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PROPOSED WQ GUIDELINES FOR SELENIUM TO PROTECT AQUATIC LIFE

N. K. Nagpal

Water Management Branch
Environmental and Resource Management Department
BC Ministry of Environment, Lands & Parks
Victoria, British Columbia

ABSTRACT

Water Management Branch (WMB) is responsible for developing water quality guidelines for contaminants to protect all water uses in BC from their adverse effects. The water uses considered in this exercise include drinking water, aquatic life, wildlife, and agriculture. Selenium is the focus of the current WMB's effort. This document reviews the critical literature and rational for the proposed water quality guidelines for selenium to protect aquatic life in BC.

INTRODUCTION

Selenium (Se) is both beneficial to animals, plants, and humans. However, selenium poisoning due to an excessive exposure has been well documented in the literature. Selenium exists in the 2^- (Se^{-2} or selenide), 0 (Se^0 or elemental Se), 4^+ (SeO_3^{-2} or selenite), and 6^+ (SeO_4^{-2} or selenate) oxidation states. Each oxidation state exhibit different chemical behaviour. The concentration, speciation, and association of Se in a given environment depend upon pH and redox conditions, the solubility of its salts, the complexing ability of soluble and solid ligands, biological interactions, and reaction kinetics. Selenide and elemental Se occur in acidic, reducing, and organic rich environments. Metallic selenides, Se-sulphides, and elemental Se are insoluble, and therefore, biological unavailable. For the pH and redox conditions of most soil and aquatic environments, SeO_3^{-2} and SeO_4^{-2} should be the dominant forms of Se (McNeal and Balistrieri 1989).

SOURCES OF SELENIUM

Volcanic emanations and metallic sulphides associated with igneous activities are primary sources of Se in nature. Secondary sources include biological pools in which Se has accumulated. Seleniferous' soils are found in the arid and semi-arid areas of the world, including the western areas of Canada and the United States (Adriano 1986).

Anthropogenic sources of Se include fuel (coal and oil) combustion, primary and secondary non-ferrous metal industry, waste disposal and incineration, manufacturing processes, mining, smelting, and refining activities, and steam electric. Selenium is released during coal mining because of the oxidation of selenium-bearing pyrite (USDHHS 1994). Compounds of selenium are used in many products: photoelectric cells;

as maroon and orange pigments; in combination with cadmium sulphide for plastics and ceramics; in increasing the resistance of rubber to heat, oxidation, and abrasion (as an accelerator and vulcanizing agent in rubber production); and as lubricants to increase the machinability of stainless steel. Selenium sulphide is also used as a catalyst in the preparation of Pharmaceuticals including niacin and cortisone, as an ingredient in anti-dandruff shampoos (selenium sulphide) for certain scalp conditions, and as a constituent of fungicides (selenium sulphide). Radioactive selenium is used in diagnostic medicine and aids in the visualization of difficult-to-study malignant tumours. Selenium is also used in glass manufacturing and as an alloy of steel and copper (Adriano 1986, USDHHS 1994).

ENVIRONMENTAL CONCENTRATION and FATE

Selenium concentrations in aquatic environments are generally low. The total Se concentration in the BC rivers was found to be less than one 0.001 mg Se/L in Fraser River, Kettle River, and Okanagan River. However, higher concentrations ranging up to 0.0025 mg Se/L were measured in the Elk River, which is located in the coal mining area of eastern BC. In seawater, concentrations of selenium were reported to range from 0.000009 to 0.00045 mg Se/L (Eisler 1985).

The mean Se concentration in the invertebrates (amphipoda, sphaeriidae, unioidae, and gastropoda) from Battle River of Alberta and Saskatchewan ranged from 0.2 to 1.6 mg Se/kg dry-weight. The highest concentration of 4.0 mg Se/kg dry-weight was recorded for unioidae at the site, which was mainly influenced by agricultural activities. The concentration in the muscle tissue of white sucker and northern pike ranged from <0.007 to 0.023 mg Se/kg wet-weight (Anderson *et al.* 1994)

Selenite (Se IV) and selenate (Se VI) are the dominant species in most fresh and marine waters. A large percentage of selenium also occurs in organic forms as result of sorption by biogenic particles and methylation. Organic metabolites of selenium are more volatile than the parent organic forms. Plants, fungi, bacteria, microorganisms, and animals can produce methylated forms of Se from inorganic and certain organic forms (Moore, 1991; Adriano 1986). The formation of methylated Se compounds by animals appears to be one mechanism for Se detoxification as the toxicity of dimethyl selenide is 500 to 1 000 times lower than the toxicity of Se^{2-} (McNeal and Balistrieri 1989).

