Environment and Climate Change Canada | Environnement et Changement climatique Canada

# *Canadian Environmental Protection Act, 1999*

# Federal Environmental Quality Guidelines

# *Selenium*

**Environment and Climate Change Canada**

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## Introduction

Federal Environmental Quality Guidelines (FEQGs) describe acceptable quality of the ambient environment. They are based solely on the toxicological effects or hazards of specific substances or groups of substances. FEQGs serve three functions: first they can be an aid to prevent pollution by providing targets for acceptable environmental quality; second, they can assist in evaluating the significance of concentrations of chemical substances currently found in the environment (monitoring of water, sediment, soil and biological tissue); and third, they can serve as performance measures of the success of risk management activities. The use of FEQGs is voluntary unless prescribed in permits or other regulatory tools. Thus FEQGs, which apply to the ambient environment, are not effluent limits or "never-to-be-exceeded" values but may be used to derive effluent limits. The development of FEQGs is the responsibility of the Federal Minister of Environment under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Government of Canada (GC) 1999). The intent is to develop FEQGs as an adjunct to risk assessment or risk management of priority chemicals identified in the Chemicals Management Plan (CMP) or other federal initiatives.

Where data permit, FEQGs are derived following CCME protocols. FEQGs are developed where there is a federal need for a guideline but where the Canadian Council of Ministers of Environment Canadian Council of Ministers of Environment (CCME) guidelines for the substance have not yet been developed or are not reasonably expected to be updated in the near future. For more information, please visit the Federal Environmental Quality Guidelines (FEQGs) page.

This factsheet describes the federal tissue quality guidelines for the protection of fish and birds for selenium (Table 1). FEQGs are not developed for the water, sediment or soil compartments; however, recent water-based guidelines developed in North America are summarized.

Table 1. Federal environmental quality guidelines for selenium

| Fish egg-ovary tissue (µg/g dry weight) | Fish (whole body) tissue (µg/g dry weight) | Bird egg (µg/g dry weight) |
|---|---|---|
| 14.7 | 6.7 | 11 |

## Substance Identity

Selenium (Se) is a naturally occurring element (CAS Number 7782-49-2), found in minerals such as pyrite, chalcopyrite, pyrrothite and sphalerite and in crude oil and coal deposits (ECCC, HC 2017). Anthropogenic sources of selenium include selenium production and processing facilities, use of selenium-containing products, and disposal and management of selenium-containing waste. Selenium occurs in various oxidation states and common species are selenate ($SeO_4^{-2}$), selenite ($SeO_3^{-2}$), elemental selenium ($Se^0$) and organic and inorganic selenides ($Se^{-2}$). Selenium is nutritionally essential for organisms; however, introduction of selenium into the environment from both natural and anthropogenic sources can lead to elevated concentrations in surface water, groundwater, soils, and vegetation (BCMOE 2014; USEPA 2016). Among all trace nutrients, the difference between essentiality and toxicity is narrowest for selenium and thus the risk of adverse impact from environmental contamination is extremely high (Luoma and Rainbow 2008).

Open-pit coal mining in British Columbia and Alberta has resulted in the mobilization of selenium from waste rock leachate with high concentrations of selenium to surface and groundwater, potentially threatening fish and bird populations (BCMOE 2014). Uranium mining in Saskatchewan has been associated with increased selenium concentrations in receiving waters and deformities observed in fish (Muscatello et al. 2006; Muscatello and Janz 2009). In Ontario and Manitoba, selenium releases have been reported from metal smelter emissions and effluents (Nriagu and Wong 1983; Manitoba Conservation 2007).

Based on the screening assessment of selenium and its compounds (ECCC, HC 2017), the Government of Canada concluded that selenium and its compounds meet the criteria under paragraph 64(a) of CEPA, as they

are entering or may enter the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity. However, it was concluded that selenium and its compounds do not meet the criteria under paragraph 64(b) of CEPA, as they are not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger to the environment on which life depends. Selenium and its compounds were also determined to meet the persistence and bioaccumulation criteria as set out in the *Persistence and Bioaccumulation Regulations* of CEPA (GC1999).

