electronics, experimental biology, and medicine as a result of their ability to emit light with specific wavelengths (Norwegian Pollution Control Authority 2008). CdSe nanoparticles belong to a group of such particles called quantum-dots or Q-dots, which are used in solar cells, LEDs, transistors, and diode lasers. CdSe nanoparticles are specifically used in fluorescence imaging to localize specific cells (e.g., locating tumor cells). However, results of studies examining the ecotoxicity of CdSe Q-dots have been variable since the effects appear to be related to several factors, such as the method of synthesis and surface coatings applied to the Q-dots (Farré *et al.* 2009). Since these nanoparticles are very small and reactive, and relatively little is known about their environmental effects, the long-term implications of introduction of these nanoparticles into aquatic environments remains a concern.

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In addition to industrial applications, Se has many pharmaceutical uses which reflect its beneficial health properties for humans and livestock. Selenium is an important antioxidant which plays a role in proper immune function. Selenium, usually in the form of sodium selenite, is used as a supplement in the treatment of diseases like AIDS, Alzheimer's, arthritis, asthma, cancer, cardiovascular, pancreatitis, reproductive problems, thyroid dysfunction, and viral infections (USGS 2009a). Selenium is commonly added to livestock feeds or as a component of mineral licks. Selenium-based shampoos, containing Se-mono or disulphide, are used to control dandruff and fungal infections of skin in humans, as well as for dermatitis or mange treatment in dogs (Eisler 1985; Haygarth 1994).

4.2 Environmental Concentrations of Selenium

All Se concentrations reported in this document have been converted to common units: ng/m^3 for air; µg/L for water; µg/g or mg/kg for soils, sediment and tissue residues, and in mg/L for blood (whole blood or serum) and other bodily fluids (urine). Where possible, studies reporting data as wet weight (ww) have been converted and reported in this document as dry weight (dw) using either the reported % moisture or an estimated % moisture, which is indicated. Details reported in published or grey literature, such as summary statistics (mean, geometric mean, or median), number of samples (n), standard error (SE), standard deviation (SD), and confidence intervals (CI) were included in this document when those data were available.

The data presented in this section is intended to provide the reader with examples of Se concentrations found in various environmental compartments including those typically measured in aquatic ecosystems (water, sediment and biotic tissues). The data presented here is not a comprehensive compilation of background and human-influenced levels of Se, but provides a cross-section of the data reported in published and grey literature that are representative of typical Se values as well as some anomalous Se concentrations in the environment.

4.2.1 Air

The global cycling of Se includes an atmospheric component that represents an important mechanism for Se transformation and redistribution, and has the potential to affect ecosystems world-wide (Haygarth 1994; Cutter and Cutter 2001). Selenium emissions to the atmosphere can

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be divided into naturally-occurring and anthropogenic sources. Nriagu (1989) estimated a total Se flux to air of 14,700 tonnes per year which is consistent with more recent estimates of 13,000 to 19,000 tonnes per year (Wen and Carignan 2009). Of these emissions, natural sources account for 57%, while anthropogenic sources account for 43% of total emissions (Nriagu 1989). Natural sources of Se to the atmosphere can result from either biological or physical processes, but approximately 90% are biogenic. These are primarily in the form of gaseous dimethyl selenide generated from the microbial methylation and subsequent volatilization of Se from soils, wetlands, marine and freshwaters, and vascular plants (Haygarth 1994; Cahill and Eldred 1998). The vast majority of natural biogenic Se comes from the ocean, estimated to be anywhere from 5,000 to 8,000 tonnes per year, and a smaller fraction, approximately 1,200 tonnes per year, from terrestrial sources (Nriagu 1989). The remaining 10% of natural sources are non-biogenic, and include volcanoes (8%), suspension of sea salt (2%) and crustal weathering (<1%) (Cahill and Eldred 1998). Taylor and Lichte (1980) measured Se in volcanic ash in the vicinity of the Mount St. Helens, Washington eruptions of 1980. The range of Se concentrations were between <0.2 and 3 μg/g depending on location, demonstrating how natural events can influence localised distribution of Se in the environment.

Anthropogenic Se emissions to air are primarily from combustion sources, including coal burning (50%), oil (9%), and other miscellaneous sources (10%), along with copper smelting (20%), lead and zinc smelting (4%), the production of Se dioxide (4%), as well as other manufacturing processes, primarily glass and ceramics (<4%) (Cahill and Eldred 1998).

Selenium concentrations in air are usually less than 1 ng/m^3, with levels in semi-urban and rural continental areas ranging between 0.3 and 1 ng/m^3 (Haygarth 1994; Mosher and Duce 1989). Unpublished data from the Canadian National Air Pollution Surveillance (NAPS) network, representing all the Provinces and Territories except Yukon, Newfoundland and PEI, was summarised by the Canadian Council of Ministers of Environment (CCME 2009). Based on fine particulate matter (PM_{10}; the fraction less than 10 μm in diameter) samples collected across the network between 2002 and 2003, they estimated a background concentration of atmospheric Se in Canada of 1 ng/m^3 (Table 4.1).The range of sample results in that data set was from 0.5 ng/m^3 measured in Iqaluit, Nunavut, to 1.7 ng/m^3 in Egbert, Ontario 75 km northwest of Toronto.

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Remote continental and oceanic areas have much lower atmospheric Se concentrations, in the range of 0.43 and 0.25 ng/m^3 total Se, respectively (Nriagu 1989). Selenium concentrations measured at remote sites in the north-western United States to the Appalachian Mountains in the east, range from 0.05 ng/m^3 to 1 ng/m^3, respectively (Cahill and Eldred 1998).

While marine biogenic Se contributes significantly to the overall concentration and fluxes, anthropogenic sources contribute substantially to concentrations in the atmosphere (Nriagu and Pacyna 1988). Anthropogenic sources of Se have increasingly become important in the mobilization and redistribution of Se into the global biosphere (Nriagu and Wong 1983; Nriagu and Pacyna 1988; Nriagu 1989; Wen and Carignan 2009).

Table 4.1 Typical and background selenium concentrations in air.

Location	Mean Air Se (ng/m^3)	Location / Description	Reference
Global	≤ 1	Typical concentration of Se in air globally	Mayland (1994)
Canada	1 (n = 721) 0.5 – 1.7 (n = 721)	Background PM_{10} Se concentrations from Canadian NAPS network, 2002-03; Range of Se in PM_{10} samples	CCME (2009)
Remote continental or oceanic regions	0.43 and 0.25	Range of atmospheric Se concentrations in remote regions	Nriagu (1989)
North-western US Appalachian Mountains	0.05 1	Typical Se concentrations in air in remote regions of the US	Cahill and Eldred (1998)

For example, in areas where high vehicle traffic volumes, petroleum refineries, or metal smelting plants contribute to the atmospheric emissions of Se, local Se concentrations in air can be significantly elevated over background (Cutter 1989). Urban or industrial areas can have the highest average concentrations of atmospheric Se. Mosher and Duce (1989) reported total Se concentrations in urban areas between 1 and 10 ng/m^3, comparable to other reported urban air concentrations in the range of 3 to 4 ng/m^3 (Marier and Jaworski 1983; Nriagu 1989).

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Near industrial point-sources such as smelters, refineries and coal-powered power plants, air-borne particulate matter (PM) can contain 120 to 6000 ng/m^3 Se, depending on the source, and may result in long-range dispersion of particulate and volatile Se (Nriagu and Wong 1983; Mosher and Duce 1989; Cutter 1989). Based on studies conducted between 1979 and 1980 in Sudbury, Ontario near five copper-nickel mining and smelting operations, Nriagu and Wong (1983) estimated the total annual atmospheric emissions of Se were about 50 tonnes/year. Within a 3 km radius of the Copper Cliff smelter stack, Se concentrations in air ranged from 100 to 6,000 ng/m^3. Lakes sampled within a 30 km radius showed that water and sediments of lakes close to the smelter had higher Se concentrations compared with more distant lakes. These data demonstrate the dramatic influence point sources of Se to the atmosphere may have on local environments (Nriagu and Wong 1983).

4.2.2 Soils

The range of Se content in soils can vary widely. Soils in areas where the parent geologic formations are of Tertiary and Upper Cretaceous age, particularly marine Cretaceous shales, have the potential for Se leaching (Lakin 1961; Presser 1994a). In areas where precipitation is greater than 64 cm per year, Se is naturally leached from surface soils thereby reducing the risk of Se accumulating in soils and plants to high concentrations (Lakin 1961). Areas particularly susceptible to Se contamination are those where both a geologic source of Se exists and the evaporation rate exceeds precipitation by a factor of 2.5 or greater (Seiler e*t al.* 1999).

Background soil Se concentrations for some provincial sites across Canada are summarized in Table 4.2. Based on results of studies conducted across Canada, the Canadian Council of Ministers of the Environment (CCME) determined the mean natural background concentration of soil Se for Canada was 0.7 µg/g (CCME 2009). Soils considered low risk for Se toxicity to plants range from 0.02 to 2.5 µg/g Se, while a risk of Se toxicity may be expected in soils with Se concentrations of 4.0 to 6.0 µg/g (Marier and Jaworski 1983). The toxicity of Se in soils is

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Table 4.2 Background soil selenium concentrations reported in the literature for locations in Canada.

Location	Mean Soil Se (µg/g dw)	Location Description	Reference
Canada	0.7 (n = 967) 0.3 (n = 173)	Background representing data from several Canadian studies; Background representing data from 173 samples across Canada	CCME (2009) McKeague and Wolynetz (1980), cited in CCME (2009)
Southern Ontario	0.46 (± 0.38) 0.1 – 3.9 (n = 294)	Mean (± SD) and range of Se in soils from 294 surface soil samples (top 25 cm)	CCME (2009)
Manitoba Saskatchewan Alberta	0.62 (± 0.44) <0.2 – 3.8 0.53 (± 0.28) 0.1 – 3.1 0.55 (± 0.28) 0.1 – 2.7	Mean (± SD) and range of Se in soils (total number of samples = 1,076)	Natural Resources Canada (unpublished data 1992), cited in CCME (2009)
Alberta	0.48 (± 0.28) (n = 258)	Mean (± SD) of 258 agricultural soil samples from 129 sites representing 43 areas across Alberta	CCME (2009)
British Columbia	0.29 (± 0.37) (n = 416)	Mean (± SD) soil Se based on analysis of 416 surficial soil samples (MDL = 0.2 µg/g)	BC MoE Background Soil Quality Database[1]

[1] Data accessed from MoE web site http://www.env.gov.bc.ca/epd/remediation/guidance/index.htm#tech, Technical Guidance document number 17, Background Soil Quality Database.

dependent on its availability to plants which is controlled by many factors (see Section 4.2.3). Canadian soil quality guidelines for the protection of environmental health are 1.0 µg/g Se for agricultural, residential or parkland uses, and 2.9 µg/g Se for both commercial and industrial land uses (CCME 2009).

Background soil Se data for BC were obtained from the MoE background soil quality database, which has archived historical surficial soil metals data used in the development of the *Contaminated Sites Regulation* soil quality standards.[2] Approximately 75% of the soil samples collected in relatively uncontaminated locations across BC had Se concentrations less than the analytical method detection limit (MDL) of 0.2 µg/g (total number of samples 448). Of the 25% of samples from all regions that reported soil Se above the MDL, the concentrations were ≤ 1.1

[2] Background Soil Quality Database, Technical Guidance document number 17, accessed from MoE web site http://www.env.gov.bc.ca/epd/remediation/guidance/index.htm#tech,

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µg/g, with slightly higher concentrations reported in a few locations in urban parks in Metro Vancouver (maximum Se concentration was 2.4 µg/g). Soil Se concentrations in BC are well below the Canadian Se soil quality guideline for agriculture, residential or parkland uses, with only a few exceptions.

A few areas in Canada might slightly exceed the lowest Canadian soil quality Se guidelines as a result of naturally-occurring Se in the surficial geology and it is not uncommon for soil Se concentrations to greatly exceed Canadian soil guidelines near anthropogenic sources. For example, soils in the vicinity of three copper-nickel smelters near Sudbury, Ontario, had elevated soil metals concentrations, including Se (Ontario MoE 2004). Soil Se concentrations ranged from less than or equal to the MDL of 0.5 µg/g in areas furthest from smelters, to 49 µg/g in adjacent areas (Ontario MoE 2004).

Manitoba Conservation measured Se in soils, vegetation, and snow pack in the vicinity of Hudson Bay Mining and Smelting located in Flin Flon. This plant has been operating from the early 1930s, and recently (2010) ceased production. Soils in close proximity to the smelter and in areas where there had been very little soil disturbance had mean Se concentrations of 177.2 µg/g, while at locations more remote from the smelter concentrations were below the MDL of 0.2 µg/g (Manitoba Conservation 2007). In 92 of the 108 sampling locations within the study area, soil Se concentrations exceeded the Canadian soil quality guideline of 1 µg/g (Manitoba Conservation 2007; CCME 2009).

4.2.3 Terrestrial Plants

It is difficult to discuss plant concentrations of Se without also discussing soil Se concentrations since the two are closely related. Selenium concentration in plants, particularly forage crops, have been used as an index of the distribution of Se in soils throughout the continental US, Mexico and Canada. Such data are also used to determine where risk to animals might exist due either to Se deficiencies or to toxicity (Marier and Jaworski 1983). The "Great Plains" region of the arid mid-western US and the Canadian Prairie Provinces are areas of relatively high Se concentrations in soils and vegetation (Marier and Jaworski 1983).

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It is unclear whether or not Se is required for plant growth; however, if Se is required it is probably at very low concentrations (Marier and Jaworski 1983; NRC 1983; Mikkelsen *et al.* 1989; Mayland 1994; Efroymson *et al.* 1997; Ellis and Salt 2003). The uptake of Se by plants is primarily through the roots, followed by translocation to leaves and stems (Mikkelsen *et al.* 1989; Mayland 1994). The uptake and concentration of Se in plants is dependent on the concentrations of Se in soils, the availability of that Se (soil pH, form of Se), and the plant species' ability to uptake and store Se (Marier and Jaworski 1983). In well-aerated, alkaline soils, Se tends to form selenates, which are easily taken up by plants (NRC 1983). Selenites are also readily absorbed by plants (Mikkelsen *et al.* 1989). Soils rich in organic matter may inhibit Se mobilization and uptake by plants (Fernández-Martínez and Charlet 2009).

Forage plants and grains considered to have low Se typically contain less than 0.05 μg/g, whereas adequate concentrations would be in the range of 0.1 μg/g (Marier and Jaworski 1983). Most agricultural crops have Se concentrations that do not exceed 1 μg/g (Mikkelsen *et al.* 1989). In areas where soils have low Se, locally grown forage may have inadequate levels of the element resultin in the need to supplement Se in livestock feeds (Mikkelsen *et al.* 1989). Alternatively, vegetation in areas with Se-rich soils may contain levels of Se that could be toxic to livestock. A diet containing 0.1 to 0.3 μg/g Se is generally thought to be adequate for most animals that require it (Mayland 1994). This data has been summarized below in Table 4.3.

Table 4.3 Summary of literature thresholds for Se concentrations in forage crops considered deficient, acceptable, or toxic to livestock.

Effect on livestock	Plant tissue Se concentration (μg/g dw)	Reference
Low (deficient)	< 0.05	Marier and Jaworski (1983)
Adequate	0.1 – 0.3	Mayland (1994)
Toxicity in livestock	3 - 15	Mayland (1994)

Since the ability of plants to accumulate and tolerate Se varies greatly, some plant species may experience Se toxicity (Mikkelsen *et al.* 1989). Direct Se toxicity to plants is influenced by the

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same factors as those that affect Se uptake – soil pH, texture, organic content and the presence of other competitive ions (CCME 2009). For example, there is an antagonistic effect between sulphate (SO_4) and selenate (SeO_4) in soils, and to a lesser extent between sulphate and selenite (SeO_3). Hence, the presence of higher soil sulphate results in reduced Se concentrations in plant tissues (Mikkelsen *et al.* 1989). The mechanism is thought to be either directly antagonistic, with SO_4 substituting for Se, or plant growth is enhanced by added SO_4 which dilutes the effect of Se (Mikkelsen *et al.* 1989). This interaction between sulphur and Se is not surprising due to the physical and chemical similarities between the two elements.

Plant Se concentrations are also influenced by differences in the ability of different plants to absorb and accumulate Se. However, toxicity can occur when Se is taken up, translocated, and incorporated into the essential sulphur compounds in the plant as Se-analogues. Toxic effects on plants can occur at soil Se concentrations as low as 1.5 µg/g; symptoms of Se poisoning include chlorosis, stunting, yellow leaves and reduction in plant biomass and yields in forage crops (CCME 2009).

Plants known as "Se accumulators" are tolerant of Se-rich soils and may incorporate in excess of 3 to 15 µg/g Se in their tissues, which is considered a dietary effect threshold above which animals begin to show signs of Se toxicity (Mayland 1994). Selenium accumulator plants are common in the genus *Astragalus, s*ome species of which can contain concentrations as high as 20,000 µg/g Se (Mikkelsen *et al.* 1989). Typically, selenate and selenite is taken up through the roots, and translocated throughout the plant where it is incorporated into amino acids, most of which (60 – 80%) is further incorporated into proteins. Accumulator plants are thought to be able to prevent incorporation of Se-amino acids into proteins, thereby avoiding a phytotoxic response that normally occurs in plants at higher Se concentrations (Mikkelsen *et al.* 1989). Accumulator plants are often found in semi-arid regions with seleniferous soils, conditions that may occur in at least 15 countries including Canada, Mexico, the United Kingdom, Ireland, Israel, Venezuela, Australia and the United States (Adriano 2001). In BC, there are approximately 32 species of *Astragalus* (milkvetches); most are native, found in a variety of climates, several are yellow-listed and some very rare species which are red-listed (Douglas *et al.* 1999). Some species of

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milkvetch may be toxic to livestock, but details about the toxicity and ability to accumulate Se of species in BC could not be found.

Coastal regions of British Columbia have forages that are deficient in Se content for livestock needs (Bittman *et al.* 1999). This is likely a reflection of the very low soil Se concentrations in coastal areas, which is consistent with data from the BC background soil quality database. Selenium levels in livestock forages grown in BC were assessed by Miltimore *et al.* (1975) and summarized in Table 4.4. The Thompson, Nicola, Cariboo, Creston, east Kootenay and south Okanagan regions all had mean Se forage (grasses and legumes) from 0.20 to 0.34 µg/g, with the Thompson having the highest mean concentrations (0.34 ±0.2 µg/g Se, n=176). The Chilcotin and Peace regions both had mean forage Se concentrations of 0.19 µg/g (n=39 and 29, respectively). The Coastal, Bulkley and Boundary regions had mean forage Se concentrations of 0.16 (n=296), 0.14 (n=27), and 0.14 (n=25) µg/g, respectively. Only two areas with samples considered high (twice the standard deviation of means) in Se were observed in the Creston Flats (mean Se 1.5 µg/g, n=11) and Miles 410-422 on the Alaska Highway (mean Se 3.04 µg/g, n=17) (Miltimore *et al.* 1975). Local veterinarians have identified this area near Mile 422 of the Alaska Highway north of Dawson Creek, known as Toad River, as having a high incidence of Se toxicity in horses (Dr. M. Ross, DVM, pers. comm., Dawson Creek Veterinary Clinic, December 2011).

Agricultural runoff and irrigation drainage waters have been known to carry elevated Se concentrations in areas like the San Joaquin Valley in California where soils have higher concentrations of Se (Presser 1994b). Southern Manitoba and parts of Saskatchewan share similar geology and possibly similar soil Se concentrations. However, studies conducted in southern Manitoba indicate that, in spite of this, the risk of Se contamination to surface water, of the scale experienced in California, is very low (Hu *et al.* 2009). The reason for this is related to relatively fewer acres of well-drained soil Manitoba irrigates compared with other heavily agriculturalised areas and the very different climate and evaporation rates (Hu *et al.* 2009).

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Table 4.4 Selenium concentrations measured in grasses and legumes in BC collected between 1966 and 1973, listed in order from highest to lowest (from Miltimore *et al.* 1975).

Region	Mean Se (± SD) (µg/g dw)
Thompson	0.34 (± 0.20), n = 176
Nicola	0.27 (± 0.14), n = 36
Cariboo	0.22 (± 0.13), n = 81
Creston	0.21 (± 0.15), n = 183
East Kootenay	0.21 (± 0.12), n = 23
South Okanagan	0.20 (± 0.18), n = 60
Chilcotin	0.19 (± 0.07), n = 39
Peace	0.19 (± 0.10), n = 29
Coastal	0.16 (± 0.09), n = 296
Bulkley	0.14 (± 0.11), n = 27
Boundary	0.14 (± 0.09), n = 25

4.2.4 Water

4.2.4.1 Marine Water

Marier and Jaworski (1983) found that seawater typically had lower Se concentrations than freshwater attributed to the increased contact freshwater has with Se-rich rock and soils. An early estimate of the Se content in marine waters was slightly over 4 µg/L at 19 ‰ salinity (Sverdrup *et al.* 1942). However, Se concentrations in open ocean water with minimal influence from human activity tend to be well under 1 µg/L. There are several other estimates of worldwide background Se concentrations in seawater, with levels ranging from 0.009 to 0.045 µg/L (Sui and Berman 1989). By comparison, Eisler (1985) reported higher Se concentrations in sea water off California (0.06 µg/L). Marier and Jaworski (1983) estimated average Se in seawater to be in the range of 0.09 ± 0.03 µg/L. Vertical profiles along a transect in the western Atlantic showed concentrations of dissolved Se in seawater varied little, ranging between 0.04 and 0.07 µg/L (Cutter and Cutter 2001). Coastal ocean waters off Brazil within the influence of the Amazon

River had dissolved Se concentrations of 0.02 µg/L (Cutter and Cutter 2001). Profiles of total dissolved Se, selenite (Se IV) and selenate (Se VI) showed nutrient-like behaviour of Se, with surface water depletion and deeper water enrichment, (Cutter and Cutter 2001). Sui and Berman (1989) found this same pattern with oceanic Se concentrations being highly correlated with micronutrients such as silica and phosphorus.

Marine microorganisms accumulate Se from water and sediment in a similar way as freshwater organisms, and like freshwater systems, marine primary producers are considered to be the first step in Se bioaccumulation. Larger marine organisms further accumulate Se from the food web and to a lesser extent from surrounding water and sediment. Uptake from water and Se upwelling from sediment may be more important sources of Se for filter feeding organisms like bivalves and corals (Sui and Berman 1989).

There are few Canadian data for Se in marine water, but those found were generally below the analytical method detection limit (MDL). Nine water quality samples from locations around Sooke Harbour in 2008 on the south west coast of Vancouver Island were all below the MDL of 0.5 µg/L total Se (BC Ministry of Environment EMS database[3]). Samples collected between 2000 and 2009 in coastal marine waters from Howe Sound, Vancouver Harbour, English Bay, Coal Harbour, False Creek and Second Narrows were all reported as less than the MDLs which ranged from 0.06 to 2.0 µg/L in 2000/2001 and from 0.04 to 2.0 µg/L in 2009.

4.2.4.2 Groundwater

Groundwater is a subset of freshwater and is often a neglected environmental compartment in many ecological assessment and monitoring programs. Globally, mean concentrations of Se in groundwater are estimated to be approximately 0.2 µg/L (Nriagu 1989). In Canadian groundwater drinking sources, typical Se concentrations were generally less than 1.0 µg/L (CCME 2009).

[3] Data accessed from the BC Ministry of Environment, Environmental Monitoring System (EMS) database, Jan 2010.

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Exceptions to these typically low Se concentrations in groundwater occur in areas where the geology is high in Se, or where anthropogenic activities elevate Se. CCME (2009) reported exceedances of the maximum acceptable drinking water guideline of 10 µg/L in Quebec, near Walkerton Ontario, Saskatchewan, southern and northern Alberta and southwest BC (Chilliwack). For example, in seleniferous geologic formations in Saskatchewan, Se concentrations in some groundwater drinking water supplies exceed the Health Canada (1992) drinking water guideline of 10 µg/L total Se (S. Ferris, pers. comm., Saskatchewan Environmental Protection Branch, June 2010). These water supply systems are all south of Saskatoon, Saskatchewan and coincident with geologic formations known to be high in Se (Marier and Jaworski 1983; Seiler *et al.* 1999; Saskatchewan Ministry of Environment 2008;).

Zubel (2000) conducted a study near Cultus Lake (Chilliwack, BC) and found that Se concentrations in two groundwater sources used for drinking were 100 and 80 µg/L, values well over the drinking water guideline. The source of contamination was thought to be from septic systems and/or fertilizers. The remainder of 28 groundwater sample results were less than the detection limit which ranged from <30 to < 60 µg/L. Such high analytical detection limits provides little information on background groundwater quality relative to normally low background Se concentrations in groundwater.

4.2.4.3 Surface (Fresh) Water

Background Se concentrations in surface waters range from 0.1 to 0.4 µg/L (USDOI 1998; Adriano 2001). Luoma and Rainbow (2008) estimated that background Se concentrations for undisturbed surface waters may be between 0.07 and 0.19 µg/L. Other estimates at the lower end of background concentrations for Se include those for lakes, between 0.001 and 0.04 µg/L (Eisler 1985), and for rivers (global), 0.06 µg/L (Nriagu 1989).

However, anthropogenic inputs from runoff or effluents can elevate Se concentrations in surface water, but increased atmospheric concentrations can also be a conduit for Se directly into large waterbodies (lakes) or indirectly into streams from soil runoff (Adriano 2001). As examples of these kinds of industrial contributions, effluent concentrations of Se in metal mining discharges in Canada can range from 4.9 to 110 µg/L (BEAK 2002), and smelters have been known to

elevate local lake water concentrations of Se through direct wet and dry deposition (Nriagu and Wong 1983; Manitoba Conservation 2007).

Typical Se concentrations in Canadian freshwaters are similar to those found elsewhere globally, typically much less than 1 µg/L, ranging from 0.01 to 4 µg/L (CCME 2009). Based on a survey of 122 municipalities across Canada, Subramanian and Méranger (1984, as cited in CCME 2009) showed that Se concentrations in typical surface drinking water supplies were less than or equal to the MDL of 0.5 µg/L (CCME 2009).

Environment Canada and provincial government agencies have collaborated since the mid-1980s, carrying out jointly funded water quality monitoring programs under the federal-provincial Water Quality Monitoring Agreements (WQMA). This database provides an effective basis for comparing water quality across diverse areas within and between provinces. This data and information on Se concentrations from other authors have been summarized in Table 4.5.

Environment Canada Atlantic Region ENVIRODAT water quality database show 2009 surface water Se concentrations generally at or below the minimum detection limit of 0.01 µg/L in the Atlantic Provinces.[4] In Newfoundland and Labrador, data collected under the Canada-Newfoundland WQMA program between 1986 and 2000, was used to generate a map showing Se concentration contours for the purpose of identifying "hot spots" across the province (NLWRMD 2010). The figure showed that surface water total Se concentrations were generally less than the existing CCME water quality guideline for aquatic life of 1.0 µg/L. The only instances of elevated Se were at urban sites or sites with saltwater intrusion, the latter highlighting the connection between surface and groundwater in coastal areas (R. Paterson, pers. comm., Newfoundland and Labrador Department of Environment and Conservation, June 2011).

Somers *et al.* (1999) reviewed water quality data collected across Prince Edward Island. Sites represented ambient environmental condition across a range of land-use (mostly forested to largely agricultural) and stream size, but did not include sites influenced strongly by effluent

[4] Data accessed through Environment Canada Atlantic Region water quality web page, http://map.ns.ec.gc.ca/envirodat/root/main/en/extraction_page_e.asp June 2010.

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Table 4.5 Summary of surface water quality data across Canada (other than in BC) for background or minimally influenced locations.

Location /Station Name	Mean Se (µg/L)	Description (period of record, if known)	Impact[1]	Reference
Canadian surface waters	≤ 0.05	Summary of surface source drinking water for 122 municipalities across Canada	U	Subramanian and Méranger (1984)
	<0.01 – 4.0	Range of Se concentrations in surface water	U	NAQUADAT(1985) (cited in CCME 2009)
Atlantic provinces	≤ 0.01 (n=231)	Summary of Environment Canada Atlantic Region ENVIRODAT 2009 water quality database	R/PI	ENVIRODAT 2009
Newfoundland and Labrador	< 1.0 µg/L	Generalized summary of surface water quality data 1986 – 2000.	R/PI	NLWRMD 2010
Prince Edward Island	0.08 (0.05) 0.16 (max)	Mean (SD) and maximum total Se conc at freshwater sites representing ambient environmental conditions (Atlantic Region ENVIRODAT)	R/PI	Somers *et al.* 1999
Nova Scotia, Cape Breton, New Brunswick	≤ 1.0	Monitoring data from 29 rivers in NS, NB and Cape Breton, 1992 to 1996. Two values over MDL, 1.16, 1.42 µg/L	R/PI	Dalziel *et al.* 1998
Quebec	< 0.05	2009 data collected at Rivière à la Pêche, a pristine location in La Mauricie National Park.	R	Environment Canada data request 2010
Ontario	< MDL (0.05 – 1.0)	Data from 14 long-term monitoring stations at background or minimally impacted sites in Ontario.	R/PI	PWQMN data request 2010
Ontario Sturgeon River (Severn Sound)	< 0.5	Sturgeon River near Severn Sound, two samples in 2002.	R	Ontario MoE data request 2012
Manitoba	< 0.4	Data collected at water quality surveillance sites in Manitoba since 2001	R	Manitoba Conservation data request 2010
Saskatchewan	0.02 - 0.1	Range of Se concentrations at reference sites in two studies near uranium mines	R	Muscatello *et al.* (2008); Pyle *et al.* (2001)
Alberta	0.3 – 0.7	Range of average Se for 19 WQ stations 2005 to 2007;	R/PI	Environment Canada data request 2010; Casey (2005)
	0.7	Median Se concentration at reference sites above coal mining activities	R	

[1]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown

discharges. Mean Se concentrations in fresh surface water sites were 0.08 (± 0.05) µg/L, with the maximum concentration of 0.16 µg/L. This demonstrates the very low Se concentrations across PEI in surface waters.

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Lake water quality surveys conducted between 1981 and 2005 by Nova Scotia Environment showed Se in lake water was consistently less than the MDL of 1.9 or 2.0 µg/L (Nova Scotia Environment 2009). Dalziel *et al.* (1998) conducted seasonal sampling on 29 rivers in Nova Scotia, Cape Breton and New Brunswick between 1992 and 1996. With the exception of two samples, measuring 1.16 and 1.42 µg/L, dissolved Se concentrations at all river sites were below the MDL of 1.0 and 1.2 µg/L. Although Se is not a wide-spread problem in Atlantic Provinces, there has been concern regarding Se in coal mining areas in the north-eastern area of the mainland, like south of Canso Strait, and Cape Breton Island. Selenium releases are also a concern at the Sydney Steel Mill which operated between approximately 1901 and 2000, releasing toxic wastes to Sydney Harbour and Tar Ponds on Cape Breton Island (D. Taylor, pers. comm., Nova Scotia Environment, May 2010).

Data collected in 2009 at a federal water quality station on Rivière à la Pêche in La Mauricie National Park in Quebec (a pristine location) show Se concentrations at or below the MDL of 0.05 µg/L. Within the freshwater fluvial reach of the St. Lawrence River at Lavaltrie, PQ, 25 samples collected between 2007 and 2009 had an average Se concentration of 0.1 µg/L (Environment Canada ENVIRODAT[5]). Although the St. Lawrence River is influenced by human activities, these sites were considered representative of minimally impacted water quality concentrations for Se for most streams in the province of Quebec (G. Tardif, pers. comm., Environment Canada, April 2010).

Water quality data collected in Ontario during the mid to late 1970s showed that Se concentrations in lakes Superior, Michigan, Huron, Erie and Ontario were generally below the MDL of 0.1 µg/L to 1.0 µg/L (IJC 1981). The exception was Lake Erie, which had Se concentrations that ranged between < 0.1 and 36 µg/L, the latter likely influenced by anthropogenic discharges (IJC 1981). Surface water quality data from the Ontario Ministry of Environment's Provincial Water Quality Monitoring Network (PWQMN) were retrieved from 14

[5] Data obtained by request through Geneviève Tardif, Fresh Water Quality Monitoring and Surveillance, Water Science and Technology, Environment Canada, Gatineau QC, May 2010.

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long-term monitoring stations at background or possibly minimally impacted sites throughout Ontario.[6]

A review of these data showed that most sites have Se concentrations consistently below the MDL, which was 1.0 µg/L during the early years of the program, and 0.05 µg/L in later years (C. Rocks, pers. comm., Ontario MoE, April 2010). At one station still being monitored on the Ottawa River near the Otto Holden Dam, there was a measurable and slightly higher average Se concentration of 0.27 µg/L for the period of record between 1989 and 1994. Although this concentration is elevated relative to other Ontario sites, it is still well under the CCME WQG of 1 µg/L (CCME 2007a). At a site on the Sturgeon River in Ontario, two samples collected in 2002 had Se concentrations < the MDL of 0.5 µg/L.[7]

Exceptions to these low levels are sites associated with point source contributions from mining effluents or atmospheric emissions. An example is in Sudbury, where lakes within a 30 km radius of the copper-nickel smelter had up to 4 times the concentration of Se (0.2 to 0.4 µg/L) compared to lakes outside the influence of the smelter (≤0.1 µg/L) (Nriagu and Wong 1983). A more recent study on nine lakes at varying distances (4 to 204 km) from the Sudbury smelter showed similar patterns in dissolved Se. At two lakes in close proximity (4 km) to the smelter, the average water column Se was 0.67 µg/L (Chen *et al.* 2001). However, Se concentrations averaged 0.1 µg/L in five lakes that were greater than 30 km from the smelter (Chen *et al.* 2001). These studies show that atmospheric deposition from metal smelters can be an important source of Se to surface waters.

Manitoba's water quality data shows surface water Se concentrations near or below the MDL (0.4 µg/L since 2001 and 2.0 µg/L prior to 2001) (K. Jacobs, pers. comm., Manitoba Water Stewardship, March 2010). Se concentrations are elevated in areas in surface waters influenced by hard rock mining and/or smelting activities. One example is surface water around the Hudson Bay Mining and Smelting (HBMS) operation near Flin Flon. In 2009, Ross Lake, which receives effluents from HBMS as well as urban runoff from the City of Flin Flon, had Se concentrations

[6] Data obtained by request though Carline Rocks, Ontario Ministry of Environment, May 2010.
[7] Data obtained by request from Georgina Kaltenecker, Ontario Ministry of Environment, August 2012.

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between 156 and 162 µg/L (K. Jacobs, pers. comm., Manitoba Water Stewardship, March 2010). As part of a human health risk assessment currently underway, HBMS has reported water, sediment, and fish tissue residues for 12 lakes in the vicinity of their operations that were likely influenced by both effluent discharges and aerial deposition from smelter stack emissions (Stantec 2009). All 12 lakes sampled met the Health Canada Se drinking water quality guideline of 10 µg/L (Health Canada 1992), and most lakes were below 2 µg/L. However, Schist Lake, which receives effluents from HBMS via Ross Lake and Ross Creek, had Se concentrations of 4 to 5 µg/L (Stantec 2009).

The western provinces of Saskatchewan and Alberta typically have very low Se concentrations in surface waters (< 1 µg/L), although there are some exceptions where Se may be elevated due localized geologic formations or anthropogenic activities. Studies in northern Saskatchewan document elevated total Se concentrations in surface waters below uranium mining operations, where Se concentrations ranged from 0.5 to 7.67 µg/L in exposed lakes and streams downstream, compared with 0.02 to 0.1 µg/L in reference areas (Muscatello *et al.* 2008; Pyle *et al.* 2001).

Data collected under the Federal-Provincial Water Quality Monitoring Agreement (WQMA) from 19 stations on major Alberta rivers between 2005 to 2007 showed the average total Se concentrations was between 0.3 and 0.7 µg/L (MDL = 0.1 µg/L).[8] These concentrations represent background levels, but in some other locations in Alberta anthropogenic sources of Se are known to elevate background concentrations. For example, a report summarizing data collected from the mid 1980s to 2003, documented Se increases below open-pit coal mining areas in Alberta (Casey 2005). Upstream of mining influences, waters had a median Se concentration of 0.7 µg/L, while in closest proximity to the mines levels ranged between 12.7 and 29.2 µg/L Se (Casey 2005). Another study conducted between 1998 and 1999 on Alberta streams influenced by large-scale open-pit coal mining showed that surface water Se concentrations were as high as 48 µg/L (Casey and Siwik 2000).

[8] Data obtained by request through Julie Boyer, Fresh Water Quality Monitoring and Surveillance, Water Science and Technology, Environment Canada, Montreal QC, July 2010.

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The oil and gas industry has also been associated with Se releases to the environment. In 2006, at an oil sand upgrader (a facility that processes crude bitumen from oil sands) near Edmonton, Alberta, waste water effluents with Se concentrations averaging 300 µg/L (range of 150 to 600 µg/L) were found entering the North Saskatchewan River (A.Wolanski, pers. comm., Alberta Environment, February 2010). Since identifying the problem, mitigation measures have reduced Se loadings by 80%, with concentrations in effluent currently in the range of 20 µg/L. In spite of significant reductions in effluents, Se in sediment and biota remain elevated in the near field area (< 50 m downstream) below the discharge (North/South Consultants Inc. 2009).

Background water quality data for BC were obtained from government databases and reports, primarily from the Federal-Provincial WQMA program, for approximately 42 sites across the province which are monitored bi-weekly or monthly.Table 4.6 summarizes Se concentrations at several of these sites. Where data was reported as less than the MDL, the value of the detection limit was used to calculate the mean. These data show that total Se concentrations are typically much less than 1 µg/L, but can be elevated above 1 µg/L in areas where there are natural Se sources from seleniferous rock and/or sources from anthropogenic activities (seeTable 4.6 for site descriptions).

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Table 4.6 Mean (SD) water concentrations of selenium measured in various river systems in BC.

Station Name	Mean Se (SD) (µg/L)	Location / Description (period of record)	Impact[1]	Reference
Moyie River at Kingsgate	0.08 [2] (0.03) (n=98)	Columbia Mtn Highlands ecoregion, some agriculture, logging and historical mining (Pb-Zn-Ag) (1984-2009)	PI	Dessouki (2009d); Environment Canada[4]
Fraser River at Red Pass	0.10 [2] (0.07) (n=385)	Western Continental Range ecoregion, close to the headwaters, no human activity (1984-2004)	R	Swain (2007a); Environment Canada[4]
Flathead River at the International Boundary	0.20 [2] (0.08) (n=376)	Northern Continental Divide ecoregion, fairly pristine wilderness, some logging, mining exploration (1984-2004)	R	Pommen (2005); Environment Canada[4]
Fraser River at Marguerite	0.14 [2] (0.10) (n=383)	Central Interior ecoregion, between Quesnel and Williams Lake BC (1985-2004)	PI	Swain (2007b); Environment Canada[4]
Kettle River	0.15 [2] (0.12) (n=590)	Okanagan Highland ecoregion, southern interior of BC near Midway above US border (1984-2009)	PI	Dessouki (2009a); Environment Canada[4]
Fraser River at Hope	0.16 [2] (0.11) (n=420)	Pacific Coastal Mtns ecoregion in southern BC, east of Vancouver (1984-2004)	PI	Swain (2007c); Environment Canada[4]
Peace River above Alces River	0.37 [2] 0.22 (n=425)	Peace Lowlands ecoregion, north-eastern BC, 45 km upstream of BC-Alberta border (1984-2002)	PI	Phippen (2003a); Environment Canada[4]
Okanagan River at Oliver	0.39 [2] (0.11) (n=404)	Okanagan Highland ecoregion, southern interior of BC near Oliver BC, above US border (1984-2009)	PI	Dessouki (2009b); Environment Canada[4]
Iskut River below Johnson River	0.54 [2] (0.22) (n=130)	Northern Coastal Mtns ecoregion, near confluence with the Stikine R, north central BC (1984-2009)	PI	Dessouki (2009c); Environment Canada[4]
Salmon River near Hyder	1.07 [2] (0.43) (n=573)	Northern Coastal Mtns ecoregion, near BC/Alaska border, mineralized with historical mining (Au, Ag, Cu, Pb, Zn) (1984-2002)	I	Phippen (2003b); Environment Canada[4]
Bear River at Stewart	1.25 [2] (0.53) (n=282)	Northern Coastal Mtns, near Stewart BC, mineralized area with historical mining (Au, Ag, Cu, Pb, Zn) (1984-1994)	I	Webber (1997); Environment Canada[4]
Elk River at Hwy 93 near Elko	2.50 [3] (1.07) (n=456)	Southern Rocky Mtn Trench ecoregion, downstream of large-scale open-pit coal mining (1991-2011)	I	Swain (2007d); Environment Canada[4]
Elk River below Sparwood	5.47 [3] (2.10) (n=179)	Northern Continental Divide ecoregion, close proximity to large-scale open-pit coal mining (2002-2011)	I	Swain (2007e); Environment Canada[4]

[1]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown

[2]No significant trend over the period of record.

[3]Significant increasing trend over the period of record. Se concentrations routinely exceed both CCME and BC guidelines for the protection of aquatic life.

[4]Data used in the calculation of average total Se concentrations was downloaded from Environment Canada's Water Quality web site accessed on-line at http://waterquality.ec.gc.ca/EN/home.htm.

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Swain (2007a; 2007b), confirmed a long-established increasing Se concentration trend at two sites on the Elk River in south eastern BC below large scale open pit coal mining operations. Selenium concentrations at both sites greatly exceed both the CCME and the BC Se guidelines for aquatic life of 1 µg/L and 2 µg/L, respectively (CCME 2007a; Nagpal 2001). Since 2009 at the Elk River monitoring site below Sparwood, monthly water quality samples have occasionally exceeded the CCME and BC drinking water guideline of 10 µg/L (Environment Canada ENVIRODAT[19]).

In contrast, a site on the Flathead River at the International Boundary, an adjacent watershed also sampled under the Canada – BC WQMA, shows no such trend with an average concentration of 0.02 µg/L total Se (Pommen 2005). The Flathead is similar in its lithography and within the same ecoregion as the Elk River, but has not had the same degree of disturbance (Pommen 2005).

4.2.5 Sediment

Sediment Se concentrations in aquatic environments are usually less than 4 µg/g (Cutter 1989) and average background concentrations in the range of 0.2 to 2 µg/g (USDOI 1998). Sediments of undisturbed surface waters typically have Se concentrations at the lower end of the range, about 0.2 µg/g (Luoma and Rainbow 2008). Eisler (1985) reported concentrations between 0.35 and 0.75 µg/g in the Great Lakes, and 0.22 µg/g in Lake George, New York. Marine sediments often contain background Se concentrations ranging from 0.1 µg/g to 2.0 µg/g (Sui and Berman 1989).

Sediment Se concentrations reported in eastern Canada are presented in Table 4.7. In the province of Quebec, anthropogenically enriched lakes (sources of Se not identified) can have sediment Se concentrations as high as 14.5 µg/g (Eisler 1985). In Ontario, sediment profiles collected near the Sudbury smelter had deposition rates of Se in lake sediments estimated to be 0.3 to 12 mg/m^2/year, mirroring smelter production and among the highest recorded in North America at that time (Nriagu and Wong 1983). Most of the reported sediment concentrations for

[9] Data used in the calculation of average total Se concentrations was downloaded from Environment Canada's Water Quality web site accessed on-line at http://waterquality.ec.gc.ca/EN/home.htm.

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relatively undeveloped and unimpacted lakes were below 1 μg/g, even where lakes had historical mining or had been developed for recreational purposes.

Sediment selenium concentrations in western Canada are summarised in Table 4.8. Minor elevations in sediment Se concentrations have been detected in the Northwest Territories at near-field locations below a tungsten mine on the Flat River, but not below a metal-mining operation (i.e., lead, zinc, copper and silver) on Prairie Creek; both are tributaries to the South Nahanni River (Spencer *et al*. 2008). In the Flat River, the upstream (background) concentration of sediment Se was 0.5 μg/g, but at the near-field site was 3.1 μg/g, approximately 6 times that of background. The metal-mine operating on Prairie Creek treated their mine-water portal effluent in polishing and catchment ponds prior to discharge to the receiving environment, which the authors believed could explain why Se was not accumulating in sediments below the mine site (Spencer *et al*. 2008).

In other areas, Se is known to accumulate in sediments, for example below uranium mining operations (Klaverkamp *et al*. 2002; Muscatello *et al*. 2008; Burnett-Seidel and Liber 2012). In a 2005 study of Se contamination below a uranium mine in northern Saskatchewan, Se concentrations in the top 10 cm of lake sediment at a reference site were less than the detection limit (< 0.01 μg/g, n=3) while the Se-exposed sediments had a mean concentration of 0.54 ± 0.01 μg/g (n=3) (Muscatello and Janz 2009a). However, in a study conducted at a different uranium mining area in northern Saskatchewan, Muscatello *et al.* (2008) found that mean lake sediment Se concentrations were 5.7 ± 0.4 μg/g (n=3) at reference sites but increased below uranium mining activities to 25.6 ± 2.9 and 62.2 ± 4.7 μg/g (n=3) at medium and high exposure sites, respectively. The two studies by Muscatello *et al.* (2008; 2009a) had very different reference site sediment Se, showing that area-to-area sediment concentrations may be variable.

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Table 4.7 Summary of sediment selenium values reported in eastern Canadian provinces.

Station Name	Sediment Se (µg/g dw)	Location / Description	Impact[1]	Reference
Nova Scotia				
Kejimkujik Lake	1.12 (± 0.12) (n= 3)	Mean (SD) sediment Se. Oligotrophic lake in southern end of NS	PI	Environment Canada (2012)[10]
East River	All < 1 or 2 (MDL)	East River near Trenton NS, in vicinity of coal-fired power plant ash lagoons	PI	Lalonde *et al.* (2011)[11]
New Brunswick				
Grand Lake	0.19 (± 0.08) (n=3)	Mean (SD) sediment Se for a lake, historical coal mining area east of Fredericton;	PI	Environment Canada (2012)[10]
Grand Lake	≤1 (n=3) 3, (n=1)	Four sites in the vicinity of old coal-fired power plant, one site at the outfall had Se over the MDL;	I	Lalonde *et al.* (2011)
Saint John Harbour	1.61 (± 0.34) (n=21)	Mean (SD) from six sites sampled 2011-12. Of 87 individual replicates, 66 samples were < MDL (1 µg/g).	I	University of New Brunswick (2012)[12]
Ontario				
Winnange Lake	0.56 (n=2)	Mean sediment Se. Undeveloped lake within a Provincial Park.	R	
Harp Lake	0.25 (± 0.25) (n=3)	Mean (SD) sediment Se. Lake in the Muskoka region east of Huntsville ON, cottage development;	PI	Environment Canada (2012)[10]
Mary Lake	0.59 (± 0.11) (n=3)	Mean (SD) sediment Se. Lake in the Muskoka region near Bracebridge ON, cottage development, regulated flow.	PI	
Quebec				
Lac Matagami	1.10 (± 0.12) (n=3)	Mean (SD)sediment Se. Relatively remote but historical metal mining (Cu, Zn, Ag, Au);	I	
Lac Edouard	0.52 (± 0.31) (n=3)	Mean (SD) sediment Se. Relatively remote and unimpacted lake;	R	Environment Canada (2012)[10]
Lac Ouescapis	0.51 (0.22) (n=3)	Mean (SD) sediment Se. Relatively remote and unimpacted lake.	R	

[1]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown

The much higher reference site Se concentration in the earlier study (5.7 ± 0.4 µg/g) may reflect possible mine influence at those sites and/or the different dynamics in accumulation of Se in sediments in those lakes. This underscores the complexity of the behaviour of Se in aquatic environments.

[10] Data obtained from Melissa Gledhill, Environment Canada CARA databases.
[11] Data not reported in publication, obtained from author. MDLs were relatively high = 1 or 2 µg/g dw.
[12] Data obtained by request from Dr. K. Kidd, Professor, Dept. of Biology, University of New Brunswick.

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Burnett-Seidel and Liber (2012) compared sediment Se data collected for environmental monitoring purposes with the lowest- and severe-effect levels for Se (LELs and SELs, respectively) derived by Thompson *et al.* (2005). They found that 75% of reported sediment Se concentrations at reference sites (n=12) were below the 1.9 µg/g LEL concentration, 25% were above the LEL but below the SEL concentrations, and none exceeded the SEL concentration of 16.1 µg/g.

Studies in Alberta show that Se emitted from coal-fired power plants can accumulate in lake sediments (Donahue *et al.* 2006). Approximately 74% of Alberta's electric energy is generated from coal-fired power plants (Statistics Canada 2010). In western Canadian coal-fired power plants, Se had one of the highest rates of emission, at 0.73 kg/day, of 10 elements of concern examined (Goodarzi *et al.* 2006). In a study on Wabamun Lake, Alberta, where four coal-fired power plants operated within a 35 km radius (two of which were located on the shoreline), sediment cores showed that Se had increased 3.4-fold in lake sediments since the 1950s (Donahue *et al.* 2006). These increases coincided with the start-up of coal-fired power plants and other industries in the Wabamun Lake watershed (Donahue *et al.* 2006).

Sediment Se concentrations on the North Saskatchewan River in Alberta, sampled between 2007 and 2009, were approximately 0.5 µg/g (n=3) at reference sites above the Scotford oil-sand upgrader discharge. However, 10 m below the upgrader discharge, sediment Se concentrations were 9.6 µg/g (n=1) (North/South Consultants Inc. 2009).

Selenium has a strong affinity to coal geologies and this is reflected in the wide range of sediment Se concentrations that may be found in coal mining areas, even at unimpacted reference sites (Yudovich and Ketris 2006). For example, whole sediment Se in the Upper McLeod River and Cold Creek basins in Alberta (unimpacted by coal mining) were between 0.3 and 4.8[13] µg/g (Casey 2005).

[13] The range in sediment selenium at reference sites in this Alberta study was 0.3 to 1.7 µg/g (dw) when the highest concentration of 4.8 µg/g (dw) was omitted since the sediment composition of this sample was suspected to contain higher amounts of organic material including small twigs, compared with other samples (Casey 2005).

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Table 4.8 Summary of sediment selenium concentrations at locations sampled in western Canada.

