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Canadian Soil Quality Guidelines
SELENIUM
Environmental and Human Health Effects

Scientific Criteria Document

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NOTE TO READERS

The Canadian Council of Ministers of the Environment (CCME) is the major intergovernmental forum in Canada for discussion and joint action on environmental issues of national, international and global concern. The 14 member governments work as partners in developing nationally consistent environmental standards, practices and legislation.

This document provides the background information and rationale for the development of the Canadian Soil Quality Guidelines for selenium. For additional technical information regarding these guidelines, please contact:

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This scientific supporting document is available in English only. Ce document scientifique du soutien n'est disponible qu'en anglais avec un résumé en français.

Reference listing;

CCME. 2009. Canadian Soil Quality Guidelines : Selenium. Environmental and Human Health. Scientific Supporting Document. Canadian Council of Ministers of the Environment, Winnipeg.

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The original Canadian Soil Quality Guidelines for selenium was developed in 2001 (EC, 2001) and published in 2002. Initial drafts of the 2001 document were prepared by Cantox Environmental Inc. in 1998-1999, with subsequent revisions by Nadec Experts and Consultants Inc. in 2000. Additional input and reviews of the 2002 guidelines were provided by: Kathie Adare, J.D. Whall, and Kelly Potter of Environment Canada; Marco Pagliarulo and Diana Piché of Ontario Ministry of Environment; and Mark Richardson of Risklogic Scientific Services, Inc. In 2007 it was revised and updated by the Contaminated Sites Division, Health Canada (Sylvie Coad), and the National Guidelines and Standards Office, Environment Canada (Kelly Potter). Review comments were provided by the CCME Soil Quality Guidelines Task Group, and Peter M. Chapman of Golder Associates Ltd.

This 2009 revised edition corrects an error made in 2007 in the calculation of the direct human health-based soil quality guideline (SQG_{DH}) for commercial and industrial land use.

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ABSTRACT

Canadian environmental quality guidelines, developed under the auspices of the Canadian Council of Ministers of the Environment (CCME), are numerical concentrations or narrative statements recommended to support and maintain designated resources uses. Canadian soil quality guidelines can be used as the basis for consistent assessment and remediation of contaminants at sites in Canada.

This report was prepared by the Contaminated Sites Division of Health Canada and by the National Guidelines and Standards Office (Environment Canada), which acts as Technical Secretariat for the CCME Soil Quality Guidelines Task Group. The Guidelines were derived according to the procedures described in *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 2006).

The 2002 selenium guideline was updated and revised in 2007 by the Contaminated Sites Division, Health Canada (Sylvie Coad), and the National Guidelines and Standards Office, Environment Canada (Kelly Potter). Review comments were provided by the CCME Soil Quality Guidelines Task Group, and Peter M. Chapman of Golder Associates Ltd.

The 2007 publication was revised in 2009 to correct an error made in 2007 in the calculation of the direct human health-based soil quality guideline (SQG_{DH}) for commercial and industrial land use. Note that the final Canadian Soil Quality Guidelines from 2007 have not changed as a result of this revision and thus there is no impact on tier 1 implementation. However, the 2007 Human Health Soil Quality Guideline (SQG_{HH}) for commercial land use has changed because it was, and still is, based on the commercial land use SQG_{DH}. The 2007 industrial land use SQG_{HH} is not affected by the current changes because it was, and still is, based on the off-site migration check ($SQG_{OM\text{-}HH}$ =1135 mg/kg).

In summary, the changes made to the Canadian Soil Quality Guidelines for Selenium (2007) are as follows (note, these changes could have implications for tier 2 implementation);

- SQG_{DH} for commercial land use is reduced from 300 mg/kg to **125 mg/kg**
- SQG_{DH} for industrial land use is reduced from 9770 mg/kg to **4050 mg/kg**
- SQG_{HH} for commercial land use is reduced from 300 mg/kg to **125 mg/kg**

Following the introduction, Chapter 2 presents chemical and physical properties of selenium and a review of the sources and emissions in Canada. Chapter 3 discusses selenium's distribution and behavior in the environment while Chapter 4 reports the effect in terrestrial biota and the toxicological effects on microbial processes, plants, and animals. Chapter 5 discusses the effects of selenium in human and experimental animals. This information is used in Chapter 6 to derive environmental and human health soil quality guidelines for selenium receptors in four types of land uses: agricultural, residential/parkland, commercial, and industrial.

The following soil quality guidelines are recommended by CCME based on the available scientific data. A draft recommended CCME soil quality guidelines fact sheet is presented in both French and English in Appendix A. For selenium, the environmental soil quality guideline (SQG_E) relative to agricultural and residential/parkland land uses is 1 µg/g whereas for

commercial and industrial land uses it is 2.9 µg/g. The human health soil quality guideline (SQG_{HH}) relative to agricultural and residential/parkland land uses is 80 µg/g, for commercial land use it is 125 µg/g, and for industrial land use it is 1135 µg/g. For specific locations with unusually high natural background concentrations that still exceed these guidelines, jurisdictions have the option to set site-specific guidelines that consider the unique geological characteristics of the particular locations (CCME 2006).

RÉSUMÉ

Les recommandations canadiennes pour la qualité de l'environnement, élaborées sous les auspices du Conseil Canadien des Ministres de l'Environnement (CCME), sont des concentrations ou des énoncés décrivant les limites recommandées dans le but d'assurer le maintien et le développement durable d'utilisations désignées des ressources. Les recommandations canadiennes pour la qualité des sols peuvent être utilisées comme base pour l'uniformisation des processus d'évaluation et d'assainissement des terrains contaminés au Canada.

Le présent document a été préparé par la Division des lieux contaminés de Santé Canada et par le Bureau national des recommandations et des normes (Environnement Canada), qui agit comme secrétaire technique pour le Groupe de Travail du CCME sur les Recommandations pour la qualité des sols. Les recommandations ont été élaborées selon les procédures décrites dans le *Protocole d'élaboration de recommandations pour la qualité des sols en fonction de l'environnement et de la santé humaine* (CCME 2006).

En 2007, la Division des lieux contaminés de Santé Canada (Sylvie Coad) et le Bureau national des recommandations et des normes d'Environnement Canada (Kelly Potter) ont actualisées et révisées les recommandations de 2002 concernant le sélénium. Des commentaires d'examen ont été fournis par le Groupe de Travail du CCME sur les Recommandations pour la qualité des sols et Peter M. Chapman de Golder Associates Ltd.

La publication de 2007 a été révisée en 2009 pour corriger une erreur commise en 2007 dans le calcul des recommandations pour la qualité des sols en fonction de la santé humaine par contact direct (RQS_{CD}) pour les utilisations commerciales et industrielles du sol. Veuillez prendre note que la version finale des Recommandations canadiennes pour la qualité des sols de 2007 n'a pas été modifiée par cette révision, et qu'il n'y a donc pas eu d'impact sur la première étape de la mise en œuvre. Toutefois, les recommandations pour la qualité des sols en fonction de la santé humaine (RQS_{SH}) de 2007 pour les utilisations commerciales du sol ont été modifiées, car elles étaient, et sont toujours, basées sur les RQS_{CD} pour les utilisations commerciales du sol. Les RQS_{SH} de 2007 pour les utilisations industrielles du sol ne sont pas touchées par les modifications, car elles étaient, et sont toujours, basées sur la vérification pour la migration hors site ($RQS_{MH\text{-}SH}$ = 1135 mg/kg).

En résumé, les modifications apportées aux Recommandations canadiennes pour la qualité des sols qui s'appliquent au sélénium (2007) sont les suivantes (veuillez prendre note que ces modifications pourraient avoir un impact sur la deuxième étape de la mise en œuvre) :

- Les RQS_{CD} pour les utilisations commerciales du sol passent de 300 mg/kg à **125 mg/kg**
- Les RQS_{CD} pour les utilisations industrielles du sol passent de 9770 mg/kg à **4050 mg/kg**
- Les RQS_{SH} pour les utilisations commerciales du sol passent de 300 mg/kg à **125 mg/kg**

Faisant suite à une brève introduction, le chapitre 2 présente les propriétés chimiques et physiques du sélénium, de même qu'un survol des sources et des émissions au Canada. Les chapitres 3 et 4 traitent du devenir et du comportement de cette substance dans l'environnement ainsi que des effets toxicologiques sur les processus microbiens, les plantes et les animaux. Le

chapitre 5 porte sur les effets toxicologiques et le comportement de cette substance chez l'humain et les animaux de laboratoire. Ces informations sont utilisées au chapitre 6 afin d'élaborer des recommandations protectrices de l'environnement et de la santé humaine pour la qualité des sols relatives au sélénium dans le cadre de quatre types d'utilisations de terrains : agricole, résidentiel/parc, commercial et industriel.

Les recommandations pour la qualité des sols suivantes, proposées par le CCME, sont fondées sur les donnés scientifiques disponibles. Pour le sélénium, les recommandations pour la qualité des sols en vue de la protection de l'environnement relatives aux terrains à vocation agricole et résidentielle/parc sont de 1 µg/g de sol et de 2.9 µg/g de sol pour les terrains à vocation commerciale et industrielle. Les recommandations pour la qualité des sols en vue de la protection de la santé humaine sont de 80 µg/g pour les terrains à vocation agricole et résidentielle/parc, de 125 µg/g pour les terrains commerciaux et 1135 µg/g pour les terrains industriels. Il est possible qu'il existe des sites aux concentrations naturelles de fond anormalement élevées et dépassant ces recommandations. Dans ces cas, les pouvoirs publics peuvent établir des recommandations propres à un site qui examinent les caractéristiques géologiques uniques de ces endroits (CCME 2006).

1. INTRODUCTION

Canadian Soil Quality Guidelines are numerical concentrations or narrative statements that specify levels of toxic substances or other parameters in soil that are recommended to maintain, improve or protect environmental quality and human health. They are developed using formal protocols to ensure nationally consistent, scientifically defensible values. The guidelines are nationally endorsed through the Canadian Council of Ministers of the Environment (CCME).

This report reviews the sources and emissions of selenium, its distribution and behaviour in the environment, and its toxicological effects on soil microorganisms, plants, animals, and humans. This information is used to derive guidelines for selenium to protect environmental and human health receptors according to the processes outlined in "A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines" (CCME 2006), for agricultural, residential/parkland, commercial and industrial land uses. In addition, various check mechanisms considering indirect pathways of exposure (e.g., nutrient and energy cycling, and off-site migration of contaminants via wind and water erosion) as elaborated in (CCME 2006), are used to ensure protection for resources and receptors not otherwise considered in the derivation of soil quality guidelines.

The following derived values should be considered for general guidance purposes; however, in the application of these values, site-specific conditions should be considered. Since the guidelines may be applied differently in various jurisdictions, the reader should consult appropriate authorities for guidance in the application of these guidelines. Every attempt was made to provide a conservative estimate that could be applied to any area in Canada. Due to geological conditions, it is possible that natural enrichment will result in exceedences of the soil quality guidelines. Thus, such exceedences do not automatically imply that the ecosystem is compromised. The guideline represents a limit below which no adverse impacts are expected, but site-specific information, such as local background concentrations, should always be considered in the application of these guidelines (See CCME (1996) for further discussion of this issue).

2. BACKGROUND INFORMATION

2.1 Physical and Chemical Properties

Physical and chemical properties of selenium are presented in Table 1. Selenium (CAS RN: 7782-49-2) is a Group 16 or VIA non-metal, and has four valence states (0, II, IV, VI). There are six natural isotopes of selenium, the most abundant being ^{80}Se (50%), and ^{78}Se (23.5%). The chemical properties of selenium resemble but are not identical to those of sulphur (Greenwood and Earnshaw 1984).

Due to the number of valence states, selenium forms a number of compounds in the environment, including bromides, fluorides, chlorides, oxides, hydrides, sulfides, and many metal compounds (i.e., selenides). 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; crystalline selenium is a deep red; the most stable form of selenium is termed gray or metallic selenium (also called crystalline hexagonal selenium), and is a lustrous gray to black colour.

Selenium has unique electrical and semiconductor properties. In the dark, its electrical conductivity is low but in the light, it is increased remarkably and a small electrical current is produced in the element (Reilly 2006).

2.2 Geochemical Occurrence

In nature, selenium usually occurs in sulfide ores of metals, hydrothermal ores, uranium ores in sandstone deposits, and to a lesser extent in pyrite, clausthalite (PbSe), naumannite (Ag_2Se), tiemannite (HgSe), and selenosulfur (Shamberger 1981).

Average crustal abundance estimates for selenium generally range from 0.05 to 0.09 µg/g (Plant *et al*. 2003; NRC1983). Some Canadian Precambrian non-nickeleferous ores are known to contain extremely high selenium concentrations (500 to 1,000 µg/g) (Hawley and Nichol 1959). Magmatic rocks such as granites and basalts, for example, contain low levels of Se, generally not exceeding 0.05 µg/g. On the other hand, some sedimentary rocks with the exception of sandstone (0.02-0.05 µg/g) contain much higher selenium concentrations (shales, 0.6 µg/g; argillaceous sediments, 0.4-0.6 µg/g); and limestones and dolomites, (0.03-0.10 µg/g) (Kabata-Pendias and Pendias, 2000). For example, the black shales of the Selwyn Basin, Yukon are reported to contain relatively high levels of Se (up to 1 µg/g in rock) in some areas (Gamberg *et al*. 2005). Dunn (1990) found much higher selenium concentrations in Cretaceous sedimentary rock formations in central Saskatchewan with levels ranging from 0.2 µg/g in the Lower Cretaceous Mannville Group to 11.7 µg/g in the White Speckled Shales. Selenium is also found in fossil fuels at concentrations ranging from 0.046 to 10.65 µg/g in coal (mean: 3.0 µg/g) and from 0.006 to 2.2 µg/g in oil (mean: 0.6 µg/g) (Marier and Jaworski 1983).

2.3 Analy tical Methods

There are a number of reliable digestion and analytical methods to determine the selenium content of soil and other environmental media (air, water and biological samples).

In soil samples, the amount of an element available for analysis varies depending on the extraction treatment of the samples prior to analysis (Ure 1995). Dissolution and digestion procedures for "total" analysis are available to extract a metal from the soil matrix which is mostly made up of silica and silicates, and of organic matter and sulfides. The release of all the selenium from soil for total selenium analysis requires digestion with hydrofluoric acid, generally used in combination with oxidizing acids such as perchloric and nitric acids (U.S. EPA

2006a; Ure 1995). On the other hand, *aqua regia* ($1HNO_3$:3HCl) digestion is used for "pseudo-total" analysis of soils and sludges. This method releases the "biologically-relevant" (R. G. Garrett, Natural Ressources Canada, pers. com.) or "pseudo-total" selenium adsorbed to soil particles, present in soluble salts and organic matter, as well as the selenium content of some weak silicates, leaving most silicates and stable mineral matrices intact (Ure 1995). Soil samples of some earlier selenium analyses by fluorometry were digested using only perchloric and nitric acids.

Different analytical methods, however, can result in different concentrations from the same sample depending on which form of selenium the method is capable of detecting, and at which physical/chemical sample parameters the method best operates. For example, the determination of selenium in complex environmental materials by hydride generation atomic absorption spectroscopy (HGAAS) produces more accurate results than flame or graphite furnace AAS (ATSDR 2003; Campbell 1992), but it only detects selenenite in solution; if total inorganic selenium is to be determined, a further step to reduce inorganic Se(VI) to Se(IV) is required (Cornelis *et al.* 2003). Hydride vapour generation techniques coupled with ICP-MS have been used successfully for the trace analysis of selenium in soils; Anderson *et al.* (1994) achieved detection limits of ≤1.0 µg/L for selenium extracted from soil with this method. The analytical method recommended for selenium by the CCME in 1993 was U.S. EPA Method 6010 revision 0 (6010A), an inductively coupled plasma-atomic emission spectroscopy (ICP-AES) method (CCME 1993a, b). This method is used for the analysis of trace amounts of a suite of inorganic parameters in both liquid and solid phase samples. However, detection limits, sensitivity, and optimum ranges will vary with the matrices and model of the spectrophotometer. CCME (1993b) reported an estimated instrumental detection limit (DL) of 75 µg Se/L and thus, is of limited use for determination of low selenium concentrations. U.S. EPA (2006a) has since revised this method once (Method 6010B).

For selenium in water and wastewater, the most sensitive methods to determine selenium in these media are hydride generation atomic absorption spectrometry (HGAAS) (manual or continuous), electrothermal (graphite furnace) atomic absorption spectrometry (ETAAS), and derivatization colorimetry (Standard Methods 2005). In most cases, a chemical pre-treatment for the reduction of selenate to selenite is required as noted above. The continuous HGAAS is a preferred method due to the quick and reproducible results that can be obtained, coupled with a low DL of less than 2 µg/L (Standard Methods 2005). In 1993, CCME (1993a, b) recommended two methods: U.S. EPA Method 3114B, a manual hydride generation atomic absorption spectrometry (HGAAS), and U.S. EPA Method 3120B, an inductively coupled plasma emission spectroscopy for the analysis of selenium in water and wastewater.

Methods such as ETAAS and HGAAS are commonly and successfully used in the determination of selenium in biological samples (Aras and Ataman 2006). While flame AAS lacks the sensitivity needed for trace amounts of selenium, flameless AAS is highly sensitive (Bem 1981). For total selenium determination in biological matrices, all selenium species in the sample should usually be converted to selenates with modifiers and by pyrolysis which in turn must be reduced to selenites. Other analytical methods such as spectrophotometry, voltammetry, and x-ray fluorescence have been successfully used in the determination of selenium in various human tissues and fluids but the most common method is fluorometry (ATSDR 2003). Neutron Activation Analysis (NAA) methods are capable of accurately detecting traces of selenium as

low as 0.01 to 0.001 μg/g in a wide variety of biological and environmental samples (Bem 1981). However, very few facilities have the necessary nuclear reactors and specialized knowledge to perform such analyses (ATSDR 2003). In the present document, unless otherwise specified, concentrations in biological materials are reported on a wet weight basis.

2.4 Production and Uses in Canada

In 2003, Canada was the second largest producer of selenium after Japan and before Belgium (Yukon Zinc Corporation 2005). In Canada, primary selenium is recovered as by-products of copper refining processes. In 2003, the Canadian selenium production amounted to 288 tonnes while its consumption was only 10.8 tonnes or 3.7% as a percentage of production (NRCan 2004). The 2005 primary selenium production from Canadian sources (Quebec, Ontario, Manitoba and Saskatchewan) is estimated to be about 216 tonnes (NRCan 2006).

The principal global markets for selenium (with estimated percentages) are glass manufacturing (35%), chemicals and pigments (24%), metallurgy (23%), electronics (10%), and other applications (8%) (George 2004).

In the glass industry, selenium is used as both a colourizing and decolourizing agent. It is also used as a solar heat reductant in architectural glass. Cadmium sulfuloselenide compounds are excellent red, orange and maroon pigments for ceramics, glazes, paints, enamels and plastics. However, this use is generally restricted owing to the toxicity of selenium-based pigments (George 2004, Yukon Zinc Corporation 2005).

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. However, selenium is being substituted by bismuth, lead and tellurium in free-machining alloys and by tellurium in lead-free brasses (Andersson 2005).

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, fertilizers, anti-dandruff shampoos, and anti-fungal agents (George 2004).

The major electronic use of selenium in the 1970s and 1980s was as the photoreceptor, arsenic triselenide, on drums of photocopying machines. This end-use has been drastically curtailed owing to the substitution of high-purity selenium compounds by amorphous silicon and organic photoreceptor compounds which are more environmentally friendly, less costly and better performers. 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). Amorphous selenium flat-panel detector systems for radiological facilities are being developed (George 2004). Selenium is also used as a vulcanizing agent in rubber processing and as a catalyst in Kjeldahl nitrogen analysis (George 2004).

It has been estimated that about 90% of the Se utilised in the US is dissipated in various forms to the environment. The remainder is recovered from photocopy machines, laser printers and rectifiers for secondary use (Butterman and Brown 2004)

2.5 Sources and Concentrations in the Canadian Environment

The assessment of soil quality for naturally occurring elements must take into consideration regional variations in background concentrations in Canada. Background concentrations and environmental fate of metals strongly depend on geological and biological characteristics and, therefore, any assessment of potential risks should take into consideration regional differences in metal content in the natural environment (Chapman and Wang 2000).

Relatively high concentrations of metals can occur naturally in Canadian soils, stream sediments, and water, blurring the distinction between anthropogenic pollution versus naturally occurring geological formations and natural bodies of ore. Selenium rich areas ($\geq$ 0.5 µg/g soil) in Canada include the southern Prairies and Ontario. Copper ores from Noranda (Quebec), Sudbury (Ontario) and Flin Flon (Manitoba) are relatively rich in selenium content (Marier and Jaworski 1983; NRCan 2006). Soils and sediments reflect the composition of parent material, resulting in higher metal concentrations in mineralized areas (Wilson *et al.* 1998). Mining districts are characterized by naturally occurring metals in soil, sediment, rock, and water at concentrations that could result in their classification as "contaminated sites" (Painter *et al.* 1994). In the determination of anthropogenic metal contamination of soils, no single guideline concentration can adequately represent the variance in background concentrations across Canada (Painter *et al.* 1994; Chapman and Wang 2000).

2.5.1 Atmosphere

Pacyna and Pacyna (2001) estimated global anthropogenic emissions of trace elements in the mid-1990s. The authors estimated global anthropogenic and natural emissions of selenium at 4,600 and 9,300 tonnes per year, respectively, the latter largely based on the work of Nriagu (1989). Coal and other fossil fuel combustion from stationary sources are estimated to be the primary source of anthropogenic emissions of selenium (ATSDR 2003). In 1999, under the Great Lakes Regional Air Toxics Emissions Project, the Ontario Ministry of the Environment (OME) published province-wide data on selenium emissions (Great Lakes Commission 2002). Annual emissions from point sources, area sources, on-road sources and off-road sources were estimated to be approximately 7650 kg, 230 kg, 280 kg and 80 kg, respectively (Great Lakes Commission 2002). Skeaff and Dubreuil (1997) estimated selenium emissions (stack and fugitive) from Canadian non-ferrous smelters to be 3.02 tons in 1993. The selenium concentrations measured by Brook *et al.* (1997) in Toronto and Montreal air particulates are attributed to the proximity of industries fuelled by coal and by coal-powered generation facilities. Laden *et al.* (2000) considered the presence of selenium in $PM_{2.5}$ as a marker for coal combustion sources. Between 62% and 85% of selenium emissions in the U.S. have been attributed to the burning of coal (Laden 2000).

Levels

There is a paucity of information on background levels of selenium in the Canadian atmosphere in the published literature. Unpublished information on selenium concentrations in air was provided by Environment Canada from the National Air Pollution Surveillance (NAPS) Canadian network of monitoring stations (T. Dann, Environment Canada, pers. com.). Additional data came from the Integrated Atmospheric Deposition Network (IADN) from a limited number of stations monitoring air quality of the Great Lakes Basin. Elemental concentrations in particulates were determined by x-ray fluorescence (Environment Canada 2005) or ICP-MS analyses. Selenium concentrations in air were often below analytical limits of detection.

Elemental concentrations in coarse particulate matter (PM_{10}) and fine particulate matter ($PM_{2.5}$) were provided for NAPS stations across Canada (T. Dann, Environment Canada, pers. com.). Particulate matter (PM_{10}) and ($PM_{2.5}$) were collected over 24-hr periods from dichotomous samplers with Teflon filters; elemental concentrations were obtained using nondestructive x-ray fluorescence spectrometry (Burnett *et al.* 2000; Dann 1994; Environment Canada 2005). Annual minimum and maximum concentrations of selenium for 2002 and 2003 in PM_{10} (n=2170 samples) and in $PM_{2.5}$ (n=2144 samples) were provided for 31 NAPS stations distributed across all provinces and territories with the exception of the Yukon, Newfoundland and PEI. The interpretation of both data sets is somewhat limited by the large number of samples with concentrations below analytical detection and is further confounded by detection limits which were not consistent over time and location (range: 0.0006 to 0.0015 ng/m^3). However, measured selenium levels were low. In PM_{10}, only 33% of the samples (n=721) were above analytical detection for selenium. Annual mean concentrations (incorporating only values above analytical detection, in 2002/03 were reported by 31 stations and ranged from 0.5 ng/m^3 (Iqaluit, Nunavut) to 1.7 ng/m^3 (Egbert, ON). An overall average selenium concentration of 1.0 ng/m^3 in PM_{10} for 2002/03 was calculated based on those means. In $PM_{2.5}$, fewer determinations for selenium concentrations above the analytical limit were reported; 28% of the samples were above analytical detection (n=606). Annual average concentrations (incorporating only values above analytical detection) in 2002/03 reported by 31 stations ranged from 0.4 to 0.9 ng/m^3 and the overall average across all stations of the mean detected values was calculated to be 0.6 ng/m^3.

Burnett *et al.* (2000) summarized selenium concentrations in fine particulate matter ($PM_{2.5}$) collected in 8 Canadian cities (Montreal, Ottawa, Toronto, Windsor, Winnipeg, Calgary, Edmonton, and Vancouver) from 1986 to 1996 inclusive, also monitored under the NAPS program. The mean and 95th percentile concentrations reported for selenium over this time period were 1 and 3.6 ng/m^3, respectively. Nearly half (49%) of the 4,255 samples collected were above analytical detection, the average detection limit reported for selenium over this time period was 0.5 ng/m^3. A few years earlier, Brook *et al.* (1997) summarized the selenium NAPS data collected from 14 urban areas between 1985 and 1993. Selenium was only detected in about 36% of the 3435 samples (typical detection limit of 0.8 ng/m^3) with a median and 95% percentile concentrations of 0.8 and 3.0 ng/m^3, respectively.

Data on selenium concentrations in PM_{10} collected up to 2000 are available under the Integrated Atmospheric Deposition Network (IADN) for 3 monitoring stations around the Great Lakes (IADN 2003). The IADN, established in 1990, has been implemented by the Canadian Federal

(Environment Canada) and Provincial (Ontario Ministry of the Environment) governments and the U.S. Environmental Protection Agency as mandated in Annex 15 of the Great Lakes Water Quality Agreement (GLWQA) to monitor air and precipitation in the Great Lakes Basin. Three Canadian monitoring stations, located on Burnt Island (Lake Superior), Egbert (rural) and Point Petre (Lake Ontario), Ontario, were selected as representative of background air data for particulates in the region. Annual mean selenium summary data (non-blank corrected) from 1998 to 2000 for the 3 locations range from 0.214 ng/m^3 in Burnt Island to 1.719 ng/m^3 in Egbert. From 1998 to 2000, the overall annual mean Se levels (rounded up to 1 significant digit) for Burnt Island, Egbert and Point Petre were 0.4, 0.5, and 0.9 ng/m^3, respectively (IADN 2003).

A pollution episode involving a copper smelter near Montreal produced a maximum air selenium concentration of 27.4 µg/m^3, with a five day average of 15.4 µg/m^3 (Burton and Phillips 1981). These air concentrations were associated with damage to several species of local vegetation.

In the United States, background ambient air concentrations of selenium are generally in the ng/m^3 range (ATSDR 2003). Based on the results of a number of air quality studies, the U.S. National Academy of Sciences (NAS 1976) estimated that the average selenium concentration in U.S. air was less than 10 ng/m^3. Metropolitan areas were thought to have slightly higher airborne selenium concentrations, with levels of less than 40 ng/m^3 expected for most cities (NAS 1976).

2.5.2 Indoor Air

Only one study on selenium concentrations measured in indoor air was located in the literature. As part of the Windsor Air Quality Study, Bell *et al.* (1994) measured low levels of airborne selenium collected in the personal breathing zone of volunteers at home and the office in the Windsor area, Ontario. A total of 47 indoor personal air samples (n=47) were collected in 1991 (summer) and 1992 (winter and early spring) and analysed for selenium by ICP-MS; at a method detection limit (MDL) of 2.5 ng/m^3, only 8 samples contained measurable levels of selenium and the maximum was 3.3 ng/m^3. Selenium levels in personal air samples (n=4) taken when volunteers visited bingo halls ranged from 0.2 to 0.8 ng/m^3 (mean: 0.5 ng/m^3). Bell *et al.* (1994) also measured selenium levels in six homes of asthmatics in the Windsor area; five of six samples contained detectable levels (MDL unspecified) with a mean of 0.6 ng/m^3 and a maximum level of 3.3 ng/m^3; cigarettes were smoked indoors in five of those homes, and likely in the bingo hall.

Limited information on selenium levels in photocopy rooms has also been published. However, this information is likely no longer pertinent to present conditions in photocopy rooms, because arsenic triselenide is no longer used as a photoreceptor in the more up-to-date machines (George 2004; Andersson 2005). Harkin *et al.* (1976) reported higher airborne selenium concentrations in photocopying rooms compared to laboratories. Selenium is released from photocopiers due to a volatilization process generated by the sparking and heating of selenium-containing toner particles entrapped by static electricity buildup (Scheuermann 1978; Vokal-Borek 1979). It was estimated that 6000 photocopies would release 0.1 mg of selenium into the air, but this was not considered likely to pose any indoor air quality problems provided that air circulation was adequate (Scheuermann 1978). However, there would be a potential for substantial selenium

accumulation in the air of poorly ventilated, high-volume photocopier rooms. Dukic-Cvijanovic (1991) reported an average concentration (±s.d.) of 2.5 ±1.3 μg/m^3 in the air of 10 photocopy rooms (presumably located in Serbia).

2.5.3 Soil and Dust

Selenium is a ubiquitous natural constituent in soil, originating from rocks in the earth's crust (Kabata-Pendias and Pendias 2000). The selenium content in soil is widely variable and is a reflection of the weathering of parent materials though atmospheric and anthropogenic inputs may also alter its composition (Neal 1995). In a study of distribution of selenium in Canadian soils, Lévesque (1974a) concluded that the main factors governing the selenium distribution in most soil profiles were the Se content of the parent material and the organic content of the upper horizons levels; the highest levels of selenium were encountered at the soil surface.

Levels

On a worldwide basis, Plant *et al*. (2003) estimated an average selenium concentration of 0.4 μg/g in surficial soils with levels typically ranging from 0.01 to 2.0 μg/g. Data specific to selenium concentrations in Canadian soils were identified for Alberta, Saskatchewan, Manitoba, Ontario, New Brunswick, Nova Scotia and Prince Edward Island, and are described below.

Canada

McKeague *et al.* (1979) analysed representative soil samples from all provinces and territories except Manitoba to evaluate background concentrations of minor elements in Canadian mineral soils. Relevant data, previously obtained under the International Joint Commission program and published elsewhere (Whitby *et al.* 1978a, 1978b), were also incorporated in the McKeague *et al.* (1979) data sets. For the determination of selenium, a total of 188 soil samples were digested with $HNO_3/HClO_4$ and analysed using a fluorometric method. The analytical results were regrouped geographically into 5 regions: Appalachian, Canadian Shield, St. Lawrence Lowlands, Interior Prairies and Cordilleran. For all regions combined, the selenium levels ranged from 0.02 to 3.7 μg/g and a mean of 0.30 μg/g was reported. On a regional basis, mean selenium levels ranged from 0.18 to 0.40 μg/g. Detailed regional data are presented in Table 2.

Lévesque and Vendette (1971) developed a modified method using fluorescence after $HNO_3/HCLO_4$ digestion to determine Se levels in soils. Ten surface soil samples from Ontario, Quebec and New Brunswick were tested for selenium with concentrations ranging from 0.155 to 0.540 μg/g. Lévesque (1974a) reported levels in 54 Canadian soil profiles according to horizon and to soil properties. Considering all types of soil and all horizons analysed, the overall selenium content ranged from 0.07 μg/g (Podsolic C horizon) to 2.1 μg/g (Gleysolic H horizon).

Prairies

In 1992, the Geological Survey of Canada (Natural Resources Canada) performed an ultra-low density regional geochemical survey of surface soils (top 25 cm) in the Prairies (R.G. Garrett 2005, Natural Resources Canada, pers. com.). A total of 1273 (as stated, but adding up to 1076) Prairie soil samples (Manitoba, n=198, Saskatchewan, n=526 and Alberta, n= 352) were

analysed for Se and other elements. The <2 mm fraction of the sieved samples was retained for near total determination by AAS following digestion in a fuming $HF\text{-}HNO_3\text{-}HCLO_4$ mixture. Manitoba surface soils contained the highest concentrations of selenium ranging from 0.1 to 4.7 µg/g with a mean(±s.d) of 0.62 µg/g (±0.44). In Saskatchewan, Se levels ranged from 0.1 to 3.1 µg/g with a mean(±s.d) of 0.53 µg/g (±0.28). Similar results were obtained for Alberta soils where Se levels ranged from 0.1 to 2.7 µg/g with a mean(±s.d) of 0.55 µg/g (±0.28). Interestingly, the same median concentration of selenium, 0.5 µg/g, was determined in each of the 3 surveyed provinces. The highest levels of selenium in the Prairie soils are attributed to the occurrence of Se-enriched Cretaceous-age shale outcrops in the Manitoba escarpment and further west (R. G. Garrett. Natural Resources Canada, 2005, pers. com.; Plant and Smith 1998).