## Uses

Canada is among the top five producers of selenium, along with the US, Japan, Belgium and Chile (BCMOE 2014). In Canada, selenium is recovered as a by-product from copper or zinc refining processes (ECCC, HC 2017). Between 2005 and 2012, Canadian production of selenium ranged between 97,000 kg and 288,000 kg (NRCan 2014). Selenium and its compounds can be used as a component of pigments (in plastics, paints, ceramics and glass), in rubber (accelerator in rubber vulcanization), agriculture (soil supplement, animal feed and pesticides), lubricants and metallurgical applications, electronic equipment, drugs including natural health products (as a medicinal ingredient in multi-vitamin/mineral supplements and anti-dandruff shampoos), supplemented foods, specific foods for special dietary uses, cosmetics and consumer products (ECCC, HC 2017). Details of these uses and sectors where a potential risk to the environment was identified are presented in ECCC, HC (2017).

## Fate, Behaviour and Partitioning in the Environment

Selenium can enter the aquatic ecosystem in any form, but the soluble forms of selenium that reside in the aquatic ecosystems generally consist of selenate ($SeO_4^{2-}$) and selenite ($SeO_3^{2-}$) (Maher et al. 2010). Selenite is typically present in larger proportions in the effluents of coal power plants and petroleum refineries, whereas selenate is most likely found in mine effluents and agricultural run-offs (Maher et al. 2010; Young et al. 2010). Many selenium compounds can react with water and enter the environment in a dissolved phase. Within the environmentally relevant pH range of 6 to 8, only selenate, elemental selenium, selenite and bi-selenite are present in water (ECCC, HC 2017). Over this pH range, the predominant dissolved forms are selenate and selenite in oxygenated freshwaters (Brookins 1988; Belzile et al. 2000; Ralston et al. 2009). At pH values below 7, in mildly reducing conditions, selenite species are reduced to elemental selenium (ATSDR 2003).

Because selenium bioavailability varies with the selenium species present, the form of selenium that aquatic organisms are exposed to is critically important. For example, increase of sulphate concentration reduces selenate toxicity to aquatic invertebrates and fish, but has no effect on selenite toxicity (Carlton 1998; USEPA 2016). Dietary selenium exposure has been widely examined and has been considered a critical exposure pathway for assessing selenium toxicity at environmentally relevant concentrations.

Partitioning of selenium between water and sediment is system specific and is controlled by several processes (Chapman et al. 2010). Selenium adsorbs to the surface of iron-or manganese-rich sediment (ECCC, HC 2017) and the precipitation of selenium upon contact with ferric compounds is pH dependent (Maher et al. 2010). Finally, certain bacteria in the sediment use selenate or selenite as terminal receptor of electrons in respiration (Oremland et al. 1989) and thus they are a very important part of selenium cycling in environment (Nancharaiah and Lens 2015). Over time, organic selenium generally transforms into an inorganic species through photo-oxidation and mineralisation (Chen et al. 2005).

Behaviour of selenium in soil is also dependent on a large number of factors: redox conditions, pH, iron hydroxide content, clay content, organic materials and the presence of competing anions (CCME 2009). For example, selenides are found in more acidic soils containing high concentrations of organic matter. Elemental selenium can be formed in moist anoxic soils, while selenate is the predominant species in alkaline, oxygenated soils (ECCC, HC 2017). Selenite is soluble but less mobile than selenate due to greater adsorption

to soil minerals and organic material (ECCC, HC 2017). Due to selenate being more mobile, remobilisation of selenium may be caused by the dissolution of selenate in irrigation waters (Chapman et al. 2010).

The atmosphere is also an important environmental compartment for selenium although the biogeochemical cycling of selenium occurs mainly in water, sediment and soil (ECCC, HC 2017). Up to 30% of the selenium present in feed coal for combustion is emitted in a vapor phase (Chapman et al. 2010). The inorganic forms are mainly selenium dioxide (due to the burning of fossil fuels) and elemental selenium adsorbed to particulates (ECCC, HC 2017). Selenium dioxide, having a vapour pressure of 12.5 mm Hg at 70°C, is more volatile than elemental selenium. Elemental selenium itself has a low vapour pressure of 1 mm Hg, and thus is not usually found in the air as vapour (ASTDR 2003). Two other gaseous organic species can be produced by biotransformation; dimethyl selenide ($(CH_3)_2Se$) and dimethyl diselenide ($(CH_3)_2Se_2$) (Terry et al. 2000; Guo et al. 2001). Selenium compounds do not have a long residence time in the air (ECCC, HC 2017).