Station Name	Sediment Se (µg/g dw)	Location / Description	Impact[1]	Reference
Yukon Lake Kusawa	0.72 (± (0.38) (n=3)	Mean (SD) sediment Se at an unimpacted lake site in southern Yukon	R	Environment Canada databases (2012)[14]
Northwest Territories Flat River	0.5 (n=1)	Sediment Se at reference site above tungsten mine, South Nahanni River watershed;	R	Spencer *et al.* (2008)
Flat River, Northwest Territories	3.1 (n=1)	Sediment Se concentrations at the near-field site below the tungsten mine.	I	
Saskatchewan Indigo Lake	< 0.01 (MDL) (n=3)	Reference lake upstream of the McClean Lake uranium mine (sampled in 2005)	R	Muscatello and Janz (2009a)
Vulture Lake	0.54 (± 0.01) (n=3)	Exposed site below effluent discharges from the McClean Lake uranium mine (sampled in 2005)	I	
Saskatchewan David Lake	5.7 (± 0.4) (n=3)	Reference site upstream of the Key lake uranium mine (sampled in 2004)	R	Muscatello *et al.* (2008)
Delta Lake	25.6 (± 2.9) (n=3)	Medium exposure site below Key Lake uranium mine (sampled in 2004)	I	
Unknown Lake	62.2 (± 4.7) (n=3)	High exposure site below Key Lake uranium mine (sampled in 2004)	I	
Saskatchewan Granite Lake	0.72 (± 0.38) (n=3)	Mean (SD) sediment Se. Relatively unimpacted lake 40 km west of Creighton (smelter).	PI	Environment Canada databases (2012)
Alberta Upper McLeod River and Cold Creek	0.3 to 4.8 (n=13)	Range of background sediment Se concentrations at reference sites sampled between 1999 and 2000	R	Casey (2005)
Alberta North Saskatchewan River	0.5 (n=3)	Mean background sediment Se measured in 2009 above Scotford oil upgrader	R	North/South Consultants Inc (2009)
	9.6 (n=1)	Sediment Se concentration (2009) immediately downstream of the Scotford oil upgrader	I	
British Columbia Fraser River basin	<0.05 – 0.6	Range of sediment Se at 44 sites sampled within the Fraser River basin 1994-96	R-I	Brewer *et al.* (1998)
British Columbia Murray River, north eastern BC	0.98 (± 0.19) (n=3)	Mean (SD) background sediment Se at three reference sites 2009 and 2011 (samples sieved, < 63 µ fraction analysed)	R	BC MoE data files
British Columbia Elk River watershed, south eastern BC	1.5 (± 1.4) <0.5 to 3.1 (n=3)	Mean (±SD) and range of background sediment Se concentrations at lentic reference sites in 2009;	R	Minnow *et al.* (2011)

[1]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown

[14] Data obtained by request from Melissa Gledhill, Environment Canada CARA databases.

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Minnow *et al.* (2011) reported whole sediment Se concentrations collected in 2009 from the top 2 cm at lentic reference sites in the Elk River, BC were between < 0.5 and 3.1 µg/g (mean 1.5 ± 1.4, n=3). However, at coal mining impacted sites in the Elk River, sediment Se concentrations measured at lentic sites in 2009 were between 2.2 and 20.5 µg/g (mean 10.4 ± 7.7, n=4).

Brewer *et al.* (1998) reported bed sediment Se concentrations for 44 individual sampling sites representing 15 reaches of the Fraser River basin in BC. The reaches were categorized into reference (little or no potential human impact) and non-reference (some degree of influence from one or more land-use activities). Based on data collected between 1994 and 1996, sediment Se concentrations ranged between < MDL (0.05) at more pristine sites, and 0.6 µg/g at sites on the Quesnel River (mid-basin tributary) and sites within the North Arm of the Fraser (lower basin).

On the Murray River in north eastern BC, where the < 63 µm fraction was analysed to reduce variability, background sediment Se concentrations at three reference sites (n=3 at each site) were ≤ 1 µg/g (± 0.19) (K. Przeczek, pers. comm., BC Ministry of Environment, March 2012).

4.2.6 Aquatic Biota

4.2.6.1 Microbes and Algae

Bacteria, fungi, and plants including attached (periphyton) and planktonic algae, easily take up waterborne Se, which is the first important step in Se accumulation and transfer to higher trophic levels in both freshwater and marine environments (Lemly 1993a; Luoma and Presser 2009; Maher *et al.* 2010). Since these microorganisms play a key role in Se biotransformation, subsequent bioaccumulation and potential toxicity higher up the food web, knowing Se concentrations in this environmental compartment is an important indicator of potential toxic effects (Swift 2002; Luoma and Rainbow 2008).

With the exception of vascular plants, Se is thought to be an essential micronutrient for most living organisms, from microorganisms to large mammals (Ellis and Salt 2003). Studies have shown that at least some aquatic algae require Se for growth (e.g., marine planktonic species of diatoms, dinoflagellates, prymnesiophytes, and a cyanophyte) (Harrison *et al.* 1988). Selenium from the water column is actively taken up and concentrated in these small aquatic plants;

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relatively small increases in available waterborne Se may cause large increases in Se concentrations in these organisms (Lemly 1993a).

The term *biofilm* was first used in the early 1980s and refers to the thin layer of microorganisms made up of bacteria, fungi, and attached algae and found on rocks and on the water-sediment interface (Casey and Siwik 2000; Casey 2005). The term periphyton also describes the assemblage of microbes, algae, and detritus found on rocks, lake bottoms (euphotic zone), and streambeds. Periphyton or biofilm has been referred to as *epilithon*, biofilm found on rocks in moving water (lotic environments), or *epipelon,* biofilm found on sediment surfaces in slower moving water depositional (lentic) environments (Wetzel 2001; Orr *et al.* 2006, 2012). A summary of Se concentrations in algae (plankton) and biofilm (periphyton) from western Canada is presented in Table 4.9.

The overall range of Se concentrations reported for marine and freshwater periphyton is from 0.4 to approximately 142 µg/g (Cutter 1989; Sui and Berman 1989). Below a uranium mine in northern Saskatchewan, plankton samples from reference areas had Se concentrations of 1.11 ± 0.11 µg/g while samples from medium and high exposure sites were 5.01 ± 0.70 and 7.21 ± 1.43 µg/g, respectively (Muscatello *et al.* 2008). Periphyton samples analysed in the same study had background Se concentrations of 0.29 ± 0.05 µg/g, and at medium and high Se exposure sites concentrations were 1.10 ± 0.26 and 3.75 ± 0.64 µg/g, respectively (Muscatello *et al.* 2008). In a study conducted in southern Alberta, rocks were scraped to obtain biofilm in reference and coal mine-affected streams (Casey 2005). The Se concentrations in biofilm at reference or minimally impacted sites were less than 1.0 µg/g, and as high as 4.0 µg/g in impacted areas (Casey 2005).

In 1996, McDonald and Strosher (2000) conducted a study on the Elk River in BC near coal mining activities and found that the Se concentration in attached algae at a reference site (0.31 µg/g) was one fifth that measured at a location just downstream of mine discharges (1.56 µg/g). In a more recent study on the Elk River, Se concentrations of biofilm established on introduced substrates (three slides analysed per site) at two lentic reference sites were 0.4 and 1.9 µg/g (Minnow *et al.* 2011). In the same study at coal mine-exposed lentic sites, the range of Se in biofilm samples were between 7.5 and 51.0 µg/g (dw).

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Table 4.9 Summary of selenium tissue concentrations in planktonic algae and biofilm from sites in western Canada.

Station Name	Se Conc. (µg/g dw)	Location / Description	Impact[1]	Reference
David Lake, north-central Saskatchewan	1.11 ± 0.11 (n=3)	**Plankton** Se concentrations from a reference lake upstream of Key Lake uranium mine, Saskatchewan	R	Muscatello *et al.* (2008)
	5.01 ± 0.70 (n=3)	**Plankton** Se from medium exposure site	I	
	7.21 ± 1.43 (n=3)	**Plankton** Se from high exposure site	I	
David Lake, north-central Saskatchewan	0.29 ± 0.05 (n=3)	**Periphyton** Se concentrations from a reference lake upstream of Key Lake uranium mine, Saskatchewan	R	
	1.10 ± 0.26 (n=3)	**Periphyton** Se concentrations from medium exposure site	I	
	3.75 ± 0.64 (n=3)	**Periphyton** Se concentrations from high exposure site	I	
Deerlick Creek, Alberta	1.0 (0.7-1.4) (n=3)	Mean (range) of **biofilm** Se concentrations in a reference stream above coal mining activities	R	Casey (2005)
Luscar Creek, Alberta	3.2 (2.3-4.0) (n=4)	Mean (range) of **biofilm** Se concentrations in an exposed stream below coal mining activities	I	
Elk River watershed, BC	0.3 (n=1)	**Periphyton** Se from a reference site collected in 1996 above open-pit coal mining activities	R	McDonald and Strosher (2000)
Elk River watershed, BC	0.8 to 1.6 (n=5)	Range of **periphyton** Se in samples collected in 1996 from exposed sites below open-pit coal mining activities	I	
Elk River watershed, BC	0.4 and 1.9 (n=2)	**Biofilm** Se concentrations from introduced substrates deployed in 2009 at 2 lentic reference sites	R	Minnow *et al.* (2011)
Elk River watershed, BC	7.5 to 51.0 (n=4)	Range of **biofilm** Se concentrations from introduced substrates deployed in 2009 at 4 lentic exposed sites	I	

[1]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown

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4.2.6.2 Macrophytes

Background Se in aquatic macrophytes is typically between 0.1 and 2.0 µg/g (USDOI 1998). Sandholm *et al.* (1973) found that aquatic plants had low background Se concentrations, ranging from 0.02 to 0.14 µg/g. This is consistent with other estimates of background Se in aquatic macrophytes from natural waters, ranging between 0.04 and 0.24 µg/g (Eisler 1985), and generally less than 0.60 µg/g (Maier *et al.* 1998). Edible seaweed is reported to have Se concentrations of between 0.16 and 0.39 µg/g (Eisler 1985).

Studies show that when additional Se is introduced into a stream, macrophytes are very effective in bioaccumulating Se, with increases in concentration occurring rapidly, sometimes within weeks (Ohlendorf *et al.* 1986; Maier *et al.* 1998). In studies conducted at the Kesterson National Wildlife Refuge in California, the mean Se concentration of rooted plants from a control area was 0.43 µg/g, compared with areas contaminated by agricultural drainwater, where concentrations were between 18 and 79 µg/g, a 120-fold increase (Ohlendorf *et al.* 1986). As Kesterson continued to be impacted by inputs from agricultural drainwater, the mean concentration of Se in aquatic macrophytes sampled in evaporation ponds in the Kesterson study area reached 273 µg/g (Presser and Ohlendorf 1987; Presser *et al.* 1994).

Along with microorganisms (biofilm and plankton), macrophytes can be an important source of Se in the food webs of some organisms, particularly herbivorous invertebrates and some water fowl (Stewart *et al.* 2010). Therefore, macrophytes also play a key role in the biotransformation and bioaccumulation of Se in the transfer of Se up the aquatic food web (Bowie *et al.* 1996; Luoma and Presser 2009; Stewart *et al.* 2010).

There are few measurements of Se content in aquatic macrophytes from Canadian waters. Casey (2005) sampled filamentous algae, aquatic macrophytes (unidentified), moss, *Potamogeton* sp., and reedgrass (*Sparagium* sp.) in Alberta between 1999 and 2000. Only filamentous algae and unidentified macrophytes were sampled at locations that were clearly above and below coal mining areas. In the reference locations, individual Se concentrations in samples of filamentous algae and macrophytes were 0.3 and 1.3 µg/g, respectively, while at the exposed locations concentrations were 5.5 and 17 µg/g, respectively (Casey 2005). At two coal mine-exposed sites in the Elk Valley BC in 2002 and 2003, the range of Se concentrations in individual macrophyte

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samples, which included *Carex* sp., *Equisetum* sp., and *Typha* sp., were between 3.9 and 12.3 µg/g (n=5) (Minnow Environmental Inc. 2004).

4.2.6.3 Invertebrates

The background range of Se in aquatic invertebrates sampled from uncontaminated water is between 0.5 and 1.5 µg/g (Eisler 1985). However, Se in top predators within the same habitats may differ greatly, reflecting the variability of Se bioaccumulation in various prey species (Luoma and Rainbow 2008). For example, Se concentrations in the benthic clam *Corbula amurensis* were tracked for approximately 15 years in the San Francisco Bay area; Se concentrations ranged between 2 and 22 µg/g in areas minimally to strongly impacted from refinery discharges (Kleckner *et al.* 2010; Luoma and Rainbow 2008). Selenium concentration data from some Canadian studies are summarized in Table 4.10.

Jardine and Kidd (2011) collected stream invertebrates in New Brunswick at 49 locations between 2006 and 2007. The taxa at each site varied, including mixtures of primary consumers (Pteronarcyidae, Hydropsychidae, and freshwater mussels) and predators (Gomphidae, Aeshnidae, Gerridae, Cordulegastridae, Megaloptera, and Perlidae). The mean Se concentrations of invertebrate tissues at 39 of the sites ranged from 0.7 to 3.5 µg/g. Most of the 39 sites (72%) had tissue Se concentrations between 1 and 2 µg/g, reflecting the effects of local geology and suggesting that Se enrichment is rare in New Brunswick (Jardine and Kidd 2011).

Near uranium mining operations in Saskatchewan detritivore, filter-feeders, and predator invertebrate taxa (in all cases n=3–5) had mean background tissue Se concentrations of 0.93 ± 0.22, 2.01 ± 1.11, and 1.23 ± 0.43 µg/g, respectively (Muscatello *et al.* 2008). In almost all cases, the mean Se concentration in these invertebrate groups at medium and high exposure sites were significantly higher, with the exception of filter-feeders at the medium exposure sites (Muscatello *et al.* 2008). Detritivores at medium and high exposure sites had mean Se concentrations of 12.39 ± 4.87 and 25.12 ± 7.07 µg/g, respectively, which were approximately 13 and 25 times background concentrations (Muscatello *et al.* 2008). A study by Weech *et al.* (2011), in the same area in northern Saskatchewan, found mean (SD) Se concentrations of invertebrate tissues at reference sites were 0.86 (± 0.05, n=19) µg/g.

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Table 4.10 Summary of invertebrate and zooplankton tissue selenium concentrations from Canadian studies.

Station Name	Se Conc. (µg/g dw)	Location / Description	Impact	Reference
New Brunswick	0.7 (± 0.2) to 3.5 (±1.09)	Range of mean (SD) Se in **mixed-taxa invertebrate** tissues collected from 49 streams across New Brunswick (n=5).	R - I	Jardine and Kidd 2011
David Lake, Saskatchewan (reference)	0.9 (± 0.2) 1.23 (± 0.4) 2.0 (± 1.1)	Mean (SE) Se in **detritivores** (n=5) Mean (SE) Se in **predators** (n=3 – 5) Mean (SE) Se in **filter-feeders** (n=5)	R	Muscatello *et al.* (2008)
Delta Lake, Saskatchewan	12.4 (±4.9) 12.7 (± 0.9) Not reported	Mean (SE) Se in detritivores (n=5) Mean (SE) Se in predator (n=3 – 5) Mean (SE) Se in filter-feeders (n=5)	I (medium exposure)	
Unknown Lake, Saskatchewan	25.1 (± 7.1) 16.0 (± 2.1) 6.0 (± 0.9)	Mean (SE) Se in **detritivores** (n=5) Mean (SE) Se in **predators** (n=3 – 5) Mean (SE) Se in **filter-feeders** (n=5)	I (high exposure)	
Key Lake area, north Saskatchewan	0.9 (± 0.05) (n=19)	Mean (SD) Se of mixed aquatic **invertebrates** from reference marsh.	R	Weech *et al.* (2011)
Deerlick & Cold creeks, Alberta	1.5 to 7.3 (4.5) (n=4)	Range and (mean) Se in **invertebrate** samples at reference sites above coal mining activities	R	Casey (2005)[2]
Luscar Creek, Alberta	6.3 to 15.0 (10.0) (n=3)	Range and (mean) Se in **invertebrate** samples at exposed sites below coal mining activities	I	
Mt Polley Mine, Williams Lake, BC	0.6 (± 0.1) to 2.5 (± 0.7)	Mean (SD) Se in **invertebrates** from four reference lakes near Mt. Polly Mine (n=5 at each site)	R	Minnow Environmental Inc.(2013)
Elk River, BC	2.74; 6.84 (n=2)	**Invertebrate** Se in samples at two reference sites above open-pit coal mining	R	McDonald and Strosher (2000)
Elk River, BC	6.82 to 10.7 (n=5)	Range of **invertebrate** Se in samples from exposed sites below open-pit coal mining	I	
Elk River watershed, BC	4.4 (± 1.6) (n=4)	Mean (SD) of composite **invertebrate** samples from Boivin, Gold and Lynx (2) creeks (assuming 75% moisture)	R	Harding and Paton (2003)
Elk River BC	3.9 (± 1.6) (n=32)	Geometric mean (SD) Se in **invertebrate** samples from lotic and lentic sites reference sites, 1996-2009	R	Calculated using data provided by Minnow Environmental Inc.
Lake Koocanusa, south eastern BC	2.9 (± 0.3) (n=5)	Mean (SD) of replicate **zooplankton** samples collected at a reference site above coal mining inputs	R	McDonald (2009)

[1]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown

[2] Mean of Se concentrations measured in mayflies (Baetidae, Heptageniidae and Ephemerellidae), stoneflies (Perlodidae, Perlidae and Chloroperlidae), caddisflies (Hydropsychidae and Rhyacophila) and the dipteran Tipulidae (Casey 2005).

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In a study, conducted between 1999 and 2001 at coal mining areas in Alberta, mean benthic invertebrate tissue Se concentrations were 4.5 µg/g at reference areas, and 10.0 µg/g[15] at mine-exposed areas (Casey 2005). Wayland *et al.* (2006) and Wayland and Crosley (2006) collected benthic invertebrate Se data from many of the same areas as Casey (2005) which would account for the tissue Se residue data from these studies being very similar. A study by Wayland *et al.* (2007) represents the combined data set.

Minnow Environmental Inc. (2013) collected benthic invertebrates using a ponar from four reference lakes in the vicinity of Mount Polley Mine north-east of Williams Lake, BC. The mean (SD) Se concentrations in invertebrate tissues ranged from 0.6 (± 0.1) to 2.5 (0.7), (n=5 at each site). In 1996, McDonald and Strosher (2000) collected benthic invertebrate samples in the Elk River BC, containing a mixture of taxa; predominantly Perlodid stoneflies and/or Hydropsychid caddisflies, as well as mayflies and stoneflies. Selenium concentrations of composite samples of benthic invertebrates collected at two references sites were 2.74 and 6.84 µg/g, while at three sites downstream of coal mining activities Se concentrations were between 6.82 and 10.7 µg/g (McDonald and Strosher 2000).Benthic invertebrate tissue Se data from reference sites sampled by Minnow Environmental Inc., including the data collected by McDonald and Strosher (2000), were used to estimate an overall geometric mean (SD) background Se concentration for the Elk River basin of 3.9 (1.6) µg/g (n=32). McDonald (2009) collected zooplankton samples in Lake Koocanusa reservoir from a reference area above inputs from coal mining and found mean (SD) Se concentrations were 2.9 (± 0.3, n=5) µg/g.

4.2.6.4 Vertebrates

Tissue Se concentrations vary in vertebrates depending on species, tissue types (whole-body, muscle, liver or egg/ovary), and other factors. Oviparous (egg-laying) vertebrate species, particularly fish and birds, are taxa most at risk to Se toxicity. While reptiles and amphibians may also be sensitive to the effects of Se, their relative sensitivity to Se is less certain (Janz *et al.* 2010). Therefore, most of the focus in this section was on fish and birds, and other oviparous wildlife where data were available.

[15] Reported as the median of mean concentrations of secondary and tertiary consumer invertebrate taxa.

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4.2.6.5 Fish

The US Department of the Interior's (US DOI) National Irrigation Water Quality Program (USDOI 1998) summarised fish tissue data from US and global sources. At sites not influenced by Se contamination, the average whole-body Se concentration in fish was between 1.6 and 2.4 µg/g. In controlled feeding studies where dietary Se was no greater than 2 µg/g, whole-body Se in fish was < 2 µg/g, and mean muscle, gonad, and egg Se concentrations were between < 2 and 4 µg/g (USDOI 1998). Eisler (1985) reported Se in freshwater whole-body fish tissues similar to the range reported by US DOI (1998) but noted that marine fish typically had higher Se concentrations than freshwater. The differences were not great, ranging from similar, to less than an order of magnitude (Eisler 1985).

DeForest (2009) recently summarized available fish tissue Se data at reference sites to define a typical background concentration (Figure 4.1). The 50th and 90th percentile Se concentrations were 2.9 and 6.8 µg/g for whole body (n=902), 1.6 and 4.8 µg/g for muscle (n=403), 8.6 and 15.2 µg/g for eggs (n=52), and 9.4 and 24.0 µg/g for ovaries (n=47), respectively (DeForest 2009).The author noted that it was not possible to verify in all cases that the data represented fish which had not been previously exposed to Se from either anthropogenic activity or as a result of fish moving in and out of contaminated areas (DeForest 2009). Fish movement, in particular those species that move over great distances, can influence exposure to Se and complicate exposure assessment (Stewart *et al*. 2010).

Selenium concentrations in fish from Canadian freshwaters show background tissue concentrations are comparable across the country, with some exceptions (Table 4.11). Mean Se concentrations for brook trout (*Salvelinus fontinalis*) in New Brunswick were fairly low, ranging between 0.6 µg/g (n=1), to 2.6 (± 0.4) µg/g, n=11). The low concentrations in New Brunswick brook trout reflect the geology of the area.

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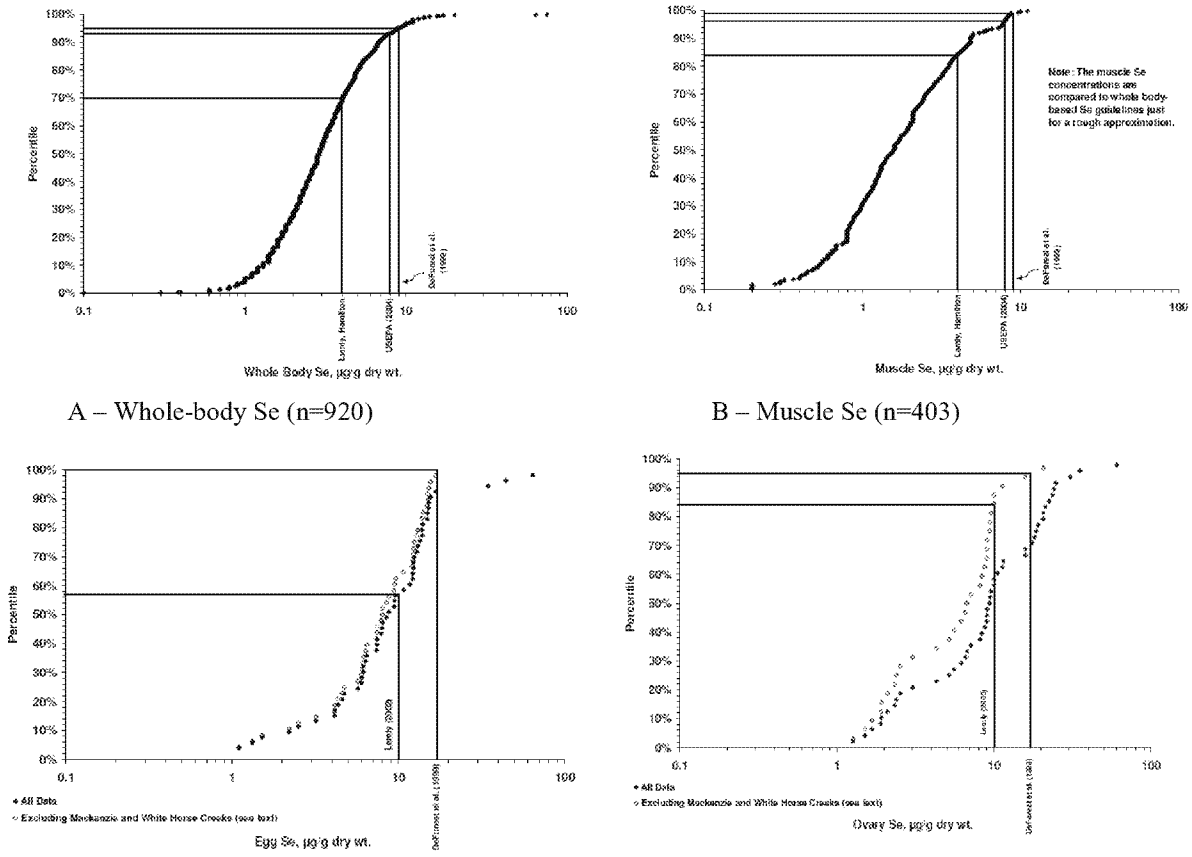

A – Whole-body Se (n=920)

B – Muscle Se (n=403)

C – Egg Se (n=52)

D – Ovary Se (n=47)

Figure 4.1 Cumulative distribution of tissue selenium concentrations in fish from reference sites in the US and Canada; A whole-body, B muscle, C egg, and D ovary tissues (from DeForest 2009, reprinted with permission from North American Metals Council).

Environment Canada's National Contaminants Monitoring and Surveillance Program (NCMSP) has collected fish tissue data at many sites across Canada for several species, including lake trout (*Salvelinus namaycush)* and walleye (*Sander vitreus*)[16]. Unpublished data from several of these sites for walleye in Quebec and lake trout in Ontario, two species representing the top predators most likely to accumulate Se, are presented in Table 4.11.

[16] Data obtained by request through Daryl McGoldrick, Canadian Centre for Inland Waters, Environment Canada, Burlington ON, August 2012.

Table 4.11 Summary of selenium in fish tissues for monitoring sites in eastern Canada (data obtained from Environment Canada, converted from ww to dw assuming 75% moisture content[1]).

Sampling Location	Se (µg/g dw)	Fish Species / Description	Impact[2]	Reference
New Brunswick 39 streams across New Brunswick	0.6 (n=1) to 2.6 (± 0.4) (n=11)	Range of mean Se (±SD) measured in **brook trout** tissues from 39 streams across New Brunswick.	R - I	Jardine and Kidd 2011
Quebec St Lawrence River at St. Nicolas	2.04 (± 0.17)	Mean (±SD) whole-body Se in **walleye** (n = 10) sampled in 2009;	I	Environment Canada (2012)[17]
Lac Matagami	2.96	Mean muscle Se in **walleye** (n = 2) sampled in 2009;	U	
Lac Ouescapis	1.84 (± 0.15)	Mean (±SD) muscle Se in **walleye** (n = 8) sampled in 2010	U	
Quebec Lac Édouard	1.26 (± 0.37)	Mean (±SD) muscle Se in **lake trout** (n = 10) sampled in 2009	I	Environment Canada (2012)[17]
	1.21 (± 0.10)	Mean (±SD) muscle Se in **lake trout** (n = 10) sampled in 2010	I	
Ontario Lake Huron (Georgian Bay)	3.63 (± 0.49)	Mean (±SD) whole-body Se in **lake trout** (n = 10) sampled in 2010-11, Cape Rich site on Georgian Bay	PI	Environment Canada (2012)[17]
Lake Ontario (eastern basin)	2.46 (± 0.33)	Mean (±SD) whole-body Se in **lake trout** (n = 29) sampled in 2009, 10 & 11 in the eastern basin of Lake Ontario;	PI	
Lake Superior (nr Pie Island)	1.94 (± 0.34)	Mean (±SD) whole-body Se in **lake trout** (n = 36) sampled in 2009 and 2011, Pie Island - Thunder Bay station;	PI	
Lake Erie (eastern basin)	2.00 (± 0.28)	Mean (±SD) whole-body Se in **lake trout** (n = 41) sampled in 2009, 10 & 11 in the eastern basin of Lake Erie - Dunkirk station;	I	

[1]Converted from wet weight to dry weight using a generic 75% moisture content as reported in Lemly (2002a).
[2]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown

At Quebec sites, the mean fish muscle tissue concentrations for Se for both walleye and lake trout do not exceed 3 µg/g. Walleye data from 2009 and 2010 at three sites in Quebec shows that

[17] Data provided by Daryl McGoldrick, Canadian Centre for Inland Waters, Environment Canada, Burlington ON, August 2012.

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the most pristine site at Lac Ouescapis has lower mean Se in walleye muscle tissues (1.84 µg/g, n = 8), than do either of the two sites which possibly have some impacts from human activity; the St. Lawrence River at Nicholas (2.04 µg/g, n = 10) and Lac Matagami (2.96 µg/g, n = 2). Lake trout data collected at a sampling site on Lac Édouard in Quebec in 2009 and 2010, showed that mean muscle Se concentrations were 1.26 and 1.21 µg/g (n=10), respectively.

At four sites on the Great Lakes in Ontario, mean lake trout muscle tissue residues were between 1.94 and 3.63 µg/g Se (n between 10 and 41, see Table 4.11). This range of Se in fish tissues likely reflects the amount of human influence at each of the sites, but the tissue Se concentrations remain fairly low.

In western Canada, Se has been measured in fish tissues at many locations (Table 4.12). For example, in the Yukon River, Alaska, northern pike (*Esox lucius*), longnose sucker (*Catastomus catastomus*) and burbot (*Lota lota*), had whole-body composite Se concentrations ranging from 0.92 to 3.4 µg/g with only one burbot sample having concentrations in excess of the literature-based risk threshold for protection of piscivorous wildlife used by the authors (3.0 µg/g Se) (Hinck *et al.* 2006). Harrison and Klaverkamp (1990) conducted lake studies in Manitoba and Saskatchewan, and reported mean Se concentrations for fish in lakes grouped into three distances from a smelter emission in Flin Flon, Manitoba. In lakes furthest from the smelter, mean muscle and liver Se concentrations for northern pike were 1.21 and 8.38 µg/g [18] respectively, and 1.17 and 4.05 µg/g for white suckers (*Catostomus commersoni*) (Harrison and Klaverkamp 1990).

Studies on the North Saskatchewan River in the vicinity of the Scotford oil sand upgrader facility in Alberta, showed that average whole-body tissue Se concentrations in longnose dace (*Rhinichthys cataractae*) captured at two reference areas were highly variable over a three year monitoring program, ranging from a minimum of 1.12 µg/g to a maximum of 5.39 µg/g[19] (North/South Consultants Inc. 2009).

[18] Converted from wet weight to dry weight using 79% and 76% moisture content for liver and muscle respectively as reported in Harrison and Klaverkamp 1990.
[19] Converted from wet weight to dry weight assuming 75% moisture content.

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Table 4.12 Summary of Se concentrations in various fish species and tissue types from western Canada.

Sampling Location	Se (µg/g dw)	Fish Species / Description	Impact[1]	Reference
Yukon River Basin, Yukon Territory	0.92 to 3.4[2]	Range of whole-body Se in **northern pike** (n=19), **longnose sucker** (n=9), and **burbot** (n=3) sampled within the basin	PI	Hinck *et al.* (2006)
Four lakes in Saskatchewan	1.21 (0.42) 1.17 (0.50)	Mean (SD) muscle Se in **northern pike** Mean (SD) muscle Se in **white sucker** (lakes upwind of smelter emissions)	R	Harrison and Klaverkamp (1990)
Five lakes near Flin Flon, Manitoba	3.67 (1.58) 4.38 (2.04)	Mean (SD) muscle Se in **northern pike** Mean (SD) muscle Se **white sucker** (lakes in close proximity to smelter)	I	
North Saskatchewan River, Alberta	1.1 (0.8) to 5.4 (1.0) (n=8)	Range of mean (SD) Se in whole-body tissue of **longnose dace** at 5 reference sites, 2007 – 2009	R, PI	North/South Consultants Inc. (2009)
Deerlick Creek, Alberta	8.96 (± 1.02)[3] (n=20)	Mean (± SE) egg Se in **rainbow trout** at a reference site above coal mining	R	Holm *et al.* (2005)
Luscar Creek , Alberta	25.34 (± 3.58)[3] (n=22)	Mean (± SE) egg Se in **rainbow trout** sampled below coal mining activities	I	
Cold Creek, Alberta	3.33 (± 0.26)[3] (n=22)	Mean (± SE) egg Se in **brook trout** at a reference site above coal mining	R	
Luscar Creek, Alberta	19.97 (± 1.79)[3] (n=30)	Mean (± SE) egg Se in **brook trout** sampled below coal mining activities	I	
North-eastern BC watersheds	1.6 to 7.9 (n=178)	Range of whole-body Se in **slimy sculpin** at reference sites inside and outside coal mining areas	R	Carmichael and Chapman (2006)
Elk River, south-eastern BC	7.59 (±1.88) (n=42)	Mean (± SE) ovary Se for **westslope cutthroat trout** in lotic reference or minimally impacted sites (1998 to 2009)	R, PI	Minnow *et al.* (2011)
	19.52 (lotic) 92.43 (lentic)	Highest individual ripe ovary Se measurements for exposed sites	I	
	4 (3.0 – 4.6) (n=10)	Mean Se (range) in muscle in westslope cutthroat trout	R	McDonald and Strosher (1998)
Flathead River, south-eastern BC	1.29 (0.28) (n=20)	Mean (SD) muscle Se in **westslope cutthroat trout** at lotic reference sites (2006)	R, PI	Henderson and Fisher (2012)
	7.04 (1.8) (n=22)	Mean (SD) whole-body Se in **slimy sculpin** sampled in 2006.		
Blind Creek, north-eastern BC	3.4 (0.5) (n=5)	Mean (SD) Se in whole-body juvenile **rainbow trout** sampled pre-coal mining development (2004), Brule Mine	R	Golder (2009)
	7.1 (1.8) (n=8)	Mean (SD) Se in whole-body juvenile **rainbow trout** sampled after coal mining development (2008), Brule Mine	I	

[1]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown

[2] Converted from wet weight to dry weight using 75% moisture content (Lemly 2002a).

[3] Converted from wet weight to dry weight using 61% moisture as reported in Holm *et al.* 2005.

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Mean Se concentration in rainbow trout (*Oncorhynchus mykiss*) egg from a reference and a mine-affected stream in western Alberta were 8.96 (± 1.02) µg/g (n=20) and 25.4 (± 3.58) µg/g (n=22) respectively[20]. In the same study, brook trout mean egg Se concentrations were 3.33 (± 0.26) µg/g (n=22) at the reference site and 19.97 (± 1.79) µg/g (n=30) at the mine-affected site (Holm *et al.* 2005). The reason for differences between these two species of salmonids in the uptake and compartmentalisation of Se in tissues was unclear but underscored the need for further site- and species-specific research (Holm *et al.* 2005). At Blind Creek in north eastern BC, mean (SD) whole-body Se concentrations in juvenile rainbow trout increased from 3.4 (0.5) µg/g prior to mining, to 7.1 (1.8) µg/g four years after mining commenced (Golder 2009).

Carmichael and Chapman (2006) carried out studies in north-eastern BC, comparing whole-body slimy sculpin (*Cottus cognatus*) tissue Se concentrations from reference sites within and outside of coal mining geology zones. Mean whole-body Se concentrations in sculpin from reference streams inside the active coal mining zone were significantly higher than those outside the zone (Carmichael and Chapman 2006). Although sculpin collected at reference sites outside the coal zone were assumed to be uninfluenced, some tissue Se concentrations were slightly elevated, exceeding the 2001 BC WQG for Se, but all were below the US EPA criteria (Carmichael and Chapman 2006). Elevated Se in sculpin tissue at reference sites inside and outside the coal mining zone was thought to be related to the influence of natural coal deposits present in the area, or due to small sample sizes or the coal-field boundaries used (Carmichael and Chapman 2006). Henderson and Fisher (2012) collected sculpin in the Flathead River in south eastern BC, in 2006 at sites considered relatively pristine and found that mean (SD) whole-body Se tissue concentrations were 7.04 (1.8) µg/g (n=22). At those same sites, Se measured in westslope cutthroat trout muscle tissue (similar to whole-body concentrations) were 1.29 (0.28) µg/g (n=20).

Spencer *et al.* (2008) investigated the effects of mining activities on the Flat River and Prairie Creek in the Northwest Territories. They reported mean muscle tissue Se concentrations in sculpin (equal numbers of male and female fish, n=40) at two reference sites were 3.28 and 5.0

[20] Converted from wet weight to dry weight assuming 61% moisture content.

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µg/g.[21] However, at the near- and far-field sites, mean muscle Se concentrations were comparable to or lower than that at reference sites. Other studies conducted on sculpin have found similar elevations in natural background whole-body Se tissue concentrations (Hamilton and Buhl 2003; EDI 2009). The relevance of elevated tissue Se concentrations in sculpin is unknown since there are currently no published Se toxicity thresholds for sculpin, but these data illustrate the species-specific nature of Se accumulation.

Based on studies in the Elk River, BC between 1998 and 2009, Minnow *et al.* (2011) reported mean Se concentrations at lotic reference or minimally impacted sites in whole-body, muscle, and ripe ovary tissue of westslope cutthroat trout (*O. clarkii lewisi*) were 5.2 (±1.0) (n=9), 4.6 (±0.8) (n=42) and 7.6 (±1.9) (n=15) µg/g, respectively. Individual muscle tissue residues of fish captured in 2009 from areas exposed to coal mining effluents were as high as 19.5 µg/g at lotic sites and 92.4 µg/g at lentic sites (Minnow *et al.* 2011). The differences in bioaccumulation dynamics between lotic and lentic waters were evident in these data, and are discussed further in Section 5.

These data indicate that slight elevations in tissue Se may be apparent at reference sites near coal geologies where large-scale mining is occurring, compared with other reported background Se concentrations (e.g., the Flathead River). This provides evidence that background conditions may vary and/or fish movement may obscure true background. Site selection should be carefully considered in a monitoring and regulatory framework. Similarly, some species such as sculpin, may accumulate Se more efficiently that others even in relatively pristine conditions. This underscores the need to affirm that reference site fish are not exposed to anthropogenic Se sources and/or development of site- or species-specific Se tissue guidelines (objectives) for some areas or species may be necessary. This is discussed further in Section 8.4.

Not all fish tissue data located was included in this document. Other BC fish tissue data collected as part of the Fraser River Action Plan, not summarised in Table 4.12, exist for peamouth chub (*Mylocheilus caurinus*), mountain whitefish (*Prosopium williamsoni*) and starry flounder

[21] Converted from wet weight to dry weight assuming 75% moisture content

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(*Platichthys stellatus*). In general, the concentrations reported by these authors for Se in muscle tissues were less than 2.4 µg/g in all three species tested (Raymond *et al.* 2001)[22].

4.2.6.6 Birds

Selenium in birds is commonly measured in feathers, blood, hepatic tissue (liver/kidney), and eggs; Se is rarely measured on a whole-body or carcass basis in birds (USDOI 1998). Some whole-body/carcass bird Se concentrations have been reported by Eisler (1985) include 0.4 to 2.0 µg/g in little green heron (*Butorides virescens*), 2.1 µg/g in blackbird (*Turdus merula*), and 0.6 µg/g in both house sparrow (*Passer domesticus*) and pheasant (family Phasianidae).[23] White *et al.* (1977, cited in Skorupa *et al.* 1996) conducted a monitoring program in 1973, sampling 51 sites across the US and reported an average carcass Se (feet, legs, feathers and beaks removed) of < 2 µg/g in starlings (*Sturnus vulgaris*). Wells *et al.* (1977) reported a background Se carcass concentration of 1.3 µg/g (assuming 65% moisture content) from a composite of five black-necked stilts (*Himantopus mexicanus*) collected in Texas.

Feathers from birds in reference areas usually have between 1 and 4 µg/g of Se, while whole blood typically contains between 0.1 and 0.4 mg/L Se (USDOI 1998). Selenium concentrations in the liver and kidney tissues of birds are similar, with reference Se concentrations generally < 10 µg/g (USDOI 1998). Avian liver tissue Se concentrations reported for background locations ranged from 2.0 – 4.3 µg/g in American coots (*Fulica americana*), 5.2 – 9.5 µg/g in dabbling ducks (family Anatidae), to 6.0 – 9.9 µg/g in stilts and avocets (Recurvirostridae) (Skorupa *et al.* 1996).

The most direct means of determining the potential for toxic effects of Se in birds is through measurement of egg Se concentrations (Adams *et al.* 1998; Fairbrother *et al.* 1999; Heinz 1996). In areas without Se contamination, typical concentrations of Se reported in bird eggs were < 5 µg/g (USDOI 1998; Skorupa *et al.* 1996; Ohlendorf *et al.* 1986). In Se-contaminated areas, birds nesting near ponds collecting agricultural drainage waters within the Kesterson Wildlife Refuge were found to have egg Se concentrations up to 20 times higher than eggs from reference areas

[22] Converted from wet weight to dry weight assuming 75% moisture content.
[23] Unable to ascertain if these were average or individual values.

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(Ohlendorf *et al.* 1986). Many eggs from contaminated areas had concentrations in excess of 40 µg/g Se, and were associated with higher incidences of embryonic mortality, hatchling mortality, and severe developmental abnormalities (Ohlendorf *et al.* 1986).

Much of the Canadian data on bird Se tissue concentrations, some of which is summarized in Table 4.13, originate from western Canada. Outridge *et al.* (1999) reviewed Se data for several species of birds in Canada and found that seven of nine species had background mean egg Se concentrations below 3 µg/g. Two species, black-legged kittiwake (*Rissa tridactyla*) and northern fulmar (*Fulmarus glacialis*), which feed in pelagic marine areas, had mean egg Se concentrations slightly over 4 µg/g (Outridge *et al.* 1999). This observation is consistent with the observation that marine bird species may have higher Se levels due to feeding habits. Another anomaly found by Outridge *et al.* (1999), (not in Table 4.13) weres the high concentrations of Se in the liver of the common merganser (*Mergus merganser*) and western grebe (*Aechmophorus occidentalis*), with the median (and maximum) concentrations of 38.6 (76.1) µg/g and 34.1 (66.2) µg/g, respectively. These birds were captured in the marine habitat of Howe Sound, BC, which may explain the relatively high values. The authors noted that further investigation was warranted (Outridge *et al.* 1999).

In Alberta (Slave Lake area) and the Northwest Territories (Yellowknife and Inuvik) between 2003 and 2004, DeVink *et al.* (2008) collected liver tissue from lesser and greater scaup (*Aythya affinis* and *Aythya marila*, respectively), scoters (*Melanitta fusca*), and ring-necked ducks (*Aythya collaris*). Geometric mean (range) Se concentrations in liver were 6.2 (5.5 – 7.0, n=71), 4.6 (4.0 – 5.6, n= 42), and 32.6 (28.4 – 37.3, n=50) µg/g in female scaup, ring-necked ducks, and scoters, respectively, from all sites and all years combined (DeVink *et al.* 2008). The higher Se in scoter livers was attributed to heavier use of marine habitats for foraging where concentrations of Se are naturally higher than in freshwater environments (DeVink *et al.* 2008).

Morrisey *et al.* (2004) studied two populations of American dippers (*Cinclus mexicanus*) in the Chilliwack area in south-western BC. The resident river dippers and more migrant tributary dipper populations had mean egg Se concentrations of 2.96 (± 0.16) and 2.67 (± 0.19) µg/g, respectively (not in Table 4.13). Wayland *et al.* (2006) studied resident American dipper

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populations near coal mining activities in the Rocky Mountain foothills of Alberta and found dippers at reference sites had mean egg Se concentrations (SD) of 4.9 (± 0.2) µg/g, versus 6.3 (± 0.2) µg/g within coal mining areas, which was significantly higher. SciWrite (2004), Harding *et al.* (2005), and Harding (2008) conducted studies on various bird species in the Elk River, south-eastern BC, between 2002 and 2005, evaluating the potential impacts of Se from large-scale open pit coal mining activities. In their study on the American dipper, there was no significant difference in mean (SE) egg Se concentrations between reference (7.4 (0.45) µg/g, n=11) and exposed areas (8.0 (± 0.44) µg/g, n=10) (Harding *et al.* 2005). However, in spotted sandpiper (*Actitis macularia*) there was a significant difference between mean (SE) egg Se concentrations at reference (3.8 (± 0.19) µg/g, n=14) and exposed sites (7.3 (± 0.43) µg/g, n=26) (Harding *et al.* 2005). Monitoring conducted in north-eastern BC on spotted sandpiper found mean (SD) egg Se concentration in reference streams were at 3.2 (± 0.3) µg/g, with a fairly narrow range in individual eggs (2.8 to 3.7 µg/g Se) (Golder 2010a).

In the Elk River basin, red-winged blackbirds (*Agelaius phoeniceus*) had mean egg Se concentrations of 2.96 µg/g in reference areas, and 21.7 µg/g in areas influenced by coal mining, with individual measurements in exposed areas reaching 40 µg/g (Harding 2008). Mean egg Se concentrations were measured in several aquatic bird species in the Elk Valley, including Canada goose (*Branta canadensis*), mallard (*Anas platyrhynchos*), American coot, hooded merganser (*Lophodytes cucullatus*), blue-winged teal (*A. discors*), green-winged teal (*A. carolinensis*), ring-necked duck, Barrow's goldeneye (*Bucephala islandica*), and bufflehead (*B. albeola*) (SciWrite 2004). Mean egg Se ranged from 1.38 µg/g, measured in Canada goose at a reference site, to 29.6 µg/g in American coot at a high Se exposure site (SciWrite 2004).

These data show that the majority of mean egg Se concentrations in birds at background sites are less than 5 µg/g. The Harding *et al.* (2005) reference site data for dipper is an exception and is much higher than reported by Wayland *et al.* (2006) for that species in Alberta studies.

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Table 4.13 Summary of selenium concentrations in bird tissues collected at sites in Canada.

Sampling Location	Se (µg/g dw)	Bird Species / Description	Impact[1]	Reference
Sites across Canada	2.56 (± 1.11) (n=134)	Mean egg Se in multiple bird species (predominantly water fowl) at sites from NWT, Ontario and Nova Scotia (n represents number of pooled samples in mean)	R, PI	Outridge *et al.* (1999)
Slave Lake, Alberta; Inuvik & Yellowknife, NWT	6.2 (5.5–7.0) (n=71)	Mean liver Se (95%CI) in female ***scaup***	R, PI	DeVink *et al.* (2008)
	4.6 (4.0–5.4) (n=42)	Mean liver Se (95%CI) in female ***ring-necked ducks***	R, PI	
	32.6 (28.4–37.3) (n=50)	Mean liver Se (95%CI) in female ***scoter*** (influenced by marine habitat and diet)	R, PI	
Chilliwack, south-western BC	2.96 (± 0.16) (n=17)	Mean (SD) egg Se in ***American dipper*** from riverine reference sites	R.PI	Morrisey *et al.* (2004)
McLeod River, Alberta	4.9 (± 0.2)	Mean (± 1SD) egg Se in ***American dipper*** from reference sites	R	Wayland *et al.* (2006)
Gregg River, Alberta	6.3 (± 0.2)	Mean (± 1SD) egg Se in ***American dipper*** from coal mine influenced sites	I	
Elk River, south-eastern BC	7.4 (0.5) (n=11)	Mean egg Se (SE) in ***American dipper*** from reference sites	R	Harding *et al.* (2005)
	8.4 (0.4) (n=10)	Mean egg Se (SE) in ***American dipper*** from coal mine influenced sites	I	
	3.8 (0.2) (n=14)	Mean egg Se (SE) in ***spotted sandpiper*** from reference sites	R	
	7.3 (0.4) (n=26)	Mean egg Se (SE) in ***spotted sandpiper*** from coal mine influenced sites	I	
Hambrook Creek, north-eastern BC	3.2 (± 0.3) (2.8 – 3.7)	Mean egg Se (±SD), and (range) of Se in individual ***spotted sandpiper*** collected from reference area near coal mining	R	Golder (2010)
Elk River, south-eastern BC	2.96 – 6.34 (0.89–6.00)	Range of mean egg Se concentrations in ***red-winged blackbird*** at reference sites	R	Harding (2008)
	3.91 – 21.7 [1] (3.60–39.9)	Range of egg Se (individual min – max egg Se concentrations) in ***red-winged blackbird*** at sites influenced by coal mining	I	
Elk River, south-eastern BC	1.38, (n=2)	Mean egg Se in ***Canada goose*** from a reference site (aqueous Se < 1.0 µg/L)	R	SciWrite (2004)
	3.23	Egg Se in individual ***mallard*** egg collected from a low Se Exposure site (aqueous Se between 1 – 10 µg/L)	I	
	17.1	Egg Se in individual ***Canada goose*** egg collected from a high Se exposure site (aqueous Se >10 µg/L)	I	
	29.6, (n=2)	Mean egg Se in ***American coot*** from a high Se exposure site (aqueous Se >10 µg/L)	I	

[1]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown.

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4.2.6.7 Amphibians and Reptiles

The background concentration of Se in amphibian and reptile tissue was reported to be between 2.9 and 3.6 µg/g in liver and 1 to 3 µg/g in other tissues (USDOI 1998). Studies conducted near a coal-fired power plant on eastern narrow-mouth toads (*Gastrophryne carolinensis*) found that reference site females had mean (SD) whole-body Se burdens of 1.85 ± 0.14 µg/g, with similar Se concentrations in eggs, 1.63 ± 0.12 µg/g (n=18) (Hopkins *et al.* 2006). Females at exposed sites had mean (SD) whole-body and egg Se concentrations of 42.40 (± 38.78) and 43.96 (± 37.62) µg/g (n=10), respectively (Hopkins *et al.* 2006).

Embryo Se concentrations measured from studies conducted between 2005 and 2009 on the Columbia spotted frog (*Rana luteiventris*) in the Elk Valley BC, at reference or minimally impacted sites, were between 2.44 to 5.26 µg/g, with a mean (SD) of 3.6 (± 0.8) µg/g (n=12) (Minnow *et al.* 2011).

4.2.6.8 Mammalian Wildlife

There are little data on Se concentrations in mammalian wildlife species. Species, such as mink (*Mustela vison*), are often near the top of the aquatic-based food web so may be at risk in areas where Se concentrations are elevated. Although mammals that rely on aquatic ecosystems for food accumulate Se, studies have not necessarily demonstrated a clear toxic response to Se, leading to the supposition that mammals may be less susceptible to Se-induced deformities than birds (Clark *et al.* 1987; Clark *et al.* 1989; Janz *et al.* 2010). While some small mammals themselves may be less at risk of Se-induced toxicity, levels of Se in these species may pose a threat to predators that may be more sensitive to Se (i.e., predatory birds) (Clark *et al.* 1987).

A summary of tissue Se residues in mammals from several North American studies is provided in Table 4.14. Puls (1994) reported that mink with an adequate Se concentration in their diet (0.2 to 0.4 µg/g), had tissue concentrations approximately 2.0 to 3.2 µg/g[24] in liver, 7.0 µg/g in kidney[25], and 0.3 to 0.41 mg/L in blood. Adequate Se concentrations in the diet of deer (no species given) and domestic sheep (*Ovis aries*) are reported to be between 0.1 and 1.0 µg/g, and

[24] Converted wet weight to dry weight assuming 75% moisture content.
[25] Converted wet weight to dry weight assuming 75% moisture content.

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0.4 to 1.0 µg/g, respectively. Puls (1994) reported tissue concentrations of Se in the muscle of deer (with adequate dietary Se), were between 0.25 and 0.49 µg/g. Additional Se tissue and blood data for domestic pets and livestock mammalian species may be found in Puls (1994).