Selenium was one of the 30 trace elements determined in a survey of agricultural soils under the Alberta Environmentally Sustainable Agriculture (AESA) Soil Quality Monitoring Program (Penny 2004). The survey was performed in 2002 to provide a valuable benchmark database regarding elemental concentrations within ecoregions, soil types and landscapes. A total of 129 sampling sites taken from 43 benchmark sites were investigated and two soil samples from different depths (0-15cm and 15-30cm) from each sampling site were collected for analysis (n=258). Soil samples were digested with aqua-regia and selenium was determined by hydride Atomic Absorption. Total selenium levels ranged from 0.1 to 1.6 µg/g with a mean (±s.d.) of 0.476(±0.278) in the 0 to 15 cm sampling depth samples, and from 0.001 to 2.3 µg/g with a mean (±s.d.) of 0.474±0.335 µg/g in the 15 to 30cm sampling depth samples. While these levels fall well within the global range for selenium in soil estimated by Plant *et al*. (2003), some small areas of Alberta (also identified in Manitoba and Saskatchewan) are known to be toxic to livestock because of much higher levels of this element (Fleming 1980). Selenium deficient soils are much more common in Alberta; the "white muscle disease" in selenium-deficient livestock is reported to be prevalent in this province (Penny 2004).

Mermut *et al.* (1996) investigated the trace elemental composition of agricultural soils from the Brown and dark Brown zones in southwestern Saskatchewan. A total of 341 soil samples from surface horizons (Ap) and associated parent materials (C) were collected from 13 sites. The investigators noted that these soils had been cultivated and fertilized for 30 years. Triplicate soils samples were treated with $HF\text{-}HClO_4\text{-}HNO_3$ and analyzed by ICP-MS. On a dry weight basis, the selenium concentrations from the Ap and the C horizons ranged from 7.12 to 10.32 µg/g (mean=8.49 µg/g) and 7.37 to 12.68 (mean=9.3 µg/g), respectively. These concentrations are elevated when compared to Se levels found in other parts of the country and elsewhere in the world. The highest Se concentrations were found in the heavy clay soils. However, selenium was reported to be the only element of the entire suite of 68 elements analyzed where the total soil clay content and the total Se amount from both horizons were not significantly correlated. It should also be noted that these levels are well above those reported by other investigators in Saskatchewan and the authors did not provide any discussion to explain this apparent aberration.

Preliminary data from an elemental survey of Saskatchewan surface soils (A horizons) by the Saskatchewan Land Resource Unit of Agriculture and Agri-Food Canada and various other partners indicate selenium levels ranging from 0.2 to 0.8 µg/g (L.M. Kozak, Agriculture and Agri-Food Canada, 2005, pers. com.).

In 2004, the U.S. Geological Survey partnered with Canada (Geological Survey of Canada, and Agriculture and Agri-Food Canada) and Mexico to initiate pilot studies for a proposed soil geochemical survey of North America (Smith *et al.* 2005). Soil was sampled along two continental-scale transects. One north-south transect extended from northern Manitoba to the U.S.-Mexican border near El Paso in 2004 to be extended into Mexico in 2005. The other transect followed the 38th parallel in the U.S. from coast to coast. A total of 32 Manitoba sites selected in both agricultural and forested areas comprised the Canadian component of the survey. At each Canadian site, soil samples were collected at various depths and analysed for about 40 elements after a near-total four-acid extraction. Samples (n=32) analysed for Se were determined by hydride generation-atomic absorption spectrometry. The selenium content of the surface soil samples (0-5 cm) ranged from <0.02 to 1.2 µg/g with a mean (calculated assuming levels <0.2 to be equal to ½ the detection limit) and median of 0.3 and 0.2 µg/g, respectively.

In 2002, Manitoba Conservation conducted a survey of produce and soil from 11 gardens to determine levels of metals in the context of a human health risk assessment (Jones and Henderson 2006). Nine sites were selected within the city of Flin Flon where soils are expected to be impacted by emissions from the Ni-Cu HBM&S smelter. In addition, 2 other sites outside Flin Flon were selected (Cranberry Portage, 38.23 km from Flin Flon, as a minimally impacted site) and The Pas (115.01 km from Flin Flon, as a control site). Selenium levels were determined by ICP/MS on strong-acid digested (HCl/HNO_3) sieved (10 mesh) surface (top 10 cm) soil samples. The mean Se content of the 9 Flin Flon sites (n=3 samples per site) ranged from 0.4 to 5.7 µg/g. The overall mean and median for all the Flin Flon sites combined were 1.8 and 1.4 µg/g, respectively. The site closest to the smelter (No: TQ0164) contained the highest Se levels (range: 5.3 to 6.3 µg/g). The mean Se content of the garden soils of Cranberry Portage and The Pas were 0.3 and 0.5 µg/g, respectively (Jones and Henderson 2006).

Haluschak *et al.* (1998) conducted a survey of southern Manitoba soils to assess background levels of trace elements in agricultural soils and to determine their geological distribution. Soil samples from 121 areas were collected from the A horizon (0-15 cm) and the C horizon (50-60 cm deep, to include parent materials). Total selenium concentrations were determined in 618 soil samples by Atomic Absorption Spectroscopy (AAS) after treatment with a mixture of HF-$HClO_4$-HNO_3, and a detection limit (DL) of 0.2 µg/g was achieved. Samples with concentrations below the DL were assigned a value equal to ½ DL in the statistical calculations. Selenium levels ranged from < 0.2 to 3.8 µg/g with a mean (± s.d.) of 0.5± 0.4 µg/g, a median of 0.4 µg/g and a 95th percentile of 1.2 µg/g. Soils with the highest selenium levels were those associated with shales as parent materials, particularly those from the Keld Member soil series and those contained in the alluvial soils of the Manitoba Escarpment. The authors also noted that the selenium levels increased with increased soil clay content, as expected.

Ontario/Quebec

A multi-element profile of indoor dust in relation to outdoor dust and garden soils (0-5 cm) was conducted for the city of Ottawa, Ontario (Rasmussen *et al.* 2001). This city represents an urban centre with a low concentration of heavy industries. Random samples of house dust as well as street dust and garden soil (within 15 m of each residence) were collected from 10 zones in the city of Ottawa. Metal content (dry weight) was determined by inductively coupled plasma mass

spectrometry (ICP-MS) after digestion by $HF/HNO_3/HCl$ (hydrofluoric acid, nitric acid and hydrochloric acid; perchloric acid was also used for the digestion of street dust samples). The minimum detection limit for selenium was 0.5 µg/g. Selenium concentrations in garden soil ranged from 0.3 to 1.2 µg/g (n=50). The concentration representing the 95th percentile was 0.9 µg/g. An arithmetic mean of 0.7 µg/g and a geometric mean of 0.6 µg/g were reported. The selenium concentrations in adjacent street dust samples (n=45) ranged from 0.1 to 1.1 µg/g, with a 95th percentile concentration of 0.8 µg/g and arithmetic and geometric mean values of 0.5 and 0.4 µg/g, respectively. In house dust (n=48), selenium concentrations ranged from 0.3 to 6.8 µg/g with a 95th percentile concentration of 2.2 µg/g and arithmetic and geometric mean values of 1.2 and 1.0 µg/g, respectively.

In 1994 Geological Survey of Canada conducted a second ultra-low density regional geochemical survey in Ontario south of Sault Ste.Marie using the same sampling and analytical techniques as those employed 2 years earlier in the Prairies. The summary statistics for Se determinations in 294 surface soil samples (top 25 cm) showed a range of 0.1 to 3.9 µg/g, a mean (± s.d.) of 0.46 µg/g (±0.38) and a median of 0.4 µg/g. The occasional higher Se levels in Ontario surface soils are attributed to the presence of both sulphite mineralization in Precambrian rocks and the presence of Cu-Ni smelters in the Sudbury area (R.G. Garrett, Natural Resources Canada, 2005, pers. com.)

In 1990, surface soils (0-5 cm) were sampled from 12 urban locations in Windsor and from 18 rural locations in Essex County (Gizyn 1994). The soil samples were collected as part of a baseline study of soil, produce and air quality prior to operation of the Detroit municipal waste incinerator. In urban soils, selenium concentrations (dry weight) ranged from 1.04 to 2.03 µg/g with an arithmetic mean of 1.59 µg/g. Selenium concentrations in rural soils were lower, ranging from 0.52 to 1.30 µg/g with an arithmetic mean of 0.89 µg/g.

Farm fields receiving no sludge were sampled, to a depth of 15 cm, at 228 locations across the agricultural belt of Ontario and analysed for selenium (Frank *et al.* 1979). The soil samples were collected from orchards and vineyards (n=38), from vegetable producing farms, from cash crop farms and from unimproved pastures. An additional 30 samples were taken from farm fields having received at least one and not more than 5 applications of sludge. Selenium concentrations in soils having received no sludge ranged from 0.10 to 1.67 µg/g dry soil with a mean (±s.d.) of 0.35(±0.22) µg/g and clay soils contained not significantly more selenium than sandy soils averaging 0.48 µg/g and 0.27 µg/g, respectively. In comparison, the sludged soil contained even lower concentrations of selenium (mean (±s.d.): 0.37(±0.22) µg/g; range: 0.10 to 1.67 µg/g). In the data analysis, Frank *et al.* (1979) indicated that agricultural activities had no significant effect on the selenium content of soils nor did the application of sludges.

In Ontario, surface soils (0-5 cm) not impacted by point sources of pollution from old urban parkland sites and rural parkland sites were analysed for selenium by Hydride Flameless Atomic Absorption Spectrometry (HYD-FAAS) (OMEE 1994). In old urban parkland soils (n=60), selenium concentrations (dry weight) ranged from 0.83 (lower concentration limit or LCL) to 1.7 µg/g (upper concentration limit or UCL). Rural parkland concentrations (n=101) ranged from 0.67 (LCL) to 2.0 µg/g (UCL). The Ontario Typical Range (OTR) 98th percentile values, encompassing 98% of the selenium concentrations in the sites sampled, were 1.3 µg/g for old

urban parkland soils and 0.93 µg/g for rural parkland soils (OMEE 1994).

In the Port Hope area, Ontario a total of 74 surface and sub-surface samples from 10 sites subjected to potential airborne uranium deposition were obtained and analyzed for about 50 elements in the context of site-specific environmental and human health assessments (Sheppard *et al.* 2004). The samples collected in 2002 were digested in aqua-regia and analyzed by ICP-MS. The selenium levels ranged from 0.00 to 7.52 µg/g with a mean (±s.d.) and median of 2.05 (±1.81) µg/g and 1.69 µg/g, respectively.

In a study of selenium uptake by plants, Lévesque (1974b) determined selenium levels in 10 untreated Ontario soils. The levels ranged from 0.20 to 0.74 µg/g with a mean of 0.50 µg/g.

Atlantic

Metal concentrations in garden soils were measured as part of a multi-media analysis of metals in urban and rural sites in New Brunswick (Pilgrim and Schroeder 1997). Selenium concentrations (dry weight) were measured in triplicate soil samples (depth not reported) collected from 9 urban gardens in East Saint John (ESJ), 2 urban gardens in West Saint John (WSJ), and 1 rural garden in Fredericton. Soil concentrations were determined by ICP-MS after nitric and hydrofluoric acid digestion. Selenium concentrations from all 3 sites were below detection (<1 µg/g).

In Nova Scotia, as part of a human health risk assessment for the area North of Coke Ovens (NOCO) Site, JDAC Environment (2001a, b) reported levels of metals, TPH/BTEX and PAHs in surface soils collected within North Sydney (urban reference area) and from an area south of Sydney (rural reference area). For metals, including selenium, surface soil composite samples (0-5 cm, 3 test holes blended) were collected and analyzed by U.S. EPA Method #3050A (treatment with nitric acid, hydrogen peroxide and weak hydrochloric acid (0.1 M), unspecified instrumentation (FLAA or ICP-MS)). A total of 90 surface samples were taken randomly from an urban area of North Sydney considered not to be significantly impacted by the historical coke oven and smelting operations (JDAC Environment 2001a). Fifty-six percent of the analyzed samples were below the limit of detection (LOD) of 1 µg/g and the maximum selenium level was 2 µg/g; an arithmetic mean of 0.70 µg/g was calculated (a value equal to ½ LOD was substituted for the samples below detection and incorporated in the calculations) and the 97.5 percentile was 2 µg/g (JDAC Environment 2001a). For the rural reference area, 91 surface soil samples were taken along 4 concentric arcs, 5 to 20 km from the centre of Sydney, in areas downwind from the industrial point sources (JDAC Environment 2001b). Similar statistical analysis of the data was performed as those from urban areas. However, 5 samples judged to be contaminated by anthropogenic inputs were excluded from the data set. The selenium levels ranged from 1.0 to 2.0 µg/g with an arithmetic mean concentration of 1.035 µg/g and the 97th percentile value was 2.0 µg/g (JDAC Environment 2001b).

Gupta and Winter (1975) analyzed 66 soil samples from 8 soil series for total selenium content in Prince-Edward-Island where soil is known to be deficient in this trace element. The reported Se levels from the 8 soil series were stratified according to 4 soil pH ranges (<5.5, 5.6-5.8, 5.9-6.1, and >6.2). Overall individual Se values ranged from 0.09 to 0.60 µg/g and the soil series means ranged from 0.208 to 0.330 µg/g for Queens clay loam and Dunstaffnage sandy loam,

respectively. The overall weighted mean (by number of sites) was calculated to be 0.229 µg/g.

2.5.4 Groundwater

Naturally occurring selenium concentrations in groundwater are generally low, typically much less than 1 µg/L. Canadian data on levels of selenium in groundwater are limited mostly to a few reports from the Prairie Provinces (excluding Manitoba) and from British Columbia, some reporting elevated levels of the element, especially in shallow wells. Lower selenium concentrations, although above the Guideline for Canadian Drinking Water Quality (Maximum Acceptable Concentration (MAC) of 10 µg/L (Health Canada 1992; 2006a), have been reported in deeper wells supplying drinking water to the municipality of Walkerton, Ontario (this data set is described in the Drinking Water section below).

Levels

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 (Alberta Agriculture, Food and Rural Development 2002). Selenium was found in shallow clay-rich nitrate containing aquitards where levels of the element usually exceeded the MAC. About 8.5% of groundwater samples from a shallow sandy aquifer below an irrigated manured field contained selenium in excess of the MAC (10 µg/L). A further analysis of groundwater data from shallow wells in southern Alberta shows that increasing levels of selenium have a tendency to be associated with increasing levels of salt and nitrate. Selenium was detected in 43% of 173 farm wells in southern Alberta, of which 8% of detected levels exceeded the MAC (10 µg/L) (Alberta, Agriculture and 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 dryland saline soils in southern Alberta. Selenium was detected in 86% of the 42 samples and levels (as dissolved Se) ranged from <141 to 6,080 µg/L; the arithmetic mean (±s.d.) and median were 1,820 ±1,520 µg/L and 1,570 µg/L, respectively. According to Outridge *et al.* (1999), the 6,080 µg/L value is among the highest selenium concentrations in water ever reported. Like much of the shallow groundwater associated with dryland saline soils in the North American Great Plains, this water supply is unsuitable for human and livestock consumption with 85.7% of the samples exceeding both the Guideline for Canadian Drinking Water Quality (10 µg/L) and the Canadian Water Quality Guideline for the protection of agricultural uses - livestock water (50 µg/L). The authors speculate that the elevated selenium content of the groundwater is related not only to localized geological formations and processes but also to the use of selenium as a feed supplement, a common agricultural practice.

Domestic wells in three areas of Northern Alberta were tested for arsenic and other elements, including selenium (Alberta Health and Wellness 2000). Annual median and average selenium concentrations measured in wells from the Aspen Regional Health Authority (RHA) were 1.0 and 0.1 µg/L, respectively; those from the Lakeland RHA were 0.2 and 0.7 µg/L, respectively; and those from the Keeweetinok RHA were 0.1 and 0.2 µg/L, respectively.

Selenium levels above the MAC were reported in Saskatchewan groundwater. A pilot project on the groundwater quality of domestic wells located in the southeastern, the southwestern and the Moose Jaw/Regina areas of the province was undertaken during 1996/97 by SaskWater, the province's Crown water utility service provider delivering potable and non-potable water supplies and other services (Shaheen, undated). Groundwater from 93 wells was sampled and analyzed for a suite of chemical parameters; 16% of the wells (n = 10) exceeded the MAC (10 µg/L) for selenium.

Outridge *et al*. (1999) also listed unpublished groundwater selenium data obtained from the Saskatchewan Department of Resource Management. Total number of readings per site, and maximum and median selenium concentrations in groundwater were reported as "total selenium" in µg/L for Webb (town well, n=4, 20 and 15), Balgonie (well, n=6, 19 and 15), Corderre (well, n=5, 22 and 10), Keeler (town well, n=2, 94 and 90), Regina (private wells, n=32, 390 and 1) and for Zehner (private wells, n=13, 130 and 75); and as "dissolved selenium" in µg/L for Zehner (private wells, n=4, 96 and 45).

In the Vanscoy/Grandora area of Saskatchewan, shallow wells (n=48, depth ≤30.5 m) tested in November/December 2004 for trace metals contained between <0.1 and 39 µg Se/L, with mean and median values of 6 and 1.2 µg Se/L, respectively; 18.7% of the wells exceeded the MAC (10 µg/L) (Saskatchewan Watershed Authority 2005). In comparison to the province as a whole, shallow wells (depth ≤30.5 m) sampled throughout the province under the Rural Water Quality Advisory Program of the Saskatchewan Watershed Authority contained comparable selenium levels, with values ranging from <1 to 410 µg/L and a mean of 10 µg/L and a median of <1 µg/L.

Selenium concentrations were determined as part of a study of groundwater conditions of the Columbia Valley Aquifer, a rural and agricultural area south-west of Chilliwak near Cultus Lake, British Columbia (Zubel 2000). Thirty-eight groundwater samples collected from different locations were tested for "total selenium" and five samples contained selenium levels above the MAC, with values ranging from 70 to 170 µg/L.

2.5.5 Sediments

In the Bay of Fundy, selenium sediment concentrations were found to range from 0.11 to 0.36 µg/g (Loring 1979). Sediments collected from two mining-impacted lakes in Northwestern Quebec (Lake Dufault and Lake Duparquet) had mean selenium concentrations of 7.2 (range 1.4 to 14.5) and 0.5 (range 0.2 to 0.8)µg/g respectively (Speyer 1980). Both lakes are similar in all respects except for the physical features of the sediments; while Lake Dufault has a heavy-metal enriched sludgy sediment layer, Lake Duparquet sediments are mainly fine silty material (Speyer 1980). Sediment selenium concentrations in the Great Lakes are normally less than 1 µg/g dry weight (IJC 1981). Traversy *et al.* (1975) reported that selenium sediment concentrations (presumed to be total selenium) in the Great Lakes ranged from 0.2 to 2 µg/g with lake averages ranging from 0.63 to 1.0 µg/g.

2.5.6 Ambient Water

Selenium concentrations in water vary considerably depending on local geological conditions and anthropogenic activities involving the use and release of selenium. Selenium concentrations in Canadian surface waters have been reported to range from 0.01 µg/L to 4 µg/L (NAQUADAT 1985). Hodson *et al.* (1983) reported that the selenium concentration in the Great Lakes ranges from 0.01 to 5 µg/L, while Traversy *et al.* (1975) reported a range of <0.1 to 0.8 µg/L and an overall average of <0.1 µg/g for selenium on the Great Lakes. The selenium concentrations in the waters of two small mining-impacted lakes in Northwestern Quebec were below a detection limit of 0.1 µg/L (Speyer 1980). However, average water column Se concentrations in coal mining areas of B.C. can reach 28 µg/L in tributaries and 13 µg/L in rivers (Chapman 1999). A water quality survey of 60 rivers in Nova Scotia and New Brunswick found selenium water concentrations to be below a detection limit of 1.0 µg/L in all but two samples; the samples exceeding the detection limit were less than 1.4 µg/L (Dalziel *et al.* 1998). Other studies have found selenium concentrations in freshwater to range from 0.1 to 400 µg/L (Wilber 1980; Lakin and Davidson 1967; Sakurai and Tsuchiya 1975). The higher concentrations were detected in waters seeping from naturally-occurring seleniferous deposits.

In seawater, selenium concentrations have been reported to range from 0.06 to 0.12 µg/L (Lakin and Davidson 1967; Burton and Statham 1982). Coastal and estuarine waters also typically contain selenium within this concentration range (Cutter 1989). Generally, under ambient conditions, selenium is detected infrequently in surface waters and groundwater (ATSDR 2003). For example, examination of the EPA STORET database for North Carolina found that only 3.3% of 657 surface water samples exceeded a detection limit of 1.0 µg/L, with a maximum reported concentration of 12 µg/L (NCDNR 1986). However, in regions of high natural selenium enrichment, groundwater concentrations as high as 600 µg/L have been reported (Glover *et al.* 1979).

Higher levels of selenium tend to be found in irrigation return waters, seeps, springs, and shallow wells where seleniferous soils may contribute to the selenium content of the water (ATSDR 2003). In a survey of 107 irrigations and 44 livestock well water supplies in California, selenium concentrations ranged from <10 µg/L to 272 µg/L (Oster *et al.* 1988). Waters that receive industrial discharges also tend to have elevated selenium concentrations. Studies have indicated elevated selenium in refinery effluents discharged into San Francisco Bay (maximum 156 µg/L) (Cutter, 1989); agricultural drainage waters at the Kesterson Reservoir (maximum 1350 µg/L) (Maier *et al.* 1988); in ponds downstream of the Kesterson Reservoir (maximum 200 µg/L) (Ohlendorf and Hothem 1995); fly ash settling basin effluent (up to 50 µg/L) and sewage outflows (up to 280 µg/L, depending on level of sewage treatment) (Lemly 1985; Baird *et al.* 1972).

2.5.7 Drinking Water

Selenium occurs naturally in drinking water, though usually in trace amounts. In Canada, drinking water is routinely monitored for the presence of selenium for surveillance purposes. As noted in the Groundwater section, the Canadian Guideline for Drinking Water Quality for selenium is expressed as a Maximum Acceptable Concentration (MAC) of 10µg/L, established in 1978 and

updated in 1992; drinking water containing 10 µg Se/L would account for 10 to 25% of the estimated total daily intake of this essential trace element (Health Canada 1992; 2006a).

Levels

Raw, treated and distributed drinking water samples from 122 municipalities across Canada (representing 36% of the population) were tested for selenium in 1982. This survey showed levels of selenium in water supplies to be equal to or below the detection limit of 0.5 µg/L (Subramanian and Méranger 1984).

A 1975 study of 120 Manitoba drinking water supplies found that, although 93% of samples were below detection (<0.5 µg/L), 7% of the samples had selenium levels between 5.0 and 10.0 µg/L (Health Canada 1992). However, in areas of natural selenium enrichment, the selenium content may be higher.

More recently, only 2 of 179 Ontario municipal drinking water distribution systems monitored for selenium in 2000, 2001 and 2002 as part of the Drinking Water Surveillance Program reported levels above the MAC, with a maximum of 16 µg/L (OMOE 2006). The majority of the samples exceeding the MAC (41 samples out of a total of 43 samples) came from the Walkerton municipal groundwater supply where selenium is a known naturally occurring element in groundwater due to its location on the Salina Formation, a rock formation documented to leach selenium in groundwater (OMOE 2006).

In Manitoba, results of the 2002 drinking water quality test results for Winnipeg were obtained and the selenium average was less than 0.4 µg/L (range: <0.2 – 0.4 µg/L) (City of Winnipeg 2004).

Saskatchewan's regulated drinking water supplies, comprising about 593 licensed waterworks in the province, were tested for selenium, a known, naturally occurring element in drinking water supplies of the province, during the fiscal years 2002-2003 to 2005-2006. In 2002-2003, three exceedances over the MAC (range: 16 – 32 µg/L) were reported during that period. A few more exceedances were observed in subsequent years: in 2003-2004, two, and in 2004-2005 and 2005-2006, each, four exceedances over the MAC (Saskatchewan Environment 2006).

In Quebec, water distribution systems provide drinking water for 88% of the population. For the period extending from 1995 to 2002, the Ministère du Développement Durable, de l'Environnement et des Parcs du Québec (MDDEP) reported only two instances across all the distribution systems where the selenium concentration exceeded 10 µg/L with a maximum of 81 µg/L (MDDEP 2003).

Bottled water is not regulated under the Guidelines for Canadian Drinking Water Quality but is subjected to the provisions of the Food and Drugs Act and Regulations (Health Canada, Office Consolidation of the Food and Drugs Act and the Food and Drugs Regulations). In a survey of 188 samples of domestic and imported bottles of water sold in Canada in 1995-1996, Dabeka *et al.* (2002) found 24 samples (13%) exceeding the MAC (10 µg/L) for selenium. The highest concentrations were detected in a sample of mineral water (130 µg/L) and a sample of Canadian spring water (293 µg/L). In the same study, 11 samples of tap water were also tested for this

element, 7 of which were below detection and the highest reported level was 22 µg/L.

2.5.8 Biota

The selenium content of vegetation generally depends on the selenium content of the soil and the solubility of the selenium species that are present (Marier and Jaworski 1983). There are several species of grasses and herbaceous plants that accumulate selenium. These plants are termed primary and secondary accumulators and can contain from 100 to 100,000 µg Se/g and 25 to 100 µg Se/g tissue (dry weight), respectively (ATSDR 2003). Some species of primary accumulators from the genus *Astragalus* growing on seleniferous soils in the central U.S. and Canada are notoriously toxic to cattle and sheep that eat them (Neal 1995; WHO 1986; amongst others). Grazing animals that chronically consume a limited number of selenium accumulator plants over a period of weeks or months may be affected by "blind staggers" a condition that is eventually fatal to the animals. In primary and secondary accumulators, selenium uptake is generally proportional to the amount of selenium in the soil or other growth medium (Shane *et al.* 1988; Arthur *et al.* 1992). Selenium accumulators accumulate selenium in the form of methylselenomethionine, a water soluble compound which is not bound to proteins which is thought to be the basis of selenium tolerance in those plants (Jacobs 1989; Neal 1995). Non-accumulators typically contain less than 25 µg/g tissue (dry weight) (Rosenfeld and Beath 1964). Limited data on selenium levels in Canadian forages and grains have been located in the literature.

In the southern portion of the Prairie Provinces, which is classified as having seleniferous soils, relatively high concentrations of selenium have been reported to occur in wheat (Marier and Jaworski 1983). Within this region, it has been estimated that 80% of all forages and grains contain >0.1 µg/g (Kubota and Allaway 1972). Lindberg (1968) reported a selenium concentration of 1.3 µg/g in Canadian wheat. Miltimore *et al.* (1975) analyzed a variety of hay, silage and grain crops for selenium content in eleven agricultural regions of British Columbia (B.C.). Mean selenium concentrations (in µg/g) were 0.22 for legumes, 0.21 for grasses, 0.15 for oat forage, 0.13 for sedge hay, 0.08 for corn silage, 0.20 for barley and oats, and 0.32 for wheat. The authors compared these concentrations to other regions in Canada, and found that selenium content of B.C. forages was approximately four times greater than Northern Ontario forages (Lessard *et al.* 1968), and 10-fold greater than forage and grain grown on Prince Edward Island (Winter *et al.* 1973). However, the mean selenium content of alfalfa grass and oat forage was comparable to levels found in Alberta (Martin *et al.* 1973), although levels in Alberta hay were only one-third that of B.C. hay (Miltimore *et al.* 1975). Walker *et al.* (1941) reported a mean selenium concentration of 0.18 µg/g in Alberta wheat.

Vegetation grown on soils with underlying Paleozoic shales (seleniferous soils) in the eastern Yukon Territory were found to contain mean selenium concentrations ranging from 0.8 to 2.9 µg/g (Fletcher *et al.* 1973). Vegetation grown on adjacent non-seleniferous soils was found to have selenium concentrations of less than 0.5 µg/g.

An analysis of Alberta barley grain and grass-legume roughage found mean selenium concentrations of 0.211 and 0.176 µg/g, respectively (Redshaw *et al.* 1978). Barley grain

selenium concentrations were highest in the south and southeastern portions of the Province. No concentration gradient was observed for grass-legume roughage.

Limited data are also available for tissue concentrations of selenium in Canadian avian and mammalian wildlife. In addition to data on the selenium content of muscle tissues, liver and kidney are routinely analysed for trace elements because they are known to be target organs for selenium accumulation and relatively good indicators of selenium status in animals.

Carcasses of loons (*Gavia immer*) and common mergansers (*Mergus merganser*) from Ontario, Quebec and the Atlantic provinces were analyzed for mercury and selenium (Scheuhammer *et al.* 1998). Of the two piscivorous species, the merganser tissues contained the lowest selenium concentrations. In general, levels of selenium in both species were highest in liver and kidney and lowest in breast tissue. On a dry weight basis, average levels (±s.d.) of selenium in the loon liver (n=30), kidney (n=31) and breast muscle (n=36) were 15±7.4 µg/g, 15±8.9 µg/g and 2.8 ±1.0 µg/g, respectively. In the mergansers, reported mean levels (±s.d.) in liver (n=33), kidney (n=37) and breast muscle (n=62) were 9.7±0.7µg/g, 8.5±0.5 µg/g and 1.8 ±0.8 µg/g, respectively.

Harding *et al.* (2005) determined the selenium content of eggs of American dippers (*Cinclus mexicanus*) and spotted sandpipers (*Actitis macularia*) harvested along the Elk River, British Columbia downstream of several coal mines (exposed populations) and some upstream tributaries (control populations). The coal mine runoffs are a major source of elevated levels of selenium of the Elk River system where an average (±s.d.) selenium concentration 34.2 ±11.9 µg/L has been reported. In contrast, the average level of the element upstream of the mines was ≤1.4 µg/L. The mean selenium concentrations per nest in the exposed dipper eggs (n=40) and the reference eggs (n=58) were 8.4 and 7.4 µg/g (dry weight), respectively, levels not significantly different. Average selenium levels per nest determined in the exposed (n=111) and reference sandpiper eggs (n=112) were significantly different with values of 3.8 and 7.3 µg/g (dry weight).

In a study of mercury and trace elements in a pelagic Arctic marine food web in Northwater Polynyna, Baffin Bay, Campbell *et al.* (2005) determined the selenium content in muscle and liver from 12 species of seabirds and from ringed seals. In seabirds, mean selenium levels were lower in muscle than in liver and ranged from 1.46 µg/g (black guillemot) to 5.79 µg/g (black-legged kittiwake). Selenium levels in liver ranged from 3.12 (black-billed murre) to 11.22 µg/g (black-legged kittiwake). Mean selenium concentrations (±s.d.) in ringed seal liver and kidney were 0.44 ±0.07 µg/g and 10.19 ±6.72 µg/g, respectively.

Yukon moose tissues collected between 1994 and 2001 were analysed for selenium by ICP-MS after nitric acid digestion (Gamberg *et al.* 2005). Kidney (n=384), liver (n=56) and muscle (n=37) contained an average (±s.d.) of 1.02 ±0.44, 1.60 ±1.63 and 0.22 ±0.34 µg Se/g tissues, respectively. The black shales of the Selwyn Basin are known for their elevated selenium content (≤ 1 µg/g) in some Yukon areas where moose are likely to accumulate elevated levels of this element in their tissues. Although hepatic selenium levels were within the range known to be considered chronically toxic in cattle, the moose showed no signs of selenium toxicity.

In Nova Scotia, Pollock (2005) determined levels of trace elements in some organs of white-tailed deer (*Odocoileus virginianus*) and moose (*Alces alces*). On a dry weight basis, levels of selenium in the liver of white-tailed deer (n=54) ranged from 0.6 to 4.0 µg/g with a geometrical mean of 1.4 µg/g. Significantly ($p<0.001$) lower selenium levels were determined in the liver of moose (n=48; range: 0.28-3.57 µg/g; geometric mean: 2.9 µg/g). The highest selenium levels were measured in the moose kidney (n=21) with values ranging from 1.8 to 5.3 µg/g and a geometrical mean of 2.9 µg/g. Pollock (2005) compared these selenium levels with those reported in the literature for cattle and found that 15% of the analyzed deer liver samples were either selenium deficient or marginally deficient while 70% of those from moose were similarly deficient.

Various muscle tissues from 12 bull bison raised in the U.S. or Canada were analyzed for selenium by fluorometry (Driskell *et al.* 1997). The selenium content of 4 different meat cuts ranged from 0.23 to 0.27 µg/g.

Cattle raised in seleniferous areas of western Canada tend to accumulate much more selenium in skeletal muscle than those produced elsewhere in the country. Hoffman *et al.* (1972) reported average selenium levels (±s.d.) in beef muscle sampled from the following Canadian locations: St. Johns, Newfoundland (0.37 ± 0.06 µg/g), in Nappan, Nova Scotia (0.21 ± 0.07 µg/g), Kapuskasing, Ontario (0.07 ± 0.02 µg/g), in Brandon, Manitoba (1.24 ± 004 µg/g) and in Swift Current, Saskatchewan (1.33 ± 0.13 µg/g).

