

This document provides Canadian soil quality guidelines for selenium for the protection of environmental and human health (Table 1). A scientific supporting document is also available (CCME 2009).

## **Background Information**

Selenium (CAS RN: 7782-49-2) is a Group 16 non-metal. There are several allotropic forms of selenium, three of which are widely recognized. Amorphous selenium is dark red in powder form and bluish-black as a vitreous solid, while crystalline selenium is a deep red. The most stable form of selenium is termed grey or metallic selenium, also called crystalline hexagonal selenium, and is a lustrous grey to black colour. Se has four valence

states (0, II, IV, VI) and forms a large number of compounds in the environment, including bromides, fluorides, chlorides, oxides, hydrides, sulphides, and many metal compounds (i.e., selenides). Selenium comprises approximately 0.05 to 0.09 mg·kg<sup>-1</sup> of the earth's crust. In nature, it usually occurs as selenides of metals, in hydrothermal sulphides, and in uranium ores in sandstone-type deposits (Shamberger 1981). It can also be found to a lesser extent in pyrite, clausthalite (PbSe), naumannite  $(Ag_2Se),$ tiemannite (HgSe), and selenosulphur. Selenium also occurs in volcanic rocks at concentrations as high as  $120 \text{ mg} \cdot \text{kg}^{-1}$ , and in coal and oil at mean concentrations of  $3.0 \text{ mg} \cdot \text{kg}^{-1}$  (range: 0.046 to 10.65) and 0.6 mg·kg<sup>-1</sup> (range: 0.06 to 2.2), respectively (Marier and Jaworski 1983).

	Land use					
	Agricultural	Residential / parkland	Commercial	Industrial		
Guideline	$1^{a}$	1 <sup>a</sup>	<b>2.9</b> <sup>a</sup>	<b>2.9</b> <sup>a</sup>		
SQG <sub>HH</sub>	80	80	125	1135		
Limiting pathway for SQG <sub>HH</sub>	Soil ingestion	Soil ingestion	Soil ingestion	Off-site migration		
$SQG_E$	1	1	2.9	2.9		
Limiting pathway for $SQG_E$	Soil contact	Soil contact	Soil contact	Soil contact		
Interim soil quality criteria (CCME 1991)	2	3	10	10		
Soil Quality Guidelines (EC 2001)	1	1	3.9	3.9		

#### Table 1. Soil quality guidelines for selenium (mg·kg<sup>-1</sup>).

Notes:  $SQG_{HH} = soil$  quality guideline for human health;  $SQG_E = soil$  quality guideline for environmental health; NC = not calculated; ND = not determined

<sup>a</sup>Data are sufficient and adequate to calculate both an SQG<sub>HH</sub> and an SQG<sub>E</sub>. Therefore the soil quality guideline is the lowest of the SQG<sub>HH</sub> and SQG<sub>E</sub>, and represents a fully integrated *de novo* guideline for this land use, derived in accordance with the Protocol. The corresponding interim soil quality criterion (CCME 1991) and soil quality guideline (EC 2001) are superseded by this new SQG.

The guidelines in this fact sheet are for general guidance only. Site-specific conditions should be considered in the application of these values. The values may be applied differently in various jurisdictions. The reader should consult the appropriate jurisdiction before application of the values.

Canadian Environmental Quality Guidelines Canadian Council of Ministers of the Environment, 2002, updated 2007, revised 2009

In Canada, primary selenium is recovered as a by-product of copper refining processes. In 2003, Canada was the second largest producer of selenium after Japan and before Belgium (Yukon Zinc Corporation 2005). Primary selenium production in 2005 from Canadian sources (Quebec, Ontario, Manitoba and Saskatchewan) was estimated to be about 216 tonnes (NRCan 2006), recovered as by-products of the copper refining processes. The principal global markets for selenium (with estimated percentages) are glass manufacturing (35%), chemicals and pigments (24%), metallurgical additives (23%), electronics (10%), and other applications (8%) (George 2004). In the glass industry, selenium is used as a colourizing and a decolourizing agent. In metallurgical applications, selenium is used as an additive to improve machinability, casting and forming properties of steel, copper and lead alloys and as a substitute for lead in brass plumbing. With respect to chemical applications, selenium is used as a catalyst in the preparation of some pharmaceuticals and as an ingredient in various pharmaceutical preparations such as dietary supplements for humans and farm animals, anti-dandruff shampoos, and anti-fungal agents (George 2004).

The major electronic use of selenium in the 1970s and 1980s was as a photoreceptor, on drums of photocopying machines. This end-use has been drastically curtailed owing to the substitution of selenium compounds for amorphous silicon and organic photoreceptor compounds and thus, selenium compounds are now only used to repair older copiers (George 2004; Andersson 2005). Other electronic applications for selenium include rectifiers (now largely replaced by silicon (Andersson 2005)), photographic toners, p-type semiconductors, arc light electrodes (Merck Index 1996), light meters, solar cells and photoelectric cells (Yukon Zinc Corporation 2005). Selenium is also used as a vulcanizing agent in rubber processing and as a catalyst in Kjeldahl nitrogen analysis (George 2004).

Both anthropogenic and natural sources contribute to the ubiquitous presence of selenium in all media of the Canadian environment. Examples of major anthropogenic sources of selenium include copper refining operations, glass manufacturing, pigments, metallurgical activities, burning of coal and oil, and agricultural/biological applications (e.g., feed additives, fertilizers, pesticides). The major natural sources are the weathering of rocks, minerals, soils, and volcanic activity.

Worldwide anthropogenic inputs of selenium to aquatic ecosystems are estimated at 10,000 to 72,000 t $\cdot$ yr<sup>-1</sup> (Nriagu and Pacyna 1988). Estimated anthropogenic inputs to soils range from 6,000 to 76,000 t $\cdot$ a<sup>-1</sup> (Nriagu

and Pacyna 1988). Additional data on anthropogenic sources are reviewed by Nagpal and Howell (2001).

Most of the recent data on the selenium content in Canadian soils came from the Prairie Provinces and In 1992, the Geological Survey of Canada Ontario. (Natural Resources Canada) performed an ultra-low density regional geochemical survey of surface soils in the Prairies (R.G. Garrett, 2005, Natural Resources Canada, pers. com.). A total of 1076 Prairie soil samples were analysed for Se. Selenium levels in Alberta soils ranged from 0.1 to 2.7 mg·kg<sup>-1</sup> (mean ( $\pm$ s.d.): 0.55( $\pm$ 0.28) mg·kg<sup>-1</sup>), those from Saskatchewan ranged from 0.1 to 3.1 mg  $kg^{-1}$  (mean: 0.53(±0.28) mg·kg<sup>-1</sup>), and those from Manitoba ranged from 0.1 to 4.7 mg·kg<sup>-1</sup> (mean:  $0.62(\pm 0.44)$  mg·kg<sup>-1</sup>). Haluschak et al. (1998) conducted a survey of southern Manitoba soils to assess soil background levels from 121 areas. Selenium levels ranged from <0.2 to 3.8 mg·kg<sup>-</sup> <sup>1</sup>with a mean ( $\pm$ s.d.) of 0.5 $\pm$  0.4 mg·kg<sup>-1</sup>. Soils with the highest selenium levels were those associated with shales as parent materials. In Alberta, an elemental survey of 258 agricultural soil samples soils from 129 sampling sites chosen from 43 benchmark locations was performed in 2002 (Penny 2004). Total selenium levels ranged from 0.1 to 1.6 mg·kg<sup>-1</sup> with a mean ( $\pm$ s.d.) of 0.476( $\pm$ 0.278) in the 0 to 15 cm sampling depth samples, and from 0.001 to 2.3  $mg \cdot kg^{-1}$  with a mean (±s.d.) of 0.474 (±0.335) in the 15 to 30cm sampling depth samples. Selenium concentrations in garden soil samples collected in Ottawa, Ontario ranged from 0.3 to 1.2 mg·kg<sup>-1</sup> (mean: 0.6 mg·kg<sup>-1</sup>) (Rasmussen et al. 2001). A second ultra-low density regional geochemical survey was performed in Ontario south of Sault Ste.Marie by the Geological Survey of Canada. The Se determinations in 294 surface soil samples (top 25 cm) ranged from 0.1 to 3.9 mg·kg<sup>-1</sup>, with a mean ( $\pm$ s.d.) of  $0.46 (\pm 0.38) \text{ mg·kg}^{-1}$  (R.G. Garrett, 2005, Natural Resources Canada, pers. com.). Surface soils were sampled from 12 urban locations in Windsor and from 18 rural locations in Essex County (Gizyn 1994). In urban soils, selenium concentrations ranged from 1.04 to 2.03 mg·kg<sup>-1</sup> with an arithmetic mean of 1.59 mg·kg<sup>-1</sup>. Selenium concentrations in rural soils were lower, ranging from 0.52 to 1.30 mg·kg<sup>-1</sup> with an arithmetic mean of 0.89 mg·kg<sup>-1</sup>. In Ontario, the 98<sup>th</sup> percentiles of selenium in soil samples from rural parkland and old urban parkland are  $0.93 \text{ mg} \cdot \text{kg}^{-1}$  and  $1.3 \text{ mg} \cdot \text{kg}^{-1}$ , respectively (OMEE 1994). A study of a variety of soil types from 53 sites across Canada, representing all provinces and territories except Manitoba, found background selenium concentrations to range from 0.03 to  $2 \text{ mg} \cdot \text{kg}^{-1}$  (mean 0.26 mg  $\cdot \text{kg}^{-1}$ ) (McKeague and Wolynetz 1980). For the purposes of developing Canadian Soil Quality Guidelines for selenium, a

conservative background soil concentration of  $0.7 \text{ mg}\cdot\text{kg}^{-1}$  has been assumed.