Bioaccumulation and trophic transfer through aquatic food webs are the major biogeochemical pathways of selenium in aquatic ecosystems (USEPA 2019). Key factors affecting selenium bioaccumulation are physical and chemical properties of the environment (e.g., pH, redox potential, temperature and hydrology), the chemical form of selenium, the ambient selenium concentration, the exposure route and duration, and the species exposed and their trophic level. Selenium can be actively taken up by the primary producers (e.g., algae, plants and microbes) and converted to organo-selenium compounds and thereby providing the base from which selenium enters the aquatic food web (ECCC, HC 2017). The absorption of selenium by organisms in aquatic systems is through direct contact with water is low compared to absorption through diet (Presser and Luoma 2010). Details on bioaccumulation and bioconcentration of selenium in aquatic organisms (algae, plants, invertebrates and fish) are presented in published assessments (BCMOE 2014, USEPA 2016, 2019; ECCC, HC 2017).

## Ambient Concentrations

Selenium concentrations in Canadian waters are similar to global concentration ranges of 0.01 to 4 µg/L (CCME 2009). Detailed monitoring data for selenium in Canadian surface waters, fish tissue and birds are presented in BCMOE (2014). The surface water monitoring data presented here are summarized from BCMOE (2014). Because these data came from multiple sources, method detection limits (MDL) varied considerably. Selenium concentrations in Newfoundland and Labrador were generally less than 1.0 µg/L. Elevated concentrations were reported at some urban sites or sites with saltwater intrusion. Environment and Climate Change Canada's monitoring data for Atlantic Provinces recorded selenium concentrations at or below the MDL of 0.01 µg/L. The reported mean and maximum concentrations for PEI were 0.08 and 0.16 µg/L, respectively. Monitoring data for 29 rivers in Nova Scotia (Cape Breton) and New Brunswick also reported concentrations below the MDLs of 1 and 1.2 µg/L respectively, with the exception of two samples. Data collected from a federal water quality monitoring station in Quebec showed selenium concentrations at or below the MDL of 0.05 µg/L. An average selenium concentration of 0.1 µg/L was reported within the freshwater fluvial reach of the St. Lawrence River, Quebec.

Selenium concentrations in the Great Lakes were generally below the MDL of 0.1 µg/L to 1.0 µg, with the exception of Lake Erie, where concentrations ranged between below detection and 36 µg/L, the higher values in Lake Erie were likely due to anthropogenic discharges. The data from 14 long-term monitoring stations of the Ontario Ministry of Environment showed that most sites have selenium concentrations below the MDL of 1.0 µg/L during the early years of monitoring and below the reduced MDL of 0.05 µg/L in later years. Exceptions to these low concentrations were sites associated with point source inputs from mining effluents or atmospheric emissions.

Selenium concentrations in surface waters of Manitoba were generally near or below the MDL of 0.4 µg/L since 2001 and the previous MDL of 2.0 µg/L prior to 2001. However, elevated concentrations were reported from areas influenced by mining and smelting activities. Typically, low selenium concentrations in water (less than 1 µg/L) were reported from Saskatchewan during the early years of monitoring, however, in some areas selenium concentrations may be elevated due to geologic formations or anthropogenic activities (e.g., uranium mining operations in northern Saskatchewan). In major rivers in Alberta, the average selenium

concentrations ranged between 0.3 and 0.7 µg/L (MDL = 0.1 µg/L). These average concentrations are representative of background, but anthropogenic activities in some areas have increased selenium concentrations in surface waters (e.g., open-pit coal mining and oil and gas industry). Selenium concentrations in British Columbia waters are typically less than 1 µg/L, but can be elevated above 1 µg/L in areas where there are natural selenium sources from seleniferous rock or inputs from anthropogenic activities (e.g., open-pit coal mining).

## Mode of Action

Selenium is required in trace amounts for the normal functioning of cells in almost all animals; however, excess amounts can have toxic effects (USEPA 2016). Among the biologically essential elements, selenium is also one of the most toxic (Chapman et al. 2010). It has been generally believed that selenium toxicity is due to elevated selenium having the capacity to substitute for sulfur in cysteine and methionine, and the non-discriminant inclusion of selenomethionine during protein synthesis, potentially causing changes in tertiary protein structure and function by altering the disulfide linkages (Janz 2012). It is possible that teratogenesis in embryos is caused by this mechanism. Further evidence of selenium toxicity relating to the replacement of sulfur by selenium is suggested when observing the effects of selenosis in adult mammals and birds. One of the main symptoms of selenosis is damage to body structures containing keratin, which is high in sulfur (Spallholz and Hoffman 2002). However, this explanation has been questioned due to the more active regulation processes for selenocysteine, and due to methionine not having a major role in tertiary protein structure, therefore selenomethionine substitution should not have a major impact on the functioning of proteins (Janz 2012).