A Canadian survey to determine levels of residues of several elements in 5 species of slaughter animals was conducted between 1982 and 1989 (Salisbury *et al.* 1991). Although cattle liver and kidney samples were analyzed for selenium by hydride generation AAS, no muscle tissues were analysed for this element. Selenium was detected in all the kidney samples (n=1379) but only in 1315 of 1378 liver samples (detection limit was not provided). Selenium levels in kidney ranged from 0.09 to 4.10 µg/g with an average (±s.d.) of 0.92 ± 0.44 µg/g. In contrast, selenium concentrations in liver were much lower than in kidney and ranged from 0.04 to 1.22 µg/g with an average of the detected levels of 0.28 ± 0.19 µg/g.

Dietz *et al.* (1996) reported selenium content for a number of marine animals in the waters off Greenland. Geometric mean selenium concentrations were 0.49 µg/g for Iceland scallops, 0.54 µg/g for muscle tissue of Polar cod, and <0.2 µg/g for Arctic cod muscle tissue.

Evans *et al.* (2005) reviewed a large number of recent studies on persistent organic contaminants and metals present in freshwater biota of the Canadian Arctic and Subarctic. This overview presents the results of several studies on the selenium content of freshwater fish species regularly consumed by local inhabitants. Average selenium content in muscle was generally lower in sea-run chars from Northern Quebec than in landlocked chars from the central Arctic Archipelago with levels ranging from 0.14 to 0.60 µg/g and from 0.37 to 1.65 µg/g, respectively. Other studies on char conducted at a later date by Muir *et al.* (2001) and Muir and Köck (2003) showed that selenium was the most abundant element of the metal suite detected in landlocked chars from the central Arctic Archipelago. In a statistical analysis of temporal trends for selenium levels in char, Muir *et al.* (2005) indicated that levels of this element determined in 2003 were lower than those reported in 1997 to 2002 but did not show a consistent trend. Evans *et al.*

(2005) also presented data from various reports on metals detected in several fish species from a number of Northern lakes and from the Mackenzie River Basin. The average selenium content in fish muscle from Northern lakes was 0.29 µg/g in cisco, 0.23 µg/g in walleye, and ranged from 0.17 to 0.73 µg/g in char and 0.27 to 0.40 µg/g in lake trout. Mean selenium levels in fish muscle from the Mackenzie River Basin ranged from 0.06 to 0.79 µg/g in whitefish, from 0.08 to 0.38 µg/g in lake trout, from <0.05 to 0.22 µg/g in northern pike and from 0.14 to 0.26 µg/g in yellow walleye.

Fish from Lake Erie and Lake Ontario were found to contain fairly low selenium concentrations ranging from 0.01 to 0.08 µg/g (Traversy *et al.* 1975), about an order of magnitude lower than similar studies. An average concentration of 0.33 (range, 0.04 to 2.0) µg/g has been reported for freshwater fish from central Canada (Beal 1974). In a multi-elemental survey of Canadian freshwater fish, Uthe and Bigh (1971) determined the selenium content in five species from the industrialized lower Great Lakes Basin and from a remote lake in Manitoba. Selenium levels in "headless, dressed fish composites" from lakes Erie, Ontario and St. Pierre ranged from 0.19 to 0.38 µg/g; comparable levels ranging from 0.17 to 0.24 µg/g were measured in fish from Moose Lake, Manitoba. Levels of various trace elements were determined in 6 fish species collected from 14 Canadian lakes around the Great Lakes (Johnson 1987). The selenium content of whole fish composites was 0.78 µg/g in lake trout, 0.84 µg/g in whitefish, 0.55 µg/g in common sucker, 0.38 µg/g in yellow perch, 0.37 µg/g in northern pike, and 0.25 µg/g in walleye. Similar selenium concentrations in freshwater fish collected from 112 monitoring sites across the continental U.S. were reported as part of the National Contaminant Biomonitoring Program (Lowe *et al.* 1985). Mean geometric selenium concentrations (and range) measured in the 1978-79 and 1980-81 surveys were 0.46 (0.09-3.65) and 0.47 (0.09-2.47) µg/g, respectively.

Northern pike from two small Northwestern Quebec lakes (Lake Dufault and Lake Duparquet) were found to have muscle tissue selenium concentrations ranging from 1.1 to 3.0 µg/g and <0.2 to 0.62 µg/g, respectively (Speyer 1980). Both lakes were impacted by mining activity at the time of the study . More recently in 2001, Laliberté and Tremblay (2002) determined levels of selenium in sediments, water and fish from 3 lakes (Lac Chibougameau, Lac Aux Dorés and Lac Obatogamau) impacted by copper and gold mines and one control lake (Lac Waconichi) in Northern Quebec. Muscle composite samples from 5 fish were analyzed by AAS for selenium. Levels of selenium were similar across all 5 species regardless of where the samples came from (lake trout, 0.34 to 0.58 µg/g; northern pike, 0.36 to 0.41 µg/g; walleye, 0.32 to 0.42 µg/g; lake whitefish, 0.44 to 0.54 µg/g; and burbot, 0.25 to 0.31 µg/g), and a median of 0.37 µg Se/g was reported. In a 2002 follow-up study of the same Northern Quebec lakes, Laliberté (2004) determined similar selenium levels in fish muscle composites from 8 fish species (range of means: 0.18 to 0.61 µg/g). In addition, a large sample of lake minnows (n=337) was composited and the selenium content of this composite was reported to be high with a value of 1.47 µg/g; no discussion was provided by the author.

Levels of industrial metals in yellow perch (*Perca flavescens*) collected in 12 lakes near Sudbury, a copper and nickel mining and smelting town in Northern Ontario, were determined by Pyle *et al.* (2004). Based on the location with respect to the smelters and water quality parameters such as alkalinity, conductivity, hardness, pH, and a gradient of waterborne metals, the lakes were grouped into 3 clusters: Group 1 were three reference lakes with background

selenium levels (means: below detection-0.6 µg Se/L), Group 2 were three highly alkaline lakes with the highest waterborne selenium levels (means: 4.7-5.8 µg Se/L), and Group 3 were five lakes with high pH but with intermediate selenium levels (means: 1.0 - 2.3 µg Se/L). Levels of selenium in perch muscle (dry weight) followed the same pattern and were the lowest in fish from Group 1 lakes (means: 1.4 - 1.6 µg/g), the highest in Group 2 lakes (means: 7.7 - 24.0 µg/g) and intermediate in Group 3 lakes (means: 2.3 - 11.8 µg/g,). In the liver, selenium levels were about 3 to 4 times higher on a dry weight basis.

Casey and Siwik (2000) measured selenium levels in rainbow trout muscle and eggs collected from the McLeod, Gregg and Smokey River Basins and two lakes impacted by 2 surface coal mines in Alberta. Reference samples were collected upstream from the mines and from a reference lake. The selenium concentrations in fish muscle from reference and impacted sites ranged from 0.02 to 4.01 µg/g and 1.10 to 26.40 µg/g, respectively.

Average selenium levels in fillet of commonly consumed marine fish species from the North-East Atlantic ranged from 0.3 to 0.6 µg/g (Oehlenschlager 1997).

Lo and Sandi (1980) reported selenium concentrations in a variety of Canadian fish and fisheries products, based on unpublished 1974 data from the Inspection Branch, Fisheries and Marine Service, and Environment Canada. Selenium levels in a variety of regularly consumed fish species ranged from 0.18 to 1.9 µg/g (marine fish species), 0.79 to 7.15 µg/g (tuna), 0.45 to 1.99 µg/g (marine shellfish), and 0.04 to 1.77 µg/g (freshwater fish).

2.5.9 Commercially Available Foods

North American food commodities originate from widespread geographical areas within the continent and abroad. 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). Hence, wide regional variations in the selenium content of the food supply are expected. It is difficult, therefore, to establish the selenium content of commercial foods available in Canada with any certainty (Reilly 1996; Diplock 1993). For example, Pennington and Young (1990) reported a wide variation in the selenium content across the U.S. food supply from the results of Total Diet Study where 234 food commodities were surveyed; the coefficients of variation on the selenium content ranged from 19% to 47% (mean, 32%), coefficients of variation being much higher than those reported for other trace elements. Even higher coefficients of variation were reported by Wolf *et al.* (1992) in a limited study of the variability of the selenium content of 88 foods collected across the U.S in the preparation of a nationwide survey of the food supply; nearly one quarter of the reported coefficients of variation were shown to be 50% or greater, the maximum being 76%. Therefore, the selenium levels and estimated intakes presented herein should be interpreted with caution.

Further uncertainty arises from the use of different methodologies for food collection, food preparation (if any) and chemical analyses but there are conflicting reports on this matter. On the one hand, analysis of commercially prepared foods and baby foods indicated that processing may reduce food selenium levels (Morris and Levander 1970; Marier and Jaworski 1983). On the other, Higgs *et al.* (1972) found that cooking or processing has little effect on the selenium content of

foods.

Brazil nuts are known to be the single richest natural source of dietary selenium (Reilley 1996, 2004; Chunhieng *et al.* 2004; amongst others). The selenium content of Brazil nuts sold in the U.K. ranged from 2.3 to 53 μg/g (Thorn *et al.* 1978) [it should be noted that there is a mistake in the text when reporting levels of selenium in "μg/kg" instead of "mg/kg" as correctly indicated in Tables 1. and 2. of the article]. The same nuts sold in the U.S. averaged 36 ±50 μg/g (Reilley 2004). Concentrations as high as 126 μg/g are found in some particular cultivars (Chunhieng *et al.* 2004). The highest concentration ever reported was 512 μg/g detected in a nut from the Manaus-Belem region of Brazil (Chang *et al.* 1995).

As presented in the previous section, fresh fish and shellfish are a good source of dietary selenium. Average selenium concentrations were reported for the following seafood purchased in Ste-Foy, Quebec: oysters (0.938 μg/g), lobster (0.300 μg/g), frozen shrimp (0.793 μg/g), and flounder fillet (0.320μg/g) (Amer and Brisson 1973). Some fish fillets and other seafood bought in Guelph, Ontario were fairly rich in selenium (Arthur 1972) with levels ranging from 1.22 and 1.87 μg /g in sole, from 1.03 – 1.64 μg /g in halibut, from 0.85 to 1.21 μg/g in haddock and from 0.80 to 0.97 μg /g in cod, and from 0.67 to 0.82 in salmon steak, from 0.53 to 1.42 μg/g in scallops, and from 0.36 to 0.86 μg/g in trout.

Prepared or processed fish and shellfish products also contain notable levels of the element. The nutritional composition of British Columbia canned pink and sockeye salmon was investigated by Vanderstoep *et al*. (1990). The selenium content was determined by hydride generation AAS and was reported in units of mg per 100g of total can contents, presumably on a wet weight basis. The pink salmon samples (n=32) contained an average (±s.d.) of 0.024±0.004 mg/100g (or 0.24 ±0.04 μg/g) ranging from 0.017 to 0.035 mg/100g (or 0.17 to 0.35 μg/g). Slightly higher selenium levels were determined in the sockeye salmon samples (n=32) with an average of 0.030±0.005 mg/100g (or 0.30 ±0.05 μg/g) and a range of 0.017 to 0.047 mg/100g (or 0.17 to 0.47 μg/g). Selenium levels in canned salmon, shrimp and lobster purchased in Guelph, Ontario contained even higher levels of the element ranging from 1.03 to 1.97, 0.98 to 2.03, and 0.86 to 1.65 in μg/g, respectively (Arthur 1972). Somewhat lower mean selenium levels were determined in canned salmon (0.441 μg/g) and in canned shrimp (0.533 μg/g) obtained in Ste-Foy, Quebec (Amer and Brisson 1973).

Higham and Tomkins (1993) determined the selenium content of 15 brands and types of canned tuna available in the U.S. using differential-pulse cathodic-stripping voltametry. The four main commercial tuna species were albacore, yellowfin, skipjack and bluefin originating mostly from the west coast of Central and South America, from the West coast of USA and the mid-Atlantic. Selenium levels ranged from 0.38 μg/g (solid light tuna) to 1.25 μg/g (Albacore solid white) with an average (±s.d.) of 0.68 ±0.268 μg/g. Holak (1976) reported an average of 0.49 μg/g in canned tuna.

Grains and cereals are reported to be a good dietary source of selenium. However, the species and the variety of grains as well as geographical origin appear to be the main determinants in the selenium content (Arthur 1972). Analyses of the spatial distribution of the reported selenium levels in the wheat crops demonstrated that the highest Se levels occurred in Southwestern

Saskatchewan and adjoining Alberta and are associated with alkaline soils in which selenium, as selenates, is the most mobile (Gawalko *et al*. 2002). Various bread types made with Canadian wheat and rye contained approximately 0.4 µg Se/g (Dabeka 1994). Even higher selenium levels (in µg/g) were measured in wheat-based breakfast cereals purchased in Guelph, Ontario (puffed wheat cereals, 1.10 – 1.46; bran cereal, 0.74 – 1.08; and Red river cereals, 0.62 – 0.64) and in uncooked noodles (0.71 – 1.13), all made from the durum variety grown in Western Canada. Similar breakfast cereals made from Ontario wheat contained much lower selenium levels (0.03 – 0.17 µg/g). Corn- and rice-based breakfast cereals were poor sources of selenium. Although baked goods and cereals are not as rich in selenium as in some animal tissues, these foods are consumed daily in fairly large amounts and thus, contribute significantly to the daily selenium intake. Health Canada estimated from the results of the 1992 Total Diet Study (TDS) that 51% of the daily selenium intake of Canadians (males and females of all age groups) comes from baked goods and cereals (Dabeka 1994).

Vegetables are generally poor sources of selenium in the human diet with the exception of asparagus, mushrooms, garlic (Amer and Brisson 1973; Morris and Levander 1970), some vegetables of the cabbage family and some protein-rich legumes (Higgs *et al.* 1972; WHO 1987). Commercially cultivated mushrooms (*Agaricus bisporus*), especially the brown strains, can be a good source of selenium depending on the culture medium. Beelman *et al*. (2004) reported that one portion (85 g) of commercially cultivated Crimini (coffee-coloured) mushrooms is enough to supply an excess of 20% of the recommended dietary allowance (RDA) for selenium in adults. The selenium content of white mushrooms cultivated in the East, Midwest and West U.S. ranged from 1.0 to 2.1 µg Se/g (dry weight) (or from 0.08 to 0.17 µg/g (wet weight), as calculated values assuming 92% moisture content (U.S. DA 2002). Brown mushrooms contained slightly more selenium with levels ranging from 1.5 to 3.4 µg/g (dry weight) (or calculated to be from 0.12 to 0.27 µg/g (wet weight) assuming the same moisture content). Similar average concentrations were reported for commercially cultivated white mushrooms (0.11 µg/g) and brown mushrooms (0.25 µg/g) grown in Finland (Mattila *et al.* 2001). Fresh mushrooms from Ste-Foy, Quebec analyzed by Amer and Brisson (1973) contained about 0.3 µg Se/g on average.

Total Diet Studies and other food surveys

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). A total of 135 food composites (including 9 infant food composites) were prepared for consumption and analyzed in triplicates by cyclic and pseudocyclic INNA instrumental neutron activation analysis (Shi *et al.* 1999). Mean selenium concentrations in food composites are presented in Table 3. The highest selenium concentrations were determined in organ meats such as liver and kidney (1.044 µg/g), nuts and seeds (0.635 µg/g), canned fish (0.413 µg/g), and white bread (0.410 µg/g). Fruit and vegetables were generally poor sources of selenium in the diet.

Amer and Brisson (1973) analyzed by fluorometry 101 fresh or canned food samples purchased locally in Ste-Foy, Quebec for selenium content; the food samples were not cooked prior to chemical analysis. Foods containing the highest mean levels of the element were kidneys, (2.008 µg/g), seafood (0.588 µg/g), meat (excluding kidneys) (0.588 µg/g), and cereal and grain products

(0.363 (μg/g). Relatively high selenium mean levels were encountered in vegetables such as asparagus (0.860 μg/g), garlic (0.300 μg/g), cauliflower (0.262 μg/g), and eggplant (0.133 μg/g).

Arthur (1972) determined the selenium content of a wide selection of Canadian food products by fluorometric analysis. Samples of breakfast cereals and baby foods were obtained directly from the manufacturers while the rest of the analyzed food items were purchased from retail outlets in Guelph, Ontario. For each food item, three independent samples were tested. All samples were analyzed without any preparation (drying or cooking) and thus, data comparisons between the more recent TDS (Dabeka 1994) have to be made cautiously. Pork and beef kidney contained the highest levels of the element ranging from 2.85 to 3.67 μg/g and 1.85 to 2.70 μg/g, respectively. Fish and shellfish were also determined to be excellent sources of the mineral with the highest levels (μg Se/g) determined in canned shrimp (0.98 – 2.03), canned salmon (1.03 – 1.97), and in sole fillet (1.22 – 1.87). As reported in the Dabeka (1994) survey, fruit and vegetables did not contribute much dietary selenium.

With the exception of cheese (up to 0.124 μg Se/g), fresh dairy products in the above three Canadian food surveys contained relatively low concentrations of the element (≤0.033 μg Se/g). Amongst the meats (muscle from beef, pork, and chicken), the three surveys reported the highest levels of selenium in pork (Toronto, ≤ 0.307 μg/g, Guelph, ≤ 0.35 μg/g) (Dabeka 1994; Amer and Brisson 1973; Arthur 1972).

Levels of selenium in Canadian wines are surprisingly high. Taylor *et al.* (2003) determined the selenium content of 95 Canadian wines produced in Okanagan Valley (59 samples from 26 vineyards) and the Niagara Peninsula (36 samples from 17 vineyards). The wines from both regions were analysed by ICP-MS and a limit of detection of 0.8 μg/L was achieved. Mean selenium levels (±s.d.) in Okanagan and Niagara wines were 1.51±1.6 and 1.34 ±0.71 μg/L, respectively.

Infant Formulas

The selenium content of commercial infant formula preparations were purchased in Ottawa in 1992 (before selenium fortification was instigated) and during 1993 and 1994 (L'Abbé *et al.* 1996). The infant formulas generally contained more selenium than found in human milk. The average Se content of the formulas purchased before the Se addition ranged from 2.7 to 21 μg/L. The mean content of the element in selenium-fortified formulas ranged from 16 to 35 μg/L. The authors noted that all selenium-fortified formulas contained more selenium than specified on the label and found a wide variation between lots of the same products. Lower selenium levels of 15 μg/L and 8 μg/L were also determined in milk-based and soya-based formulas, respectively, as part of Health Canada's Total Diet Study of food commodities purchased in Toronto in 1992 (Dabeka 1994). Lowly (2004) reviewed the nutritional content of 12 milk-based infant formulas available in Canada as specified on the labels (not determined by independent chemical analysis). The selenium levels were zero (n=3 brands), 15 μg /L (n=3), 15.5 μg/L (n=2) and 20μg/L (n=4), In a similar survey, Clark Lowry (2005) reported the selenium content of formulas made from soy-proteins (range: 10 to 20 μg/L, n=5 brands) or from protein hydrolysates made with hydrolyzed casein with added essential amino acids (range: 19 to 20 μ/L, n=3).

2.5.10 Consumer Products

Selenium sulfide has anti-seborrhoeic and antifungal properties when used topically. This chemical compound is used at concentrations of 1% and 2.5% in shampoo for the treatment of dandruff and seborrhoeic dermatitis of the scalp, and of 2.5 % in lotions for the treatment of tinea versicolor, a common fungal infection of the skin (MedlinePlus 1993; Health Canada 2006b). Antidandruff shampoo containing selenium sulfide is classified as a natural health product in Canada (Health Canada 2006b). There is a paucity of information on the percutaneous absorption of selenium sulfide but data suggest that it is not likely absorbed through this route of exposure (WHO 1986; ATSDR 2003).

Selenium dietary supplements are also classified as natural health products in Canada (Health Canada 2004a; b). Several forms of selenium are used in commercial mineral and vitamin food supplements sold in Canada. Selenium sources in nutritional supplements include selenium salts such as the citrate and the selenate, selenium chelates from hydrolysed vegetable protein (HVP) and from hydrolysed animal protein (HAP) which are also known as selenium proteinates. Selenium supplementation is also available in the form of selenised yeast at label concentrations reaching 200 µg/tablet in over the counter supplements sold in Canada (Health Canada 2006c). Generally, the selenium content of multivitamin/multimineral supplements is $\leq$ 50 µg/tablet, usually taken once a day (Health Canada 2006c).

2.5.11 Human Tissues and Biological Fluids

Selenium is an essential nutrient and therefore, this element is expected to be detected at trace levels in all tissues and biological fluids in the human body. Selenium concentrations measured in the tissues and biological fluids of normal, healthy human subjects from Canada and elsewhere are presented in Table 6. The Canadian selenium values reported in Table 6 appear to be consistent with levels reported in the U.S.

Biological monitoring to determine nutritional selenium status or exposure has been used extensively and successfully in the past. The selenium content of whole blood is an adequate marker of nutritional status given a relatively steady dietary intake of selenium and may be of value for long-term selenium intake and status (Hambidge 2003). Plasma is another selenium biomarker being especially useful to determine the response to selenium supplementation in a relatively short time period (Hambidge 2003). Hair is not regarded as a good marker of selenium status in North America and other developed countries because selenium sulfide-based shampoo may falsify results; selenium is deposited on scalp hair where it is potentially absorbed into the shaft (ATSDR 2003; Combs and Combs 1986). However, selenium determination in hair has been a useful tool in epidemiological studies in selenium-poor areas of China where good correlations between selenium levels in hair and selenium status have been reported (Combs and Combs 1986). Urine is not always a good marker of selenium nutritional status because it may reflect short-term variations in exposure; determination of selenium in urine collected over 24 hours and expressed as an amount per day provides a more accurate indication of nutritional status. (ATSDR 2003; IOM 2000; WHO 1986; Iyengar and Wolttiez 1988; amongst others).

Breast Milk

Samples of Canadian breast milk and commercial infant formulas were analyzed for selenium by L'Abbé *et al.* (1996). Selenium levels were determined by a diamino-naphthalene (DAN) fluorometric method. The mature breast milk samples were collected in 1992 from Eastern Ontario donors and contained an average (±s.d) of 18 ±3.3 µg Se/L and ranged from 13 to 25 µg/L. After reviewing the literature on the selenium content of human milk, L'Abbé *et al.* (1996) concluded that the levels measured in Eastern Canada fall well within the upper range of those reported in the U.S., Europe and elsewhere. IOM (2000) reported a range of 15 to 20 µg Se/L human milk from Canadian and American mothers, based on studies performed in the 1980s. The selenium content of breast milk is influenced by the selenium status of mothers which is generally affected by the geographic location of the lactating women (Flynn 1992; Levander 1989). For example, selenium levels of milk from donors residing in 17 U.S. states were reported to be higher (28 µg/L) in areas with high soil selenium content and lower (13 µg/L) in areas with a low soil selenium with an overall mean of 18 µg/L (Shearer and Hadjimarkos 1975). In China, it is a very well known fact that there are some areas where human milk is known to contain extreme levels of selenium at both ends of the spectrum corresponding to the selenium content of the soil where the women live. An average of only 2.6 µg Se/L milk is reported in areas where soils are known to be selenium poor and levels as high as 283 µg/L are reported from a region where the soil contains elevated levels of the element (Levander 1989).

2.6 Existing Soil and Water Quality Criteria and Guidelines

Soil and water quality criteria and guidelines for selenium have been developed by several agencies, and are summarized in Table 7.

3. ENVIRONMENTAL FATE AND BEHAVIOUR IN SOIL

The environmental fate and behaviour of selenium 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. The most common selenium compounds in air include selenium dioxide, dimethyl selenide, dimethyl diselenide and hydrogen selenide; the latter is rapidly oxidized to elemental selenium and water as a result of its high reactivity (NAS 1976).

In soil pore water, the expected forms of selenium are the salts of selenic and selenious acids (selenates and selenites, 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 the selenates (NAS 1980). Elemental selenium is essentially insoluble. In acidic, organic-enriched soils, metal selenides, selenium sulfides and selenites are the predominant species. Selenides and selenium sulfide are also insoluble and tend to be immobile in soils (ATSDR 2003). In neutral, well-drained 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 (Mikkelsen *et al.* 1989; Geering *et al.* 1968). 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 sulfate 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 sulfate (Mikkelsen *et al.* 1989). Mixed results have been obtained with soil 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). It has been noted that accumulator types of plants are capable of accumulating more chemical forms of selenium than non-accumulator plants which typically accumulate selenates only (Underwood 1977). Mikkelsen *et al.* (1989) concluded that although

there are species-specific differences in the potential of plants to accumulate selenium, all plants are capable of accumulation of selenium when grown in soil containing moderate levels of water soluble selenium species.

Uptake of selenates by plants is an energy-dependent process whereas selenite uptake occurs passively (Shrift and Ulrich 1969; Ulrich and Shrift 1968). Selenate uptake in the root appears to follow the same transport pathway as sulfate, with the two ions competing for binding sites in plant root cells (Leggett and Epstein 1956). Once in the root, selenates are translocated unchanged in the xylem to the leaves, similar to sulfate translocation (Peterson *et al.* 1981). Selenites however, are rapidly converted to selenates or organic selenium compounds prior to translocation in the xylem (Asher *et al.* 1977).

Once accumulated by terrestrial plants, selenates can be converted to a variety of organic selenium compounds; mainly selenomethionine, selenocysteine and other seleno-amino acids which can become incorporated into plant proteins. This process is believed to occur mainly in the leaves, and the first step is the reduction of selenates to selenites, which subsequently bind to plant amino acids (Anderson and Scarf 1983). These conversions tend to occur most readily in primary accumulator plants (Marier and Jaworski 1983). However, the accumulator plants possess mechanisms that prevent the incorporation of seleno-amino acids into plant proteins, thus avoiding selenium-induced phytotoxicity (Brown and Shrift 1982). These organic selenium compounds, as well as selenates and selenites, can be released back to the soil environment when plants die and decay. In some accumulator plant species, selenates are slowly converted to organic forms and may comprise 40 to 50% of the total plant selenium content (Cappon 1981). In secondary accumulator and non-accumulator plants, total selenium concentrations are typically greatest in the roots, while in primary accumulators, total selenium concentrations are usually greatest in the foliage and stems (Marier and Jaworski 1983). The presence of sulfur in soils, such as through the use of sulfur-containing fertilizers, can decrease the uptake of selenium by terrestrial plants (Marier and Jaworski 1983; NAS 1976).

In a field study with winter wheat, Zhao *et al.* (2007) found that at higher levels of irrigation, the concentration of selenium in the wheat grains was decreased. Three potential reasons were suggested for this observation. First, there may have been a dilution effect because at higher irrigation there was also increased grain yield. Second, there was sulfur present in the irrigation water which could have inhibited the uptake of selenium. And third, there could have been increased leaching losses of available selenium from the soil due to the irrigation (Zhao *et al*. 2007).

Elemental selenium, organic, and inorganic selenium species may be methylated by soil microorganisms, with the methylated species subsequently volatilized to the atmosphere (Doran 1982; Fishbein 1983; Shamberger 1981). *Aeromonas* spp., *Flavobacterium* spp., and *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 (Fishbein 1983; Reamer and Zoller 1980; Zieve and Peterson 1981). Dimethyl selenone and dimethyl 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). These authors found that a greater amount of methylated selenium was volatilized from soil during the Spring, than in the Winter, Summer or Fall. 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). Some plant species, such as the accumulator plant *Astralagus*, also produce volatile methylated selenium compounds (Reamer and Zoller 1980; Zieve and Peterson 1984). Plants can also absorb volatile selenium compounds from the atmosphere, in addition to releasing them; however, this is a minor uptake pathway compared to root uptake from soil (Zieve and Peterson 1987). Biomethylation of selenium in soils occurs rapidly as long as it is present in a soluble state, or is present in high enough concentrations that microorganisms 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). Microbial oxidation of elemental selenium in soil can also occur, producing SeO_3^{2-} and SeO_4^{2-}. However, this process appears to occur at a relatively slow rate (Losi and Frankenberger 1998).

Selenium is well known to bioaccumulate in aquatic food webs and also appears to be capable of bioaccumulating in terrestrial organisms. Soil dwelling organisms such as earthworms can accumulate selenium to concentrations several times higher than concentrations in soil (Beyer *et al.* 1987). 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 the Kesterson reservoir area. While there has been some contradictory evidence as to whether selenium biomagnifies in food webs (Kay 1984; Lemly 1985), the bioaccumulation measurements by Wu *et al.* (1995) showed a clear increase in selenium concentration from the soil to plants, and from grasshoppers to mantis. The authors concluded that selenium biomagnification is occurring in the Kesterson region, but that it does not appear to be a simple stepwise magnification phenomenon. Further research was recommended to reveal the dynamics of selenium bioaccumulation in the insects and to better understand the complexity of soil chemical and biological factors.

4. BEHAVIOUR AND EFFECTS IN TERRESTRIAL BIOTA

4.1 Terrestrial Plants

4.1.1 Uptake, Metabolism and Elimination

As discussed above, the most important factors in determining the uptake of selenium by plants is the form and concentration of selenium in the soil. In soil, the phytoavailability of selenium is several times greater for selenate (Se^{+6}) than for selenite (Se^{+4}), while elemental selenium is largely unavailable (Mikkelsen *et al.* 1989). Plant uptake of selenate is believed to be active, while selenite uptake is thought to be passive (Peterson *et al.* 1981). Selenium uptake by plants is 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).

4.1.2 Bioaccumulation

Plants may accumulate selenium in amounts of less than 1 µg/g plant tissue up to several thousand µg/g plant tissue (James *et al.* 1989). Selenium accumulator plants (e.g., *Astragalus*, *Stanleya*, *Haplopappus* and *Xylorhiza*) can accumulate extremely high concentrations of selenium (up to at least 5 mg/g dry weight) (Salisbury and Ross 1985) and have frequently poisoned livestock. An unpleasant odour is often associated with accumulator species and livestock will tend to avoid eating these plants if other species are available (Olson 1978). Agricultural crops typically have a much lower tolerance for selenium (Mikkelsen *et al.* 1989).

The US DOE (1998) reviewed plant uptake factors (concentration in plant / concentration in soil) for selenium from 14 studies (156 observations) on various grasses and crop species. Uptake factors ranged from 0.02 to 77 (mean of 2.3), with the median value of 0.7 being adopted for guideline development (U.S. DOE 1998; Efroymson *et al.* 2001). In the regression of soil selenium concentrations against plant selenium concentrations, the model fit was significantly improved (i.e., r^2 was increased from 0.63 to 0.85) when pH was also considered, suggesting that pH significantly influences selenium accumulation (Efroymson *et al.* 2001).

4.1.3 Toxicity

It is thought by some researchers that selenium may have an essential role in plant growth, but this has not yet been confirmed (Mikkelsen *et al.* 1989; Efroymson *et al.* 1997a). The mechanism of selenium toxicity appears to be related to the replacement of sulfur, which is very similar to selenium in its chemical properties in cellular components (Mikkelsen *et al.* 1989). Selenium accumulators appear to tolerate high concentrations of selenium since these plants form mainly seleno-amino acids that are not toxic themselves, although this substitution for sulfur in proteins may disrupt normal metabolism, and are not incorporated into certain proteins

that could become toxic (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 25 to 30 µg/g tissue (Se^{+6} in soil); burseem >10 µg/g tissue (Se^{+4} in soil); pea/mustard/wheat 3 µg/g tissue (Se^{+6} in soil); wheat 10 to 15 µg/g tissue (Se^{+4} soil); and rice 2 to 67 µg/g tissue (Se^{+6} in soil).

Selenium (Se^{+6}), in the form of Na_2SeO_4, was shown to reduce shoot weight in alfalfa at 1.5 µg/g in soil, while 0.5 µg/g in soil had no effect (Wan *et al.* 1988). Shoot weight of alfalfa also was reduced when grown in soil containing 2 µg/g selenite (Se^{+4}), with greatest reductions in soils with the lowest organic matter (Soltanpour and Workman 1980). In wheat, 2.5 µg/g (lowest concentration tested) selenium in soil as Na_2SeO_3 resulted in decreased biomass and yield after 50 days (Singh and Singh 1978). At a selenium concentration of 1 µg/g, Carlson *et al.* (1991) demonstrated reductions of shoot weight by up to 59% on sorgrass seeds. In forage cowpea (*Vigna sinensis*), dry matter was reduced at concentrations of 2.5 µg/g when selenium was added as either elemental selenium, $Na_2SeO_3 \cdot H_20$, or H_2SeO_3 (Singh and Singh 1979). Selenium appeared to be more toxic in the form of Na_2SeO_4, resulting in reduced dry matter of forage cowpea at concentrations as low as 1 µg/g (Singh and Singh 1979). The toxicity of selenium to plants is summarized in Table 8.