In the western United States, the mean Se concentration in the blood of free-ranging bighorn sheep (*Ovis canadensis*) captured between 1980 and 1986, was 0.25 ± 0.25 mg/L (n=457) (Kock *et al.* 1987). The mean blood Se concentration of these wild sheep is comparable to the range of reported blood Se concentrations for domestic sheep of 0.12 to 0.5 mg/L, reported by Puls (1994).

Clark (1987) published the results of studies conducted on 10 species of wild mammals collected in 1984 at the Kesterson Wildlife Refuge area (Se-impacted) and the Volta Wildlife area (reference) in California. In general, they found that carnivorous species or species with diets closely linked to ponds containing Se-contaminated drainage (n=193), had much higher tissue Se concentrations in liver, than those in reference areas (n=139) (Clark 1987). Although the study focused on California vole (*Microtus californicus*), several other species were sampled, including: three species of mice (house mouse (*Mus musculus)*, deer mouse (*Peromyscus maniculatus)*, and western harvest mouse (*Reithrodontomys megalotis*)), ornate shrew (*Sorex ornatus*), muskrat (*Ondatra zibethicus*), California ground squirrel (*Spermophilus beecheyi*), dessert cottontail (*Sylvilagus audubonii*), long-tailed weasel (*Mustela frenata*), and Norway rat (*Rattus norvegicus*) (Clark 1987). Of note were the accumulations of Se in the liver tissues of voles from Kesterson, which had mean liver Se as much as 522 times those measured in voles at the reference sites (Clark 1987).

Clark *et al.* (1989) compared Se in racoon (*Procyon lotor)* captured at the Kesterson Reservoir and Volta Wildlife Area. The Kesterson racoons had liver and blood Se concentrations at least an order of magnitude greater than those captured at Volta, with hair and feces Se concentrations 30 and 21 times greater, respectively (Clark *et al.* 1989). Clark *et al.* (1992) continued studies on small mammals in the San Francisco Bay area, characterizing several contaminant levels including Se, in mice and voles in pickleweed marsh habitats. At Calaveras Point, the most contaminated sampling location in the Bay area, Se liver concentrations in mice were elevated

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(range of mean Se were1.54 – 4.75 µg/g, n=3), but still well below those measured at Kesterson (maximum liver Se was 60 µg/g in deer mouse) (Clark *et al.* 1992).

In Canada, several studies reported Se analysis on various wildlife species (Wren 1984; Gamberg *et al.* 2005a, 2005b; Lemke and Schwantje 2005). Wren (1984) conducted studies on beaver (*Castor canadensis*), racoon, and river otter (*Lutra canadensis*) in the Tadenac Lake area, an uninhabited watershed in Ontario. Of the tissues sampled, Se concentrations were highest in the liver of all three species, compared with kidney, intestine and muscle tissues. The mean (± SD) liver Se concentrations in racoon, otter, and beaver were 2.8 (± 1.2), 2.1 (± 0.3), and 0.2 (± 0.0) µg/g (n=4) [26], respectively. The results also indicated that liver concentrations of mercury and Se were highly correlated (r=0.96, n=12) in all species analysed. In a subsequent study by Wren *et al.* (1986), Se liver concentrations were measured in otter captured from four lakes in north-western Ontario; mean concentrations ranged from 1.21 µg/g (n=9) to 2.21 µg/g (n=26).

Trace mineral concentrations in bighorn sheep were compiled and evaluated using data collected between 1978 and 2004 from three meta-populations of California bighorn sheep and one meta-population of Rocky Mountain bighorn sheep, in British Columbia (Lemke and Schwantje 2005). Serum Se concentrations for all individuals ranged between 0.01 to 0.89 mg/L; the mean serum Se for California and Rocky Mountain bighorn sheep were 0.09 mg/L and 0.16 mg/L, respectively. The concentrations of Se in liver tissue for all individuals were from 0.24 to 10.48 µg/g; mean concentrations for California and Rocky Mountain bighorn sheep were 1.36 and 1.44 µg/g, respectively[27]. The authors stated that all mean serum and liver Se values were within established normal ranges for domestic sheep, published by Puls (1994).

[26] The author did not specify if Se concentrations were reported in wet wright or dry weight.
[27] Converted wet weight tissue concentrations to dry weight assuming 75% moisture content.

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Table 4.14 Summary of selenium concentrations in mammalian species measured in tissues at various locations across North America (units in dry weight unless otherwise stated).

Sampling Location	Se (µg/g dw or mg/L)	Mammal Species / Description	Impact[1]	Reference
Literature summary[2]	0.2–0.4 0.5–0.8 (ww) 1.4 (ww) 0.3–0.41 mg/L	Mink, adequate dietary Se Mink, liver tissue Mink, kidney Mink, whole blood	U	
Literature summary[2]	0.1–1.0 0.25–0.49	Deer, adequate dietary Se Deer, muscle tissue	U	Puls (1994)
Literature summary[2]	0.4–1.0 0.12–0.5 mg/L 0.25-1.5 (ww)	Domestic sheep, adequate dietary Se Domestic sheep, whole blood Se Domestic sheep, liver Se	U	
Western US	0.25 ± 0.2 mg/L (n=457)	Bighorn sheep blood Se (mean ± SD)	R/U	Kock *et al.* (1987)
British Columbia	0.01–0.89 mg/L (n=598) 0.06-2.62 (ww) (n=283)	Bighorn sheep blood Se, range of individual blood samples Bighorn sheep liver Se, range of individual samples	R/U R/U	Lemke and Schwantje 2005
Kesterson Reservoir, Pond 2	119 (61–250) (n=5)	California vole liver Se (mean and (range))	I	Clark *et al.* (1987)
Volta Wildlife Area (two sites)	0.23 (ND–1.4) (n=10)	California vole liver Se (mean and (range))	R	
Kesterson Reservoir, Pond 6	38.2 (19–73) (n=4)	Western harvest mouse liver Se (mean and (range))	I	
Volta Wildlife Area, Pond 7	1.69 (1.2–2.2) (n=4)	Western harvest mouse liver Se (mean and (range))	R	
Kesterson Reservoir	19.9 (12–31) (n=8)	Racoon liver Se (mean and (range))	I	Clark *et al.* (1989)
Volta Wildlife Area	1.69 (0.9–5.9) (n=4)	Racoon liver Se (mean and (range))	R	
San Francisco Bay, CA	4.75–1.56 (n=3)	Mice liver Se (range of mean conc at most contaminated sites)	I	Clark *et al.* (1992)
Tadenac Lake, south central Ontario	2.8 (± 1.2) [3] 2.1 (± 0.3) [3] 0.2 (± 0.0) [3] (n=4)	Racoon liver Se (mean ± SD) Otter liver Se (mean ± SD) Beaver liver Se (mean ± SD)	R	Wren (1984)
Yukon, CA	5.60 ± 3.36 (n=98)	Mink liver Se (mean ± SD) (assuming 75% moisture content)	R/PI	Gamberg *et al.* (2005a)
Yukon, CA	4.08 ± 1.67 (n=384) 6.40 ± 6.52 (n=56) 0.88 ± 1.36 (n=37)	Moose kidney Se, (mean ± SD) (assuming 75% moisture content) Moose liver Se (mean ± SD) (assuming 75% moisture content) Moose muscle Se (mean ± SD) (assuming 75% moisture content)	R/PI	Gamberg *et al.* (2005b)

[1]R = reference (unimpacted), PI = possibly impacted, I = impacted, U = unknown
[2] Number of samples and location of samples, not reported.
ND = Se not detected, entered as 0.1 for calculations.
[3] The author did not specify if results were reported in wet weight or dry weight.

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Gamberg *et al.* (2005a) measured Se in mink captured in the Yukon between 2001 and 2002. Mean liver Se was 5.60 ± 3.36 (n=98, assuming 75% moisture content). In another study conducted by Gamberg *et al.* (2005b) in the Yukon between 1994 and 2001, moose (*Alces alces*) tissues (kidney, liver, and muscle) were analysed for several metals, including Se. The data was summarised to evaluate: a) which, if any, elements were of toxicological concern, b) whether any temporal or geographic trends were evident, and c) assess whether tissue concentrations in moose kidney could be linked to environmental trace element concentrations. Selenium concentrations in moose kidney (n=384), liver (n=56), and muscle (n=37) were 4.08 ± 1.67, 6.40 ± 6.52, and 0.88 ± 1.36 µg/g, respectively[28]. The authors found that moose kidney Se was negatively correlated with age, as was arsenic, copper and molybdenum, while cadmium was positively correlated with age. They also concluded that concentrations of metals in moose reflected the natural geology of the areas they were captured.

5.0 Environmental Fate and Persistence

Three main processes govern the release of Se: natural weathering; biomethylation, which accounts for biogenic Se sources from terrestrial plants as well as freshwater and marine biota; and, accelerated release due to human activities such as mining, irrigation of Se-rich soils, fossil fuel combustion, or smelting of other minerals (Maier and Knight 1994). From a global perspective, the most significant pathway for the mobilization of Se, representing the largest fluxes, is from land to the aquatic environment (Figure 5.1; Haygarth 1994). However, the greatest contribution of Se both globally and to the aquatic environment originates from human activities often associated with areas having seleniferous soils or a geology of marine sedimentary rock or shales (Maier and Knight 1994; Luoma and Rainbow 2008).

[28] Assumed 75% moisture content to convert tissue concentrations from wet weright to dry weight.

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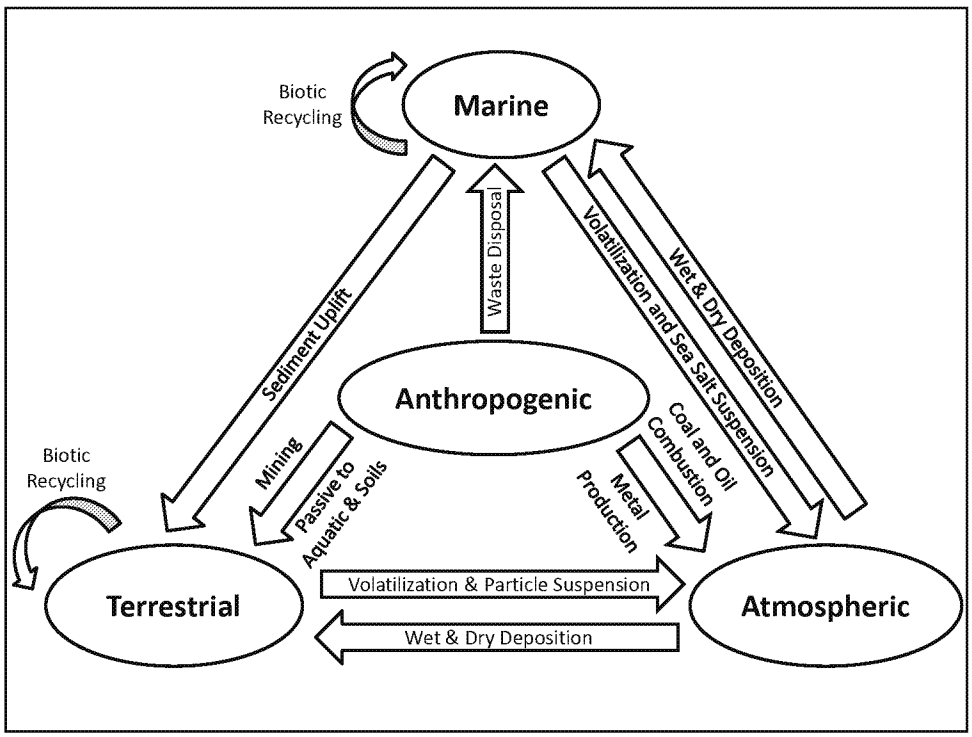

Figure 5.1 Global selenium cycle showing the major compartments and pathways for the movement of selenium (adapted from Haygarth 1994).

5.1 Processes in the Aquatic Selenium Cycle

While there is information on Se cycling on a global scale (e.g., Haygarth 1994), this section focuses on the cycles and processes that affect aquatic ecosystems. Selenium enters aquatic environments directly and indirectly through wet and dry deposition from both natural biogenic and anthropogenic (industrial combustion) sources in dissolved or particulate form (Maier and Knight 1994; Maher *et al.* 2010). Biogenic sources contribute 50 to 66% of the global Se. Natural random events, such as wildfires and volcanoes can also contribute to local increases of Se in air, but human activities are responsible for an increasing proportion of Se released to the environment (Nriagu and Pacyna 1988; Wen and Carignan 2009). Although the atmospheric contributions and speciation of particulate Se are not well studied, there is growing concern about the increasing use of coal for energy production world-wide and the potential consequences of greater contributions from this activity to the global Se cycle (Maher *et al.* 2010). Atmospheric Se is quickly photo-oxidized and returned to Earth's surface, some of which falls directly into surface waters as inorganic Se (Maier and Knight 1994).

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The concentration of Se in surface soils is primarily driven by the geologic origin of the parent rock formation. The availability of Se in soils to plant life varies depending on other soil characteristics such as alkalinity, pH, and sulphur content. However, soil microbes also play an important role in transforming elemental, inorganic, and organic forms of Se, which are then more available for plant uptake (CCME 2009). Methylated selenides formed by soil microbes and by the plants themselves, may then volatilize to the atmosphere. It is the wet and dry deposition of this biogenic Se from the atmosphere that is thought to be a major source in the cycling and redistribution of Se back to soils, surface water and groundwater (CCME 2009).

Land releases of Se into water occur from natural and human activities, either indirectly through overland runoff or directly from industrial discharges to the environment, with water being the most important vector for Se entering aquatic systems (Maher *et al.* 2010). As Se is transported, distributed, and redistributed in surface waters, the form of Se changes, playing an important role, along with overall Se loading, in the availability and fate of Se, and ultimately the severity of its effects on aquatic life (Luoma and Rainbow 2008).

Figure 5.2 shows the three major processes observed in the cycling of Se in aquatic environments (Maher *et al.* 2010):

- microbial processing and transformation of Se (selenate, selenite, elemental Se, selenide and gaseous dimethylselenide and dimethyldiselenide);
- trophic transfer of Se involving algae, plants, and animals (selenomethionine, selenocysteine and reduced diselenides);
- deposition and resuspension of multiple species of Se (selenate, selenite, elemental Se, and reduced diselenides).

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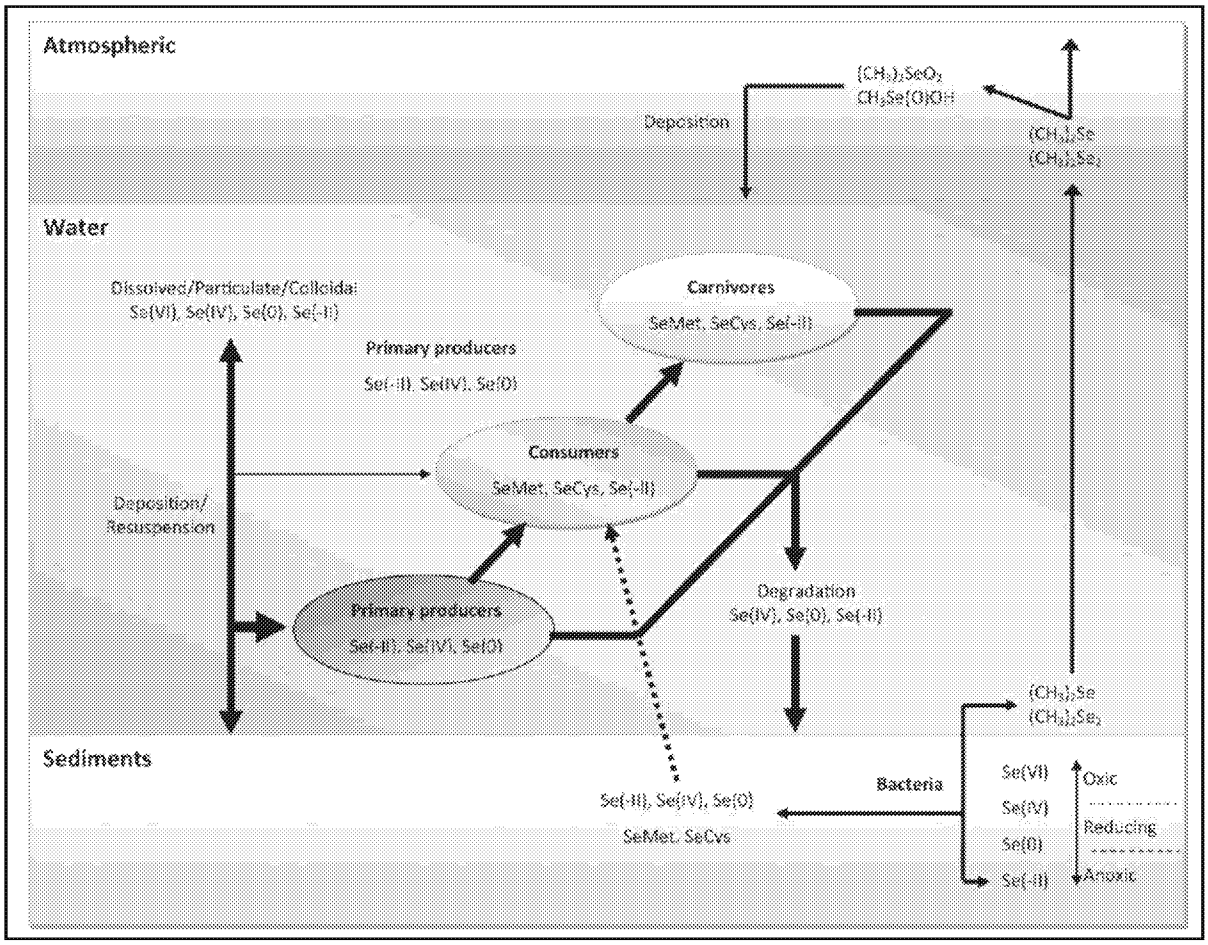

Figure 5.2 Schematic showing the cycling of selenium in aquatic environments. The major processes in an aquatic system are shown in bolded arrows (from Maher *et al*. 2010).[29]

5.2 Physical and Biological Processes

The biogeochemistry of Se in an aquatic environment is complex. Three processes influence the fate of dissolved Se in an aquatic environment: biological uptake, where Se is absorbed or ingested by organisms; binding with particulate matter, colloids, or surficial sediments; and, ongoing dissolution in aqueous solution (Lemly and Smith 1987; Simmons and Wallschläger 2005). Most of the Se entering the aquatic environment binds to particulate matter or is taken up by organisms (Maier and Knight 1994), ultimately becoming detritus and sediments through death, decay, and deposition of organisms and settling of particulates (Lemly and Smith 1987). Sediments play an important role in the cycling of Se in the aquatic environment. Selenium moves into and out of the top layers of sediment and detritus, governed by several physical,

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biological, and chemical processes (Simmons and Wallschläger 2005; Lemly and Smith 1987). The main processes that govern the partitioning of Se are: deposition and resuspension of selenate, selenite, elemental Se, and selenides; trophic transfer of selenomethionine, selenocysteine, and organo-selenides in algae, plants, and animals; and, microbial processes on selenate, selenite, elemental Se, organo-selenides and the gaseous Se forms dimethyl selenide and dimethyl diselenide (Maher *et al.* 2010).

Selenium is immobilized in aquatic environments in several ways (Lemly and Smith 1987; Simmons and Wallschläger 2005), including:

- biological uptake and reduction of oxidized forms of dissolved and particulate Se (selenate and selenite) to stable elemental Se by microbes in the top layer of sediments and overlying detritus;
- chemical adsorption or binding of selenate and selenite onto Fe-Mn oxyhydroxides and dissolved organic carbon at sediment surfaces;
- further chemical or biological reduction in sediments that result in insoluble organic, mineral, elemental, or adsorbed Se;
- uptake of organic Se by macrophytes, bacteria, and algae which is then methylated and released to the atmosphere as volatile Se; and,
- uptake, death, and decay of organisms followed by deposition to detrital and sediment layers.

Biological and chemical processes partitioning Se into particulate forms may account for as much as 90% of the total Se in an aquatic environment (Lemly and Smith 1987; Simmons and Wallschläger 2005). This degree of Se partitioning in sediment is consistent with results of experiments on the use of wetlands for treating Se-contaminated waste-water. Hansen *et al.* (1998) found that 89% of Se from refinery effluents were removed in a constructed wetland treatment area; most were immobilized into sediments and plant tissues, and 10 to 30% were removed through biological volatilization by vegetation and microbes. Bowie *et al.* (1998) analysed Se partitioning in the Hyco Reservoir, North Carolina, and reported that 97% of the total Se were found in lake sediment, about 3% in the water column and less than 0.1% in biota. A model used to calculate the fate of Se inputs predicted that 50% of the total loadings (inputs)

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into the reservoir were in sediments, 45% were flushed out in the outflows, and less than 5% were volatilized by algae and bacteria from the lake surface.

Although Se is immobilized by incorporation into biota and sediments, these compartments of Se represent a temporary repository (Lemly and Smith 1987; Simmons and Wallschläger 2005). Some Se will also be remobilized through several processes, which include:

- uptake, oxidation, and methylation of inorganic and organic Se by plant roots and microbes, followed by release to the atmosphere as volatile selenides which are then photo-oxidized and returned to land and water surfaces;
- the physical mixing and subsequent oxidation of reduced forms of Se by bioturbation (burrowing invertebrates) and disturbance related to the feeding activities of fish and other wildlife at sediment surface layers;
- direct physical mixing of sediments by water currents or upwellings;
- oxidation of sediments through plant photosynthesis; and,
- uptake and further cycling of Se by rooted plants and invertebrates which are then consumed by higher organisms.

In a study that compared two slightly different lentic areas in the Elk Valley BC, emergent vegetation was the most significant factor dictating sediment redox conditions in addition to the corresponding speciation and flux of Se across the sediment-water boundary (Martin *et al.* 2008). Rooted plants may be one of the main drivers for both redox conditions and the flux of Se between sediment pore water and overlying bottom waters (Martin *et al.* 2011).

5.3 Factors Affecting the Fate of Selenium

The form and availability of Se to aquatic organisms is controlled by both geochemical processes and kinetically-driven biological processes, such as species-specific mechanisms of uptake (Luoma and Presser 2009). Selenium speciation in various environmental compartments is influenced strongly by the oxidation-reduction potential (ORP), pH, flushing rates, and biological productivity of the particular environment, all of which play a role in the biological availability of Se (Simmons and Wallschläger 2005). The ratio of selenium species in solution depends on the source (natural or industrial), any treatment that was implemented prior to

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release, complexation with other ions, oxygenation of water, and biological activity (Maher *et al.* 2010; Bowie *et al.* 1996). Selenate is soluble, fairly stable and not particularly reactive with particulate matter so is taken up slowly especially in the presence of sulphate which competes directly with selenate for binding sites (Luoma and Presser 2009). Selenite and selenides are more reactive and may be taken up more readily by biota. Microorganisms readily convert selenate into elemental selenium in sediments and will also convert any form of aqueous Se into organo-selenides (Luoma and Presser 2009). The more reactive, reduced particulate selenium species accumulate in sediment and biota, creating a risk to vulnerable organisms in the food web (Luoma and Presser 2009).

The form and partitioning of Se is dependent on adsorption of dissolved ions onto particles, pH, and redox potential, but also may be influenced by temperature. Colder temperatures tend to decrease adsorption and increase partitioning of Se to sediments, relative to adsorption to competitive ions (such as sulphates, nitrates, or carbonates) (Wright 1999; Simmons and Wallschläger 2005; Maher *et al.* 2010). The speciation of Se may also be influenced by the concentration of organic matter, productivity (microbial activity), and physical mixing processes present (Lemly and Smith 1987). Studies have demonstrated that lentic waters are typically more biologically productive and, due to their slow moving nature, favour the establishment of reducing conditions. Under such conditions, the transport of Se into sediments is enhanced, increasing exposure and enhancing uptake of Se by bottom-dwelling benthic organisms (Simmons and Wallschläger 2005; Orr *et al.* 2006). This enhanced mobilization and bioavailability of Se in lentic environments at the base of the food web, leads to greater uptake and cycling of Se and higher overall bioaccumulation and risk to higher predators (Figure 5.3; Orr *et al.* 2006; Simmons and Wallschläger 2005). Redox potential and biological activity in sediments largely drive the flux of Se between water and the food web, perpetuating long-term toxic effects in aquatic systems even when Se inputs into the system have been reduced (Simmons and Wallschläger 2005).

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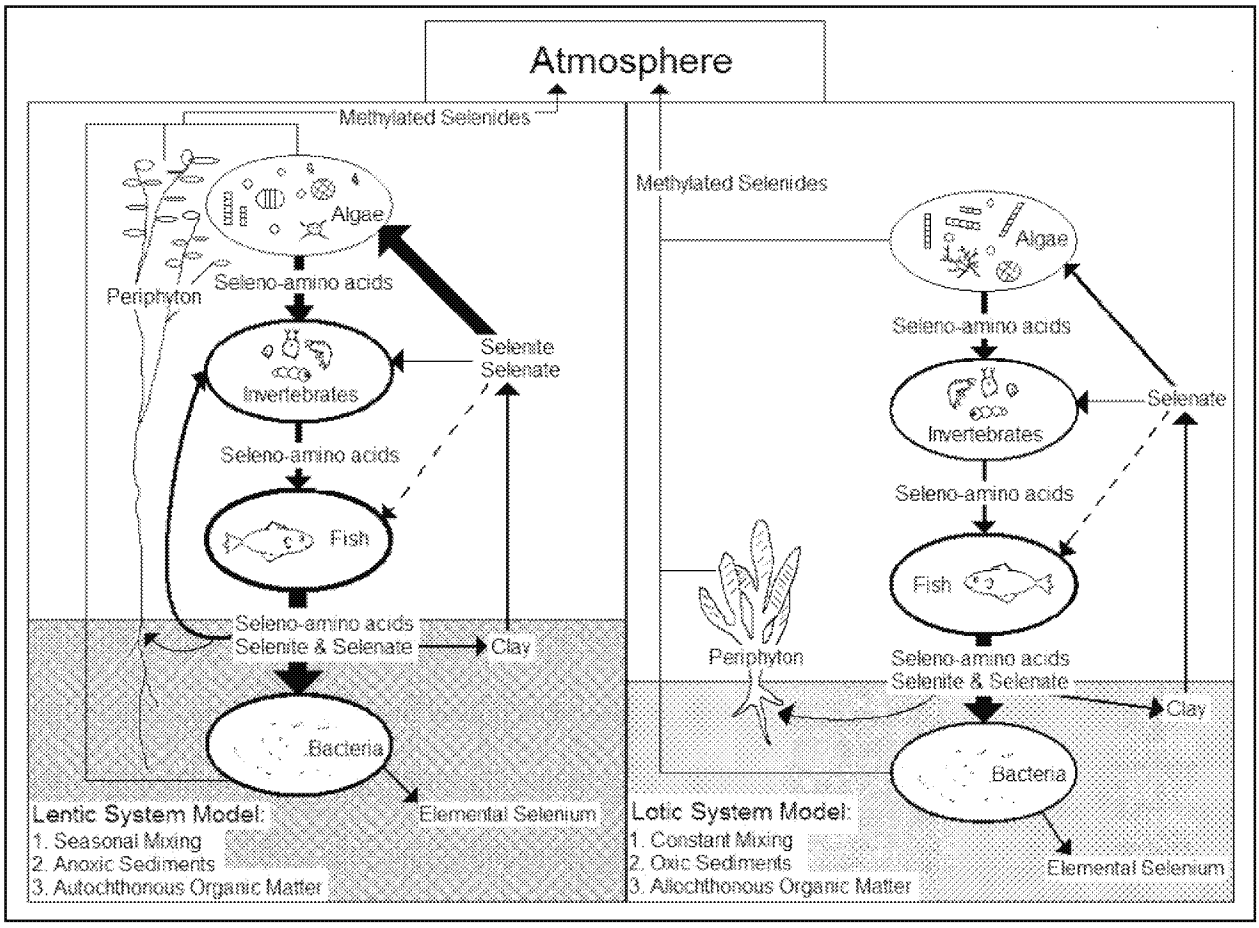

Figure 5.3 A simplified Se biogeochemical cycling in lentic & lotic aquatic environments. Thickness of ellipse = relative Se concentration in trophic level; thickness of arrows = magnitude of Se transfer or bioconcentration (from Simmons and Wallschläger 2005).

Based on studies comparing two lentic areas, Martin *et al.* (2008, 2011) concluded that speciation and recycling of Se is dependent on redox conditions. Furthermore, the presence or absence of emergent vegetation appears to be the dominant driver in sedimentary redox and hence the recycling and persistence of reduced Se in waters adjacent to sediments. These findings support those of Simmons and Wallschläger (2005).

Several important conclusions can be drawn from these models that predict the behaviour of Se in the aquatic environment, including:

- sediments are important in the biogeochemical cycling (and accumulation) of Se through the benthic food web, and represent a major repository for overall Se loadings to an aquatic system, as well as a source of Se exposure to benthic organisms;

- organisms play a major role in Se cycling. The biological processing and high flux rates of Se in an aquatic system can be determinants for Se speciation in the water column and microbial action drives much of the accumulation of Se in sediments;
- bacteria and primary producers bioconcentrate Se from water by several orders of magnitude, responding quickly to changes in water concentrations of Se. Higher trophic levels receive their Se though diet, some responding slowly to changes in Se. Sediments also respond more slowly to changing Se loadings;
- loss of Se through volatilization may be small in most aquatic environments. Wetlands with shallow, productive ecosystems can have more substantial losses; and,
- the type of environment (lotic versus lentic) plays a role in the cycling, partitioning and bioavailability of Se, as does the structure of the aquatic food web.

(Bowie *et al.* 1996; Ohlendorf 2003; Maher *et al.* 2010).

5.4 Selenium Behaviour in the Environment

Once Se is introduced into an aquatic environment and taken up in sediments and biota, it can continue to cycle and persist for many years if not decades (Simmons and Wallschläger 2005). Ecosystem recovery is just as complex as the mechanisms of bioaccumulation, and dependent on many factors including the severity of contamination, habitat type, annual productivity, flushing rates, and climate (Lemly and Smith 1987). Even after concentrations have been reduced in the water column, Se will continue to cycle from sediments and through the food web, heightening the risk associated with Se-contaminated ecosystems (Lemly and Smith 1987; Lemly 1997a; Lemly and Ohlendorf 2002).

Hermanutz *et al.* (1996) conducted a series of studies on bluegill sunfish (*Lepomis macrochirus)* using outdoor experimental streams dosed with 2.5, 10 or 30 µg/L sodium selenite, then after approximately a year, ceased Se dosing and continued to track effects. Streams dosed at 2.5 or 10 µg/L showed no adverse effects on progeny one year after Se addition ended. However, streams dosed with 30 µg/L Se took two years to recover (i.e., no significant adverse effect on adult or progeny after two years following the end of Se dosing).

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A study of Belews Lake was conducted 10 years after concentrations of Se in the water column dropped to less than 1 μg/L (Lemly 1997a). Selenium concentrations in the sediment-detrital food pathway still represented a risk to fish and aquatic life, as evidenced by the continued occurrence of Se-induced effects.

Maier *et al.* (1998) investigated the effects of a single application of Se fertilizer to enhance deer winter range on streams within the treatment area. Their findings were surprising given there was only a short-term elevation in waterborne Se, from below detectable concentrations (< 1μg/L) before the application, to 10.9 ± 0.7 μg/L three hours after the application. Concentrations of Se returned to below detection 11 days after the fertilizer treatment. Selenium concentrations measured in stream invertebrate tissues prior to treatment were 1.67 ± 1.65 μg/g, increased to 4.74 ± 1.73 μg/g three hours post-treatment (demonstrating the very rapid uptake into the food web), and remained at 4.54 ± 0.52 μg/g 11 months following the treatment.

Swift (2002) found similar results in experimental stream mesocosms dosed with sodium selenite for two to three years, followed by a two to three year period with no dosing. Selenium accumulated very rapidly in sediment and macroinvertebrate tissues at the onset of dosing and either reached a plateau, or in the case of some macroinvertebrates, continued to increase depending on species and dose. When dosing ceased, Se concentrations in sediment, rooted macrophytes, macroinvertebrates and fish decreased slowly. In the medium exposure mesocosm (10 μg/L), it took approximately one year for Se concentrations in these compartments to return to near that of control levels, and in the high exposure mesocosm (30 μg/L) it took almost two years. Only after several years, did Se concentrations in all compartments of the ecosystem approach concentrations considered to be non-toxic to fish and wildlife.

Hamilton *et al.* (2004) conducted studies in the Colorado River to evaluate the effect of lowering waterborne Se concentrations on various ecosystem compartments in a Se-contaminated backwater channel. A comparison of samples taken before (1995-96) and after (1997-98) Se was reduced demonstrated that substantial decreases in Se concentrations were seen in water, sediment, aquatic invertebrates and some forage fish. However, Se concentrations did not

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appreciably decrease in the endangered Colorado pikeminnow (*Ptychocheilus lucius)* and instead may have increased slightly (Hamilton *et al.* 2004).

Paveglio and Kilbride (2007) conducted follow-up studies in 2005 on resident birds at the North and South Grassland areas in the San Joaquin Valley, California, where Se-contaminated agricultural drainage water was used for wetland management between 1978 and 1985 (see Ohlendorf *et al.* 1986). The practice of using drainage water to augment wetland inputs into Kesterson Reservoir increased Se in surface water ulitmately resulting in reproductive failure and teratogenic deformities in several avian species (Presser and Ohlendorf 1987; Ohlendorf *et al.* 1996). Paveglio and Kilbride (2007) conducted their studies 20 years after changes were instituted to dilute the Se-laden drainage water with freshwater. They found that while Se concentrations in the livers of birds were generally lower than concentrations measured in 1986-87, Se concentrations in livers of pintail ducks inhabiting the more contaminated North Grasslands were no different than previously measured. As well, black-necked stilts had liver Se concentrations which still overlapped the thresholds for potential reproductive effects, demonstrating that Se continued to cycle through the wetland environment long after mitigative actions had been implemented.

These studies and others (e.g., Driessnack *et al.* 2011; Franz *et al.* 2011), provide good examples of the challenges facing environmental managers in the control, mitigation, and remediation of existing Se contamination, and the considerations that must be taken into account when issuing authorizations for new industrial, municipal, or agricultural activities.

6.0 Selenium Bioaccumulation

Bioaccumulation is a general term used to describe the accumulation of a chemical substance in an organism potentially involving multiple routes of exposure including respiration, dermal contact, and ingestion (CCREM 1987). The accumulation of a substance occurs because the rate of intake exceeds the organism's ability to use and remove excess concentrations of the contaminant (NLM 1993). Bioconcentration is a related term but refers only to the net accumulation of a substance resulting from the simultaneous uptake and excretion or elimination in an organism from water-only exposure, or the environmental media it is in direct contact with

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(CCREM 1987). Biomagnification refers to a systematic increase in the concentration of a substance in tissues as it passes up through two or more trophic levels (CCME 1999).

Selenium bioaccumulation is the key to toxic effects in an aquatic ecosystem. Selenium accumulates in biotic and abiotic environmental compartments which contribute, to different degrees, to elevated Se body burdens (Ohlendorf 2003; Presser and Luoma 2009; Stewart *et al.* 2010). Selenium residues in tissues can vary, even within the same species at the same site, and may not always be predictably correlated with Se in water (Buhl and Hamilton 2000; Hamilton 2002). Selenium concentrations in aquatic life can range between 100 and 35,000 times the concentration in water where waterborne Se is between 2 and 16 µg/L (Lemly 1996a; Lemly and Smith 1987). Relatively low water column Se concentrations, in the range of 3 to 10 µg/L, may result in elevated levels of Se in sediments, primary and secondary producers, and higher trophic level consumers, causing reproductive impairment in fish, aquatic birds, and amphibians (Lemly and Smith 1987; Peterson and Nebeker 1992; USDOI 1998; Lemly and Skorupa 2007). In a majority of cases, low water column Se concentrations (≤ 2 µg/L) will not result in significant accumulation through the food web. However, there are rarer instances where water Se < 1 µg/L results in significant bioaccumulation and apparent chronic effects (Pease *et al.* 1992; Barwick and Maher 2003; Rudolph *et al.* 2008).

Hamilton (2004) stated that fish studies published as early as 1957, identified accumulation of Se in the food webs and corresponding toxic effects. Barnhart (1957, cited in Skorupa 1998) found that seven species of stocked game fish in Sweitzer Lake, Colorado, were not reproducing, where benthic fauna had Se concentrations as high as 20 µg/g and fish livers contained 40 µg/g Se (Skorupa 1998). Later studies began to distinguish between the relative uptake and toxicity of different chemical forms of Se (Hamilton 2004). However, it should be noted that Se body burden may be almost entirely from dietary intake in some aquatic species. In other species, some invertebrates for example, uptake of soluble Se from water may account for 20% to 60% of the total body burden (Stewart *et al.* 2010).

Studies conducted in San Francisco Bay between 1998 and 1999 by Purkerson *et al.* (2003), found Se values ranged from 1.02 to 6.07 µg/g in estuarine zooplankton. Among their

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conclusions, the authors found that smaller herbivorous-omnivorous zooplankton had higher Se concentrations than larger omnivorous-carnivorous zooplankton, suggesting that trophic level and size may play an important role in regulating zooplankton Se concentrations (Purkerson *et al.* 2003).

For planktonic invertebrates in general, if water concentrations of Se are < 1.0 µg/L, tissue residues also tend to be low. Some macro-invertebrates, such as molluscs or benthic stream invertebrates, may accumulate a limited amount of Se by direct contact with water and sediments (Sui and Berman 1989). There is evidence that some detritus–eating benthic invertebrates may have accumulated relatively more Se from ingestion of epipelon (microorganisms living within the sediment-water interface) than was ingested directly from water and sediments (Orr *et al.* 2006). The concentration of Se in different tissues within the same organism is not necessarily consistent. Sometimes large differences are found between tissue types, for example Se in the mantle, kidney and digestive glands of the American oyster (*Crassostrea virginica*) (Sui and Berman 1989; Eisler 1985). Interestingly, with the exception of fish, some marine organisms can accumulate higher concentrations of Se than freshwater species without apparent ill effects (Stewart *et al.* 2010).

6.1 Quantifying Bioaccumulation

Bioconcentration factors (BCFs) and bioaccumulation factors (BAFs) are tools commonly used to better understand the fate of Se, predict toxic effects, and evaluate risk to ecological receptors (DeForest *et al.* 2007). Bioconcentration factors are calculated as ratios of Se concentrations in tissue to Se concentrations in water, or the ratio of Se in sediment to water. Bioaccumulation factors are ratios between tissue Se concentrations and dietary or sediment Se concentrations (Besser *et al.* 1993; Cleveland *et al.* 1993). However, some researchers may also also refer to the ratio of tissue Se to water Se as a bioaccumulation factor. Bioaccumulation factors are more appropriately used when chemical exposure routes include water, dietary items, and incidental ingestion of particles containing the chemical (DeForest *et al.* 2007).

Biomagnification is considered to have occurred when the ratio of contaminant concentrations between successive trophic levels is greater than 1.0 (Stewart *et al.* 2010). There are conflicting

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opinions as to whether Se biomagnifies through the food chain. Some investigators believe that Se may not biomagnify between trophic levels when considered on a whole-body to whole-body basis (Ohlendorf 2003) or when specific predator-prey relationships are not considered (Luoma and Rainbow 2008). Others have found that, in some instances, Se does biomagnify (Lemly 1996; Presser and Luoma 2006; Luoma and Rainbow 2008; Barwick and Maher 2003), may biomagnify (Stewart *et al.* 2004), may biomagnify depending on the size of the organism (Zhang and Wang 2007), or will biomagnify between some trophic levels and not others (Jasonsmith *et al.* 2008; Muscatello *et al.* 2008; Sanders and Gilmore 1994). Of the field studies conducted in Canada, McDonald and Strosher (1998), Casey (2005) and Orr *et al.* (2006) all reported bioaccumulation factors (BAFs) or bioconcentration factors (BCFs) in one or more compartments of the food web, and based on their data suggested biomagnification was occurring. The summary of bioaccumulation data in Table 6.1 shows variation in Se bioaccumulation related to the form of Se or other factors in the natural environment, making Se bioaccumulation in aquatic environments difficult to predict.

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Table 6.1 Summary of bioaccumulation and bioconcentration factors found in various compartments based on studies on freshwater organisms.

Species/media	Exposure concentration, duration, and form of Se (if known)	BAF/BCF	Reference
	Field exposure (lotic)		
Westslope cutthroat trout (*O. clarkii lewisi*)	8.6 µg/L total Se in water [1]	BAF = 1087 (n=10)	McDonald and Strosher (1998)
	10.7 µg/g Se in invertebrates (dietary)[1]	BAF = 0.9 (n=10)	
	Field exposure (lotic)		
Sediment	10.7 µg/L total Se in water [2]	BCF = 224 (n=4)	Casey (2005)
Biofilm		BCF = 299 (n=4)	
Filamentous algae		BCF = 514 (n=1)	
Rainbow trout (ripe ovary)	10.0 µg/g dietary Se [2]	BAF = 3.4 (n=3)	
	Field exposure		
Lentic sediment	Range 7.15 - 88 µg/L total Se in water at 3 lentic sites, n=1	BCF = 0.06 - 1.11	Orr *et al.* (2006)[3]
Lentic cutthroat trout		BAF = 0.9 - 3.6	
Lotic sediment	20.1 and 20.9 µg/L total Se in water at 2 sites, n=1	BCF = 0.1 (both sites)	
Lotic cutthroat trout		BAF = 0.34 - 0.4	
	Lab exposure		
Green algae (*C. reinhardtii*)	24 h, 10 µg/L selenate	BCF = 428 (±44) (n=3)	Besser *et al.* (1993)
	24 h, 10 µg/L selenite	BCF = 1440 (±38) (n=3)	
	24 h, 10 µg/L Se-methionine	BCF = 2320 (±181) (n=3)	
	24 h, 1 µg/L Se-methionine	BCF = 15,700 (371), (n=3)	
Daphnids (*D. magna*)	96 h, 10 µg/L selenate	BCF = 293 (±23) (n=3)	
	96 h, 10 µg/L selenite	BCF = 570 (±77) (n=3)	
	96 h, 10 µg/L Se-methionine	BCF = 30,300 (±3,860) (n=3)	
	96 h, 1 µg/L Se-methionine	BCF = 229,000 (±14,000) (n=3)	
Bluegill sunfish (*L. macrochirus*)	30 d, 100 µg/L selenate	BCF = 20 (n=3)	
	30 d, 10 µg/L selenite	BCF = 56 (n=3)	
	30 d, 10 µg/L Se-methionine	BCF = 5,000 (n=3)	
	30 d, 1 µg/L Se-methionine	BCF = 8,000 (n=3)	

[1] Field data collected from site on the Fording River near Swift Creek, BC, below coal mining.
[2] Field data collected from site on Luscar Creek, AB, below coal mining.
[3] BCFs and BAFs calculated using data from Table S6 of the published Supplemental Data.

While there may be differing opinions regarding biomagnification, there is no disagreement that the most significant bioaccumulation of Se occurs at the microorganism level as bacteria and

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algae take up available Se from the water column and sediments (Ohlendorf 2003; Lemly 1987). This first step in bioaccumulation not only represents the largest concentration of Se but can also be the most variable likely due to species-specific and site-specific variables (Schlekat *et al.* 2004). Selenium is also taken up from the water column by fish and wildlife through the gills, epidermis, gut, and diet (Hamilton 2004). Presser and Luoma (2010) suggest the aqueous route of exposure typically makes up less than 5% of the overall body burden in tissues of consumer organisms. Sandholm *et al.* (1973) were the first to report that Se accumulated in fish primarily through the food web. This study examined the food chain uptake of Se in fish, demonstrating that Se uptake through food organisms, such as phytoplankton and zooplankton, was the most efficient exposure route, while Se accumulation directly from water was not substantial. Dietary Se uptake is also important in invertebrates. For example, water boatmen (*Trichocorixa reticulate*) exposed to high concentrations of Se in water did not accumulate any more Se than control exposure groups, but did accumulate significantly more Se from diet than controls, suggesting accumulation in this species is solely from diet (Thomas *et al.* 1999).

Biotransference factors (BTFs), defined as the ratio of chemical concentration in organisms from a food source to its consumer or between trophic levels, have been estimated by Barwick and Maher (2003) and later by Jasonsmith *et al.* (2008). These researchers calculated the BTFs between successive trophic levels to evaluate whether or not biomagnification was occurring in various food pathways. Barwick and Maher (2003) examined bioaccumulation of Se within an estuarine seagrass ecosystem, finding that 29 of 35 trophic interactions demonstrated positive biotransference (i.e., BTFs >1) indicating that biomagnification had occurred. Despite Se concentrations in the water column being low (0.3 to 0.5 μg/L), the muscle of four predatory fish species demonstrated biomagnification of Se and had tissue levels exceeding the maximum Australia New Zealand Food Authority (ANZFA1992) permitted level of Se considered safe for human consumption (5 μg/g Se) (Barwick and Maher 2003). Jasonsmith *et al.* (2008) concluded that biomagnification was occurring within a freshwater ecosystem, in the phytoplankton and sediment/detrital pathways, likely explaining the Se biomagnification seen in both rainbow trout and flathead gudgeon (*Philypnodon grandiceps*).

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Since Se accumulation is influenced by more than just exposure to ambient concentrations, researchers have added biokinetic or biodynamic terms to models which more accurately reflect the accumulation and passage of Se through the food web (Luoma and Rainbow 2005; Zhang and Wang 2007; Luoma and Presser 2009; Presser and Luoma 2010). These models quantify the mechanistic components from one trophic level to the next, including not only the concentration of Se in food or ingested particles, but also ingestion rate, and constants for assimilation efficiency, growth, and efflux (excretion) rates (Luoma and Rainbow 2005; Zhang and Wang 2007; Luoma and Presser 2009; Presser and Luoma 2010). Luoma and Presser (2009) have advocated a systematic approach that breaks down the overall bioaccumulation (BAFs) into smaller steps that consider the uptake of Se in the aquatic food web progressively though each successive trophic level. In their model, enrichment factors (EFs), which are equivalent to BCFs between water and primary producers, as well as trophic transfer factors (TTFs), calculations of accumulation between trophic levels, may be derived experimentally or from field observations. These models should consider site- and species-specific differences in bioaccumulation and may sometimes require incorporation of an aqueous exposure route for some species (DeForest *et al.* 2007; Zhang and Wang 2007; Luoma and Presser 2009).

Many uncertainties and gaps in our understanding of the biokinetics and biodynamics of Se bioaccumulation remain. Research is needed on the biotransformation of Se at the base of the food web, mechanisms of uptake, ingestion and assimilation efficiency rates, sequestration and inter-organ transfer of Se, and the induction of toxic effects (Stewart *et al.* 2010). In addition, some of the basic assumptions that are the foundation of this approach, have been questioned, such as to what degree, if at all, an individual of any given species is capable of regulating Se. If biota have the ability to regulate Se (i.e., TTFs are not fixed), then values used for ingestion, assimilation and excretion are not constant (Beckon *et al.* 2010). Once again, this area of research is progressing and better ways of predicting Se bioaccumulation are being sought.

6.2 Factors Affecting Bioaccumulation

Selenium bioaccumulation can vary widely within and between species and is dependent on many biotic and abiotic factors, including the amount, form of Se, the presence of other elements and compounds, food preferences, temperature, type of habitat, species sensitivity, life stage, and

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trophic position or food web structure (Lemly and Smith 1987; Coulliard *et al.* 2008; Stewart *et al.* 2010). Although more research is needed on the mechanisms of Se bioaccumulation for various organisms, some general findings are apparent. For example, at low ambient Se concentrations, assimilation and accumulation of Se is often highest, declining as aqueous or dietary concentrations increase. This biphasic dose-response pattern is seen in nutrition, agriculture, pharmacology, and toxicology describing responses to essential compounds, where there is an optimum range and a toxic range, as concentrations increase in excess of optimum (Beckon *et al.* 2008).This pattern suggests that a mechanism for regulation of Se may exist in some species (Ohlendorf 2003; DeForest *et al.* 2007; Harding 2008; Minnow *et al.* 2011).

6.2.1 Selenium Speciation

Differences in the bioconcentration of organic and inorganic Se are apparent, with the accumulation of organic Se (seleno-L-methionine and selenocysteine) one to two orders of magnitude greater than inorganic selenate or selenite (Simmons and Wallschläger 2005). Besser *et al.* (1993) found that organic Se, in the form of seleno-L-methionine, accumulated more readily than either inorganic selenate or selenite in the algae *Chlamydomonas reinhardtii*, the water flea *Daphnia magna* and in bluegill sunfish (Table 6.1). Bioconcentration factors estimated for the three species based on an exposure of 1 µg/L Se-methionine were approximately 16,000 in algae, 200,000 in daphnids and 5,000 in bluegill sunfish, as compared with bioaccumulation of selenite and selenate in the same species between 270 and 3,600 (Besser *et al.* 1993) (Table 6.1). Kiffney and Knight (1990) found similar accumulation patterns in studies on the cyanobacteria *Anabaena flos-aquae*. Maier and Knight (1993) found seleno-DL-methionine accumulation was significantly higher than selenate or selenite based on laboratory experiments exposing larval midge *Chironomus decorus* for a 48-hour period. Similar studies were conducted by Franz *et al.* (2011), who compared accumulation of Se in larval *C. dilutus* exposed for10-days to aqueous selenate, selenite, and seleno-DL-methionine, versus controls. These authors found no appreciable difference in body Se concentrations of control larva and those exposed to 4.8 µg/L selenate, but found substantial increases resulted from exposure to 3.8 and 1.8 µg/L of selenite and seleno-DL-methionine, respectively.

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In a study on macroinvertebrates, Se speciation analyses showed that organic selenides and diselenides, modelled as selenomethionine and selenocysteine, accounted for more than 85% of the total Se measured in all nymph and larval insects analysed (Andrahennadi *et al.* 2007). In the same study, small fractions of selenite were found in invertebrate tissue, but selenate was not found in significant quantities despite selenate being the dominant form of Se in the water column. Interestingly, the researchers also found that biofilm samples showed fairly high concentrations of selenite and moderate concentrations of elemental Se. This supports other study results which suggest that the conversion of selenate to selenite, the initial step in Se bioaccumulation, is important in making Se more available to consumers, while bacterial conversion to elemental Se renders some fraction of total Se in a potentially less available form (Simmons and Wallschläger 2005; Andrahennadi *et al.* 2007; Luoma and Presser 2009). Studies on birds and fish have shown similar trends in the relative bioaccumulation and toxicity of organic and inorganic Se forms, with the organic forms of Se (organic selenides, selenomethionine and selenocysteine) bioaccumulating more readily than inorganic forms (Heinz *et al.* 1987; Ohlendorf and Heinz 2011).