Naturally occurring selenium concentrations in groundwater are generally low, typically much less than  $0.001 \text{ mg L}^{-1}$ . Wells in southern Alberta tend to contain more selenium than elsewhere in the province because this element is a natural component of the native bedrock. Selenium was detected in 43% of 173 farm wells in southern Alberta, of which 8% of detected levels exceeded the MAC (0.010 mg·L<sup>-1</sup>) (Alberta Agriculture, Food and Rural Development 2002). Miller et al. (1996) conducted a study between 1990 and 1992 on the elemental content of shallow groundwater associated with dry land saline soils in southern Alberta. Selenium was detected in 86% of the 42 samples and levels (as dissolved Se) ranged from <0.141 to  $6.080 \text{ mg}\cdot\text{L}^{-1}$ ; the arithmetic mean ( $\pm$ s.d.) was 1.820  $\pm$ 1.520 mg·L<sup>-1</sup>. Shallow groundwater associated with dryland saline soils in the North American Great Plains is known to be unsuitable for human and livestock consumption (Outridge et al. 1999).

Drinking water supplies in Canada rarely exceed the Canadian Guideline for Drinking Water Quality, expressed as a Maximum Acceptable Concentration (MAC) of 0.01 mg·L<sup>-1</sup>, established in 1978 and updated in 1992 (Health Canada 1992; 2006a). A 1982 survey of Canadian drinking water supplies from 122 municipalities across Canada, representing 36% of the Canadian population, found that selenium was at or below a detection limit of  $0.0005 \text{ mg} \cdot \text{L}^{-1}$  (Subramanian and Méranger 1984). The analysis included raw, treated and distributed water samples. However, in areas of natural selenium enrichment, or anthropogenic contamination, selenium concentrations in drinking water may be higher. A 1975 study of Manitoba drinking water supplies found that, although 93% of samples were below the detection limit of 0.005 mg·L<sup>-1</sup>, 7% of the samples had selenium concentrations between 0.005 and 0.010 mg·L<sup>-1</sup> (Health Canada 1992). A concentration of 0.0005  $mg \cdot L^{-1}$  was considered to be the typical selenium level in Canadian drinking water supplies, based on extensive drinking water quality surveys performed across Canada.

Levels of selenium in the Canadian atmosphere are extremely low. The background selenium level used for exposure estimates by inhalation was 1.0 ng  $\cdot$ m<sup>-3</sup>. This value corresponds to the overall mean concentration of selenium in PM<sub>10</sub> samples (n=2170) collected across Canada in 2002 and 2003 from 31 National Air Pollution Surveillance stations (T. Dann, Environment Canada, pers. com.)

Levels of selenium in food commodities are a reflection of the selenium content of the soil where crops are produced and animals are raised (Reilly 1996, 2004; Arthur 1972). However, wide regional and seasonal variations in the selenium content of the food supply are expected because commercially available foods originate from widespread geographical areas within the North American continent and abroad. Selenium concentrations were determined in commercial food purchased in retail outlets in Toronto in July 1992 as part of the Total Diet Study (Dabeka 1994). One hundred and thirty-five food composites were prepared for consumption before testing for selenium. The highest selenium concentrations were determined in organ meats such as liver and kidney (1.044 mg·kg<sup>-1</sup>), nuts and seeds  $(0.635 \text{ mg}\cdot\text{kg}^{-1})$ , canned fish  $(0.413 \text{ mg}\cdot\text{kg}^{-1})$ , and white bread  $(0.410 \text{ mg} \cdot \text{kg}^{-1})$ . Fruits and vegetables were generally poor sources of selenium in the diet

Selenium sulfide is used topically to treat dandruff, seborrhoeic dermatitis of the scalp, and tinea versicolor, a common fungal infection of the skin. (MedlinePlus 1993; Health Canada 2006b). Several forms of selenium in various amounts are available in commercial mineral and vitamin supplements sold in Canada (Health Canada 2006c).

Selenium concentrations have been measured in vegetation, fish, avian and mammalian wildlife. This information is summarized in CCME (2009).

The analytical method recommended for selenium by the CCME in 1993 was US EPA Method 6010A, an inductively coupled plasma-atomic emission spectroscopy method. This method is used for the analysis of a variety of inorganic parameters in both liquid and solid phase samples. Details of this method can be found in CCME (1993).

## **Environmental Fate and Behaviour in Soil**

The environmental fate and behaviour of selenium in the environment is influenced largely by its oxidation state and the consequent differences in behaviour of its different chemical species. The oxidation state of selenium is dependent on a number of ambient environmental parameters, such as pH, Eh, and biological activity (Maier et al. 1988). The bulk of selenium in soil is the result of weathering and leaching processes, with a lesser contribution from wet and dry deposition of selenium compounds present in the atmosphere.

In soil pore water, the expected forms of selenium are the salts of selenic and selenious acids, selenates (Se<sup>+6</sup>) and selenites ( $Se^{+4}$ ), respectively. Selenates are among the most mobile selenium compounds, due to their high solubility and inability to adsorb onto soil particles (NAS 1976; Kabatas-Pendias and Pendias 2000). Selenites are less soluble than selenates (NAS 1980). Elemental selenium is essentially insoluble. In acidic, organicenriched soils, metal selenides, selenium sulphides and selenites are the predominant species. Selenides and selenium sulphide are also insoluble and tend to be immobile in soils (ATSDR 2003). In neutral, welldrained soils, sodium and potassium selenites dominate, with soluble metal selenites occurring to a lesser extent (ATSDR 2003). Selenites are typically complexed to iron oxides/hydroxides and clays in acidic and neutral soils and are of extremely low solubility in this form (Geering et al. 1968; Mikkelsen et al. 1989). In alkaline soils (pH > 7.5) that are well-oxidized, selenates are the major selenium species. As selenates are highly mobile, they are readily taken up by microorganisms in the soil or leached through the soil (Klaassen et al. 1991). Under highly reduced conditions, elemental selenium tends to dominate in soils but is of minimal bioavailability due to its low water solubility.

Selenium may be taken up by terrestrial plants when the soil environment favours the soluble species (i.e., alkaline and well-oxidized). While both selenates and selenites are accumulated by plants, selenates are more readily taken up. This may reflect the tendency of selenates to be less adsorbed to soil particles and organic matter than selenites (Banuelos and Meek 1990). Selenium uptake by plants is influenced by a number of factors including soil type, soil texture, pH, colloidal content, Eh, organic matter, clay content, soil sulphate and phosphate concentration, total level of selenium in the soil, and the capacity of plant species to accumulate selenium (i.e., accumulator or non-accumulator). For example, selenium phytoavailability generally increases at higher pH values, and decreases with increasing amounts of clay, iron oxides, organic matter, and soil sulphate (Mikkelsen et al. Mixed results have been obtained with soil 1989). phosphate concentrations (Mikkelsen et al. 1989). Although much of the total selenium present in the soil may occur in other forms, soluble selenates appear to be responsible for the majority of selenium accumulation by plants (NAS 1976). Water-soluble organic species may also be taken up by plants (Shamberger 1981).

Elemental selenium, organic, and inorganic selenium species may be methylated by soil microorganisms, with the methylated species subsequently volatilized to the

# Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health

atmosphere (Shamberger 1981; Doran 1982; Fishbein Aeromonas spp., Flavobacterium spp., and 1983). Pseudomonas spp., as well as several genera of fungi, are believed to be responsible for the methylation of elemental, organic, and inorganic selenium compounds to dimethyl selenide and dimethyl diselenide (Reamer and Zoller 1980; Zieve and Peterson 1981; Fishbein 1983). Dimethyl selenone and methyl methyl selenite may also be formed to a lesser extent. The methylation process is temperature-dependent, with significant inhibition of methylation activity occurring at lower temperatures (Chau et al. 1976; Zieve and Peterson 1981). Production of volatile methylated selenium species is also dependent on such factors as microbiological activity, moisture, time, concentrations of soluble selenium within the soil matrix and season (Zieve and Peterson 1981). Microorganisms appear to methylate organic selenium species more readily than selenates, selenites or elemental selenium, with methylation of elemental selenium occurring the least rapidly (Maier et al. 1988). Biomethylation of selenium in soils occurs rapidly as long as it is present in a soluble state, or is present in high concentrations that microorganisms enough use biomethylation as a detoxification mechanism (Reamer and Zoller 1980). The methylation of selenium in soils (and plants), its volatilization to the atmosphere, and its subsequent return to soil via wet and dry deposition processes is believed to be the major natural process through which selenium cycles in the environment (Doran 1982).

## **Behaviour and Effects in Biota**

## Soil Microbial Processes

Selenium toxicity has been demonstrated in bacteria, fungi and algae; however, data appear to suggest an essential role for selenium in procaryotic and eucaryotic cells, indicating that there may be a soil concentration below which adverse effects may result from deficiency (Janda and Fleming 1978). The lowest observed effect concentration (LOEC) identified was 198 mg Se·kg<sup>-1</sup> soil, where arylsulphatase activity was reduced in seleniumcontaining soils (Al-Khafaji and Tabatabai 1979). At 484 mg·kg<sup>-1</sup>, selenium was reported to reduce respiration in native soil microflora by 43% (Lighthart et al. 1977). At 1975 mg·kg<sup>-1</sup>, selenium was reported to reduce amidase activity as well as soil acid and alkaline phosphatase activities (Juma and Tabatabai 1977; Frankenberger and Tabatabai 1981).