4.2 Soil Microbial Processes

As described in Section 3, the soil microbial community may convert inorganic forms of selenium to methylated species which subsequently volatilize from the soil matrix into the atmosphere. This biomethylation process is believed to be a key process in the environmental cycling of selenium. The biomethylation process may also be a detoxification mechanism for some soil microbial species.

4.2.1 Toxicity

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 would be a soil concentration below which adverse effects may result from deficiency (Janda and Fleming 1978).

The effects of selenium on various soil microbial processes are presented in Table 9. The lowest effects concentration identified was 198 µg/g, where arylsulfatase activity was reduced in soils (Al-Khafaji and Tabatabai 1979). At 484 µg/g, selenium was reported to reduce respiration in native soil microflora by 43% (Lighthart *et al.* 1977). At 1975 µg/g, selenium was reported to reduce amidase activity in addition to soil acid and alkaline phosphatase activities (Frankenberger and Tabatabai 1981; Juma and Tabatabai 1977).

4.3 Terrestrial Invertebrates

4.3.1 Uptake, Metabolism and Elimination

The biological half-life of selenium in earthworms and ants was reported to be 64 days and 15 days, respectively (Wilber 1983).

4.3.2 Bioaccumulation

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 µg/g in *Aporrectodea tuberculata* and *Aporrectodea turgida*, respectively, in soil that contained <0.1 µg/g selenium. When soil selenium concentrations were increased to 6.7 µg/g, the earthworm selenium concentrations increased by approximately 5-fold. This same study also found that earthworm selenium concentrations were negatively correlated with soil calcium concentrations.

4.3.3 Toxicity

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 µg/g selenium (as sodium selenite) (Fischer and Koszorus 1992).

4.4 Livestock and Terrestrial Wildlife Species

4.4.1 Uptake, Metabolism and Elimination

Selenium absorption from the gastrointestinal tract varies with the chemical form and the amount ingested (NRC 1980). 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). This may offer a partial explanation of why ruminants are sensitive to dietary deficiencies of selenium.

Following administration of selenium in the diet of various animal species, such as beagle dogs, 9.5% has been observed to be absorbed through the gut (Weissman *et al.* 1983). Studies with rats suggest a higher absorption from selenides and elemental selenium (Franke and Painter 1938; Smith *et al.* 1938). A net absorption of radiolabelled selenium was observed to be 35% in sheep administered 0.35 µg/g in their diet and 85% in pigs administered 0.50 µg/g in their diet (Wright and Bell 1966).

In beagle dogs, it was observed that 20% of inhaled selenium metal aerosols were deposited in the lungs and upper respiratory tract (Weissman *et al.* 1983). Two hours following exposure, it

was determined that 80% of the radiolabelled selenium was absorbed though the lungs into the bloodstream (i.e., 80 of 20%, or 16%).

After absorption of selenium, the highest concentrations in animal tissues are found in the liver and kidney, however high levels also may be found in the pancreas, spleen, heart or lungs (Clayton and Clayton 1994; Marier and Jaworski 1983). Many selenium compounds are biotransformed in the liver to excretable metabolites (Friberg *et al.* 1986). Within 24 hours of the injection of female mallard ducks with small amounts of ^{75}Se as selenious acid, the concentration of the selenium isotope in the ovaries had more than doubled, which was considered to indicate a kinetic mechanism that may in part explain reproductive effects in waterfowl (Wilson *et al.* 1997). Selenium has been shown to cross the placenta in rats, dogs, mice and humans (Clayton and Clayton 1994).

Transfer of animals from seleniferous to non-seleniferous diets is followed by rapid, and then slow, loss of selenium from the tissues via bile, urine and/or expired air (NRC 1980). In monogastric species and ruminants urinary excretion is the main excretory pathway for selenium (NAS 1980). In poultry, it has been reported that selenium disappears from the liver within 8 to 14 days once the source is removed (Puls 1994). Biological half-lives have been reported for selenium in various species: 26 days in the mallard ducks (Halford *et al.* 1983); 10 days in pheasants (Wilber 1983); and 13 days in voles (Wilber 1983).

4.4.2 Bioaccumulation

Selenium concentrations in animal tissues tend to reflect dietary selenium concentrations, especially 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 a review of soil-to-small mammals bioconcentration factors, and derived a final mean and median soil-to-small mammal uptake factor of 0.3464 and 0.1619, respectively for selenium. In an earlier document, soil-to-mammal uptake factors were calculated at 0.143 and 0.109 for *Peromyscus leucopus* and *Oryzomys palustais*, respectively (Sample *et al.* 1996a). 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.

4.4.3 Toxicity

The environmental hazard of selenium was brought to the forefront as a result of findings at the Kesterson National Wildlife Refugee in California where thousands of waterfowl and shorebirds were either killed or deformed as a result of selenium contamination (Lemly 1997). No adverse impacts on wild mammals however, were demonstrated at this site (Clark 1987).

Selenium is nutritionally required by several animal species in small amounts (e.g., ruminants, chicks, 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). In addition to nutritional requirements, selenium has been reported to reduce the toxicity of arsenic, cadmium, mercury, silver and thallium (Marier and Jaworski 1983). Selenium deficiency diseases can occur in animals in

environments where selenium concentrations in soils and vegetation are low. Selenium dietary requirements for domestic animals typically range from 0.1 to 0.3 µg/g in dry matter (NAS 1980).

4.4.3.1 Mammalian Toxicity

In farm animals (cattle, sheep, pigs and horses), toxicity from intake of feed containing excessive selenium or from consumption of accumulator plants has resulted in blind staggers (i.e., disorientation, abnormal gait, circling) and/or alkali disease, which is characterized by emaciation, lameness, hair loss, and hoof malformations (NRC 1980; Clayton and Clayton 1994). Diets containing 1 to 44 µg/g dry weight have been reported to induce chronic selenium poisoning with symptoms including liver cirrhosis, lameness, loss of hair and hooves, emaciation, reduced conception and increased fetal resorption in various mammalian species (Harr 1978; NRC 1980). Other pathological signs of selenium toxicity in domestic animals include hepatic necrosis, nephritis, hyperemia, and ulceration in the upper GI tract. (Clayton and Clayton 1994). In domestic animals, lethal doses range from 1.5 to 3.0 µg/g bw in cats, dogs, and rabbits, and 9 to 20 µg/g bw in cattle and sheep (Puls 1994).

Sheep appear to be the most sensitive livestock species to selenium intoxication. A chronic oral LOAEL of 0.08 µg/g bw/day was reported for sheep orally administered selenium in the diet for one year (Puls 1994). Blind staggers and/or alkali disease have been reported in sheep exposed to dietary selenium concentrations ranging from 5 to 25 µg/g dry weight (Puls 1994). Cows exposed to various levels of selenomethionine through their diet for 120 days developed tissue lesions at a dose of 0.8 µg/g bw/day (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 µg/g bw/day, i.e., an exposure concentration of 8 µg Se/g food (Goehring et al. 1984a). A maximum tolerable dietary level of selenium of 2 µg/g was reported for protection of domestic animals (NAS 1980), which translates into a dose of 0.08 µg/g bw/day for dairy cattle. Eisler (1985) reported that `livestock toxicity is prevented if dietary concentrations of selenium do not exceed 5 µg/g in natural forage or 2 µg/g in feeds supplemented with purified selenium.

Five studies in rodents considered reproductive effects following oral exposure to organic or inorganic selenium compounds and are discussed below because of their relevance in the derivation of exposure limits for terrestrial mammals (Rosenfeld and Beath 1954; Schroeder and Mitchener 1971b; Nobunaga *et al.* 1979; Chiachun *et al.* 1991; Tarantal *et al.* 1991).

Rosenfeld and Beath (1954) exposed rats for 2 generations to 1.5, 2.5 and 7.5 mg/L of inorganic selenium as potassium selenate in their drinking water. No adverse effects on reproduction were observed in rats exposed to 1.5 mg/L in drinking water. In the 2.5 mg/L group, however, there was a 50% reduction in the fecundity of females, and fertility, juvenile growth and survival were adversely affected in the 7.5 mg/L group (Rosenfeld and Beath 1954). A NOAEL of 0.20 and a LOAEL of 0.33 µg/g bw/day were established by Sample *et al.* (1996b) based on this study. In a study by Lijinski *et al.* (1989), young rats (7 weeks of age) were exposed for 28 weeks to 0, 1.4 and 2.1 µg/g selenium as sodium selenite in the diet. At the 1.4 µg/g dose, rats displayed slower growth rates and reached a smaller maximum weight; however, the magnitude and statistical

significance of this effect were not documented. The results of a 3-generation study of mice exposed to a single dose of inorganic selenium as selenate in both their food and drinking water (0.056 µg/g and 3 mg/L, respectively), indicated an inhibition of reproductive success and failure to breed (Schroeder and Mitchener 1971b). Mice exposed to concentrations of 0.9 mg/L inorganic selenium as sodium selenite in drinking water for 30 days prior to reproduction and through day 18 of gestation showed no adverse effects on reproduction (Nobunaga *et al.* 1979). In a second group of mice exposed to 1.8 mg/L, offspring weight was reduced, but this effect was not considered biologically significant (Sample *et al.* 1996b).

Chiachun *et al.* (1991) exposed mice to 0.25 mg/L organic selenium as k-selenocarageenan in drinking water and reported no adverse reproductive effects. The duration of this study was unclear, but dosing appears to have occurred during gestation. Tarantal *et al.* (1991) reported no adverse effects in long-tailed macaques exposed to 0.025 µg/g bw/day organic selenium (L-selenomethionine) by nasogastric intubation for 30 days during gestation. Fetal mortality and adult toxicity were observed in macaques exposed to 0.15 and 0.3 µg/g bw/day for the same dosing period. However, these effects occurred within the range observed among the macaque colony at large and they could not therefore be attributed to the selenium treatment (Sample *et al.* 1996b).

A summary of NOAELS (no-observable-adverse-effect levels) and LOAELs (lowest-observable-adverse-effect-levels) for livestock and terrestrial wildlife identified in the literature are presented in Table 10.

4.4.3.2 Avian Toxicity

Environmental levels of selenium have been associated with embryonic mortality and teratogenesis in birds, particularly in the Western U.S. (Clayton and Clayton 1994). However, the evaluation of selenium toxicity is complicated by its occurrence in many different chemical forms that differ greatly in their toxicity to birds (Heinz 1996). Heinz (1996) notes that because elemental selenium is virtually insoluble in water, it presents little risk to birds; however, both selenite and selenate are toxic to birds. Organic selenium compounds, particularly selenomethionine, have been shown to be highly toxic to birds.

In mallard ducks fed a diet supplemented with selenium (as seleno-DL-methionine), no reproductive effects were reported at 3.5 µg/g, while the lowest effect level was 7 µg/g (Stanley *et al.* 1996). No significant histologic lesions were identified in adult male mallard ducks fed 0, 10 or 20 µg/g selenium in the diet (as seleno-DL-methionine) for 16 weeks (Green and Albers 1997). Suppression of certain aspects of the immune system was reported to occur in mallard ducks at 2.2 mg/L selenomethionine in drinking water (Fairbrother and Fowles 1990). Following exposure to 10 or 25 µg/g sodium selenite for 78 days, mallard ducks were found to have a significantly higher frequency of lethally deformed embryos compared to those exposed to 1 or 5 µg/g sodium selenite (Heinz *et al.* 1987). No adverse effects were reported at the 5 µg/g dose level. In a 6-week exposure of newly hatched mallard ducklings to sodium selenite or to selenomethionine, adverse effects on mortality were observed at 40 µg/g 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 µg/g (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 µg/g 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 µg/g (Heinz and Sanderson 1990).

In a feeding study in which mallard ducks were fed diets containing 8 and 16 µg/g selenomethionine for 100 days, malformations in unhatched eggs at incidences of 6.8 and 67.9%, respectively, were observed, compared with 0.6% for controls (Heinz *et al.* 1989). The most common malformations were of eyes, bill, legs and feet. In addition, ducklings hatched from the eggs of exposed ducks experienced reduced survival and growth even though fed a control diet following hatching. No adverse effects were seen in this study at dietary concentrations of 1, 2, or 4 µg/g selenomethionine. Screech owls exposed to 30 µg/g (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 (Wiemeyer and Hoffman 1996). However, owls exposed to 10 µg/g (dry weight) selenomethionine in their diet had no adverse effects on reproduction. Black-crowned Night Herons exposed to dietary concentrations of 10 µg/g 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 µg/g (dry weight) 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 µg/g (dry weight) 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 µg/g, 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).

A summary of NOAELS (no-observable-adverse-effect levels) and LOAELs (lowest-observable-adverse-effect-levels) for avian species identified in the literature are presented in Table 9.

5. BEHAVIOUR AND EFFECTS IN HUMANS AND EXPERIMENTAL ANIMALS

5.1 Human Exposure Estimates

The total Estimated Daily Intake (EDI) is expressed in units of "µg/kg bw/day" and is intended to represent the average exposure that a Canadian may receive of selenium. The normal sources from which a person may receive exposure to selenium is primarily thought to include foods, soil, air and water. Two types of consumer products, shampoo and cream containing selenium sulfide and selenium containing dietary supplements, were identified as potential sources of additional background selenium exposure in the general Canadian population. Employing average concentrations of selenium in the various media, and the typical rates of intake of those media for the Canadian population, the EDI for selenium was derived.

For the purpose of this exposure assessment, five age classes of the general population were considered: adults, teenagers, school aged children, toddlers and infants. Reference body weights and standard intakes of air, drinking water, and soil for each specified age class of the general population are presented in Table 13. The total daily selenium intake via food was calculated using food consumption rates for various age groups of Canadians (Table 4.) and mean concentrations for selenium determined in the 1992 Canadian Total Diet Study (Table 3.). Dietary intake estimates for selenium are provided for all age groups in Table 5.

No information on the use of dietary supplements by Canadians was located in the literature. American statistics on the use of supplements among adults (n=4862) aged 20 years and older are provided by the 1999-2000 National Health and Nutrition Examination Survey (NHANES) (Radimer *et al*. 2004). The prevalence of any dietary supplement use was 52%, of multivitamin/multimineral supplements use was 35% but the use was only 1.1% for supplements containing only selenium. Tablets of multivitamin/multimineral supplements available in Canada generally contain 50 µg Se or less, to be taken once a day (Health Canada 2006c). Assuming similar multivitamin/multimineral supplements use patterns by Canadian adults and that supplement tablets contain 50 µg of selenium, it is estimated that the average Canadian adult is taking 17.5 µg Se/day from dietary supplements. For toddlers, children and adolescents, selenium intakes from multivitamin/multimineral dietary supplements were not estimated because it appears that those supplements formulated for children do not contain any selenium. This information was obtained from websites of popular children brands of multivitamin/multimineral supplements such as "One-A-Day Kids", "Flintstones", and "Centrum Kids and Centrum Junior" formulations.

A background soil concentration of selenium of 0.7 µg/g is assumed, both for the purpose of deriving estimated daily (background) intake from soil, and for the derivation of the human health-based soil quality guidelines. This concentration is reflective of mean selenium concentrations measured in background soils collected in Alberta (Penny 2004; R.G. Garrett, Natural Resources Canada, 2005, pers.com.), Saskatchewan (R.G. Garrett, Natural Resources Canada, 2005, pers.com.), Manitoba (R.G. Garrett, Natural Resources Canada, 2005, pers.com.); Haluschak *et al.* 1998; and Smith *et al*. 2004), and Ontario (OMEE 1994; R.G. Garrett, Natural Resources Canada, 2005, pers.com.; Gizym 1994; Rasmussen *et al*. 2001). It appears that the

earlier data sets, such as those from McKeague *et al.* (1979), Lévesque (1974a, b) and Gupta and Winter 1975), report levels that are lower than those determined at a later date for the same areas; losses of volatile forms of selenium or other methodological problems may have occurred in these earlier determinations. As noted above, soil concentrations of selenium vary according to local geology. Although no single soil concentration can adequately represent the variance in background soil concentrations across Canada (Painter *et al.*, 1994), it is also essential to define a reasonable value for purpose of generic, national guidelines development. This background concentration is likely to be conservative for some regions in Canada but it is based on the most complete and reliable recent data sets available at the present time. Refer to Table 2 for more details on background selenium concentrations in Canadian soils. For indoor dust, an average background level of 1.2 µg/g measured in 48 Ottawa homes (Rasmussen *et al.* 2001) was used to estimate exposure from dust ingestion, assuming that an average person spends about 21 hours a day indoors and the rest of the time outdoors or in a vehicle (Leech *et al.* 1996; U.S. EPA 1997).

The typical selenium level used in the exposure estimates for air was 1.0 ng/m^3. This air concentration is considered representative of background selenium concentrations in Canada. This value is equal to the overall mean concentration of selenium in PM_{10} samples (n=2170) collected across Canada in 2002 and 2003 from 31 National Air Pollution Surveillance stations (T. Dann, Environment Canada, pers. com.). Limited data on selenium concentrations in indoor air were identified for the Windsor area (Bell *et al.* 1994); these proved to be essentially the same as those measured in ambient air. There was no need, therefore, to apportion inhalation exposure between indoor and outdoor environments for this exposure assessment. It is assumed that the majority of Canadians live in urban environments (Statistics Canada 2005) and that complete retention and absorption of inhaled selenium occurs.

For drinking water consumption, an urban exposure scenario is the most common situation expected to arise since 80% % of Canadians live in cities (Statistics Canada 2005) and 84% of these urban dwellers receive treated water supplies, mostly from surface water sources (Environment Canada 2005b). A concentration of 0.5 µg/L was considered to be the typical selenium level in Canadian drinking water supplies, based on extensive drinking water quality surveys performed across Canada. Slightly higher selenium concentrations were reported for drinking water from groundwater sources in the Prairies and from one municipality in Ontario.

Table 14 summarizes the daily intake estimates for total selenium via all media for five age classes of the Canadian general population. The estimated daily intakes (EDI) for adults, teenagers, school aged children, toddlers and infants were 135.7, 132.6, 112.9, 69.3, and 13.5 µg/day, respectively, which correspond on a body weight basis to 1.92, 2.22, 3.43, 4.20 and 1.65 µg/kg bw/day, respectively. The main source of selenium exposure comes from the diet constituting more than 99% of the EDI for all age classes.

5.2 Pharmacokinetics

5.2.1 Absorption

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). Virtually all of the

absorption of orally administered selenium compounds occurs in the small intestine, primarily the duodenum (Whanger *et al.* 1976), and also in the caecum and colon (Reilly 1996). Absorption is usually not regulated by the selenium status and thus, does not appear to be under homeostatic control (IOM 2000; WHO/FAO 2004). Generally, inorganic forms of selenium are not absorbed as efficiently as organic forms (U.S. EPA 1984). Selenate is nearly all absorbed but only a fraction of the dose is incorporated into tissues, the rest being eliminated quickly via urine (IOM 2000). Selenite, on the other hand, is not absorbed as well as selenate. Usually more than half of the ingested selenite is absorbed, but it is better retained into the tissues than selenate (IOM 2000). Both selenite and selenate are commonly used to fortify foods and as selenium sources in dietary supplements (IOM 2000).

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 is relatively low, often less than 25% (Combs 2001a; Navarro-Alarcón and López-Martínez 2000). The bioavailability of selenium from a mixed diet is estimated to be between 60 and 80% (Daniels 1996). The bioavailability of selenium from water is lower than from food (Valentine 1997). The bioavailability of organic selenium compounds in food supplements is much lower than those containing of inorganic forms (Navarro-Alarcón and López-Martínez 2000).

Similarly, selenium absorption does not appear to be under homeostatic control in rats (Levander 1986). Rats fed selenite absorbed 95% of the dose regardless of their usual dietary selenium intake, whether deficient or mildly toxic (Brown *et al*. 1972). In humans, it also appears that with both inorganic and organic forms, the degree of absorption is independent of the exposure level, but may be greater when a selenium deficiency exists (ATSDR 2003). Oral doses of selenomethionine, the main form of selenium in plants, appear to be retained more readily and metabolised more slowly in the human body. Furthermore, selenomethionine becomes incorporated with protein tissues whereas inorganic forms are absorbed into other tissues in humans (Levander 1986; WHO 1987, 1996).

When administered orally as solid sodium selenite, selenium absorption from the gastrointestinal tract ranges from 44 to 80% (Thomson 1974; Thomson and Stewart 1974; Stewart *et al.* 1978). Gastrointestinal absorption or orally administered aqueous sodium selenite was reported to range from 46 to 95% (Thomson 1974; Robinson *et al.* 1978; Thomson *et al.* 1978). Thomson (1974) observed 90 to 95% absorption of a low dose (0.01 mg/person) of aqueous sodium selenite. Absorption of a larger oral dose (1.0 mg/person) of either sodium selenite or selenomethionine was 90 to 95% and 97%, respectively (Thomson *et al.* 1978). Griffiths *et al.* (1976) reported 96 to 97% absorption of a single dose of 0.002 mg selenium administered as selenomethionine in solution. Robinson *et al.* (1978) found that 75% of selenomethionine, but only 46% of sodium selenite, was absorbed during a 10-11 week administration of solutions providing a dose of 0.0013 to 0.0023 mg/kg bw/day to New Zealand women. Thompson and Robinson (1986) reported an apparent absorption of selenate to be 95%, compared with 62% for selenite.

Experimental animals also appear to efficiently absorb selenium when administered by the oral route. Absorption efficiencies of 80 to 100% have been reported for rats administered selenium as sodium selenite, sodium selenate, selenomethionine, and selenocysteine (Furchner *et al.* 1975;

Thomson and Stewart 1973). Furchner *et al.* (1975) also reported that greater than 90% of an oral dose of selenious acid was absorbed in mice and dogs. However, an oral study with beagle dogs found that only 9.5% of administered selenium was absorbed in the gut (Weissmann *et al.* 1983). Virtually all of the absorption of orally administered selenium compounds occurs in the small intestine, primarily the duodenum (Whanger *et al.* 1976).

Selenium absorption in humans following inhalation exposure has only been studied in occupational settings. While these studies indicate that humans absorb selenium compounds in the lungs, no quantitative data are available. Weissman *et al.* (1983) reported that 20% of selenium inhaled as metal aerosols was deposited in the lungs and upper respiratory tract of beagle dogs. Of the deposited selenium, 80% was absorbed in the lungs within two hours. Thus, 16% of the administered dose was absorbed in the lungs. The authors also reported that 97% of selenious acid retained in the lungs was absorbed within two hours. The rate of selenium absorption from the inhalation route depends on the chemical form, with absorption of selenious acid aerosols occurring approximately twice as rapidly as absorption of elemental selenium aerosols (Weissman *et al.* 1983; Medinsky *et al.* 1981).

No dermal absorption of selenomethionine was observed in women tested with a maximum dose of 0.0029 μg/g selenium in a 0.05% L-selenomethionine lotion (Burke *et al.* 1992). However, as the concentrations tested were 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). Mice treated with a 0.02% selenomethionine lotion (0.29 μg/g bw/day) three times a week for 30 weeks showed significantly higher concentrations of selenium in the liver and ventral skin (away from application site) than controls (Burke *et al.* 1992). In rats, 9 to 27% of dermally applied ^{75}Se - selenious acid was absorbed (Medinsky *et al.* 1981).

5.2.2 Distribution

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). In humans, absorbed selenium is transported in the blood from the gastro-intestinal tract to the liver where it is reduced to selenide in erythrocytes (Reilly 2006). From there, it is carried in the blood, bound to α and γ-globulins, to various organs and tissues. Protein-bound selenium in human blood is usually found in the very low-density β-lipoprotein fraction (Reilly 2006).

Sodium selenite, administered in drinking water or the diet of rats and dogs, has been found to be widely distributed in the body, with the highest concentrations occurring in the liver and kidney (Furchner *et al.* 1975; Sohn *et al.* 1991; Thomson and Stewart 1973). Oral exposures to selenium compounds have also been found to result in the occurrence of elevated concentrations of selenium in the central nervous system (rats), the pancreas (in poultry), spleen, heart, lungs, and the milk of humans and various laboratory animals (Zi-Jian Jie and An 1992; Cantor *et al.* 1975; Archimbaud *et al.* 1992; Choy *et al.* 1993; Moser-Veillon *et al.* 1992; Marier and Jaworski

1983). Very low concentrations tend to be found in muscle, bone and blood, but extremely high selenium concentrations can often be detected in fingernails and hair (Marier and Jaworski 1983).

There are limited data available on the distribution of selenium following inhalation and dermal exposure. Burke *et al.* (1992) detected elevated selenium concentrations in liver and skin of mice subjected to dermal treatment with a selenomethionine-containing lotion. After inhalation of aerosols of elemental selenium and selenious acid, selenium concentrated in the liver, kidney, spleen, and lungs of beagles (Weissman *et al.* 1983).

A number of studies have found that selenium can be transferred to offspring both transplacentally and through lactation in rats, dogs, monkeys, mice and hamsters (Archimbaud *et al.* 1992; Choy *et al.* 1993; Willhite *et al.* 1990). Selenium has also been shown to readily cross the human placenta (Barlow and Sullivan 1982). It has been suggested that selenium may be transported between tissues via selenoproteins that carry selenium through the bloodstream (Daniels 1996; Magos and Webb 1980).

While most researchers have found that selenium intake occurs independently of the exposure level, others have observed it to be a linear relationship between dietary selenium and the amount of selenium in tissues (Marier and Jaworski 1983). In addition, the pharmacokinetics of selenium do not appear to be under homeostatic regulation; rather, selenium levels are dependent on excretory pathways (Marier and Jaworski 1983). However, there is evidence for lower-bound homeostatic control of absorption, as evidenced by increased absorption of selenium by healthy human males on a selenium-deficient diet (Martin *et al.* 1989).

5.2.3 Metabolism

The metabolism of selenium involves pathways for the incorporation of selenium into selenium-dependent enzymes (selenium is an essential trace element in all mammals), as well as pathways for the elimination of selenium from the body.

Selenium (as selenocysteine) is an integral component of the active site of the enzyme glutathione peroxidase (GSH-Px). This enzyme occurs in most animal and human tissues and is involved in the reduction and inactivation of hydrogen peroxide and lipid hydroperoxides (Stryer 1988). Thus, GSH-Px protects cellular and organelle membranes from peroxidative damage, and together with vitamin E, maintains membrane integrity (Shamberger 1986; Koller and Exon 1986). Other enzymes and proteins that incorporate or require selenium have also been identified in humans and animals (ATSDR 2003).

In general, the metabolic elimination pathway of selenium involves a series of mainly reductive reactions that ultimately result in the respiratory or urinary excretion of the products (Marier and Jaworski 1983). Selenites are the most studied selenium compound, with considerably less information available on the metabolism of selenates and organic selenium compounds. Metabolic reduction of selenite is believed to occur by catalysis of electron transfer involving low molecular weight sulfhydryl groups in the glutathione cycle (Vokal-Borek 1979). The reduction of selenite and selenate requires glutathione, the methylating agent S-

adenosylmethionine, NADPH, coenzyme A, ATP, and magnesium salts to provide optimal conditions for the reactions (Ganther 1979). The first step is the nonenzymatic reaction of selenite with glutathione to form selenotrisulfide derivatives. The selenotrisulfides are then reduced nonenzymatically in the presence of glutathione or enzymatically by glutathione reductase in the presence of NADPH to yield selenopersulfide (ATSDR 2003). Selenopersulfide is unstable and decomposes to glutathione and selenium, or is enzymatically reduced by glutathione reductase, in the presence of NADPH to form hydrogen selenide (Ganther 1980; Hsieh and Ganther 1975). Hydrogen selenide is then methylated by S-adenosylmethionine, in the presence of selenium methyltransferase to yield di- and trimethyl selenides (ATSDR 2003). The methylation of hydrogen selenide is believed to be a detoxification process with S-adenosylmethionine acting as the methyl group donor (Marier and Jaworski 1983). Methylated selenide species are then excreted through mainly the urinary or exhalation pathways. The majority of selenium metabolism appears to occur in the liver, based on the localization of selenium metabolites in liver endoplasmic reticulum (Marier and Jaworski 1983).

Selenates do not appear to be as readily converted to methyl selenides as selenites (ATSDR 2003). Studies of selenate metabolism are limited in mammals, but bacterial studies have indicated that selenate requires an activation step, prior to its reduction to selenite (Bopp *et al.* 1982).

5.2.4 Excretion

Excretion of selenium metabolites and end-products occurs primarily through the urinary route. Approximately 50-80% of absorbed selenium is eliminated in the urine (Marier and Jaworski 1983). Various selenium compounds are detected in urine such as selenomethionine, selenocysteine, selenite, selenate and selenocholine (Robinson *et al*. 1985). For a long time, trimethylselenide (TMSe) was believed to be the most important metabolite detected in human urine, representing 30-50% of the total selenium excreted via this route (Marier and Jaworski 1983). However, in the light of improved analytical technology, TMSe was recently found not to be a major constituent of human urine under normal conditions; however, this metabolite is produced in larger amounts when intake increases and thus, may be an important biomarker of excessive intake (Reilly 2006). It is now believed that two selenosugars, selenosugar **1** and its deacylate analogue, selenosugar **3**, are the most important constituents of human urine; a third selenosugar **2**, an analogue to selenosugar **1**, is believed to be a minor constituent (Suzuki *et al.* 2005). In humans, urinary excretion of selenium can be used to estimate the dietary intake [2 x (total selenium concentration excreted in 24 hr) = dietary intake] (Thomson and Robinson 1980); however, a number of other dietary factors can influence this relationship (e.g., sulfate supplementation increases urinary selenium excretion) (Greger and Marcus 1981).

In rats, the predominant urinary metabolite of selenium is trimethylselenonium, regardless of the form of selenium administered (Palmer *et al.* 1970). This metabolite has been reported to account for 30 to 50% of the total selenium excreted in the urine in those experimental animals (Palmer *et al.* 1970). However, a recent study also found selenium-containing sugars (selenosugars) in urine of rats fed selenite (Kobayashi *et al.* 2002). No studies on the metabolic formation of trimethylselenonium were identified in the literature but it does not appear to be a methylation product of dimethyl selenide (Obermeyer *et al.* 1971). The chemical form of

selenium is a key determinant in how rapidly selenium is excreted in urine (ATSDR 2003). In rats, urinary excretion of selenium was greater following oral administration of sodium selenite than that of selenomethionine (Thomson and Stewart 1973). It has been suggested that this may be a mechanism that contributes to the greater tissue retention of selenium from selenomethionine than from inorganic forms (Martin *et al.* 1989).

The main metabolite excreted via exhalation is dimethyl selenide. This elimination pathway is considered minor except in cases where extremely high levels of selenium are absorbed, such as acute intoxication scenarios (Marier and Jaworski 1983). Humans exposed to high concentrations of selenium have been reported to have a strong garlic odor to their breath, likely caused by the dimethyl selenide (Bopp *et al.* 1982; Civil and McDonald 1978), probably formed in the liver.

In mice, dimethyl selenide and dimethyl diselenide have been detected in expired air, following ingestion of unspecified amounts of sodium selenite, DL-selenomethionine, or DL-selenocysteine in drinking water (Jiang *et al.* 1983). One study found that increased dietary intake of protein and methionine can increase the amount of dimethyl selenide expired in air (Ganther *et al.* 1966).

Excretion of selenium metabolites in the feces or bile is considered insignificant, accounting for no more than 20% of the ingested selenium (Wilber 1980; NAS 1976). Faecal selenium consists mostly of unabsorbed dietary selenium with some selenium contained in biliary, pancreatic and intestinal secretions (Levander and Baumann 1966). However, some researchers have found that urinary and faecal excretion are similar in humans, with each route accounting for approximately 50% of the total selenium excretion (Stewart *et al.* 1978).

In general, the proportion of selenium excreted by any route (i.e., exhalation, faecal, urinary) is dependent on several factors, including level of exposure, chemical form, time since exposure, level of exercise, dietary selenium status, and lactation (ATSDR 2003). The exhalation route becomes more significant at high selenium exposure levels, and lactating women decrease their urinary and faecal excretion of selenium (ATSDR 2003; Moser-Veillon *et al.* 1992). Vigorous exercise appears to double urinary excretion of selenium (Oster and Prellwitz 1990). A greater proportion of selenium is retained within the bodies of individuals on selenium-deficient diets; thus reduced urinary and faecal excretion appears to be the homeostatic mechanism by which the body retains needed selenium (Martin *et al.* 1989).

In both humans and animals, the elimination of selenium appears to be triphasic, with the first phase characterized by rapid selenium excretion, the second phase slower, and the third phase much slower (ATSDR 2003). Average half-lives for sodium selenite in humans are approximately 1 day (phase 1), 8 to 9 days (phase 2), and 115 to 116 days (phase 3) (Thomson and Stewart 1974). Selenomethionine elimination in humans is also triphasic but slower than that of selenite, with average half-lives of approximately 0.4 to 2 days (phase 1), 5 to 19 days (phase 2), and 207 to 290 days (phase 3) (Griffiths *et al.* 1976).