6.2.2 Physical Environment

Selenium behaves differently in lotic (fast moving) and lentic (very slow moving or still) waters (Lemly and Smith 1987; Simmons and Wallschläger 2005; Hillwalker *et al.* 2006; Orr *et al.* 2006). As mentioned in Section 5.2, lentic aquatic environments have lower flushing rates and higher productivity and favour reducing conditions, all of which enhance Se bioaccumulation. Orr *et al.* (2006) compared the accumulation of Se in lentic and lotic environments and found that lentic habitats accumulated Se to a greater extent than lotic exposure areas (Table 6.1). Some investigators have used the water flow regime of an aquatic system, along with primary productivity and sediment characteristics, to estimate retention capacity of Se (the ability of a system to accumulate and conserve Se) to assess the degree of hazard Se may pose to fish and wildlife (Lemly 2002c; Lemly 2007).

Lemly (1993b) found temperature was a significant factor in Se bioaccumulation. In a study evaluating the effects of Se exposure, when water temperature was decreased to 4 °C to simulate winter conditions, whole-body Se concentrations in juvenile bluegill sunfish (*Lepomis*

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macrochirus) increased by approximately 30% compared with a control group held at 20 °C. At the termination of the test, 40% mortality was observed in the exposed group – an effect later dubbed "*winter stress syndrome*" (Lemly 1993b). McIntyre *et al.* (2008) attempted to replicate the study and while their study design was not exactly the same, they found the temperature-related effect thresholds were slightly higher than reported by Lemly (1993b). However, McIntyre *et al.*'s (2008) results were similar to that of Lemly (1993b) in that juvenile bluegill appeared to accumulate more Se and were more sensitive to Se toxicity in the test groups that were lowered to colder water temperatures (20 to 4 °C). The mechanism for increased Se accumulation found in the Lemly (1993b) study may have been related to a combination of reduced feeding (reduced photoperiod) and lipid depletion as energy stores were used (resulting in reduced weight and decreased condition factor). McIntyre *et al.* (2008) did not find similar decreases in lipid content or condition factor in his studies. Janz (2012) noted that the evidence for increased Se toxicity over winter periods has not been found under field conditions. For example, Driedger *et al.* (2009) did not find evidence of a temperature effect on bioaccumulation in field investigations on northern pike. On the other hand, Saiki *et al.* (2001) conducted field studies on Se body burdens in both bluegill and green sunfish (*Lepomis cyanellus*) and found that waterborne Se was the single most important predictor of Se body burden, but temperature was also a significant predictor. Since the literature is equivocal, this may be an area where more research is needed to determine what the mechanism and effect of colder water may be on Se bioaccumulation and toxicity.

6.2.3 Influences of Other Compounds and Elements

Studies have shown that the presence of dissolved sulphate can reduce selenate uptake by algae, cladocera and midges, probably due to the chemical similarity of the two compounds (Hansen *et al.* 1993; Williams *et al.* 1994; Ogle and Knight 1996; Riedel and Sanders 1996). The mechanism for this interaction is rooted in the competition between sulphate and selenate for transport across cell membranes (William *et al.* 1994). However, while sulphate may reduce the uptake of selenate, it does not appear to affect uptake of selenite or selenomethionine (Ohlendorf 2003). On the other hand, Severi (2001) examined the effects of sulphate on sodium selenate and sodium selenite concentrations in the aquatic plant *Lemna,* reporting that the toxicity of both Se salts appeared to be inversely correlated with sulphate concentration. The relationship between

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sulphate and selenate bioaccumulation may not be as evident in some species, which may affect Se bioavailability and toxicity in species such as *Hyallela azteca* (Brix *et al.* 2001).

In the draft US EPA Se criteria document, the authors derived a sulphate-correction for an acute exposure criterion for selenate (US EPA 2004). However, there appears to be conflicting evidence of a consistent relationship between decreasing selenate bioaccumulation and increasing sulphate in zoobenthos (deBruyn and Chapman 2007). These authors point out that the selenate:sulphate relationship in *Gammarus* reported by Brix *et al.* (2001) was driven by a single low LC50 value at low ambient sulphate concentrations; a similar analysis for *Hyalella* revealed no such relationship. They also point out that while Hansen *et al.* (1993) reported the bioconcentration of Se was reduced as sulphate increased, that decrease in bioconcentration was modest (less than a factor of 2) over a very large range of sulphate (Se/sulphur ratios from 1:0 to 1:480) (DeBruyn and Chapman 2007). Hansen *et al.* (1993) offered a caveat to their findings, stating that the presence of sulphate does not eliminate selenate uptake and added that more research is needed before a sulphate-corrected Se criterion should be adopted.

Ohlendorf (2003) mentions organic carbon and sediment grain size as factors influencing Se concentration and bioavailability. Wiramanaden *et al.* (2010) also found that whole sediment Se concentrations were closely related to sediment total organic carbon. However, this may reflect the eventual fate of Se that is taken up by microorganisms or complexed with organic and inorganic particulate matter which is then incorporated into sediments (Simmons and Wallschläger 2005). Hillwalker *et al.* (2006) conducted a three-year study examining the possible connection between organic carbon and Se accumulation in various compartments of lotic and lentic environments. Although the pond and creek had fairly similar water Se and similar total organic carbon (TOC) content in sediments, the pond sediment and detritus (top 2 cm) had 1 to 7 times higher levels of Se. They also found that Se was more efficiently accumulated in invertebrates at the lentic (pond) site compared with lotic. The study revealed that site-specific relationships between organic content and Se were apparent in sediment and detritus. However, organic carbon did not explain differences found in site-specific Se accumulation in invertebrates between lotic and lentic sites, suggesting that factors beyond

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organic carbon (other site-specific biogeochemical factors) influence bioaccumulation in lotic and lentic food web (Hillwalker *et al.* 2006).

In experiments using green algae (*C. reinhardtii)*, Riedel and Sanders (1996) found that phosphate suppressed the uptake of selenite. Studies by Yu and Wang (2004) also suggest that increased phosphate concentrations in water may suppress the uptake of selenite by algae due to competition between the selenite and phosphate ions. Wright (1999) proposed that increased nitrate might be associated with increased oxidation and mobilization of Se, a possibility that could have profound implications for many industrial discharges containing high levels of nitrate.

Metals such as copper, arsenic, and iron can influence Se bioaccumulation (Hamilton 2004; Barceloux 1999). In a laboratory feeding study, Atlantic salmon fed a copper-supplemented diet had reduced liver Se concentrations (Lorentzen *et al.* 1998). These researchers found a strong negative correlation between dietary copper and liver Se concentrations, yet at the same time observed a significant positive correlation between liver concentrations of copper and Se (Lorentzen *et al.* 1998). Reduced Se levels in the liver with increased dietary Cu was thought to be the result of: a) redistribution of Se for synthesis of glutathione peroxidase in response to oxidative stress (a detoxification mechanism to address increased Cu) or, b) the formation and excretion of Cu-Se complexes from the liver or intestines of fish (Lorentzen *et al.* 1998). Hilton and Hodson (1983), found a similar relationship with increasing Se and Cu concentrations in rainbow trout liver.

There is a great deal of literature reporting the antagonistic effect of Se on mercury (Hg) bioaccumulation, particularly in marine fish (Chen *et al.* 2001; Ohlendorf 2003; Klaverkamp 2002; Hamilton 2004). Studies conducted on northern pike demonstrated that waterborne Se had no effect on Hg concentrations in fish tissue, but dietary Se reduced Hg concentrations in both total body burden and muscle tissue (Turner and Swick 1983). Chen *et al.* (2001) found similar results; there was a strong negative correlation between muscle Se and Hg in perch (*Perca flavescens*) and walleye (*Stizosedion vitreum*) in nine lakes in the vicinity of a Sudbury Ontario smelter. However, it should be noted that not all literature report an antagonistic effect between

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Se and Hg. Some authors have reported no effect or even a synergistic effect (greater than the sum of individual effects) on the bioaccumulation and toxicity of Hg in the presence of Se (Stewart *et al.* 2010; Khan and Wang 2009). The relationship between Se and Hg, while thought to be strictly antagonistic, needs further research.

Arsenic (As) has also been shown to have an antagonistic effect with Se, reducing the toxic of Se in several species under varying conditions, including teratogenic effects (Levander 1977). The mechanism is thought to be the increased excretion of Se in bile rather than As blocking Se accumulation. Arsenic toxicity can also be reduced by the presence of Se (Marier and Jaworski 1983). Sufficient concentrations of iron in solution has been shown to decrease Se availability through co-precipitation of Se and iron as insoluble ferric selenite (Barceloux 1999).

Aquatic environments receiving industrial discharges contain a mixture of contaminants. Available metals such as chromium, cadmium and nickel may confound the expected effects of elevated Se perhaps through formation of complexes and explaining an apparent lack of toxicity in fish (Lohner *et al.* 2001a). For example, Pollack and Machin (2009) found that the effect of Se on the reproductive indices of male lesser and greater scaup were attenuated when Se was bound with cadmium, but not with mercury.

6.2.4 Species-Specific Variation in Selenium Uptake

One of the many challenges of understanding Se bioaccumulation is the variation of accumulation across different environments and between species (Figure 6.1). Studies have compared Se uptake and depuration of the bivalve *Potamocorbula amurensis* and two common crustacean zooplankton species, including the effect on Se concentrations in their predators (Stewart *et al.* 2004). These studies demonstrated that basic differences in physiology, in this case the relatively slow rate of Se loss in molluscs compared to crustaceans, resulted in a much higher bioaccumulation of Se in molluscs suggesting that some organisms and their predators may be at higher risk of Se-induced effects.

Hillwalker *et al.* (2006) conducted accumulation studies in a lotic and lentic ecosystem and found that along with site-specific differences in accumulation of Se (lotic versus lentic) there

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were also organism-specific differences in Se accumulation within basic classification levels (Order, Genera). They underscored the importance of investigating organism-specific factors in understanding the transfer of Se to higher trophic levels.

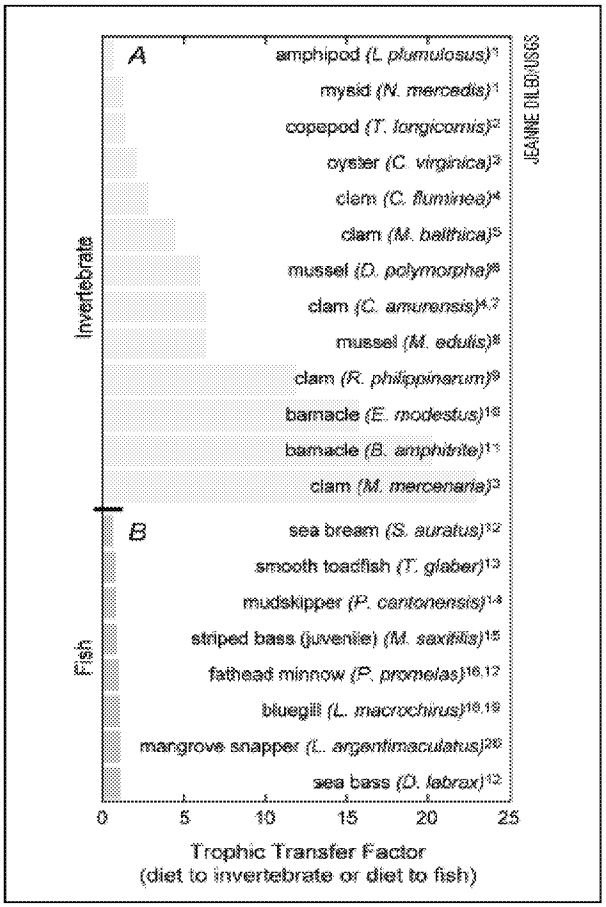

Figure 6.1 Trophic transfer function (TTF) estimates based on laboratory data for several invertebrate and fish species (from Luoma and Presser 2009).

Conley *et al*. (2011) found that a species of mayfly (*Centroptilum triangulifer*) fed a lower ration (*1x*) of cultured periphyton had higher Se body burdens and higher trophic transfer factors (TTFs) than mayflies on double the ration (*2x*). Several possible explanations were put forward to explain this disparity: a) mayflies receiving the *2x* ration became larger possibly diluting the accumulated Se; b) the *1x* ration group were nutritionally limited resulting in higher Se assimilation efficiencies, as well as higher TTFs and body burdens; or, c) because they were nutritionally limited, the *1x* group were unable to avoid more highly contaminated food prey

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species (Conley *et al.* 2011). This study demonstrates the complexity and uncertainty that exists when predicting Se bioaccumulation in natural environments. In an attempt to address these complexities, biokinetic controls of Se accumulation have been the basis for recent modelling approaches to estimate the trophic transfer of Se in aquatic ecosystems (Luoma and Presser 2009).

Selenium does not necessarily show an allometric (proportional) relationship of increasing concentration with increasing size, length, or age of the individual species as do some metals like mercury, indicating that other physiologically-based pharmacokinetic factors may be involved in driving bioaccumulation (Newman and Unger 2003; Hamilton 2004; Zhang and Wang 2007; Gantner *et al.* 2009; Stewart *et al.* 2010). These within- and between-species differences underscore the importance of site-specific and species-specific data.

7.0 Deficiency and Toxicity of Selenium

7.1 Effects on Human Health

Globally, the Se status in human populations varies widely ranging from deficient to toxic; variation is due primarily to the amount of Se in our diets (Rayman 2012; Combs 2001). Selenium intake through food is dependent upon various factors within our diets including the consumption composition of different food groups and the locations where our food is produced due to geologic and environmental variables (MacPherson 1997; Fairweather–Tait *et al.* 2010; Rayman 2012). The body's ability to absorb, metabolize, and eliminate Se also contributes to Se status. Recent studies have focussed on the Se species that humans are exposed to in our diets because bioavailability, toxicity, and beneficial effects vary with different Se species (Rayman *et al.* 2008; Fairweather-Tait *et al.* 2010; Rayman 2012).

7.1.1 Exposure in Humans

For the vast majority of people living in North America, the primary source of exposure to Se is through food and dietary supplements (Health Canada 1992; ATSDR 2003; Health Canada 2010c). Food is estimated to account for more than 98% of the daily intake of Se (Health Canada1992). Although people may also be exposed to Se compounds in ambient air, drinking water, and soil, typically (though not always) these are insignificant contributions (IOM 2000).

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7.1.1.1 Dermal

Selenium is an ingredient in some antidandruff shampoos and antifungal skin creams (CCME 2009). Selenium compounds are not easily absorbed through the skin; there is little information concerning systemic effects of dermal exposure to Se compounds in humans (ATSDR 2003). Use of antidandruff shampoo containing 1% selenium sulphide by 150 individuals found no adverse effects after six weeks (Neumann *et al.* 1996). Acute dermal exposure to selenous acid or selenium dioxide causes burns and rashes (ATSDR 2003; Calello 2010).

7.1.1.2 Air

Exposure through inhalation is typically insignificant for the general population; however, occupations where people may be exposed via inhalation are the metal industries, Se-recovery processes, paint manufacturing and special trades (ATSDR 2003). Selenium compounds encountered in these settings include dusts of elemental Se, hydrogen selenide, and Se dioxide; the respiratory tract is the primary site of injury after inhalation but gastrointestinal, cardiovascular, and irritation of skin and eyes may also occur (ATSDR 2003). Quantitative data is not available primarily because of the possibility of concurrent exposure to other substances (ATSDR 2003). However, there have been reported exposures in occupational settings. At a copper refinery, exposure exceeding 0.2 mg Se/m^3 (amongst other contaminants in the air) increased nose irritation and sputum (Holness *et al.* 1989). CCME considers that typical background Se concentration in Canada is approximately 1.0 ng/m^3 (CCME 2009).

7.1.1.3 Selenium Species in Foods

Excellent reviews of the various species and concentrations of Se in foods and food supplements and associated health effects are provided by Rayman *et al.* (2008) and Fairweather-Tait *et al.* (2011). While all dietary forms of Se tend to be absorbed quite efficiently in the human body, the retention and use of organic forms tend to be higher than for inorganic forms (IOM 2000; Finley 2006; Calello 2010; Fairweather-Tait *et al.* 2010).

The Se content of the food we consume is a reflection of complex geologic and environmental factors (Combs 2001; Fairweather-Tait *et al.* 2010). Selenium concentrations in soils on which crops are grown (e.g., grains and cereals) or in fodder consumed by animals is a large contributor

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to the amount of Se found in our food (MacPherson *et al.* 1997; ASTDR 2003; Fairweather-Tait *et al.* 2011; Rayman 2012). The biosynthesis and metabolism of Se in plants and animals will affect the Se concentration and species present in plant and animal foods (Rayman 2012). For example, selenomethionine is the dominant Se form in cereals and grains; it is less prevalent in vegetables and some animal and fish species (Rayman *et al.* 2008; Fairweather-Tait *et al.* 2010). Selenomethionine accounts for approximately 55-85% of Se species in bread and wheat, 50-60% in meat, and 29-70% in fish (Fairweather-Tait *et al.* 2011). The major species in plant sources are selenate, selenomethionine and smaller amount of selenocysteine (Rayman *et al.* 2008). There is less information on the species of Se in dietary sources of animal origin (Rayman *et al.* 2008).

Grains and cereals grown in Canada, Australia, Japan and the US are known to be excellent sources of Se compared to similar crops grown in Europe (MacPherson 1997; Finley 2006; Rayman 2012). In fact, reductions in the importation of North American wheat to European countries has been linked to less than optimal Se status in humans in Finland and the UK thus prompting government to find strategies to increase levels (MacPherson 1997; Rayman 2000; Finley 2005; Reilly 2006).

7.1.1.4 Selenium Intake from Food

Through the Total Diet Study, Health Canada analyzes contaminants and chemicals in Canadian foods; in other countries these surveys are often called 'Market Basket Surveys Studies' (Health Canada 2012b). The Canadian Total Diet Study conducted by Dabeka (1994) found that 51% of the daily selenium intake (males and females of all age groups) was derived from baked goods and cereals. Dietary studies of the US population published by the Food and Drug Administration (FDA 1982) estimated that approximately 52% of Se intake was obtained from grains and cereals, followed by meat, fish, and poultry (36%), and dairy products (10%). A study in the United Kingdom estimated 26% of Se was obtained from bread and cereals, 26% from meat, 21% from dairy products, 10% from fish, with fruit, vegetables, eggs and other products accounting for the remaining contribution (Fairweather-Tait *et al.* 2011). Average dietary Se intake of Canadians was assessed in the most recent three surveys of trace elements (Table 7.1).

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Selenium status corresponds to intake; the Canadian Health Measures Survey measures Se in blood and urine of participating Canadian populations from the ages of 6 – 79 years (Health Canada 2010c). During the 2007-2009 survey cycle, the geometric mean and 90th percentile of Se in blood concentrations of populations of both males and females aged 6-79 years were 0.198 mg/L and 0. 236 mg/L, respectively (Health Canada 2010a). Calello (2010) reported the following as normal concentrations in US populations: whole blood 0.1 – 0.34 mg/L, serum 0.04 – 0.6 mg/L, urine < 0.03mg/L, and hair < 0.4 µg/g.

Table 7.1 Average dietary intake of Se has been published by Health Canada (2005; 2006; 2007a) for various age-sex groups.

Age-Sex Group	Reference Body Weights (kg)[30]	2005 (Toronto) (µg/kg bw/day)	2006 (Halifax) (µg/kg bw/day)	2007 (Vancouver) (µg/kg bw/day)
0-1 Mo (M&F)	7	5.0	4.9	7.9
2-3 Mo (M&F)	7	4.4	4.2	7.1
4-6 Mos (M&F)	7	4.2	4.2	8.2
7-9 Mo (M&F)	13	4.0	3.9	7.1
10-12 Mo (M&F)	13	4.0	4.1	7.0
1-4 Years (M&F)	13	5.7	5.7	8.7
5-11 Years (M&F)	27	4.6	4.4	6.5
12-19 Years (M)	57	3.1	3.1	4.5
12-19 Years (F)	57	2.3	2.2	3.2
20-39 Years (M)	70	2.6	2.7	3.7
20-39 Years (F)	70	2.1	2.1	2.8
40-64 Years (M)	70	2.1	2.1	3.0
40-64 Years (F)	70	1.7	1.7	2.4
All ages (M and F)		2.2	2.2	3.2

[30] Health Canada. 1994. Human Health Risk Assessment for Priority Substances. Minister of Supply and Services Canada, Ottawa, Ontario.

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7.1.1.5 Selenium Intake from Dietary Supplements

Dietary supplements can be a significant source of Se; generally the content of Se in multivitamins/multiminerals is ≤ 50 µg/tablet (Health Canada 2007b). Selenium supplementation in the form of selenised yeast can be as high as 200 µg/tablet in over-the-counter supplements (Health Canada 2007b). Other forms of Se are available in commercial mineral and vitamin foods sold in Canada.

7.1.1.6 Selenium Intake from Drinking Water

Drinking water was not considered as a significant exposure source of Se when the IOM developed dietary reference intakes (IOM 2000); these have been adopted by Canada (Health Canada 2003). Drinking water sources in Canada and the US usually contain very low or only trace amounts of Se (Health Canada 1992; ATSDR 2003; CCME 2009). For example, a survey of 122 drinking water supply systems in Canada found that Se levels were at or below 0.5 µg /L (Health Canada 1992). Source water sampling results provided by the Interior Health Authority from 761 small water systems (ie. serving less than 500 people) indicated that 13 systems had source water Se concentrations greater than 10 µg/L, 349 systems had concentrations between 1 and 10 µg/L, while the remaining 399 systems had concentrations less than 1 µg/L (J. Norlin, pers. comm., Interior Health Authority, September 2013). There are circumstances where the Se contribution from drinking water can be significant either due to natural or anthropogenic processes. For example, Valentine (1997) listed specific geographic regions in the United States where Se concentrations in surface, springs, and ground water sources were 8 to 180 times the EPA drinking water standard of 50 µg/L. Drinking water at these concentrations can result in dietary exposures exceeding the tolerable upper intake level of 400 µg/day. Inorganic forms of Se (e.g., selenate, selenite) are the primary water-soluble forms found in drinking water which are considered more toxic than organic forms (Health Canada 1992; OEHHA 2010).

7.1.2 Pharmacokinetics in Humans

Both organic and inorganic forms of Se are available in the human diet; absorption varies with species (IOM 2000; Calello 2010). Rayman *et al.* (2008) provides an excellent review of the species of Se and associated concentrations found in foods and food supplements. Absorption of all forms is efficient; elemental Se is least bioavailable (<50%) followed by inorganic selenite

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and selenate (75%), and organic Se (95%) (IOM 2000; ATSDR 2003; Finley 2006; Rayman *et al* 2008). Absorption may be affected by factors such as its chemical form, presence of protein, vitamin E, and vitamin A (Fairweather-Tait 1997). Animal studies have demonstrated that the primary site of absorption is the lower end of the small intestine (Reilly 2006). The specific mechanisms of absorption are not fully understood, however there is evidence that selenomethionine is absorbed by sharing an active transport mechanism with methionine, selenite is absorbed via passive diffusion, while other species such as selenate are absorbed from action of sodium-mediated carrier transport shared with sulphate (Vendeland *et al.* 1994; Fairweather-Tait 1997; Barceloux 1999; Reilly 2006).

Of interest in terms of health effects is the amount of Se that is bioavailable. This is defined by Fox *et al.* (2004) as that fraction of ingested nutrient that is utilised for normal physiological functions. Organic forms of Se such as selenomethionine are more bioavailable than inorganic forms such as selenate and selenite (Rayman *et al.* 2008; Fairweather-Tait *et al.* 2010). The bioavailability of Se in fish is not well studied, however recent research suggests it is dependent on species rearing location factors (Yoshida *et al.* 2011). Studies have found that Se bioavailability is low in tuna and other seafood products (Alexander *et al.* 1983; Wen *et al.* 1997; Yoshida *et al.* 2001; Fox *et al.* 2004) whereas Fox *et al.* (2004) found that in farmed trout, Se bioavailability to humans is comparatively high.

Selenium metabolism is partially determined by its chemical form (Rayman *et al.* 2008; Finley 2006; Combs 2001). Finley (2006) describes three potential fates of ingested Se: 1) as selenomethionine, it may be inserted into general proteins (i.e., not Se specific) as a substitute for methionine; 2) in salt form (selenite and selenate) it may be reduced to the selenide and then inserted into Se specific proteins (i.e., selenoproteins such as glutathione peroxidase; and 3) Se compounds (e.g., the species found in allium vegetables) may be reduced to selenide and ultimately excreted primarily through urine but a lesser amount in the breath.

Soluble Se compounds such as sodium selenate and sodium selenite are considered more toxic to humans than the organic forms (Yang *et al.* 1983; Health Canada 1992; Nagpal and Howell 2001). The amount of information available on the human toxicity of inorganic forms of Se in

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drinking water, independent of intake of organic Se from food sources is however limited (Health Canada 1992; OEHHA 2010). According to the IOM (2000) inorganic Se in humans can cause toxicity at much lower levels than is observed with organic forms of Se.

7.1.3 Essentiality and Deficiency

Selenium is known to be of fundamental importance to human health (IOM 2000; Rayman 2000; Reilly 2006). Selenium is a critical component of selenoproteins such as glutathione peroxidase; these function primarily in oxidation-reduction reactions (IOM 2000; ATSDR 2003). Selenoproteins are also a catalyst for the production of active thyroid hormone (Rayman 2000). There is substantial evidence that Se deficiency or low Se status is accompanied by loss of immunocompetence, disease progression of viral infections, risk of miscarriage, and cardiovascular disease (Rayman 2000; Reilly 2006). For example, low Se status in the general population of Finland has been linked to high cancer and cardiac mortality rates (Reilly 2006).

Selenium deficiency is rare in the modern North American diet because generally food is obtained from different geographic areas and food choices are plentiful. However, Se deficiency has been observed in areas of China where the soil Se levels are very low (< 0.125 mg Se/kg soil), the diet is almost entirely composed of locally produced food, and there is limited food variety (Whanger 1989; Combs 2001). Deficiency occurs when daily intake falls below 20 μg/day (Calello 2010). Keshan disease was discovered in the Keshan county of northeast China in the mid 1930s and it is linked to Se deficiency (<25 μg/day) in humans (Whanger 1989; IOM 2000). It is a cardio-myopathy characterized by an enlarged heart as well as abnormal ECG patterns, cardiogenic shock, and congestive heart failure, with multifocal necrosis of the myocardium (IOM 2000; ATSDR 2003). The disease is almost exclusively observed in children.

There is also evidence that Se deficiency may be related to a condition called Kashin-Beck disease which is characterized by atrophy, degeneration, and necrosis of cartilage tissue (IOM 2000; ATSDR 2003). The disease is endemic in an area of Asia consisting of east Siberia, North Korea, North Vietnam and northeast China, primarily in the Shaanxi province (Whanger 1989). The disease only occurs in Se deficient children, however, it appears to be triggered by an additional stress and not when in isolation (IOM 2000). Other causative factors hypothesized

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include high levels of fulvic acid in drinking water or deficiencies of other nutrients such as manganese (Reilly 2006). Improved Se nutritional status does not prevent Kashin-Beck disease and therefore the role of Se in this disease still remains somewhat uncertain (IOM 2000).

Since 1995, Health Canada harmonized the development of nutrient-based recommendations with the Food and Nutrition Board of the Institute of Medicine (IOM), National Academy of Sciences (Health Canada 2013a). Dietary reference intakes (DRIs) for Se were published in 2000 by the IOM (Table 7.2). According to Health Canada, DRIs are established using functional indicators of good health and prevention of chronic disease, as well as adverse health effects from excessive nutrient intakes. The IOM did not identify any age group as being more susceptible to the toxic effects of selenium when developing dietary reference intakes (IOM 2000).

Table 7.2 Selenium dietary reference intake values for humans (IOM 2000; Health Canada 2003).

Population	**Dietary Reference Values; Se (µg/day)**		
	EAR[1]	**AI[2] or RDA[3]**	**UL[4]**
infants 0 to 6 months	Has not been determined	*15**	45
infants 7 to 12 months	Has not been determined	*20**	60
children 1 to 3 years	17	20	90
children 4 to 8 years	23	30	150
children 9 to 13 years	35	40	280
adolescents 14 to 18 years	45	55	400
adults	45	55	400
pregnant women	49	60	400
lactating women	59	70	400

[1]*Estimated Average Requirements (EAR): a nutrient intake value that is estimated to meet the requirement of half the health individuals in a life stage and gender group.*
[2]*Adequate Intake (AI): a recommended intake value based on observed or experimentally determined approximations or estimates of nutrient intake by a group (or groups) of health people that are assumed to be adequate – used when a RDA cannot be determined.*
[3]*Recommended Dietary Allowances (RDA): the dietary intake level that is sufficient to meet the nutrient requirement of nearly all (97 to 98 %) healthy individuals in a particular life stage and gender group.*
[4]*Tolerable Upper Intake Levels (UL): the highest level of a nutrient intake that is likely to pose no risk of adverse health effects for almost all individuals in the general population. As intake increases above the UL, the risk of adverse effects increases.*

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7.1.4 Human Toxicity and Toxicological Reference Values

Ingestion of elemental and organic Se compounds is not known to cause acute toxicity (Calello 2010). Acute toxicity can occur following ingestion of inorganic forms: sodium selenite, sodium selenate, selenium dioxide, hydrogen selenide, selenic acid, and selenous acid (Calello 2010). Observations of acute oral toxicity (several 1,000 times the recommended dietary allowance) can induce vomiting, diarrhoea, nausea, and occasionally, cardiovascular issues in humans and laboratory animals (ATSDR 2003). For example, an accidental death occurred in Australia when a 75-year-old man consumed 10 g of sodium selenite (See *et al.* 2006). Three and half hours following ingestion, symptoms were abdominal pain, poor perfusion, and hypertension. The man died six hours after ingesting the sodium selenite of cardiac arrest. Experiments using laboratory animals indicate that sodium selenite is the most acutely toxic Se compound: oral LD_{50} for sodium selenite (mg/ Se kg body weight) reported as 4.8 – 7 (rats), 1.0 (rabbits), 3.2 (mice), 2.3 (guinea pigs) (ATSDR 2003).

Information about chronic Se intoxication effects in humans following oral ingestion is derived primarily from observations of selenosis in the Hubei Province of China (Brozmanová *et al.* 2010; ATSDR 2003). Daily dietary Se intake was estimated to range from 3,200 to 6,690 µg (Yang *et al.* 1983). Symptoms observed in individuals exposed to chronically high levels of dietary Se include loss of hair and nails, skin lesions, tooth decay, and abnormalities of the nervous system (ATSDR 2003). Unusually high levels of Se were measured in locally grown foods (e.g., vegetables and cereals) and drinking water (although few samples were collected, concentrations ranged from 117 – 159 µg/L from surface supplies). Based on studies from this region, Yang and Zhou (1994) determined that the onset of selenosis occurs at or above consumption of 910 µg Se per day; a NOAEL of 800 µg Se/day was derived. In developing dietary reference intakes, the Institute of Medicine (IOM) applied an uncertainty factor of 2 to Yang and Zhou's (1994) NOAEL, resulting in a tolerable upper intake level of 400 µg/day. The IOM (2000) considers this protective of sensitive individuals based on the toxic effects being not severe but also not necessarily reversible.

In 1991, EPA's Integrated Risk Information System (IRIS) developed an oral reference dose (RfD) of $5x10^{-3}$ mg/kg-day for selenium based on NOAEL for selenosis reported by Yang *et al.*

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(1989). RfD's are estimates (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

The ATSDR (2003) derived a chronic oral minimum risk level (MRL) of $5x10^{-3}$ mg/kg-day based on a NOAEL of 0.015 mg/kg-day for disappearance of symptoms of selenosis in recovering individuals reported by Yang and Zhou (1994). An oral MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure (ASTDR 2003).

Health Canada developed recommended dietary allowances for Se recognizing the margins between desirable and undesirable intakes and associated health benefits and adverse affects (Table 7. 2; Health Canada 2013a). Health Canada's (2010d) toxicological reference values (TRVs) provide a benchmark with which to assess the risks posed by environmental contaminants. For trace elements, the agency recommends using the upper tolerable intake level (UL) as the reference exposure level; the UL is applied as the tolerable daily intake. The department provides TDI's for various age groups (Table 7.3).

Table 7.3 Tolerable daily intake of selenium by age group (Health Canada 2010d).

Age-group (years)	Standard Body Weights (kg) [1]	Tolerable daily intake (µg/kg bw/d)
0-0.5	8.2	5.5
0.6-4	16.5	6.2
5-11	32.9	6.3
12-19	59.7	6.2
20+	70.7	5.7

[1] Standard body weights were derived from Health Canada (2010d).

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7.2 Toxicity in Aquatic Organisms

7.2.1 Mode of Action of Selenium

The importance of Se in the healthy functioning of organisms is based on the physiological association and involvement of selenoproteins. Selenium is incorporated as selenocysteine in a range of physiologically important selenoproteins which are required by all living organisms with the exception of higher plants and yeasts (ATSDR 2003). In mammals, there could be as many as 100 selenoproteins, 30 of which have been characterized and 15 of which have known biological function (Brown and Arthur 2001). Some of the first selenoproteins to be identified were a major class of glutathione peroxidase enzymes which act as antioxidants in cell cytosol, cell membranes, blood plasma, and the gastrointestinal tract, protecting cells from lysis or damage due to lipid peroxidation (the breakdown of cell membrane lipids by free radicals) (Brown and Arthur 2001; ATSDR 2003; Lemly 1998).

Selenium is an essential component of a major group of iodothyronine deiodinase enzymes involved in mediating production of thyroid hormone triiodothyronine (T3) from its precursor thyroxine (T4) (Brown and Arthur 2001). Thyroid hormones have a crucial role in cell function, tissue development, and physiology; they are required for proper immune system function, have a role in cell differentiation, and regulate the metabolic breakdown of protein, fat and carbohydrates (Brown and Arthur 2001, ATSDR 2003; Janz *et al.* 2010). Another selenocysteine-containing enzyme, thioredoxin reductase, catalyzes the reduction of thioredoxin which controls intracellular redox reactions and regulates the proliferation of normal cells. Thioredoxin reductase has also been associated with cancer cells when thioredoxin is in high concentrations (Brown and Arthur 2001).

Other selenoproteins are involved in Se transporting and mediating synthesis of selenocysteine (selenoprotein P), muscle metabolism (selenoprotein W), and the regulation of Se bioavailability by converting dietary selenocysteine and selenomethionine into other functional selenoproteins (ATSDR 2003; Janz *et al.* 2010; Brown and Arthur 2001).

An acute toxic response in aquatic organisms occurs at very high aqueous concentrations of Se, in the range of 100 mg/L and over (see Section 7.4.1, and Table 7.4), with the dietary exposure

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pathway in this case posing a much lower risk. Although the mechanism of acute toxicity is not completely understood, it is believed that oxidative stress is involved as the cellular mechanism in much the same way as chronic selenosis (Janz 2012). Chronic Se toxicity occurs at much lower Se concentrations, primarily from dietary sources (see Section 7.4.3.4). There have been three common theories on the mechanism for chronic Se toxicity published: 1) Se substitutes for sulphur (S) in proteins; 2) the inhibition of Se methylation results in the accumulation of reduced hydrogen selenide; and, 3) the theory which has gained acceptance as probably the fundamental cellular mechanism of toxicity, is oxidative stress resulting from high Se concentrations (Spallholz and Hoffman 2002; Miller *et al.* 2007; Janz *et al.* 2010).

The Se substitution theory is based on the knowledge that the proper functioning of proteins relies on a helical protein structure created by the disulphide, or sulphur-to-sulphur (S-S), linkages (Lemly 2002a). When Se is in surplus, sometimes not much over background, excess dietary Se is substituted for S into proteins. These Se-substituted proteins contain triselenium (Se-Se-Se) or selenotrisulphide (S-Se-S, also called 2-seleno-1,3-disulphide) linkages which, when translated during protein synthesis and incorporated into proteins, prevent the S-S bonds from forming thereby altering the structure of the proteins (Lemly 2002a). It is thought that the flawed proteins which are transferred maternally into eggs cause teratogenesis in the developing embryo leading to a variety of characteristic lethal and sublethal deformities (Lemly 2002a). These malformed proteins may also be responsible for pathological changes in the organs and tissues of juvenile and adult fish (Sorensen 1991; Lemly 2002a).

Substitution of Se for S in proteins however, is not be the only toxic mechanism (Janz *et al.* 2010). Spallholz and Hoffman (2002) suggest Se toxicity, in particular hepatic toxicity, may arise in organisms exposed to excess dietary selenocysteine. This leads to the inhibition of Se methylation (Se-detoxification process) of both organic and inorganic Se compounds and the subsequent accumulation of hydrogen selenides, an intermediate metabolite of the Se methylation process (Spallholz and Hoffman 2002). Inhibition of Se methylation leads to an excess of hydrogen selenides, a known contributor to hepatotoxicity (Nakamuro *et al.* 2000).

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Finally, the third theory suggests tissue damage is related to oxidative stress, which occurs when bio-reactive superoxides are produced in response to high Se concentrations (Lemly 2002a). This mechanism is now being proposed as possibly the initiating step in embryo mortalities and deformities in fish and birds (Spallholz and Hoffman 2002; Hoffman 2002; Palace *et al.* 2004; Miller *et al.* 2007; Janz *et al.* 2010; Janz 2012). The one common interaction that occurs in organisms during metal toxicity is the reaction between Se and thiols (sulphur containing organic compounds, R-SH), resulting in the formation of reactive oxygen species called superoxides (Spallholz and Hoffman 2002). Glutathione is a thiol with antioxidant properties which easily combines with some forms of Se, resulting in the production of free radicals that bind with and inhibit the normal function of cellular enzymes and proteins (Spallholz and Hoffman 2002; Palace *et al.* 2004). Glutathione peroxidase is a Se-containing enzyme responsible for reducing lipid hydroperoxides to their corresponding alcohols and reducing free hydrogen peroxide to water (Janz *et al.* 2010). Glutathione peroxidase however, is involved in both the removal of reactive oxygen species and their production, depending on the amount and chemical form of Se an organism is exposed to (Spallholz and Hoffman 2002). Selenium toxicity occurs when oxidative damage from reactive oxygen species exceeds the organism's antioxidant defence mechanisms (Spallholz 1994). More research is needed to determine the importance of oxidative stress as a mode of toxic action for Se in both teratogenic and other effects such as histopathological changes (lesions) and immune system dysfunction in juvenile and adult organisms (Palace *et al.* 2004; Janz *et al.* 2010).

7.2.2 Teratogenicity

Selenium can elicit reproductive effects in the form of teratogenic deformity, a more visually obvious biomarker of Se toxicity restricted to the embryo/larval life stages which can lead to reproductive impairment or failure in fish and birds (Lemly 1997b; Palace *et al.* 2004; Hamilton 2004). Evaluating the teratogenic effects resulting from the maternal transfer of Se to the offspring of fish and birds is a commonly researched reproductive toxic end point (DeForest 2008). For example, studies on resident rainbow trout conducted by Holm *et al* (2005) in Alberta documented teratogenic effects in fish captured below coal mining areas where water column Se concentrations were between 1 and 32 µg/L. The egg Se concentrations associated with a 15%

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increase in skeletal deformities above reference sites, were between 22.6 and 27 μg/g[31](Holm *et al.* 2005). Based on their results, Holm *et al.* (2005) recommended against calls for higher Se criteria for cold-water fish compared to warm-water species (Kennedy *et al.* 2000; Chapman 2007).

Selenium exposure can cause deformities in juvenile fish which have not been previously exposed through maternally transferred Se. A study by Teh *et al.* (2004) demonstrated that non-reproductive deformity in juvenile fish could occur, leading the authors to speculate whether the mechanism of the observed effects were related to excessive lipid peroxidation (Teh *et al.* 2004). Regardless, this strongly suggests Se exposure can result in teratogenic effects through reproductive and non-reproductive pathways. There is more discussion on this in Section 8.

The significance of teratogenic effects (embryonic deformity) in fish has been demonstrated in reproductive studies showing that few of the deformed individuals survive past the larval or juvenile stages (Gillespie and Baumann 1986; Hermanutz *et al.* 1992; Lemly 1993a). Hermanutz *et al.* (1992) found that larval fish with lordosis and haemorrhaging failed to survive more than one day, and edematous larvae surviving five days post-hatch failed to absorb their yolk sac and died before reaching swim-up stage. Other studies on fish embryo and larvae have also found that the presence of deformities in individuals resulted in significant increases in overall mortalities (Hamilton *et al.* 2005). Minnow and Paine, Ledge and Associates (2006) studied dwarf longnose sucker in coal mine-affected lentic waters in the Elk Valley BC. While no significant correlation was found between egg Se and larval mortality or deformity, correlation analysis suggested that larvae in groups that had high mortality tended to have more severe deformities, while survivors with lower mortality were associated with more mild or moderate deformities. Buhl and Hamilton (2000) found that Colorado pikeminnow (*Ptychocheilus lucius*) embryos with deformities may be more sensitive than normal embryos to fungal infection resulting in increased mortality in these individuals. The possible secondary effects of Se toxicity (deformity causing higher mortality through increased predation or higher incidence of fungal infection) requires further research.

[31] Converted wet weight to dry weight using 61% moisture content consistent with Holm *et al.* (2005).

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In fish, both Se-related deformity and reproductive failure may eventually alter the population structure through an absence of year classes (particularly young-of-year) and increases in the average age of the population (May *et al.* 2001). These population-level consequences are also dependent on many factors such as species fecundity, survival, reproductive life-span and immigration/emigration of species (May *et al.* 2001).

In birds, teratogenic effects are usually associated with low survival of hatchlings (Ohlendorf 2003). Direct and indirect mortality may lead to reduced recruitment, population declines and the eventual disappearance of species (Lemly 2002a; Ohlendorf 2003).

7.3 Factors Affecting Selenium Toxicity in the Aquatic Environment

There are many factors that influence the bioaccumulation of Se (see Section 6.2) and since many of these also control an organism's exposure, they can affect the toxic response to Se (Janz *et al.* 2010). Factors controlling physical and dietary Se exposure, such as mobility of the species feeding within or outside of Se contaminated areas, can result in a high degree of variability in Se exposure and, hence, the resultant ranges in Se concentrations in diagnostic tissues (Holm *et al.* 2003; Fairbrother *et al.* 1999). However, while mobility of an organism may complicate evaluation of Se exposure, it does not necessarily alter the relationship between Se accumulation and effects. The mobility of organisms has implications in evaluating site-specific exposure for some species, leaving uncertainty in environmental risk assessments.

Surface waters, particularly those that receive contaminants, contain a mixture of chemical constituents which can bind with Se, potentially attenuate or magnify toxic responses, or have no effect on Se toxicity depending on the substance (Eisler 1985). Constituents that may reduce Se toxicity include arsenic, antimony, bismuth, cadmium, copper, lead, germanium, mercury, silver, thallium, tungsten and zinc (Hamilton 2004; Janz *et al.* 2010; Heinz 1996). However, interactions between Se and other compounds are often unclear and inconsistent (see Section 6.2). For example, mercury accumulation has been shown to be blocked by excess Se. In bird studies, it has also been shown that the combination of Se with mercury may have an antagonistic toxic effect (Hamilton 2004). Heinz and Hoffman (1998) found that dietary Se had a protective effect against methylmercury poisoning in adult mallards, but in contrast combined Se

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and mercury diets had a synergistic effect demonstrated as reduced reproductive success and increased deformity in embryos.

Lohner *et al.* (2001a) hypothesized that an apparent lack of expected toxicity of Se to fish was due to the presence metals like arsenic, chromium, copper and nickel, which potentially formed complexes that interfered with Se accumulation and toxicity. Other elements such as antimony, bismuth, cyanide, germanium, silver and tungsten, have been shown to reduce the accumulation and/or toxicity of Se (Marier and Jaworski 1983; Hamilton 2004). Selenium has also been reported to decrease the toxicity of elements such as cadmium and thallium (Marier and Jaworski 1983). Some other elements such as chromium, cobalt, fluorine, molybdenum, nickel, tellurium, uranium, vanadium and zinc, may have little or no effect on Se toxicity (Marier and Jaworski 1983; Hamilton 2004).

Selenium is also thought to be erroneously taken up into cells via the sulphate uptake mechanisms, due to the similarity of selenate and sulphate (Ogle and Knight 1996). In the presence of elevated sulphate concentrations, the bioaccumulation and toxicity of selenate can be ameliorated (Ogle and Knight 1996; Hansen *et al.* 1993). However, the relationship between the reduction of Se bioaccumulation and toxicity in the presence of sulphate is not consistent (Besser *et al.* 1989; see the discussion on sulphate and selenate in Section 6.2.3).

Many other factors can influence Se toxicity including temperature, the chemical form of Se, exposure duration, nutritional status of the organism, differences in the sensitivity of species and life stages, differences between freshwater and marine environments, and other environmental stresses (Lemly and Smith 1987; Hamilton 2004). Organic forms of dietary Se, particularly selenomethionine, are more toxic to organisms than inorganic forms (Heinz *et al.* 1987; Kiffney and Knight 1990; Ingersoll *et al.* 1990; Maier and Knight 1993). Kiffney and Knight (1990) found that sublethal effects (reduced chlorophyll *a* production) in cyanobacteria occurred at exposure concentrations of 100 µg/L seleno-L-methionine, but for both selenite and selenate exposures, those effects were not apparent until concentrations reached 3,000 µg/L.

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Differences in toxicity observed in studies may not always be easily explained. Hamilton *et al.* (1990) conducted partial life cycle laboratory feeding studies on Chinook salmon (*Oncorhynchus tshawytscha*) to compare the effects of naturally incorporated forms of Se in diet with that of fish meal spiked with selenomethionine. The naturally Se-enriched diet resulted in a greater effect (reduced growth) in juvenile fish at both lower concentrations and over a shorter time period, than did a diet of Se-fortified fish meal. The authors offered three possible explanations for the increased toxicity in the diet with environmentally incorporated Se: 1) the diet may have contained other environmental contaminants (i.e., boron, chromium, and strontium); 2) other forms of Se (e.g., selenocysteine) may have been present in the diet of natural incorporated Se; and, 3) there could have been differential uptake, distribution or elimination of Se in fish fed the naturally incorporated Se diet (Hamilton *et al.* 1990).

7.4 Toxicity of Selenium to Aquatic Organisms

The most obvious effects of Se toxicity to aquatic biota are on reproduction which may be manifested as reduced survival and increased deformity in developing embryos (Lemly 1998). In general, the threshold for adverse effects in vertebrate animals may begin at Se concentrations less than an order of magnitude above normal background ambient concentrations (Eisler 1985; USDOI 1998; Ohlendorf 2003). Other estimates put that margin even smaller at only 2 to 5 times that of normal background (Maier and Knight 1994).

Aquatic species not only have species-specific sensitivity to Se, but may also have different chronic effect thresholds for non-reproductive and reproductive toxicological endpoints due to differences in exposure routes, exposure durations, forms of Se they are exposed to and modes of toxicity for these effects (Hamilton 2004; Janz *et al.* 2010). In studies that evaluate several reproductive endpoints (e.g., hatchability, deformity, and larval survival), hatchability has been shown to be a more sensitive reproductive endpoint than larval deformity in birds (Ohlendorf 2003) and fish (Crane *et al.* 1992; NewFields 2009; Nautilus Environmental and Interior Reforestation Co. Ltd. 2011). For example, hatching success was a more sensitive toxicological end point than larval development and survival in a study on perch, grass carp, and stickleback (Crane *et al.* 1992). NewFields (2009) conducted studies on brown trout (*Salmo trutta*) and determined EC10 for post-hatch survival was 17.68 (95% CI, 13.44 – 23.25) µg/g egg Se, while

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the EC10 for deformity was 19.33 (95% CI, 15.07 – 24.79) µg/g egg Se. Similarly, in a study on westslope cutthroat trout, hatchability (larval survival) was the more sensitive toxicity endpoint (Nautilus Environmental and Interior Reforestation Co. Ltd. 2011). However, deformity was the more sensitive endpoint than survival (hatchability) in studies by Holm *et al.* (2005) and McDonald *et al.* (2010). More research is needed to understand the relative sensitivity of Se in fish and wildlife species, as well as reproductive versus non-reproductive toxicological endpoints (Hamilton 2004; Janz *et al.* 2010).

7.4.1 Short-Term (Acute) Toxicity

Short-term Se exposure resulting in acute mortality typically occurs at much higher aqueous concentrations than chronic effects and results primarily from waterborne Se not dietary sources (Luoma and Presser 2009; Janz 2012). However, responses to short-term exposure of very high water Se concentrations will vary by species. In addition to species sensitivity, the form of Se, duration of exposure and endpoint being measured are factors that could influence final acute values.

Aquatic invertebrates are, for the most part, thought to be tolerant to aqueous Se, at least at concentrations that might result in dietary Se toxicity to fish and birds (deBruyn and Chapman 2007; Janz *et al.* 2010). Yet, some freshwater invertebrates are among the most sensitive and the most tolerant to acute concentrations of Se (USEPA 1987). Some acute toxicity thresholds for invertebrates are presented in Table 7.4. Water-only acute toxicity thresholds (96 hour LC50s for mortality) for selenite range from 210 µg/L for *Daphnia magna* (Adams and Heidolph 1985, as cited by USEPA 2004) to 203,000 µg/L for the leech *Nephelopsis obscura* (Brooke *et al.* 1985, as cited by USEPA 1987, 2004). Pieterek and Pietrock (2012) found differences between sensitivity in lab-reared versus field-collected *H. azteca*, to selenate, with the lab-reared individuals being approximately an order of magnitude more sensitive. In both groups however, mean survival times decreased as the exposure period increased to 10 days.

Bringmann and Kuhn (1977, as cited by USEPA 1987, 2004) reported a 24 hr EC50 of 9.9 µg/L selenite for immobilization of *D. magna,* indicating these invertebrates may indeed be much more sensitive to waterborne Se concentrations than other invertebrates. LeBlanc (1980) found

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that *D. magna* had a 24-hour LC50 of 660 µg/L which was a slightly higher value compared to a 48-hour LC50 of 430 µg/L (the form of the Se was not reported by LeBlanc (1980) but is listed as selenous acid in USEPA (2004) Table 1a).

Some marine invertebrate species may have a higher tolerance to Se. Martin *et al.* (1981) exposed embryonic Pacific oyster (*Crassostrea gigas*), blue mussel (*Mytilus edulis),* and Dungeness crab (*Cancer magister)* to Se dioxide (selenous acid), and reported all species had 96 hour LC50 values greater than 10,000 µg/L. On the other hand, juvenile bay scallop (*Aropecten irradians*) exposed to selenite had a short-term LC50 concentration of 255 µg/L based on static 96 hour laboratory tests (Nelson *et al.* 1988).

For fish species, short-term toxicity thresholds (Table 7.4) typically occur at much higher Se concentrations than those for chronic exposures. The US EPA (2004) reported that genus mean acute values (GMAV) for freshwater fish species exposed to aqueous selenite ranged from 1,783 µg/L for striped bass (*Morone saxatilis*), to 28,500 µg/L for bluegill sunfish (Table 7.5). However, there are reports of relatively low short-term Se thresholds. For example, larval haddock (*Melanogrammus aeglefinus*) exposed to selenous acid for 96 hours had a LC50 concentration of 600 µg/L (Cardin 1986, as cited by USEPA 1987). Cardin (1986, as cited by USEPA 2004) reported a LC50 for the more tolerant larval winter flounder *(Pseudopleuronectes americanus)* of 14,649 µg/L in water-only acute exposures of selenous acid.