### Terrestrial Plants

The most important factors influencing plant uptake of selenium are the form and concentration of selenium in the soil. In soil, the phytoavailability of selenium is several times greater for selenate (Se<sup>+6</sup>) than for selenite (Se<sup>+4</sup>), while elemental selenium is largely unavailable (Mikkelsen et al. 1989). Selenium uptake by plants is also influenced by soil properties such as pH, soil texture, organic matter, and the presence of competitive ions (Mikkelsen et al. 1989). In general, selenium phytoavailability is reduced with increasing amounts of clay, iron oxide, organic matter in soil, and decreased pH (Mikkelsen et al. 1989). Selenium is translocated to all parts of the plant, with concentrations typically greater in plant seeds than leaves, and with smaller amounts in plant stems (Olson 1978; Efroymson et al. 1997a). Plants may accumulate selenium in amounts ranging from less than  $1 \text{ mg} \cdot \text{kg}^{-1}$  plant tissue to as much as several thousand mg·kg<sup>-1</sup> plant tissue (James et al. 1989). Seleniumaccumulating plants (e.g., Astragalus, Stanleya, Haplopappus and Xylorhiza) can accumulate extremely high concentrations of selenium, up to at least 5 mg·g<sup>-1</sup> dry weight (Salisbury and Ross 1985) and have frequently poisoned livestock. Agricultural crops typically have a much lower tolerance for selenium (Mikkelsen et al. 1989). The US DOE (1998) reviewed 156 plant uptake factors (concentration in plant/ concentration in soil) for selenium from 14 studies on various grasses and crop species. Uptake factors ranged from 0.02 to 77, with the median value of 0.7 being adopted for Canadian Soil Ouality Guideline development.

Selenium may play an essential role in plant growth, but this has not yet been confirmed (Mikkelsen et al. 1989; Efroymson et al. 1997a). Selenium-accumulating plants appear to tolerate high concentrations of selenium. These plants form a variety of seleno-amino acids that are not toxic to the plants themselves, although the substitution of selenium for sulphur in amino acids and proteins may disrupt normal metabolism (Brown and Shrift 1982; Bollard 1983). Indications of selenium toxicity in plants include chlorosis, stunting, and yellowing of the leaves (Efroymson et al. 1997a). Plant tissue concentrations of selenium associated with a 10% yield reduction were summarized by Mikkelsen et al. (1989) as follows: alfalfa to  $30 \text{ mg} \cdot \text{kg}^{-1}$ tissue  $(Se^{+6})$ 25 in soil);  $(Se^{+4})$ burseem >10 mg·kg<sup>-1</sup> tissue in soil): pea/mustard/wheat  $3 \text{ mg} \cdot \text{kg}^{-1}$  tissue (Se<sup>+6</sup> in soil); wheat 10 to 15 mg·kg<sup>-1</sup> tissue (Se<sup>+4</sup> soil); and rice 2 to 67 mg·kg<sup>-1</sup> tissue (Se<sup>+6</sup> in soil).

Selenium, as Na<sub>2</sub>SeO<sub>4</sub>, was shown to reduce shoot weight in alfalfa at 1.5 mg·kg<sup>-1</sup> in soil, while 0.5 mg·kg<sup>-1</sup> in soil had no effect (Wan et al. 1988). Shoot weight of alfalfa also was reduced when grown in soil containing  $2 \text{ mg} \cdot \text{kg}^{-1}$  selenite (Se<sup>+4</sup>), with greatest reductions in soils with the lowest organic matter (Soltanpour and Workman 1980). In wheat,  $2.5 \text{ mg} \cdot \text{kg}^{-1}$  (lowest concentration tested) selenium in soil as Na<sub>2</sub>SeO<sub>3</sub> resulted in decreased biomass and yield after 50 days (Singh and Singh 1978). A reduction of shoot weight by up to 59% on sorgrass (Sorghum vulgare) seeds was observed at a selenium concentration of 1 mg·kg<sup>-1</sup> (Carlson et al. 1991). In forage cowpea (Vigna sinensis), dry matter was reduced at concentrations of 2.5 mg·kg<sup>-1</sup> when selenium was added as either elemental selenium, Na<sub>2</sub>SeO<sub>3</sub>·H<sub>2</sub>0, or H<sub>2</sub>SeO<sub>3</sub> (Singh and Singh 1979). Selenium appeared to be more toxic in the form of Na<sub>2</sub>SeO<sub>4</sub>, resulting in reduced dry matter of forage cowpea at concentrations as low as  $1 \text{ mg} \cdot \text{kg}^{-1}$  (Singh and Singh 1979).

### Terrestrial Invertebrates

Selenium appears to be capable of bioaccumulating in terrestrial invertebrate organisms. Wu et al. (1995) reported average selenium bioaccumulation factors of 44, 44, and 75 for soil-to-plants, plants-to-grasshoppers, and grasshoppers-to-praying mantis, respectively, from seven sites in a California, US reservoir area. Sample et al. (1998a) conducted a review of soil to earthworm uptake factors, and determined a literature-derived media soil to earthworm uptake factor of 0.985 for selenium. Beyer et al. (1987) observed worm selenium concentrations of 16 and  $22 \text{ mg} \cdot \text{kg}^{-1}$  in Aporrectodea tuberculata and Aporrectodea turgida, respectively, in soil that contained  $<0.1 \text{ mg}\cdot\text{kg}^{-1}$ selenium. When soil selenium concentrations were increased to  $6.7 \text{ mg} \cdot \text{kg}^{-1}$ , the earthworm selenium concentrations increased by approximately 5-fold.

The survival of the adult beetle (*Tenebrio molitor*) was reported to be reduced when transferred to a nutrient medium containing 0.125% sodium selenite (Hogan and Razniak 1991). Reproductive effects (i.e., a decrease in the number of cocoons per worm) were reported in the earthworm (*Eisenia fetida*) when exposed to 77 mg·kg<sup>-1</sup> selenium (as sodium selenite) (Fischer and Koszorus 1992).

## Livestock and Wildlife

Terrestrial mammals and birds are exposed to selenium primarily through the food chain. Selenium absorption from the gastrointestinal tract varies with the chemical form and the amount ingested (NRC 1980; Marier and Jaworski 1983). In monogastric species, selenium has been reported to be almost completely absorbed from the diet, while ruminants have a relatively low dietary selenium absorption rate (Marier and Jaworski 1983).

Selenium concentrations in animal tissues tend to reflect selenium concentrations, especially dietary when provided by natural dietary ingredients as compared to selenate or selenite (NRC 1980; Heinz et al. 1989; Stowesand et al. 1990). Sample et al. (1998b) conducted review of soil-to-small mammal selenium а bioconcentration factors, and derived a final mean and median soil-to-small mammal uptake factor of 0.35 and 0.16, respectively. Santolo et al. (1999) estimated selenium accumulation factors in American kestrel from diet-to-blood of 1.0 and from diet-to-eggs of 2.2.

Selenium is nutritionally required by animals in small amounts (e.g., ruminants, chickens, quail, mice, swine) (NAS 1980), but can become toxic in slightly greater amounts (Lemly 1997). For livestock, the threat of selenium deficiency is considered by some researchers to be a greater threat than selenium toxicity (Eisler 1985). Selenium dietary requirements for domestic animals typically range from 0.1 to 0.3 mg·kg<sup>-1</sup> in dry matter (NAS 1980).

Sheep appear to be the most sensitive mammalian species to selenium intoxication. A chronic oral LOAEL of 0.08 mg·kg<sup>-1</sup> bw·day<sup>-1</sup> was reported for sheep orally administered selenium in the diet for one year (Puls 1994). Cows exposed to various levels of selenomethionine through their diet for 120 days developed tissue lesions at a dose of 0.8 mg·kg<sup>-1</sup> bw·day<sup>-1</sup> (O'Toole and Raisbeck 1994). Similarly, pigs fed diets with various levels of sodium selenite for 5 weeks demonstrated reduced weight gain and food intake at doses as low as approximately 0.8 mg·kg<sup>-1</sup> bw·day<sup>-1</sup>, i.e., an exposure concentration of 8 mg Se·kg<sup>-1</sup> food (Goehring et al. 1984). A maximum tolerable dietary level for selenium of 2 mg·kg<sup>-1</sup> was reported to be protective of domestic animals (NAS 1980), which corresponds to a daily dose of 0.08 mg·kg<sup>-1</sup> bw·day<sup>-1</sup> for cattle.

Selenium has been associated with embryonic mortality and teratogenesis in birds, particularly in the Western US (Clayton and Clayton 1994). However, the evaluation of selenium toxicity is complicated by its occurrence in many different chemical forms which differ greatly in their toxicity to birds (Heinz 1996). Organic selenium compounds, particularly selenomethionine, have been shown to be highly toxic to birds.

In a study where mallard ducks were fed a diet supplemented with selenium (as seleno-DL-methionine), no reproductive effects were reported at 3.5 mg·kg<sup>-1</sup> while the lowest effect level was 7 mg·kg<sup>-1</sup> (Stanley et al. 1996). Suppression of the immune system was reported to occur in mallard ducks at 2.2 mg $\cdot$ L<sup>-1</sup> selenomethionine in drinking water (Fairbrother and Fowles 1990). Heinz et al. (1987) observed no adverse effects in ducks orally administered 5 mg $\cdot$ kg<sup>-1</sup> selenium as sodium selenite for 78 days. No adverse effects were seen in a study in which mallard ducks were fed diets containing selenium concentrations of 1, 2, or 4 mg·kg<sup>-1</sup> as selenomethionine for 100 days (Heinz et al. 1989). In a 6-week exposure of newly hatched mallard ducklings to either sodium selenite or selenomethionine, adverse effects on mortality were observed at 40 mg kg<sup>-1</sup> of selenium in the diet for both forms (Heinz et al. 1988). Similarly, both forms of selenium resulted in reduced food consumption and reduced body weight at a dietary concentration of 20 mg·kg<sup>-1</sup> (Heinz et al. 1988). In another study with mallard ducklings, survival, body weight, and food consumption were all affected at a dietary exposure of 30  $mg \cdot kg^{-1}$  for two weeks, with selenium added to food as either seleno-DL-methionine or seleno-L-methionine (Heinz et al. 1996). Seleno-L-methionine appeared to be more toxic than seleno-DL-methionine (Heinz et al. 1996). Mallard ducks have demonstrated food avoidance at dietary concentrations of selenium as low as 10 mg·kg<sup>-1</sup> (Heinz and Sanderson 1990).