Aquatic organisms accumulate selenium from both water and food. The bioaccumulation of selenium through the diet, however, is usually greater than the direct uptake from water, particularly when the Se

occurs in natural dietary ingredients (Ohlendorf 1989). Evidence for selenium biomagnification through the food chain is contradictory (Ohlendorf 1989). In aquatic food chain from the Lower San Joaquin River, California, Saiki *et al.* (1993) observed that Se concentration was lower in filamentous algae than in invertebrates and fishes. However, Se levels in or on detritus were similar to or higher than in invertebrates and fishes. They concluded that Se in invertebrates and fishes accumulated through food-chain transfer from Se-enriched detritus rather than from filamentous algae.

CARCINOGENICTY, MUTAGENICITY, and TERATOGENICITY

Earlier studies with seleniferous corn and wheat in the diet, sodium selenite or sodium selenate in drinking water, and sodium selenate in the diet reported that selenium was carcinogenic in animals. However, subsequent studies revealed that the earlier studies were flawed and that there was no association between selenium intake and the incidence of cancer in humans and animals (USDHHS 1994). The only selenium compound that has been shown to be carcinogenic is selenium sulphide administered orally to mice and rats. Hepatocellular carcinomas and adenomas (in rats and mice), and alveolar/bronchiolar carcinomas and adenomas (in mice) were reported in the animals following chronic oral exposure to selenium sulphide. Selenium sulphide is a pharmaceutical compound used in antidandruff shampoos. However, it is not absorbed through the skin and the use of shampoos containing this compound should be safe, unless one has cuts or sores on the scalp or hands (USDHHS 1994).

TOXICITY TO FRESHWATER BIOTA

Selenium toxicity to aquatic organisms is governed by several factors: form and concentration of selenium, type and characteristics (species and life stage) of organism, type and duration of exposure (chronic and acute), and environmental factors (water hardness, temperature, other variables, etc.).

The lowest selenite-Se concentration in water, causing 50% mortality in adult fathead minnows *(Pimephalespromelas),* was reported to be 0.62 mg Se/L (USEPA 1980, 1987).

In a study with the swim-up fry (8-12 weeks) of coho salmon *(Oncorhynchus kisutch),* Hamilton and Buhl (1990) found that selenite-Se was more toxic than selenate-Se. The fry showed 50% mortality at 7.8 mg/L of selenite-Se or 32.5 mg/L of selenate-Se. In the same study, Hamilton and BuM also concluded that the joint acute toxicity of the binary mixtures of selenate and selenite selenium to the fish could be characterized as strictly additive,

The available data indicated that chronic toxicity of selenium ranged from a low of 0.005 to 40 mg Se/L for several species of freshwater fish. Sorensen (1988) reported abnormalities of both liver and ovaries in redear sunfish *(Lepomis microlophus)* from Martin Lake contaminated with selenium at 0.005 mg Se/L. Cumbie and Van Horn (1978) reported complete reproduction failure for bluegill *(Lepomis macrochirus),* green sunfish *(L. cyanellus),* largemouth bass *(Micropterus salmoides)* and flat bullhead *(Ictalurus platycephalus)* from Belews Lake containing 0.01 mg Se/L at pH 7.5 and water hardness of 38.2 mg $CaCO_3$/L. A total of 29 species of fish in this lake decreased in standing crop from 86 7 to 27 1 g/ha for this same time period. Because the levels of selenium in the water were relatively low for the severity of the effects on the fish, it was hypothesized that these deleterious effects were a result of bioaccumulation through the food chain; the phytoplankton from two sites at Belews Lake contained an average of 15 to 70 µg Se/g dry-weight.

In a three-trophic level and a continuous flow-through test system, Dobbs *et al.* (1996) exposed alga *(Chlorella vulgaris),* rotifers *(Brachionus calyciflorus),* and fathead minnows *(Pimephales promelas)* for 25 days to various levels of selenium (0, 0.110, 0.207, and 0.396 mg Se/L). They found that the fathead minnow growth was impaired after seven days at 0.207 and 0.396 mg Se/L levels, with 100% mortality by day sixteen. A reduction in the fish biomass, compared to the control, was also apparent at 0.110 mg Se/L after day twenty.

Gillespie and Baumann (1986) studied bluegill larvae collected from crosses of adults from Hyco Reservoir with relatively high selenium in water (0.009-0.012 mg Se/L) and in diet (25-45 µg Se/g dry-weight), and adults exposed to much lower levels of selenium. They noted that the exposure to Se in the Hyco Reservoir environment resulted in larvae with gross abnormalities and general edema. They concluded that the symptoms seen in the larvae were due to selenium transferred to the egg from the female parent. Hermanutz *et al.* (1992) with bluegills in experimental streams, and Schultz and Hermanutz (1990) with fathead minnows *(Pimephalespromelas)* observed similar effects when exposed to 0.01 mg/L Se. These studies, looking at the uptake of selenite in embryos after hatching, concluded that the high levels seen in embryos were not taken up from water initially upon hatching but were in fact passed on from the parent.