Selenium toxicity thresholds for rainbow trout range from approximately 4,500 to 9,000 µg/L (Adams 1976, as cited by USEPA 2004; Buhl and Hamilton 1991). Short-term thresholds for coho salmon (*Oncorhynchus kisutch*) and Chinook salmon are reported to be 7,830 µg/L and 8,000 µg/L respectively (Hamilton and Buhl 1990; Buhl and Hamilton 1991).

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Table 7.4 Examples of short-term (acute) toxicological thresholds reported for some invertebrate species to water-only exposures of selenium.

Species	Toxicological Endpoint	Se concentration in µg/L (form of Se)	Reference[1]
Hyallela azteca (amphipod)	LC50 (96 hr mortality)	340 (selenite)	Halter *et al.* (1980) cited in USEPA (2004)
Hyallela azteca (amphipod)	LC50 (95% CI) (10-day, mortality) (strictly speaking not an acute test)	86 (55 – 180), (lab-reared) 574 (408 – 820), (field-collected), (selenate)	Pieterek and Pietrock (2012)
Cerodaphnia dubia (cladoceran)	LC50 (< 24 hr)	440 (selenite)	USEPA (2004)
Nephelopsis obscura (leech)	LC50	203,000 (selenite)	Brooke *et al.* (1985) cited in USEPA (2004)
Daphnia magna (cladoceran)	LC50 (mortality)	210 (selenite)	Adams and Heidolph (1985) cited in USEPA 2004
Daphnia magna (cladoceran)	LC50 (24 hr, mortality) LC50 (48 hr, mortality)	660 (selenous acid) 430 (selenous acid)	LeBlanc (1980)
Daphnia magna (cladoceran)	EC50 (24 hr immobilization)	9.9 (selenite)	Bringmann and Kuhn (1977) cited in USEPA (2004)
Mytilus edulis (blue mussel) *Crassostrea gigas* (Pacific oyster) *Cancer magister* (Dungeness crab)	LC50 (96 hr) (marine species, embryonic/larval)	> 10,000 (selenium dioxide/selenous acid)	Martin *et al.* (1981)
Aequipecten irradians (Atlantic bay scallop)	LC50 (96 hr)	255 (selenite)	Nelson *et al.* (1988)

[1]It should be noted that literature included in Table 7.4 was not classified since there are no short-term guidelines being proposed for Se. These data provide examples of the range of toxicity threshold reported in the literature.

Table 7.5 Examples of short-term toxicity thresholds for mortality in fish species in water-only exposures of selenium.

Species	Toxicological Endpoint	Se concentration in µg/L (form of Se)	Reference[1]
Oncorhynchus mykiss (rainbow trout, juvenile)	LC50 (96 hr)	4,500 – 9,000 (selenate)	Adams (1987) cited in USEPA (2004); Buhl and Hamilton (1991)
Oncorhynchus Tshawytscha (Chinook, fry)	LC50 (96 hr)	3,578 – 13,600 (selenite)	Hamilton and Buhl (1990)
Oncorhynchus kisutch (coho, fry)	LC50 (96 hr)	8,150 – 23,400 (selenite)	Hamilton and Buhl (1990)
Lepomis macrochirus (bluegill sunfish)	LC50 (96 hr)	28,500	Cardin (1986) cited in USEPA (2004)
Melanogrammus aeglefinus (haddock)	LC50 (96 hr), larval	600 (selenite)	USEPA (2004)
Morone saxatilis (striped bass)	LC50 (96 hr)	1,783 (selenite)	USEPA (2004)
Polyprion americanus (stone bass)	LC50 (96 hr)	14,649	USEPA (2004)

[1]Please note that literature included in Table 7.5 was not classified since there are no short-term guidelines being proposed for Se. These data provide examples of the range of acute toxicity threshold reported in the literature.

The US EPA (2004) acute criterion for aquatic organisms for selenite is 258 µg/L and for selenate (sulphate-adjusted) is 410 µg/L where sulphate is 100 mg/L. When environmental concentrations of Se reach potentially acutely toxic concentrations, which might occur below major industrial discharges, swift action must be taken by environmental managers to mitigate and remediate the effects.

7.4.2 Chronic Toxicity

Since ambient Se concentrations rarely reach levels that result in acute effects, the more common situation resulting in Se toxicity occurs at much lower chronic exposures (Sorenson 1991; Maier and Knight 1994; Lemly 1998). Environmental guidelines focus on chronic exposure concentrations to protect against sublethal effects.

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7.4.2.1 Microorganisms and Invertebrates

It is generally thought that bacteria, fungi, algae, and invertebrates are fairly tolerant to elevated Se concentrations, and the more important role these organisms play is in the rapid transformation and transfer of Se into the aquatic food web (Janz *et al.* 2010). However, these organisms should not be thought of as simple conduits for Se bioaccumulation, insensitive to Se toxicity. There is a high degree of variability in the toxic effects on algae and invertebrate taxa based on water Se concentrations, suggesting that Se uptake is very different among species at a given water concentration. In fact, microorganisms may also have differing nutritional requirements for Se. Doblin *et al.* (1999) conducted lab experiments on three estuarine phytoplankton species (*Gymnodinium catenatum*, *Alexandrium minutum* (Dinophyta) and *Chaetoceros* cf. t*enuissimus* (Bacillariophyta)) which commonly bloom in southern Australian waters. Phytoplankton were cultured in Se-deficient mediums to determine the requirements for Se based on growth rate and biomass yield. The authors found that the dinoflagellates *G. catenatum* and *A. minutum* had obligate and intermediate Se requirements, respectively, while the diatom *C. tenuissimus* showed no changes in growth or biomass after eight weeks in Se-deficient media.

Chronic toxicity thresholds for green algae species exposure to sodium selenite for greater than four days, ranged between 522 and 70,000 µg/L (USEPA 2004). However, Vocke *et al.* (1980) exposed four species of freshwater algae to sodium selenate and reported the 14-day EC50s for reduced growth were from 33 µg/L for *Ankistrodemus falcatus,* to 277 and 284 µg/L for *Selenastrum capricornutum* and *Scenedesmus obliquus*, respectively, and 8,511 µg/L for *Microcoleus vaginatus,* demonstrating the broad range in sensitivities of algae to Se (Vocke *et al.* 1980).

deBruyn and Chapman (2007) found that Se body burden concentrations in invertebrates resulting in sublethal toxic effects showed a relatively narrow range; 1 to 30 µg/g Se. *Chironomus* was shown to be among the most sensitive invertebrate taxon, when considered on a body burden basis (deBruyn and Chapman 2007, based on data in Ingersoll *et al.* 1990 and Maier and Knight 1993). Some invertebrate species may be at risk when Se is in the lower range of concentrations (3 to 11 µg/g Se) thought to elicit sublethal effects on fish and birds predators

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(deBruyn and Chapman 2007). By extrapolation, using a BAF of 1,000, deBruyn and Chapman (2007) estimated the range of water Se concentrations associated with sublethal effects were between 1 to 30 µg/L. These findings are important in the context of setting protective water quality guidelines.

There is evidence suggesting lethal and sublethal effects in some freshwater invertebrate species occur at Se concentrations below the acute and chronic LC50s reported in the literature. For example, Malchow *et al.* (1995) found that midge larvae fed a diet of ≥ 2.11 µg/g (seleniferous algae) showed significantly reduced growth at body burdens of ≥ 2.55 µg/g after only 96 hours of exposure. Anastasia *et al.* (1998) found that the early survival of fiddler crab larvae (*Uca pagnax*) may be reduced at body burden concentrations ranging from 2.4 to 268.5 µg/g.

Some researchers have found significant decreases in algal species diversity and benthic invertebrate diversity and abundance in streams below open-pit coal mining, related to increases in Se, nitrate, and sulphate. Hauer and Sexton (2010) observed decreased algal diversity and abundance at sites with the highest mean values of Se (10 µg/L), nitrate (2,000 µg/L), and sulphate (400 µg/L). Frenette (2008) conducted an invertebrate study below coal mining areas using a multimetric approach and demonstrated subtle changes in community structure, namely the loss of sensitive *Ephemeroptera* taxa and reduced number of EPT (total number of *Ephemeroptera*, *Plecoptera* and *Trichoptera*).

It should be noted that some field studies often do not distinguish between the effects of Se and the possible effects of another contaminant or the mixture of contaminants present. In studies using stream mesocosms, Swift (2002) found that density of isopods and tubificid worms was significantly decreased by exposure to both medium (10 µg/L) and high (30 µg/L) aqueous Se treatments but density was unaffected at 2.5 µg/L. More recently, Pond *et al.* (2008) found negative impacts to benthic invertebrate communities below coal mining activities in West Virginia, where several metrics such as *Ephemeroptera* generic richness, family richness, and the total number of *Ephemeroptera* and *Plecoptera* were strongly negatively correlated with Se and other water quality parameters.

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Not all invertebrates are susceptible to chronic low Se levels. For example, Brix *et al.* (2004) found that brine shrimp (*Artemia franiciscana*) had a NOEC and LOEC for Se of 3,000 µg/L and 8,000 µg/L, respectively, for both 11-day growth and 21-day reproduction endpoints. Brasher and Ogle (1993) found that *H. azteca* had 10-day LC50s to both selenite and selenate that were lower, 502 and 1135 µg/L, respectively.

7.4.2.2 Vertebrates

Oviparous (egg-laying) vertebrates are among the most sensitive organisms to Se toxicity (US DOI 1998; Palace *et al.* 2004; Janz *et al.* 2010). Of those, fish and bird species have the highest sensitivities to both Se-related embryo mortality and developmental deformity, although amphibians and reptiles may also be sensitive to Se (Janz *et al.* 2010).

7.4.2.3 Fish

Reproductive and non-reproductive toxic effects may be seen in fish from chronic Se exposure; (Lemly 2008; Janz *et al.* 2010). Reproductive effects are those originating from the maternal transfer of Se, while non-reproductive effects refer to the direct toxic impacts Se may have on juveniles and adults (Lemly 2008). Both reproductive and non-reproductive effects result primarily from the dietary intake of Se (DeForest and Adams 2011). There is also evidence that waterborne Se can elicit non-reproductive effects albeit at higher aqueous concentrations (Hunn *et al.* 1987; Cleveland *et al.* 1993; Hamilton 2004; Miller *et al.* 2007).

Chronic effects via multiple exposure routes can simultanously occur and be expressed at the molecular, biochemical, and cellular level right up to individual and population levels (Sorenson 1991; Lemly 1998; Lemly 2002a; Janz *et al.* 2010). Sublethal effects may occur at low Se concentrations, for example reduction in calcium concentrations in the vertebrae of rainbow trout fry, eye cataracts, and changes in gill and organ histopathology and blood characteristics (Hodson *et al.* 1980; Hilton and Hodson 1983; Hicks *et al.* 1984; Sorenson *et al.* 1984; Sorenson 1991). Other reported effects on early life stage and juvenile fish from chronic Se exposure at relatively low water (3–8 µg/L) and whole-body Se concentrations (1–15 µg/g), include reduced condition factor, growth and survival (Sorenson 1991; Lemly 2002a; Hamilton 2003).

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Lemly (2002a; 1997a) documented chronic effects on fish populations from Belews Lake, North Carolina, between 1975 and 1986. These included:

- swelling and inflammation of gill lamellae;
- elevated lymphocytes;
- reduced hematocrit and haemoglobin (anemia);
- pathological alterations in livers kidneys, hearts, and ovaries (e.g. vacuolization of parenchymal hepatocytes, intracapillary proliferative glomerulonephritis, severe pericarditis and myocarditis, necrotic and ruptured mature egg follicles);
- cataracts present on the cornea and lens;
- reproductive disruption including lack of fertilization, lower hatchability, and higher mortalities of eggs and alevins;
- exopthalus (protruding eyes) caused by edema[32] or fluid build-up within the eye socket;
- abdominal edema caused by fluid in the visceral cavity or abdomen;
- a range of teratogenic edema of the head; and,
- skeletal deformities commonly seen in the head, jaw, mouth and fins, and spinal deformities in the lumbar and caudal areas (kyphosis, lordosis and scoliosis).

While research has demonstrated the range of observed reproductive and non-reproductive effects in fish may be very broad, the literature also suggests that chronic effects observed in the early life and juvenile stages of fish are not consistent, likely as a result of differential rates of Se accumulation and/or different species sensitivity (Hamilton 2003). The most sensitive life stage to the toxic effects of contaminants is often the early life stage (Newman and Unger 2003). In studies conducted on larval razorback suckers (*Xyrauchen texanus*) fed Se-laden zooplankton, dietary exposures of 3 to 5 µg/g Se resulted in a fairly rapid increase in mortality (Hamilton *et al.* 2005). In a study on northern pike exposed to uranium mill effluent in lakes, significant increases in developmental deformities were associated with the medium and high Se concentration sites (approximately 0.7 and 2.7 µg/L, respectively) (Muscatello *et al.* 2006; 2008). However, there were no apparent differences in time to eyed-embryo, hatchability and swim-up stages among all

[32] Non-teratogenic edema versus that related to deformity is distinguished by excess fluid forming behind the eye and in the abdomen as a result of excess Se which distorts selenoproteins in the cell membrane structure causing damage to cell permeability making organs "leaky" (Lemly 2002).

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Se treatments, suggesting that embryonic deformity was the more sensitive indicator of toxicity in that fish species. Crane *et al.* (1992) found variable hatchability in perch (*Perca fluviatilis*), grass carp (*Ctenophryngodon idella*) and stickleback (*Gasterosteus aculeatus*) eggs raised in experimental ponds with Se concentrations up to 10 µg/L, but hatchability dropped to zero for all species at 25 µg/L. In this experiment, hatchability was a more sensitive reproductive endpoint than larval development, survival or teratogenic effects.

Although dietary Se is the primary route of exposure in fish, the water exposure route should not be ignored. Research assessing toxic responses in fish from water-only exposures has shown that early life stage and juvenile fish may be sensitive to Se when based on whole-body tissue accumulation (Hodson *et al.* 1980; Hamilton and Wiedmeyer 1990; Cleveland *et al.* 1993). In a study using water-only Se exposures Hodson *et al.* (1980) found a significant increase in eyed egg mortality of rainbow trout at Se exposures at or above 26 µg/L, decreases in red blood cell volume and serum iron concentrations at 53 and 16 µg/L, respectively, and a decrease in mean time to hatch at Se concentrations greater than 4.4 µg/L (significant decrease in mean time to hatch at 16.0 µg/L). At 44 weeks post-hatch, the mean whole-body tissue Se concentration estimated for rainbow trout exposed to the highest water Se concentration (53 µg/L) was 1.7 µg/g compared with control (0.4 µg/L Se) fish which had whole-body Se concentrations of 0.64 µg/g Se.[33]

Hamilton and Wiedmeyer (1990) conducted studies on Chinook salmon using water-only exposures of boron, molybdenum and Se. Boron and molybdenum body burdens in test fish were less than analytical detection limits leading the authors to conclude that these elements were not bioaccumulating and did not pose a significant risk of adverse effects. However, there was a strong correlation between increased body burden of Se and increasing water Se, with significant decreases in survival and growth in fish exposed to water concentrations of selenate and selenite at 50 µg/L or greater, and 9.3 µg/L or greater, respectively. Resulting whole-body no-effect concentrations in these studies were between 3 and 5 µg/g (Hamilton and Wiedmeyer 1990).

[33] Converted wet weight to dry weight assuming 75% moisture content.

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Cleveland *et al.* (1993) exposed juvenile bluegill sunfish to both water and dietary Se and found that water-only exposures decreased swimming behaviour at 170 µg/L, and significantly increased mortality at 680 µg/L Se (mostly as selenate).

Miller *et al.* (2007) exposed juvenile rainbow trout to a range of aqueous sodium selenite concentrations, incorporating both 96-hour acute test concentrations (390 and 2,670 µg/L, measured Se), and 30-day chronic test concentrations (50 and 160 µg/L, measured Se). Several physiological stress indicators such as plasma cortisol, plasma glucose, plasma T3 and T4 hormones, were activated in both acute and chronic exposures suggesting oxidative stress is an important mechanism in non-reproductive effects. Miller *et al.* (2009) conducted a study on white suckers exposed to agricultural drain water in Lethbridge Alberta. At Se concentrations in receiving waters of between 0.4 and 26.7 µg/L, plasma glucose concentrations were significantly positively correlated with Se concentrations in muscle between 1.5 and 6.4 µg/g ($p < 0.05$). Although white suckers did not exhibit the classic stress response (i.e., increased cortisol with increasing Se), the results suggested that the fish were mobilizing energy reserves as Se increased. These studies suggest waterborne Se as a component of contaminated effluents, is important to overall exposure, particularly in streams that are in close proximity to industrial discharges where fish reside.

Lohner *et al.* (2001a; 2001b; 2001c) conducted field studies on tolerant sunfish species in streams receiving coal fly ash, finding that fish in exposed streams had significant differences in some biochemical indicators and significant hematological changes, but without apparent changes in fish health condition indices at even the highest Se concentrations (32 ± 13 µg/L).

Thomas and Janz (2011) investigated behavioural and physiological changes in adult zebrafish (*Danio rerio*) fed a diet spiked with selenomethionine at 0 (control), 3.7, 9.6 and 26.6 µg/g over 90 days. They found that swimming performance was reduced significantly in all feeding groups, but particularly in the highest feeding group which had an almost 50% reduction in performance compared with control fish. Mean mortality was significantly greater in fish fed the highest Se diet, and all fish fed Se spiked diets showed increased energy stores (measured as triglycerides and glycogen), an indication that Se might interfere with normal energy homeostasis in fish.

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These studies demonstrate that chronic non-reproductive effects of Se on adult and juvenile fish can result from relatively low exposures of waterborne and dietary Se. Higher aqueous (and dietary) Se exposure concentrations may be relevant in areas in close proximity to industrial discharges where fish reside. While increased mortality responses are easily interpreted, the cost of changes in behaviour and physiology to an individual's health, and ultimately their success, is not known but may have negative consequences from an energetics perspective (Thomas and Janz 2011).

7.4.2.4 Birds

Many of the sublethal effects of Se in fish are similar to those found in birds (Lemly 1993a). Non-reproductive chronic effects in birds from Se exposure include reduced immune function, excess feather loss, liver lesions and necrosis, muscle atrophy and weight loss (Ohlendorf *et al.* 1988; Fairbrother and Fowles 1990; Ohlendorf and Heinz 2011).

The more sensitive chronic effects in birds are related to reproductive impairment. Reproductive impairment is a general term including decreased fertility, reduced egg hatchability (embryo mortality) and increased incidence of deformity in embryos including eyes, feet, legs, beak and skull (Ohlendorf and Heinz 2011). Studies on birds show that thresholds for reduced hatchability are usually below those for teratogenic effects (Ohlendorf 2003). Egg fertility in some bird species, such as American kestrel (*Falco sparverius*), is considered a more sensitive toxicological endpoint often not reported but distinct from embryotoxicity (Ohlendorf and Heinz 2011).

Egg Se concentration is the most sensitive and reliable measurement of avian Se exposure, with thresholds for reproductive impairment estimated to be between 3 and 8 μg/g, depending on the species and form of Se in the diet (Heinz 1996; Ohlendorf and Heinz 2011). It is thought that aquatic birds are more susceptible to Se toxicity than terrestrial birds species based on the lack of reproductive effects seen in meadow larks (*Sturnella neglecta*), and barn swallows (*Hirundo rustica*) studied at Kesterson Reservoir (Santolo and Ohlendorf 1994). This observation could be the result of lower exposure (different diet preferences) and/or species-specific differences in how Se is metabolised. It should be noted that sea birds are often less sensitive to Se than

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freshwater birds due to mechanisms thought to protect marine birds against metal toxicity (Burger *et al.* 1994).

In studies of water fowl in the Elk Valley, American coots accumulated more Se in eggs (mean egg Se = 29.6 µg/g) than Canada geese at the same site (mean egg Se = 17.1 µg/g). The authors suggest this was the result of the differences in feeding patterns (SciWrite 2004). Canada geese successfully hatched and raised four goslings to day 31, while the coots hatched seven eggs but no young were found. There was no conclusive evidence linking failure to successfully fledge young coots with Se concentrations in eggs (SciWrite 2004). However, other studies have reported comparable Se concentrations that were sufficient to cause toxicity to coots, which are among the most sensitive bird species to Se toxicity (Ohlendorf *et al.* 1986; Hoffman *et al.* 1988; Ohlendorf *et al.* 1988).

7.4.2.5 Amphibians and Reptiles

There is a scarcity of literature on the effects of Se on amphibians and reptiles (Hopkins *et al.* 2006; Janz *et al.* 2010). Amphibians and reptiles may respond in much the same way as fish and birds to dietary intake, bioaccumulation, and maternal transfer of Se to eggs and this may be important in the transfer of Se through food webs (Stewart *et al.* 2010).

In studies on the toxicological effects of Se on amphibians and reptiles, the majority have examined simple Se bioaccumulation (Hopkins *et al.* 2004, 2006; Bergeron *et al.* 2010) or effects from mixtures of contaminants in which case causal relationships with Se cannot be determined conclusively (Hopkins *et al.* 2006; Minnow Environmental 2006; Unrine *et al.* 2007; Janz *et al.* 2010). For example, Minnow Environmental (2006) conducted a study on Columbia spotted frog in the Elk Valley BC, below coal mining activites and found that higher Se concentrations may have contributed to increased mortality and deformity in tadpoles. However, the authors were unable to estimate toxicity thresholds due to the high variability in tadpole response at low Se concentrations. More research is needed to develop toxicologal thresholds for amphibians and reptiles.

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7.4.2.6 Mammals

Mammals are not as sensitive to Se as fish and birds (Ohlendorf 1989; Janz *et al.* 2010). In studies conducted between 1984 and 1986 at Kesterson National Wildlife Refuge in central California, concentrations of Se were measured in various tissues (blood, liver, hair) and feces of 10 species of small mammals (Ohlendorf 1989). Results demonstrated a strong relationship to environmental Se concentrations, with the highest tissue Se concentrations associated with the most contaminated sites (Ohlendorf 1989). In spite of higher Se bioaccumulation in the exposed mammals, there were no apparent changes in the health of the organisms between exposed and reference areas. The exception was that no pregnant voles or mice were found in Kesterson, whereas pregnant individuals were found in the reference area. Although this finding might suggest reproductive failure, Se could not be definitively linked to the observation (Ohlendorf 1989). In comparison, at the same locations there were overt and often severe signs of acute and chronic Se toxicity observed in both fish and birds (Ohlendorf 1989).

7.4.3 Chronic Toxicity Thresholds for Selenium

There are several documents that summarize the existing toxicity data and provide effect thresholds or risk benchmarks for evaluating Se concentrations in the various environmental compartments including water, sediment, and tissue (Skorupa *et al.* 1996; Lemly 1996a, 1996b; US DOI 1998; Fairbrother *et al.* 1999; DeForest *et al.* 1999; Hamilton 2003; Ohlendorf and Heinz 2011; DeForest and Adams 2011).

7.4.3.1 Water

Adams *et al.* (1998) recommend a water Se criteria of 6.8 µg/L (lower 10th percentile of the distribution) which was the concentration the authors associated with a threshold concentration for reproductive effects in bird of 20 µg/g egg Se, based on modelling of data from different bird species and sites across the US. However, the majority of research supports a waterborne Se effect threshold for both fish and birds of approximately 2 µg/L or less (Lemly and Smith 1987; DuBowy 1989; Peterson and Nebeker 1992; Maier and Knight 1994; Lemly 1996a; Skorupa *et al.* 1996; Hamilton and Lemly 1999; Swift 2002; Hamilton 2003; Paveglio and Kilbride 2007). Table 7.6 provides a summary of these data.

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Hodson *et al.* (1980) exposed newly fertilised rainbow trout eggs from naive parental stock to water-only lethal and sublethal concentrations of sodium selenite over 44 weeks. The results showed that Se was lethal to trout at concentrations between 2,000 and 16,000 µg/L, but sublethal physiological effects were evident above 4.3 µg/L. Chronic responses included decreased median time to hatching at ≥4.4 µg/L, increased developmental rate at ≥16 µg/L, and increased in eyed-egg mortality at ≥28 µg/L Se. Exposed fish had increased body burden Se with increasing water Se concentrations, demonstrating Se bioaccumulated in tissue from a water-only exposure.

Dubowy (1989) developed dietary bioaccumulation models to predict the water concentration required to protect sensitive hervbivorous birds. The results showed that water Se greater than 2.8 µg/L would result in dietary Se (10 µg/g) sufficient to cause negative reproductive effects in ducks. Hermanutz *et al.* (1996) conducted studies on bluegill sunfish using stream mesocosm. They found adverse effects on early life stage and juvenile fish at Se test concentrations of 2.5 and 10 µg/L. Swift (2002) conducted experiments using stream mesocosms and found that isopod and *Tubifex* populations were dramatically reduced at the high (30 µg/L) and medium (10 µg/L) Se concentrations[34] but not at low (2.5 µg/L) exposures, although other invertebrate species were not affected even at high concentrations. Based on this study, Swift (2002) recommended a water quality criterion of 2 µg/L to protect sensitive fish and invertebrate species. This research demonstrates that a range of sublethal and lethal effects can occur in sensitive organisms at environmentally-relevant aqueous Se concentrations resulting in negative consequences to long-term health, survival and productivity.

The toxicity of Se is dependent on bioaccumulation primarily driven by dietary uptake (Luoma and Rainbow 2008). However, certain studies indicate a water concentration of 2 µg/L may not be sufficiently protective when bioaccumulation of Se in the food web is considered. Some researchers have found negative effects on biota in ecosystems with very low water Se concentrations, at or below 2 µg/L (Pease *et al.* 1992; Crane *et al.* 1992; Peterson and Nebeker 1992; Skorupa *et al.* 1996; Swift 2002; Rudolph *et al.* 2008). Crane *et al.* (1992) conducted ecosystem-scale studies on Se bioaccumulation and the effects on biota using freshwater

[34] Mesocosms dosed with selenium as sodium selenite.

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experimental ponds. They reported that spinal deformities in larval perch increased in eggs that were laid in ponds with 2 µg/L and 10 µg/L compared to control ponds; none of the eggs from ponds with 25 µg/L Se hatched.

Rudolph *et al.* (2008) found that at O'Rourke Lake, a small reference lake with Se concentrations <1.0 µg/L, the mean concentration of westslope cutthroat trout egg tissues was 14.11 (± 1.42) µg/g. The mean (SD) hatchability in fish eggs collected from O'Rourke Lake was 72.6 % (± 27.2%), which was lower (but not significantly different) than mean (SD) hatchability in eggs collected at the exposed lake 83.3 % (± 13.9%). Although this data may suggest that very low waterborne Se (<1 µg/L) can lead to reproductive effects from bioaccumulation of Se in diet, there is uncertainty in the interpretation of these results. O'Rourke Lake contained no resident fish (Elkford Rod and Gun Club 1984) prior to a stocking program which introduced westslope cutthroat trout on three occasions between 1985 and 1992 (BC Environment, Fish and Wildlife Branch internal files). Therefore, classifying O'Rourke Lake as a natural reference site may be questionable as the lake did not support fish prior to their introduction.

Lemly (1996a) cautioned that where circumstances favour transformation and uptake of Se from water, concentrations as low as, or in the case of O'Rourke Lake, less than 1 µg/L, could lead to enhanced bioaccumulation and unanticipated adverse effects. Peterson and Nebeker (1992) developed a model using existing data to estimate waterborne Se toxicity thresholds for a range of wildlife that included marsh wrens (*Cistothorus palustris*), belted kingfisher (*Ceryle alcyon*), mallard, osprey (*Pandion haliaetus*), bald eagle (*Haliaeetus leucocephalus*), bats (*Myotis* spp.), shrews (*Sorex* spp.), mink (*Neovison vison*) and river otter (*Lontra canadensis*). They found that the waterborne toxicity estimates for sensitive birds and mammals clustered around 1 µg/L; the range for all species evaluated was 0.7 to 2.1 µg/L.

Based on this work, authorities in California have identified the San Francisco Bay estuary as one that is extremely sensitive to bioaccumulation of Se in the food web (Pease *et al.* 1992). The San Francisco Bay Regional Water Quality Control Board recommended a site-specific water quality criterion of 0.1 to 0.8 µg/L, limiting Se bioaccumulation and tissue concentrations to below levels that might harm fish, birds and humans (Pease *et al.* 1992).

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Table 7.6 Examples of literature-based chronic effect thresholds, risk benchmarks and concentrations of concern for water.

Water thresholds	Selenium concentration (µg/L)	Reference
San Francisco Bay estuary site-specific Se criteria	0.1 – 0.8	Pease *et al.* (1992)
Freshwater thresholds for the protection of fish, birds and other wildlife	≤ 1[1]	Crane *et al.* (1992); Peterson and Nebeker (1992); Lemly (1996)
	2	DuBowy (1989); Maier and Knight (1994); Lemly (1996); Skorupa *et al.* (1996); USDOI (1998); Hamilton and Lemly (1999); Swift (2002); Hamilton (2003); Paveglio and Kilbride (2007)
Lower 10th percentile water Se associated with mallard egg Se = 20 µg/g	6.8	Adams *et al.* (1998)

[1]Lemly (1996a) recommended <1 µg/L for organic forms of water column Se as the threshold for food-chain bioaccumulation and reproductive failure in fish and wildlife. Organic Se forms will represent on a fraction of total water column Se.

7.4.3.2 Sediment

Sediments represent an important repository for Se cycling in aquatic ecosystems. While sediment Se concentrations can be highly variable, both temporally and spatially, evaluation of concentrations is extremely important in understanding the potential environmental threats of Se (Canton and Van Derveer 1997; Lemly 2003). Research conducted on lakes influenced by uranium mining in northern Saskatchewan identified strong relationships between dissolved Se in the water column and total Se concentrations in sediments ($r^2 = 0.98$, $p < 0.05$, Figure 7.1) (Wiramanaden *et al.* 2010). In spite of the data variability, this study was able to show that sediment Se increased proportionally with increasing water Se, suggesting that the major mechanism for incorporation of Se into sediments is through the transformation of Se from the water column by primary producers. The authors also found a strong relationships between dissolved Se in pore-water and overlying surface water ($r^2 =- 0.95$, $p < 0.05$). The same study showed strong relationships between mean whole-body chironomidSe concentrations and dissolved Se in the water ($r^2 = 0.91$, $p < 0.05$), and with whole sediment Se concentrations ($r^2 = 0.80$, $p < 0.05$). A very strong relationship between chironomid tissue Se and dissolved Se in

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sediment pore-water was also reported ($r^2 = 0.99$, $p < 0.05$) (Wiramanaden *et al.* 2010). These results demonstrate the importance of water column Se as the source for sediment and sediment pore-water in Se cycling and the subsequent uptake and bioaccumulation of Se in invertebrates.

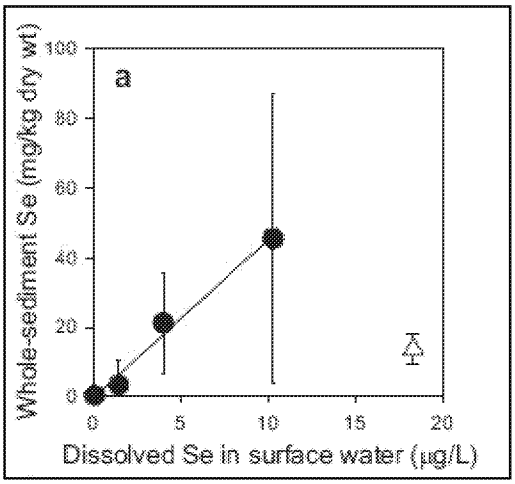

Figure 7.1 Relationship between selenium concentrations in whole sediment and mean surface water selenium for lakes influenced by uranium mining in northern Saskatchewan. Error bars = 1 SD. Hollow triangle is data for Wolf Lake, which was not included in the regression, $r^2 = 0.95$, $p < 0.05$ (from Wiramanaden *et al.* 2010).

Recognizing the link between Se in sediment and bioaccumulation of Se in the food web, some authors have suggested toxicity thresholds or concentrations of concern related to sediment Se concentrations (IJC 1981; Lemly and Smith 1987; Van Derveer and Canton 1997; US DOI 1998). These are summarised in Table 7.7. Some of the earliest recommendations for sediment quality guidelines for Se came from the International Joint Commission's Aquatic Ecosystem Objectives Committee, who stated that sediment Se concentrations should not exceed 5 µg/g for the protection of aquatic life (IJC 1981). Lemly and Smith (1987) recommended that sediment Se concentrations greater than or equal to 4 µg/g represent a concentration of concern. Hamilton (2004) suggested a sediment toxicity threshold for Se of 4 µg/g, but cautioned that emerging research might reveal that the threshold is lower in some ecosystems. Van Derveer and Canton (1997) compiled data from 25 study sites across the US and calculated a sediment Se toxicity threshold of 2.5 µg/g.

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Table 7.7 Examples of literature-based sediment selenium toxicity thresholds suggested for the protection of fish and wildlife.

Sediment Thresholds	Selenium Concentration (µg/g dw)	Reference
Lowest effect level (LEL)	0.9 – 1.9[1]	Thompson *et al.* (2005)
Toxic threshold value	2.0	Lemly (2003)
EC10 (fish and birds)	2.5	USDOI (1998)
Predicted effects threshold Observed effects threshold	2.5 > 4	Van Derveer and Canton (1997)
Level of concern	≥ 4	Lemly and Smith (1987)

[1]Range of lowest effect levels (LEL) for benthic invertebrates in uranium mining-affected sites derived using "closest observation" and "weighted " methods (Thompson *et al.* 2005).

Reliance on sediment concentrations as a predictor of Se toxicity is problematic since sediment Se and conditions that result in Se bioaccumulation can be highly variable. Studies comparing sediment Se concentrations to effects show that toxicity is affected by many factors such as total organic carbon (Van Derveer and Canton 1997;USEPA 1998; Hamilton 1999; Hamilton and Lemly 1999; Wiramanaden *et al.* 2010). However, multiple indicators of exposure such as Se concentrations in water, suspended particulates, sediments, and diet, and their associated benchmarks, are important in identifying potential risks, especially if Se concentrations in reproductive tissues are unavailable (Hodson *et al.* 2010). Currently, the only existing Canadian federal or provincial sediment Se quality guidelines are those developed by the BC MoE of 2 µg/g (Nagpal and Howell 2001).

7.4.3.3 Microorganisms and Invertebrates

Chronic toxicity thresholds for microorganisms and invertebrates are presumed much higher than thresholds for fish or birds that consume them (Section 7.4.3.4). There are no general published toxicity thresholds for invertebrates, partly because there is such a wide range of responses and a similarly wide range of reported values for effects across species. Table 7.8 summarises some of the published invertebrate effect thresholds. However, as previously mentioned, there is some evidence that sensitive invertebrate taxa may have chronic toxicity thresholds for Se that are similar to or below the dietary toxicity thresholds of their predators, based on whole-body Se

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concentrations (deBruyn and Chapman 2007). Other examples of benthic community shifts and losses of sensitive invertebrate taxa below coal mining areas (Section 7.4.2.1) suggest that more research is necessary to determine a chronic effect thresholds for Se in sediment applicable to a broad array of invertebrate taxa and their life stages (Frenette 2008; Janz *et al.* 2010).

Table 7.8 Examples of some suggested literature-based selenium toxicity thresholds for invertebrates.

Invertebrate Thresholds	Selenium Concentration	Reference
Internal Se tissue concentration associated with sublethal effects	1 – 30 µg/g dw	deBruyn and Chapman (2007)
Water concentration to protect sensitive taxa	2 µg/L	Swift (2002)

7.4.3.4 Dietary

Dietary Se toxicity thresholds are not a direct a measure of toxicity, but measurements in prey organisms can provide an alternative to direct monitoring efforts (Lemly 1996). Minnow (2009) and Minnow *et al.* (2011) recommend targeting macroinvertebrates because 1) they can be frequently sampled with less environmental impact than is associated with gathering fish or bird egg tissues; 2) are not considered charismatic fauna; and, 3) concentrations are a direct reflection of dietary Se levels providing a better indication of localised changes in food web Se, than can be provided by monitoring fish species with large foraging areas.

Reported thresholds for Se in dietary tissues to prevent bioaccumulation leading to toxic responses in sensitive fish species (i.e., Se concentrations in prey organisms) range from 10 and 11 µg/g for warm and cold water fish species respectively, to 3 and 4 µg/g or less, for freshwater and anadromous fish to (DeForest *et al.* 1999; US DOI 1998; Lemly 1996). The dietary thresholds for fish reported in literature are summarised in Table 7.9, demonstrating that a majority of these are 3 to 4 µg/g Se (Hamilton 2004).

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Hilton *et al.* (1980) conducted a 20-week study on rainbow trout to investigate the physiological response to dietary Se deficiency and toxicity, and attempt to quantify the dietary requirement for Se. These authors stated that optimum dietary Se was between 0.15 and 0.38 µg/g Se based on maximum plasma glutathione peroxidase activity. Fish fed a diet supplemented with 13 µg/g Se displayed reductions in growth, poor feed efficiency and higher mortalities than all other test groups. Based on lower glutathione peroxidase activity in the test groups fed the 3.67 and 13 µg/g Se diets, the authors believed that dietary Se in excess of 3 µg/g, over long term exposures, may be toxic to rainbow trout. Hilton and Hodson (1983) conducted a 16-week feeding study on juvenile rainbow trout and found that kidney tissues had significantly higher calcium and phosphorus levels in test groups that had diets supplemented with 5 and 10 µg/g Se, indicating that renal calcinosis was occurring. They also found that trout in both the low and high carbohydrate test groups having diets supplemented with highest level of Se (10 µg/g Se) had significant reductions in body weight and a higher feed:gain ratio than all other test groups. Cleveland *et al.* (1993) found that juvenile bluegill sunfish fed a diet enriched with seleno-L-methionine for 90 days in flow-through experiments experienced a significant increase in mortality at 6.5 µg/g or greater, although the response was not always consistent.

Hamilton *et al.* (1990) fed swim-up larval Chinook salmon diets containing either Se-contaminated fish meal (fish from San Luis Drain, SLD diet) or diets fortified with selenomethionine (SeMet diet) for a 90 day period. They found that the growth of larval fish fed the SLD diet was significantly reduced at dietary concentrations between 3.2 and 5.3 µg/g; these same larval fish had mean whole-body Se concentrations of 4.0 µg/g (Hamilton *et al.* 1990). The Hamilton *et al.* (1990) study was criticised by DeForest *et al.* (1999) since the San Luis Drain fish may have contained other co-contaminants. However, fish fed 9.6 µg/g Se in either diet (SLD or SeMet diets) had significantly increased mortality at mean whole-body Se levels between 5.4 and 6.5 µg/g (Hamilton *et al.* 1990). Once again, the results for the 90-day study were criticised due to increased mortality between 60- and 90-days intervals in all groups including the controls, suggesting other factors may have been in part, responsible for mortalities (DeForest *et al.* 1999). Therefore, use of the 60-day results reported by Hamilton *et al.* (1990) has been considered acceptable (DeForest *et al.* 1999; DeForest and Adams 2011).

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Table 7.9 Examples of literature-based dietary Se toxicity thresholds for the protection of fish and birds.

Dietary Thresholds	Selenium Concentration (µg/g dw)	Reference
For protection of fish	3 3 – 9 12 – 14	Lemly (1993a, 1996) Hilton *et al.* (1980); Hilton and Hodson (1983); Hamilton *et al.* (1990); Cleveland *et al.* (1993); Jasonsmith *et al.* (2008) Janz *et al.* (2010)
For protection of fish and/or birds	4[1,2]	Moore *et al.* (1990); Maier and Knight (1994); Wayland *et al.* (2007)
For protection of poultry	5	Puls (1994)
For protection of sensitive bird species	4.8 (CI 3.6 – 5.7) 4.4 (CI 3.8 – 4.8)	Ohlendorf (2003) Ohlendorf (2007) Ohlendorf and Heinz (2011)

[1]Dietary concentrations below 3 µg/g were not associated with adverse effects and concentrations above 4 µg/g were associated with adverse effects (Maier and Knight 1994).
[2]Dietary EC10 for hatching failure published by Wayland *et al.* 2007.

In a more recent study, Jasonsmith *et al.* (2008) found that deformity rates were between 11–12% (gross spinal deformity) in flathead gudgeon at dietary concentrations for that species that ranged from 3.1 to 8.3 µg/g. These examples represent some of the lower dietary toxicity thresholds reported in the literature for fish.

Dietary toxicity thresholds for birds are summarized in Table 7.9. Wayland *et al.* (2007) carried out an assessment of the potential risk that may be posed by dietary Se by combining invertebrate Se data and modelling Se exposure to evaluate possible toxicity for two aquatic bird species. They found that 4.0 µg/g (95% CI of <0.5 - 7.3) was the dietary EC10 for reduced hatchability in American dipper and harlequin ducks (*Histrionicus histrionicus*). Puls (1994) reported that poultry, which are particularly sensitive to Se, have an adequate dietary intake of 0.3 to 1.1 µg/g; diets between 3 and 5 µg/g are excessive but not necessarily toxic. Dietary concentrations over 5 µg/g do not necessarily affect adult laying hens but results in reduced hatchability and embryonic deformity (Puls 1994). Ohlendorf *et al.* (2008) and Ohlendorf and

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Heinz (2011) provide good summaries of the dietary thresholds for birds ranging from 3.0 to 8 µg/g, with most EC10 values in the range of 4.0 to 4.9 µg/g.

In a study conducted on spotted sandpiper in the Elk Valley, BC, there was a highly significant 15% decrease in hatchability at exposed sites compared with reference (X^2=9.6, $p < 0.01$) (Harding *et al.* 2005). The mean Se concentrations measured in the diet (benthic invertebrates) of sandpipers at the two exposed sites were 4.7 and 10.2 µg/g. The lower of these two sites is within the low range of the recommended dietary toxicity thresholds for birds (Harding *et al.* 2005). This is a good example of the applicability of a dietary threshold for resident BC birds.

7.4.3.5 Fish Tissues

The bioaccumulation of Se is important in determining toxicity, therefore, both tissue and dietary Se threshold concentrations are considered more appropriate and reliable than water or sediment for predicting the effects from Se (DeForest *et al.* 1999; Hamilton 2003; Ohlendorf and Heinz 2011). For fish, whole-body, liver, muscle, ripe ovary, and egg tissues have been used to evaluate dose-response relationships and estimate toxicity thresholds. These thresholds are summarised in Table 7.10.

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Table 7.10 Examples of literature-based selenium toxicity thresholds for fish.

Fish Thresholds	Selenium Concentration (µg/g dw)	Reference
Whole-body	4	Lemly (1996a); Hamilton (2002)
	4 –6	Ohlendorf *et al.* (2011)
	6 (cold water fish) 9 (warm water fish)	DeForest *et al.* (1999); Brix *et al.* (2000)
	8.1	DeForest and Adams (20121)
Muscle	8	Lemly (1996a)
Liver	12	Lemly (1996a)
Egg/ovary	10	Lemly (1996a)
	17	DeForest *et al.*(1999); Brix *et al.* (2000); DeForest&Adams (2011)
	20	DeForest *et al.* (2012)
Modelled population density effect threshold (approximate EC10) as egg Se in westslope cutthroat trout	28	deBruyn (2009)

Reported whole-body tissue toxicity thresholds for fish range from 3 to 4 µg/g, not distinguishing between warm or cold-water fish (Lemly 1996a; Hamilton 2003) to 6 µg/g for larval cold-water anadromous fish and 9 µg/g for warm-water fish (DeForest *et al.* 1999). DeForest and Adams (2011) recommended a whole-body Se toxicity threshold for fish of 8.1 µg/g.

Different species of fish may have different toxicity thresholds for Se. Hamilton *et al.* (1990) conducted feeding studies on Chinnok salmon and after 60 days of exposure (SeMet diet) found mortality increased significantly at mean whole-body Se concentrations between 5.4 and 6.5 µg/g. On a whole-body tissue basis, two studies on mosquitofish (*Gambusia affinis*) suggest this species is fairly tolerant to Se (Lemly 2002a; Saiki *et al.* 2004). In Belews Lake, North Carolina, mosquitofish were the single resident species of fish that persisted after 19 other species were

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extirpated due to Se toxicosis (Lemly 2002a). Another study in California showed no observed negative consequences to reproduction in adult mosquitofish with whole-body Se as high as 17.5 µg/g (Saiki *et al.* 2004). On the other hand, bluegill sunfish, in the same Order (Cyrpinodontiformes) as mosquitofish, have much lower whole-body toxicity thresholds reported for reduced winter survival of between 5.9 and 9.6 µg/g (Lemly 1993b; McIntyre *et al.* 2008). On an egg Se basis, McDonald *et al.* (2010) reported an EC10 for larval deformity in Dolly Varden char (*Salvelinus malma*) of 54 µg/g Se. Brown trout and rainbow trout and were found to be highly sensitive to Se toxicity, with egg Se EC10s for larval survival and deformity in the range of 17 to 22µg/g Se, respectively (Holm *et al.* 2005; NewFields 2009). This shows the range of Se toxicity, even within fairly closely related genera, may be relatively wide.

The US EPA (2004) released a draft Se tissue criterion for whole-body fish tissue of 7.91 µg/g, and 5.85 µg/g as a summer and fall concentration that should trigger further monitoring in winter. These numbers were based on the results of Lemly (1993b), which showed increased sensitivity to Se toxicity in bluegill sunfish when fish were additionally stressed by low temperatures. Previously, the BC MoE published a whole-body tissue Se guideline for fish of 4 µg/g (Nagpal and Howell 2001).

Tissue effect thresholds for egg and/or gravid ovary tissue in fish have also been suggested, ranging from 10 µg/g for overall health and reproductive vigour (Lemly 1996), to 17 µg/g for larval mortality (DeForest *et al.* 1999; DeForest 2008; DeForest and Adams 2011). Most recently 20 µg/g was recommended by DeForest *et al.* (2012) as a possible guideline for Canada using a species sensitivity distribution approach. While these are meant to be generally applicable thresholds, there is a range of species sensitivity to Se which is evident when comparing published studies reporting egg tissue thresholds. Holm *et al.* (2005) compared Se-related deformity in two species of salmonids from streams in Alberta and found that rainbow trout (*Oncorhynchus mykiss*) had lower toxicity thresholds than brook trout (*Salvelinus fontinalis*).

Figure 7.2 shows the distribution of reported toxicity thresholds for fish species (Janz *et al.* 2010; DeForest and Adams 2011). While salmonids are thought to be among the most sensitive fish species to Se toxicity, a study on Dolly Varden char (*Salvelinus malma*) reported an EC10 for

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larval deformity of 54 µg/g Se in eggs, not shown on the figure (McDonald *et al.* 2010). Although some char species may be more tolerant than other salmonids, studies indicate that rainbow trout, cutthroat trout and brown trout are much more sensitive with toxicity thresholds in the range of 17 µg/g egg Se (DeForest and Adams 2011). The EC10 for alevin mortality in brown trout was 17.7 µg/g egg Se (NewFields 2009), a value that was later revised to 20.8 µg/g egg Se (Formation Environmental 2011). The EC10 published by Elphick (2009) for larval survival in westslope cutthroat trout was 19 µg/g egg Se. This value was also later revised to 24.8 µg/g egg Se due to a discrepancy in laboratory analytical results (Nautilus Environmental and Interior Reforestation Co. Ltd. 2011). Some non-salmonid species also have lower thresholds than char. Northern pike were reported to have an EC10 for larval deformity of 20.4 µg/g, which is more similar to those reported for other salmonids (Muscatello *et al.* 2006). What is interesting about Figure 7.2 is that it demonstrates the narrow range of Se toxicity thresholds for fish, with some exceptions.

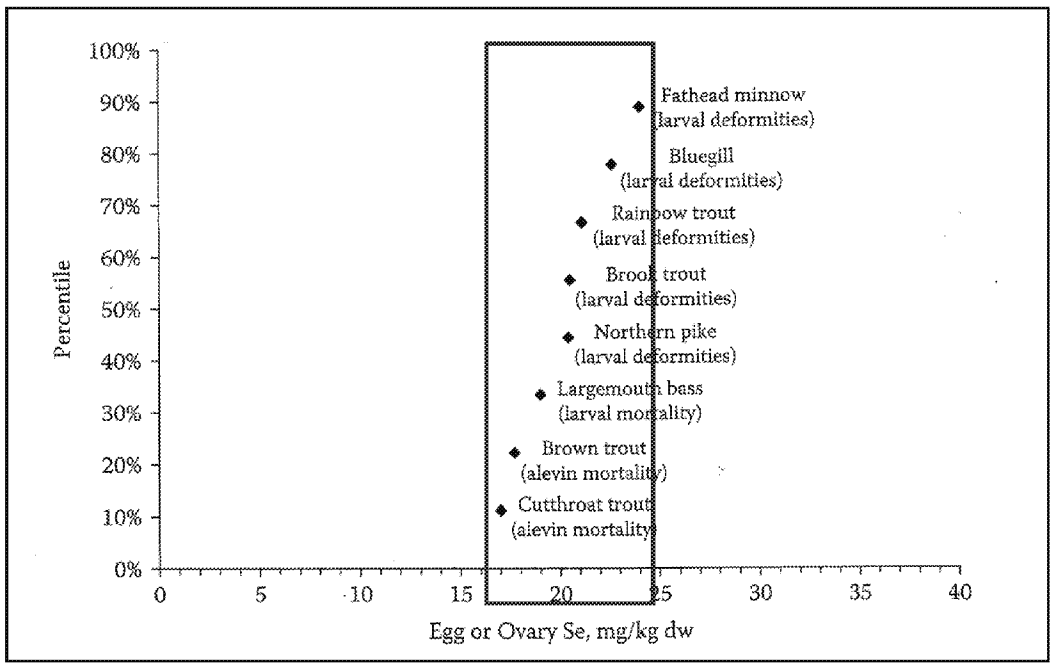

Figure 7.2 Species sensitivity distribution for egg or ovary selenium toxicity thresholds for larval deformity or alevin mortality (EC10 or equivalent) for several species of fish. Note the narrow range of toxicity thresholds in red (from Janz *et al.* 2010).[35]

[35] Reprinted with permission from *Ecological Assessment of Selenium in the Aquatic Environment*. Copyright 2010, Society of Environmental Toxicology and Chemistry (SETAC), Pensacola, FL, USA. ISBN 978-1-4398-2677-5

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Besser *et al.* (2012), recently published a full life-cycle study on desert pupfish (*Cyrpinodon macularius*), reporting that Se exposure had minimal results on survival and growth of juvenile and adult pupfish except in the highest Se dietary exposure (52 µg/g dw). They also reported that reduced egg production, although variable in treatment groups, was reduced in all Se exposure groups with egg Se of 4.4 µg/g or greater. This indicates that although pupfish may be among the least sensitive freshwater fish for survival and growth endpoints, based on egg production results, this species may be highly sensitive. It is thought that of the reproductive endpoints which are typically measured, larval survival and deformity are among the most sensitive (Janz *et al.* 2010). The results of Besser *et al.* (2012) indicate that more research is needed on a broader range of toxicological endpoints and species to better delineate the effects of Se.