Screech owls exposed to 30 mg·kg<sup>-1</sup> (dry weight) selenomethionine in their diet for almost 14 weeks through reproduction experienced reductions of 38 and 88% in egg production and hatchability, respectively, as well as a 100% reduction in nestling survival (Wiemever and Hoffman 1996). However, owls exposed to 10 mg·kg<sup>-1</sup> (dry weight) selenomethionine in their diet had no adverse effects on reproduction. Black-crowned night-herons exposed to dietary concentrations of 10 mg·kg<sup>-1</sup> selenomethionine for 94 days through reproduction had no adverse effects on reproduction (Smith et al. 1988). In American kestrels exposed to seleno-L-methionine through their diet for 11 weeks, concentrations of 9 mg·kg<sup>-1</sup> dw had no effect on body weight and did not produce any signs of toxicity in adult kestrels (Yamamoto et al. 1998). Dietary exposure to 12 mg·kg<sup>-1</sup> dw over 11 weeks also had no adverse effects on various reproductive measures in kestrels (Santolo et al. 1999).

In domestic chickens, dietary concentrations as low as  $5 \text{ mg} \cdot \text{kg}^{-1}$ , administered for up to 28 weeks, were observed to cause wiry feathers in hatched chicks, as well as increased chick mortality and decreased hatchability (Moxon 1937; Ort and Latshaw 1978). This concentration corresponds to a daily dose of 0.34 mg \cdot \text{kg}^{-1} bw  $\cdot \text{day}^{-1}$ .

## Human and Experimental Animal Health Effects

For the general population, exposure to selenium is mainly through ingestion of food and food supplements (IOM 2000) Selenium intake from the diet was estimated using food concentration data from the 1992 Toronto Total Diet Survey (Dabeka 1994) and using food consumption rates for various age groups of Canadians (Health Canada 1994). Total daily intakes of selenium by all routes of exposure (food, water, air, and soil/dust) were calculated by age class. The estimated daily intakes (EDI) for adults, teenagers, school aged children, toddlers and infants were 0.1357, 0.1326, 0.1129, 0.0693, and 0.0135 mg Se·day<sup>-1</sup>, respectively, which correspond on a body weight basis to 0.00192, 0.00222, 0.00343, 0.00420 and 0.00165 mg Se kg bw<sup>-1</sup> day<sup>-1</sup>, respectively. The main source of selenium exposure comes from the diet constituting more than 99% of the EDI for all age classes.

Oral absorption of selenium is extensive (ATSDR 2003). Several selenium compounds appear to be readily absorbed from the human gastrointestinal tract, with the degree of absorption dependent on the chemical form (e.g., organic, inorganic), physical state (e.g., solid, solution), and the dosing regimen (ATSDR 2003). Absorption is usually not regulated by the selenium status and thus, does not appear to be under homeostatic control; rather, selenium levels are dependent on excretory pathways (IOM 2000; WHO/FAO 2004). Selenium is more readily available from foods of plant origin (greater than 85%) than those from animal origin (on average 15%). Although fish contains relatively high amounts of selenium, the bioavailability of selenium from this food source appears to be relatively low, often less than 25% (Combs 2001; Navarro-Alarcón and López-Martínez 2000). The bioavailability of selenium from a mixed diet is estimated to be between 60 and 80% (Daniels 1996).

Studies in humans regarding the absorption of selenium following inhalation exposure are limited to occupational studies. Selenium has been measured in the urine of workers occupationally exposed to selenium; however, an estimate of the extent or rate of absorption was not given (ATSDR 2003).

No dermal absorption of selenomethionine was observed in women tested with a maximum dose of  $0.0029 \text{ mg}\cdot\text{kg}^{-1}$ selenium in a 0.05% L-selenomethionine lotion (Burke *et al.* 1992). However, as the concentrations tested were relatively low, the authors concluded that dermal absorption may occur at higher doses. Users of shampoos containing 1% selenium disulfide did not experience dermal uptake (ATSDR 2003; NAS 1980).

The route of exposure appears to have no significant impact on the distribution of selenium within the body (ATSDR 2003). In addition, similar distribution patterns for both inorganic and organic forms of selenium have been reported in most studies, with selenomethionine and other organic selenium compounds retained in tissues at higher concentrations than inorganic selenium compounds (ATSDR 2003).

The metabolism of selenium involves pathways for the incorporation of selenium into selenium-dependent enzymes, as well as pathways for the elimination of selenium from the body (ATSDR 2003). Excretion of selenium metabolites and end-products occurs primarily through the urinary route.

Some 20 mammalian functional selenoproteins have been characterised so far (Arthur et al. 2003). There are three "families" of selenoenzymes: the glutathione peroxidases deiodinases (GSPxs). iodothyronine (IDs) and thioredoxin reductases (TRs) (Reilly 2004). The four known forms of GSPxs operate in different cellular compartments to protect cell membranes against oxidative stress. The IDs interact with iodine to prevent abnormal thyroid hormone metabolism and the TRs participate in the reduction of nucleotides and the binding of transcription factors in DNA and in numerous key cellular activities such as cell growth, inhibition of cell death, regeneration of proteins inactivated by oxidation and regulation of redox reactions (Rayman 2000; WHO/FAO 2004; Reilly 2004; 2006).

The first evidence for the essentiality of selenium in human diets came from the association of Keshan disease (a form of cardiomyopathy endemic to certain areas of China diagnosed in children and women of childbearing age) and Kashin-Beck disease (osteoarthropathy endemic to Eastern Siberia and other districts of Russia, China, Tibet, Japan, and North Korea), where diet is deficient in selenium (Levander 1986; WHO 1996; Reilly 2004, 2006).

Selenium is a paradoxical element in that there is a small margin of safety between levels of selenium that

constitute dietary deficiency and those that result in toxicity (Reilly 2004, 2006). The Food and Nutrition Board of the Institute of Medicine of the National Academies with the participation of Health Canada established Dietary Reference Intakes (DRI) for Vitamin C, Vitamin E, Selenium and Carotenoids (IOM 2000). DRIs replace the (RDAs) in the U.S. and Recommended Nutrient Intakes (RNIs) in Canada (IOM 2000; Health Canada 2003). Until the development of DRIs, Health Canada had not established RNIs for all the known essential trace elements, selenium being one of them.

In the context of setting DRIs, considerations were given to the required daily intake of selenium to reduce the risk of chronic disease, to maintain homeostasis based on biochemical indicators and to replenish daily losses. DRIs consider bioavailabilty as well as all nutrient and dietary interactions (Mertz 1995; WHO 2002; IOM 2000, 2001; amongst others). For selenium and other essential trace elements, there is a safe range of intakes between deficiency and toxicity called Acceptable Range of Oral Intake (AROI) from various sources (IOM 2000). Four DRI values within the AROI include the following, as defined by IOM (2000) for selenium and other essential trace elements: the Recommended Dietary Allowance (RDA), the Adequate Intake (AI), the Estimated Average Requirement (EAR) and the Tolerable Upper Intake Level (UL). The DRIs for selenium are presented in Health Canada (2003).

The specific mechanisms of toxicity for selenium and selenium compounds are not well understood. However, it is generally believed that only the soluble, readily absorbed, forms of selenium are capable of causing toxicity (ATSDR 2003). A suggested toxic mechanism is that selenium inactivates the sulphydryl enzymes that are necessary for oxidative reactions during respiration (Lombeck et al. 1987; Mack 1990). In addition, evidence that selenium can replace sulphur in biological macromolecules (especially when the Se:S ratio is high), is thought to be a possible mechanism for chronic toxicity (Stadtman 1983; Tarantal et al. 1991). A number of studies have shown that soluble selenium compounds are the most toxic and that these compounds tend to exert a cumulative toxicity, with lower doses causing death when administered over longer periods of time (ATSDR 2003).

Details of the human and mammalian toxicology of selenium are discussed elsewhere (CCME 2009). In general, people exposed to very high levels of selenium through their diet have reported dizziness, fatigue, irritation, collection of fluid in the lungs, and severe bronchitis. The exact levels at which these effects occur are not known. Upon contact with skin, selenium compounds have caused rashes, swelling, and pain (ATSDR 2003).

While some animal studies have shown reproductive and developmental effects from oral selenium exposure (e.g., Rosenfeld and Beath 1954; Schroeder and Mitchener 1971), the mutagenicity and genotoxicity database on selenium compounds is inconclusive, with many studies producing conflicting results for a number of selenium compounds (Environment Canada 2002). There is no human evidence of reproductive effects, teratogenesis, or developmental abnormalities. In addition, in humans, the majority of studies have demonstrated either an inverse or no association between environmental selenium concentrations and cancer incidence and/or cancer mortality rates (ATSDR 2003; US EPA 1991).

Based on the results of the available epidemiology, animal, mutagenicity, and genotoxicity data, selenium compounds have not been classifiable as carcinogenic to humans, according to the International Agency for Research on Cancer (IARC 1987). Only one selenium compound, selenium sulphide, has been shown to be carcinogenic in animal studies; thus it has been designated a probable human carcinogen by the IARC and the US EPA. However, this compound is not present in soils, foods or other environmental media to any significant extent; thus human environmental exposure to selenium sulphide would be minimal (CCME 2009).