5.3 Selenium Essentiality and Deficiency

There is scientific consensus that selenium is an essential trace element in both animal and human nutrition (NAS 1976; Bennett 1982; WHO 1987; Levander 1982; Robinson 1982; Foster and Sumar 1997). Selenium has even been reported to be an essential trace element in all vertebrates (Bowen 1979). The essential nutritional role of selenium was first discovered in farm animals in the 1950s and in humans in 1973 (Foster and Sumar 1997; Rotruck *et al.* 1973). Selenium plays significant roles in human metabolic processes (WHO/FAO 2004).

The strongest 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 (Chen *et al.* 1980; Xu *et al.* 1985; Sokoloff 1985, Levander 1986; WHO 1996 and Reilly 2004;2006). It has been suggested that Keshan disease may actually have a viral origin (*Coxsachie*), and that the disease is exacerbated by selenium deficiency, or a combination of selenium deficiency and low dietary protein (Beck *et al.* 1995; Guanquing 1979; Reilley 2004). .A low intake of iodine, mycotoxin contamination of cereals by molds (*Fusarium sp.*), poor diet and harsh living conditions have also been implicated in the aetiology of Kashin-Beck disease (Reilly 2004). Xia *et al.* (1993) have postulated that low selenium intake and oxidative stress are necessary for the occurrence of Keshan and Kashin-Beck disease. Insufficient levels of other trace nutrients and inflammatory processes are also believed to play a role (Reilly 2004). Administration of selenium selenite supplements to afflicted individuals resulted in the virtual elimination of both diseases from areas where they had previously been endemic (ATSDR 2003). The addition of selenium to table salt in Keshan disease districts in China was also a very successful intervention (Reilly 2006). More recently, Kashin-Beck disease has also been strongly associated with goiter in some regions of Tibet where people are deficient in both selenium and iodine (Moreno-Reyes *et al.* 1998). The latter investigaters have determined that iodine deficiency is a major co-factor in the development of Kashin-Beck disease.

In addition to Keshan and Kashin-Beck diseases, selenium deficiency has been associated with numerous other diseases, conditions and effects, including muscular dystrophy, malaria, cardiovascular diseases, loss of hair pigment, liver necrosis, hemolytic anemia, pre-eclapsia, spontaneous abortions, Sudden Infant Death Syndrome, macrocytosis, and hospital patients on intravenous total parenteral feeding solutions that contain no selenium (van Rij *et al.* 1979; Foster and Sumar 1997; Marier and Jaworski 1983; Rayman 2000; U.S. EPA 1991)

There have also been reports that supplementary selenium relieves a number of human health problems and diseases such as muscular discomfort, cardiomyopathy, arthritis, cataracts, cystic fibrosis, hemolytic anemia, multiple sclerosis, Kwashiorkor (a protein-calorie malnutrition), night blindness, and immunodeficiencies (Foster and Sumar 1997; van Rij *et al.* 1979; Johnson *et al.* 1981; Marier and Jaworski 1983).

Selenium and Cancer Protection

Since the 1970s, some studies in both human and animals have shown that selenium may have a

protective function against certain types of cancers (Foster and Sumar 1997; Marier and Jaworski 1983; Reilly 2004). Although there is ever-increasing evidence from recent human studies that higher selenium status or selenium supplementation may reduce cancer risk, the association between cancer and selenium is still not completely established (Reilly 2004, 2006). In her recent review, Rayman (2005) stated that evidence of the cancer preventing properties of selenium from geographic, prospective, and intervention studies in humans and from experimental animal studies is becoming quite strong, especially in the case of prostate cancer.

A relationship between selenium and cancer has been shown in a nested case-control study within the Health Professionals' Cohort Study that demonstrated an inverse relationship between higher selenium status (as measured in selenium content of toe nail clippings) and prostate cancer (Yoshizawa *et al.* 1998). That study involved nearly 34,000 men and showed that men in the highest quintile of selenium status were less likely of developing advanced prostate cancer as those in the lowest quintile. On the other hand, the Nurses' Health Study Cohort involving more than 60,000 American nurses in the early 1980s failed to show any relationship between higher toenail selenium and a reduced cancer risk (Garland *et al*. 1995). In another case-control study on the potential role of selenium (as measured by selenium content of toe nail clippings) in the etiology of breast, colon and prostate cancer in Montreal between 1989 and 1993, Ghadirian *et al.* (2000) found no association between toenail selenium and breast or prostate cancer but observed a statistically significant inverse association between toenail selenium and the risk of colon cancer in both men and women.

In China, several large-scale intervention trials involving supplementation of various combinations of vitamins and trace elements were undertaken (Reilly 2004; 2006). Two of these trials were conducted in the Linxian area of China where the highest rates of oesophageal cancer have been reported worldwide. More than 30,000 subjects were recruited and assigned into 4 groups receiving different combinations of various vitamins and trace elements, some at levels two to three times the U.S. RDAs (Blot *et al.* 1993). Cancer mortality was significantly reduced in those receiving high levels of a mixture of β-carotene, vitamin E and selenium. However, the investigators were unable to attribute the reduced cancer mortality to selenium alone (Blot *et al.* 1993). In a follow-up survey of nutritional intake of Linxian residents, Zou *et al.* (2003) suggested that the reduction in oesophageal cancer was not due to selenium supplementation alone but to an overall improvement of the nutritional status through a better diet. However, in a subsequent prospective study of a smaller sample of randomly selected individuals, Wei *et al.* (2004) did find significant inverse associations between baseline serum selenium and death from esophageal squamous cell carcinoma and gastric cardia cancer. Another large-scale intervention trial in Qidong, China, a high risk area for primary hepatocellular carcinoma (HHC), was conducted between 1984 and 1990 (Huang *et al.* 2003). The trial involved more than 130,000 subjects where selenium-fortified salt was provided to the people of one township and none to the four other townships (Li *et al.* 1992). After six years, a significant reduction of HHC was observed in the group receiving fortification while no change was observed in those receiving no fortification (Li *et al.* 1992). Although it appeared at the time that there was a direct association between selenium and a reduction in HHC, this cancer has also been shown to be mainly due to exposure to hepatitis B virus and Aflotoxin B1 (Lunn *et al*. 1997; Hsia *et al.* 1992). It was also known that the population of Qidong was highly exposed to both the virus and the toxin (Huang *et al.* 2003). In addition, exposure to high concentrations of Aflotoxin B1 has been potentially

associated with a genetic mutation found in various human cancers (Niu *et al.* 2002). Consequently, the anti-cancer properties of selenium could not be fully attributed to the fall in liver cancer (Sakoda *et al.* 2005) in the Qidong population.

Large-scale intervention studies have also been conducted in the U.S. The Nutrition Prevention of Cancer (NPC) Trial was to investigate if selenium supplementation could reduce the risk of two forms of non-melanoma skin cancer, basal cell and squamous cell carcinoma (Clark *et al*. 1996). A total of 1,312 adults with a history basal cell or squamous cell carcinoma were recruited between 1983 and 1991 and given either 200 µg/day selenized yeast or a placebo. The results of the trial did not show a significant decrease in non-melanoma skin cancer with selenium supplementation. However, an analysis of secondary end points indicated significant reductions in total cancer mortality (37% reduction) and in the incidence of lung (46% reduction), colorectal (58% reduction), and prostate (63% reduction) cancers. The Selenium and Vitamin E Cancer Prevention Trial (SELECT) to study prostate cancer and the international Prevention of Cancer with Selenium in Europe and America Trial (PRECISE) (several types of cancer) are underway (Reilly 2004; 2006).

In laboratory animals, "supranutritional" levels of selenium, doses at least an order of magnitude higher that those needed to prevent deficiency, have been used successfully to protect against chemically-induced and spontaneously occurring tumours in laboratory animals (Combs and Combs 1986; Combs 2001a; Ip and Ganther 1992; Whanger 1983, Ip 1998; amongst others). Levander (1987) hypothesized that the "anti-cancer" protective effects of selenium are due to its roles in alleviating oxidative damage, altering carcinogen metabolism, and selective toxicity against rapidly dividing tumour cells. It is believed that the chemopreventive properties of selenium are probably due to the production of anticancer selenium metabolites (Ip 1998). Methylated forms of selenium such as methyl selenol appear to be important with respect to cancer prevention (Reilly 2004; Rayman 2005). Moreover, Rayman (2005) postulated that selenoproteins may also be directly implicated in the anti-cancer process by reducing DNA damage, oxidative stress, inflammation and other cellular activities.

Biological Roles of Selenium

Some 20 mammalian functional selenoproteins have been characterised in recent years and there is evidence from molecular studies that up to 40 selenoproteins may be present in mammals (Arthur 2003). There are three "families" of selenoenzymes: the glutathione peroxidases (GSPxs), iodothyronine deiodinases (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 and to act as the body's storage depot for selenium since their role in metabolism is dependent on an individual's selenium status (Sunde 1993; Foster and Sumar 1997; Reilly 2004). The GSHPxs function interdependently with the antioxidant vitamins A, C, and E in catalyzing the decomposition of hydrogen peroxide, lipid peroxides and hydroperoxides in the presence of glutathione (Combs and Combs 1986; Duthie *et al.* 1993). The three types of IDs interact with iodine to prevent abnormal thyroid hormone metabolism by regulating and producing active thyroid hormones; types 1 and 2 are involved in the synthesis of active T_3 whereas types 3 are involved in the catalysis of T_4 to inactive T_3 hormones (Foster and Sumar 1997; Reilly 2004; 2006). The TRs participate in the reduction of nucleotides and the binding of transcription factors in DNA. Furthermore, TR withTrx (thioredoxin) forms a powerful system known as TR/Trx system that is involved 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). Other important selenoproteins are known to play important biological roles. Selenoprotein P, the major selenoprotein in blood, is probably involved in the protection of endothelial cells against perioxinitrite, a reactive nitrogen species formed during inflammation as it may also be a selenium transporter between the liver and other sites where selenium is needed to produce other selenoproteins (Rayman 2000; Reilly 2004; 2006). Selenoprotein W is found mainly in muscle tissues, brain, testis and spleen and it appears to be involved in skeletal and heart muscle metabolism (Rayman 2000; Reilly 2004; 2006). Other selenoproteins are involved in the function of the male reproductive system and fertility by protecting developing and mature sperm cells from oxidative damage, by playing a role in sperm maturation, and possibly, by protecting prostate secretory cells against the development of carcinoma (Rayman 2000; Reilly 2004; 2006).

Selenium and the Immune Function

There is convincing evidence suggesting a potential association between selenium and the immune function. Although not fully understood, adequate selenium intake is essential for the full expression of the immune function, and reduced selenium status (Reilly 2006). Even in selenium-replete subjects, supplementation has noticeable effects in stimulating the immune system, including improved activation of T cell and proliferation of *B* lymphocytes (Rayman 2000; Hawkes *et al.* 2001). The protective role of selenium has been implicated in the immune response to inflammation, and to various bacterial, mycological, viral infections (Rayman 2000). For example, selenium containing GSHPxs has an effect on neutrophil function by protecting the neutrophils from their own radical production while also protecting cells against invaders (Arthur *et al.* 2003).

Selenium Requirements

Selenium is a rather 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. Selenium essentiality was recognised by the U.S. National Academy of Sciences, the U.S. EPA, and U.S. Food and Nutrition Board who recommended a safe and adequate range of 50 to 200 µg per person per day for adults, with correspondingly lower ranges for infants and children (Robinson 1982; NAS 1980; U.S. EPA 1984; Lemly 1997). The safe and adequate range was extrapolated from human balance studies and laboratory animal studies (NAS 1980). A few years later, the U.S. National Research Council (NAS 1989) established Recommended Dietary Allowances (RDAs) for selenium of 70 µg/day for adult men and 55 µg/day for adult women, based on a daily dose 0.87 µg/kg bw/day derived from a series of depletion studies carried out in Chinese males (Yang *et al.* 1989a,b; Yang *et al.* 1988; Levander 1991). RDAs for children and infants were extrapolated from the adult RDAs on the basis of body weight; for children, the RDA is set at 0.87 µg/kg bw/day (NAS1989). Selenium dietary requirements for pregnant or lactating mothers are greater than those for non-pregnant or non- lactating women, with RDAs of 65 µg/day and 75 µg/day respectively (NAS 1989).

More recently, 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) and for other vitamins and trace elements (IOM 2001). 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). In addition, an in-depth analysis of the health benefits and adverse effects of selenium were taken into consideration for setting DRIs. DRIs are normally developed for specific age and gender groups and physiological states for almost all population groups (IOM 2000, 2001). Hence, different values can protect sub-population groups at risk without being over-protective for the rest of the general population (Mertz 1998; Munro 1999). 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 (food and fortified food, drinking water, beverages and supplements), that is maintained under homeostasis in healthy populations (IOM 2000, 2001; Barr 2006).

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). Table 12 presents the selenium DRI values for various life stage groups.

The RDA is defined as "the average daily nutrient intake level sufficient to meet the nutrient requirement of nearly all (97 to 98 percent) healthy individuals in a particular life stage and gender group" (IOM 2000, 2001). The RDA for selenium is based on the amount of the element needed to maximize the synthesis of the selenoprotein gluthathione peroxidase, which is indicated by the plateau of activity of the plasma isoform of this enzyme (IOM 2000). No RDA was determined for infants up to one year of age for lack of data. Instead, an AI was determined, defined as "the recommended average daily intake level based on observed or experimentally determined approximations or estimates of nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate which is used when an RDA cannot be determined". The AI for infants up to 6 months of age was based on the estimated average selenium intake from breast milk on the basis that no cases of American or Canadian exclusively breast-fed infants showing signs of selenium deficiency had been reported. The IA for those from 7 months to 1 year of age is estimated from the selenium provided by 0.6L breast milk per day and by usual complementary weaning foods consumed by that age group (IOM 2000).

The EAR is defined as "the average daily nutrient intake level estimated to meet the requirement of half the healthy individuals in a particular life stage and gender group" (IOM 2000, 2001). EARs have not been determined for infants during their first year of life. For children and adolescents, no data were available to determine EARs; instead, the EAR is extrapolated downward from adult values with adjustments for metabolic body size and growth while taking into consideration the prevention of the onset of Keshan disease. For adults, the setting of the EAR was based on the plateau concentration of the plasma selenoproteins measured in two intervention supplementation studies that were designed similarly and conducted in both China and New Zealand (IOM 2000).

Some upward adjustments on the EAR and RDA values were made for pregnancy and lactation. The EAR was increased by 4 µg/day, to account for the fetal deposition of selenium during pregnancy. The RDA for this life stage was set at 60 µg/day to provide 120% of the EAR value for selenium, based on an estimated coefficient of variation (CV) of 10% (by definition the RDA is the EAR plus twice the CV to cover the needs of 97-98% of individuals in a group, rounded to the nearest 5 µg). For lactating females, an additional 14 µg/day were added to the EAR to provide for the estimated 14 µg selenium per day excreted in breast milk. The RDA was adjusted upwards to 70 µg/day, using the same assumptions made for pregnant females (IOM 2000).

The UL is defined as "the highest average daily nutrient intake level that is likely to pose no risk of adverse health effects to almost all individuals in the general population (IOM 2000, 2001). As intake increases above the UL, the potential risk of adverse effects may increase". The ULs for all age groups are presented in Section 5.6, Overall Human Toxicological Evaluation. The human and mammalian toxicology of selenium is discussed in detail in the following section (Section 5.4).

5.4 Human and Mammalian Toxicology

The specific mechanisms of toxicity by which selenium and selenium compounds produce toxicity are not well understood. However, it is generally believed that only the soluble forms of selenium are capable of causing toxicity as they are absorbed readily (ATSDR 2003). One theory for the mechanism of acute selenium toxicity is that selenium inactivates the sulfhydryl enzymes that are necessary for oxidative reactions during respiration (Mack 1990; Lombeck *et al.* 1987). In addition, evidence that selenium can replace sulfur 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).

It should be noted that many manifestations of selenium toxicity are remarkably similar to those observed in cases of selenium deficiency, thus complicating the association of selenium levels in various media with adverse effects on organisms.

5.4.1 Mammalian Toxicology

5.4.1.1 Acute Toxicity

Oral Studies

The most acutely toxic selenium compounds by the oral route appear to be soluble selenates and selenites (Olson 1986). Oral LD_{50} values for sodium selenite have been reported to range from 4.8 to 7 mg/kg bw in rats, 1.0 mg/kg bw in rabbits, 3.2 to 3.5 mg/kg bw in mice, and 2.3 mg/kg bw in guinea pigs (Cummins and Kimura 1971; Pletnikova 1970). Rabbits orally administered

sodium selenite exhibited pulmonary congestion, hemorrhages, edema, labored respiration, muscular weakness and asphyxial convulsions (Smith and Westfall 1937).

LD_{50} values of 17 and 49 mg/kg bw were reported for mice and rats, respectively, after oral administration of selenium dioxide (Singh and Junnarkar 1991). Elemental selenium is of low acute toxicity due to its low solubility; a 10 day oral LD_{50} of 6700 mg/kg bw was reported by Cummins and Kimura (1971). An LD_{50} for 1-20% selenium sulfide dissolved in aqueous 0.5% methylcellulose, administered by gavage to rats, was 138 mg/kg bw (Cummins and Kimura 1971). By contrast, Henschler and Kirschner (1969) reported an oral LD_{50} value of 3.7 mg/kg bw for selenium sulfide administered by gavage to mice in aqueous 0.5% carboxymethylcellulose. An oral LD_{50} of 78 mg/kg bw in rats has been reported for 1% selenium sulfide shampoo (Cummins and Kimura 1971). The authors observed that anorexia and diarrhea occurred in rats at doses at or near the LD_{50} for selenium sulfide. Smyth *et al.* (1990) observed degenerative kidney and pancreatic changes in sheep acutely exposed to a single oral dose of sodium selenite at 5 mg/kg bw. Acute oral exposure of mice to selenium dioxide dissolved in water at 1.7 mg/kg bw caused a moderate reduction in alertness, spontaneous activity, touch response, muscle tone, and respiration (Singh and Junnarkar 1991).

Inhalation Studies

LC_{50} values for guinea pigs exposed via inhalation to hydrogen selenide for 1, 4, and 8 hours, were 12.7, 9, and 1 to 4 mg/m^3, respectively (Dudley and Miller 1941). Hall *et al.* (1951) observed no mortality in rabbits and guinea pigs exposed to elemental selenium dust at concentrations up to 31 mg/m^3 for four hours, every other day for eight days. However, surviving animals displayed a variety of effects, including pneumonitis, emphysema, and spleen and liver congestion. This same study found that rats exposed via inhalation to 33 mg/m^3 selenium dust displayed severe respiratory effects, such as hemorrhage and edema of the lungs; several test animals died.

Dermal Studies

No acute dermal studies for selenium compounds were identified in the literature.

5.4.1.2 Subchronic and Chronic Toxicity

Oral Studies

Male and female F344/N rats and B6C3F1 mice were orally administered sodium selenite and sodium selenate in drinking water for 13 weeks (NTP 1994). In the selenate group, 10 male and 10 female rats and mice received 0, 3.75, 7.5, 15, 30, or 60 mg/L. The selenite group of 10 male and female rats and mice received 0, 2, 4, 8, 16, or 32 mg/L. All rats exposed to 60 mg/L selenate died, while two female rats exposed to 32 mg/L selenite died. Rats and mice exposed to 30 mg/L selenate and 32 mg/L selenite had decreased body weights. Rat and mouse water consumption was found to decrease as the dose levels increased. Selenate concentrations greater than 7.5 mg/L, and all selenite concentrations were associated with increased incidence of renal papillary degeneration in rats. No lesions were observed in mice as a result of selenate or

selenite administration. Increases in estrous cycle length were also observed in female rats and mice during this study. A NOAEL of 0.4 mg/kg bw/day in rats for both selenate and selenite (15 and 16 mg/L, respectively), was estimated, based upon a consideration of all observed effects. In mice, a NOAEL of 0.8 and 0.9 mg/kg bw/day was estimated for selenate and selenite, respectively, based on body weight depression and decreased water consumption.

Post-weaning, 60-70 g male Sprague-Dawley rats were fed selenite or seleniferous wheat *ad libitum* at dose levels of 1.6, 3.2, 4.8, 6.4, 8.0, 9.6, and 11.2 mg/kg in a subchronic study (Halverson *et al.* 1966). A NOAEL of 4.8 mg/kg (40 µg/kg bw/day) was observed, based on a lack of effects at selenium levels up to 4.8 mg/kg. At selenium concentrations greater than 8.0 mg/kg, effects such as decreased liver and spleen weights, and reduced hemoglobin were observed. Mortality was observed in the three highest dose groups fed both selenite and seleniferous wheat. Mortality was 100% for the dose group fed wheat with a selenium concentration of 11.2 mg/kg. In addition, a significant growth reduction was observed for animals fed both selenite and wheat containing 6.4 mg/kg selenium or higher.

Koller *et al.* (1986) found that rats administered sodium selenite in drinking water at a dose of 0.7 mg/kg bw/day for 10 weeks displayed a reduction in humoral antibody production in response to an administered antigen. Reduced prostaglandin synthesis was also observed. At lower doses (0.07 and 0.28 mg/kg bw/day) natural killer cell (NKC) cytotoxicity was enhanced, while prostaglandin activity and delayed-type hypersensitivity were reduced. The authors reported a LOAEL of 0.7 mg/kg bw/day. A NOAEL could not be identified in this study due to the conflicting nature of enhanced NKC activity occurring at the same dose level as reduced prostaglandin activity and delayed-type hypersensitivity.

Administration of sodium selenite in drinking water at a dose of 0.28 mg/kg bw/day for 58 days resulted in the death of 25/50 male rats (Schroeder and Mitchener 1971a). There was no increase in mortality for female rats receiving the same dose for the same duration. Previously, Rosenfeld and Beath (1954) found that rats could tolerate a dose of 1.05 mg/kg bw/day in drinking water as potassium selenate for eight months; no mortalities were recorded. By contrast, decreased survival was reported in rats fed sodium selenate or selenite at a dose of 0.4 mg/kg bw/day for two years (Harr *et al.* 1967; Tinsley *et al.* 1967). At a dose of 0.25 mg/kg bw/day, hepatitis was commonly observed, with liver lesions occurring at doses as low as 0.10 mg/kg bw/day. Liver weights showed a dose-dependent decrease with increasing levels of selenate or selenite in the diet (Harr *et al.* 1967; Tinsley *et al.* 1967). Harr *et al.* (1967) also reported that sodium selenate or selenite caused softening of bones to occur in rats at doses as low as 0.2 mg/kg bw/day. A study with hamsters found no mortality associated with the dietary administration of sodium selenite at a dose of 0.42 mg/kg bw/day for 122-144 weeks (Birt *et al.* 1986).

Lifetime exposure of mice to sodium selenate in drinking water at a dose of 0.57 mg/kg bw/day resulted in amyloidosis of the lung, heart, and kidney in some animals (Schroeder and Mitchener 1972). Nelson *et al.* (1943) had previously reported that no effects on lungs occurred in rats administered 0.50 mg/kg bw/day in drinking water for two years. However, doses ranging from 0.25 to 0.5 mg/kg bw/day produced cirrhosis of the liver in rats in this study.

An NTP study found no respiratory or musculoskeletal effects in mice and rats administered selenium sulfide at doses of 464 and 31.6 mg/kg bw/day, respectively, by gavage once daily for 13 weeks (NTP 1980a). There was also a lack of gastrointestinal and renal effects in rats at the 31.6 mg/kg bw/day dose level. However, rats displayed liver necrosis at 31.6 mg/kg bw/day but not at a lower dose of 17.6 mg/kg bw/day. No hepatic effects were observed in mice dosed with 464 mg/kg bw/day but an increased incidence of interstitial nephritis was observed at this dose level; a dose of 216 mg/kg bw/day produced no renal effects in mice (NTP 1980a).

Rats fed selenite for 24-51 days at a dose of 0.75 mg/kg bw/day displayed red cell hemolysis (Halverson *et al.* 1970). Pigs administered sodium selenite in feed for eight weeks displayed hepatic effects at doses of 0.59 and 1.07 mg/kg bw/day (Mihailovic *et al.* 1992). A previous study had found a lack of hepatic effects in pigs at a dose of 0.47 mg/kg bw/day, administered as sodium selenite in feed for 35 days (Mahan and Magee 1991).

In another subchronic study, guinea pigs fed either organic selenium or inorganic sodium selenite were reported to have decreased serum protein levels, decreased SGOT enzyme activity and increased erythrocytic glutathione activity (Das *et al.* 1989a). In addition, animals fed sodium selenite were observed to have increased incidences of histopathological lesions of the liver, bile duct epithelium, lung, spleen, adrenal gland, testis and lymphoid organs. Microcytic hypochromic anaemia was also observed; this condition was most prominent in guinea pigs fed organic selenium (Das *et al.* 1989b). The authors found that co-administration of 10 mg/kg sodium arsenite in the diet had a protective effect against the selenium toxicity (Das *et al.* 1989a,b).

Salbe and Levander (1990) administered selenium to rats as selenomethionine or as sodium selenate. Rats given a diet deficient in methionine had a decrease in final body weight, with the most significant decrease observed in rats fed inorganic sodium selenate. Decreased rates of body weight gain are a common observation associated with subchronic or chronic oral administration of selenium compounds to experimental animals (ATSDR 2003).

In general, the available subchronic and chronic oral animal bioassays with selenium compounds are inadequate for determining a reliable NOAEL or LOAEL, due to the use of single doses, relatively short study durations, poor characterization of effects at certain dose levels, or because effects were observed at none or at all dose levels.

Inhalation Studies

No studies investigating health effects in animals following subchronic or chronic inhalation exposure to selenium or selenium compounds were identified in the literature.

Dermal Studies

No studies investigating health effects in animals following subchronic or chronic dermal exposure to selenium or selenium compounds were identified in the literature.

5.4.1.3 Reproductive and Developmental Toxicity Studies

The developmental toxicity of selenomethionine was studied by Tarantal *et al.* (1991) in long-tailed macaques. Forty pregnant macaques were dosed daily by nasogastric intubation with 0, 0.025, 0.15, or 0.3 mg/kg bw/day as selenomethionine through gestational days 20-50. There were no significant differences in pregnancy loss between treated and control animals. In addition, no statistically significant treatment-related effects were observed upon necropsy at gestational day 100. The authors found no evidence of any significant fetal developmental, teratogenic, or maternal effects at any of the doses tested.

Schroeder and Mitchener (1971b) orally administered 3 mg/L selenium as selenate (0.76 mg/kg bw/day) in drinking water to CD mice over three generations. No maternal toxicity was observed. However, there was a significant increase in young deaths in the F1 generation, as well as an increased number of runts in generations F1 through F3. In addition, breeding events were found to be decreased by the F3 generation.

Potassium selenate was orally administered to male and pregnant female rats through five breeding cycles at dose levels of 1.5, 2.5, or 7.5 µg/g selenium (Rosenfeld and Beath 1954). At the lowest dose tested, no effects on reproduction or the number of young were observed. At the 2.5 µg/g (125 µg/kg bw/day) dose level, there was a 50% reduction in the number of offspring produced. At the 7.5 µg/g (375 µg/kg bw/day) dose level, female fertility was decreased, as was the number of surviving offspring and the growth rate of the pups. Male fertility appeared unaffected.

Nobunaga *et al.* (1979) orally administered 3 or 6 µg/L selenium as selenite in drinking water to IVCS mice for 30 days prior to mating and throughout the gestation period. Upon sacrifice of the mice at day 18 of gestation, no significant differences between treated and control animals were observed with respect to fertility, number of litters, total implants, number of implants per dam, fetal and embryonic mortality, resorptions, litter size, gross or skeletal malformations. The only significant effect observed was a reduced body weight in surviving fetuses of mice administered 6 µg/L (780 µg/kg bw/day) selenium.

5.4.1.4 Carcinogenicity Studies

Generally, carcinogenicity studies with selenates, selenites, and organic selenium compounds have shown negative results. The animal carcinogenicity database is generally poor with a number of conflicting results that are difficult to interpret because of apparent anticarcinogenic activity, high toxicity of some selenium compounds, and flawed study designs. Furthermore, comparison of the available studies is made difficult because several different selenium compounds with varying degrees of bioavailability were used in the cancer bioassays (U.S. EPA 1991).

Selenium (as selenite, selenate, or organic selenium in forage) was first reported to be a possible carcinogen in rat studies conducted by Nelson *et al.* (1943), Seifter *et al.* (1946), Tsuzuki *et al.* (1960), and Volgarev and Tscherkes (1967). However, these studies suffered from a number of design flaws, short durations, incomplete quantification of results, and other inconsistencies that

make their findings questionable. For example, Nelson *et al.* (1943) reported tumours only in animals with cirrhotic livers, the investigators had problems discerning malignant tumours from non-malignant tumours, and a large number of animals had died of liver cirrhosis prior to the appearance of liver tumours. Volgarev and Tscherkes (1967) and Tsuzuki *et al.* (1960) had no control groups of animals. A follow up experiment by Volgarev and Tscherkes (1967), this time involving the use of a control group, found no increase in tumour incidence in 100 rats administered sodium selenate in the diet at a dose of 0.22 mg/kg/day for 25 months.

Wistar rats fed sodium selenite or selenate in their diet at concentrations ranging from 0.5 to 16 mg/kg for their lifetime developed no tumours (Harr *et al.* 1967; Tinsley *et al.* 1967). However, non-neoplastic liver effects, including hepatocyte hyperplasia were observed at dietary concentrations greater than 4 mg/kg selenium. As this study employed a large number of animals (1,437 were used in this study and 1,126 of them were necropsied), and both positive and negative controls, the negative result for carcinogenicity is considered to be a robust finding (ATSDR 2003).

Schroeder and Mitchener (1971b) administered 2 mg/L sodium selenite or selenate in drinking water to Long-Evans rats for one year, then 3 mg/L for the remainder of the animals' lifespan. Incidence of all tumours and of malignant tumours was significantly increased in the selenate-treated rats versus controls. Selenite-treated rats showed small number of tumours; however, this may have been due to the greater toxicity of selenite versus selenate which shortened the survival time of the selenite-treated group (U.S. EPA 1991). This study is considered inadequate as only heart, lung, liver, kidney, and spleen tissues were examined histologically, and an increase in longevity was observed in selenate-treated female rats (U.S. EPA 1991). Furthermore, this study was confounded by a virulent pneumonia epidemic (ATSDR 2003).

A subsequent study (Schroeder and Mitchener 1972), where Swiss mice were administered 3 mg/L sodium selenate or selenite in drinking water showed no significant increase in total tumour or malignant tumour incidence in treated versus control animals. Longevity was increased in male and female mice administered selenate, while the selenite-dosed females showed a decrease relative to control animals.

The only selenium compound that has been found to be carcinogenic in animals is selenium sulfide. This compound is not readily found in foods or the environment but is a pharmaceutical used primarily in anti-dandruff shampoos (ATSDR 2003). Chronic oral exposure to selenium sulfide was found to produce statistically significant increases in hepatocellular carcinomas and adenomas, and alveolar/bronchiolar carcinomas and adenomas in mice (NTP 1980a). However, dermal studies with Swiss mice where selenium sulfide was applied to the skin of males and females at doses of 0, 0.5, or 1.0 mg/kg bw/day for 86 weeks showed no significant difference in tumour incidence versus the control group (NTP 1980b). It should be noted however, that the application sites were not covered; thus, some ingestion of the selenium sulfide may have occurred through licking (ATSDR 2003). Another dermal assay tested Selsun, a prescription dandruff shampoo containing 2.5% selenium sulfides (NTP 1980c). Groups of 50 male and female Swiss mice were dermally exposed to 0%, 25%, or 50% solutions of Selsun in distilled water 3 days/week for 86 weeks. The equivalent doses were 0, 0.31, and 0.625 mg/kg bw/day, respectively. Incidences of alveolar/bronchiolar adenomas or carcinomas were significantly

increased over vehicle control male mice, but not untreated control males. No significant differences in tumour incidence were observed for female mice. This study may also have been confounded by animals licking the application sites.

Anticarcinogenicity of Selenium Compounds

Selenium supplementation has been shown to significantly inhibit tumours induced by chemicals, viruses, or ultraviolet radiation (ATSDR 2003). Clayton and Baumann (1949) were the first to report anticarcinogenic effects of selenium. They observed a 50% reduction in dimethylaminobenzene-induced tumour incidence in rats fed a diet supplemented with 5 µg/g selenium as selenite. Numerous studies since have described reduced incidence of induced tumours and neoplasms following the administration of selenium. Detailed discussion of these studies is beyond the scope of this document, however a comprehensive review of a large number of studies which have investigated the anticarcinogenic and anti-tumorigenic properties of selenium compounds can be found in Milner and Fico (1987).

5.4.1.5 Mutagenicity and Genotoxicity

Data on the mutagenicity of selenium compounds are inconclusive. Selenate and selenite, at a concentration of 12 mM, were found to be mutagenic in a reverse mutation assay using *Salmonella typhimurium* strains TA98, TA100, and TA1537 in the absence of rat hepatic homogenates (Noda *et al.* 1979). In a similar *Salmonella* assay, Lofroth and Ames (1978) found sodium selenate, but not selenite to be mutagenic. The actual strains used in this assay were not reported. Selenite was observed to produce DNA damage in *Bacillus subtilis* strains 17A and 45T while selenate produced a negative result in the recombinant assay (Nakamuro *et al.* 1976).