Another feature of Se toxicity is the steepness of the concentration-response curve for fish and birds species. Figure 7.3 from Nautilus Environmental and Interior Reforestation Co. Ltd. (2011) shows Se concentration-response curve for westslope cutthroat trout for larval survival based on egg Se concentrations. The near vertical slope of the curve is a good demonstration that small increments above the toxicity threshold (EC10) result in very large increases in response. This narrow range between onset of effects and complete mortality, underscores the need for caution in establishing "safe limits" for Se in the environment.

Site-specific and species-specific relationships between whole-body, muscle and egg Se, make it possible to convert between tissue types (Casey and Siwik 2000; Casey 2005; Holm *et al.* 2005; Minnow *et al.* 2007). There are some techniques in use or under development, which allow non-destructive fish sampling to determine Se tissue residues. Non-destructive muscle plug sampling allows for greater flexibility in long-term monitoring of vulnerable or listed populations (Casey and Siwik 2000; Casey 2005; Minnow 2004; Minnow *et al.* 2007). There are also techniques being developed to better understand the temporal exposure to Se in highly mobile fish species using analysis of the annual otolith growth rings, although this technique requires the fish is sacrificed (Palace *et al.* 2007; Palace *et al.* 2011).There are additional recommendations for monitoring in Section 9.0.

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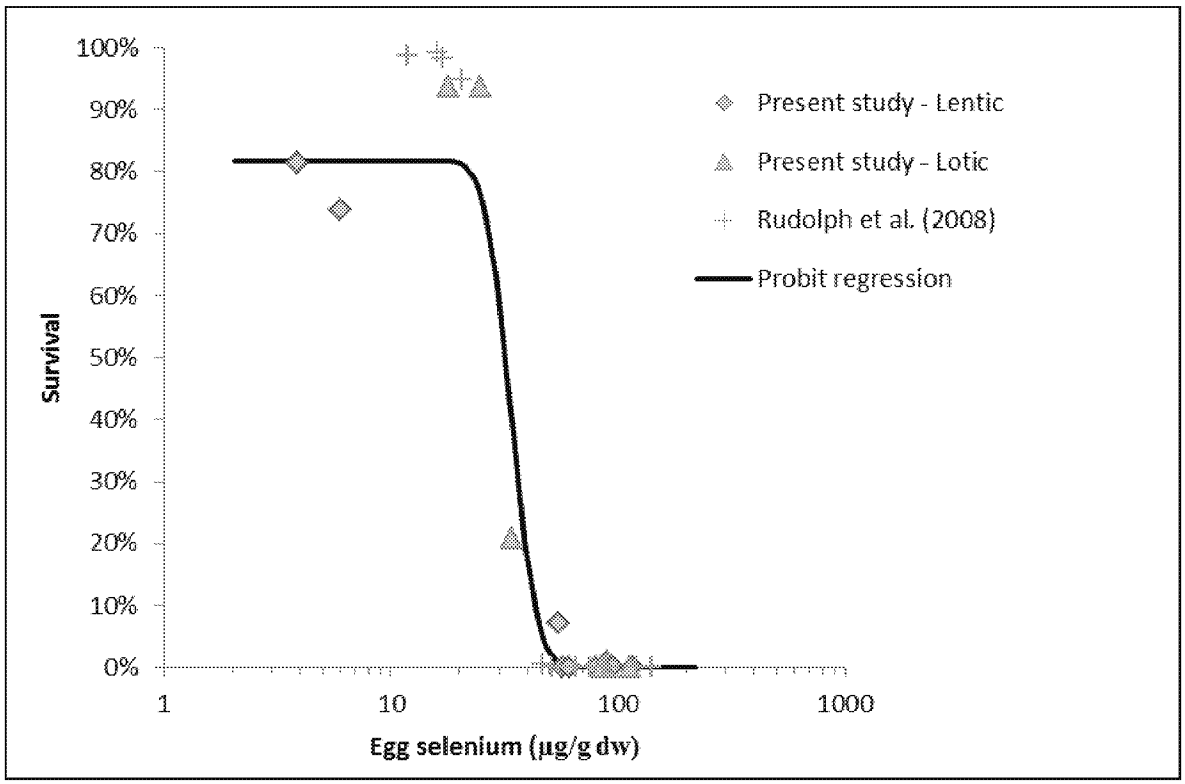

Figure 7.3 The dose-response curve for a study on westslope cutthroat trout field-collected, lab-fertilised gametes. Note the very steep slope of the curve, indicating a narrow range between the onset of effects and complete mortality (EC10 = 24.8 µg/g, 95% CI 12.0 – 30.5) (from Nautilus Environmental and Interior Reforestation Co. Ltd. 2011).

7.4.3.6 Bird Tissues

Selenium tissue residues for birds have been published for whole-body, diet, egg, liver, kidney, muscle, blood, and feathers. The most commonly measured tissue Se toxicity threshold concentrations are for diet and egg (Puls 1994; Skorupa *et al.* 1996; US DOI 1998; Ohlendorf and Heinz 2011). Published thresholds for birds are summarised in Table 7.11. The most relevant indicator of toxicity effects in birds is egg Se concentration (Skorupa 1998; Janz *et al.* 2010; Ohlendorf and Heinz 2011).

Dietary Se toxicity thresholds in birds are between 3 and 8 µg/g; some EC10 (95% CI) values reported for selected species were 4.0 µg/g (<0.5 - 7.3 µg/g), 4.4 µg/g (3.8 - 4.8 µg/g), and 4.9 µg/g (3.6 - 5.7 µg/g) (Ohlendorf and Heinz 2011). Puls (1994) reported that nutritionally adequate dietary Se for poultry and other birds (0.3 - 1.1µg/g Se) results in whole-egg concentrations between 0.67 and 5.0 µg/g Se. Egg Se greater than 5.0 µg/g Se is considered high,

and 8.3 to 50 µg/g can be toxic. The toxicity thresholds for egg Se concentrations related to reproductive impairment in wild birds are between 6 and 7 µg/g for sensitive black-necked stilts (Ohlendorf and Heinz 2011). Comparable toxicity thresholds for the American avocet, considered to be a more tolerant species, are in the order of 37 µg/g mean egg Se. Using various regression techniques, researchers analysed mallard egg viability and duckling mortality data generated from lab exposures and estimated EC10s between 12 and 15 µg/g for those endpoints (Adams *et al*. 2003).

Table 7.11 Examples of literature-based selenium toxicity thresholds for birds.

Bird thresholds	**Selenium concentration (µg/g dw)**	**Reference**
Dietary	0.3 – 1.1 (adequate) 3.0 – 5.0 (high) 5.0 – 80.(toxic)	Puls (1994)
Liver	10-20[1]	Ohlendorf and Heinz (2011)
	> 12[2]	Heinz (1996)
Eggs	6-7	Skorupa (1999)
	10	Heinz (1996)
	12-15	Adams *et al*. (2003)

[1]Considered suspicious of Se toxicosis when accompanied by symptoms listed for toxic effects.
[2]Possible reproductive impairment if egg-laying females have liver [Se] > 3 µg/g wet weight (assuming 75% moisture).

7.4.3.7 Amphibians and Reptiles

As mentioned previously, there is very little toxicity information on amphibians and reptiles and even fewer estimates of Se thresholds. Those data that exist have been summarised in Table 7.12. This lack of data for amphibians and reptiles is a critical gap in our understanding of Se effects leading several authors to suggest this is an area for future focus (Hopkins *et al*. 2006; Janz *et al*. 2010).

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Table 7.12 Some published selenium toxicity thresholds for amphibians and reptiles.

Amphibian and reptile thresholds	Selenium concentration (µg/g dw)	Reference
Whole-body	≥ 20[1]	USDOI (1998)
Eggs	≥ 10[1]	

[1] Presumptive thresholds for adverse effects based on whole-body concentrations (10x normal), and presumptive reproductive impairment threshold based on egg concentrations.

8.0 Guidelines for Selenium

8.1 Summary of Existing Environmental Quality Guidelines for Selenium

Existing water quality guidelines or criteria from North America, Australia, New Zealand, and Europe, are summarised in Tables 8.1 through 8.6, organized by water use. Guidelines for the protection of human health, presented in Table 8.1, are discussed in more detail in Section 8.3. It should be noted that each of the jurisdictions developing environmental quality guidelines or criteria use their own particular decision criteria and protocols, so the values may not be directly comparable or reflect the same level of protection.

Alberta, Saskatchewan, and Manitoba have published their own provincial aquatic guidelines which for the most part, have been adopted from the CCME (AENV 1999; Manitoba Conservation 2002; Saskatchewan Environment 2006). Prince Edward Island, Nova Scotia, New Brunswick, Newfoundland and Labrador, and the Yukon Territory use existing CCME water quality guidelines.

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Table 8.1 Summary of human health drinking water and tissue residue guidelines and criteria.

Water Use	Jurisdiction	Guideline/Criteria/Objective	Reference
Drinking water	Health Canada	10 µg/L	Health Canada (1992)
	World Health Organization	40 µg/L	WHO (2003)
	Japan Ministry of Environment	10 µg/L	Japan MoE (1993)
	US Environmental Protection Agency	50 µg/L	USEPA (2009)
	US Environmental Protection Agency, and Ministère du Développement durable, de l'Environnement et des Parcs du Québec	170 µg/L (water + organism)[1] 4200 µg/L (organisms only)[1]	USEPA (2009) MDDEP (2009)
Water-based human health consumption	US Environmental Protection Agency, Mid-Atlantic Region	6.9 µg/g wet weight (ww)	USEPA Mid-Atlantic Region (2010)
Tissue consumption of fish, birds, and mammals for human health	US Environmental Protection Agency	20 µg/g ww, recreational fishers 2.457 µg/g ww, subsistence fishers	USEPA (2000)
	US Department of Interior	2 µg/g ww, limited consumption for healthy populations and no consumption by children and pregnant women 5 µg/g ww, complete ban for all populations	USDOI (1998)
	Office of Environmental Health Hazard Assessment (California EPA)	Fish Contaminant Goal (FCG) – 7.4 µg/g ww (based on an individual consuming sport fish at a rate of 8 oz/week over a lifetime.)	OEHHA (2009)

[1] US EPA 2009 states that a more stringent maximum contaminant level has been issued by EPA. Refer to drinking water regulations (40 CFR 141).

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Table 8.2 Summary of existing selenium water quality guidelines, criteria or objectives for the protection of freshwater and marine aquatic life.

Jurisdiction	Guideline/Criteria/Objective	Reference
Aquatic life – freshwater		
International Joint Commission	≤ 1 µg/L	IJC (1981)
Canadian Council of Ministers of Environment	1 µg/L	CCREM (1987) CCME (2007a)
BC MoE	2 µg/L	Nagpal and Howell 2001
Ontario Environment and Energy	100 µg/L	MoEE (1987, 1994)
Ministère du Développement durable, de l'Environnement et des Parcs du Québec	Same as US EPA (see below)	MDDEP (2009)
US EPA	5 µg/L (chronic)[1]	US EPA (1987; 2004)
Australia/New Zealand	5 µg/L trigger value to protect 99% of species[2]	ANZECC (2000)
The Netherlands	0.09 µg/L, target value (long-term) 5.4 µg/L, max permissible conc. (short-term)	Warmer and van Dokkum (2002)
Aquatic life – marine water		
BC MoE	2 µg/L	Nagpal and Howell (2001)
US EPA	290 µg/L, saltwater acute[3,4] 71 µg/L, saltwater chronic[3,4]	USEPA (2004)

[1]US EPA acute Se criteria 258 µg/L for selenite, and a sulphate-corrected algorithm for selenate, exp(0.5812[ln(sulphate)]+3.357), e.g., at 100 mg/L sulphate, selenate should not exceed 417 µg/L Se.
[2]The ANZECC recommends for chemicals that bioaccumulate the low risk 99% trigger values be used.
[3] US EPA 2004 criteria document states that because Se may be as toxic to saltwater fishes as it is to freshwater fishes, the status of the fish community should be monitored, if Se exceeds 5.85 µg/g dw in summer or fall, or 7.91 µg/g dw during any season in the whole-body tissue of salt water fishes.
[4]US EPA 2009 states if Se is as toxic to saltwater fishes in the field as it is to freshwater fishes in the field, the status of the fish community should be monitored whenever the Se concentration exceeds 5.0 g/L in saltwater because the saltwater chronic criteria does not take into account uptake via the food chain.

The Province of Quebec has adopted US EPA water quality criteria for Se, including those for marine waters (MDDEP 2009). Ontario and BC have developed their own guidelines for Se (MoEE 1994; Nagpal and Howell 2001). Ontario's provincial water quality objective (PWQO) for Se (100 µg/L) stands out as much higher than others. This is because the objective was originally developed in 1979 (MoEE 1979) and was based only on acute toxicity data since chronic data was not available at that time. All provinces and territories in Canada participate in

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the development and technical review of CCME environmental quality guidelines as members of the inter-provincial Water Quality Task Group (CCME 2007b).

Selenium water quality guidelines or criteria for the protection of freshwater aquatic life have been developed by several other jurisdictions, including: the International Joint Commission (IJC 1981), Canada (CCME 2007a), the US EPA (USEPA 2004), Australia and New Zealand (ANZECC 2000), and the Netherlands (Warmer and van Dokkum 2002) (Table 8.2). Both BC MoE and the US EPA have also developed water column guidelines/criteria for the protection of marine aquatic life (Table 8.2).

Since Se is listed as a priority toxic pollutant under the US *Clean Water Act*, many state jurisdictions have either adopted the EPA aquatic life criteria (5 µg/L), or developed Se water quality standards (WQS) based on the EPA criteria and/or other scientific information (USEPA 1986). These are summarised in Table 8.3.

Table 8.3 Summary of US State water quality standards or site-specific standards developed for water.

Jurisdiction	Standard/Criteria	Reference
California EPA	0.1 – 0.8 µg/L[1]	Pease *et al.* (1992)
Colorado	4.6 µg/L	(Colorado DPHE 2007)
Indiana	35 µg/L	Indiana WPCB (2009)
Illinois	1,000 µg/L undesignated waters 5 µg/L Lake Michigan and tributaries	Illinois PCB (2009)
Iowa	10 – 70 µg/L	Iowa DNR (1992)
West Virginia	5 – 62 µg/L	WV DEP (2009)

[1]Site-specific water column quality criterion for San Francisco Bay.

State-derived standards for the protection of aquatic life are usually consistent with the US EPA Se criteria, but may deviate slightly. These exceptions include:

- California EPA and the California Water Quality Control Board – set a site-specific standard for Se in San Francisco Bay of 0.1 to 0.8 µg/L (Pease *et al.* 1992).
- Colorado – established a chronic Se WQS of 4.6 µg/L (Colorado DPHE 2007);

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- Indiana – a 4-day average chronic aquatic criteria (CAC) concentration of 35 µg/L was established (Indiana WPCB 2009);
- Illinois – 1,000 µg/L was established as a "not to be exceeded" concentration in undesignated waters except in mixing zones. A WQS of 5 µg/L was established for Lake Michigan or waters tributary to Lake Michigan (Illinois PCB 2009);
- Iowa – established chronic Se standards that range from 10 to 70 µg/L, depending on the classification of water (Iowa DNR 1992); and,
- West Virginia – adopted the US EPA Se chronic water quality criteria of 5 µg/L, but has also established site-specific numeric criterion of 62 µg/L for watersheds receiving coal mining drainage (WV DEP 2009).

The European Commission, (United Kingdom, Ireland, Portugal, Germany, Spain, France, Belgium and the Netherlands) has not listed Se as a priority substance and, therefore, has not developed environmental quality standards for Se (EC 2008). The Netherlands have published their own water column (dissolved Se) and sediment environmental quality objectives for Se (Warmer and van Dokkum 2002). The maximum permissible concentration (short-term) and target (long-term or chronic) values for total Se in surface waters are 5.4 and 0.09 µg/L, respectively (Table 8.4). Environmental quality objectives for Se in sediment are maximum permissible concentrations and target values of 2.9 and 0.7 µg/g (dw), respectively (Table 8.4; Warmer and van Dokkum 2002). Several other jurisdictions in North America have developed Se guidelines for sediment and soils which are summarised in Table 8.4.

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Table 8.4 Summary of selenium sediment and soil quality guidelines, criteria or objectives developed by other jurisdictions and previously by BC MoE for the protection of freshwater and marine aquatic life and soil organisms (respectively).

Jurisdiction	Guideline/Criteria/Objective	Reference
Sediment		
BC MoE	2 µg/g (dw)	Nagpal and Howell (2001)
International Joint Commission	≤ 5 µg/g (dw)	IJC (1981)
The Netherlands	0.7 µg/g (dw) target value (long-term) 2.9 µg/g (dw) max permissible conc. (short-term)	Warmer and van Dokkum (2002)
Soil		
Canadian Council of Ministers of Environment	1 µg/g and 2.9 µg/g[1]	CCME (2009)

[1]CCME soil quality Se guideline 1 µg/g for agricultural, residential or parklands and 2.9 µg/g for commercial and industrial lands.

In the early 1980s, the International Joint Commission published a guideline for Se in sediment (IJC 1981). The BC MoE also published a sediment quality guideline for sediments to protect aquatic life (Nagpal and Howell 2001). The CCME (2009) recently issued an environmental quality guideline for soil that protects receptors for agriculture, residential, recreational, commercial, and industrial land uses (Table 8.4). The soil guideline for sensitive agricultural, residential and parkland uses (1 µg/g) was derived using thresholds for soil contact and/or soil ingestion by wildlife or livestock, while for commercial and industrial land uses (2.9 µg/g) it was derived primarily on toxicity data for soil contact by plants, soil microorganisms, and invertebrates (CCME 2009).

Tissue based guidelines or site-specific criteria for protection of fish and wildlife are summarised in Table 8.5. The BC MoE, the San Francisco Regional Water Quality Control Board, and the US EPA have developed water column and/or tissue-based guidelines or site-specific criteria for the protection of sensitive fish species. Recently, DeForest *et al.* (2012) proposed a fish egg Se guideline of 20 µg/g Se, derived using the CCME (2007) species sensitivity distribution model. However, the CCME (2007) protocol was developed to deal with the concentration of substances in the water column and the toxic effects resulting from direct exposure, not bioaccumulation.

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BC MoE developed a guideline for wildlife in 2001 (Nagpal and Howell 2001) using birds as representative sensitive wildlife. The Utah Department of Environmental Quality (DEQ) recently developed a site-specific standard for the Great Salt Lake also using birds as surrogates (Table 8.5). The Se wildlife standard arrived at by the Utah DEQ (12.5 µg/g dw as egg Se) did not represent a consensus of all members of the Science Panel, a group of experts struck to review and recommend site-specific Se criteria. Some members of the Science Panel believed the standard was not sufficiently protective (CH2M Hill 2008). To address this, the Utah DEQ and the Utah Division of Water Quality developed assessment procedures as part of the standard, called Footnote (14) which were adopted by the Utah Water Quality Board and later submitted to the US EPA for approval (USEPA 2011a). The assessment procedures are a series of bird egg Se thresholds which, if exceeded, institute higher levels of response by the Utah Division of Water Quality if those thresholds are surpassed (USEPA 2011a). The trigger thresholds commence at the 5 µg/g mean egg Se, at which time monitoring is increased to assess Se loadings, address data gaps and areas of uncertainty identified during development of the standard (USEPA 2011a). If monitoring demonstrates that mean Se concentrations are ≥ 6.4 µg/g, a Level II Antidegradation review is launched by the State; at 9.8 µg/g Se studies are initiated to develop total maximum daily loads (TMDLs) for discharges; and at ≥ 12.5 µg/g mean egg Se, impairment is declared and TMDLs are formalized and implemented (USEPA 2011a; Table 8.5).

The Science Panel also recommended a bird dietary Se standard for the Great Salt Lake of between 3.6 and 5.7 µg/g, representing the 95% confidence interval of the dietary EC10 for bird egg hatchability estimated to be 4.9 µg/g Se (CH2M Hill 2008).

The San Francisco Bay Regional Water Quality Control Board derived a water column site-specific criterion for the protection of fish, birds, and humans (Pease *et al.* 1992). Based on modelling data from the San Francisco Bay estuary, the Board estimated that a water concentration of between 0.1 and 0.8 µg/L would limit Se uptake in primary producers and reduce the potential for bioaccumulation of Se in fish, birds, and humans consuming wildlife tissues (Pease *et al.* 1992).

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Table 8.5 Summary of water and tissue residue selenium guidelines and site-specific criteria developed for the protection of fish and wildlife.

Jurisdiction	Guideline/Criteria/Standard	Reference
Fish (whole-body)		
BC MoE	4.0 µg/g	Nagpal and Howell (2001)
US EPA	5.85 µg/g (summer or fall), 7.91 µg/g (any season)[1]	USEPA (draft 2004)
Wildlife (fish and/or birds)		
BC MoE guidelines for birds as wildlife surrogates	4 µg/L (water, maximum conc) 7µg/g (mean egg Se, alert level)	Nagpal and Howell (2001)
San Francisco Bay Regional Water Quality Control Board site-specific criteria for fish and birds	0.1 – 0.8 µg/L[2]	Pease *et al.* (1992)
Utah Department of Environmental Quality dietary Se standard for birds	3.6 – 5.7 µg/g (95% CI of EC10) 4.9 µg/g (dietary EC10 for hatchability)	CH2M Hill 2008
Utah Department of Environmental Quality bird egg site-specific criteria for Great Salt Lake	> 5.0 µg/g (increased monitoring)[3] 6.4 µg/g (Level II Antidegradation review) 9.8 µg/g (TMDL process initiated) ≥ 12.5 µg/g (waterbody declared impaired, TMDLs implemented)	USEPA (2011a)

[1] US EPA 2004 criteria document states that if whole-body fish tissue Se exceeds 5.85 µg/g during summer or fall, fish should be monitored to determine whether Se exceeds 7.91 µg/g whole-body (WB) during winter.
[2]This represents a modelled Se concentration in water that would limit uptake in primary producers and reduce bioaccumulation in fish, birds, and humans.
[3]The Utah Water Quality Board adopted Footnote (14); a series of triggers based on mean egg Se in birds.

Several jurisdictions have developed Se water quality guidelines for the protection of agricultural uses, summarised in Table 8.6. The Canadian federal guidelines for Se in irrigation water and livestock water, originally published in 1987, were established to protect crops and/or livestock foraging on crops and livestock watering (CCREM 1987; CCME 2005). Alberta (AENV 1999) and Manitoba (Manitoba Conservation 2002) adopted the CCREM (1987) agricultural guidelines, but BC derived slightly lower guideline values for both irrigation and livestock watering (Nagpal and Howell 2001). Agriculture and Agri-Foods Canada recently published a recommended maximum Se concentration for drinking water specific to horses, due to higher

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watering requirements for horses and their overall higher sensitivity to contaminants (Olkowski 2009).

Table 8.6 Summary of selenium guidelines and objectives developed by other jurisdictions and previously by BC MoE, for the agricultural water uses of crop irrigation and livestock watering.

Jurisdiction	Guideline/Criteria/Objective	Reference
Canadian Council of Ministers of Environment	20-50 µg/L, irrigation[1] 50 µg/L, livestock watering	CCME (2005)
BC MoE	10 µg/L , irrigation water 30 µg/L, livestock watering	Nagpal and Howell (2001)
Agriculture and Agri-Food Canada	10 µg/L, drinking water for horses[2] 50 µg/L, drinking water for all other livestock	Olkowski (2009)

[1]CCME Se guideline of 20 µg/L for continuous irrigation, 50 µg/L for intermittent irrigation.
[2]Ministry of Agriculture and Agri-Foods Canada recommended safe upper level of water contaminants for horses and other livestock.

8.2 Guideline Approach

The updated Se guidelines proposed in this section follow a similar approach as that used in 2001 by Nagpal and Howell (2001). Guidelines for various water uses and media are presented and accompanied by supporting rationale. For the human health (tissue residue), and aquatic life guidelines, screening values and alert concentrations (respectively) have been developed. These concentrations provide guidance to environmental managers using a tiered approach to Se management. Since Se bioaccumulation is complex and can be variable, it should be recognized that there is some degree of uncertainty in concentrations that may be considered safe for different media.

There has previously been no clear consensus on what measure of chronic Se toxicity is most relevant. Differences in interpretation of available toxicity data may have been responsible for the disparity in how Se has been managed (historically using water-based guidelines or criteria) in Canada and the US (Lemly 1996b; Adams *et al.*1998; Hamilton and Lemly 1999; Deforest *et al.* 1999; Fairbrother *et al.* 1999; Skorupa 1999; Hamilton and Palace 2001; McDonald and Kennedy 2002; Luoma and Rainbow 2008). Many of the authors cited above would agree

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however, that water column Se concentrations are not the most reliable measure for a complete understanding of Se bioaccumulation or its potential adverse effects. This is largely due to research showing that direct uptake of dissolved Se in higher animals, regardless of its form, is slow and often has no direct relationship to bioaccumulation in tissues or toxic responses in organisms (Coyle *et al.* 1993; Cleveland *et al.* 1993; Sappington 2002; Luoma and Presser 2009).

Despite water Se concentrations in general being a poor predictor of effects, some studies have defined strong relationships between water Se concentrations and tissue Se in fish (Skorupa and Ohlendorf 1991; Casey and Siwik 2000; Golder 2010b; deBruyn 2009). Hence, water guidelines can be very useful as alert concentrations or triggers for further action and management before Se increases to levels in the food chain which could lead to toxic responses in exposed organisms (Canton *et al.* 2008).

There is a consensus regarding the need to develop tissue-based Se guidelines and criteria (Lemly 1996; DeForest *et al.* 1999; Adams *et al.* 2003; Hamilton 2003; USEPA 2004; Janz *et al.* 2010). Tissue-based guidelines provide a more direct causal link between Se exposure and toxic effects, integrating the many factors affecting accumulation at any given site. Selenium concentrations in the prey of higher trophic level consumers, as well as tissues of the consumers themselves, are commonly recommended for monitoring (DeForest *et al.* 1999; Lemly 2002a; Ohlendorf and Heinz 2011). Many researchers advocate the use of eggs or ripe ovary tissue in fish and birds in Se risk assessment, but there can be limitations to relying on a specific tissue (Janz *et al.* 2010). The main disadvantage of using tissues for evaluating Se exposure is often the need for destructive sampling (sacrificing the organism to obtain a sample) which, in itself, can put a small population at risk. Sampling may be restricted or extremely difficult if the study includes rare or endangered species, if mature ovary tissues are difficult to obtain (e.g., small-bodied fish) or if early-life stages of fish (e.g., immature, non-breeding fish) are at risk (Hamilton 2002; USEPA 2004).

Guidelines should consider the possible logistical, regulatory or administrative limitations in monitoring and assessment programs, accommodating options for investigators to compare a

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wider array of indicator species, tissues, and media for a flexible approach (Coyle *et al.* 1993; Lemly 1996b). For example, the utility of guidelines based solely on dietary concentrations for fish and birds may be restricted since knowledge of the foraging habits, identifying actual diet composition, and hence getting accurate dietary Se estimates may be difficult (Fairbrother *et al.* 1999).

Since Se accumulation and toxicity varies widely between and within sites and species, site-specific assessments may be necessary (Lemly 1999; Adams *et al.* 1998; deBruyn *et al.* 2008). A generic water quality guideline may be appropriate for one aquatic system, or for one species, or one time of year, but may not be adequately protective of another adjacent system with different Se cycling dynamics (e.g., mainstem reaches versus side channels of the same river) (Lemly 1999). While development of site-specific assessment of Se is recommended, there is also a fundamental need for broadly applicable general guidelines to alert environmental managers to the potential threat Se may pose to aquatic resources and humans (Lemly 1999; Canton *et al.* 2008). Therefore a suite of Se guidelines for different media and tissues have been developed for use in BC to protect human health and aquatic life.

8.3 Guidelines for the Protection of Human Health

8.3.1 Drinking Water Guideline

In 1992, Health Canada established a drinking water quality guideline for Se; maximum acceptable concentration (MAC) of 10 µg/L (Health Canada 1992; Table 8.7). However, this guideline is currently being updated (P. Hamel, pers. comm., Health Canada, July 2012).

The rationale for the 1992 guideline was that drinking water containing a MAC of 10 µg/L would be the source of between 10 and 25% of total Se intake and thus providing a reasonable factor of safety from adverse health effects (Health Canada 1992). Health Canada based the total intake on the National Academy of Sciences recommended safe and adequate range of 50-200 µg Se per person per day for adults, and a minimum toxic intake level of 500 – 700 µg per person per day for adults (Health Canada 1992). In BC, Provincial Health Authorities have adopted the Health Canada Guidelines for Canadian Drinking Water Quality to assess the risk from chemical contaminants in drinking water (Drinking Water Leadership Council 2007).

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Table 8.7 Drinking water guidelines for selenium from various jurisdictions.

Jurisdiction	Regulation or Guideline	Value
Health Canada (1992)	Guideline -maximum acceptable concentration	10 µg/L
US EPA Office of Drinking Water	Standard – maximum contaminant level	50 µg/L
California Environmental Protection Agency (CEPA 2010)	Public Health Goal	30 µg/L
Alabama, Colorado, Delaware, Kentucky	Primary Drinking Water Standard	10 µg/L
Louisiana	Ground water protection- maximum contaminant level	10 µg/L
Massachusetts	Groundwater protection maximum contaminant level	10 µg/L
Minnesota	Drinking water guideline	30 µg/L
Mississippi	Water quality criterion	10 µg/L
Oklahoma	Public and private standard	10 µg/L
Virginia	Maximum contaminant level	10 µg/L
Wyoming	Water quality for human health standard	10 µg/L
World Health Organization	Health-based drinking water guideline	40 µg/L
Australia (National Health and Medical Research Council and Natural Resource Management Ministerial Council (2011)	Drinking water guideline	10 µg/L

The US EPA Office of Drinking Water recommends a maximum contaminant level (MCL) of 50 µg/L (US EPA 2009). This is a legally enforceable nation-wide standard under the *Safe Drinking Water Act*. Several US States have implemented more conservative standards (ATSDR 2003, Table 8.7). The World Health Organization established a provisional health-based drinking water guideline of 40 µg/L Se (WHO 2011). The WHO based this guideline on an allocation factor of 20% of the upper tolerable intake of 400 µg/day; this is a provisional guideline due to uncertainties inherent in the scientific database. The National Health and Medical Research Council and the Natural Resource Management Ministerial Council in Australia established a drinking water guideline of 10 µg Se/L (National Health and Medical Research and the Council and the Natural Resource Management Ministerial Council 2011).

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To protect drinking water sources and human health, Se concentrations should not exceed 10 µg /L at any time. This guideline is adopted from Health Canada's drinking water guideline and any future revisions to that number will be reviewed and may result in revisions to the BC WQG.

8.3.2 Health-based Selenium Screening Value in Fish Tissue

8.3.2.1 Exposure Assessments in Fish Tissue in Canada and the US

Jurisdictions in both Canada and the US have developed guidance protocols and procedures to address excessive Se accumulation in wild fish and waterfowl harvested for human consumption. The Government of Alberta assesses risks to human health from contaminants in fish tissue through a formal process lead by the Public Health Advisory Committee (PHAC). Alberta Health and Wellness, Regional Health Authorities, and Alberta Environment all have representation on the PHAC (Environment Canada 2010; W. Zhang, pers. comm., Alberta Health and Wellness, July 2011). Advisories are published in the Alberta Guide to Sportfishing Regulations (Government of Alberta 2011). For example, in February 2000, Alberta's Public Health Advisory Committee completed an initial human health risk assessment on Se in fish from four water courses near Hinton Alberta: Lac Des Roches Lake, West Jarvis Creek, Luscar Creek and Gregg Creek (Chen 2000). A consumption advisory from the Provincial Health Officer was not necessary because it was determined that people were not consuming fish from these watercourses (W. Zhang, pers. comm., Alberta Health and Wellness, July 2011).

The US EPA's Office of Water prepared four guidance documents for state, local, regional, and tribal environmental health officials responsible for issuing fish and shellfish consumption advisories in an effort to standardize the human health risk assessment approach amongst states (USEPA 2000a, 2000b, 2000c, 2000d). Volumes 1 and 2 provide information on monitoring and assessing. Volumes 3 and 4 address risk management and communication. The Volume 1 monitoring strategy recommends a two-tiered approach; screening studies (Tier 1) and intensive studies (Tier 2). The objectives of the Tier 1 are to identify areas where concentrations of contaminants in edible portions of consumed fish indicate the potential for health risks to humans. The guidance documents provide recommendations on the sampling program (size and classes of target species, sampling locations, sampling times, number of replicates, sample

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analysis, data analysis, and reporting). Observed concentrations of contaminants are compared to 'screening values' (USEPA 2000a; Table 8.8). According to the US EPA, exceeding a screening value is an indication that site-specific human health risk assessments may be warranted (Tier 2).

A screening value of 20 µg/g Se ww, has been established for recreational fishers and 2.457 µg/g, Se ww, for subsistence fishers (USEPA 2000b). Screening values are calculated using a risk-based procedure: a reference dose of 5 x 10^{-3} (mg/kg-day), an adult body weight of 70 kg, and consumption rates of 17.5 g/day for the general population and 142.4 g/day for subsistence fishers (USEPA 2000a). The following equation was used to calculate the EPA's screening values:

$$SV = [(\text{Reference Dose})(\text{Body Weight})]/\text{Consumption Rate}$$

Screening values are useful as they provide default values when site specific information such as fish consumption rates is not available. The EPA (USEPA 2000b) recommends that monitoring Se concentrations in fish and shellfish should be part of a monitoring program in areas where Se occurs in geological formations, or where oil and coal combustion occurs or has historically occurred.

Table 8.8 US EPA selenium screening values for sport and subsistence fishers.

Population	**Screening value[1] µg/g Se in fish tissue (wet weight)**
Adult, Sports Fisher	20.0
Adult, Subsistence Fisher	2.457

[1]Screening values are calculated using a risk-based procedure: a reference dose of 5 x 10^{-3} (mg/kg/-day), an adult body weight of 70 kg, and consumption rates of 17.5 g/day for the general population (i.e., sports fishers) and 142.4 g/day for subsistence fishers (USEPA 2000a).

More recently, the California Environmental Protection Agency's Office of Environmental Health Hazard (OEHHA) developed Fish Contaminant Goals (FCG) and Advisory Tissue Levels (ATL) for Se (OEHHA 2008). Analysis of Se concentrations in fish from several California waterbodies is part of the agency's surface water ambient monitoring program (OEHHA 2009). The key difference between FCGs and ATLs is that the latter are calculated with adjustments to

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incorporate the benefits of fish consumption (OEHHA 2008). FCGs are estimates of contaminant levels in fish that pose no significant risk to individuals consuming sport fish at a standard rate of 32 g/day, prior to cooking over a lifetime. The OEHHA (2008) uses FGC as a reference point to assist agencies with pollution mitigation and elimination through the development of fish tissue-based criteria. The OEHAA used the following equation to calculate the FCG, for Se (a nutrient with a non-carcinogenic effect) where the consumption exposure is equal to the reference dose:

$$\text{Tissue Concentration} = \frac{[(\text{Reference dose})(\text{Body Weight})]-(\text{Background Dietary Level})}{(\text{Standard Consumption Rate})}$$

The FCG for Se in fish tissue is 7.4 µg/g ww; this is based on an adult weighing 70 kg, a standard consumption rate of 32 g/day over a life-time, a reference dose of 5×10^{-3} mg/kg-day, and a background dietary consumption rate of 114 g/day (Table 8.9). According to OEHHA (2008), selenium concentrations are not reduced by cooking or cleaning techniques and therefore no reduction factors were applied in determining FCG and ATLs.

ATLs for Se were calculated using the same general equation as for FCGs, however to balance the risks and benefits of fish consumption the OEHHA determined that the average exposure should be equivalent to the reference dose. ATLs are recommended fish servings per week based on contaminant concentrations in fish tissue. They are designed to ensure that consumers are not exposed to a contaminant at levels that are beyond established safe intake levels (Table 8.10).

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Table 8.9 Fish contaminant goals for selenium in California sport fish are levels that pose no significant risk to individuals consuming sport fish at a standard rate (32 g/day).

Population	Fish Contaminant Goal[1] µg/g Se in fish tissue (wet weight)	Standard Consumption Rate
Adult, Sport Fisher	≤7.4	32 g/day, prior to cooking, over a life-time

[1]Fish contaminant goals are based on an adult weighing 70 kg, and a reference dose of 5 x 10^{-3}(mg/kg/day). They are designed to prevent consumers from being exposed to more than the average daily reference dose (OEHHA 2008).

Table 8.10 Advisory tissue levels for a range of selenium concentrations in California sport fish and corresponding consumption recommendations.

Population	Advisory Tissue Level[1] µg/g Se in fish tissue (wet weight)	Recommended Consumption Rate
Adult, Sport Fisher	>15	No consumption
	> 4.9-15	32.4 g/day
	>2.5-4.9	64.9 g/day
	<2.5	97.2 g/day

[1]Advisory tissue levels are calculated with adjustments to incorporate the benefits of fish consumption (OEHHA 2008).

Public health consumption advisories due to elevated Se in fish tissue or wildlife have been issued in several US states including Idaho, California, Nebraska, Colorado (S. Fontenelle, pers. comm., US Public Health Service Commissioned Corps US EPA, Office of Water Standards and Health Protection), Utah, West Virginia, and Michigan (Appendix B).

8.3.2.2 BC Health-based Screening Value for Selenium in Fish Tissue

In cases where aquatic contaminants have the potential to accumulate in wild fish, game, or other exposed foods, the Ministry of Environment with the Ministry of Health may determine that development of health-based guidelines is prudent for use in environmental monitoring programs. Health-based screening values (SV) for Se in fish tissue using a risk-based approach are provided below. These were developed collaboratively by the Ministry of Environment and the Ministry of Health. Screening values are defined in this document as threshold values against which levels of selenium in the ambient environment can be compared and assessed for potential risks to human health.

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The SVs were calculated based on conservative estimates of the receptor population's fish consumption rates, days of exposure, and Se bioavailability. Screening values have been calculated based on three fish consumption scenarios: high, moderate, and low. Determining which screening value to use in a regional monitoring program will depend on what is known about local consumption habits. For example, if only seasonal, sports fishing occurs, the 'low' consumption pattern SV may be adequate. However, if subsistence fishing is occurring, then a high consumption SV would be the appropriate threshold. Monitoring programs must be undertaken with consideration of several factors to ensure that food samples are representative of those consumed by local populations. Sampling and monitoring considerations and protocols have been outlined by the BC MOE (BC MOE 2012b) and Health Canada (2004a).

Exceedances of a screening value may indicate that detailed monitoring and evaluation of risks to human health are appropriate; this would be determined by the Ministry of Health or local Health Authorities. To assess potential exposure to local populations all exposure pathways including food, water, and air must be evaluated (M. Zemanek, pers. comm., BC Ministry of Health, June 2011).

It is recognized that consumption of fish has benefits for health outcomes; this would also be considered by public health professionals in any decisions regarding consumption advice (M. Zemanek, pers. comm., BC Ministry of Health, June 2011; McAuley and Knopper 2011). Fish are excellent sources of polyunsaturated fatty acids, vitamins and minerals; there is evidence that a diet containing fish has numerous health benefits including a reduction in the risk of coronary heart disease and stroke (FAO/WHO 2011).

Screening values were calculated using Health Canada's (2004b) recommend general equation for predicting ingestion of a contaminant via ingestion of contaminated food, as follows:

$$\text{Dose (mg/kg bw/day)} = [\Sigma\ [C_{Foodi} \times IR_{Foodi} \times RAF_{Orali} \times D_i\ ]]/[BW \times 365]$$

Where:

- Dose=predicted intake of contaminant

- C_{Foodi} = concentration of contaminant in food *i* (mg/kg)
- IR_{Foodi} = receptor ingestion rate for food *i* (kg/day)
- RAF_{Orali} = relative absorption factor from the gastrointestinal tract for contaminant *i* (unitless)
- D_i = days per year during which consumption of food *i* will occur
- *BW* = body weight (kg)
- 365 = total days per year (constant)

The above equation is adjusted to:

$$C\ (mg/kg) = [Dose \times BW] / [IR_{Foodi} \times RAF_{Orali}]$$

Where:

- *C* = highest concentration of Se in fish tissue (µg/g) that will not allow *[Dose x BW] / [IR_{Food} x RAF_{Orali}] to exceed 1*,
- Dose = tolerable daily intake: Health Canada's recommends using the upper tolerable intake level of dietary reference intakes as the tolerable daily intake for selenium (see Table 7.3). Adults are used as the default receptors; IOM (2000) did not identify one particular age group as being more susceptible to the toxic effects of Se.
- BW = body weight: 70.7 kg standard weight for an adult,
- IR_{Food} i – ingestion rate: calculated based on the following fish consumption rates:
    - 220 g/day Canadian First Nations (Health Canada 2004b),
    - 111 g/day Canadian general population (Health Canada 2004b),
    - 21.5 g/day based on 2 recommended servings/week (Health Canada 2011).
- $RAF_{Orali=}$ relative absorption factor ; 100% used as a cautious upper bound, adsorption of all forms of Se is considered relatively high in humans.

Note: For the purpose of setting screening values, it is assumed that fish are consumed on a daily basis throughout the year (365 days).

Note: mg/kg = µg/g (these are equal; no conversion necessary).

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Fish Consumption (IR_{Food} i)

Extensive fish consumption surveys for BC coastal and inland communities are not available to provide BC specific rates. The Columbia River Treaty Inter-Tribal Commission (1994) fish consumption survey of four communities of the Columbia River Basin found that the average rate of consumption for all species of fish was 58.7 g/day. The 90th percentile was between 97.2 and 130 g/d, the 95th percentile was 170 g/d, and the 99th percentile was 389 g/d. Chen (2000) conducted a survey in the Swan Hills area and determined that 2% of the population consumed >100 g/d, 13% consumed 30-99 g/d, 28% consumed 3-29 g/d, and 57% consumed < 4g/d. Richardson (1997) provided receptor characteristics for fish consumption rates for the Canadian general and native populations. While dated, this information continues to be recommended for use by Health Canada (2004b) for preliminary quantitative risk assessments. Although these rates may be considered conservative, they will be used in the calculation of BC's screening values. Site-specific assessments would likely need to refine these rates.

Screening values are provided for three populations: high fish consumption patterns (greater than 220 g/day), moderate consumption (111 g/day) and low consumption (21 g/day) (Table 8.11). The high and moderate consumption rates correspond to Health Canada's rates for Canadian First Nation populations and the general population (Health Canada 2004b).

Table 8.11 Screening values for rates of average, moderate and high fish consumption.

Consumption Pattern	Ingestion Rate (g/day)	Reference Dose (µg/ kg bw/day)	Standard Body Weight (kg)	Relative Absorption Factor (%)	Screening Value (µg/g, wet weight)
High	220	5.7	70.7	100	1.83
Moderate	111	5.7	70.7	100	3.63
Low	21.5	5.7	70.7	100	18.74

A screening value for populations consuming the recommended two servings per week or 21.5 g/day (Health Canada 2011) is also provided. These default consumption rates provide guidance when food use by local community populations is not available.

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If screening values are exceeded, it is protocol that the Ministry of Environment reports the information to the Ministry of Health and local Health Authorities. The latter agencies determine what action and public communication may be necessary.

To protect human health, the BC Ministry of Environment and the Ministry of Health recommend the use of the following screening values in environmental monitoring programs where elevated Se concentrations due to natural or anthropogenic activities in aquatic environments is a concern:

- ***1.8 µg/g (wet weight) or 7.3 µg/g (dry weight) for high fish intake (0.22 kg/day)***
- ***3.6 µg/g (wet weight) or 14.5 µg/g (dry weight) for moderate fish intake (0.11 kg/day)***
- ***18.7 µg/g (wet weight) or 75.0 µg/g (dry weight) for low fish intake (0.03 kg/day).***

Sampling and monitoring considerations and protocols have been outlined by the BC MoE (BC MoE 2012b) and Health Canada (2004). Exceeding screening values may lead to site-specific investigations to assess possible health risks.

8.4 Aquatic Life

Selenium presents a challenge in application of BC's typical protocol for deriving guidelines, since Se is a required nutrient but becomes toxic at concentrations not much higher than background. Short-term guidelines were not developed for Se since acute toxicity occurs at relatively high concentrations associated with industrial activities and effluent discharges. Rather, long-term guidelines were derived to protect aquatic life from the more typical and potentially hazardous effects resulting from the bioaccumulation of Se at much lower concentrations in water. Published chronic Se toxicological endpoints were reviewed with a focus on the harmful effects of Se based on water quality, sediment, dietary tissue, and receptor tissues for a range of aquatic organisms and their consumers. The use of standard BCFs or BAFs in deriving a guideline for Se was not possible since Se behaves differently in different environments. While Se relationships between water and other environmental compartments, including tissue, are site-specific, generic relationships appear to be relevant when compared with site-specific data reported at sites in BC (see the following sections on guideline derivation for details).

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The guidelines include long-term values for several environmental compartments: water column, sediment, and tissues (dietary tissues for fish and birds, whole-body, muscle and egg tissues of fish, and bird egg tissue). Multi-media guidelines provide more information and greater flexibility in a monitoring and management framework aimed at protecting aquatic ecosystems. This approach was based on several factors:

- transformation of aqueous Se and subsequent bioaccumulation of dietary Se is the primary route of exposure in aquatic ecosystems;
- chronic exposure of waterborne Se also contributes to overall exposure and may result in negative effects on aquatic biota, ranging from increased mortality and deformity, to subtle changes in behaviour and physiology (Hodson *et al.* 1980; Hilton *et al.* 1982; Hamilton and Wiedmeyer 1990; Cleveland *et al.* 1993; Miller *et al.* 2007; Palace *et al.* 2004);
- sediment is an important repository for Se and may contribute to the long-term cycling of Se and adverse impacts in an ecosystem long after the Se source is removed (Lemly 2002a), and;
- a full characterisation of Se in all critical environmental compartments (water, sediment, and biota) should be conducted to assist in the evaluation, interpretation, and management of Se in aquatic ecosystems (Lemly 1996a; Sappington 2002; Presser and Luoma 2006; Ohlendorf *et al.* 2008).

The guidelines were developed based on taxa shown to be most sensitive to Se exposure, namely fish and birds. Differences in the propensity of Se to accumulate exist for different natural aquatic settings (i.e., lentic versus lotic). The guidelines adopt the principle of protecting the most sensitive hydrologic units within an exposed watershed, since fast moving (lotic) streams are connected with and have within them, slower moving, depositional (lentic) areas such as pools, back-eddies, back-channels, lakes, and wetlands (Lemly 1999).

In deriving the guidelines, BC MoE protocols (2012a) were followed, along with guidance provided in MacDonald (1993). The guidelines are based primarily on laboratory studies where test organisms have been exposed to a single contaminant to reduce potential confounding effects of co-occurring contaminants. Since Se is a complex element with many factors

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influencing its toxicity, many published field studies were also considered in the derivation of the guidelines.

Published Se concentrations were converted to common units for water (µg/L), and sediment and tissues (µg/g dry weight) where possible. Tissue concentrations reported in wet weight were converted to dry weight using the reported % moisture content, or if no conversion factor was reported an assumed % moisture (usually 75% for muscle, whole-body, and organs) was used.

Selenium toxicity thresholds for fish species represented in BC and/or Canada were considered in the derivation of the guideline. Studies used to derive guideline values were evaluated and classified as primary, secondary, or unacceptable for guideline derivation as per the BC protocol (BC MoE 2012a). Classification of toxicological studies on the effects of Se presented some challenges. Selenium is not a typical element with a typical mode of action. Guidelines for bioaccumulative substances typically take into consideration a bioaccumulation factor (BAF), but for Se the reported BAFs vary widely making it difficult to establish a "general" BAF (Peterson and Nebeker 1992). Since Se bioaccumulates through the food web, the exposure mechanisms and toxicity can be complex and require novel study designs that accommodate this unique element. Although controlled feeding studies (e.g., mesocosm studies) would not be classified as acceptable since there are no standard federal or provincial protocols, these studies were considered primary if they met all other classification criteria. This is consistent with the approach taken by other jurisdictions such as the US EPA (C. Delos, pers. comm., US EPA, May 2012)

Minimum data requirements for setting long-term (chronic) guidelines were met (BC MoE 2012a). However, the existing literature supports the theory that invertebrate, plant, and algal species are not as sensitive to Se toxicity as are fish and birds. Therefore, greater emphasis was placed on fish and bird studies, or amphibian studies if available. Published studies were classified according to BC MoE (2012a).

In deriving aquatic life and wildlife guidelines, a minimum of three acceptable controlled laboratory feeding studies on fish or bird species present in BC or Canada were required. Since

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Se bioaccumulation occurs primarily through dietary exposure, long-term laboratory feeding studies (or preferably combined dietary and water Se) were necessary to reliably establish toxicity thresholds. The American Society for Testing and Materials (ASTM 2005) have published protocols for controlled feeding studies. Environment Canada and the US EPA have not yet developed protocols for fish and wildlife feeding studies (C. Buday, pers. comm., Pacific Environmental Science Centre April 2012; C. Delos, pers. comm., US EPA, May 2012). Therefore, the evaluation of many of these studies relied on professional judgement based on the study design details provided in the publications.

Controlled laboratory or mesocosm feeding experiments that generate toxicological endpoints for reproductive effects are inherently more difficult and much more expensive to conduct (e.g., Hamilton *et al.* 1990; Hermanutz *et al.* 1992; Doroshov *et al.* 1992; Coyle *et al.* 1993; Hardy *et al.* 2010). However, if control group mortality remains low and/or, there is a clear concentration response in the larval test endpoints such as edema or deformity, they provide a greater level of control than field-generated data.

Many of the fish egg/ovary toxicity threshold estimates reported in the literature are based on field-collected gametes which have an unknown history of Se exposure (e.g., Kennedy *et al.* 2000; deRosemond *et al.* 2005; Holm *et al.* 2005; Muscatello *et al.* 2006; Rudolph *et al.* 2008, Elphick *et al.* 2009; NewFields 2009). Field-collected gametes may have also been exposed to co-contaminants which may have altered a toxic response. These studies do not meet the BC or CCME criteria for classification as primary or secondary literature. Regardless of their limitations, some field studies demonstrate a clear dose-response for endpoints which are symptomatic of Se toxicity. Since these studies provide a large body of evidence, they were considered in setting tissue-based guidelines. The US EPA has also made judicious use of field-generated data in the derivation of their Se criteria (C. Delos, pers. comm., US EPA, May 2012).