Health Canada (1992) did not derive a TDI (Tolerable Daily Intake), for selenium as a basis for setting the Guideline for Canadian Drinking Water Quality for selenium (MAC of  $0.10 \text{ mg} \cdot \text{L}^{-1}$ ). The IOM (2000) developed Tolerable Upper Intake Levels (ULs) for selenium applicable to various life group stages. The "UL is the highest level of daily nutrient intake that is likely to pose no risk of adverse healthy effects in almost all individuals" (IOM 2000). Like RfDs (reference doses) or TDIs, ULs are derived using well established principles of the risk assessment methodology using various data sources such as epidemiological studies with excessive intake of essential trace elements, clinical trials and experimental studies (WHO 2002). Adverse health effects of end-points from excessive nutrient intakes such as a NOAEL and/or a LOAEL are identified and used for the derivation of ULs for chronic daily intake of essential trace elements. Uncertainty factors (UFs) are applied to NOAELs and/or LOAELs in the calculation of ULs (WHO 2002). Even though these UFs tend to be lower than those used in TDI or RfD derivations, usually less than 10, because of the availability of reliable human data

(Becking 1998; Dourson and Erdreich 2001; Munro 1999), they remain fully protective of human health (Mertz 1995). ULs consider risks from both nutrient deficiencies and toxicity and the variability between individuals (WHO 2002). The use of large UFs could conceivably lead to a reference intake potentially associated with nutritional deficiencies (Munro 2006).

ULs for selenium are applicable to selenium intake via food and supplements, in both organic and inorganic forms of the element. The IOM (2000) did not, however, take into account intake from drinking water in the UL for this element because the Institute considered this exposure pathway not to be significant.

The ULs were derived on a re-examination of Chinese individuals recovering from selenosis (Yang and Zhou 1994). These individuals had been studied in an earlier epidemiological study investigating the occurrence of selenosis in populations with a range of selenium intakes due to selenium content of locally-grown foods. At that time, blood selenium levels were shown to reflect observed clinical signs of selenosis (i.e., garlic odour breath, thickened and brittle nails, hair and nail loss, reduced hemoglobin, mottled teeth, skin lesions, limb pain, and peripheral anesthesia (Yang et al. 1989). The IOM (2000) selected a NOAEL of 0.800 mg·day<sup>-1</sup>, a level associated with recovery, deemed protective of both U.S. and Canadian populations. An uncertainty factor (UF) of 2 was selected to protect sensitive individuals, the toxic effect being not severe but not necessarily being completely reversible. A UL of 0.400 mg·day<sup>-1</sup> was established for adults, aged 19 years and older, for pregnant and lactating females, as well as for older children aged 14 to 18 years (IOM 2000). For infants (birth to 6 months) and older infants (7 to 12 months), the ULs were set at 0.045 and 0.060 mg·day<sup>-1</sup>. ULs of 0.090, 0.150, and 0.280 mg day<sup>-1</sup> were established for children aged 1 to 3 years, 4 to 8 years and 9 to 13 years (IOM 2000). The UL for the toddler, aged 7 months to 4 years, as defined in the CCME (2006) protocol was recalculated to 0.103mg day<sup>-1</sup> as a duration weighted average. On a body weight basis, the ULs for adults, teenagers, school aged children, toddlers and infants are calculated to be 0.0057, 0.0062, 0.0063, 0.0062, and 0.0055 mg kg bw <sup>1</sup>·day<sup>-1</sup>, respectively.

#### **Guidelines Derivation**

Canadian soil quality guidelines are derived for different land uses following the process outlined in CCME (2006) using different receptors and exposure scenarios for each land use (Table 1). Detailed derivations for selenium soil quality guidelines are provided elsewhere (CCME 2009).

#### Soil Quality Guidelines for Environmental Health

The environmental soil quality guidelines (SQG<sub>E</sub>) are based on soil contact using data from toxicity studies on plants and invertebrates and in the case of agricultural land, include soil and food ingestion toxicity data for mammalian and avian species. To provide a broader scope of protection, a nutrient and energy cycling check for soil microbes and an off-site migration check for commercial and industrial land uses are also calculated. The off-site migration check is to ensure that commercial and industrial sites do not contaminate adjacent agricultural or residential/parkland lands. For soluble organic substances, soil concentrations are also calculated that will prevent contamination of groundwater for the protection of freshwater life, irrigation, and livestock watering.

The lowest of the various applicable soil quality guidelines and check values for each land use is recommended as the  $SQG_E$ .

In the case of selenium, soil contact guidelines were derived using the LOEC method. Data were insufficient to derive a nutrient and energy cycling check for all land uses. However, available data suggest that these soil processes are less sensitive than other ecological receptors. The soil and food ingestion guideline for agricultural uses was not as low as the soil contact guideline. Thus, the soil contact guidelines are recommended as the SQG<sub>E</sub> for agricultural and residential/parkland land uses (See Table 2). Data were sufficient to calculate an off-site migration check value for commercial and industrial land uses. However, this check value was not lower than the soil contact guideline; therefore, the soil contact guideline value is recommended as the SQG<sub>E</sub> for commercial and industrial land use (See Table 2). Guidelines for protection of groundwater were not calculated for selenium because the models are not applicable to inorganic substances.

#### Soil Quality Guidelines for Human Health

For potential risks posed at federal contaminated sites in Canada by exposure to contaminants that are also considered to be essential trace elements, Health Canada recommends the use of 'Tolerable Upper Intake Levels' (ULs) from IOM (2000; 2001) as the reference exposure

### Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health

levels for risk assessment. Since selenium is an essential trace element for human health and selenium compounds do not appear to be carcinogenic, the ULs from IOM (2000) for the most sensitive receptor designated for a land use (i.e., a toddler for agricultural, residential/parkland and commercial lands, and an adult for industrial lands) are proposed for use in the derivation of the human health soil quality guidelines for selenium).

The CCME soil quality protocol (CCME 2006) recommends the application of various check mechanisms, when relevant, in order to provide a broader scope of protection. The lowest of the soil ingestion guideline, the off-site migration check, and the guideline for the protection of potable water is recommended as the SQG<sub>HH</sub> (Table 2). In the case of selenium, the only check mechanism for which sufficient data were available to calculate a check value was the off-site migration check for commercial and industrial land use.

The human health soil quality guidelines (SQG<sub>HH</sub>) for selenium are based on the direct contact pathway for agricultural, residential/parkland and commercial land uses (Table 2). The off-site migration check value was lower than the calculated human health soil quality guideline for industrial land use, based on the direct contact pathway. Therefore, the off-site migration check value is recommended as the SQG<sub>HH</sub> for industrial land uses (Table 2).

#### Soil Quality Guidelines for Selenium

The soil quality guidelines are intended to be protective of both environmental and human health, and are therefore the lower of the  $SQG_E$  and  $SQG_{HH}$  guidelines. Where sufficient and adequate data exist for both, the CCME interim soil quality criteria can be superseded (Table 1).

For agricultural, residential/parkland, commercial and industrial land uses, the soil quality guidelines are the soil concentrations calculated for the  $SQG_E$  which are based on the soil contact guidelines.

Because there are sufficient data to derive both the  $SQG_E$ and the  $SQG_{HH}$ , the soil quality guidelines represent fully integrated *de novo* guidelines for each land use, derived according to the protocol (CCME 2006). The interim soil quality criteria for selenium (CCME 1991), and previous soil quality guidelines derived for selenium (EC 2001) are replaced by the SQGs recommended here.

For specific locations with unusually high natural

background concentrations that still exceed these guidelines, jurisdictions have the option to set sitespecific guidelines that consider the unique geological characteristics of the particular locations (CCME 2006). CCME (1996) provides guidance on potential modifications to the final recommended soil quality guidelines when setting site-specific objectives.

#### References

Alberta Agriculture, Food. And Rural Development. 2002. Agricultural impacts on groundwater quality in the irrigated areas of Alberta. Available at:

http://www1.agric.gov.ab.ca/\$department/deptdocs.nsf/all/irr4452

- Al-Khafaji, A.A., and M.A. Tabatabai. 1979. Effects of trace elements on arysulfatase activity in soils. Soil Sci. 127(3):129-133. (Cited In Efroymson et al. 1997b).
- Andersson, E. 2005. Hazardous substances in electrical and electronic equipment (EEE) – Expanding the scope of the RoHS directive. Document prepared for the Swedish Chemicals Inspectorate at the Department of Risk Reduction in Sundybberg as part of a course for the Department of Applied Environmental Science at Göteborg University, Sweden. Available at http://forum.europa.eu.int/Public/irc/env/weee\_2008/library?l=/charact eristics/hazardous\_substances/\_EN\_10\_&a=d
- Arthur, D. 1972. Selenium content of Canadian foods. Can. Inst. Food Sci. Technol. J. 5(3):165-169.
- Arthur, J.R., R.C. Mackenzie, and G.J. Beckett. 2003. Selenium on the immune function. J. Nutr. 133: 1457S-1459S.
- ATSDR. (Agency for Toxic Substances and Disease Registry). 2003. Toxicological profile for selenium. Prepared for US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta, GA.
- Banuelos, G.S., and D.W. Meek. 1990. Accumulation of selenium in plants grown on selenium-treated soil. J. Environ. Quality. 19(4):772-777. (Cited in ATSDR 2003).
- Becking, G.C. 1998. The effect of essentiality on risk assessment. Biol. Trace Elem. Res. 66(1-3):423-38.
- Beyer, W.N., G. Hensler, and J. Moore. 1987. Relation of pH and other soil variables to concentrations of Pb, Cu, Zn, Cd and Se in earthworms. Pedobiologia. 30:167-172.
- Bollard, E.G. 1983. Involvement of unusual elements in plant growth and nutrition. In: Lauchli, A. and Bieleski, R.L. (eds.). pp. 695-744. (Cited in Salisbury and Ross 1985).
- Brown, T.A., and A. Shrift. 1982. Selenium: toxicity and tolerance in higher plants. Biol. Rev. 57:59-84. (Cited in Mikkelsen et al. 1989).
- Burke, K.E., R.G. Burford, G.F. Combs Jr, I.W. French, and D.R. Skeffington.1992. The effect of topical L-selenomethionine on minimal erythema dose of ultraviolet irradiation in humans. Photodermatol Photoimmunol Photomed 9(2):52-57. (Cited In ATSDR 2003).
- Carlson, C.L., D.C. Adriano, and P.M. Dixon. 1991. Effects of soilapplied selenium on the growth and selenium content of forage species. J. Environ. Qual. 20: 363-368.
- CCME. (Canadian Council of Ministers of the Environment). 1991. Interim Canadian environmental quality criteria for contaminated sites. CCME, Winnipeg.
- 1993. Guidance manual on sampling, analysis, and data management for contaminated sites. Volume II. Analytical method summaries. CCME, Winnipeg.