It has been more recently theorised that selenium is toxic because in excess it leads to the production of the superoxide anion and further reactive oxygen species, causing oxidative stress (Palace et al. 2004; Janz 2012). Selenium can oxidise thiols such as one of the functional groups found in glutathione, thus creating a metabolic intermediate, such as methylselenol or dimethyselenide. The selenide can then be oxidised by oxygen ($O_2$) to create superoxide anion ($O_2^-$) (Spallholz and Hoffman 2002; Palace et al. 2004; Janz 2012). At high concentrations of selenium, these species are found in quantities too great for antioxidant defenses to prevent oxidative damage (Janz 2012).

## Federal Environmental Quality Guidelines Derivation

### Federal Tissue Quality Guideline for Fish

Selenium distribution through the aquatic food web (e.g., plants, invertebrates and fish) has been shown to result in bioaccumulation of selenium in aquatic-dependent wildlife and causing reproductive impairments and malformations (Ohlendorf et al. 1986; Hoffman et al. 1988; Hothem and Ohlendorf 1989). There is a general agreement that freshwater fishes are more sensitive to selenium than other aquatic organisms (BCMOE 2014; USEPA 2016; ECCC, HC 2017). Chronic exposure to selenium causes toxicity to fish at concentrations only slightly above essentiality (Lemly 1997). Reproductive impairments in fish are well documented and the most sensitive life stages are egg and larvae. Exposure to selenium in fish during the larval stage primarily occurs through maternal transfer to the eggs and yolk sac absorption. Reduced hatching, teratogenicity (deformities) and edema are the most common effects observed in early life stages of fish (Lemly 2002; Janz et al. 2010). Significant correlation of selenium concentration measured in fish ovaries and eggs with these endpoints make them reliable predictors of selenium toxicity to fish (ECCC, HC 2017). ECCC, HC (2017) derived a predicted no-effect concentration (PNEC) for selenium based on selenium residues in egg-ovary.

In addition to a fish egg-ovary PNEC, a whole-body tissue PNEC for fish was also derived since monitoring data for egg-ovary are sparse and their collection is limited by the time of year. The data for selenium concentration in adult fish muscle or whole-body are more frequently available (ECCC, HC 2017). The concentration of selenium in fish tissues is an indicator of selenium bioavailability and represents accumulation from all possible exposure pathways. Both fish egg-ovary (14.7 µg/g dw) and whole body

tissue (6.7 µg/g dw) PNECs are adopted as federal environmental quality guidelines for selenium. The methodology used for their derivation, including toxicity data considered, are summarized here from ECCC, HC (2017).

Fish egg-ovary tissue residue guideline:

Available toxicity data for fish egg and ovary were compiled and evaluated in ECCC, HC (2017). When multiple acceptable endpoints of the same type were available for an individual species, a geometric mean was calculated following the principles of the CCME protocol (2007). Similar to the PNEC derivation (ECCC, HC 2017), selenium residues in eggs and ovaries are considered to be at a ratio of one-to-one for guideline development. Egg-ovary selenium concentrations associated with toxic effects ranged from 16.2 to 54 µg/g dw (Table 2). Bluegill sunfish (*Lepomis macrochirus*), white sturgeon (*Acipenser transmontanus*) and brown trout (*Salmo trutta*) were the most sensitive species, whereas dolly varden (*Salvelinus malma*) was the most tolerant species.

Acceptable toxicity data for each species were ranked according to sensitivity in a species sensitivity distribution. Several cumulative distribution functions were fit to the dataset and the logistic model provided the best fit among the models considered (ECCC, HC 2017). The 5$^{th}$ percentile ($HC_5$) of the SSD (14.7 µg/g dw) is the fish egg-ovary guideline (ECCC, HC 2017). The federal fish egg-ovary guideline is very similar to guidelines published by other jurisdictions: 11 µg/g dw by British Columbia (BCMOE 2014) and 15.1 µg/g dw by the USEPA (2016).