Consistent with BC MoE (2012a) derivation protocols, care was exercised in selecting the appropriate species information to make guidelines applicable in BC and Canada. The available Se toxicity data spans several different species, some resident in BC or Canada and others not. In BC, *Oncorhynchus*, represented by Chinook salmon, rainbow trout and cutthroat trout, is the

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most common genus found to be very sensitive to Se toxicity (DeForest and Adams 2011). They are fairly abundant, distributed in many medium and large coastal and inland river systems in BC (Scott and Crossman 1973; McPhail 2007). Data was available for Chinook salmon, rainbow trout (Goettl and Davies 1978; Hilton *et al.* 1980; Hilton and Hodson 1983; Hunn *et al.* 1987; Hamilton *et al.* 1990; Holm *et al.* 2005; Vidal *et al.* 2005), and westslope and Yellowstone cutthroat trout (Kennedy *et al.* 2000; Rudolph *et al.* 2008; Elphick *et al.* 2009; Nautilus Environmental and Interior Reforestation Co. Ltd. 2011; Hardy *et al.* 2010). Other fairly common species in BC for which there is Se toxicity data are brook trout (Holm *et al.* 2005), white sucker (deRosemond *et al.* 2005), and Dolly Varden char (McDonald *et al.* 2010).

Toxicity data for other fish species with either limited or very limited presence in BC and/or Canada (McPhail 2007) were also reviewed: brown trout (*Salmo trutta*) (NewFields 2009), northern pike (Muscatello *et al.* 2006); white sturgeon (*Acipenser transmontanus*) (Tashjian *et al.* 2006), bluegill sunfish (Finley *et al.* 1985; Doroshov *et al.* 1992; Hermanutz *et al.* 1992; Cleveland *et al.* 1993; Coyle *et al.* 1993; Lemly 1993b; Hermanutz *et al.* 1996; McIntyre *et al.* 2008), fathead minnow (Schulz and Hermanutz 1990), largemouth bass (*Micopterus salmoides*) (Carolina Power and Light 1997, as cited in USEPA 2011b), and striped bass (*Morone saxitilis*) (Coughlan and Velte 1989). There are also Se toxicity data that exist for species not found in Canada: Sacramento splittail (*Pognichthys macrolepidotus*) (Teh *et al.* 2004). Data for these species was reviewed but more emphasis has been placed on data for cold-water salmonid taxa common to BC and/or Canada.

8.4.1 Water Column Guideline

Since bioaccumulation and toxicity of Se to organisms cannot always be predicted consistently from the concentration of Se in water, many scientists advise against the sole use of a water column guideline (Luoma and Presser 2009; Stewart *et al.* 2010). However, higher Se in water increases the risk of higher Se levels in biota, even if the absolute relationship is weak. Some researchers have shown a strong relationship exists between water Se concentrations and fish tissue Se, making water a reasonably good predictor of Se accumulation in fish and an important assessment tool (Skorupa and Ohlendorf 1991; Casey and Siwik 2000; deBruyn 2009; Golder 2010b). Moreover, water is probably the most sampled media in environmental monitoring

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programs since it is easy, relatively inexpensive, and does not require sacrificing organisms which may be rare or endangered. Therefore, a BC water column guideline was retained in the suite of Se guidelines and where water:tissue relationships are established can be used as a surrogate for biological sampling, and a trigger for further assessment and action.

The current CCME Se water quality guideline for protection of aquatic life (1 µg/L) was published in 1987 and reflected the available science at the time. Comparison of the current CCME guideline with many of the published toxicity thresholds (Table 7.6) and water quality guidelines from other jurisdictions (Table 8.2) indicates this guideline is likely protective for the most sensitive environments. The previous BC MoE water column guideline of 2 µg/L was applicable to both fresh and marine water. This guideline was based on several assumptions including the chronic effects of Se (accumulation) as well as studies classified as primary. Studies used in the derivation included several reported lowest observed effect concentrations (LOECs) for a number of fish species (Cumbie and Van Horn 1978; Schultz and Hermanutz 1990; Hermanutz *et al.* 1992; Hermanutz 1992; Gillespie and Baumann 1996), and an EC50 for *Daphnia magna* (Bringmann and Kuhn 1977, as cited in Nagpal and Howell 2001), all of which converge around 10 µg/L Se. This value was then divided by an uncertainty factor of 5 to derive the 2 µg/L BC water column guideline to protect aquatic life (Nagpal and Howell 2001).

Many jurisdictions recommend a concentration of approximately 2 µg/L as the threshold beyond which there is risk of adverse effects to aquatic life (DuBowy 1989; Maier and Knight 1994; Lemly 1996; Skorupa *et al.* 1996; Hermanutz *et al.* 1996; USDOI 1998; Hamilton and Lemly 1999; Swift 2002; Hamilton 2003; Paveglio and Kilbride 2007). Some studies however, demonstrate that water concentrations over 1 µg/L may be cause for concern to sensitive species in environments and/or food webs due to enhanced Se bioaccumulation (Crane *et al.* 1992; Peterson and Nebeker 1992; Skorupa *et al.* 1996; Swift 2002; Stewart *et al.* 2004). Based on a generalized dietary intake model, Peterson and Nebeker (1992) recommended a dissolved Se threshold of no greater than 1 µg/L to protect sensitive piscivorous birds and mammals. Data reviewed by deBruyn and Chapman (2007) showed that sublethal effects to aquatic insects (as much as 50% declines in some taxa) have been observed in field studies where Se concentrations were between 5 and 10 µg/L. In studies using experimental streams, Swift (2002) found

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population reductions in stream isopods exposed to water concentrations of 10 µg/L or greater, and in tubificid worms at the 30 µg/L exposure concentration. Hermanutz *et al.* (1996) found that the progeny of adult bluegill sunfish exposed to 2.5 and 10 µg/L waterborne Se in experimental streams were adversely affected. Skorupa and Ohlendorf (1991) reviewed and analysed field and laboratory data relating embryo toxicity in birds to water concentrations of Se and recommended 2.3 µg/L as a reasonable provisional goal for waterborne Se to protect sensitive aquatic birds. Collectively, these results provide support of the BC water quality guideline of 2 µg/L or lower, for very sensitive environments and/or species.

The San Francisco Bay Regional Water Quality Control Board developed site-specific Se criteria for San Francisco Bay estuary to protect fish, birds, and humans (Pease *et al.* 1992). Their rationale was the recognition that the earliest US EPA water quality criterion for Se (USEPA 1987) did not consider the persistence and bioaccumulation of Se through the food chain. The criterion developed for the Bay was 0.1 to 0.8 µg/L selenite to limit the uptake of Se at the base of the food chain and reduce Se bioaccumulation in consumers. The California State Water Resources Control Board (SWRCB) developed a water column criterion to protect fish from adverse effects using relationships between fish tissue and water Se concentrations. Based on a mean low-effect concentration adjusted for background in fish tissue of 1.1 µg/g wet weight, the estimated water column criteria for the protection of fish was 0.9 µg/L. In addition, the SWRCB recommended a water criterion of 0.8 µg/L to protect humans consuming fish tissue (Pease *et al.* 1992). The recommended site-specific criterion is currently under review as part of the process for developing a total maximum daily loading (TMDL) for the Bay (B. Baginska, pers. comm., San Francisco Bay Regional Water Quality Control Board, April 2012). The San Francisco Bay estuary is an example of a particularly sensitive environment and food web to bioaccumulation of Se.

A study on northern pike conducted by Muscatello *et al.* (2006) in northern Saskatchewan lakes receiving uranium mining/milling effluent provides compelling evidence for a low water Se guideline. These researchers found significant increased deformity in fry from the medium and high Se exposure sites compared with reference. The mean Se concentrations at the medium and high exposure sites measured at the end of the study were approximately 0.5 and 1.0 µg/L

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(Muscatello *et al.* 2006). In a similar field study on westslope cutthroat trout, Rudolph *et al.* (2008) selected O'Rourke Lake, a small reference lake with Se concentrations <1.0 µg/L, where mean (SD) hatchability in fish eggs was 72.6 % (± 27.2%). This was lower (but not significantly different) than mean (SD) hatchability in eggs collected at the exposed lake 83.3 % (± 13.9%). This data may suggest that very low waterborne Se (<1 µg/L) can lead to reproductive effects, however, the interpretation of this study may be compromised since no fish resided in O'Rourke Lake prior to fish stocking programs.

A bioaccumulation model for westslope cutthroat trout in the Elk Valley, BC (Golder 2010b) was developed between water and egg Se concentrations. Golder's water-to-egg general linear model for lentic environments, which represent the most sensitive to Se accumulation, had a reasonably good fit (r^2= 0.75). This equation[36] was used, along with the average EC10 egg Se toxicity threshold for larval survival and deformity in cutthroat trout (21 µg/g egg Se), to calculate a corresponding water concentration of 2.2 µg/L. Using the same equation with the average rainbow trout egg Se EC10 estimate of 22 µg/g for larval deformity, yielded a corresponding water concentration of 2.5 µg/L. Although water column concentrations of 2 µg/L Se may be broadly applicable as a guideline, the results of Muscatello *et al.* (2006), Rudolph *et al.* (2008), and Golder's (2010b) BAF model, suggest that in sensitive lentic areas water Se concentrations over 1 µg/L may represent a risk to aquatic life.

An alert concentration of 1 µg/L is recommended to identify and evaluate environments where enhanced Se bioaccumulation may be occurring. The extremely low site-specific Se water criterion developed by the San Francisco Bay Water Quality Control Board (0.1 – 0.8 µg/L) is one of several examples illustrating environments where Se uptake is particularly efficient resulting in greater potential risk to organisms higher up in the food web (Pease *et al.* 1992). An alert concentration of 1 µg/L Se can be used as part of a tiered, adaptive management approach. Exceedance of the alert concentration in sensitive environments indicates the need for increased monitoring of water and other ecosystem compartments (i.e., sediment, periphyton or biofilm, and dietary items). This tool will support early detection of potential Se bioaccumulation

[36] The equation derived for the relationship between water Se and westslope cutthroat trout egg Se in lentic waters was $\log(Se_{egg}) = 0.36 \times \log(Se_{water}) + 1.2$ from Golder (2010b).

problems and provide earlier opportunities to commence proactive management actions. The alert concentration is consistent with the current CCME environmental quality guideline of 1 µg/L Se in water.

The 30-day average alert concentration for the protection of aquatic life in sensitive ecosystems is 1 µg/L determined as the mean concentration of 5 evenly spaced samples collected over 30 days, and measured as total Se.

The proposed guideline of 2 µg/L was compared to typical background Se water concentrations to assess its applicability. Water Se concentrations at reference sites in BC and Alberta are less than 2 µg/L and typically less than 1 µg/L (Minnow *et al.* 2011; Casey and Siwik 2000). The routine monitoring data from the Elk River upstream of mining activity showed that background concentrations of Se in water were at or below 1 µg/L (Minnow *et al.* 2011). A review of water quality data collected near coal mines in Alberta, representing a larger data set over a longer time frame, showed that at 11 reference sites, the range of mean water Se was between ≤ 0.25 and 2.2 µg/L with a median water Se concentration of 0.7 µg/L, well below the 2 µg/L guideline value (Casey 2005). Background water quality at sites across Canada (Table 4.5) is below the Se WQG of 2 µg/L. The water column guideline for aquatic life is applicable to both fresh and marine waters, since marine water typically has lower Se concentrations than freshwater and Se behaves similarly in both environments (Sui and Berman 1989).

The 30-day average water quality guideline for protection of aquatic life is 2 µg/L determined as the mean concentration of 5 evenly spaced samples collected over 30 days, and measured as total Se.

8.4.2 Sediment Guideline

Selenium in suspended and bed sediments is an important exposure route for organisms at the base of the food web (Lemly and Smith 1987; Fan *et al.* 2002). Nagpal and Howell (2001) developed an *interim* sediment Se quality guideline (2 µg/g) due to limited available data at that time. This guideline was evaluated based on current information.

Thompson *et al.* (2005) reported very low sediment effect concentrations of between 0.9 and 1.9 µg/g, based on two different derivation methods. The Netherlands Institute for Inland Water

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Management and Waste Water Treatment (known as RIZA in the Netherlands) published an environmental quality standard for sediment Se of 0.7 µg/g as a target value (long-term chronic objective), and 2.9 µg/g as a maximum permissible concentration (short-term objective) (Warmer and van Dokkum 2002).

Effect levels assessed by Thompson *et al.* (2005) were based on field data related to sediment quality and co-occurring benthic invertebrate communities near uranium mines in Saskatchewan. The response of the benthic community could therefore be related to other contaminants present or the mixture of contaminants. In addition, the effect concentrations do not consider indirect toxic effects of Se bioaccumulation on higher trophic level organisms. So, while informative, Thompson *et al.* (2005) does not qualify as primary literature, but does provide a possible range of effect levels in sediment.

The Se sediment environmental quality objectives developed by RIZA (Warmer and van Dokkum 2002) for freshwater and marine environments were originally derived using a statistical extrapolation (a species sensitivity distribution) of global toxicological data (van de Plassche and de Bruijn 1992). However, since no soil or sediment toxicological data could be found, sediment objectives for Se were derived by estimating a partition co-efficient (K_p) from measurements of river water and suspended particulate matter. The authors stated that many of the assumptions, including whether the K_p's truly represented an equilibrium, were questionable, making the sediment objectives highly uncertain (van de Plassche and de Bruijn 1992).

Other published benchmarks or concentrations of concern are not appreciably higher than 2 µg/g. For example, the US DOI (1998) summarized a number of field-collected Se sediment concentrations associated with adverse effects to fish or other wildlife populations ranging from 0.9 to 5 µg/g. The US DOI (1998) adopted a value of 2.5 µg/g as their EC10 for sediment Se effects on fish and wildlife. Based on an analysis of data collected over more than a decade at Belews Lake, NC, Lemly (2003) stated that sediments above 2 µg/g Se pose a high risk for accumulation of Se in benthos to levels that may be toxic to fish and birds.

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Although these published sediment thresholds and the original publication used to establish the BC interim sediment guideline in 2001 (Van Derveer and Canton (1997)) suggest that a sediment threshold of 2 μg/g would be protective, there is still insufficient primary data and much uncertainty for derivation of a full or an interim guideline.

Adding to this uncertainty is the inherent variability in sediment metals data, including those for Se. Seiving sediments and analysing only the fine (< 63 μm) fraction can reduce that variability (BC MoE 2013). However, sediment data may have other sources of variability due to analytical or sample collection methods used. For example, one site in the Elk Valley (Barnes Lake wetland) was sampled in 2002 and again in 2006 over which time the Se concentration in sediment nearly doubled (from 2.0 to 3.9 μg/g). The difference may reflect the different collection methods used in the surveys. In 2002, a ponar was used and three subsamples of the top 3 cm were analysed, but in 2006, a 2-inch corer was used and seven to eleven subsamples of the top 1 cm were analysed (Minnow Environmental Inc 2004; Minnow *et al.* 2007). Alternatively, the difference may indicate a potential source(s) of Se in the Barnes Lake wetland as coal mining expands in the valley. This example underscores the difficulty in using sediment data, in isolation of other indicators of Se accumulation in the environment, since data variability could be the result of several factors. More information on monitoring techniques and approaches are provided in Section 9.0.

The sediment guideline concentration of 2 μg/g was compared with background levels measured in eastern (Table 4.7) and western Canada (Table 4.8). The sediment Se concentrations in Tables 4.7 and 4.8 represent whole samples (not sieved prior to analysis), with one reported value for Murry River sediment Se representing analysis of the < 63 μm fraction (Table 4.8). Most reference sites in Tables 4.7 and 4.8 had Se sediment concentrations below 2 μg/g, with only a few exceptions in areas having naturally high Se.

Since background is typically below 2 μg/g, and the published benchmarks, thresholds and objectives for sediment Se are 2 μg/g or less, this sediment concentration should be protective of the most sensitive organisms. Because there is no new primary literature available for an updated sediment Se guideline and uncertainty in the existing information, the status of the guideline will

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change from *interim*, to an *alert concentration*. No uncertainty factor was applied to this value since it is not a guideline. For most environments, the sediment alert concentration is protective and, along with data from other ecosystem compartments, will provide an early indication of the increased risk of impacts to aquatic organisms. Since background concentrations of Se in marine sediments are also typically well below 2 µg/g, the sediment alert concentration will also apply in marine environments (Sui and Berman 1989). In regions where true background sediment Se exceeds the alert concentration, site-specific water quality objectives may be considered in consultation with BC MoE.

The chronic sediment quality alert concentration for the protection of aquatic life is 2 µg/g, calculated as the mean concentration of at least 5 samples collected in a representative area (i.e., site).

8.4.3 Tissue Guidelines

The previous BC tissue residue Se guideline of 4 µg/g (dry weight) (Nagpal and Howell 2001) for whole-body fish was based on two key published studies. In the first, Hamilton and Wiedmeyer (1990) studied Se effects on growth and survival in Chinook salmon, reporting a range of no-effect water Se concentrations (35 to 70 µg/L) and no-effect whole-body tissue Se concentrations of 2 to 5 µg/g. Brix *et al.* (2000) re-evaluated these same data along with similar published data, and suggested the whole-body Se toxicity threshold (EC10) for cold-water fish of 6 µg/g. Nagpal and Howell (2001) converted the whole-body tissue EC10 of 6 µg/g to a wet weight concentration of 1.2 µg/g (assuming 80% moisture content), which was rounded to 1 µg/g, with no uncertainty factor applied. This value was then converted to a dry weight whole-body tissue guideline of 4 µg/g Se (also assuming 80% moisture content).

Dietary exposure is the predominant route of uptake (DeForest and Adams 2011). Waterborne exposure studies on fish accounts for some degree of uptake, with reported effects being associated with water-only exposures (Hodson *et al.* 1980; Hilton *et al.* 1982; Hicks *et al.* 1984; Cleveland *et al.* 1993; Hamilton 2004; Miller *et al.* 2007). Therefore, both routes of exposure were considered in updating the Se tissue guidelines.

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There are some differences in background tissue concentrations between freshwater and marine aquatic environments. Although it varies by species, some marine animals, such as birds and fish, often have higher tissue concentrations of Se than freshwater animals (Sui and Berman 1989; DeVink *et al.* 2008). Therefore, the tissue residue guideline for aquatic life applies only to freshwater fish.

8.4.3.1 Dietary Tissue

Selenium measurements in lower trophic level organisms may be used as a trigger for further action and may be valuable for environmental managers and practitioners (Lemly and Smith 1987; Lemly 1996; US DOI 1998; Wayland and Crosley 2006; Wayland *et al.* 2006, 2007; Canton *et al.* 2008). There are many advantages to sampling dietary organisms:

- periphyton and benthic invertebrates may be more Se tolerant;
- invertebrates are more abundant, and easier to sample than higher trophic levels;
- sampling non-commercial or non-charismatic species for Se risk assessment does not put populations at risk;
- invertebrates may be alternate bioindicators when target species are rare or agencies limit their collection; and,
- evaluating subtle changes to aquatic benthic invertebrate communities and if possible relating those changes to Se exposure can provide a means of assessing overall ecosystem impacts (e.g., Swift 2002; Pond *et al.* 2008).

For these reasons, concentrations of Se in the tissue of fish and other wildlife prey organisms will provide another compartment of the ecosystem to monitor Se bioaccumulation. The direct effects of Se on the prey organisms themselves can also be evaluated.

Some criticisms of using a dietary chronic Se guideline include: dietary Se is thought of as an indirect measure of toxicity, the observed responses can be highly variable, Se concentrations in some trophic levels can be highly variable, and characterisation and selection of appropriate indicator dietary species can be problematic (USEPA 2004; Malloy *et al.* 1999; DeForest and Adams 2011). There are also logistic problems associated with collecting enough dietary tissue for analysis if a laboratory requires larger volumes. Despite these limitations, diet is the critical

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exposure pathway for those organisms most at risk, so an understanding of dietary Se can aid in predicting bioaccumulation and toxicity (Lemly 1996; Canton *et al.* 2008; Ohlendorf and Heinz 2011). Some studies have shown that fish Se body burdens can be accurately predicted based on dietary Se intake, with an almost 1:1 relationship between dietary and body burden Se concentrations (Stewart *et al.* 2004; Hopkins *et al.* 2004; Stewart *et al.* 2010; Presser and Luoma 2010). Comprehensive monitoring programs evaluating the effects of Se include this important dietary component to provide data for site-specific modelling (Orr et al 2006, 2012). Hence, there is adequate justification across the literature to establish a dietary guideline for Se.

A summary of studies on the effects of dietary Se exposure to fish and birds (and their classification) is provided in Table 8.12. The estimates of dietary Se toxicity thresholds for fish and birds, range from 3 to 14 µg/g, with the majority of threshold values being less than 7 µg/g (see Table 7.9). Both the US DOI (1998) and Presser *et al.* (2004) suggest that a dietary Se concentration between 3 and 7 µg/g would present a marginal risk to aquatic life, while dietary Se concentrations above 7 µg/g present a substantive risk to higher trophic level consumers.

As discussed in Section 7.4.3.4, the assessment of deBruyn and Chapman (2007) suggests invertebrates may be at risk of sublethal effects when Se body burdens between 1 and 30 µg/g, which is within or lower than the range of the sublethal dietary effect thresholds for fish and birds. They reported that a dietary threshold of 3 µg/g resulted in a Se body burden that would cause a 15-40 % reduction in the growth of *Chironomus*. More research is needed to develop clearer toxicity thresholds for invertebrates before a guideline can be based on the analysis provided in deBruyn and Chapman (2007). Their evaluation does, however, suggest that a dietary Se guideline to protect fish and birds may protect invertebrate prey organisms. There are currently no published Se dietary guidelines, however, the lowest dietary threshold predictions and estimates for fish and birds are approximately 3 to 5 µg/g Se, respectively (Hilton *et al.* 1980; Hilton and Hodson 1983; Lemly 1996b; Hamilton 2004; Ohlendorf *et al.* 2010). Most of the reported dietary thresholds in Table 8.12 are NOEC and /or LOEC concentrations with only one reported EC10 estimate.

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The estimated geometric mean of the dietary Se NOEC/LOEC (i.e. MATC) for rainbow trout mortality was 4.8 µg/g, one of the lowest published toxicity thresholds (Goettl and Davis 1978). Hilton *et al.* (1980) exposed rainbow trout fry to six different diets containing between 0.07 and 13 µg/g sodium selenite, at water Se concentrations of 0.4 ± 0.2 µg/L. Based on the observations from the 20-week study, they suggested that long-term diets over 3 µg/g could ultimately be toxic to rainbow trout. In a more recent study, a LOEC for reduced growth of 4.6 µg/g dietary Se was reported for rainbow trout (Vidal *et al.* 2005). These studies suggest that a dietary guideline of between 3 and 5 µg/g for sensitive fish species would be protective.

Since an aquatic dietary guideline should also consider aquatic-dependent wildlife, thresholds for birds were evaluated. Most bird Se toxicity studies prior to the 1980s used domestic poultry (Ohlendorf 2003). Ort and Latshaw (1978) conducted laboratory feeding studies on adult chickens and found that a diet of 5.5 µg/g Se, over 28 weeks, resulted in significantly reduced egg hatchability.

After 1983, many laboratory studies were conducted on mallard ducks as a result of toxicosis in birds identified at Kesterson Reservoir (Ohlendorf 2003). Ohlendorf (2003) analysed laboratory toxicity data from six studies on mallard ducks, a species considered to be relatively sensitive to Se, and developed a dietary Se EC10 effects threshold for hatchability of 4.9 (3.6 – 5.7) µg/g. At approximately the same time, Adams *et al.* (2003) published mallard Se thresholds for deformity and hatchability using the same data but different statistical methods. Prompted by the re-analysis by Adams *et al.* (2003), Ohlendorf (2007) revised his dietary threshold estimate to 4.4 (3.8 – 4.8) µg/g. The studies on mallard were considered acceptable for development of a dietary tissue guideline based on an evaluation of the reports using CCME's guidance criteria for guidelines for the protection of wildlife (CCME 1998).

A study of spotted sandpiper in the Elk Valley, BC, showed a significant decrease in hatchability at two exposed sites where the mean dietary Se concentrations were 4.7 and 10.2 µg/g (Harding and Paton 2003). Wayland *et al.* (2007) studied the dietary risk to American dippers and harlequin ducks on coal mine-impacted streams in Alberta. Based on existing published toxicological literature, a simulation model, and risk assessment these authors predicted a dietary

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EC10 for reduced hatchability of 4 µg/g with a fairly wide 95% confidence interval of 0.5 – 7.3 µg/g Se (Wayland *et al.* 2007).

Dietary thresholds for fish and birds are similar (Doroshov *et al.* 1992; Harding and Paton 2003; Tashjian *et al.* 2006; Wayland *et al.* 2007). Hamilton *et al.* (2005) suggested that a dietary threshold of 4 µg/g would not be overly conservative for sensitive fish and bird species. Of the studies listed in Table 8.12, Hilton and Hodson (1980), Hilton *et al.* (1980), Hamilton *et al.* (1990), and Tashjian *et al.* (2006), were classified as primary data. Only the 60-day results for SeMet diet published in Hamilton *et al.* (1990) were used to derive the dietary guideline due to possible contamination of the diet containing San Luis Drain fish (DeForest *et al.* 1999). The study by Teh *et al.* (2004) was classified as secondary. Bird studies by Ort and Latshaw (1978), Stanley *et al.* (1996) and Heinz *et al.* (1989) were classified as primary according to CCME (1998) protocols for wildlife tissue guidelines. The remainder of the studies listed in Table 8.12 were unacceptable due to control mortality exceeding allowable limits (Doroshov *et al.* 1992; Vidal *et al.* 2005; Hardy *et al.* 2010), and/or no clear concentration response detected (Hardy *et al.* 2010).

An expert scientific panel reviewing Se studies in the Elk Valley BC agreed an area-specific dietary (benthic invertebrate) trigger of 5 µg/g would be protective of aquatic invertebrates, as well as fish and wildlife species (Canton *et al.* 2008).

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Table 8.12 Published dietary effect thresholds for selenium toxicity on fish and bird species.

Species	Se in diet (µg/g) dw	Effect	Study Classification	Reference
Acipenser transmontanus (white sturgeon)	10	Dietary Se "threshold" for histopathological alterations in kidney of juvenile sturgeon	1°	Tashjian *et al.* (2006)
Oncorhynchus tshawytscha (Chinook salmon)	9.6	EC10 reduced growth in larval fish, 60 day exposure (approximate dietary concentration for both SLD and SeMet diets)	1°	Hamilton *et al.* (1990) (cited in USEPA (2011a))
Oncorhynchus mykiss (rainbow trout)	4.6	LOEC for reduced growth in juveniles	U	Vidal *et al.* (2005);
	4.8	Geometric mean of dietary NOEC/LOEC for mortality (MATC)	1°	Goettl and Davies (1978) (cited in DeForest *et al.* (1999));
	>3.7/< 13.1	NOEC/LOEC for decreased body weight in juvenile fish exposed for 20 wks;	1°	Hilton *et al.* (1980)
	> 6.6/< 11.4	NOEC/LOEC for renal calcinosis in juvenile rainbow trout on low carb diet;	1°	Hilton and Hodson (1983)
Oncorhynchus clarkii bouvieri (Yellowstone cutthroat trout)	11.2	Dietary concentration associated with NOEC for larval mortality and deformity (no LOEC could be estimated)	U	Hardy *et al.* (2010)
Lepomis macrochirus (bluegill sunfish)	5.5	NOEC for edema and delayed yolk sac resorption	U	Doroshov *et al.* (1992)
	13.9	LOEC for edema		
Pogonichthys macrolepidotus (Sacramento splittail)	6.6	LOEC for deleterious effects on juvenile fish not exposed maternally to Se (> 50% deformity in larval fish at this dietary exposure concentration)	2°	Teh *et al.* (2004)
Poultry	5.5[1]	LOEC – Significant reduction in hatchability in laying hens	1°	Ort and Latshaw (1978)
Falco sparverius (American kestrel)	6 - 12	Dietary NOEC/LOEC for reductions in body mass after a six month feeding study	1°	Yamamoto and Santolo (2000)
Anas platyrhynchos (mallard ducks)	3.9[1]	NOEC for reproductive effects;	1°	Stanley *et al.* (1996)
	7.8[1]	LOEC – 33% reduced hatching and teratogenic effects rise sharply above this threshold;	1°	
	4.4[1]	NOEC for reproductive effects;		Heinz *et al.* (1989)
	8.9[1]	LOAEL – approx 17% reduction in duckling survival, 43% decrease in mean number of 6-day old ducklings		

[1]Dry weight calculated based on 10% moisture in diet as reported in Ort & Latshaw (1978), Stanley *et al.*(1996) and Heinz *et al.* (1989). Studies classified for guideline derivation as primary (1°), secondary (2°) or unacceptable (U).

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In 2008, a group of Se toxicology experts developed a site-specific standard for the Great Salt Lake in Utah, and using a summary of toxicological data, recommend a dietary EC10 for hatchability in birds of 4.9 µg/g, with a 95% confidence interval of 3.6 to 5.7 µg/g (CH2M Hill 2008).

Fish and bird dietary Se concentrations greater than 5 µg/g may exceed the thresholds for teratogenic effects (Ohlendorf *et al.* 2011). Since there is a narrow margin between adequate dietary Se concentrations and those thought to pose a risk to fish and wildlife, and because the form of Se is a determinant in the degree of risk for toxic effects, it may be difficult to accurately predict Se toxicity from dietary intake. However, the above evaluations suggest that dietary Se concentrations above 4 µg/g constitute a risk for excess bioaccumulation resulting in reproductive and non-reproductive effects to sensitive receptor fish and wildlife species. Since fish and birds may be consuming a mix of invertebrates and fish, the fish whole-body tissue residue guideline of 4 µg/g should align with the dietary guideline. Therefore, the BC interim dietary guideline is 4 µg/g.

While most reference area concentrations of invertebrate tissue will not exceed an interim dietary guideline of 4 µg/g, some areas with naturally high Se may have background tissue concentrations that are close to, or slightly exceed this guideline (see Table 4.7). The average of all invertebrate tissue Se data collected between 1996 and 2009 in the Elk Valley BC, at combined lotic and lentic reference sites was 3.9 (± 1.6) µg/g (calculated from data summary provided by Minnow Environmental Inc.). In regions where true background dietary tissue Se exceeds this value a careful examination of environmental conditions is warranted to evaluate the need to develop site-specific water quality objectives in consultation with BC MoE.

This guideline is designated interim because additional data are needed to verify the protection afforded by this value (BC MoE 2012a). Dietary concentrations exceeding this value would serve as a trigger for further investigation. While there are some studies that suggest this guideline may not protect highly sensitive invertebrate species, more definitive research is needed to define Se toxicity thresholds before a full guideline for protection of invertebrates may be proposed. No uncertainty factor was applied to this value because Se is a dietary requirement,

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some background levels of dietary Se approach this value, and there is need for additional data to confirm the guideline. Dietary Se evaluation should target known or likely prey organisms in the diet of sensitive receptor species, including other fish, and evaluate the presence of other contaminants.

The interim chronic dietary guideline to protect fish and aquatic-dependent wildlife is 4 µg/g Se (dry weight) measured as the mean concentration of at least eight replicate (composite) tissue samples representing appropriate invertebrate or other prey species. Further guidance on sample collection is provided in BC MoE (2012b).

8.4.3.2 Egg/Ovary Tissue

A Se guideline must consider both the reproductive effects resulting from the maternal transfer of Se and non-reproductive effects on early life stages (immediately after the onset of exogenous feeding) and juveniles. Both result primarily from the ingestion of dietary Se, but also from direct uptake of Se from water (Hermanutz 1992; DeForest 2008). Toxicity thresholds for non-reproductive effects in early life stages and juvenile fish are not as well defined as those for reproductive effects, but some researchers suggest the thresholds are similar (DeForest 2008; Janz *et al.* 2010; DeForest and Adams 2011; Table 8.13).

Egg or ripe ovary Se concentrations provide the most direct basis for predicting reproductive effects in fish and other wildlife (Sections 7.4.3.5 and 7.4.3.6) and are the preferred tissues for environmental assessments (deBruyn *et al.* 2008; Janz *et al.* 2010; DeForest and Adams 2011; Ohlendorf and Heinz 2011). While tissue guidelines may be more ecologically relevant than water or sediment, it presents several challenges in terms of implementation (Lemly and Skorupa (2007). In some cases, constraints on sampling fish, whether seasonal or regulatory, may preclude egg/ovary sampling, in which case analysis of whole-body, muscle, or muscle plug tissues can provide a reasonable indication of risk for reproductive effects from Se toxicity (DeForest and Adams 2011). While generic tissue relationships have been defined, species-specific and site-specific correlations (the most reliable) between tissue types are often developed and may be used to translate Se concentrations between tissue types to predict reproductive effects (deBruyn *et al.* 2008). Differences in tissue Se relationships do exist even

between closely related species, as demonstrated by Holm *et al.* (2005) who found a 7-fold increase in rainbow trout egg Se compared with muscle Se, while brook trout had only a 2-fold increase in egg Se over muscle Se at the same sites.

Some of the literature reporting Se toxicity thresholds measured as egg/ovary concentrations have been summarized in Table 8.13. Egg Se toxicity is evident in several fish species at concentrations of 12.7 µg/g and above (USEPA 2011b). DeForest and Adams (2011) recommended a combined egg/ovary threshold for fish of 17 µg/g, which considered data for several species including bluegill sunfish, brook trout, rainbow trout, brown trout, cutthroat trout, Dolly Varden char, northern pike, and white sucker. DeForest *et al.* (2011) recently proposed an egg/ovary Se guideline for fish of 20 µg/g, using a species sensitivity distribution (SSD) model with most of the same data.

The US EPA estimated genus mean chronic values (GMCV) for Se based on EC10s for the four most sensitive fish genera as *Oncorhynchus* (22.6 µg/g), *Micropterus* (20.4 µg/g), *Lepomis* (18.4 µg/g) and *Salmo* (17.8 µg/g) (C. Delos, pers. comm., US EPA, August 2011). The GMCVs for *Micropterus* and *Salmo* were taken from single published values (see Table 8.13). The GMCVs for *Oncorhynchus* were based on the geometric means of toxicity thresholds calculated from Holm *et al.* (2005) and Rudolph *et al.*(2008), and the GMCVs for *Lepomis* were derived from Doroshov *et al.* (1992), Hermanutz *et al.* (1996), and Coyle *et al.* (1993) (C. Delos, pers. comm., US EPA, August 2011).

The studies in Table 8.13 relate to fish species found in Canadian waters. However, much of this literature represents laboratory studies conducted on field-collected gametes (i.e., Kennedy *et al.* 2000, Holm *et al.* 2005, deRosemond *et al.* 2005, Muscatello *et al.* 2006, Rudolph *et al.* 2008, Elphick 2009, NewFields 2009, Nautilus Environmental and Interior Reforestation Co. Ltd. 2011) so are classified as unacceptable for guideline derivation. The basis for this classification is that the exposure of wild adults was not measured and not consistent, therefore the gametes represent variable exposure concentrations. In addition, adult females may have possibly been exposed to and influenced by other co-contaminants. However, field studies provide valuable information and were used as part of the weight of evidence in the derivation of the egg Se

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guideline. Hermanutz *et al.* (1992), Coyle *et al.* (1993), Carolina Power and Light (1997), were classified as primary literature since they were controlled laboratory feeding studies and met all other criteria.

Hardy *et al.* (2010) conducted a two and a half year feeding trial using Yellowstone cutthroat trout and calculated a NOEC of > 16.0 µg/g egg Se, for reproductive endpoints. Limitations of the study included low number of replicates, high variability, Se doses insufficient to elicit a clear toxic response, and high (19.5%) mortality in the control group in weeks 48-80. These limitations resulted in Hardy *et al.* (2010) being classified as unacceptable for direct use in guideline derivation.

The lowest Se egg tissue toxicity threshold based on the available primary EC10 estimates, including those for species less common in BC, is 12.7 µg/g (8.5 – 19.0) for bluegill sunfish, reported by Hermanutz *et al.* (1992, 1996) (based on reanalysis of this data by US EPA 2011b). However, there is some uncertainty associated with this estimate since toxicity estimates reported for this species by other researchers were higher; 16 to 24.6 µg/g egg Se. Developing a guideline using data for the genus *Oncorhynchus* may be more representative of fish in BC, and the EC10 data are slightly higher, more in line with other bluegill sunfish estimates. As depicted in Figure 7.2, there is a fairly narrow range of egg Se toxicity thresholds for fish (17 – 24 µg/g).

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Table 8.13 Summary of egg/ovary toxicity thresholds for fish from studies with combined water and dietary exposure.

Fish Species	Egg/Ovary Se Effect Threshold (µg/g dw)	Effect	Study Classification[b]	Reference
Oncorhynchus mykiss (rainbow trout)	22.6–26.9	Estimated EC15, 15% probability of larval deformities (61% moisture)	U	Holm *et al.* (2005)
	21.1[a] (13.0 – 34.2)	EC10 (95% CI) for skeletal deformity (reanalysis of Holm *et al.* 2005);	U	USEPA (2011a)
	23	EC10 for larval deformity (95% CI not reported) (reanalysis of Holm *et al.* 2005)	U	DeForest & Adams (2011)
Oncorhynchus clarkii bouvieri (Yellowstone cutthroat trout)	> 16.0	NOEC for larval mortality and deformity (no LOEC could be estimated)	U	Hardy *et al.* (2010)
Oncorhynchus clarkii lewisi (westslope cutthroat trout)	> 21.2	NOEC for larval mortality & deformity;	U	Kennedy *et al.* (2000)
	> 20.6	NOEC for larval deformity;	U	Rudolph *et al.* (2008)
	24.1[a] (16.0–36.3)	EC10 (95% CI) for alevin mortality (reanalysis of Rudolph *et al.* 2008);	U	USEPA (2011b)
	17	EC10 estimate for alevin mortality (95% CI not reported) (reanalysis of Rudolph *et al.* 2008);	U	DeForest & Adams (2011)
	19.0 (6.8–22.7)	EC10 (95% CI) for larval survival;	U	Elphick *et al.* (2009)
	24.8 (12.0–30.5)	EC10 (95% CI) for larval survival, revised based on egg Se analysis from alternate lab	U	Nautilus Environmental & Interior Reforestation Co. Ltd. (2011)
Salmo trutta (brown trout)	17.7 (13.4–23.3)	EC10 (95% CI) for alevin survival (15 d post swim-up);	U	NewFields (2009)
	17.8[a] (14.5–22.0)	EC10 (95% CI) analysis including hatchery fish (reanalysis of NewFields 2009)	U	USEPA (2011a)

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Table 8.13 (con't)

Fish Species	Egg/Ovary Se Effect Threshold (µg/g dw	Effect	Study Classification[1]	Reference
Salvelinus fontinalis (brook trout)	>20	NOEC for larval fish (61% moisture) (reported as EC06 by DeForest and Adams 2011)	U°	Holm *et al.* (2005)
Esox lucius (northern pike)	20.4 (11.1–29.7)	EC10 (95% CI) for larval deformity	U	Muscatello *et al.* (2006)
Catostomus commersoni (white sucker)	25.6	EC12, mean Se concentration associated with 12% larval deformity	U°	deRosemond *et al.* (2005)
Lepomis macrochirus (bluegill sunfish)	3.9/21.1 (9.1)	NOEC/LOEC (geometric mean) for larval edema;	1°	Doroshov *et al.* (1992)
	20.1[2] (6.3–63.8)	EC10 (95% CI) for larval edema (reanalysis of Doroshov *et al.* 1992);	1°	USEPA (2011b)
	16	EC10 for larval edema (reanalysis of Doroshov *et al.* 1992);	1°	DeForest and Adams (2011)
	12.7[2] (8.5–19.0)	EC10 (95% CI) for larval edema (reanalysis of Hermanutz *et al.* 1992, 1996);	1°	USEPA (2011b)
	24.6[2] (21.2–28.5)	EC10 (95% CI) for larval survival (reanalysis of Coyle *et al.* 1993)	1°	USEPA (2011b)
Micropterus salmoides (largemouth bass)	20.4[2] (13.8–30.1)	EC10 (95% CI) for larval survival (reanalysis of Carolina Power and Light 1997)	1°	USEPA (2011b)

[1]Studies classified for guideline derivation as primary (1°), secondary (2°) or unacceptable (U).
[2]Denotes endpoints of the four most sensitive fish genus used by US EPA to derive their egg Se tissue criteria (not published), rounded to one significant decimal.

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Determining an appropriate uncertainty factor to apply to the lowest EC10 concentration, is a balance between ensuring Se levels that meet nutritional needs while avoiding levels that may cause adverse effects; this margin is very narrow. The minimum uncertainty factor of 2 results in a value that meets the balance between adequacy and protection, while addressing the inherent uncertainties in published toxicity threshold estimates.

The mean EC10 for rainbow trout (22.05 µg/g egg Se) of studies by Holm (2002) and Holm *et al.* (2003, 2005) divided by an uncertainty factor of 2 results in a fish egg Se WQG of 11.03 µg/g. Applying an uncertainty factor of 2 to the mean of reported EC10s for westslope cutthroat trout (21.97 µg/g), results in a guideline of 10.99 µg/g, and to the US EPA GMCV for *Oncorhynchus* (22.56 µg/g), results in a value of 11.28 µg/g. These estimates converge around 11 µg/g Se, which supports this value as an egg/ovary Se guideline for BC.

EC10 estimates for other species common in Canada including rainbow trout, brook trout, brown trout, northern pike, white sucker, bluegill sunfish, and largemouth bass, have EC10 egg Se toxicity thresholds that range from 12.7 µg/g for bluegill sunfish, to 25.6 µg/g for white suckers (Table 8.13). Although Se toxicity data is lacking for a broad cross-section of fish species, the existing information indicates that 11 µg/g egg Se would be protective of the more sensitive fish species until more data are available.

This proposed Se WQG for egg/ripe ovary Se may be compared with the reported confidence intervals for some of the published EC10 estimates. The lower confidence intervals associated with reproductive EC10 estimates for rainbow and cutthroat trout (Table 8.13), range between 13 and 16 µg/g egg/ovary Se, which are higher than a guideline value of 11 µg/g indicating the guideline will be protective. Additionally, a guideline of 11 µg/g is within the US DOI (1998) range of thresholds that represent a marginal risk of reproductive impairment in sensitive fish species (7 – 13 µg/g egg/ovary Se).

The dose-response curve for Se is steep (Figure 7.3) which heightens the risk of effects to populations at concentrations not much above individual EC10 levels. Models have been developed to predict the population response based on established individual-level toxicity

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thresholds (Van Kirk and Hill 2007; deBruyn 2009). deBruyn (2009) estimated the population-level EC10 threshold for decline in westslope cutthroat trout to be approximately 28 µg/g egg Se, which is only 3 to 5 µg/g Se greater than the individual-level fish EC10 egg toxicity threshold for that species. The overlapping 95% confidence intervals for individual-level cutthroat trout EC10s reported by US EPA (2011b) (CI = 16 – 36.3) and Nautilus Environmental and Interior Reforestation Co. Ltd. (2011) (CI = 12.0 – 30.5), with the population effect threshold of 28 µg/g is further demonstration of the rapid transition from individual- to population-level effects.

DeForest (2009) summarised background Se fish tissue data (egg, muscle and whole-body) but stated there was some uncertainty in asserting that data used in the summary were all truly "reference" and that some fish could represent mixed exposure. This uncertainty can also be the case in fish captured in BC and other Canadian waters. Background fish tissue concentrations for trout reported from reference sites in BC and Canadian waters are less than the 11 µg/g egg/ovary guideline, even in areas with relatively high background Se. Egg Se tissue concentrations for rainbow and brook trout in reference areas of studies published by Holm *et al.* (2005) were below the egg Se guideline of 11 µg/g (8.96 ± 1.02 and 3.33 ± 0.26 µg/g, respectively, Table 4.11). The same holds true for westslope cutthroat trout eggs sampled at sites in BC (7.59 ± 3.79 µg/g, Table 4.11).

There are however, some exceptions where background fish egg Se concentrations are close to, or slightly above the guideline. Since many fish species move extensively throughout a watershed, dietary Se exposure can be varied and Se tissue concentrations may reflect movement in and out of Se-contaminated areas. Fish captured in reference areas may not always represent truly unexposed fish. Rudolph *et al.* (2008) compared two lentic areas in their study examining toxicity thresholds for westslope cutthroat trout in the Elk River, BC. O'Rourke Lake, the reference area, had egg Se tissue concentrations (12.3 to 16.8 µg/g dw) which overlapped with those found at the exposed site, Clode Pond (11.8 to 140.0 µg/g dw). The egg concentrations in O'Rourke Lake are not typical of an unexposed site and might be considered an anomaly given that water concentrations of Se in the lake were less than the detection limit of 1.0 µg/L. Lentic areas can be extremely sensitive to Se bioaccumulation which may account for the higher than expected background. Adding to this, is the fact that O'Rourke Lake was stocked with westslope

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cutthroat trout on three occasions between 1985 and 1992 (BC Environment, Fish and Wildlife Branch internal files), and did not have any resident fish prior to the stocking program (Elkford Rod and Gun Club 1984). Therefore, there is uncertainty in the use of data from this site as a reflection of typical egg Se background in fish.

Minnow *et al.* (2007) reported mean egg Se concentrations in westslope cutthroat trout at lotic and lentic reference sites collected between 1996 and 2006, were 6.5 µg/g at and 8.1 µg/g, respectively. In this period, individual egg Se levels exceeded 11 µg/g on one occasion in 2006 (egg Se was 11.5 µg/g) at a lentic site at the Barnes Lake wetland site (Minnow *et al.* 2007). However, sediment Se at Barnes Lake in 2002 was 2 µg/g, yet four years later it had nearly doubled to a concentration of 3.9 µg/g (Minnow *et al.* 2007). This suggests that the site may not be truly reference, or was receiving some anthropogenic source of Se. The investigators could not rule out that females captured at reference sites may have been exposed to coal mining influences due to their large home range (Minnow *et al.* 2007). This data underscores the difficulty in accurately characterizing Se concentrations in organisms able to move in and out of contaminated zones.

McDonald *et al.* (2010) conducted a study on Se toxicity in Dolly Varden char in north eastern BC. They reported that mean Se egg concentrations at reference sites were between 5.4 and 11 µg/g. The egg Se concentrations at two of the three reference sites included in the study analysis were 10.5 and 11.0 µg/g which are relatively high compared with unexposed fish from other locations in BC. A reference clutch of eggs with 11 µg/g Se was removed from the analysis due to its very low survival rate (3%), which suggests uncertainty as to whether these reference fish with high egg Se may be been previously exposed.

The evaluation of background data suggests that at most reference sites a Se WQG of 11 µg/g would not be exceeded. The guideline does acknowledge some background tissue Se concentrations for some tissue types or species may be naturally elevated (e.g., sculpin). In areas where true background fish tissue Se exceeds the guideline, and unexpected sources of Se have been evaluated and ruled out, site-specific water quality objectives may be considered in consultation with the BC MoE.

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The chronic egg/ovary tissue guideline for the protection of fish is 11 µg/g, calculated as the mean concentration of at least eight samples (eggs or ripe ovary from 8 individual females) collected at a representative area (site), and reported as dry weight.

8.4.3.3 Whole-Body Tissue

A whole-body Se guideline is broadly applicable, and may be more appropriate for practical reasons (USEPA 2004; DeForest and Adams 2011). For example, when investigating non-reproductive effects of Se on early life-stage and juvenile fish, whole-body Se concentrations are the most appropriate measure. In situations where juvenile or small-bodied fish species are of interest, whole-body Se analysis may be the only option for monitoring. While whole-body Se concentrations may not be the most direct measure of potential reproductive effects in adults, for the reasons stated above, it has been retained as a guideline. The existing BC whole-body tissue Se guideline of 4 µg/g (Nagpal and Howell 2001) was compared to reported data for both reproductive and non-reproductive studies.

Selenium toxicity studies reporting thresholds as whole-body Se concentrations are presented in Table 8.14. Of these, Hilton *et al.* (1980), Hamilton *et al* (1990) (only the 60-day results for SeMet diet), Cleveland *et al.* (1993), Lemly (1993b), Coyle *et al.* (1993), Hermanutz *et al.* (1996) and McIntyre *et al.* (2008) were all classified as primary literature. The studies by Hodson *et al.* (1980) and Hunn *et al.* (1987) were primary and although they did not incorporate a dietary exposure component, they were considered in guideline derivation. Hilton and Hodson (1983) was considered primary but the authors reported toxicity thresholds measured as liver Se concentrations which had to be converted to whole-body tissue residues (MATC adopted from USEPA 2004). Two studies were deemed unacceptable; Vidal *et al.* (2005) and Hardy *et al.* (2010) had higher than acceptable control mortality. Additionally, Hardy *et al.* (2010) was unable to demonstrate a clear toxic response to Se at the dietary exposures used in their study.

There were uncertainties noted in some of the published whole-body toxicity thresholds for fish. Vidal *et al.* (2005) studied the effects of Se on larval rainbow trout, and found a whole-body toxicity threshold (LOEC) for reduced growth was < 4.8 µg/g Se[37]. However, the reductions in

[37] Conversion of wet weight to dry weight based on 75% moisture content.

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growth at the highest dietary exposure were not statistically different than controls, and whole-body Se concentrations were variable in test fish at 60 days and 90 days of exposure (DeForest *et al.* 2006). Vidal *et al.* (2005) acknowledged that Se body burden decreased between 60 and 90 days and suggested it may have been caused by growth dilution.

Although Vidal *et al.* (2005) was classified as unacceptable for derivation of WQGs, their results were consistent with many other primary studies (Hamilton *et al.* 1990 (60-day results for SeMet diet); Hilton *et al.* 1980; Hilton and Hodson 1983; Lemly 1993b; Hunn *et al.* 1993; Cleveland *et al.* 1993).

The lowest whole-body tissue Se thresholds for species relevant to BC were for Chinook salmon and rainbow trout. The US EPA (2004) estimated a whole-body EC20 for skeletal deformity in rainbow trout of 5.85 µg/g, using data from Holm (2002) and Holm *et al.* (2003). Of the studies listed in Table 8.14, some of the lowest effect thresholds for salmonids are reported by Hamilton *et al.* (1990), Hilton *et al.* (1980), and Vidal *et al.* (2005). Acknowledging the criticism from DeForest *et al.* (1999; 2006), only the 60-day results for SeMet diet from the Hamilton *et al.* (1990) study used for guideline derivation. Hilton *et al.* (1980) reported Se concentrations for fish carcasses (minus organs) at the dietary exposure concentrations, so Se no-effect levels on a whole-body basis would be expected to be higher.

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Table 8.14 Summary of whole-body Se toxicity thresholds for reproductive and non-reproductive end points in fish, including studies with dietary and water-only Se exposure.