Malchow *et al.* (1995) reported significant reduction in larval growth of the midge *(Chironomus decorus)* exposed to 0.01 mg Se/L, as selenite or selenate, in water for a period of 96 hours. In these tests, it was

found that the larvae were feeding on algae *(Selenastrum capricornutum)* which had bioaccumulated selenium from water to concentrations of ≥2.84 μg selenite-Se/g dry-weight and ≥2.11 μg selenate-Se/g dry-weight, respectively.

Maier *et al.* (1993) studied mortality in neonate of the water flea *(D. magnd)* exposed to different forms of selenium in water. The 48-h LCsoS were as follows: 2.84, 0.55, 0.31 and 2.01 mg/L for selenate-Se, selenite-Se, seleno-DL-methionine, and seleno-DL-cystine, respectively. These data show that organic forms of selenium are as toxic, if not more, as the inorganic forms. Immobilization as a sublethal acute response (or 48-h ICso) was observed at concentrations of 0.045 mg/L for seleno-DL-methionine and 0.52 mg/L for seleno-DL-cystine.

Davis *et al.* (1988) reported *that* 0.006 mg Se/L as selenite resulted in 50% mortality of the cladoceran *(Daphnia pulicaria)* in 96 hours. The same results were obtained with water flea *(D. magna)* exposed to 0.04 mg/L selenium as seleno-DL-methionine by these investigators.

Davis *et al* (1988) reported a decrease in reproduction of protozoan *Entosiphion sulcatum* when exposed to 0.003 mg/L selenite-Se. Crane *et al.* (1992) reported changes in abundance of some freshwater invertebrates exposed to Se for 514 days. Midges *(Chironomidae)* and water fleas *(Eurycercus lamellatus* and *Graptoleberis testudinaria)* were shown to decrease in abundance at concentrations 0.002 to 0.025 mg Se/L. The decrease in abundance was also observed between 0.010 and 0.025 mg Se/L for the water flea *(Chydorus ovalis),* and 0.025 mg Se/L for the water flea *(Acroperus harpae),* the parasitic copepod *(Ergasilus sp.),* and the tubifitid worm *(Tubifex tubifex).* It was not apparent whether the decrease in abundance was due to a decrease in reproduction or in growth to adult stage.

Davis *et al.* (1988) reported a descrease in cell division for the algae *(Ankistrodesmus falcatus)* at a concentration of 0.010 mg Se/L as selenate. Kiffney and Ejiight (1990) exposed cyanobacterium *(Anabaenaflos-aquae)* to seleno-L-methionine, selenate-Se, and selenite-Se for 10 days. The first sub-lethal effect (decrease in chlorophyll *a)* was observed at 0.1, 3.0, and 3.0 mg Se/L, respectively.

TOXICITY TO MARINE BIOTA

The lowest EC_{50} or LC_{50} value for the larvae of haddock *(Melanogrammus aeglifinus)* was reported as 0.599 mg/L selenite-Se (USEPA 1987). Chapman (1992) exposed striped bass *(Morone saxatilis)* to

selenite-Se and selenate-Se, during different life stages and conditions of inflated or uninflated bladder. Their results indicated that selenate is significantly more toxic to marine fish than selenite; this trend is opposite to what was found in the fresh water environment. The USEPA (1987) reported a significant incidence of developmental anomalies of the lower jaw in striped bass *(Morone saxatilis)* exposed to a concentration of 0.039 mg/L selenate-Se for 9 to 65 days.

In a life cycle test with opossum shrimp *(Mysidopsis bahid),* the USEPA (1980) reported an LC_{50} of 0.127 mg Se/L for the egg stage. Nelson *et al,* (1988) reported that a concentration of 0.255 mg Se/L as selenite resulted in a 50% mortality of the bay scallop *(Argopectin irradians).* Few chronic toxicity data were found in the literature for marine invertebrates. Micallef and Tyler (1990) reported a reduction in filtration rates by 86-100% when the blue mussel *(Mytilus edulis)* was exposed to 0.05 mg Se/L in the laboratory. The USEPA (1987) reported a life cycle chronic value of 0.212 mg/L selenite-Se for the mysid *Mysidopsis bahia.*

The effects of selenium on algae depend on the type of algae, the concentration and form of selenium, and the concentration of sulfate (USEPA 1987). In experiments where the organisms were pre-treated with sulfate concentration (850 mg/L) normally present in seawater, the most sensitive of the algae tested was the red algae *(Porphyridium cruentum)* which showed a decrease in growth at 0.01 mg/L selenate-Se. The growth of most of the other alga including the green alga *(Dunaliella primolectd), (Platymonas subcordiformis),* and *(Platymonas sp.),* and the diatom *(Tetraselmis chuii)* was reduced between 0.1 and 1.0 mg/L selenate-Se with the exception of the green algae *(Chlorella sp.).*

TOXICITY TO WILDLIFE

In the Kesterson Reservoir, Ohlendorf *et al.* (1988) reported adverse effects of Se on wildlife (mallards and American coot) inhabiting the area. The water entering the Kesterson reservoir/ponds, from the contaminated San Luis Drain, had an average level of 0.3 mg/L selenium. The concentration of selenium in plants, invertebrates and fish collected from the ponds ranged from 22-175 μg Se/g.