Some studies have found that selenium appears to decrease the mutagenicity of some mutagenic chemicals in rat liver cells (Gairola and Chow 1982; Schillaci *et al.* 1982).

In the Chinese hamster ovary cell assay for unscheduled DNA synthesis, sodium selenide, sodium selenite, and sodium selenate (listed in order of decreasing activity) caused an increase in unscheduled DNA synthesis with and without the presence of glutathione (Whiting *et al.* 1980). Sodium selenite was also found to cause an increased number of chromosomal abnormalities at a concentration of 10^{-5} M in rat lymphocytes (Newton and Lilly 1986). In human lymphocytes, selenite, selenious acid, selenic acid, and selenium dioxide produced chromosomal aberrations at a concentration of 2.6×10^{-6} M (Nakamuro *et al.* 1976). Elemental selenium, selenium dioxide, sodium selenide, and sodium selenite (in order of decreasing activity) induced an increase in sister chromatid exchanges in human whole blood cultures; sodium selenate tested negative in this assay (Ray and Altenburg 1980).

In general, data on the genotoxicity of selenium compounds are inconclusive. Inorganic selenium compounds have been observed to have both genotoxic and antigenotoxic effects, with antigenotoxic effects generally occurring at lower exposure levels than the genotoxic effects (ATSDR 2003). The presence of glutathione in the test mixtures has been found to enhance the genotoxicity of sodium selenite, sodium selenate, and sodium selenide in bacterial test systems

(Whiting *et al.* 1980). This has also been observed in a number of mammalian test systems (ATSDR 2003).

5.4.2 Human Toxicology

5.4.2.1 Acute Toxicity

Oral Studies

A few cases of accidental selenium poisonings in humans have been reported. A 3-year old boy died 1.5 hours after ingestion of an unknown quantity of selenious acid contained in a gun-blueing preparation. The boy displayed such clinical signs as excessive salivation, garlic odor of the breath, and shallow breathing (Carter 1966). A 15-year old female survived ingestion of a solution of sodium selenate at an estimated dose of 22.3 mg/kg bw/day, probably because she was forced to vomit soon after exposure (Civil and McDonald 1978). Clinical signs included garlic odor of the breath, diarrhea, and abnormal serum bilirubin and alkaline phosphatase levels. Acute oral exposure of humans to selenium compounds has also been reported to result in such effects as pulmonary edema, lung lesions, tachycardia, nausea, vomiting, diarrhea, abdominal pain, aches and pains, irritability, chills, tremors (Carter 1966; Koppel *et al.* 1986; Civil and McDonald 1978; Sioris *et al.* 1980).

Inhalation Studies

No studies were identified in the literature regarding human mortality following acute inhalation exposure to selenium compounds. Most reported cases of acute human inhalation exposure have been in occupational settings. Sudden acute inhalation of large amounts of selenium dioxide powder can produce pulmonary edema due to the irritant effect of the selenious acid that is formed when selenium dioxide comes into contact with moisture (Glover 1970). Bronchospasms, slight asphyxia, and bronchitis have been reported in workers briefly exposed to high concentrations of selenium dioxide (Wilson 1962; Kinnigkeit 1962). Acute inhalation exposure to elemental selenium dust was reported to cause irritation of mucous membranes in the nose and throat, coughing, nosebleeds, loss of smell, and in highly exposed workers, dyspnea, bronchospasms, bronchitis and chemical pneumonia (Clinton 1947; Hamilton and Hardy 1949). Other effects associated with the acute inhalation exposure of selenium compounds include quickened pulse, low blood pressure, indigestion, nausea, headaches, dizziness, and malaise (Wilson 1962; Glover 1970; Clinton 1947).

Dermal Studies

No studies were identified in the literature regarding human mortality following acute dermal exposure to selenium compounds. The most commonly reported effects of acute dermal exposure in humans have included rashes, burns, inflammation and contact dermatitis (Middleton 1947; Pringle 1942). However, some of these effects may have been due to caustic effects of selenious acid (ATSDR 2003).

5.4.2.2 Subchronic and Chronic Toxicity

In a follow-up to a study by Yang *et al.* (1983), Yang *et al.* (1989b) studied a population of 359 individuals from an area of China with unusually high environmental concentrations of selenium. This population was exposed to selenium primarily through soil, food and drinking water. Selenium concentrations in soil and 30 typical food types showed a positive correlation to blood and tissue levels in the exposed individuals. Average daily selenium intakes (based on lifetime exposure) from low, medium, and high selenium areas were 70, 195, and 1438 µg/day for adult males, and 62, 198, and 1238 µg/day for adult females, respectively. Yang *et al.* (1989b) noted that children aged 12 and under, never showed symptoms of selenosis (Yang *et al.* 1989b). "Long-persisting distinct clinical signs" of selenosis were observed in only 5 individuals from a potentially sensitive sub-population, and included garlic odor breath, thickened and brittle nails, hair and nail loss, reduced hemoglobin, mottled teeth, skin lesions, and central nervous system abnormalities (e.g., limb pain, peripheral anesthesia). Blood selenium concentrations in these individuals ranged from 1054 to 1854 µg/L, with a mean of 1350 µg/L. Based on the blood selenium concentrations that were shown to reflect clinical signs of selenosis, the authors concluded that a whole blood selenium concentration of 1350 µg/L, which corresponds to a daily intake of 1261 µg/day, is indicative of the lowest selenium intake causing overt signs of selenium toxicity. The authors also concluded that a whole blood selenium concentration of 1000 µg/L, corresponding to a daily intake of 853 µg/day, produced no clinical signs of selenosis. Therefore, the reported NOAEL and LOAEL from this study were 15 and 23 µg/kg bw/day, respectively, assuming an average adult body weight of 55 kg.

A more recent epidemiological study was conducted by Longnecker *et al.* (1991). In this study, 142 volunteers from known seleniferous regions of Wyoming and South Dakota were subjected to questionnaires, physical examinations, as well as blood, hair and urine analysis. The average daily selenium intake was estimated at 239 mg/day, approximately 2 to 3-fold above the U.S. national average. High correlations were observed between selenium concentrations in blood, urine, toenails and diet. Blood selenium concentrations were highly correlated to daily intake, similar to what Yang *et al.* (1989b) reported. No signs of selenium toxicity were observed in this population, including individuals for whom daily selenium intake was as high as 724 µg/day. The results of this study corroborate those of Yang *et al.* (1989b), which reported a NOAEL of 853 µg/day.

In Venezuela, the effects of high selenium intakes were studied in 65 women living in 3 known seleniferous areas in that country (Brätter and Negretti de Brätter 1996). Selenium intake was estimated by a regression equation from the selenium content of breast milk (Brätter *et al.* 1991). Serum levels of TSH (thyroid stimulating hormone) and T_3 and T_4 were also determined in the women. Although estimated daily selenium intakes ranged from 170 to 980 µg, none of the women displayed symptoms of selenosis. Free T_3 levels were significantly inversely correlated with serum selenium levels but levels of T_3, T_4 and THS were all within normal ranges and consequently, the biological significance in these observations at sub-clinical levels could not be interpreted.

Selenomethionine was orally administered to both cretin and normal schoolchildren for a period of two months (Contempre *et al.* 1991a, b). Both groups of children showed decreased levels of

T4 thyroid hormone. The authors concluded that selenomethionine increases sensitivity to iodine deficiency.

A woman who had ingested highly potent selenium supplemental tablets containing 31 mg total selenium as sodium selenite and elemental selenium per tablet for a period of 77 days, experienced hair loss and deformity and loss of fingernails (Jensen *et al.* 1984).

A family exposed to selenium in well water at a concentration of 9 mg/L (0.26 mg/kg bw/day) for three months displayed such symptoms as listlessness, lack of mental alertness, and other signs of selenosis (i.e., selenium toxicity) (Rosenfeld and Beath 1964). All symptoms disappeared after the family stopped obtaining their drinking water from this well. As the authors did not estimate the family's dietary intake of selenium, it is not possible to identify the daily dose that was associated with the observed symptoms.

5.4.2.3 Reproductive and Developmental Toxicity Studies

No studies were identified in the literature regarding reproductive effects in humans following oral, inhalation or dermal exposure to selenium compounds. Roy *et al.* (1990) found no correlation between seminal plasma selenium concentrations and sperm counts and mobility in sperm samples from 211 men. Robertson (1970) and Zierler *et al.* (1988) found no association between prenatal exposure to selenium compounds and birth defects or developmental abnormalities. However, there were many confounding variables that were not adequately accounted for in these studies (ATSDR 2003).

5.4.2.4 Epidemiological Studies

Selenium compounds have been given an IARC classification of Group 3, not classifiable as to carcinogenicity to humans due to conflicting animal, mutagenicity, and genotoxicity studies (IARC 1987). In addition, the available human epidemiological studies are of limited value in that conflicting results were reported, specific selenium compounds were not assessed, study designs and statistical analyses were flawed, or selenium exposures were not correlated with cancer risk (U.S. EPA 1991; ATSDR 2003). However, the evidence for selenium sulfide is sufficient for a B2 classification; probable human carcinogen (U.S. EPA 1993).

Several investigators have studied the association between serum selenium and cancer risk through prospective, case-control, and nested case-control epidemiology studies. In general, patients with gastrointestinal cancer, pancreatic cancer, or Hodgins lymphoma had significantly lower blood selenium concentrations than healthy patients (U.S. EPA 1991).

Shamberger and Frost (1969) reported an inverse relationship between cancer mortality rates and selenium concentrations in the food crops of several Canadian provinces. Cancer mortality rates (per 10^{-5} risk level) were found to be greatest in the provinces with the lowest amounts of selenium in food crops. In a similar study conducted in California, Shamberger and Willis (1971) reported a correlation between decreased cancer death rates and an increase in the selenium content of forage crops. The authors also investigated the ratio of observed to expected cancer mortality rates by anatomic site for men in 17 paired cities which included both

seleniferous and non-seleniferous areas. Gastrointestinal cancers and bladder cancer showed a substantially lower rate ratio in the high-selenium cities versus the low-selenium cities. A number of other studies have also reported an inverse relationship between cancer incidence and elevated environmental selenium concentrations and are reviewed in U.S. EPA (1991) and ATSDR (2004). Some recent large-scale long-term intervention studies involving the potential anti-carcinogenic properties of selenium through dietary supplementation have already been described in Section 5.3.

In a case-control study of lung cancer patients, Menkes *et al.* (1986) found that the risk of lung cancer was not associated with serum selenium levels (i.e., 0.113 and 0.110 mg/L in cases and controls, respectively). Some prospective studies have reported an association between low serum selenium levels and an increased incidence of cancer (Salonen *et al.* 1984; 1985; Willett *et al.* 1983), while others have found no correlation between cancer incidence and low blood selenium concentrations (Coates *et al.* 1988; Virtamo *et al.* 1987). A recent review of breast cancer epidemiological studies (Garland *et al.* 1993) found reports of inverse correlations, positive correlations, and no correlations between tissue selenium concentrations and breast cancer incidence. A recent cohort study by Vinceti *et al.* (1995) reported that a strong inverse relationship exists between dietary intake of selenium and cancer mortality.

Furthermore, epidemiological data do not support a causal association between the inhalation of elemental selenium dusts or selenium compounds and the induction of cancer in humans (Gerhardsson *et al.* 1986; Wester *et al.* 1981). Postmortem tissue samples in these studies showed lower concentrations of selenium compounds in lung and kidney tissues than those from controls or workers who died from non-cancer causes. However, as chronically ill and/or older individuals tend to have lower organ and tissue selenium concentrations than younger, healthier individuals, it is difficult to draw firm conclusions (Martin *et al.* 1991).

5.5 Interactions of Selenium with Other Substances

Selenium interacts with a wide variety of essential and non-essential elements, vitamins, xenobiotics and sulfur-containing amino acids. These interactions are complex and poorly understood for the most part, with the relevance to human health unknown. In general, these interactions tend to result in the reduced toxicity of selenium and/or the interacting substance. Table 15 presents some of the major interactions of selenium with other substances. This topic has been reviewed in detail by Combs and Combs (1987), Hansen (1988), and Marier and Jaworski (1983).

5.6 Overall Human Toxicological Evaluation

The U.S. EPA (1991) critically reviewed numerous studies investigating selenium exposure and its effects on experimental animals and humans. In determining the oral RfD (Reference Dose) for selenium, the U.S. EPA selected the epidemiology study by Yang *et al.* (1989b) as the principal and supporting study. The study NOAEL of 15 µg/kg bw/day was used to calculate an oral RfD of 5 µg/kg bw/day; a 3-fold safety factor was applied to account for sensitive individuals (U.S. EPA 1991). A 10-fold safety factor was not deemed necessary because of the high level of confidence in the Yang *et al.* (1989b) and additional supporting studies (U.S. EPA

1991). The results of Longnecker *et al.* (1991) strongly corroborate the NOAEL identified by Yang *et al.* (1989b). In addition, numerous other epidemiological studies and animal studies also support the findings of Yang *et al.* (1989b) (U.S EPA 1991).

Health Canada (1992) did not derive a TDI for selenium as a basis for setting the Guideline for Canadian Drinking Water Quality for selenium; a MAC of 10 µg/L, a level at which drinking water would represent between 10 and 25 percent of total selenium intake, was recommended. The World Health Organization (2003) used the same approach as Health Canada to develop the WHO Guideline for Drinking-Water Quality for selenium and did not propose a TDI.

More recently, 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 or TDIs (Tolerable Daily Intakes), 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 presence of hair and nail brittleness and loss, the most common and consistent clinical sign of chronic selenosis in people exposed to high selenium levels in their diet, was chosen as a critical end-point to support the derivation of ULs (IOM 2000). Chinese scientists have correlated human selenium blood concentrations with high dietary intakes of the trace element; chronic selenosis was diagnosed in individuals with a blood selenium concentration > 1000 µg/L, corresponding to a daily dietary selenium intake > 850 µg (Yang and Zhou 1994). For the identification of a NOAEL, the IOM (2000) selected the Yang and Zhou (1994) results of their 1992 re-examination of the same 5 individuals that previously showed signs of selenosis in Yang *et al.* (1989b). The average blood selenium levels of affected individuals had fallen from 1346 µg/L measured in 1986 (Yang *et al.* 1989b) to 968 µg/L in 1992 (Yang and Zhou 1994) corresponding to dietary intakes of 1261 and 819 µg Se/day, respectively (ATSDR 2003). In the latter study, the lower limit of the 95% confidence interval was 600 µg/day. The lower exposure levels were attributed to improvement in living conditions and by avoiding consumption of locally-grown high-selenium foods. Hence, Yang and Zhou (1994) proposed a new LOAEL of 913 µg/day, a marginally toxic daily intake of selenium, and a new NOAEL of 819 µg/day, a

level associated with recovery (ATSDR 2003). In agreement with those benchmarks, the IOM (2000) selected a NOAEL of 800 µg/day, a value 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 400 µg/day was determined for adults, 19 years and older (IOM 2000). For pregnant and lactating females, the same UL was chosen because of the absence of reports of teratogenesis and selenosis in infants born to mothers with high but not toxic selenium intakes. The NOAEL for derivation of the UL for infants (0-6 months) was conservatively based on a breast milk concentration of 60 µg Se/L known to be not toxic to infants, according to the review by Shearer and Hadjimarkos (1975). Adjusting for the daily breast milk consumption of infants during their first 6 months of life, the NOAEL was determined to be 47 µg Se/day (IOM 2000). An uncertainty factor (UF) of one was selected because no toxic effects in the infant or the mother were associated with a maternal selenium intake required to produce breast milk at a concentration of 60 µg Se/L (IOM 2000). The UL of 7 µg Se/kg bw/day was used to calculate ULs by extrapolation on a body weight basis for older infants (60 µg/day), children aged 1 to 3 years (90 µg/day), 4 to 8 years (150 µg/day), and 9 to 13 years (280 µg/day), and for adolescents, the same value as for adults (400 µg/day) (IOM 2000).

While some animal studies have shown reproductive and developmental effects from oral selenium exposure (e.g., Schroeder and Mitchener 1971b; Rosenfeld and Beath 1954), there is no evidence of reproductive effects, teratogenesis, or developmental abnormalities in humans.

The mutagenicity and genotoxicity database on selenium compounds is inconclusive, with many studies producing conflicting results for a number of selenium compounds.

The animal carcinogenicity database for selenium compounds is weak, with a number of conflicting results that are difficult to interpret because of apparent anticarcinogenic activity, high toxicity of some selenium compounds, and flawed study designs. Furthermore, comparison of the available studies is made difficult because several different selenium compounds with varying degrees of bioavailability were used in the cancer bioassays (U.S. EPA 1991). Generally, animal carcinogenicity studies with selenates, selenites, and organic selenium compounds have shown negative results.

The available human epidemiological cancer studies are of limited value in that conflicting results were reported, specific selenium compounds were not assessed, study designs and statistical analyses were flawed, or selenium exposures were not correlated with cancer risk (U.S. EPA 1991; ATSDR 2003). Nonetheless, the majority of studies have shown either an inverse relationship or no association between environmental selenium concentrations and cancer incidence and/or cancer mortality rates. Based on the results of the available epidemiology, animal, mutagenicity, and genotoxicity data, selenium compounds have been given an IARC classification of 3; not classifiable as to carcinogenicity in humans (IARC 1987). Only one selenium compound (i.e., selenium sulfide) has been shown to be carcinogenic in animal studies; thus it has been given an IARC classification of B2 - a probable human carcinogen (U.S. EPA 1993). However, this compound is not present in soils, foods or other environmental media to any significant extent; thus human environmental exposure to selenium sulfide would be negligible (ATSDR 2003).

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) as the reference exposure levels for risk assessment. Since selenium is an essential trace element in human health and selenium compounds do not appear to be carcinogenic, the ULs from IOM (2000) for all life stage groups are proposed for use in the derivation of the human health soil quality guidelines for selenium (see Table 12). However, the IOM (2000) ULs for all life stage groups do not correspond exactly to all the age classes of the Canadian general population (see Table 13) adopted by CCME (2006). Hence, some UL values were recalculated as duration-weighted average of applicable IOM age categories. The ULs for adults, teenagers, school aged children, toddlers and infants are 400, 370 (recalculated), 206 (recalculated), 103 (recalculated) and 45 µg/day, respectively. On a body weight basis, the ULs for adults, teenagers, school aged children, toddlers and infants are calculated to be 5.7, 6.2, 6.3, 6.2, and 5.5 µg/kg bw/day, respectively.

6. DERIVATION OF ENVIRONMEN TAL AND HUMAN HEALTH SOIL QUALITY GUIDELINES

6.1 Environmental Soil Quality Guidelines

Canadian Soil Quality guidelines are derived for the protection of receptors under four different land uses: agricultural, residential/parkland, commercial and industrial. The following derivation is based on the protocols described in CCME (2006).

All data selected for use in the following derivations have been screened for ecological relevance and are presented in preceding sections. Data with a soil pH below 4 were not selected for the purpose of soil quality guideline derivation. These data are considered outside the normal pH range of most soils in Canada. In addition, data were not selected if soil pH was not recorded; if no indication of soil texture was provided; if inappropriate statistical analysis was used; if the test was not conducted using soil or artificial soil; if the test soil was amended with sewage sludge or a mixture of toxicants; or if the test did not use controls.

Lowest-observed-effect concentration (LOEC) and effective concentration (EC) data used in the following derivations were considered to be statistically significant according to the study from which the data were taken.

6.1.1 Soil Quality Guidelines for Agricultural and Residential/Parkland Land Uses

6.1.1.1 Soil Quality Guideline for Soil Contact

The derivation of the soil quality guideline for soil contact (SQG_{SC}) is based on toxicological data for vascular plants and soil invertebrates. The toxicological data for plants and invertebrates are presented in Sections 4.1.3 and 4.3.3. The preferred method for determining the SQG_{SC} is to use a weight-of-evidence method with EC25 and LC25 data, or when these types of data are not available, with LOECs, NOECs, EC50s and LC50s (CCME 2006). For selenium,

there were insufficient data for use in the weight-of-evidence derivation, but there were sufficient data for use in the lowest observed effect concentration (LOEC) method. In the LOEC method, the threshold effects concentration is considered to be below the lowest LOEC of a dataset consisting of a minimum of 3 data points (1 data point for each group of receptors). The TEC is calculated according to the following formula:

$$TEC = \text{lowest LOEC} / UF$$

where

TEC = threshold effects level for plants and soil invertebrates (µg/g soil)
LOEC = lowest observed effect concentration (µg/g soil)
UF = uncertainty factor (unitless)

The lowest LOEC reported in the toxicological literature was 1 µg/g (Carlson *et al.* 1991). The endpoint for this LOEC was reduced shoot growth (approximately 60%) in sorgrass (*Sorghum vulgare*) over a 42-day exposure period. Singh and Singh (1979) also reported a LOEC of 1 µg/g for reduced dry matter yield in cowpea (*Vigna sinensis*) over a 50-day exposure period. No uncertainty factor was applied as the critical study was chronic, more than three studies were consulted and three taxonomic groups were represented (CCME 2006). Therefore, the TEC was calculated to be 1 µg/g soil.

Nutrient and Energy Cycling Check

There were no specific studies of nitrification or nitrogen fixation by soil microorganisms for selenium, however there were studies of respiration (oxygen consumption) and reduction in enzyme activities in selenium-treated soils. As discussed in CCME (2006), sulphatase and phosphatase enzyme activities vary in soils with phosphate and sulphate concentrations in the soil, and may be stabilized in soil outside the cell. The use of respiration data in the assessment of nutrient and energy cycling is also limited; there is functional redundancy in respiratory processes in soils, and that, as a result, significant impacts may have occurred before respiration is affected (CCME 2006).

Therefore it was not possible to complete a nutrient and energy cycling check for selenium.

Conclusions

Based on the foregoing, the SQG_{SC}, protective of soil contact by plants, invertebrates and microorganisms would be 1 µg/g for agricultural and residential/parkland land uses.

6.1.1.2 Soil Quality Guidelines for Soil and Food Ingestion

The following section provides the derivation of soil and food ingestion guidelines (SQG_I) for agricultural land uses, for domestic animals and wildlife that are primary consumers.

There were sufficient toxicological data to derive LOAELs to fulfill the minimal requirements for derivation of the SQG_I (two mammalian oral studies, one avian oral study). Avian toxicity

studies were available for mallard ducks, screech owls, American kestrels, black-crowned night-herons, and chickens (Table 10). Mammalian toxicity studies were available for sheep, pigs, goats, cows, ponies, pronghorn antelope, long-tailed macaques, rats, and mice (Tables 10 and 11). It was determined from the published literature that sufficient data were available to determine soil to plant bioconcentration factors (BCF_{SP}).

Development of Daily Threshold Effect Dose (DTED)

The lowest effects doses available were 0.08 µg/g bw/d for sheep (Puls 1994), 0.15 µg/g bw/d for long-tailed macaques (Tarantal *et al*. 1991), 0.25 µg/g bw/d for cows (Kaur et al. 2003), and 0.3 µg/g bw/d for chickens (Moxon 1937; Ort and Latshaw 1978). The study on sheep was rejected due to insufficient information about many aspects of the study (e.g., type of adverse effects observed, form of selenium used, etc.). The macaque study was rejected because it was based on exposure through nasogastric intubation, rather than ingestion. The cow study was rejected because only one test concentration was used, and many details of the study were unclear. Therefore, the LOAEL of 0.3 µg/g bw/d for reduced hatchability and chick survival rate in chickens (Moxon 1937; Ort and Latshaw 1978) was selected as the critical study for deriving the SQG_I. No uncertainty factor was applied to the DTED because data were available for a variety of taxonomic groups.

Using the formula provided by CCME (2006):

$$DTED = \text{lowest ED} / UF$$

where: DTED = daily threshold effect dose (µg/g bw/d);
ED = lowest effects dose (0.3 µg/g bw/d) (Ort and Latshaw 1978);
UF = uncertainty factor (none).

The DTED for chickens is thus 0.3 µg/g bw/d.

Receptor Parameters

The literature provided the following data for the receptor of concern in the derivation of soil quality guidelines:

Receptor	Body Weight (g)	Diet	Dry Matter Ingestion Rate DMIR (g/d)	Proportion of Soil Ingestion (as % of DMIR)	Reference
Chicken	1600	100% grain and vegetation	110	7.7	US EPA 1988; McMurter 1993

Bioavailability

For the purpose of environmental guideline derivation, the bioavailability term is meant to represent the difference between the bioavailability of a chemical present in food used in the reference experiment and the bioavailability of that chemical when present in food from natural

sources. This information was not available and thus a bioavailability factor of one will be assumed.

Bioconcentration Factors

The concentration ratio for selenium in soils to plants ranges from 0.02 to 77 (US DOE 1998). A median bioconcentration factor of 0.7 (US DOE 1998) was adopted for the derivation of the soil quality guideline for soil and food ingestion (SQG_I).

Determination of the Rate of Soil Ingestion

Animals will consume soils, whether as a result of selecting food items in close association with soil (e.g., eating worms from the soil), soil adhering to forage, through preening behaviours, etc. The data describing soil ingestion can in turn be used to derive the rate of soil ingestion, according to the following calculation:

$$SIR = DMIR \times PSI$$

where

SIR = soil ingestion rate (g/d on a dry weight basis)
DMIR = dry matter ingestion rate, calculated as the geometric mean of available DMIR, (110 g/d on a dry weight basis) (US EPA 1988)
PSI = percentage soil in the diet, on a dry weight basis (7.7%) (McMurter 1993)

Therefore, the SIR for the receptor of concern is as follows:

Receptor	**SIR (g/d) as dry weight)**
Chicken	8.5

Determination of the Rate of Food Ingestion

The rate of food ingestion is calculated as the proportion of the diet not consisting of soil, that is, the difference between the DMIR and the SIR, as follows:

$$FIR = DMIR - SIR$$

where

FIR = food ingestion rate (g/d dry weight)
DMIR = dry matter ingestion rate, calculated as the geometric mean of available DMIR, (110 g/d on a dry weight basis) (US EPA 1988)
SIR = soil ingestion rate (8.5 g/d dry weight) (calculated above)

Therefore, the FIR for the receptor of concern is as follows:

Receptor	**FIR (g/d) as dry weight)**
Chicken	101.5

Calculation of the Soil Quality Guideline for Ingestion

As required by the CCME (2006) protocol, 25% of the DTED is apportioned for exposures via drinking water and dermal absorption. Therefore the total exposure via ingestion of food and soil should not exceed 75% of the DTED.

In the calculation of the soil quality guideline for ingestion, the exposure via soil ingestion is calculated via the combination of the SIR with the relevant bioconcentration factors along with the body weight, as follows:

$$\text{Exposure via Soil Ingestion} = SIR \times SQG_I \times BF / BW$$

where

SIR = soil ingestion rate (g/d as dry weight);
SQG_I = soil quality guideline for soil and food ingestion (µg/g);
BF = bioavailability factor (unitless);
BW = body weight (g)

In the calculation of the soil quality guideline for ingestion, the exposure via food ingestion is calculated via the combination of the FIR with the relevant bioconcentration factors and the body weight, as follows:

$$\text{Exposure via Food Ingestion} = FIR \times SQG_I \times BCF_{food} \times BF / BW$$

where

FIR = food ingestion rate (g/d as dry weight);
SQG_I = soil quality guideline for soil and food ingestion (µg/g);
BCF_{food} = soil to food (plant or invertebrate or small mammal) bioconcentration factor (unitless);
BF = bioavailability factor (unitless);
BW = body weight (g)

These two equations can be combined and rearranged to solve for SQG_I, based on the assumption that the sum of the exposure via soil and food ingestion equals the exposure limit (0.75 x DTED x BF):

$$SQG_I = (0.75 \times DTED \times BF \times BW) / [(SIR \times BF) + (FIR \times BCF_{food} \times BF)]$$

where

SQG_I = soil quality guideline for soil and food ingestion (µg/g);
DTED = daily threshold effects dose (0.3 µg/g bw/d) (calculated above);
FIR = food ingestion rate (101.5 g/d as dry weight) (calculated above);
SIR = soil ingestion rate (8.5 g/d as dry weight) (calculated above);
BCF_{food} = soil to plant bioconcentration factor (0.7 unitless) (US DOE 1998);
BF = bioavailability factor (unitless); For the purpose of environmental guideline derivation, the bioavailability term is meant to represent the difference between the bioavailability of a chemical present in food used in the reference experiment and the bioavailability of that chemical when present in food from natural sources. This information was not

available and thus a bioavailability factor of one will be assumed.
BW = body weight (1600g) (US EPA 1988).

Therefore, the calculated SQG_I for the receptor of concern is as follows:

Receptor	**SQG_I (µg/g)**
Chicken	4.5

Therefore, the SQG_I is 4.5 µg/g for agricultural land uses. It should be noted that this concentration of selenium in soil may not be protective against adverse effects if plants that hyperaccumulate selenium are growing in the soil and are consumed by livestock and wildlife.

6.1.1.3 Summary and Selection of the SQG_E for Agricultural and Residential/Parkland Land Use

As stated earlier, the SQG_{SC} for soil contact (protective of plants, soil microorganisms and soil invertebrates) for selenium is 1 µg/g for agricultural and residential/parkland land uses. The SQG_I for the soil and food ingestion check (protective of domestic livestock, resident and transitory wildlife) for agricultural lands was 4.5 µg/g which is greater than the SQG_{SC}. Therefore, the SQG_E for agricultural and residential/parkland land use is 1 µg/g.

6.1.2 Soil Quality Guidelines for Commercial and Industrial Land Uses

6.1.2.1 Soil Quality Guideline for Soil Contact

The derivation of the soil quality guideline for soil contact (SQG_{SC}) is based on toxicological data for vascular plants and soil invertebrates. The toxicological data for plants and invertebrates is presented in Sections 4.1.3 and 4.3.3. There were insufficient data for use in the weight-of-evidence derivation, but there were sufficient data for use in the lowest observed effect concentration (LOEC) method, in which the threshold effects concentration is considered to be below the lowest LOEC of a dataset consisting of a minimum of 3 data points (1 data point for each group of receptors). The ECL is calculated using the geometric mean according to the following formula:

$$ECL = (LOEC_1 \times LOEC_2 \times ... LOEC_n)^{1/n}$$

where ECL = effects concentration low for plants and soil invertebrates (µg/g soil)
LOEC = lowest observed effect concentration (µg/g soil)

The LOECs reported in the toxicological literature for plants were: 1 µg/g (Carlson *et al.* 1991), 1 µg/g (Singh and Singh 1979), 1.5 µg/g (Wan *et al*. 1988), 2 µg/g (Soltanpour and Workman 1980), and 2.5 µg/g (Singh and Singh 1978) while that for invertebrates was 77 µg/g (Fischer and Koszorus 1992).

Therefore, the ECL was calculated to be 2.9 µg/g soil.

Nutrient and Energy Cycling Check

There were no specific studies of nitrification or nitrogen fixation by soil microorganisms for selenium, however there were studies of respiration (oxygen consumption) and reduction in enzyme activities in selenium-treated soils. As discussed in CCME (2006), sulphatase and phosphatase enzyme activities vary in soils with phosphate and sulphate concentrations in the soil, and may be stabilized in soil outside the cell. The use of respiration data in the assessment of nutrient and energy cycling is also limited, in that there is functional redundancy in respiratory processes in soils, and that, as a result, significant impacts may have occurred before respiration is affected (CCME 2006).

Therefore it was not possible to complete a nutrient and energy cycling check for selenium.

Conclusions

Based on the foregoing, the SQG_{SC} for commercial and industrial land uses, protective of soil contact by plants, invertebrates and microorganisms, would be 2.9 µg/g.

6.1.2.2 Soil Quality Guideline for Soil and Food Ingestion

As discussed in the protocol (CCME 2006), the use of commercial and industrial sites by wildlife is considered to be greatly reduced, in comparison to that observed on agricultural or residential/parkland scenarios. In addition, the normal land use activities on commercial and industrial sites do not depend on the maintenance of ecological functioning to the same degree. Therefore, as discussed in CCME (2006), soil contact is considered to represent the most significant pathway of exposure for ecological receptors under commercial/industrial land use. Because data do not permit the estimation of toxicity to wildlife via direct contact, it is assumed that the guidelines protective of soil invertebrates and plants would be protective of wildlife, based on differences in mobility and degree of direct soil contact.

6.1.2.3 Off-site Migration Guidelines for Commercial and for Industrial Land Uses

When deriving soil quality guidelines for commercial and industrial sites, exposure scenarios consider only on-site exposure. Transfers of contaminated soil from one property to another is possible by environmental occurrences such as wind and water erosion (CCME 2006).