Fish whole-body tissue residue guideline:

Because of the scarcity of monitoring data for fish eggs and ovaries, it is practical to derive a whole-body tissue guideline for fish. The concentration of selenium in adult fish muscle or whole-body homogenate is more frequently available, and is an excellent measure of exposure to selenium in fish. The more sensitive and significant reproductive-based endpoints from the egg-ovary data (Table 2) were extrapolated to whole-body tissue values using species-specific egg-ovary to whole-body conversion factors developed by the USEPA (2016).

The toxicity-modifying factors that may affect the bioavailability of selenium were not separately taken into account, because the data used are tissue residues and therefore inherently account for the influence of these factors on the toxicokinetics of selenium (ECCC, HC 2017).

The whole-body selenium concentrations (Table 2) were plotted in a SSD and the logistic model provided the best fit of the models tested (ECCC, HC 2017). The $HC_5$ of the SSD was 6.7 µg/g dw and this value was selected as the whole-body tissue guideline for the freshwater fish (ECCC, HC 2017). Similar to the egg-ovary guideline, this whole-body tissue guideline is comparable to whole-body tissue guidelines developed by the other jurisdictions: 4 µg/g dw by BCMOE (2014) and 8.5 µg/g dw by the USEPA (2016).

**Federal Tissue Quality Guideline for Bird Egg**

The bird egg guidelines for selenium are recommended as the most direct estimate of selenium toxicity in aquatic-dependent wildlife and can be used as a surrogate for all wildlife species (BCMOE 2014, USEPA 2019). In the absence of a CCME protocol for developing bird egg guidelines, *ad hoc* approaches have been used for developing the federal tissue residue guidelines for bird eggs. The USEPA (2019) has recently developed a bird egg selenium criterion (guideline) of 11.2 µg/g dw. This USEPA guideline is adopted as the federal tissue quality guideline for bird eggs. The toxicity data and methodology considered by the USEPA (2019) are summarized here (Table 3).

Table 2. Chronic toxicity data used for deriving fish egg-ovary and whole-body tissue guidelines for selenium.

| Species | Endpoint | Concentration (mg/kg dw) | Egg or Ovary | E-O/WB[a] | WB[b] (mg/kg dw) | Reference |
|---|---|---|---|---|---|---|
| Bluegill (*Lepomis macrochirus*) | $EC_{10}$ larval edema | 16.22 | O | 2.13 | 7.62 | Hermanutz et al. 1992, 1996; Doroshov et al. 1992 |
| White Sturgeon (*Acipenser transmontanus*) | $EC_{10}$ larval deformities | 16.27 | E | 1.69 | 9.63 | Linville 2006 |
| Brown trout *(Salmo trutta*) | $EC_{10}$ larval survival | 18.09 | E | 1.45 | 12.48 | Formation Environmental 2011; US EPA 2016 |
| Brook trout (*Salvenius fontinalis*) | NOEC larval deformities | 20 | E | 1.38 | 14.49 | Holm 2002; Holm et al. 2003, 2005 |
| Largemouth bass (*Micropterus salmoides*) | $EC_{10}$ larval survival & mort | 20.35 | O | 1.42 | 14.33 | Carolina Power & Light 1997 |
| Northern pike (*Esox lucius*) | $EC_{10}$ larval deformities | 20.4 | E | 2.39 | 8.54 | Muscatello et al. 2006 |
| Rainbow trout (*Oncorhynchus mykiss*) | $EC_{10}$ skeletal deformities | 21.1 | E | 2.44 | 8.65 | Holm 2002; Holm et al. 2003, 2005 |
| Fathead minnow (*Pimephales promelas*) | LOEC larval deformities | 23.85 | O | 2 | 11.93 | Schultz and Hermanutz 1990 |
| Westslope cutthroat trout (*Oncorhynchus clarki lewisi*) | $EC_{10}$ alevin mortality | 24.06 | E | 1.96 | 12.28 | Rudolph et al. 2008; Nautilus Environmental 2011 |
| Yellowstone cutthroat trout (*Oncorhynchus clarki bouvieri*) | MATC alevin mortality | 25 | E | 1.96 | 12.76 | Formation Environmental 2012 |
| Dolly varden (*Salvelinus malma*) | $EC_{10}$ larval deformities | 54 | E | 1.61 | 33.54 | McDonald et al. 2010 |