Fish Species	Whole-body Se effect threshold (µg/g dw)	Effect	Study Classification[1]	Reference
Oncorhynchus tshawytscha (Chinook salmon)	5.3/10.4	NOEC/LOEC for reduced juvenile growth (SeMet diet, 60-days)	1°	Hamilton *et al.* (1990)
	4.3	EC10 for increased mortality on seawater challenge (smolts) (reanalysis of Hamilton *et al.* 1990)	1°	DeForest and Adams (2011)
Oncorhynchus mykiss (rainbow trout)	> 5	NOEC for increased juvenile mortality (feeding study);	1°	Hilton *et al.* (1980)
	< 1.8	LOEC for reduced growth in juveniles (water-only exposure);	1°	Hodson *et al.* (1980)
	8	MATC for reduced juvenile growth (carcass [Se] equivalent to whole-body, see USEPA (2004) methods);	1°	Hilton and Hodson (1983)
	< 4.3	LOEC for increased mortality in fry, (water-only exposure);	1°	Hunn *et al.* (1987)
	5.9	EC20 for craniofacial deformity (reanalysis of Holm *et al.* 2005);	U	USEPA (2004)
	<4.8	LOEC for reduced larval growth (assuming 75% moisture)	U	Vidal *et al.* (2005)
Oncorhynchus clarkii lewisi (westslope cutthroat trout)	11.7[2] (4.6 – 13.8)	EC10 (95% CI) for larval survival based on egg Se EC10 = 19 (6.8 – 22.7);	U	Elphick *et al.* (2009)
	13.8[2] (7.7 – 18.1)	EC10 (95% CI) revised based on egg Se analysis from different lab based on egg Se EC10 = 24.8 (12 – 30.5).	U	Nautilus Environmental & Interior Reforestation (2011)
Oncorhynchus clarkii bouvieri (Yellowstone cutthroat trout)	> 11.4	NOEC for larval mortality and deformity, no LOEC could be estimated.	U	Hardy *et al.* (2010)
Esox lucius (northern pike)	9.46	Whole-body EC10 for larval deformity	U	Muscatello *et al.* 2006
Lepomis macrochirus (bluegill sunfish)	<5.9	LOEC for reduced survival based on winter temp regime (4° C);	1°	Lemly (1993b)
	9.6	EC10 for increased mortality at winter temp regime (4° C);	1°	McIntyre *et al.* (2008)
	3.8/5.0	NOEC/LOEC for increased mortality (water only exposure);	1°	Cleveland *et al.* (1993)
	7/16	NOEC/LOEC reduced survival in larvae;	1°	Coyle *et al.* (1993)
	8	EC10 reduced larval survival;	1°	DeForest and Adams (2011)
	4.4/21.8	NOEC/LOEC for larval edema;	1°	Hermanutz *et al.* 1996
	7.7	EC10 for larval edema (reanalysis of Hermanutz *et al.* 1996)	1°	DeForest and Adams (2011)

[1]Studies classified for guideline derivation as primary (1°), secondary (2°) or unacceptable (U).
[2]See Appendix A for tissue conversions (random effects log-log regression model) used for deriving egg to whole-body Se estimates.

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Research assessing toxic responses in fish from water-only exposures has shown that early life stage and juvenile fish may be sensitive to Se when based on whole-body tissue accumulation (Hodson *et al.* 1980; Hamilton and Wiedmeyer 1990; Cleveland *et al.* 1993). Some authors exclude water-only Se exposure studies on juvenile fish when deriving toxicity thresholds, stating those studies have limited relevance to natural Se exposure (i.e., lacking dietary exposure component) (DeForest *et al.* 1999; USEPA 2004; deBruyn *et al.* 2008; DeForest 2008; DeForest and Adams 2011). However, excluding such data has been criticized by other researchers who state that this approach is selective and may result in erroneous conclusions (Skorupa 1999; Hamilton 2003).

Despite this controversy regarding juvenile fish toxicity threshold predictions based on dietary versus water-only exposures to Se, more recent studies have shown that physiological changes can result when early life-stage and juvenile rainbow trout are exposed to waterborne Se (Palace *et al.* 2004; Miller *et al.* 2007). Water sources of Se can contribute to toxicity and, since Se residues in fish are the sum total of dietary and aqueous routes of exposure, water-only exposure evaluations of Se should not be disregarded as irrelevant (Hamilton 2003; Janz *et al.* 2010). Since water contributes at least in part to toxic responses in fish, water-only exposure studies were considered in the derivation of the whole-body guideline.

Hodson *et al.* (1980) and Hunn *et al.* (1987) conducted studies using water-only Se exposures on early life stages of rainbow trout. The results of Hodson *et al.* (1980) suggest a whole-body Se toxicity threshold of < 1.8 µg/g, which was the reported LOEC for reduced growth in juveniles after a 44-week exposure at the highest experimental dose of 53 µg/L. Hodson *et al.* (1980) reported effects at lower experimental doses for other endpoints such as decreased calcium in bone (12 µg/L), reduced median time to hatch (16 µg/L) and reduced survival of eyed eggs (26 and 47 µg/L exposure groups), but did not report the associated whole-body Se residues for these exposure groups. Hunn *et al.* (1987) reported a LOEC for increased mortality in fry of < 4.3 µg/g after a 90-day exposures ≥ 47µg/L Se. Hilton *et al.* (1980) conducted laboratory feeding studies on juvenile rainbow trout and found increased mortalities evident at body burdens in excess of 5 µg/g. Although some of these studies did not include dietary exposures, they suggest that a whole-body tissue Se guideline of 4 µg/g may only marginally protect sensitive life stages of

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fish. More research is needed to establish a more precise estimate of the toxicity thresholds for early life stage and juvenile rainbow trout and other sensitive fish species.

There are uncertainties regarding some of the published whole-body toxicity thresholds for fish. Vidal *et al.* (2005) studied the effects of Se on larval rainbow trout, and found a whole-body toxicity threshold (LOEC) for reduced growth was < 4.8 µg/g Se[38]. DeForest *et al.* (2006) expressed concern regarding the variability in the concentration-response relationship reported by Vidal *et al.* (2005). DeForest (2008) pointed out the uncertainty in determining non-reproductive effects on naive larval or juvenile stages of fish (e.g., Hilton *et al.* 1980, Hilton and Hodson 1983, Hamilton *et al.* 1990, Vidal *et al.* 2005) and questioned whether some studies represented realistic environmental exposure conditions and responses.

Based on toxicity data for several fish species, DeForest and Adams (2011) proposed a whole-body Se tissue EC10 of 8.1 µg/g using a species sensitivity distribution (SSD) approach. The authors noted however, that the whole-body Se EC10 estimate reported for Chinook salmon is 4.3 µg/g (Hamilton *et al.* 1990), suggesting this species may have a much lower Se threshold for juvenile mortality. Based on the work of Hamilton *et al.* (1990), Hilton *et al.* (1980), Hunn *et al.* (1987) and Vidal *et al.* (2005), the general whole-body Se EC10 of 8.1 µg/g proposed by DeForest and Adams (2011), if adopted as a guideline, would not protect the most sensitive species, such as Chinook juveniles and rainbow trout. Following BC's protocol for deriving guidelines, an uncertainty factor would need to be applied to DeForest and Adams's (2011) EC10 of 8.1 µg/g. Applying the minimum uncertainty factor of 2 results in a whole-body Se tissue guideline of 4 µg/g. Although this is only slightly below the EC10 estimate reported for Chinook salmon, given the uncertainties of the Hamilton *et al.* (1990) study, the weight of evidence continues to support a WQG of 4 µg/g to protect the majority of sensitive species and life stages.

Other published evaluations of salmonid data suggest whole-body Se toxicity thresholds lower than that recommended by DeForest and Adams (2011). Van Kirk and Hill (2007) modelled cutthroat trout population-level response based on several studies reporting individual-level

[38] Conversion of wet weight to dry weight based on 75% moisture content.

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responses to Se exposure based on whole-body tissue residues, many of which were primary literature. Through modelling, the authors determined that population-level response thresholds may be lower than predicted for individual toxicity thresholds due to density-dependent factors and the unpredictable spatial and temporal natural environmental conditions. The authors suggested that cutthroat trout populations would be protected at whole-body Se concentrations less than 7.0 µg/g (Van Kirk and Hill 2007). In a subsequent publication, Gledhill and Van Kirk (2011) modelled the effects of Se exposure on long-term effects to population size in bluegill sunfish, hoping to refine and expand the usefulness of the model. Their model showed that at a whole-body Se concentration of 4 µg/g, the predicted mean mortality response was 9.54% with a 95% prediction range of 0.65 to 63.13%. They stated that while the population-level response would be small at a mean individual-level response of 10%, if the first-year survival rate is low, equilibrium population sizes may be near or below 50% of carrying capacity when whole-body Se concentrations are 4 µg/g. The authors concluded that their model supports a whole-body Se threshold for fish of 4 µg/g.

Background whole-body Se concentrations from monitoring data collected across Canada were compared to the current guideline. Whole-body tissue residues at reference sites, even in areas of high Se geology, are typically less than the 4 µg/g guideline (refer to Table 4.11 and 4.12). There are exceptions in some fish species at reference locations where geological Se is naturally elevated or in environments where Se accumulation is enhanced. An example of this is in north eastern BC where whole-body sculpin tissue Se concentrations at some reference sites slightly exceeded 4 µg/g (Carmichael and Chapman 2006). However, sculpin may be an exception to typical background Se whole-body tissues found in other species. Data collected in reference areas in the Flathead River BC, also had relatively high mean whole-body Se concentrations (7.04 µg/g) for slimy sculpin (Henderson and Fisher 2012). Little is known about the toxicity effect thresholds for sculpin.

In 2004, at Blind Creek in northern BC, whole-body rainbow trout tissues were collected prior to coal mining activities where average tissue Se concentrations were 3.37 µg/g, (Golder Associates Ltd. 2009). Baseline data collected in 2010 for the proposed Chu Molybdenum Mine south of Vanderhoof, BC (not in Table 4.11) demonstrated that mean whole-body tissue Se for rainbow

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trout was 2.9 (± 1.78) µg/g (data submitted by Warren Robb, TTM Resources, Vancouver, BC). However, at two of the 13 monitoring sites, mean Se tissue concentrations were over 4 µg/g (4.55 ± 0.78 and 8.18 ± 0.75 µg/g Se). This suggests that a whole-body Se guideline of 4 µg/g is within background Se tissue concentrations for a majority of sites, with only some exceptions.

The whole-body Se tissue guideline was also compared to data for alternate tissue types (egg and/or muscle) using published conversion relationships. Several years of monitoring westslope cutthroat trout in the Elk Valley, BC, has resulted in the development of fairly robust relationships between Se tissue concentrations for different tissue types (egg, muscle, and calculated whole-body) (Minnow *et al.* 2007; Schwarz 2011). Tissue conversion models can be helpful, particularly where data do not exist for a specific tissue type. However, since model uncertainty cannot be eliminated, caution should be exercised when using Se threshold or guideline concentrations for one tissue type to estimate Se values in other tissue types. Using a random effects log-log tissue regression to translate an egg Se guideline of 11 µg/g (based on reproductive endpoints), to whole-body and muscle tissue concentrations, the values become 7.1 and 6.5 µg/g, respectively (Schwarz 2011).

The translations of toxicity thresholds from egg to whole-body (and muscle) might suggest there could be some upward adjustment of a whole-body (and muscle) guideline to approximately 6 µg/g. However, this translation does not take into consideration model uncertainties and may not account for possible differences in toxicity related to maternal transfer of Se versus those from direct dietary and waterborne exposure to early life stages and juvenile fish. As well, there are several reported toxicity thresholds for sensitive species that are below 6 µg/g.

The toxicity studies discussed above on juvenile rainbow trout (Vidal *et al.* 2005; Hilton *et al.* 1980; Hilton and Hodson 1983) and Chinook salmon (Hamilton *et al.* 1990) report very low whole-body thresholds. These low whole-body EC10 thresholds suggests that 4 µg/g Se in whole-body tissue would protect sensitive fish species and life stages. A whole-body guideline of 4 µg/g is consistent with recommendations in Presser *et al.* (2004), who suggested that whole-body Se concentrations between 4 to 6 µg/g represents marginal risk of harmful effects to fish. Ohlendorf *et al.* (2011) state that negative effects are known to occur at whole-body concentrations in fish as low as 4 to 6 µg/g. Lemly (1996a) recommended 4 µg/g Se in whole-

body tissue as an effect threshold for fish. An uncertainty factor was applied in the original whole-body fish tissue Se WQG published by Nagpal and Howell (2001) so no additional uncertainty factor is necesary. In some cases, background whole-body concentrations in fish may exceed 4 µg/g Se, and development of a site-specific or species-specific objective may be considered in consultation with BC MoE.

The chronic whole-body tissue guideline for the protection of fish is 4 µg/g calculated as the mean concentration of at least eight tissue samples collected at a representative area and reported as dry weight.

8.4.3.4 Muscle Tissue

Muscle tissue has been used to evaluate the exposure of fish to Se as an alternative to egg and whole-body analysis, though it may not be the most direct measure of toxic response (Waddell and May 1995; deBruyn *et al.* 2008). Muscle can be a reasonable and useful surrogate, particularly if reliable species-specific tissue relationships have been developed (deBruyn *et al.* 2008) such as those for westslope cutthroat trout in the Elk River BC (Minnow *et al.*2007) and for rainbow trout in Alberta (Holm *et al.* 2005).

Toxicity thresholds relating specifically to muscle tissue residues are limited and rarely consider species native to BC (Table 8.15). These include striped bass (Coughlan and Velte 1989), and bluegill sunfish (Finley 1985; Hermanutz *et al.* 1992, 1996). Coughlan and Velte (1989) determined a muscle Se LOEC of < 15.2 µg/g in striped bass for effects which included increased mortality, reduced growth and condition factor, as well as changes in behaviour (food avoidance) and histopathology in liver and kidney tissue. Hermanutz *et al.* (1992) exposed bluegill to 10 and 30 µg/L in outdoor stream mesocosms and observed reduced hatching and larval survival as well as increased larval deformity at muscle Se concentrations of 7.2 and 11.2 µg/g, respectively. In a related study, Hermanutz *et al.* (1996) used stream mesocosms to expose bluegill sunfish to 2.5 and 10 µg/L. In this study, larval deformities were significantly higher in the 2.5 and 10 µg/L treatments than in the controls with a resulting LOEC of approximately 4 µg/g mean muscle tissue Se. Finley (1985) found increased mortality in adult bluegill sunfish fed a diet of Se-contaminated mayfly nymphs from Belews Lake (13.6 µg/g Se) which was associated with muscle Se concentrations of 20 to 32 µg/g (assuming 75% moisture). Of the

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studies mentioned, only the two studies by Hermanutz *et al.* (1992, 1996) were classified as "primary" for the purpose of guideline derivation. Coughlan and Velte (1989) used a diet augmented with fish from Belews Lake, which may have contained co-contaminants so was deemed unacceptable. Similarly, Findley (1985) was deemed unacceptable as a result of the exposure diet containing mayfly nymphs from Belews Lake (possible co-contaminants) and poor test replication.

Other studies in Table 8.15 provide supporting evidence for a fish tissue guideline based on muscle Se concentrations. Holm *et al.* (2005) found that in rainbow trout, mean egg Se concentrations were 7-fold higher than mean muscle Se (reported as wet weight concentrations). Using this simple relationship, the reported egg-based toxicity threshold (EC15 of 8.8 to 10.5 µg/g egg Se, wet weight) for larval deformity in rainbow trout is calculated to be 5 to 6 µg/g (dry weight, assuming 75% moisture) in muscle tissue (Holm *et al.* 2005)[39]. Presser and Luoma (2006) published a rainbow trout Se toxicity threshold for muscle of 4.3 µg/g based on converting the ovary threshold reported by Holm *et al.* (2003) to a muscle tissue value. This value was also cited by Ohlendorf *et al.* (2008; 2011, supplemental data). This provides additional supporting evidence for a Se guideline of 4 µg/g in fish muscle tissue.

The Se egg to whole body translation for rainbow trout (Schwarz 2011) is based on data from Holm *et al.* (2005), Casey and Siwik (2000), and Mackay (2006). The random effects log-log regressions in Schwarz (2011) for rainbow trout were used to translate egg to muscle toxicity thresholds reported by Holm *et al.* (2005). Based on egg tissue EC15s for skeletal deformity (22.6 to 29.6 µg/g, Table 8.13), the resulting range of muscle tissue thresholds using the random effects model, was 7.2 to 9.4 µg/g. Translating the US EPA (2011a) estimate of an egg EC10 for rainbow trout larval deformity reported by Holm *et al.* (2005) the resulting muscle EC10 (95% CI) was 6.7 (4.1 – 10.9) µg/g (Table 8.15). While there was some variability in translated estimates for rainbow trout muscle tissue toxicity thresholds, the low range was from 4.3 to 7.2 µg/g, which supports a muscle guideline for fish being slightly lower than that, at 4 µg/g Se.

[39] The reported EC15 range of 8.8 to 10.5 µg/g egg Se wet weight, was divided by seven to yield the corresponding Se effect threshold range for muscle (1.25 to 1.5 µg/g wet weight). This was then converted to dry weight using 75% moisture content.

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Other researchers have relied on existing literature or species-specific tissue relationships developed during their studies or through monitoring programs to translate thresholds from one tissue type to another. For example, Muscatello *et al.* (2006) reported a muscle tissue EC10 (95% CI) for larval deformity in northern pike of 13.85 µg/g Se (3.54 – 24.16 µg/g) which was converted from an egg Se concentration estimate using the US EPA's (2004) tissue conversion equations. Using the random effects regression in Schwarz (2011), the conversion from muscle to egg EC10 (95% CI) for pike deformity was 11.5 µg/g (5.9 – 17.3 µg/g) (Table 8.15). For brown trout, a Se-sensitive species, NewFields (2009) reported an egg EC10 (95% CI) for alevin survival of 17.7 µg/g Se (13.4 – 23.3 µg/g). This was converted to a muscle Se estimate using the random effects regression, for an EC10 of 4.3 µg/g Se (4.0 – 4.7 µg/g) (Table 8.15).

The proposed egg Se guideline of 11 µg/g was converted to a muscle concentration for two sensitive BC species, rainbow and cutthroat trout using the species-specific regressions in Schwarz (2011), resulting Se residue estimates of 3.5 and 6.5 µg/g Se, respectively. The evaluation of the low toxicity thresholds based on muscle in Chinook salmon (Hamilton *et al.* 1990), brown trout (NewFields 2009), rainbow trout (Holm *et al.* 2005) and westslope cutthroat trout (Rudolph *et al.* 2008) all support a muscle guideline of 4 µg/g Se.

Muscle Se residue data from reference sites in BC and Alberta demonstrates that for many areas and fish species, muscle Se concentrations are less than 4 µg/g, with a few exceptions (see Table 4.12). Data collected in 2009 in the Elk Valley, BC, showed that westslope cutthroat trout at one reference lake site (Elk Lake) had a mean (SD) muscle tissue Se concentration of 2.98 (± 0.78) µg/g, n=4 (Minnow *et al.* 2011). Data collected by MoE staff in 2006 on the Flathead River, an adjacent watershed to the Elk, showed that mean (SD) concentrations of Se in whole-body samples of westslope cutthroat trout were 1.29 (± 0.28) µg/g, n=20 (Henderson and Fisher 2012).

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Table 8.15 Summary of toxicity thresholds based on muscle selenium concentrations for various fish species.

Fish Species	Muscle Se Effect Threshold (µg/g dw)	Effect	Study Classification[1]	Reference
Oncorhynchus mykiss (rainbow trout)	4.6 – 7.2	EC15 for larval deformity based on 7-fold increase from muscle to egg Se;	U	Holm *et al.* (2005)
	7.2 – 9.4[2]	EC15 for larval deformity translated to muscle using random effects conversion model;		
	6.66[2] (4.08 – 10.86)	EC10 (95% CI) estimate converted from egg to muscle Se based on reanalysis of Holm *et al.*(2005);	U	USEPA (2004) Presser and Luoma (2006) Ohlendorf *et al.* (2008, 2011)
	4.3	Muscle translation of egg toxicity threshold from Holm *et al.* (2005)	U	
Oncorhynchus clarkii lewisi (westslope cutthroat trout)	> 11.57[2]	NOEC for larval mortality & deformity;	U	Kennedy *et al.* 2000
	> 11.09	NOAC for larval deformity;	U	Rudolph *et al.* 2008
	13.01 (9.1 – 18.6)	EC10 (95% CI) for alevin mortality (reanalysis of Rudolph *et al* 2008);	U	USEPA 2011a
	9.57	EC10 estimate for alevin mortality (reanalysis of Rudolph *et al.* 2008);	U	DeForest and Adams (2011)
	10.55[2] (4.3 – 12.3)	Muscle EC10 (95% CI) larval survival, random effects model conversion;	U	Elphick *et al.* (2009)
	13.34[2] (7.1 – 16.0)	Muscle EC10 (95% CI) revised based on egg Se analysis from different lab , random effects model conversion.	U	Nautilus Environmental and Interior Reforestation Co. Ltd. (2011)
Salmo trutta (brown trout)	4.32[2] (3.99 – 4.68)	EC10 (95% CI) for alevin survival (15 d post swim-up);	U	NewFields (2009)
Oncorhynchus clarkii bouvieri (Yellowstone cutthroat trout)	> 11.37	NOEC for larval mortality and deformity, no LOEC could be estimated (reproductive)	U	Hardy *et al.* (2010)
Esox lucius (northern pike)	11.51[2] (5.9 – 17.3)	Muscle EC10 (95% CI) for larval deformity based on reported egg Se	U	Muscatello *et al.* (2006)
Lepomis macrochirus (bluegill sunfish)	< 11.2	LOEC for reduced hatching & larval survival & increased larval deformity;	1°	Hermanutz *et al.* (1992)
	4	LOEC for larval abnormalities;	1°	Hermanutz *et al.* (1996)
	20	LOEC for increased mortality in adult fish	U	Finley (1985)

[1]Studies classified for guideline derivation as primary (1°), secondary (2°) or unacceptable (U).

[2]See Appendix A (Schwarz (2011) for tissue conversion using the random effects log-log regression model for egg to muscle Se translation.

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Lotic reference site data can also present challenges when comparing tissue concentrations to guidelines. Due to the broad home range of species like westslope cutthroat trout, lotic reference sites in the Elk Valley and elsewhere may have resident fish which have foraged in Se-contaminated areas, confounding the conclusions regarding background tissue Se concentrations (Minnow *et al*. 2007; DeForest 2009; Minnow *et al*. 2011). Therefore, caution must be exercised if reference area tissue concentrations are unexpectedly high relative to other reference values or in excess of the guideline.

Holm *et al*. (2005) found that mean muscle Se in rainbow trout from reference sites at Deerlick Creek in Alberta, was 2.27 µg/g, assuming 78 % moisture content. In other studies conducted between 1999 and 2001 in southern Alberta, rainbow trout at a lentic reference site (Fairfax Lake) was 0.61 µg/g, assuming 75% moisture (Mackay 2006). In that same study, lotic reference muscle tissue concentrations for rainbow trout were 2.8, 3.7, 3.8 and 3.5 µg/g Se for Wampus, Whitehorse, MacKenzie, and Muskeg watersheds, respectively (Mackay 2006). These background muscle Se concentrations at sites within the coal geology, are all below 4 µg/g.

Based on the low effect concentrations for rainbow trout, brown trout and bluegill sunfish, 4 µg/g in fish muscle tissue is the recommended guideline for sensitive species (e.g., rainbow trout, westslope cutthroat trout), or fish species for which there are no toxicity data. This is an *interim* guideline since there remains some uncertainty in the estimates and little primary toxicity data directly linking effects to muscle tissue concentrations. Since we assume that whole-body and muscle Se concentrations in fish are approximately the same, and an uncertainty factor was previously applied to whole-body guidelines, an additional uncertainty factor was not applied to the interim muscle tissue guideline. In regions where natural background fish muscle tissue Se exceeds the guideline consideration of unexpected Se sources may be warranted to evaluate the need to develop site-specific water quality objectives.

The interim muscle tissue Se guideline for the protection of fish is 4 µg/g, calculated as the mean of at least eight tissue samples from individual fish collected at a representative area, and reported as dry weight.

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8.5 Guidelines for the Protection of Wildlife

The previous wildlife guideline developed for BC used birds as the surrogate to represent all sensitive wildlife (amphibians, reptiles), excluding fish and aquatic life (Nagpal and Howell 2001). The 2001 guidelines included a water-based maximum concentration of 4 µg/L, as well as an alert concentration for Se in bird eggs of 7 µg/g (Nagpal and Howell 2001). Since dietary accumulation at the base of the food web is the critical link to body burden in higher trophic levels, the aquatic life guideline (2 µg/L) for the water column has also been adopted for the protection of wildlife.

The previous bird egg tissue guideline of 7 µg/g was reviewed in light of more recent toxicity studies. Unfortunately, toxicity data on amphibians and reptiles is still limited (Section 7.4.3.7). There are studies that suggest effects may be occurring, but the results fall short of defining a Se concentration-response relationship that would allow for comparison with fish or birds (Janz *et al.* 2010; Hopkins *et al.* 2004; Minnow 2006). For example, Hopkins *et al.* (2004) conducted a laboratory study on brown house snakes (*Lamphrophis fuliginosus*), and were able to conclude that snakes readily transferred dietary Se to kidney, liver, ovary, and egg tissues, but no significant differences were found in survival, food consumption, growth, body condition, or reproductive activity in female snakes. They did find that female snakes fed 20 µg/g of dietary Se on average were less likely to reproduce, had fewer eggs, and lower total egg mass than control snakes, but the differences were not significant due to high variability in the reproductive output among all females (Hopkins *et al.* 2004). The mean (± 1 SE) Se concentration in snake eggs associated with the highest dietary treatment was 22.65 (± 0.49), exceeding embryotoxicity thresholds for birds and suggesting that birds are comparatively more sensitive (Hopkins *et al.* 2004). Similarly, Minnow (2006) conducted a study on Columbia spotted frog from the Elk Valley, BC, but failed to conclusively link effects with Se exposure from coal mining activities.

There are also toxicological studies on aquatic-dependent mammals or other small mammal species exposed to Se contamination, yet concentration-response relationships with Se have not been established for mammalian wildlife. However, the studies to date suggest that aquatic-dependant mammals may be less sensitive to Se than are fish or birds (Janz *et al.* 2010). Ohlendorf *et al.* (1989) found that of the wildlife species evaluated at Kesterson Reservoir,

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aquatic birds had the most frequent and extreme signs of Se toxicity, while small mammals showed almost none. One explanation may be due to the much wider margin between essential and toxic doses of Se in mammals compared with fish and birds (Janz *et al.* 2010). Since birds are known to be more sensitive to chronic Se effects, the updated wildlife guideline was developed using bird data as a surrogate for all wildlife species.

The CCME protocol for deriving tissue residue guidelines for wildlife calls for the calculation of tolerable daily intake to be applied to the highest known trophic level at which the most sensitive species of aquatic-dependent wildlife feeds (CCME 1998). The consideration of diet specifically for birds was incorporated into the interim dietary tissue guideline recommended for BC of 4 µg/g Se (see Section 8.4.3.1). However, for a more direct estimate of the potential for Se toxicity, most researchers recommend the use of bird egg analysis (Skorupa 1998; Adams *et al.* 1998; Ohlendorf and Heinz 2011; Table 8.16). Therefore, rather than establish a guideline for total daily intake of Se in sensitive birds as per the CCME (1998) protocol, mean egg Se concentration for birds was selected for guideline development. The Science Panel developing a site-specific Se standard for the Great Salt Lake agreed that diet and egg Se concentrations in birds would best serve as the basis for a water quality standard (Ohlendorf *et al.* 2009). This and the lack of toxicity data for other wildlife (amphibians, reptiles, and mammals), supports the bird egg guideline approach for wildlife that BC has chosen.

Some investigators have suggested that the intra-specific variability in bird egg Se concentrations is low (Heinz *et al.* 1987) while others have shown that in some species, the maternal transfer of Se to eggs may be highly variable, even within the same clutch (Bryan *et al.* 2003). Studies on common grackles (*Quiscalus quiscala*), where whole clutches of eggs were collected in nests from reference areas and at coal ash settling basins, found high inter- and intra-clutch variation in mean egg Se concentrations (Bryan *et al.* 2003). However, the variation in Se concentrations in clutches from reference areas were much lower (coefficient of variation 7.9 – 18.9 %) than in exposed areas (coefficient of variation 10.8 – 34.8 %), which was likely a reflection of the variation in dietary Se concentrations (Bryan *et al.* 2003).

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Weech *et al.* (2011) conducted studies on birds nesting near a uranium mill in northern Saskatchewan and also found that intra-clutch egg Se was highly variable in mallard ducks (*Anas platyrhychos*) and tree swallows (*Tachycineta bicolour*). These studies suggest that a single random egg sampled from a nest in higher Se-exposed areas may not be truly representative of Se in all eggs from the same clutch. While these studies may add some level of uncertainty to the estimates of toxic thresholds, most bird surveys adopt a design in which one random egg per nest is collected thereby reducing to some degree any bias in the Se estimates (Skorupa 1998; Seiler *et al.* 2003). With adequate sample sizes, a strictly random selection of a single egg from any given clutch, along with an appropriate study design and statistical analysis, can address potential biases (Dr. C. Schwarz, pers. comm., Simon Fraser University, Sept 2011). All things considered, bird egg Se is still the most direct measure of embryotoxicity in birds (Ohlendorf 2003). Liver Se concentrations in birds may also provide a reasonably good estimate of Se exposure (Ohlendorf and Heinz 2011; Table 7.11).

Since the Se poisoning of birds that occurred at Kesterson National Wildlife Refuge in California, a great deal of knowledge has been gained about the effects of Se on birds (Ohlendorf and Heinz 2011). There is a similar range of variability in the sensitivity of bird species to Se, as has been shown in fish (Ohlendorf *et al.* 1986). Domestic poultry are thought to be among the most sensitive birds (Puls 1994), but much information exists on wild species, on which many toxicity threshold estimates have been based (Table 7.11, Table 8.16).

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Table 8.16 Toxicity thresholds for various bird species based on mean egg selenium concentrations (µg/g dw).

Bird Species	Mean Egg Se Effect Threshold (µg/g dw)	Effect	Study Classification[1]	Reference
Anas platyrhychos (mallard duck)	≥ 16.67	Increased likelihood of reproductive impairment (70% moisture content);	1°	Heinz *et al.* (1989)
	12 - 16	EC10 for duckling mortality based on 6 studies, various statistical approaches;	1°	Adams *et al.* (2003)
	12 (6.4 - 16)	EC10 (95% CI) for egg hatchability based on 6 studies, logistic regression;	1°	Ohlendorf (2003)
	11.5 (9.7 – 13.6)	EC10 (95% CI) for duckling mortality;	1°	Ohlendorf (2007)
	23	EC10 for teratogenic effects ;	-	USDOI (1998)
	7.7	EC10 egg hatchability reanalysis of one study biphasic model regression;	1°	Beckon *et al.* (2008)
Actitis macularia (spotted sandpiper)	7.3 (± SE 0.43)	Significant (15%) reduction in hatchability & significantly higher (approximately double) MES at exposed sites compared to reference.	2°	Harding *et al.* (2005)
Cinclus mexicanus (American dipper)	8.4 (± SE 0.44)	15% depression in egg viability at the exposed site, although there was no significant difference in MES or hatchability due to low sample sizes	2°	Harding *et al.* (2005)
Himantopus mexicanus (black-necked stilt)	14	EC11.8 for reduced hatchability based on meta-analysis of data;	2°	Ohlendorf *et al.* (2011)
	6-7	EC03 egg viability, corrected for background;	2°	Skorupa (1999); USDOI (1998)
Agelaius phoneniceus (red-winged blackbird)	22	Approximate effects threshold for hatchability based on mean egg Se at exposed sites.	2°	Harding (2008)
Multiple bird species (data synthesis)	12	Threshold for reproductive effects based on field and lab studies, not necessarily a safe concentration;	2°	Heinz (1996)
	6	Bird egg Se guideline for evaluating toxic response in NIWQP	2°	Seiler *et al.* (2003)

[1]Studies classified for guideline derivation as primary (1°), secondary (2°) or unacceptable (U).

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Ohlendorf *et al.* (1986) plotted the frequency of embryonic mortality and deformity in chicks of several species of aquatic birds nesting in the Kesterson Wildlife Refuge, which were grouped into coots, ducks, stilts and grebes. Although there was no statistical analysis, it was apparent that coots and grebes had the highest percentages of mortality and deformity so were thought to be highly sensitive to the effects of Se contamination, while ducks and stilts were considered to be less sensitive (Ohlendorf *et al.* 1986). Studies conducted in the Elk Valley BC, on American dippers and spotted sandpipers suggest that sandpipers are more sensitive than dippers to the chronic effects of Se (Harding *et al.* 2005). Red-winged blackbirds may be slightly more Se tolerant than both dippers and sandpipers (Harding 2008).

There has been much debate over the last decade concerning an appropriate avian egg Se toxicity threshold (Skorupa 1998; Fairbrother *et al.* 1999, 2000; Skorupa 1999; Adams *et al.* 2003; Ohlendorf 2003, 2007; Presser and Luoma 2006; Beckon *et al.* 2007; Ohlendorf and Heinz 2011). A general agreement among researchers is that hatchability is a more sensitive endpoint for Se toxicity than is deformity – reductions in hatchability will be evident in birds at lower egg Se concentrations than would be the case for deformities (Skorupa 1999; Ohlendorf 2003; Janz *et al.* 2010). For some species, such as American kestrel, fertility may be a more sensitive endpoint than hatchability (Santolo *et al.* 1999). Skorupa (1998) suggested a bird egg toxicity threshold between 6 and 7 µg/g Se based on data for black-necked stilt.

Toxicological studies on mallard ducks, thought to be one of the more sensitive bird species, have provided a good starting point for development of a guideline for wildlife based on bird toxicity (Fairbrother *et al.* 1999; Skorupa 1999; Fairbrother *et al.* 2000; Ohlendorf 2003; Ohlendorf 2007; Ohlendorf and Heinz 2011; Ohlendorf *et al.* 2011;Table 8.16). Mallard duck studies were assessed using CCME's guidance on evaluation of toxicological data and were deemed acceptable for derivation of a BC guideline (CCME 1998).

Fairbrother *et al.* (1999) re-analysed data from two mallard studies (Heinz *et al.* 1989 and Stanley *et al.* 1996), estimating an EC10 for duckling mortality of 16 µg/g egg Se – a much higher estimate than the 6 – 7 µg/g Se previously proposed by Skorupa (1998). This EC10 estimate was criticized for not including important data, using incompatible response endpoints,

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and not evaluating the more sensitive endpoint of egg viability (Skorupa 1999). Fairbrother *et al.* (2000) responded to this criticism. Subsequently, Ohlendorf (2003) calculated an EC10 of 12.5 µg/g for egg hatchability based on data generated from six lab studies on mallard ducks.

Seiler *et al.* (2003) stated that an EC10 of 12.5 µg/g egg selenium (with 95% confidence boundaries of 6.4 to 16.5 µg/g) may be appropriate as a high-risk Se exposure level. A more conservative 6 µg/g egg Se, which was the approximate lower confidence limit, was used in their assessment as the toxicity benchmark for evaluating Se concentrations in eggs (Seiler *et al.* 2003). Their rationale was related to the applicable federal wildlife laws (such as the US *Migratory Bird Treaty Act* and *Endangered Species Act*) which do not allow *any* foreseeable, human-caused mortality of protected populations (Seiler *et al.* 2003).

As part of the development of a Se water quality standard for the Great Salt Lake, the mallard egg Se threshold was re-examined by a panel of experts (Ohlendorf *et al.* 2007). The Utah Water Quality Board, and Utah Department of Environmental Quality (DEQ), finally adopted the site-specific water quality criterion of 12.5 µg/g egg Se for the Great Salt Lake, which was approved by the US EPA (USEPA 2011a). However, to be more protective, the Utah Water Quality Board and Utah DEQ, incorporated a series of bird egg Se thresholds in Footnote (14), which used lower egg Se thresholds as triggers for management action (USEPA 2011a). The trigger values in Footnote (14) commence at egg Se concentrations of 5.0 µg/g, with increasing regulatory action at 6.4 and 9.8 µg/g trigger values (USEPA 2011a, see Table 8.5 for summary of trigger points/ actions).

Ohlendorf and Skorupa (1991) reviewed available avian egg Se data and determined a background concentration of about 3 µg/g. Bird egg Se is typically close to 3 µg/g concentrations in reference areas; where local geology is high in Se, the concentrations are usually 6 µg/g or less (Table 4.13). Caution should be exercised when verifying reference areas and interpreting data that appear to be anomalously high, since birds or their prey may forage in Se-exposed areas, elevating their dietary intake and resulting egg tissue residues (Ohlendorf *et al.* 2011).

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Comparing several bird studies conducted in the Elk Valley, BC, one species stands out. Harding *et al.* (2005) suggested that spotted sandpipers may be a particularly sensitive species to Se toxicity, based on their study which showed a significant 14% reduction in hatchability (compared to referenence) at a mean egg Se concentration of 7.3 µg/g. The number of failed eggs at the exposed sites were three time higher than at reference site. However, the number of eggs per clutch, showed no significant reduction at exposed sites, likely because the sample size was too small (Harding *et al.* 2005).

Beckon *et al.* (2008) re-analysed mallard data from Heinz *et al.* (1989), comparing a standard monotonic log-logistic regression model of the EC10 for mallard hatchability with two alternative biphasic regression models describing a hormetic dose-response effect. A wide range of EC10 estimates were generated; the standard log-logistic estimate was 28.6 µg/g, and the two biphasic log-logistic models were 7.3 and 3.4 µg/g. Beckon *et al.* (2008) cautioned that a biphasic dose-response model is not always the best representation of the potential effect but that this comparison demonstrated that for some effects, such as hatchability, a biphasic model may describe the response variable more accurately and yield a more protective EC10. The authors suggested the EC10 of 7.3 µg/g, generated by one of the two biphasic models, provided a more moderate estimate and best described the data. While hormetic dose-response relationships have been identified (Harding 2008; Beckon *et al.* 2008), more research is needed in the application of bi-phasic toxicological models in aquatic ecology.

The EC10 egg Se estimate for hatchability in mallard of 12.5 µg/g, could be used as the critical value for a wildlife guideline. However, several field studies suggest that at least three species of birds are more sensitive than mallard; coots, grebes and spotted sandpiper (Ohlendorf *et al.* 1989; Harding *et al.* 2005). Since toxicity data exist for only a limited number of bird species (and other wildlife species), the minimum uncertainty factor of 2 was selected and applied to the critical egg Se toxicity value for mallards of approximately 12 µg/g. This results in a guideline value of 6 µg/g bird egg Se, a concentration which is sufficiently above typical background levels in bird eggs. Comparing this value with the Footnote 14 provisions established as part of the Great Salt Lake site-specific bird egg criteria and other guideline recommendations, a guideline of 6 µg/g is adequately protective of sensitive bird species. Studies of other wildlife species would be beneficial in establishing additional tissue guidelines for sensitive species.

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Some authors (Skorupa and Ohlendorf 1991; Skorupa 1999) mention the secondary dietary hazard posed to predators feeding on bird eggs that may be in excess of dietary thresholds. It is important that a protective wildlife guideline value consider these potential secondary hazards to predators and considered in setting a wildlife guideline (e.g., other birds, some reptiles and larger mammals like marten, coyote, fox, and bear). A guideline of 6 µg/g for wildlife is slightly higher than the 4 µg/g dietary guideline recommended in this document. Since there is great uncertainty about the risk posed to predators from consuming bird eggs, and there are too few studies to determine a wildlife consumer guideline, none is proposed at this time.

The water column guideline of 2 µg/L, and the dietary guideline of 4 µg/g in food items, are applicable to wildlife species. The chronic tissue guideline for the protection of wildlife, using birds as a surrogate, is 6 µg/g (dw) in bird egg tissue, calculated as the mean concentration of at least 8 eggs (from 8 individual nests) in a representative area, reported as dry weight.

8.6 Recreational Use and Aesthetics

No information was found regarding recreational or aesthetic guidelines specifically for selenium. No evidence was found linking waterborne Se to risks associated with recreational or aesthetic uses of water. Therefore no guidelines for these water uses are proposed.

A water quality guideline for Se in recreational waters or for aesthetics is not proposed at this time due to the lack of available information.

8.7 Irrigation and Livestock Watering

The existing guidelines for agricultural water uses, specifically irrigation and livestock watering, have not been updated at this time. Details on their derivation and rationale are provided in Nagpal and Howell (2001).

The approved BC water quality guideline for irrigation water is 10 µg/L, and for livestock watering the guideline is 30 µg/L.

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8.8 Industrial Uses

No data could be found regarding selenium guidelines for industrial water uses. Therefore, no guidelines are proposed here.

A water quality guideline for Se in industrial use waters is not proposed at this time due to the lack of available information.

9.0 Monitoring and Analytical Considerations for Selenium Analysis

A group of studies were conducted in the Elk Valley, BC, all of which attempted to define a toxicity threshold for westslope cutthroat trout (Kennedy *et al.* 2000; Rudolph *et al.* 2008; Elphick *et al.* 2009; Nautilus Environmental and Interior Reforestation Co., Inc. 2011, see Section 8.4). These studies demonstrated that field-based study outcomes on the same population of fish often differ, resulting in very different toxicity threshold estimates. Additionally, laboratory results from the same study may also differ (Elphick *et al.* 2009, Nautilus Environmental and Interior Reforestation Co., Inc. 2011), adding to the uncertainty in these estimates. Other examples of toxicity threshold estimates for hatchability in mallard ducks have resulted in a range of estimates which differed based on different statistical techniques applied to the same set of data (see Section 8.5). These scientific and analytical uncertainties form the basis for distinguishing between toxicity threshold estimates, which represent concentrations at which adverse effects are apparent, and safe concentrations (Hamilton 2003, 2004).

These uncertainties can be minimized by careful design and execution of a monitoring and assessment program. A good summary of the potential monitoring pitfalls along with recommendations for conducting sound monitoring and assessment programs for Se is contained in two documents prepared for the North American Metals Council (NAMC); Ohlendorf *et al.* (2008) and a subsequent publication, Ohlendorf *et al.* (2011). Ralston *et al.* (2008) prepared a companion NAMC document on the biogeochemistry of Se, which includes advice on analytical techniques for Se and its chemical species.

Establishing data quality objectives, along with a conceptual plan, is recommended at the outset of any monitoring and assessment program for Se (Ohlendorf *et al.* 2008, 2011). During the

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collection of data, careful site selection should ensure that samples are representative of the area being sampled (control or background versus exposed sites). Sample handling, preservation or other preparation, and shipping should be conducted according to standardised procedures appropriate for each media (water, sediment, or biological). The appropriate number of sample blanks, spiked samples, certified reference materials, and duplicates should be incorporated into the sampling and monitoring program design. Ralston *et al.* (2008), and documents prepared by the BC Ministry of Environment (Cavanagh *et al.* 1998; Ministry of Water, Land and Air Protection 2003) provide more detail on proper procedures for sampling and monitoring programs.

The variability in fish tissue monitoring data at sites where there is no apparent disturbance or source of Se contamination may be explained by unanticipated Se sources, complex bioaccumulation dynamics that enhance Se uptake, and/or species-specific enhanced Se uptake. In locations where unexpectedly high Se concentrations result in one or more environmental compartments, or for species that accumulate high levels of Se in undisturbed reference areas, closer examination of the data and the site conditions are recommended. Laboratory quality assurance should be checked carefully, as well as the numbers and representativeness of samples. Highly mobile species may be moving in and out of Se-contaminated areas resulting in variable exposure and higher than expected tissue Se. Some fish species, such as sculpin, could have habitat preferences or physiologies that put them at greater risk of accumulating Se. Some locations may be more prone to Se bioaccumulation as a result of the natural geology of the area. Any one, or a combination of these factors may result in Se concentrations elevated above guidelines, in which case site-specific water quality objectives may be warranted. Contact the BC Ministry of Environment for assistance in determining if and how water quality objectives should be developed (see guidance document at: http://www.env.gov.bc.ca/wat/wq/pdf/wqo_2013.pdf).

A critical monitoring consideration when developing a comprehensive environmental risk assessment is incorporation of a thorough inventory and assessment of organisms potentially at risk in the area of concern. This should include the lowest trophic levels to the highest. Knowing what organisms may be at risk, and where, helps define the study area and identify key indicator species.

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Presser and Luoma (2006) recommended that a full characterisation of Se in the critical environmental compartments will greatly enhance the evaluation, interpretation and management of Se in aquatic ecosystems. Co-located sampling of various compartments in the environment (different media) in both exposed and reference areas, and/or across a gradient of Se concentrations provides optimal information (Ohlendorf *et al.* 2008). This will assist in long-term studies where the objective is to compare results over time to determine trends. This will also facilitate development of important site-specific and species-specific relationships within and between the various environmental compartments and tissue types.

The number and types of samples should consider other possible contaminants, the type of environment being sampled, natural variability (adequate numbers of samples), interactions between media, and target organisms (i.e., dietary organisms and key surrogate species at the top of the food web) (Ohlendorf *et al.* 2008). Assessments should target the correct season to coincide with spawning of important target species, nesting of birds, and/or worst case Se concentrations. As well, establishing relationships between Se concentrations in multiple tissue types within an individual fish species (e.g., egg/ripe ovary with whole-body or muscle tissues) can improve the understanding of Se toxicokinetics and also provide some flexibility in monitoring programs (use of non-destructive sampling techniques) once relationships between tissue types is established. For example, muscle plug tissue samples in westslope cutthroat trout in the Elk River in BC, were found to be highly correlated to muscle fillet tissues, with a correlation coefficient (r^2) of 0.996, making muscle plug samples a reliable non-destructive sampling alternative (Minnow 2004).

Sediment concentrations of Se, by nature, may be highly variable and may not provide a link to Se levels in other environmental compartments, such as tissues (Hamilton and Lemly 1999; Malloy *et al.* 1999). Sampling methods and study designs may help control the high degree of spatial and temporal variability in sediment Se concentrations (Malloy *et al.* 1999). Selenium tends to adsorb to fine-textured, organic-rich sediments, making these characteristics important to define when analysing sediment samples (Besser *et al.* 1989; Wiramanaden *et al.* 2010). Restricting stream sample locations to depositional zones and using only the fine grain size fraction of sediment for metals analysis may reduce this variability (Rex and Carmichael 2002).

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The BC MoE has recently released a guidance document that recommends sediments less than 63 μm be evaluated for metals analysis, including Se, to reduce the variability in reported metals concentrations (BC MoE 2012b). It is important when collecting sediments, to analyze key variables such as particle size distribution and total organic carbon (TOC) content. Sediment samples should be composites of at least five individual samples per location so variability between sites and changes over time can be evaluated. It is also recommended that collection and analytical methods remain constant, or careful evaluation of changes are incorporated in a monitoring program to ensure comparability of data over the long-term.

D'Ulivo (1997) stated that the most common problems associated with accurate analysis of Se involve sample preparation, storage and manipulation procedures to liberate Se compounds (mainly the protein-bound fractions) from the sample matrix. This is especially important for speciation of Se in environmental samples. Another common problem is adequately sensitive instrumentation and equipment for low and accurate detection of Se (D'Ulivo 1997).

Typically, accredited labs will use one of the US EPA methods for analyzing Se in environmental media (Ralston *et al.* 2008). For example, EPA method 200.8 is used for water or waste water. EPA methods 3050B and/or 3052 are often used for sediment, sludge, and soil samples, which involve sample digestion with both nitric acid and hydrogen peroxide (USEPA 1994; USEPA 1996a,b). The later method is typically the most commonly used for these media, as well as for biological tissue, since the hydrogen peroxide digestion step is most efficient at breaking down organically-bound Se (F. Chen, pers. comm., Maxxam Analytics, July 2011).

The use of inductively coupled plasma mass spectrometry (ICP-MS) has become the instrument of choice for determining Se in all matrices (water, soils, tissues) due to its sensitivity and precision (D'Ulivo 1997; Ralston *et al.* 2008). It does, however, have the disadvantage of being prone to interferences. For example, a seawater matrix presents difficulties unless the sample is first diluted prior to analysis (M. Melnychuk, pers. comm., Maxxam Analytics, July 2010). Over the past decade, collision/reaction cell (CRC) technology has been coupled with ICP-MS to reduce, if not eliminate, interference from polyatomic ions and further improve the performance of the ICP-MS instrument (Ralston *et al.* 2008).

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Other instruments and methods used to analyse Se are reviewed in D'Ulivo (1997), Ohlendorf *et al*. (2008) and Ralston *et al*. (2008). Use of equipment and techniques which provide minimum detection limits 10 times lower than the range of interest (water quality guideline, objective or management goal) are best for meaningful results, particularly if Se speciation is being conducted (Ralston *et al*. 2008). Where possible, analysis of Se species has been advised to gain additional information about the site-specific dynamics and transformation of Se for all environmental compartments (Ohlendorf *et al*. 2008).

Further guidance and information specific to mining-related monitoring and assessment can be found in *Water and Air Baseline Monitoring Guidance Document for Mine Proponents and Operators* (BC MoE 2012b).

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Appendix A - Fish Tissue Regression Model

Estimating tissue transfer functions for Se in fish

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This report, the associated tables and figures, and the Excel spreadsheets for the simple (separate line for each species) and the random effects tissue transfer models may be accessed on-line at BC MoE's website for water quality guidelines under selenium:
http://www.env.gov.bc.ca/wat/wq/wq_guidelines.html

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Appendix B - Summary of Consumption Guidelines for Utah, Michigan and West Virginia

Table B.1 Safe eating guideline for fish, State of Utah (Utah Department of Health, Division of Wildlife Resources, and the Utah Department of Environmental Quality, 2011).

Waterbody	**County**	**Contaminant**	**Species**	**Pregnant Women & Children** *4 oz. meals/month	**Adults** 8 oz. meals/month
Lower Ashley Creek drainage & Stewart Lake	Uintah	Selenium	Fish/Ducks	Avoid Consumption	No more than 1 6 oz. serving/month

Table B.2 Fish consumption advisory for selenium, Mount Storm and Upper Mud Lakes; Pinnacle Creek, West Virginia (West Virginia Department of Health and Human Resources, 2011).

When the concentration of selenium reaches these levels in fish tissue, the recommended fish consumption advice should restrict intake amounts to:			
Minimum (mg/kg or ppm)	**Maximum (mg/kg or ppm)**	**Group**	**Meal Restriction**
	<2.50	1	Up to 225 meals per year (no restrictions)
2.50	10.83	2	Up to 1 meal per week
>10.83	23.47	3	Up to 2 meals per month
>23.47	46.93	4	Up to 1 meal per month
>46.93	93.86	5	Up to 6 meals per year
>93.86		6	DO NOT EAT

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Table B.3 Interim consumption screening values for total selenium, Goose Lake, Michigan (Michigan Department of Community Health 2011).

Meal Category	**Total meals per year**	**Selenium Fish Consumption Screening Values µg/g wet weight**
No Restrictions	Not applicable	less than or equal to (≤) 2.5
One meal per week	52	great than (>) 2.5 to ≤ 7.4
One meal per month	12	> 7.4 to ≤ 32
Six meals per year	6	> 32 to ≤ 64
Do Not Eat	0	> 64

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