	Land use				
	Agricultural	Residential/ parkland	Commercial	Industrial	
Guideline	1 <sup>a</sup>	1 <sup>a</sup>	<b>2.9</b> <sup>a</sup>	<b>2.9</b> <sup>a</sup>	
Human health guidelines/check values					
SQG <sub>HH</sub>	80 <sup>b</sup>	80 <sup>b</sup>	125 <sup>b</sup>	1135 <sup>b</sup>	
Direct contact (SQG <sub>DH</sub> )	80	80	125	4050	
Protection of indoor air quality - basement (SQG <sub>IAQ</sub> )	NC <sup>c</sup>	NC <sup>c</sup>	NC <sup>c</sup>	NC <sup>c</sup>	
Protection of indoor air quality - slab-on-grade (SQG <sub>IAQ</sub> )	NC <sup>c</sup>	NC <sup>c</sup>	NC <sup>c</sup>	NC <sup>c</sup>	
Off-site migration check (SQG <sub>OM-HH</sub> )	_	_	1135	1135	
Protection of potable water (SQG <sub>PW</sub> )	$NC^d$	$NC^d$	$NC^d$	$NC^d$	
Produce, meat and milk check (SQG <sub>FI</sub> )	NC <sup>e</sup>	NC <sup>e</sup>		_	
Environmental health guidelines/check values					
SQG <sub>E</sub>	$1^{\mathrm{f}}$	$1^{\mathrm{f}}$	$2.9^{\mathrm{f}}$	2.9 <sup>f</sup>	
Soil contact	1	1	2.9	2.9	
Soil contact confidence $rank^h$	F	F	F	F	
Soil and food ingestion	4.5 <sup>i</sup>	_	_		
Protection of freshwater life	$NC^d$	$NC^d$	$NC^d$	$NC^d$	
Livestock watering	$NC^d$	$NC^d$	$NC^d$	$NC^d$	
Irrigation water	$NC^d$	$NC^d$	$NC^d$	$NC^d$	
Nutrient and energy cycling check	$\mathbf{NC}^{\mathrm{g}}$	NC <sup>g</sup>	NC <sup>g</sup>	NC <sup>g</sup>	
Off-site migration check	—	—	5.0	5.0	
Interim soil quality criteria (CCME 1991)	2	3	10	10	
Soil Quality Guideline (EC 2001)	1	1	3.9	3.9	

Table 2. Soil quality guidelines and check values for selenium (mg·kg<sup>-1</sup>).

Notes:

 $SQG_{HH}$  = soil quality guideline for human health;  $SQG_E$  = soil quality guideline for environmental health; NC = not calculated; — The dashes indicate guidelines/check values that are not part of the exposure scenario for that land use and therefore are not calculated.

<sup>a</sup> Data are sufficient and adequate to calculate an  $SQG_{HH}$  and an  $SQG_E$  for this land use. Therefore the soil quality guideline represents a fully integrated de novo guideline for this land use, derived in accordance with the Protocol (CCME 2006). The corresponding interim soil quality criterion (CCME 1991) is superseded by the adoption of the soil quality guideline.

<sup>b</sup> The SQG<sub>HH</sub> is the lowest of the human health guidelines and check values.

<sup>c</sup> The inhalation of indoor air check applies to volatile organic compounds and is not calculated for inorganic contaminants.

<sup>d</sup> The groundwater check applies to organic compounds and thus is not calculated for inorganic contaminants. Concerns about inorganic contaminants should be addressed on a site-specific basis.

<sup>e</sup> The produce, meat and milk check applies to organic compounds and thus is not calculated for inorganic contaminants. Concerns about inorganic contaminants should be addressed on a site-specific basis.

<sup>f</sup>The SQG<sub>E</sub> is the lowest of the environmental health guidelines and check values.

<sup>g</sup>Data are insufficient/inadequate to calculate these environmental guidelines/check values.

<sup>h</sup> For an explanation of the soil contact confidence rank, refer to CCME (2006).

<sup>i</sup> This value may not be protective if livestock or wildlife are consuming plants that hyperaccumulate selenium from the soil.

- —. 1996. Guidance manual for developing site-specific soil quality remediation objectives for contaminated sites in Canada. CCME, Winnipeg. [Reprinted in Canadian environmental quality guidelines, Chapter 7. Canadian Council of Ministers of the Environment Winnipeg]
- 2006. A protocol for the derivation of environmental and human health soil quality guidelines. CCME, Winnipeg. [A summary of the protocol appears in Canadian environmental quality guidelines, Chapter 7, Canadian Council of Ministers of the Environment, 2006, Winnipeg.]
- ——. 2009. Canadian Soil Quality Guidelines: Selenium. Environmental and Human Health. Scientific Criteria Document. Canadian Council of Ministers of the Environment, Winnipeg.
- CCREM. (Canadian Council of Resource and Environment Ministers). 1987. Canadian water quality guidelines. Prepared by the Task Force on Water Quality Guidelines.
- Chau, Y.K., P.T.S. Wong, B.A. Silverberg, P.L. Luxon, and G.A. Bengert. 1976. Methylation of selenium in the aquatic environment. Science. 192:1130-31.
- Clayton, G.D., and F.E. Clayton. 1994. Patty's Industrial Hygiene and Toxicology. Fourth Edition. Volume II, Part A. John Wiley & Sons, Inc. Toronto.
- Combs, G.F. Jr. 2001. Selenium in global food systems. Br. J. Nutr. 85: 517-547.
- Dabeka, R.W. 1994. Unpublished report on selenium and iodine levels in total diet samples. September 17, 1994. Food Research Division, Health Canada, Ottawa, Ontario
- Daniels, L.A. 1996. Selenium metabolism and bioavailability. Biol. Trace Elem. Res. 54:185-199.
- Dann, T. 2004. Personal communication from Tom Dann, Head, Air Toxics, Analysis and Air Quality, Environmental Toxicology Centre, Science and Technology Branch, Environment Canada. April 15, 2004
- Doran, J.W. 1982. Microorganisms and the biological cycling of selenium. Adv. Microbial. Ecol. 6:1-32. (Cited in ATSDR 2003.)
- Dourson, M. L., and L.S. Erdreich. 2001. Using human data to develop risk values. Hum. Ecol. Risk Assess. 7(6): 1583-1592
- EC. (Environment Canada). 2001. Canadian Soil Quality Guidelines for Selenium. Scientific Supporting Document. National Guidelines and Standards Office. Environmental Quality Branch, Environment Canada. Ottawa.
- Efroymson, R.A., M.E. Will, G.W. Suter II, and A.C. Wooten. 1997a. Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision. Prepared for the US Department of Environment and Energy. Oak Ridge National Laboratory. ES/ER/TM-85/R3.
- Efroymson, R.A., M.E. Will, and G.W. Suter II. 1997b. Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Soil Litter Invertebrates and Heterotrophic Process: 1997 Revision. Prepared for the US Department of Environment and Energy. Oak Ridge National Laboratory. ES/ER/TM-126/R2.
- Eisler, R. 1985. Selenium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Patuxent Wildlife Research Centre. US Fish and Wildlife Services. Biological Report 85(1.5), Contaminant Hazard Review Report No. 5.
- Fairbrother, A., and J. Fowles. 1990. Subchronic effects of sodium selenite and selenomethionine on several immune functions in mallards. Arch. Environ. Contam. Toxicol. 19:836-844.
- Fischer, E., and L. Koszorus. 1992. Sublethal effects, accumulation capacities and elimination rates of As, Hg, and Se in the manure worm, *Eisenia fetida* (Oligochaeta, Lumbricidae). Pedobiologia. 36:172-178. (Cited in Efroymson et al. 1997b).
- Fishbein, L. 1983. Environmental selenium and its significance. Fundam. Appl. Toxicol. 3:411-419.