The Universal Soil Loss Equation and the Wind Erosion Equation are utilized to estimate the transfer of soil from one property to another. The following equation allows us to calculate the concentration in eroded soil from the site that will raise the contaminant concentration in the receiving soil to equal the agricultural guideline within a specific time frame. This concentration is referred to as the environmental soil quality guideline for off-site migration (SQG_{OM-E}). If the guidelines for commercial or industrial sites are found to be above C_i, then potentially the adjacent property could become unacceptably contaminated from off-site deposition (CCME 2006). The following equation has been derived to allow the calculation of SQG_{OM-E}.

$$SQG_{OM-E} = 14.3 \times SQG_A - 13.3 \times BSC$$

where,

SQG_{OM-E} = environmental soil quality guideline for off-site migration (i.e., the concentration of contaminant in eroded soil (µg/g)
SQG_A = soil quality guideline for agricultural land uses (1 µg/g)
BSC = background concentration of contaminant in the receiving soil (0.7 µg/g)

Therefore, the SQG_{OM-E} for commercial and for industrial land uses was determined to be 5.0 µg/g.

6.1.2.4 Summary and Selection of the SQG_E for Commercial and Industrial Land Use

As stated earlier, the SQG_{SC} (protective of plants, soil microorganisms and soil invertebrates) for selenium is.2.9 µg/g. The results of the off-site migration check for industrial land uses were greater than this value. Therefore, the SQG_E for commercial and industrial land uses is 2.9 µg/g .

6.1.3 Data Gaps in the Derivation of Environmental Soil Quality Guidelines

There were several areas in which data were lacking, including the following:

- Studies of the effects of selenium on nitrogen fixation, nitrification, nitrogen mineralization, decomposition and/or respiration studies were lacking in the published literature.

- In general, there were few data for the derivation of soil contact guidelines, particularly for terrestrial plants and soil-dwelling invertebrates, as well as for the derivation of DTED for domestic ungulates.

- There were no data on the bioavailability of selenium in avian species.

6.2 Human Health Soil Quality Guidelines

6.2.1 Estimated Daily Intakes

A background selenium soil concentration of 0.7 µg/g is assumed, both for the purpose of deriving estimated daily (background) intake from soil, and for the derivation of the human health-based soil quality guidelines. This concentration is reflective of mean selenium concentrations measured in background soils collected in Alberta (Penny 2004; R.G. Garrett, Natural Resources Canada, 2005, pers.com.), Saskatchewan (R.G. Garrett, Natural Resources Canada, 2005, pers.com.), Manitoba (R.G. Garrett, Natural Resources Canada, 2005, pers.com.; Haluschak *et al.* 1998; and Smith *et al*. 2004), and in Ontario (OMEE 1994; R.G. Garrett, Natural Resources Canada, 2005, pers.com.; Gizym 1994; Rasmussen *et al*. 2001). It appears that the earlier data sets, such as those from McKeague *et al.* (1979), Lévesque (1974a, b) and Gupta and Winter (1975), report levels that are lower than those determined at a later date for the same areas; losses of volatile selenium or other methodological problems may have occurred in these earlier determinations. As noted earlier, soil concentrations of selenium vary according to local geology. Although no single soil concentration can adequately represent the variance in background soil concentrations across Canada (Painter *et al.* 1994), it is also essential to define a reasonable value for the purpose of generic, national guidelines development. This background concentration is likely to be conservative for some regions in Canada but it is based on the most complete and reliable recent data sets available at the present time. Refer to Table 2 for more details on background selenium concentrations in Canadian soils.

The typical selenium level used in the exposure estimates for air was 1.0 ng/m^3. This air concentration is considered representative of background selenium concentrations in Canada. This value is equal to the overall mean concentration of selenium in PM_{10} samples (n=2170) collected across Canada in 2002 and 2003 from 31 National Air Pollution Surveillance stations (T. Dann, Environment Canada, pers. com.). It is assumed that the majority of Canadians live in urban environments (Statistics Canada 2005) and that complete retention and absorption of inhaled contaminants occurs. Since no Canadian data on selenium concentrations in indoor air were identified, ambient air concentrations were used as best estimates of concentrations in indoor air.

For drinking water consumption, an urban exposure scenario is the most common situation expected to arise since 80% of Canadians live in cities (Statistics Canada 2005) and 84% of these urban dwellers receive treated water supplies, mostly from surface water sources (Environment Canada 2005b). A concentration of 0.5 µg/L was considered to be the typical selenium level in Canadian drinking water supplies, based on extensive drinking water quality surveys performed across Canada and in Manitoba, Slightly higher selenium concentrations were reported for drinking water from groundwater sources in the Prairies and from one municipality in Ontario.

The selenium exposure from diet was based on selenium determinations of 135 foods purchased in Toronto in 1992 and prepared for consumption as part of Health Canada's Total Diet Study.

6.2.2 Exposure Limit for Human Receptors

The IOM (2000) ULs for selenium adopted by Health Canada (2003) were considered to be

appropriate for use in the development of a Canadian SQG_{HH}. The UL values from IOM (2000) were adapted to the age classes of the Canadian general population and calculated on a body basis (adults, 5.7; teenagers, 6.2; school aged children, 6.3; toddlers, 6.2; and infants, 5.5 µg/kg bw/day). The RfD dose of 5 µg/kg bw/day derived by the U.S. EPA (1991) was based on the first set of epidemiological data published by Yang *et al.* (1989b) whereas the UL was based on a follow-up re-examination of the same Chinese population (Yang and Zhou 1994).

6.2.3 Soil Inhalation Rates

Soil inhalation rates were determined by multiplying air inhalation rates for a particular age group by the average soil particle concentration in air over a particular land use. Average air inhalation rates for toddlers (6 months to five years) and adults are 9.3 and 15.8 m^3/d, respectively (Richardson 1997; Health Canada 2004; CCME 2006). Health Canada (2004) reports dust concentrations of 0.76 µg/m^3 can be conservatively assumed (except in cases where regular vehicle traffic over unpaved soils or construction activities or other dust generating activities are expected). Consequently, the following soil inhalation rates were used:

Agricultural land use (toddler receptor)	=	7.1 µg/d or 7.1 x 10^{-9} kg/d
Residential/Parkland land use (toddler receptor)	=	7.1 µg/d or 7.1 x 10^{-9} kg/d
Commercial land use (toddler receptor)	=	7.1 µg/d or 7.1 x 10^{-9} kg/d
Industrial land use (adult receptor)	=	12.0 µg/d or 1.2 x 10^{-8} kg/d

6.2.4 Relative Absorption Factors

Relative absorption factors may be applied when the critical toxicological study has used a different medium than that under investigation, in order to account for the difference in absorption of the contaminant by the body in the two different media. In this case, the critical study was an epidemiological study where the dietary intake (i.e., administered oral dose) was back-calculated from a selenium blood level known to cause clinical selenosis (NOAEL) to an estimated dietary intake, so relative absorption factors are not needed to account for the difference in absorption of selenium in soil compared to selenium in water.

It is noted that most of the available information indicates that selenium is unlikely to be absorbed through human skin. ORNL (2005) recommends a value of 0.1% as an absolute absorption factor but it is a generic default assumption for metals in general and is not specific for selenium. For the purposes of this assessment, a relative absorption factor of 0.001 (i.e., 0.1%) was used.

6.2.5 Agricultural and Residential/Parkland Uses (Toddler Receptor)

For determining an agricultural and a residential/parkland soil guideline it was assumed that the most appropriate receptor to use would be the toddler, aged 6 months to 4 years, due to a large exposure per unit mass. In accordance with the CCME guideline derivation procedures (CCME 2006), a preliminary soil quality guideline was derived for three exposure pathways combined (ingestion, inhalation, and dermal):

$$SQG_{DH} = \frac{[(TDI - EDI) \times SAF \times BW]}{[(AF_G \times SIR) + (AF_S \times SR) + (AF_L \times IR_S) \times ET_2] \times ET_1} + BSC$$

where,

SQG_{DH} = direct human health-based soil quality guideline (µg/g)
TDI = tolerable daily intake (6.2 µg/kg bw/d) (adapted from Health Canada 2003; IOM 2000)
EDI = estimated daily intake for the toddler (4.2 µg/kg bw/d) (based on various data – see Table 14)
SAF = soil allocation factor of 20%, by default (CCME 2006)
BW = body weight for toddler (16.5 kg) (CCME 2006)
SIR = soil ingestion rate for toddler (0.08g/d) (CCME 2006)
SR = soil dermal contact rate for toddler (0.069g/d) [hands surface area of 0.043 m^2 (CCME 2006) × soil adherence factor of 0.001 $kg/m^2/d$ plus arms/legs surface area of 0.26 m^2 (CCME 2006) × soil adherence factor of 0.0001 $kg/m^2/d$ (CCME 2006)]
IR_S = soil inhalation rate for toddler (7.1 x 10^{-6} g/d) [i.e., inhalation rate for toddler = 9.3 m^3/d x suspended soil dust concentration of 7.6 x 10^{-10} kg/m^3 (Health Canada 2004)]
BSC = background soil concentration (0.7 µg/g) (average based on R.G. Garrett, Natural Resources Canada, 2005, pers.com.; Haluschak *et al.* 1998; Smith *et al*. 2004; OMEE 1994; Gizyn 1994; and Rasmussen *et al*. 2001)
AF_G = relative absorption factor for soil: food and water in the gut (100%, assumed by default)
AF_L = relative absorption factor for soil: water in lung tissue (100%, by default)
AF_S = relative absorption factor for soil: water on skin (0.1%, by default from ORNL 2005)
ET_1 = exposure term 1 (unitless) – days per week/7 x weeks per year/52 at the site (=1.0) [i.e., 7 days per week, 52 weeks per year assumed at the site (CCME 2006)]
ET_2 = exposure term 2 (unitless) – hours per day/24 at the site (=1.0) [i.e., 24 hours per day assumed at the site (CCME 2006)] for inhalation exposure. For soil ingestion and dermal contact, it is assumed that 1 event occurs per day regardless of time spent at the site (i.e., ET_2=1.0)

The direct human health-based soil quality guideline (SQG_{DH}) for selenium in agricultural and residential/parkland soil was calculated to be 83.1, rounded down to **80** µg/g. Therefore, for agricultural and residential/parkland land uses, the SQG_{DH} is **80** µg/g.

6.2.6 Commercial Land Use (Toddler Receptor)

Commercial land sites are generically defined as sites at which commercial activities predominate, such as a shopping mall. There are no manufacturing activities or residential sites present.

For threshold contaminants such as selenium, the toddler is assumed to be the most sensitive receptor. The commercial land use calculation is exactly the same as the Agricultural and Residential/Parkland calculations, the only differences being:

- exposure term 1 (ET_1) is 0.66 (based on 5 d/wk and 48 wk/y) due to the reduced amount of time the receptor spends on a commercial site.

- exposure term 2 (ET_2) is 0.42 (based on 10 h/d) due to the reduced amount of time the receptor spends on a commercial site for inhalation exposure. For soil ingestion and dermal contact, it is assumed that 1 event occurs per day regardless of time spent at the site (i.e., ET_2=1.0)

The direct human health-based soil quality guideline (SQG_{DH}) for selenium on commercial lands was calculated as 125.8 rounded down to **125** µg/g. Therefore, for commercial land uses, the SQG_{DH} is **125** µg/g.

6.2.7 Industrial Land Use (Adult Receptor)

In an industrial scenario, occupational exposure will be the primary route of exposure, hence the use of an adult receptor. Exposure for an adult at an industrial site is assumed to be 10 h/d, 5 d/wk and 48 wk/y. Examples of industrial lands could be manufacturing plants.

$$SQG_{DH} = \frac{[(TDI - EDI) \times SAF \times BW]}{[(AF_G \times SIR) + (AF_S \times SR) + (AF_L \times IR_S) \times ET_2] \times ET_1} + BSC$$

where,

SQG_{DH} = direct human health-based soil quality guideline (mg/kg)
TDI = tolerable daily intake (*5.7 µg/kg bw/d*) (adapted from Health Canada 2003; IOM 2000)
EDI = estimated daily intake for adult (*1.9 µg/kg bw/d*) (based on various data – see Table 14)
SAF = soil allocation factor of *20%*, by default (CCME 2006)
BW = body weight for adult (*70.7 kg*) (CCME 2006)
SIR = soil ingestion rate for adult (0.02*g/d*) (CCME 2006)
SR = soil dermal contact rate for adult (*0.11g/d*) [hands surface area of 0.089 m^2 (CCME 2006) × soil adherence factor of 0.001 $kg/m^2/d$ plus arms surface area of 0.25 m^2 (CCME 2006) × soil adherence factor of 0.0001 $kg/m^2/d$ (CCME 2006)]
IR_S = soil inhalation rate for adult (*1.2 x 10^{-5} g/d*) [i.e., inhalation rate of 15.8 m^3/d x suspended soil dust concentration of 7.6 x 10^{-10} kg/m^{-3} (Health Canada 2004)]
BSC = background soil concentration (*0.7 µg/kg*) (average based on Garrett 2005; Haluschak *et al.* 1998; Smith *et al*. 2004; OMEE 1994; Gizyn 1994; and Rasmussen *et al*. 2001)
AF_G = relative absorption factor for soil: diet and water in the gut (100%, assumed by default)
AF_L = relative absorption factor for soil: water in lung tissue (100%, by default)
AF_S = relative absorption factor for soil: water on skin (0.1%, generic default value from ORNL (2005))
ET_1 = exposure term 1 (unitless) – days per week/7 x weeks per year/52 at the site (0.66) [i.e., 5 days per week, 48 weeks per year assumed at the site (CCME 2006)]

ET_2 = exposure term 2 (unitless) – hours per day/24 at the site (0.42) [i.e., 10 hours per day assumed at the site (CCME 2006)] for inhalation. For soil ingestion and dermal contact, it is assumed that 1 event occurs per day regardless of time spent at the site (i.e., ET_2=1.0)

Using this equation, the direct human health-based soil quality guideline (SQG_{DH}) for selenium on industrial land was calculated as 4051 rounded down to **4050** µg/g. Therefore, for industrial land uses, the SQG_{DH} is **4050** µg/g.

6.2.7.1 Guideline for Protection of Groundwater

No guideline for protection of groundwater was derived for selenium due to restrictions on the mathematical model when applied to metals (CCME 2006).

6.2.7.2 Off-site Migration Guidelines for Commercial and and Industrial Land Uses

When deriving soil quality guidelines for commercial and industrial sites, exposure scenarios consider only on-site exposure. Transfers of contaminated soil, from one property to another are possible by environmental occurrences such as wind and water erosion (CCME 2006).

The Universal Soil Loss Equation and the Wind Erosion Equation are utilized to estimate the transfer of soil from one property to another. The following equation allows us to calculate the concentration in eroded soil from the site that will raise the contaminant concentration in the receiving soil to equal the agricultural guideline within a specific time frame. This concentration is referred to as the human health soil quality guideline for off-site migration ($SQG_{OM\text{-}HH}$). If the guidelines for commercial or industrial sites are found to be above $SQG_{OM\text{-}HH}$, then potentially the adjacent property could become unacceptably contaminated from off-site deposition (CCME 2006). The following equation has been derived to allow the calculation of $SQG_{OM\text{-}HH}$.

$$SQG_{OM-HH} = 14.3 \times SQG_A - 13.3 \times BSC$$

where,

$SQG_{OM\text{-}HH}$ = human health soil quality guideline for off-site migration (i.e., the concentration of contaminant in eroded soil) (µg/g)
SQG_A = soil quality guideline for agricultural land uses (80 µg/g)
BSC = background concentration of contaminant in the receiving soil (0.7 µg/g)

Therefore, the $SQG_{OM\text{-}HH}$ for commercial and for industrial land uses was determined to be 1135 µg/g, which is less than the SQG_{DH} of 4050 µg/g for the industrial scenario. Therefore, the industrial SQG_{HH} should be set to **1135** µg/g.

6.3 *Summary of Environmental and Human Health Soil Quality Guidelines*

The soil quality guidelines derived for environmental and human health, based on the above protocol, are summarized in Table 16. For specific locations with unusually high natural background concentrations that still exceed these guidelines, jurisdictions have the option to set site-specific guidelines that consider the unique geological characteristics of the particular locations.

6.4 Discussion of Uncertainties Associated with the SQG_{HH}

The SQG_{DH} provided in this section are felt to be protective of human health at most sites. Some of the issues most important to the analysis and development of the selenium SQG_{HH} are described below.

To determine an acceptable level of exposure to selenium for development of the SQG_{HH}, the scientific position of both Health Canada (2003) and IOM (2000) regarding the essentiality of selenium as a dietary reference intake (i.e., the calculated tolerable upper intake levels (ULs) of 6.2 and 5.7 µg/kg bw/day for toddlers and adults, respectively) were considered instead of more conservative toxic reference levels like a TDI, RfD or MRL. As indicated before, 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. So far, no TDI for selenium has been set by either Health Canada or the World Health Organization. A value of 5 µg/kg bw/day has been set as a chronic oral RfD by the U.S. EPA (1991) and as a chronic oral MRL by ATSDR (2003). Both reference values were based on the same NOAEL value of 15 µg/kg bw/day and and an uncertainty factor of 3 but the NOAEL did not come from the same data sets; U.S. EPA (1991) identified a NOAEL from Yang *et al.* (1989b) while ATSDR (2003) based their NOAEL on Yang and Zhou (1994).

The essentiality and toxicological database for selenium is relatively sound even though it is based on a single epidemiological study comprising relatively few individuals. The IOM (2000) ULs rely on the most recent data obtained when the same Chinese individuals were re-assessed in 1992 when no longer suffering from selenosis whereas the U.S. EPA (1991) only considered the first assessment of those individuals showing signs of the disease. Selenium levels measured in 1986 and 1992 in these Chinese individuals were employed to determine the LOAEL and NOAEL by the IOM (2000).

In order to assist in the interpretation of potential health risks, exposures that may result from the SQG_{DH} were compared to other benchmarks of exposure and potential health effects. Based on procedures described in this document, a toddler exposed to a selenium soil concentration of 80 µg/g at a residence (24 hours per day, 7 days per week, 52 weeks per year) would have an estimated exposure rate of about 0.4 µg/kg bw/day which is about 10% of the exposure that a toddler would receive from the typical background food supply (see Table 14.). In addition, exposures to soils with concentrations equal to the residential SQG_{DH} of 80 µg/g would result in exposures that are about 6% of the IOM (2000) and Health Canada (2003) tolerable upper intake

level and 8% of the U.S. EPA RfD.

As shown in the equation provided in this document, the soil ingestion route is the dominant pathway whereas uncertainties that may be associated with various aspects of dermal and inhalation exposures are unlikely to drive a risk assessment. In the case of dermal exposure, it is felt that dermal absorption of selenium would typically be a negligible exposure pathway (when compared to soil ingestion). In the case of inhalation of dusts, this pathway was evaluated using the IOM (2000) ULs. ATSDR (2003) did not develop MRLs (Minimal Risk Levels) for exposure to selenium by inhalation for lack of adequate data from animal and human studies. The California Office of Environmental Health Hazard Assessment has derived an inhalation REL (Reference Exposure Level) of 20 $\mu g/m^3$ for selenium and selenium compounds (except hydrogen selenide) extrapolated from the U.S. EPA oral RfD of 5 µg/kg bw/day (OEHHA 2001). Using this REL, the dust concentrations expected from sites at the SQG_{DH} would be much less than any value of concern (e.g., an industrial site with a $SQG_{OM\text{-}HH}$ of 1135 µg/g and a particulate concentration of 0.76 $\mu g/m^3$ would have a selenium concentration of 0.0013 $\mu g/m^3$, which is comparable to the mean selenium background level measured in ambient air across Canada, i.e., 0.001 $\mu g/m^3$). At contaminated sites where unusually high dust suspension is observed (e.g., construction activities and/or where there is excessive vehicular traffic on dirt roads), a lower value may need to be considered on a site-specific basis.

With respect to the soil ingestion route, the oral biovailability of selenium in soil was essentially assumed to equal the bioavailability of soluble forms of selenium in drinking water (selenite or selenate) and organic forms of selenium in the diet. As noted earlier, no definitive data were available and the assessment did not quantitatively account for the potential reduced bioavailability of selenium in soil.

Although localized areas within Canada may have different soil background concentrations, the SQG_{HH} developed for selenium should be protective of most situations. Drinking water, air and soil concentrations contributed relatively small amounts to the EDI calculations (i.e., $\leq$ 1% of the total EDI for all age classes) such that variations from the assumed average Canadian concentrations will typically have only a minor impact on the EDI and SQG_{HH}. In the case of soil, it is noted that the selenium background soil concentration (BSC) was assumed to be 0.7 µg/g for estimating exposure of the general Canadian population to selenium in soil. This was considered to be a reasonable value, although somewhat conservative, based on the available data.

Although the SQG_{HH} are felt to be protective at most sites, certain exposure pathways have not been evaluated in the development of the SQG_{HH}. More specifically, the SQG_{HH} have not evaluated garden produce consumption or drinking water consumption. At sites where appreciable amounts of garden produce are grown and consumed, a lower value may need to be considered, especially if locally grown produce include asparagus, mushrooms, garlic, some vegetables of the cabbage family, and some protein-rich legumes, all of which are known to bioaccumulate selenium from soil. In the case of protection of potable water, it is noted that this pathway, as it is the case for all inorganics, has not been evaluated in the development of the SQG_{HH} provided above. At sites where groundwater from nearby wells is used as a source of potable water, a lower value may need to be considered, especially if water is pumped from shallow wells.

As a result, the SQG_{HH} derived herein should be considered to be conservative even though uncertainties with some data exist. Nevertheless, as new toxicological and other data become available, the SQG_{HH} should be re-evaluated to ensure adequate protection of human health. With the above in mind, the SQG_{HH} are felt to be protective of human health at most sites.

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TABLES

Table 1. Physical and Chemical Properties of Selenium (CASRN: 7782-49-2)

Property	Value	Reference
Atomic number	34	Merck Index 1996
Molecular weight	78.96 g/mol	Merck Index 1996
Ground state electron configuration	$[Ar].3d^{10}.4s^2.4p^4$	WebElements 1998
Vapour pressure	1 mmHg at 356°C	ATSDR 2003
Solubility	insoluble in water and alcohol, slightly soluble in carbon disulfide; soluble in ether	Merck Index 1996
Melting point[1]	217°C	Merck Index 1996
Boiling point[1]	685°C	CS ChemFinder 1998
Heat of vaporization[1]	20.6 kcal/mol	Merck Index 1996
Specific gravity[1]	4.81	Merck Index 1996

[1] Values are for the most stable form of selenium (gray or metallic)

Table 2. Background Concentrations of Selenium (µg/g) in Canadian Surface Soil

Location	Number of Samples	Soil Concentrations Mean (±s.d.) (Range)	Analytical Technique	Reference
Canada – all regions	188	0.30 (0.02-3.7)	HNO_3-$HClO_4$	McKeague *et al*. 1979
Appalachian Region	45	0.23 (0.02-2.20)	Fluorometric analysis	
Canadian Shield	12	0.18 (0.06-0.71)		
St. Lawrence Lowlands	58	0.28 (0.02-3.70)		
Interior Plains	54	0.40 (0.05-2.20)		
Cordilleran Region	19	0.30 (0.07-0.78)		
Canada – all provinces and territoires except Manitoba	54 soil profiles	(0.07 – 2.1)	HNO_3-$HClO_4$ Fluorometric analysis	Lévesque 1974a
Alberta (sites throughout the province)	258 (0-15cm) (15-30cm)	0.476(±0.278) 0.474(±0.335)	Aqua regia Hydride generation AAS	Penny 2004
Alberta (sites throughout the province)	352	0.55 (±0.28) (0.1-2.7)	HF-HNO_3-$HClO_4$ AAS	R.G. Garrett, NRCan., pers. com. 2005
Saskatchewan (sites throughout the province)	526	0.53 (±0.28) (0.1-3.1)	HF-HNO_3-$HClO_4$ AAS	R.G. Garrett, NRCan., pers. com. 2005
Southwestern Saskatchewan	341 samples from 13 sites	*Ap horizon:* 8.49 (7.12-10.32) *C horizon :* 9.30 (7.37-12.68)	HF-HNO_3-$HClO_4$ AAS	Mermut *et al.* 1996 [unusually high levels – see text]
Saskatchewan	NR	(0.2-0.8)	NR	Agriculture and Agri-Food Canada 1996
Manitoba (sites throughout the province)	198	0.62 (±0.44) (0.1-4.7)	HF-HNO_3-$HClO_4$ AAS	R.G Garrett, pers. com. NRCan. 2005
Flin Flon	9 sites, n=3	1.8 (0.4-5.7)	HCl-HNO_3 ICP-MS	Jones and Hendenson 2006
Cranberry Portage	1 site, n=3	0.3 (0.2-0.3)		
The Pas	1 site, n=3	0.5 (0.5)		
Southern Manitoba	618 samples from 121 sites	0.5 (±0.4) (<0.2-1.2)	HF-HNO_3-$HClO_4$ AAS	Haluschak *et al.* 1998
Southern Manitoba	32	0.3 (<0.2-1.2)	Hydride generation AAS	Smith *et al*. 2004

Table 2. Background Concentrations of Selenium (µg/g) in Canadian Surface Soil

Location	Number of Samples	Soil Concentrations Mean (±s.d.) (Range)	Analytical Technique	Reference
Ontario				
Old urban parkland	60	1.3[a]	Hydride generation FAA	OMEE 1993
Rural parkland	101	(0.76[b]-1.7[c]) 0.93[a] (0.67[b]-2.0c)		
South of Sault Ste. Marie	294	0.46 (±0.38) (0.1-3.9)	$HF-HNO_3-HClO_4$ AAS	R. Garrett, NRCan, pers. com. 2005
Windsor, urban	18	1.59 (1.4-2.03)	Hydride generation FAA	Gizym 1994
Windsor, rural	12	0.89 (0.52-1.30)		
Ottawa, urban garden	50	0.7 (0.3-1.2)	$HF-HNO_3-HClO_4$ ICP-MS	Rasmussen *et al*. 2001
Port Hope	77	2.1 (<0.1-7.5)	Aqua regia ICP-MS	Sheppard *et al*. 2004
Agricultural Belt	228 (no sludge) 30 (with sludge)	0.35 (±0.22) (0.10-1.67) 0.37 (±0.15) (0.21-0.59)	Hydride generation Flame AAS	Frank *et al*. 1979
New Brunswick				
East St. John (urban)	18	<1	ICP-MS	Pilgrim and Schroeder 1997
West St. John (urban)	4	<1		
Fredericton area (rural)	2	<1		
Nova Scotia				
Sydney, N of Coke Ovens Area urban	90	0.77 (0.5 – 2.0)	U.S. EPA Method 3050A	JDAC Environ. Ltd 2001b JDAC Environ. Ltd 2001a
Rural	91	1.0 1.0 – 2.0		
Prince Edward Island	66 sites	0.229 (0.09-0.60)	HNO_3-HClO_4 Fluorometric analysis	Gupta and Winter 1975

Key:
NR – not reported
[a] 98th percentile
[b] lower concentration limit
[c] upper concentration limit
ICP-MS – Inductive Coupled Plasma Mass Spectrometry
FAA – Flameless Atomic Absorption Spectrometry
AAS - Atomic Absorption Spectrometry

Table 3. Average Selenium Concentrations in Canadian Foods[1]

Number	Food Composite	Se (µg/g)	Number	Food Composite	Se (µg/g)
A01	Milk, Whole	0.015	F04	Cake	0.066
A02	Milk, 2 %	0.015	F05	Cereals, Cooked Wheat	0.071
A03	Milk, 1%	0.013	F06	Cereals, Corn	0.028
A04	Milk, Skim	0.020	F07	Cereals, Oatmeal	0.032
A05	Evaporated Milk, Canned	0.029	F08	Cereals, Wheat and Bran	0.077
A06	Cream, 10 - 12 % bf	0.013	F09	Cookies	0.072
A07	Ice Cream	0.033	F10	Crackers	0.152
A08	Yogurt	0.014	F11	Danish and Donuts	0.151
A09	Cheese	0.124	F12	Flour, Wheat	0.383
A10	Cheese, Cottage	0.059	F13	Muffins	0.185
A11	Cheese, Processed, Cheddar	0.123	F14	Pancakes	0.132
A12	Butter	0.025	F15	Pasta, Mixed Dishes	0.176
B01	Beef, Steak	0.168	F16	Pasta, Plain	0.176
B02	Beef, Roast and Stewing	0.222	F17	Pie, Apple	0.056
B03	Beef, Ground	0.138	F18	Pie, Other	0.056
B04	Pork, Fresh	0.307	F19	Rice	0.052
B05	Pork, Cured	0.185	F20	Rolls and English Muffins	0.394
B06	Veal	0.127	G01	Baked Beans	0.024
B07	Lamb	0.077	G02	Beans	0.002
B08	Cold Cuts and Luncheon Meats	0.207	G03	Beets	0.004
B09	Luncheon Meats, Canned	0.102	G04	Broccoli	0.012
B10	Organ Meats, Liver and Kidney	1.044	G05	Cabbage	0.007
B11	Wieners	0.102	G06	Carrots	0.014
C01	Eggs	0.251	G07	Cauliflower	0.005
C02	Poultry, Chicken and Turkey	0.227	G08	Celery	0.010
D01	Fish, Marine, Fresh or Frozen	0.392	G09	Corn	0.014
D02	Fish, Fresh Water, Fresh or Frozen	0.133	G10	Cucumbers	0.013
D03	Fish, Canned	0.413	G11	Lettuce	0.004
D04	Shellfish, Fresh or Frozen	0.391	G12	Mushrooms, Canned	0.096
E01	Soups, Meat, Canned	0.032	G13	Onion	0.011
E02	Soups, Cream of (Name of Veg), Canned	0.009	G14	Peas	0.027
E03	Soups, Dehydrated	0.015	G15	Peppers	0.004
F01	Bread, White	0.410	G16	Potatoes, Raw	0.007
F02	Bread, Whole Wheat	0.392	G17	Potatoes, Baked	0.027
F03	Bread, Rye	0.393	G18	Potatoes, Boiled, Skins on	0.011

Table 3. Average Selenium Concentrations in Canadian Foods[1]

Number	Food Composite	Se (µg/g)	Number	Food Composite	Se (µg/g)
G19	Potatoes, Boiled, Without Skins	0.008	J08	Sugar, White	0.006
G20	Potatoes, Chips	0.009	J09	Syrup	0.007
G21	Rutabagas or Turnip	0.004	J10	Seeds, Shelled	0.635
G22	Tomato Juice, Canned	0.001	K01	Alcoholic Drinks, Beer	0.006
G23	Tomatoes	0.003	K02	Alcoholic Drinks, Wine	0.003
G24	Tomatoes/sauce Canned & Ketchup	0.028	K03	Coffee	0.003
H01	Apple Juice, Canned, Unsweetened	0.001	K04	Soft Drinks	0.002
H02	Applesauce, Canned, Sweetened	0.002	K05	Tea	0.001
H03	Apples, Raw	0.002	L01	Cereals	0.022
H04	Bananas	0.012	L02	Desserts	0.024
H05	Blueberries	0.002	L03	Dinners (Cereal + Veg + Meat)	0.041
H06	Cherries	0.003	L04	Dinners (Meat or Poultry + Veg)	0.027
H07	Citrus Fruit, Raw	0.003	L05	Formulae Milk Base	0.015
H08	Citrus Juice, Frozen	0.001	L06	Formulae Soya Base	0.008
H09	Citrus Juice, Canned	0.011	L07	Fruit	0.120
H10	Grape Juice, Bottled	0.007	L08	Meat, Poultry or Eggs	0.011
H11	Grapes	0.004	L09	Vegetables (Peas)	0.011
H12	Melons	0.002	M01	Popcorn (Microwave)	0.230
H13	Peaches	0.007	M02	Frozen Entrees (Boil)	0.107
H14	Pears	0.012	M03	Frozen Entrees (Microwave)	0.117
H15	Pineapple, Canned	0.019	M04	Frozen Entrees (Oven)	0.088
H16	Plums, Dried Prunes, & Canned Plums	0.003	M05	Frozen Entrees (Microwave)	0.112
H17	Raisins	0.005	M06	Frozen Dinner (Beef, Vegetable, Dessert)	0.046
H18	Raspberries	0.006	N01	Pizza	0.184
H19	Strawberries	0.005	N02	French Fries	0.046
I01	Cooking Fats and Salad Oils	0.012	N03	Hamburger	0.242
I02	Margarine	0.011	N04	Fish Burger	0.233
J01	Candy, Chocolate Bars	0.019	N05	Chicken Burger	0.216
J02	Candy, Suckers	0.011	N06	Hot Dog	0.260
J03	Gelatin Dessert	0.008	N07	Chicken (Breaded, Fried, Nuggets or Pieces)	0.198
J04	Honey	0.004	N08	Egg Breakfast on a Bun, Bagel, Muffin or Croissant	0.279
J05	Jams	0.007			
J06	Peanut Butter and Peanuts	0.035			
J07	Puddings	0.013			

[1] From the 1992 Total Diet Study (Dabeka 1994)

Table 4. Mean Consumption Rate of Various Food Groups by Canadians, from the 1970-72 Nutrition Canada Survey [1]

Food Group	Daily mean food intake[2] (g/person/day)			
	7 mo-4 yrs	5-11 yrs	12-19 yrs	20 yrs+
Milk & dairy	676.79	622.15	589.82	296.8
Meat, poultry & eggs	125.75	154.44	194.08	224.25
Fish & shellfish	3.35	8.37	11.22	13.85
Root vegetables	81.64	127.94	166.82	142.32
Other vegetables	78.63	116.8	147.87	161.07
Fruits & juices	189.21	201.68	159.5	185.89
Cereals & grains	168.09	299.73	324.87	246.84
Sugar & sweets	45.63	57.15	66.62	57.19
Fats, nuts & oils	6.86	14.42	18.91	14.7
Non alcoholic drinks	115.28	227.76	406.29	811.81
Alcoholic drinks	1.24	2.66	23.28	144.59

1 Health Canada (1994)

2 Infants are assumed to exclusively consume 0.75 L of breast milk/day (Health Canada 1994)

Table 5. Estimated Daily Selenium Intake via Food Consumption for the Canadian General Population[1]

Food Group Error! Bookmark not defined.	**Range of means**[2] **(**µg/g) (fresh weight)	**Daily mean intake from consumption of food**[3] (µgSe/person/day)			
		7 mo-4 yrs	5-11 yrs	12-19 yrs	20 yrs+
Milk & dairy	0.013 - 0124	11.70	11.22	11.32	6.67
Meat, poultry & eggs	0.032 – 1.044	20.16	25.94	36.01	40.62
Fish & shellfish	0.133 – 0.413	1.03	3.04	4.20	5.19
Root vegetables	0.004 – 0.046[4]	1.40	1.95	2.66	2.01
Other vegetables	0.001 – 0.028	1.03	1.15	1.96	1.62
Fruits & juices	0.001 – 0.019	0.82	0.90	0.67	0.82
Cereals & grains	0.032 – 0.410	31.81	66.64	72.82	56.95
Sugar & sweets	0.004 – 0.019	0.46	0.54	0.65	0.50
Fats, nuts & oils	0.011 – 0.635	0.23	0.64	0.84	0.60
Non alcoholic drinks	0.001 – 0.003	0.23	0.45	0.81	1.62
Alcoholic drinks	0.003 – 0.006	0.01	0.01	0.13	0.80
	Total (µg/day) [5]	**68.88**	**112.48**	**132.07**	**117.40**

1 Infants are assumed to exclusively consume 0.75 L of breast milk/day (Health Canada 1994) at an average concentration of 18 µg/L (L'Abbé *et al.* 1996); total selenium daily intake estimated to be 13.5 µg

2 Data from Table 3. (Dabeka 1994)

3 Calculated using food consumption rates from Health Canada (1996)

4 French fries contained 0.046 µg/g but the rest of root vegetables contained ≤0.0014 µ Se/g

5 22 food composites from Table 3. (L01-L09, M01-M06 and N01, N03-N08) were not used in the calculations to match the 112 food composites intake estimates from Health Canada (1994).