Peterson and Nebeker (1992) modeled thresholds for waterborne selenium toxicity to aquatic birds and mammals. The model considered the duration of time the birds spent in the contaminated areas, the proportion of diet comprised of foods from contaminated aquatic environment, trophic position, food ingestion rates (function of body mass), and energy demands associated with activities such as

reproduction. The toxicity thresholds for the marsh wren *(Cistothorus palustris),* belted kingfisher *(Ceryle alcyon),* osprey *(Pandion haliaetus),* bald eagle *(Haliaeetus leucocephalus)* and the mallard were estimated to be 0.8, 0.9,1,3, 1.9, and 2.1 μg/L, dissolved selenium, respectively. The toxicity thresholds for mammals in the same environment were 0.9 μg Se/L for bats and shrews, 1.1 μg Se/L for mink, and 0.7 μg Se/L for river otter. Independent efforts by Lemly and Smith (1987), DuBowy (1989), and Skorupa and Ohlendorf (1991), as reponted in Peterson and Nebeker (1992), suggested that waterborne selenium (dissolved) concentrations between about 1 and 3 μg/L were protective of aquatic birds.

Heinz *et al.* (1989) reported that female mallards fed control diets with no added selenium had an average of eight ducklings compared to females fed 8 μg/g of selenomethionine-Se who had an average of 4.6 young; there were no young born to females fed 16.0 μg/g selenomethionine-Se. The authors concluded that the dietary threshold necessary to impair reproduction in mallards is between 4 and 8 mg/g (dry-weight) selenium as selenomethionine. They also suggested that when eggs from a wild population contain ≥1 μg Se/g (wet-weight), reproductive impairment might be possible. At 5 μg Se/g (wet-weight) in eggs, reproductive impairment is much more likely to occur.

PROPOSED GUIDELINE

It is recommended that the maximum concentration of selenium in water for the protection of freshwater and marine aquatic life should not exceed 0.001 mg Se/L.

RATIONALE: FRESHWATER LIFE

The proposed guideline was determined using the lowest observed effect level of 0.01 mg Se/L and the Canadian Council Ministers of the Environment recommended application factor of 0.1 for chronic data. Many investigators (Hermanutz 1992, Schultz andHermanutz 1990, Hermanutz et al. 1992, Gillespie and Baumann 1986, Cumbie and VanHorn 1978, Bringmann *and* Kuhn 1977) reported adverse effects at 0.01 mgSe/L

RATIONALE: MARINE LIFE

The proposed guideline was determined using the chronic LOEL of 0.01 (USEPA 1987) mg Se/L and the recommended application factor of 0.1.

Several studies reported adverse effects of selenium at concentrations lower than 0.01 mg Se/L in both fresh and marine waters. These studies were not used for derivation of the water quality guidelines because: (i) they lacked in details about the data and its original source; (ii) they failed to provide details on experimental methodology (re: Se effects) which made the data suspect; (iii) chronic endpoints were indefinite or could have been influenced by factors other than those considered in the study, and (iv) test organisms, while exposed to waterborne selenium, were fed a diet containing selenium.

RATIONALE: WILDLIFE

Based on single-species toxicity data, bioaccumulation of Se in aquatic food webs, and exposure to the contaminant, Peterson and Nebeker (1992) estimated a threshold of about 0.001 mg/L waterborne selenium for birds and mammals with food habits that likely lead to high exposure to bioaccumulative contaminants in aquatic systems (e.g., piscivorous birds and mammals).

Heinz *et al.* (1989) noted that the dietary threshold of selenium necessary to impair reproduction in mallards is between 4 and 8 μg Se/g (dry-weight). Given the safe concentration of 0.001 mg/L selenium, the bioaccumulation factor (BAF) was estimated to be = (4 μg/g -f 0.001 mg/L) to (8 μg/g -f 0.001 mg/L), or 4000 to 8000. The estimated BAF of 4000-8000 is close to the upper limit of those reported in the literature for aquatic plants and insects. It suggests that the proposed guideline of 0.001 mg/L selenium is protective of the wildlife (e.g., Mallards) feeding on aquatic organisms.

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