- Frankenberger, W.T., and M.A. Tabatabai. 1981. Amidase activity in soils: IV. Effects of trace elements and pesticides. Soil Sci. Soc. Am. J. 45:1120-1125. (Cited in Efroymson et al. 1997b).
- Garrett, R.G., Regional and Biogeochemistry Research, Geogical Survey of Canada, Natural Ressources Canada, pers.com., January 4, 2005.
- George, M.W. 2004. Minerals Yearbook 2004: Selenium and Tellurium. U.S. Geological Survey. Available at: http://minerals.usgs.gov/minerals/pubs/commodity/selenium/selenmyb 04.pdf
- Geering, H.R., E.E. Cary, L.H.P. Jones, and W.H. Allaway. 1968. Solubility and redox criteria for the possible forms of selenium in soils. Soil Science Society of America Proceedings. 32:35-40. (Cited in ATSDR 2003). Gizyn, W.I. 1994. Windsor Air Quality Study: Soil and Garden Produce Survey Results. Phytotoxicology Section, Standards Development Branch, Ontario Ministry of Environment and Energy.
- Gizyn, W.I. 1994. Windsor Air Quality Study: Soil and Garden Produce Survey Results. Phytotoxicology Section, Standards Development Branch, Ontario Ministry of Environment and Energy.
- Goehring, T.B., I.S. Palmer, O.E. Olson, G.W. Libal, and R.C. Wahlstrom. 1984. Toxic effects of selenium on growing swine fed corn-soybean meal diets. J. Animal Sci. 59(3): 733-737.
- Health Canada. 1992. Guidelines for Canadian Drinking Water Quality Supporting document for selenium. April 1979 (updated 1992).
- ——. 1994. Human health risk assessment for priority substances. Priority substances list assessment report. Cat. No. En40-215/41E. Ottawa.
- ———. 1996. Guidelines for Canadian Drinking Water Quality, Sixth Edition - Supporting Documentation: Selenium. Prepared by the Federal-Provincial Subcommittee on Drinking Water of the Federal-Provincial Committee on Environmental and Occupational Health.
- 2003. Dietary Reference Intakes. Cat. H44-49/2003E-HTML ISBN 0-662-34958-X. Available at: <u>http://www.hc-sc.gc.ca/fn-an/nutrition/reference/dri\_using-util\_anref\_e.html</u>
- 2006a. Guidelines for Drinking Water Quality Summary Table Water Quality – Summary Table. Published by Health Canada. 2006a. Guidelines for Drinking Health Canada on behalf of the Federal-Provincial-Territorial Committee on Drinking Water of the Federal-Provincial-Territorial Committee on Health and the Environment. March 2006.
- —\_\_\_\_.2006b. Monograph Anti-dandruff products. Available at:<u>http://www.hc-sc.gc.ca/dhp-mps/prodnatur/applications/licen-</u> prod/monograph/mono\_antidandruff\_anitpelliculaire\_e.html
- 2006c. Drug Products Database (DPD). DPD online query for active ingredient"selenium", last modified: 2006-01-11. Available at: <u>http://hc-sc.gc.ca/dhp-mps/prodpharma/databasdon/index\_e.html</u>
- Haluschak, P., R.G. Eilers, G.F. Mills., and S. Grift. 1998. Status of Selected Trace Elements in Agricultural Soils of Southern Manitoba. Technical Report 1998-6E, Land Resource Unit, Brandon Research Centre, Research Branch, Agriculture and Agri-Food Canada. April, 1998.
- Heinz, G.H., D.J. Hoffman, A.J. Krynitsky, and D.M.G. Weller. 1987. Reproduction in mallards fed selenium. Environ. Toxicol. Chem. 6:423-433. (Cited in Sample et al. 1996).
- Heinz, G.H., D.J. Hoffman, and L.G. Gold. 1988. Toxicity of organic and inorganic selenium to mallard ducklings. Arch. Environ. Contam. Toxicol. 17: 561-568.
- Heinz, G.H., D.J. Hoffman, and L.G. Gold. 1989. Impaired reproduction of mallards fed an organic form of selenium. J. Wildl. Mgmt. 53:418-428. (Cited in Sample et al. 1996).
- Heinz, G.H. and C.J. Sanderson. 1990. Avoidance of selenium-treated food by mallards. Environ. Toxicol. Chem. 9: 1155-1158.

- Heinz, G.H. 1996. Selenium in Birds. In: W.N. Beyer and G.H. Heinz (eds.). Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. CRC Press: Washington.
- Heinz, G.H., D.J. Hoffman, and L.J. LeCaptain. 1996. Toxicity of seleno-L-methionine, seleno-DL-methionine, high selenium wheat, and selenized yeast to mallard ducklings. Arch. Environ. Contam. Toxicol. 30: 93-99.
- Hogan, G.R., and H.G. Razniak. 1991. Selenium-induced mortality and tissue distribution studies in *Tenebrio molitor* (Coleoptera: Tenebrionidae). Environmental Entomology. 20(3):790-794.
- IARC (International Agency for Research on Cancer). 1987. Selenium and selenium compounds. Monograph volume 9, supplement 7. Last updated 21 March 1998. Available at: http://monographs.iarc.fr/ENG/Monographs/vol9/volume9.pdf

IOM (Institute of Medicine). 2000. Dietary reference intakes for vitamin C, vitamin E, selenium and carotenoids. Panel on Dietary

- Antioxidants and Related Compounds, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of DRIs, Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Food and Nutrition Board of the Institute of Medicine of the National Academies. Washington, DC: National Academy Press, 2000.
- ——.2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, manganese, Molybdenum, Nickel, Silicon, Vanadium and Zinc. Panel on Dietary Antioxidants and Related Compounds, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of DRIs, Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Food and Nutrition Board of the Institute of Medicine of the National Academies. Washington, DC: National Academy Press, 2001.
- James, L.F., K.E. Panter, H.F. Mayland, M.R. Miller, and D.C. Baker. 1989. Selenium Poisoning in Livestock: A Review and Progress. Selenium in Agriculture and the Environment. Special Publication. No. 23. pp 123-131.
- Janda, J.M. and R.W. Fleming. 1978. Effect of selenate toxicity on soil mycoflora. J. Environ. Sci. Health. 13(9):697-706.
- Juma, N.G., and M.A. Tabatabai. 1977. Effects of trace elements on phosphatase activity in soils. Soil Sci. Soc. Am. J. 41:343-346. (Cited in Efroymson et al. 1997b).
- Kabatas-Pendias, A., and H. Pendias. 2000. Trace elements in soils and plants. Third edition. Boca Raton, FL: CRC Press.
- Klaassen, C.D., M.O. Amdur, and J.E. Doull. (eds.). 1991. Casarett and Doull's Toxicology, The Basic Science of Poisons. 4<sup>th</sup> Edition. New York, NY: MacMillan Publishing Company.
- Lemly, A.D. 1997. Environmental implication of excessive selenium: A review. Biomedical and Environmental Sciences. 10:415-435.
- Levander, O.A. 1986. Selenium . In: W. Mertz (Ed.), Trace Elements in Human and Animal Nutrition. Academic press, London. pp. 139-197.
- Lighthart, B., H. Bond, and M. Ricard. 1977. Trace Elements Research Using Coniferous Forest Soil/Litter Microcosms. USEPA-600/3-77-091. (Cited In Efroymson *et al.* 1997b).
- Lombeck, I., H. Menzel, and D. Frosch. 1987. Acute selenium poisoning of a 2-year-old child. Eur J Pediatr 146(3):308-312. (Cited In ATSDR 2003).
- Mack, R.B. 1990. The fat lady enters stage left. Acute selenium poisoning. NC. Med. J. 51(12):636-638. (Cited in ATSDR 2003).
- Maier, K.J., C. Foe, R.S. Ogle, M.J. Williams, A.W. Knight, P. Kiffrey, and L.A. Melton. 1988. The dynamics of selenium in aquatic ecosystems. In Hemphill, D.D. (ed.). Trace substances in environmental health. XXI Proceedings. Columbia, MO: University of Missouri. pp. 361-408. (Cited in ATSDR 2003).
- Marier, J.R., and J.F. Jaworski. 1983. Interactions of Selenium. National Research Council Canada, Associate Committee on

Scientific Criteria for Environmental Quality, Subcommittee on Heavy Metals and Certain Other Elements. NRCC No. 20643.

- McKeague, J.A., and M.S. Wolynetz. 1980. Background levels of minor elements in some Canadian soils. Geoderma. 24:299-307.
- MedlinePlus. 1993. Drug information: selenium sulfide topical. U.S. National Library of Medicine and National Institutes of Health. Revised: 07/26/1993. Available at: <u>http://www.nlm.nih.gov/medlineplus/print/druginfo/uspdi/202520.ht</u> ml
- Merck. 1996. The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals. Twelfth Edition. Merck and Co., Inc. Rathway, NJ.
- Mertz, W. 1995. Risk assessment of essential trace elements: new approaches to setting recommended dietary allowances and safety limits. Nutr. Rev. 53(7):179-85.
- Mikkelsen, R.L., A.L. Page, and F.T. Bingham. 1989. Factors Affecting Selenium Accumulation by Agricultural Crops. In: Selenium in Agriculture and the Environment. SSSA Special Publication Number 23. ISBN 0-89118-789-8. pp. 65-94.
- Miller, J.J., B.J. Read, D.J. Wentz, and D.J. Heaney. 1996. Major and trace element content of shallow groundwater associated with dryland saline soils in Southern Alberta. Water Qual. Res. J. Can. 31(1): 101-117.
- Moxon, A.L. 1937. Alkali disease or selenium poisoning. S. Dak. Agric. Exp. Stn. Bull. No. 311. South Dakota State College of Agriculture and Mechanic Arts, Agricultural Experiment Station, Brookings. 91 pp. (Cited in NRC 1980).
- Munro, I. 1999. Perspective of the Food and Nutrition Board Subcommittee on upper reference levels of nutrients. Proceedings of the Annual Summer Meeting of the Toxicology Forum, July 12-16, Aspen, Colorado.
- Munro, I.C. 2006. Setting tolerable upper intake levels for nutrients. J. Nutr. 136: 490S-492S.
- Nagpal, N.K. and K. Howell. 2001. Water quality guidelines for selenium. Technical appendix. Water Protection Branch, Water, Lands and Air Protection, Victoria, British Columbia. (http://www.env.gov.bc.ca/wat/wq/BCguidelines/selenium)
- NAS. (National Academy of Sciences). 1976. Selenium. Washington, D.C.: National Academy of Sciences. (Cited in ATSDR 2003).
- 1980. Recommended dietary allowances. 9<sup>th</sup> Revision. Washington, DC: Food and Nutrition Board, National Academy of Science. pp. 162-164. (Cited in ATSDR 2003).
- Navarro-Alarcón, M., and M.C. López-Martínez. 2000. Essentiality of selenium in the human body: relationship with different diseases. Sci. Total Env. 249: 347-371.
- NRC. (National Research Council). 1980. Selenium. In: Mineral Tolerance of Domestic Animals. National Academy of Sciences (NAS), Washington, DC. pp.392-415.
- NRCan (Natural Resources Canada) 2006.. Minerals and Mining Statistics On-Line. Accessed August 2006. Available at: http://mmsd1.mms.nrcan.gc.ca/mmsd/production/default e.asp
- Nriagu, J.O. and J.M. Pacyna. 1988. Quantitative assessment of worldwide contamination of air, water and soils by trace metals. Nature 333: 134-139.
- Olson, O.E. 1978. Selenium in Plants as a Cause of Livestock Poisoning. In: Effects of Poisonous Plants on Livestock. Keeler et al. (eds.). pp. 121-133.
- OMEE. (Ontario Ministry of the Environment and Energy). 1994. Ontario typical range of chemical parameters in soil, vegetation, moss bags and snow. April, 1994 (Version 1.0a). PIBS 2792. 212 pp. +app.
- Ort, J.F., and J.D. Latshaw. 1978. The toxic level of sodium selenite in the diet of laying chickens. J. Nutr. 1081114. (Cited in NRC 1980).

#### Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health

- O'Toole, D. and M.F. Raisbeck. 1995. Pathology of experimentally induced chronic selenosis (alkali disease) in yearling cattle. J. Vet. Diagnostic Investigation 7(3): 364-373.
- Outridge, A.M., Scheuhammer, G.A. Fox, B.M. Braune, L.M. White, L.J. Gregorich, and C. Keddy. 1999. An assessment of the potential hazards of environmental selenium for Canadian water birds. Environ. Rev. 7: 81–96.
- Penny, D. 2004. The Micronutrient and Trace Element Status of Forty-Three Soil Quality Benchmark Sites in Alberta. Report prepared for the AESE (Alberta Environmentally Sustainable Agriculture) Soil Quality Monitoring Program, Alberta Agriculture, Food and Rural Development, Conservation and Development Branch, Edmonton, Alberta.. July
- Puls, R. 1994. Mineral Levels in Animal Health Diagnostic Data. 2<sup>nd</sup> Edition. Sherpa International, Clearbrook, B.C.
- Rasmussen, P., K. Subramanian, and B. Jessiman. 2001. A multi-element profile of housedust in relation to exterior dust and soils in the city of Ottawa, Canada. Science of the Total Environment 267(1-3): 125-140
- Rayman, M.P. 2000. The importance of selenium to human health. The Lancet 356: 233-241.
- Reamer, D.C., and W.H. Zoller. 1980. Selenium biomethylation products from soil and sewage sludge. Science 208:500-502.
- Reilly, C. 1996. Selenium in Food and Health. Blackie Academic and Professional, London
- Reilly, C. 2004. The Nutritional Trace Elements. Blackwell Publishing,
- Reilly, C. 2006. Selenium in Food and Health, 2<sup>nd</sup> edition. Blackie Academic and Professional, London
- Richardson, G.M. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment. O'Connor Associates Environmental Inc., 14 Clarendon Ave., Ottawa, ON K1Y 0P2.
- Rosenfeld, I., and O.A. Beath. 1954. Effect of selenium on reproduction in rats. Proc. Soc. Exp. Biol. Med. 87:295-297.
- Salisbury, F.B., and C.W. Ross. 1985. Plant Physiology. Third Edition. Wadsworth Publishing Company, California. pp. 102.
- Sample, B.E., D.M. Opresko, G.W. Suter II. 1996. Toxicological Benchmarks for Wildlife: 1996 Revision. Prepared by the Risk Assessment Program Health Sciences Research Division. Oak Ridge, Tennessee. Prepared for the US Department of Energy Office of Environmental Management. ES/ER/TM-86/R3.
- Sample, B.E., J.J. Beauchamp, R.A. Efroymson, G.W. II Suter, and T.L. Ashwood. 1998a. Development and Validation of Bioaccumulation Models for Earthworms. Prepared for the US Department of Energy. Office of Environmental Management. Oak Ridge National Laboratory. ES/ER/TM-220.
- Sample, B.E., J.J Beauchamp, R.A. Efroymson, and G.W. II Suter. 1998b. Development and Validation of Bioaccumulation Models for Small Mammals. Prepared for the US Department of Energy. Office of Environmental Management. Oak Ridge National Laboratory. ES/ER/TM-219.
- Santolo, G.M., J.T. Yamamoto, J.M. Pisenti, and B.W. Wilson. 1999. Selenium accumulation and effects on reproduction in captive American kestrels fed selenomethionine. J. Wildlife Manage. 63(2): 502-511.
- Schroeder, H.A., and M. Mitchener. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch. Envir. Health. 23:102-106. (Cited in Sample et al. 1996).
- Shamberger, R.J. 1981. Selenium in the environment. Sci. Total Environ. 17:59-74.
- Singh, M., and N. Singh. 1978. Selenium toxicity in plants and its detoxification by phosphorus. Soil Sci. 126:255-262.
- Singh, M. and N. Singh. 1979. The effect of forms of selenium on the accumulation of selenium, sulphur, and forms of nitrogen and phosphorus in forage cowpea (*Vigna sinensis*). Soil Science 127(5): 264-269.

- Smith, G.J., G.H. Heinz, D.J. Hoffman, J.W. Spann, and A.J. Krynitsky. 1988. Reproduction in black-crowned night-herons fed selenium. Lake Reservoir Manage. 4:175-180. (Cited in Sample et al. 1996).
- Soltanpour, P.N., and S.M. Workman. 1980. Use of NH<sub>4</sub>HCO<sub>3</sub>-DTPA soil test to assess availability and toxicity of selenium to alfalfa plants. Commun. Soil Sci. Plant. Anal. 11(12):1147-1156. (Cited in Efroymson et al. 1997a).
- Stadtman, T.C. 1983. New biological functions--Selenium-dependent nucleic acids and proteins. Fundam Appl Toxicol 3:420-423. (Cited In ATSDR 2003).
- Stanley, T.R. Jr., G.J. Smith, D.J. Hoffman, G.H. Heinz, and R. Rosscoe. 1996. Effects of boron and selenium on mallard reproduction and duckling growth and survival. Environ. Toxicol. Chem. 15(7):1124-1132.
- Stowesand, G.S., J.L. Anderson, L.H. Weinstein, J.F. Osmloski, W.H. Gutenmann, and D.J. Lisk. 1990. Selenium in tissues of rats fed rutabagas grown on soil covering a cola fly ash landfill. Bull. Environ. Contam. Toxicol. 44:681-685.
- Subramanian, K.S., and J.C. Méranger. 1984. A survey of sodium, potassium, barium, arsenic, and selenium in Canadian drinking water supplies. At. Spectrosc. 5:34.
- Tarantal, A.F., C.C. Willhite, B.L. Lasley, C.J. Murphy, C.J. Miller, M.J. Cukierski, S.A. Brooks, and A.G. Hendrickx. 1991. Developmental toxicity of 1-selenomethionine in Macaca fascicularis. Fund Appl Toxicol 16:147-160. (Cited In Sample *et al.* 1996b).
- US DOE. (United States Department of Energy). 1998. Empirical Models for the Uptake of Inorganic Chemicals from Soil by Plants. BJC/OR-133.
- US EPA. (United States Environmental Protection Agency). 1991. Integrated Risk Information System (IRIS) for Selenium and Compounds. Online. National Center for Environmental Assessment, Cincinnati, OH.
- Wan, H.F., R.L. Mikkelse, and A.L. Page. 1988. Selenium uptake by some agricultural crops from central California soils. J. Environ. Qual. 17(2):269-272. (Cited in Efroymson et al. 1997a).
- WHO (World Health Organization). 1996. Trace Elements In Human Nutrition And Health. World Health Organization, Geneva.
- WHO (World Health Organization) 2002. Principles and methods for the assessment of risk from essential trace elements. International Programme on Chemical Safety (IPCS).
- WHO (World Health Organization) and FAO (Food and Agriculture Organization of the United Nations). 2004. Vitamin and mineral requirements in human nutrition. Second edition. Joint FAO/WHO Expert Consultation on Human Vitamin and Mineral Requirements (1998: Bangkok, Thailand). Available at:

http://whqlibdoc.who.int/publications/2004/9241546123.pdf

- Wiemeyer, S.N. and D.J. Hoffman. 1996. Reproduction in eastern screech-owls fed selenium. J. Wild. Manage. 60(2): 332-341.
- Wu, L., J. Chen, K.K. Tanji, and G.S. Banuelos. 1995. Distribution and biomagnification of selenium in restored upland grassland contaminated by selenium from agricultural drain water. Environ. Toxicol. Chem. 14(4):733-743.
- Yamamoto, J.T., G.M. Santolo, and B.W. Wilson. 1998. Selenium accumulation in captive American kestrels (Falso sparverius) fed selenomethionine and naturally incorporated selenium. Environ. Toxicol. Chem. 17(12): 2494-2497.
- Yang, G.-Q., S. Yin, R.-H. Zhou, Gu, B. Yan, Y. Liu, and Y. Liu. 1989. Studies of safe maximal daily dietary selenium intake in a seleniferous area in China. Part II. Relation between selenium intake and the manifestations of clinical sings and certain biochemical alterations in blood and urine. J. Trace Elemen. Electrolytes Health Dis, 3:123-130 (Cited In IOM 2000).
- Yang, G.-Q. and R.-H. Zhou. 1994. Further observations on the human maximum safe dietary intakes of selenium in a seleniferous area of China. J. Trace Elemen. Electrolytes Health Dis. 8: 159-165. (Cited In IOM 2000).

## SELENIUM

Yukon Zinc Corporation. 2005. Selenium Market Overview. Posted online, November 2005. Available at: <u>http://www.yukonzinc.com/documents/Selenium2005-11-10.pdf</u> Zieve, R., and P.J. Peterson. 1981. Factors influencing the volatilization of selenium from soil. Sci. Total Environ. 19:277-284.

#### Reference listing:

Canadian Council of Ministers of the Environment. 2009. Canadian soil quality guidelines for the protection of environmental and human health: Selenium (2009). In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

For further scientific information, contact:

Environment Canada National Guidelines and Standards Office 200 Sacré-Coeur Blvd., 7<sup>th</sup> Floor Gatineau, QC K1A 0H3 Phone: (819) 953-1550 Facsimile: (819) 994-3120 E-mail: ceqg-rcqe@ec.gc.ca Internet: http://www.ec.gc.ca/ceqg-rcqe

© Canadian Council of Ministers of the Environment 2009 Excerpt from Publication No. 1299; ISBN 1-896997-34-1 For additional copies:

www.ccme.ca

Aussi disponible en français.