[a] egg-ovary to whole-body conversion factors developed by the USEPA (2016);
[b] converted whole-body tissue concentration

Egg-laying vertebrates such as fish and birds are most sensitive to selenium effects (Janz et al. 2010). For example, among the wildlife species studied at Kesterson Wildlife Refuge (California), the most frequent and extreme toxicity was observed in aquatic birds, whereas small mammals showed almost no effects (Ohlendorf et al. 1989). This difference is likely due to a much wider range between biologically essential and toxic exposure concentrations of selenium in mammals than in birds (Janz et al. 2010). Egg selenium concentrations of less than 0.66 µg/g dw in birds may indicate inadequate selenium in the diet, resulting in poor adult health and reproduction (USEPA 2019).

The diet and subsequent maternal transfer of selenium in birds is the main exposure route for selenium. The most effective measurement of potential toxic effects of selenium in birds is through selenium measurement in eggs (Adams et al. 1998; Ohlendorf and Heinz 2011). Moreover, due to rapid selenium accumulation and loss, including maternal transfer, observed in birds, selenium levels measured in eggs most likely represent contamination of the local environment. In areas not receiving selenium contamination, reported selenium concentrations in bird eggs were typically less than 5 µg/g, whereas eggs from contaminated areas (Kesterson Reservoir) contained over 40 µg/g. Measuring selenium in eggs is also advantageous because eggs are easier to collect than adult birds, and the loss of an egg from the nest likely has lesser effect on a population. Moreover, an egg sample is a representative of an integration of exposure of adult females ranging from few days to weeks before egg laying.

Birds are very sensitive to selenium toxicity and the sensitive chronic effects are related to reproductive impairments, such as decreased fertility, reduced egg hatchability and increased frequency of deformity in embryos (Ohlendorf et al. 1986; Ohlendorf and Heinz 2011). Selenium exposure may cause many deformities in bird embryos, including hydrocephaly, missing eyes, twisted bills and deformed limbs (USEPA 2019). Toxicity studies on birds show that thresholds for reduced egg hatchability are usually below those for teratogenic effects (Ohlendorf 2003).

Chronic toxicity data were available for eleven bird species and among them mallard (*Anas platyrhynchos*) was the most sensitive, whereas red-winged blackbird (*Agelaius phoeniceus*) was the least sensitive species. Hatchability was consistently the most sensitive endpoint for mallard. Among the six mallard studies (Heinz et al. 1987, 1989; Heinz and Hoffman 1996, 1998; Stanley et al. 1994, 1996), hatchability data from three studies (Heinz et al. 1987, 1989; Stanley et al. 1996) were used to derive the USEPA bird egg guideline (Table 3).. Duckling growth, weight and production were all equally sensitive to hatching success in Stanley et al. (1996), and the number of normal hatchlings and nestling weight were similar in sensitivity to hatchability in Heinz et al. (1989).

The USEPA (2019) used toxicity data from three mallard studies (Table 3) for calculating a bird egg $EC_{10}$ of 11.2 µg/g dw. This calculated $EC_{10}$ is the selenium bird egg guideline with the lower and upper 95% confidence limits of 7.4 and 15 µg/g dw, respectively. The selenium $EC_{10}$ was derived using a four-parameter generalized linear model instead of the toxicity relationship analysis program (USEPA 2015) because this program was not designed to work with data pooled from multiple studies (USEPA 2019).

Table 3. Effect of dietary selenium on hatchability of mallard eggs and associated selenium concentrations in mallard eggs. Source: USEPA 2019

| Diet Se mg/kg[a] | N (hens) | Egg Hatchability % | % Hatchability as % Control | Percent Moisture | Egg Se (mg/kg dw) | Reference |
|---|---|---|---|---|---|---|
| Control | 11 | 64.4 | 100 | 71 | 0.17 | Heinz et al. 1987 |
| 10 | 5 | 34.6 | 54 | 71 | 15.9 | Heinz et al. 1987 |
| Control | 32 | 57.3 | 100 | 70 | 0.60 | Heinz et al. 1989 |
| 1 | 15 | 65.0 | 114 | 70 | 2.77 | Heinz et al. 1989 |
| 2 | 15 | 59.6 | 104 | 70 | 5.33 | Heinz et al. 1989 |
| 4 | 15 | 54.3 | 95 | 70 | 11.3 | Heinz et al. 1989 |
| 8 | 15 | 42.3 | 74 | 70 | 36.7 | Heinz et al. 1989 |
| 16 | 9 | 7.4 | 13 | 70 | 60.0 | Heinz et al. 1989 |
| Control | 33 | 62 | 100 | 71 | 0.93 | Stanley et al. 1996 |
| 3.5 | 29 | 61 | 98 | 71 | 12.1 | Stanley et al. 1996 |
| 7 | 34 | 41 | 66 | 71 | 24.5 | Stanley et al. 1996 |

[1]Selenium concentration in diet is presented as nominal; control diets typically contained 0.4 mg Se/kg dw

**Recent Water-based Guidelines from other Jurisdictions**

The tissue-based environmental quality guidelines described above have the greatest certainty, as this metric is most directly related to the site of toxic action. However, water quality guidelines are generally the most common metric used to monitor environmental conditions and regulatory performance. In part, this may be because it is an easier compartment to sample. Consequently, although no water PNECs were developed as part of the risk assessment (ECCC, HC 2017), water quality guidelines developed recently in North America are presented here for possible use in Canada.

In addition to fish whole-body tissue and egg-ovary based guidelines, the British Columbia Ministry of Environment (BCMOE 2014) also developed water based selenium guidelines. They compared the CCME (1987) water quality guideline for the protection of aquatic life (1 µg/L) with the published toxicity thresholds and water quality guidelines of other jurisdictions. They concluded that the CCME guideline of 1 µg/L is likely protective for the most sensitive environments and recommended the guideline as an alert concentration. The alert guideline exceedances at sensitive sites can help in early detection of potential selenium concerns, in setting increased monitoring efforts and as part of a tiered, adaptive management approach to initiate early proactive management actions (BCMOE 2014). The BCMOE (2014) also proposed a water column guideline for the protection of aquatic life of 2 µg/L and assessed its applicability by comparing the guideline value to ambient selenium surface water concentrations in British Columbia, Alberta, and across Canada. Collectively, with the review of existing toxicological data, these results provide support to BC's water quality guideline of 2 µg/L, or a lower value for very sensitive environments or species. Based on jurisdiction's needs and policy goals, BC water quality guidelines can be used for Canadian sites.

The USEPA (2016) derived water column criteria for lentic (1.5 µg/L) and lotic (3.1 µg/L) waters by translating the fish egg-ovary concentration to an equivalent water column concentration using a mechanistic bioaccumulation model of Presser and Luoma (2010). This model quantifies bioaccumulation in fish tissue by assuming that net bioaccumulation is a balance between assimilation efficiency from diet, ingestion rate, rate of direct uptake in dissolved forms, loss rate and growth rate. Because the potential of selenium bioaccumulation can depend on several site-specific biogeochemical factors, translation of the egg-ovary criterion to the water column criterion can be improved by deriving a site-specific criterion using site-specific selenium data and information on food-web dynamics. For their modelling exercise, the USEPA calculated selenium concentrations in the water column for a large number of study sites (26 lentic and 39 lotic) for fish species. The USEPA modelling approach can be considered for deriving the site-specific water guideline if the user is able to come up with the required site-specific data for the model. Similar to USEPA the water

guideline is only to be used when the whole-body tissue guideline is not available and that the egg-ovary guideline supersedes both whole-body tissue and water guideline.

Site-specific water quality guidelines have long been recognized as a mechanism of increasing the precision and applicability of general water quality guidelines (CCME 2003). Accordingly, a number of approaches are available to calculate water concentrations using site-specific transfer factors. Details on background concentrations, recalculation, water effect ratio and resident species procedures are presented in detail in CCME (2003).

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## List of Acronyms and Abbreviations

ATSDR – Agency for Toxic Substances and Disease Registry
CAS – Chemical Abstracts Service
CCME – Canadian Council of Ministers of Environment
CEPA – Canadian Environmental Protection Act
CMP – Chemicals Management Plan
dw – dry weight
$EC_x$ – effect concentration to x % of test species
FEQG – Federal Environmental Quality Guideline
FWQG – Federal Water Quality Guideline
GC– Government of Canada
MATC – maximum acceptable toxicant concentration (geometric mean of the NOEC and LOEC)
MDL – method detection limit
PNEC – predicted no-effect concentration
SAR – screening assessment report
SSD – species sensitivity distribution