Table 6. Selenium Concentrations in Human Tissues and Biological Fluids[1]

Tissue or Fluid	Concentration Ranges	Country	Reference
liver	0.20 - 0.65 mg/kg	Canada, ON	Subramanian and Méranger 1982
	0.18 - 0.66 mg/kg	Canada	Underwood 1977
muscle	0.26 - 0.59 mg/kg	Canada	Underwood 1977
skin	0.12 - 0.62 mg/kg	Canada	Underwood 1977
whole blood	0.182 mg/L	Canada, ON	Dickson and Tomlinson 1967
	0.221 mg/L (geo. mean)	Canada, PQ	INSPQ, 2003
	0.1 - 0.3 mg/L	U.S.	Allaway *et al.* 1968
	0.070 – 0.237 mg/L	U.S.	Combs and Combs (1986)
	0.076 - 0.140 mg/L	Italy	Minoia *et al.* 1990
serum	0.134 mg/L (geo. mean)	Canada, PQ	INSPQ, 2003
	0.143 ±0.016 mg/L (mean ±s.d.)	Canada	Lalonde *et al.* 1982
	0.081± 0.016 mg/L (mean ±s.d.)	Canada	Dickson and Tomlinson 1967
	0.123 – 0.363 mg/L	U.S.	Longnecker *et al.* 1991
	0.092 – 0.168 mg/L (1^{st} – 99 th percentile)	U.S.	NHANES III 1988-94 in IOM (2000)
plasma	0.144 mg/L (mean)	Canada	Dickson and Tomlinson 1967
	0.081 - 0.225 mg/L	U.S.	Clark *et al.* 1984
	0.056 - 0.105 mg/L	Italy	Minoia *et al.* 1990
urine	0.0291 – 0.198 mg/L	Canada	Lalonde *et al.* 1982
	0.091 mg/L (geo. mean)	Canada, PQ	INSPQ, 2003
	0.02 - 0.113 mg/L	Japan	Hojo 1981
	0.002 - 0.031 mg/L	Italy	Minoia *et al.* 1990
Hair[2]	0.44 – 0.72 mg/kg	Canada, ON and NB	Holzbecher and Ryan 1978; Ryan *et al.* 1982. Gibson 1983
	0.36 - 0.57 mg/kg	U.S., China, Greece	Schroeder *et al.* 1970; Yang *et al.* 1983; Bratakos *et al.* 1990
Nails[3]	0.54 - 1.46 mg/kg	U.S., Netherlands, Greece	Longnecker *et al.* 1991; Hunter *et al.* 1990; van't Veer *et al.* 1990; Bratakos *et al.* 1990
dental enamel	0.12 - 0.9 mg/g	U.S.	Underwood 1977
saliva	1.1 - 5.2 mg/L	U.S.	Underwood 1977

1 Breast milk concentrations are described in section 2.5.11

2 Range of the reported mean hair concentrations from three separate studies.

3 Range of the reported mean nail concentrations from four separate studies.

Table 7. Existing Soil and Water Quality Criteria Proposed by Various Jurisdictions for Selenium

Medium	Jurisdiction	Description	Maximum concentration		Reference
SOIL (µg/g dry wt)	Canada	Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health	1	Agr	EC, 2001
			1	R/P	
			3.9	C/I	
	British Columbia	Generic Numeric Soil Standards for Contaminated Sites	2	Agr	BCMOE, 2005
			3	R/P	
			10	C/I	
	Ontario	Surface Soil Remediation Criteria. Potable/ Non-potable Groundwater Situation	2, --	Agr	OMOE, 1998
			10	R/P	
			10	C/I	
		Sub-surface Soil Remediation Criteria Potable/ Non-potable Groundwater Situation	2500	R/P	
			2500	C/I	
	Quebec	Generic Numeric Criteria for Soils	1	**A**	MDDEP, 2002
			3	**B**	
			10	**C**	
	U.S. EPA Region 9	Preliminary Remedial Goals for Soil	390	R	U.S. EPA 2004
			5100	I	
	U.S. EPA Region III	Risk-Based Concentrations for Soil Ingestion	390	R	U.S. EPA 2006b
			5100	I	
GROUNWATER (µg/L)	Canada	Canadian Environmental Quality Guidelines for the Protection of Aquatic Life and the Protection of Agricultural Water Uses	1	FAQL	CCREM, 1987
			20[a],50[b]	IR	
			50	LW	
	Ontario	Potable Groundwater Criterion for Contaminated Sites (all land uses)	10		OMOE, 1998
		Non-potable Groundwater Criterion for Contaminated Sites (all land uses)	50		
	British Columbia	Generic Numerical Water Standards for Contaminated Sites	10	FAQL	BCMOE, 2005
			540	MAQL	
	Quebec	Generic Groundwater Criterion for Contaminated Sites	20	SW	MDDEP, 2002
		Generic Potable Groundwater Criterion for Contaminated Sites	10	DW	
DRINKING WATER (µg/L)	Canada	Guidelines for Canadian Drinking Water Quality	10	MAC	Health Canada 1992
	British Columbia	Generic Numerical Water Standards for Contaminated Sites	10		BCMOE, 2005

Key: -- : not established

[a] Standard for continuous application on crops; [b] Standard for intermittent application on crops

A : background concentrations; **B** : moderate soil contamination which requires additional study;

C : threshold value that requires immediate cleanup

Agr: agricultural land use; R: residential land use; P: parkland land use; C: commercial land use; I: industrial land use;

FAQL: freshwater aquatic life; MAQL: marine (and/or estuarine) aquatic life

IR : irrigation LW : livestock watering

DW: groundwater used as drinking water

SW: groundwater seeping into surface water or infiltrating sewers

MAC: maximum acceptable concentration

Table 8. Toxicity of Selenium to Plants and Soil-Dwelling Invertebrates

Species	Effect	End-point	Effect Concentration µg/g	Exposure Period	Test Substrate	Reference
Selected Studies						
wheat (*Triticum aestivum*)	23% decrease in biodiversity 22% decrease in biomass and 28% decrease in grain yield	LOEC	2.5 (selenium as Na_2SeO_3) (Note: 2.5 was the lowest concentration tested)	50 days 135 days (maturity)	sandy soil; ; pH = 7.9; 0.1% organic carbon	Singh and Singh 1978
alfalfa	reduced shoot weight (91%, 74%, 23% and 27% reductions); greatest reduction in soils with lowest organic matter	LOEC	2 (selenium^{+6} as Na_2SeO_4)	NA	silty clay loam soils; pH range = 6.9 to 7.8; organic matter 3.1%, 3.7%, 5% and 6.5%	Soltanpour and Workman 1980
	no effect; reduced shoot weight (94%)	NOEC LOEC	2 4 (selenium^{+6} as Na_2SeO_4)		silty clay loam soil; pH = 7.0; organic matter 6.3%	
alfalfa	no effect	NOEC	0.5 (selenium^{+6} as Na_2SeO_4)	NA	sandy loam soil (pH 6.7; 13% organic matter); two clay loam soils (pH 5.6, 6.9%; organic matter 15%, 13%)	Wan *et al.* 1988
	reduced shoot weight (83%, 33% and 56%)	LOEC	1.5 (selenium^{+6} as Na_2SeO_4)			
sorgrass (*Sorghum vulgare*)	reduction in shoot weight (59% and 53%)	LOEC	1 selenium^{+6} as Na_2SeO_4) (Note 1, was lowest concentration tested)	42 days	loamy sandy soil; 19% organic matter; pH 5.5 and 6.0	Carlson *et al.* 1991
sorgrass (*Sorghum vulgare*)	no effect on shoot weight	NOEC	up to 4 (selenium^{+4} as Na_2SeO_3)		loamy sandy soil; 19% organic matter; pH 5.5 and 6.0	Carlson *et al.* 1991
sorgrass (*Sorghum vulgare*)	reductions in shoot weight (64% and 61%, respectively)	LOEC	1 (selenium^{+6} as Na_2SeO_4) 2 (selenium^{+4} as Na_2SeO_3),		sandy soil; 11% organic matter; pH = 4.9	Carlson *et al.* 1991
sorgrass (*Sorghum vulgare*)	no effect on shoot weight	NOEC	up to 4 (selenium^{+4} as Na_2SeO_3)		sandy soil; 11% organic matter; pH = 6.5	Carlson *et al.* 1991
Cowpea (*Vigna sinensis*)	Dry matter yield	NOEC LOEC	1.0 2.5 (as elemental Se, $Na_2SeO_3 \cdot H_2O$, H_2SeO_3)	50 days	sandy soil, pH 8.0, 0.08% organic carbon	Singh and Singh 1979
Cowpea (*Vigna sinensis*)	Dry matter yield	LOEC	1.0 (as Na_2SeO_4)	50 days	sandy soil, pH 8.0, 0.08% organic carbon	Singh and Singh 1979
Studies Consulted But Not Used	Reduction in reproductive output (number of cocoons per worm)	LOEC	77 selenium as sodium selenite	NA	soil	Fischer and Koszorus 1992
Studies Consulted But Not Used						

Table 8. Toxicity of Selenium to Plants and Soil-Dwelling Invertebrates

Species	Effect	End-point	Effect Concentration µg/g	Exposure Period	Test Substrate	Reference
Beetle (*Tenebrio molitor)*	reduced survival	LOEC	0.125% sodium selenite	NA	nutrient medium	Hogan and Razniak 1991
Indian mustard (*Brassica juncea*)	Reduced dry matter yield reported for plants grown in water culture, but not for plants grown in soil	NA	NA (as Na_2SeO_4)	60 days	commercial compost mixture, pH = 6.5 to 7.0	Bañuelos *et al.* 1997
Abyssinian mustard (*Brassica carinata*)	Reduced dry matter yield reported for plants grown in water culture, but not for plants grown in soil	NA	NA (as Na_2SeO_4)	60 days	commercial compost mixture, pH = 6.5 to 7.0	Bañuelos *et al.* 1997
Coffee (*Coffea arabica*)	Reduced growth (height, leaf area, dry mass)	LOEC	1000 µM aqueous solution added to soil once a week (as Na_2SeO_3)	14 weeks	soil and sand (1:1)	Mazzafera 1998
Coffee (*Coffea arabica*)	Reduced growth (height, leaf area, dry mass)	LOEC	100 µM aqueous solution added to soil once a week (as Na_2SeO_3)	14 weeks	soil and sand (1:1)	Mazzafera 1998

NA = not available

Table 9. Toxicity of Selenium to Soil Microbial Processes

Effect	Endpoint	Concentration (µg/g)	Soil pH	Test Substrate	Reference
reduced arylsulfatase activity	LOEC	198	NA	soil with lowest clay content	Al-Khafaji and Tabatabai 1979
reduced oxygen consumption	LOEC	484	NA	NA	Lighthart *et al.* 1977
reduced amidase activity	LOEC	1975	NA	soil with lowest pH and organic matter	Frankenberger and Tabatabai 1981
reduced soil acid and alkaline phosphatase activity	LOEC	1975	NA	NA	Juma and Tabatabai 1977

NA = not available

Table 10. Toxicity of Selenium to Livestock and Terrestrial Wildlife

Organism	Body Weight (kg)	Ingestion Rate (g/d)	Effect	End-point	Estimated dose (µg/g bw per day)	Exposure Concentrations (µg/g bw per day)	Source of selenium	Exposure period	Exposure route	Reference
Mallard duck (*Anas platy-rhynchos*)	1	100[b]	Adult weight Adult survival Adult survival Duckling survival Deformed embryo Deformed embryo	NOEL NOEL LOEL LOEL NOEL LOEL	1 1 10 2.5 0.5 1	0.1, 0.5, 1.0, 2.5, 10	$Na_2SeO_3 \cdot 5H_20$	78 days	diet	Heinz *et al.* 1987
Mallard duck (*Anas platy-rhynchos*)	1	100[b]	Duckling survival Duckling survival	NOEL LOEL	0.4 0.8	0.1, 0.2, 0.4, 0.8, 1.6	Selenomethionine	100 days	diet	Heinz *et al.* 1989
Mallard duck (*Anas platy-rhynchos*)	Approx. 0.025 at start, 0.95 at 6 wks in control	From 30 at start to 150 by 6 wks	Mortality Food consumption Body weight Liver weight	NOEL LOEL NOEL LOEL NOEL LOEL LOEL	3.2[j] 6.3[j] 1.6[j] 3.2[j] 1.6[j] 3.2[j] 1.6[j]	0, 10, 20, 40, 80 µg/g	Sodium selenite	6 weeks	diet	Heinz *et al*. 1988
Mallard duck (*Anas platy-rhynchos*)	Approx. 0.025 at start, 0.95 at 6 wks in control	From 30 at start to 150 by 6 wks	Mortality Food consumption Body weight	NOEL LOEL NOEL LOEL NOEL LOEL	3.2[j] 6.3[j] 1.6[j] 3.2[j] 1.6[j] 3.2[j]	0, 10, 20, 40, 80 µg/g	Selenomethionine	6 weeks	diet	Heinz *et al*. 1988

Table 10 (continued). Toxicity of Selenium to Livestock and Terrestrial Wildlife

Organism	Body Weight (kg)	Ingestion Rate (g/d)	Effect	End-point	Estimated dose (µg/g bw per day)	Exposure Concentrations (µg/g bw per day)	Source of selenium	Exposure period	Exposure route	Reference
Mallard duck (*Anas platy-rhynchos*)	1.2[i] (adult males)	115	Food avoidance	NOEL LOEL	0.48 0.96	0, 5, 10, 20 µg/g	Seleno-DL-methionine	4 days	diet	Heinz and Sanderson 1990
Mallard duck (*Anas platy-rhynchos*)	0.03 at hatch, 0.19 at 2 wks	9 to 62 (varied with treat-ment)	Food consumption Body weight	NOEL LOEL NOEL LOEL	4.9[j] 9.8[j] 4.9[j] 9.8[j]	0, 15, 30 µg/g	Selenomethionine (L and DL isomers)	2 weeks	diet	Heinz *et al*. 1996; Hoffman *et al*. 1996
Mallard duck (*Anas platy-rhynchos*)	0.03 at hatch, 0.19 at 2 wks	9 to 62 (varied with treat-ment)	Survival	NOEL LOEL	4.9[j] 9.8[j]	0, 15, 30 µg/g	Seleno-L-methionine	2 weeks	diet	Heinz *et al*. 1996; Hoffman *et al*. 1996
Screech owl	0.2 [0.176-0.183 (M); 0.208-0.219 (F)]	25[c]	Adult body mass Egg size Reproduction (eggs laid, hatchability, nestling survival)	NOEL LOEL NOEL LOEL NOEL LOEL	1.25 3.75 1.25 3.75 1.25 3.75	0, 4.4, 13.2 µg/g ww (equivalent to 0, 10, 30 µg/g dw)	Seleno-DL-methionine	13.7 weeks through reproduction	diet	Wiemeyer and Hoffman 1996
Black-crowned night-heron	0.883[d]	160.6[e]	Reproductive	NOEL	1.8[f]	1.8, 5.5	Selenomethionine	94 days through reproduction	diet	Smith *et al.* 1988
American Kestrel (*Falso sparverius*)	0.12[i]	36[i]	Body weight, signs of illness	NOEL	2.7	0, 5, 9 µg/g dw	Seleno-L-methionine	77 days	diet	Yamamoto *et al*. 1998
American Kestrel (*Falso sparverius*)	0.12[i]	36[i]	Reproduction	NOEL	3.6	0, 6, 12 µg/g dw	Seleno-L-methionine	11 weeks (until the end of egg laying)	diet	Santolo *et al*. 1999

Table 10 (continued). Toxicity of Selenium to Livestock and Terrestrial Wildlife

Organism	Body Weight (kg)	Ingestion Rate (g/d)	Effect	End-point	Estimated dose (µg/g bw per day)	Exposure Concentrations (µg/g bw per day)	Source of selenium	Exposure period	Exposure route	Reference
Chicken, adult	1.6[g]	110[h]	Wirey chick down Chick mortality	NOEL LOEL	0.17 0.34	0.17, 0.34, 0.69, 1.8	Na_2SeO_3 at highest dose (1.8); seleniferous corn, barley, and wheat for all other doses.	several weeks	diet	Moxon 1937
			Hatchability Deformed embryo	NOEL LOEL	0.34 0.69					
			Food consumption Body weight Egg production	NOEL LOEL	0.69 1.8					
Chicken, adult	1.6[g]	110[h]	Hatchability	NOEL	0.2	0.007, 0.07, 0.2, 0.3, 0.6	Na_2SeO_3	16-28 weeks	diet	Ort and Latshaw 1978
			Egg production Egg weight Fertility	NOEL	0.3					
			Hatchability	LOEL	0.3					
			Egg weight Egg production	LOEL	0.6					
Pony	NR	NR	Acute selenosis and mortality	NOEC LC100	4 6	0, 2, 4, 6 µg/g bw (single dose)	Sodium selenite	Single exposure, observed for 2 weeks	Stomach tube	Stowe 1980
Pony	NR	NR	Acute selenosis and mortality	NOEL LC100	6 8	6, 8 µg/g bw (no control) (single dose)	Sodium selenite	Single exposure, observed for 2 weeks	Stomach tube	Stowe 1980
Nubian goat	NR	NR	Mortality	LC100	<40	0, 40, 80, 160 µg/g bw (single dose)	Sodium selenite	Single exposure	Oral drench	Ahmed et al. 1990
Nubian goat	NR	NR	Clinical signs of selenosis, mortality	NOEL LOEL	1 5	0, 0.25, 0.5, 1, 5, 20	Sodium selenite	220 days	Oral drench	Ahmed et al. 1990

Table 10 (continued). Toxicity of Selenium to Livestock and Terrestrial Wildlife

Organism	Body Weight (kg)	Ingestion Rate (g/d)	Effect	End-point	Estimated dose (µg/g bw per day)	Exposure Concentrations (µg/g bw per day)	Source of selenium	Exposure period	Exposure route	Reference
Sheep	23-38	NR	Reduced food intake, depression, tachypnea	NOEL LOEL	1 2	0, 1, 2, 3, 4 µg/g bw (single dose)	Sodium selenite	Single exposure, observed for 7 days	Stomach tube	Tiwary et al. 2006
Sheep	23-38	NR	Reduced food intake, depression, tachypnea	NOEL LOEL	3 4	0, 1, 2, 3, 4, 6, 8 µg/g bw (single dose)	Seleno-DL-methoionine	Single exposure, observed for 7 days	Stomach tube	Tiwary et al. 2006
Sheep	NR	NR	Mortality	LC50	5	0, 5 µg/g bw (single dose)	Sodium selenite	Single exposure. Observed for 60 hours	Oral	Smyth et al. 1990
Sheep	NR	NR	Mortality	LC75	5	0, 5 µg/g bw (single dose)	Sodium selenite	Single exposure, observed for 60 hours	Intraperitoneal injection	Smyth et al. 1990
Sheep	33-56	1000-2700[a]	Not specified	LOEL	0.08	Not specified	Not specified	1 year	Not specified	Puls 1994
Pig	24.7 (initial weight)	2380	Body weight, hair loss and separation of hoof at coronary band site	NOEC LOEC	0.48[J] 0.96[J]	0, 5, 10, 15, 20 µg/g food	Sodium selenite	12 weeks	diet	Kim and Mahan 2001
Pig	24.7 (initial weight)	2380	Hair loss and separation of hoof at coronary band site	NOEC LOEC	0.96[J] 1.45[J]	0, 5, 10, 15, 20 µg/g food	Se-enriched yeast (selenomethionine)	12 weeks	diet	Kim and Mahan 2001
Pig	8.0 (initial weight)	790	Reduced weight gain and food intake	NOEC LOEC	0.4[J] 0.8[J]	0, 4, 8, 12, 16, 20 µg/g food	Sodium selenite	5 weeks	diet	Goehring et al. 1984a
Pig	NR	1000	Neurological signs of selenosis	EC40	25 µg/g dw food	0.4 (control), 25 µg/g dw food	Seleno-DL-methoionine	6 weeks	diet	Panter et al. 1996
Pig	NR	1000	Neurological signs of selenosis	EC80	25 µg/g dw food	0.4 (control), 25 µg/g dw food	Sodium selenate	6 weeks	diet	Panter et al. 1996
Pig	NR	1000	Paralysis or other	EC100	25 µg/g dw food	0.4 (control), 25 µg/g dw food	Organic selenium from the plant	6 weeks	diet	Panter et al.

Table 10 (continued). Toxicity of Selenium to Livestock and Terrestrial Wildlife

Organism	Body Weight (kg)	Ingestion Rate (g/d)	Effect	End-point	Estimated dose (µg/g bw per day)	Exposure Concentrations (µg/g bw per day)	Source of selenium	Exposure period	Exposure route	Reference
			neurological signs of selenosis				*Astragalus bisulcatus*			1996
Pig	8.0 (initial weight)	1090	Weight gain and food intake,	NOEC	> 1.14 [J]	0.5 ,2.6, 5.6, 8.4 µg/g food	Seleniferous wheat and oats	6 weeks	diet	Goehring et al. 1984b
Pig	8.4 (initial weight)	2090	Weight gain and food intake,	NOEC	> 2.07 [J]	0.5, 2.6, 5.7, 8.3 µg/g food	Sodium selenite	17 weeks	diet	Goehring et al. 1984b
Cow	42	NR	Lassitude, inappetance, inability to rise, mortality	NOEL LOEL	1 2	0, 1, 2 µg/g bw (single dose)	Sodium selenite	Single exposure, observed for 7 days	Intramuscular injection	MacDonald et al. 1981
Cow	70-120	NR	Clinical signs of selenosis	LOEL	0.25	0, 0.25	Sodium selenite	16 weeks	Oral	Kaur et al. 2003
Cow	NR	NR	Tissue lesions	NOEL	> 0.80	0, 0.15, 0.28, 0.80	Sodium selenite	120 days	Diet	O'Toole and Raisbeck 1994
Cow	NR	NR	Tissue lesions	NOEL LOEL	0.28 0.80	0, 0.15, 0.28, 0.80	Seleno-methionine	120 days	Diet	O'Toole and Raisbeck 1994
Pronghorn Antelope	NR	NR	Clinical signs of selenosis	NOEL	> 15 µg/g dw food	0.3, 15 µg/g dw food	Organic selenium in grass hay and alfalfa	164 days	Diet	Raisbeck et al. 1996

[a] Water consumption rate (mL/d).
[b] Calculated by Sample *et al.* (1996b) using allometric equation from U.S. EPA (1988).
[c] Calculated by Sample *et al.* (1996b) using food consumption data from Pattee *et al.* (1988).
[d] As determined by Dunning (1993).
[e] Calculated by Sample *et al.* (1996b) using equation for herons by Kushlan (1978).
[f] Data on reproduction incomplete for highest dose level, therefore lowest dose level was selected as NOEL.
[g] As determined by U.S. EPA (1988).
[h] Calculated using allometric equation by U.S. EPA (1988).
[I] As determined from U.S. EPA (1993).
[J] Estimated doses were calculated based on body weights and ingestion rates from controls.

Table 11. Toxicity of Selenium to Other Mammalian Species

Organism	Body Weight (kg)	Ingestion Rate (g/ d)	Effect	Endpoint	Estimated dose (µg/g bwper day)	Exposure Concentrations (µg/g bw per day)	Source of selenium	Exposure period	Exposure route	Reference
Long-tailed macaques	4.25	7.5 *,§	Fetal mortality Fetal mortality Adult toxicity Adult toxicity	NOEL LOEL NOEL LOEL	0.025 0.15 0.025 0.15	0.025, 0.15, 0.3	L-seleno-methionine	days 20-50 of gestation	nasogastric intubation	Tarantal *et al.* 1991
Rat	0.35*	46 §,ψ	No. second generation young reduced by 50%	NOEL LOEL	0.20 0.33	0.20, 0.33, 0.99	Potassium selenate	1 yr through 2 generations	drinking water	Rosenfeld and Beath 1954
Rat, F344 (female)	0.124*	14**	Growth rate Body weight	NSF	<0.16	0, 0.16, 0.24	Na_2SeO_3	28 weeks	diet	Lijinski *et al.* 1989
Rat	0.0614	14**	Weight gain	NOEL LOEL	0.59^ 1.28^	0.5 ,2.6, 5.6, 8.4 µg/g food	Seleniferous wheat and oats	4 weeks	diet	Goehring et al. 1984b
Rat	0.060	14**	Weight gain	NOEL LOEL	0.61^ 1.33^	0.5, 2.6, 5.7, 8.3 µg/g food	Sodium selenite	4 weeks	diet	Goehring et al. 1984b
Mouse	0.03*	5.5 ψ	Incidence of runts Failure to breed	LOEL	<0.76	0.76	SeO_4	3 generations	diet and drinking water	Schroeder and Mitchener 1971b
Mouse	0.03*	3.5 §	Offspring weight	NOEL	>0.21	0.10, 0.21	$Na_2SeO_3 \cdot 5H_2O$	30 d prior to reproduction and through day 18 of gestation	drinking water	Nobunaga *et al.* 1979
Mouse	0.034	7.5 §,ψ	Reduced gestation period Larger litter	NOEL	>0.055	0.055	K-seleno-carageenan	unclear - appears to be through gestation	drinking water	Chiachun *et al.* 1991

* Body weight was determined by the U.S. EPA (1988) for the same organism.
§ Water consumption rate (ml/d).
ψ Calculated by Sample *et al.* (1996b) using allometric equation from U.S. EPA (1988).
** Ingestion rate was determined by the U.S. EPA (1988) for the same organism.
^ Estimated doses were calculated based on body weights and ingestion rates from controls.
NSF Not Statistically Significant.

Table 12. Dietary Reference Intakes (DRIs) for Selenium (adapted from IOM 2000)

Life Stage Group	RDA[1] (µg/day)	AI[2] (µg/day)	EAR[3] (µg/day)	UL[4] µg/day
Infants (M & F)				
0-6 months	ND[5]	15	ND	45
7 – 12 months	ND	20	ND	60
Children (M & F)				
1 – 3 years	20	ND	17	90
4 – 8 years	30	ND	23	150
Males and Females				
9 – 13 years	40	ND	35	280
14 – 18 years	55	ND	45	400
19 – 30 years	55	ND	45	400
31 – 50 years	55	ND	45	400
50 – 70 years	55	ND	45	400
>70 years	55	ND	45	400
Pregnancy				
≤ 18 years	60	ND	49	400
19 – 30 years	60	ND	49	400
31 – 50 years	60	ND	49	400
Lactation				
≤ 18 years	70	ND	59	400
19 – 30 years	70	ND	59	400
31 – 50 years	70	ND	59	400

1 Recommended Dietary Allowance
2 Adequate intake
3 Estimated Average Requirement
4 Tolerable Upper Intake Level
5 Not determined

Table 13. Typical Values for Average Body Weights and Intakes of Air, Water and Soil by the Canadian General Population

Age class (years)	Body weight[1] (kg)	Air intake[1] (m^3/day)	Water intake [1] (L/day)	Soil/dust intake[2] (g/day)
0-6 mo. (infant)	8.2	2.1	0.75 (water mixed with formula or breast milk)[3]	0.02
7 mo.-4 yrs (toddler)	16.5	9.3	0.6	0.08
5-11 yrs (child)	32.9	14.5	0.8	0.02
12-19 yrs (adolescent)	59.7	15.8	1.0	0.02
20+ yrs (adult)	70.7	15.8	1.5	0.02

1 Richardson 1997

2 CCME 2006

3 Health Canada 1994; Public Health Agency 2000

Table 14. Estimated Average Total Daily Intake of Selenium Via All Routes of Exposure by the General Canadian Population

Medium	Typical Selenium Levels	Daily selenium intake (µg/person/day)				
		0-6mo	7mo-4yrs	5-11yrs	12-19yrs	20+yrs
Air[2]	0.001 µg/m^3	0.0021	0.0093	0.0145	0.0158	0.0158
Drinking water[3]	0.05 µg/L	ND/ exclusively breast-fed	0.3	0.4	0.5	0.75
Soil[4]	0. 7 µg/g	0.002	0.007	0.002	0.002	0.002
Dust[5]	1.2 µg/g	0.021	0.084	0.021	0.021	0.021
Food[6]	See Table 3	13.5[7]	68.9	112.5	132.1	134.9[8]
	Total intake (µg/day)	13.5251	69.3003	112.9375	132.6388	135.6888
	Total intake (µg/kg bw/day)	1.65	4.20	3.43	2.22	1.92

1 Based on body weights and intake rates in Table 12

2 Based on Ontario background air concentration of 1.0 ng Se/m^3.

3 Based on 0.05 µg Se/L in Ontario drinking water.

4 Assuming a background soil concentration of 0.7 µg/g – see text "human exposure estimates", and time apportionment of 3hsr /day outdoors (Leech *et al*. 1996; U.S. EPA 1997)

5 Assuming a background indoor dust concentration of 1.2µg/g – see text "human exposure estimates", and time apportionment of 21hrs/day indoors (Leech *et al*. 1996; U.S. EPA 1997)

6 Based on food intake rates in Table 4.

7 Based on average concentration of 18 µg/L breast milk from Eastern Ontario donors and a milk intake of 0.75 L/day

8 Based on food intake and multivitamin/multimineral supplements

Table 15. Selenium Interactions with Other Substances

Chemical	Nature of Interaction	References
arsenic	-protects against and reduces selenium toxicity; synergism with methylated selenium metabolites; interferes with gastrointestinal absorption and expiratory and biliary excretion of selenium	Levander 1977; Levander and Baumann 1966; Obermeyer *et al.* 1971
cadmium	-selenium protects against cadmium toxicity in rats; protective effects are believed to be due to formation of a selenium-cadmium complex of high molecular weight	Flora *et al.* 1982; Jamall 1983; Ohta and Imamiya 1986
lead	-selenium decreases lead toxicity; organ lead and selenium levels concurrently following simultaneous exposure	Andersen and Nielsen 1994
fluoride	-may increase toxicity of selenium to rats	Moxon and Dubois 1939; Hadjimarkos 1969
iodine	-apparent antagonistic relationship between iodine and selenium	Foster 1993; Contempre *et al.* 1991a,b
mercury	-simultaneous administration of equivalent doses of mercury and selenium decreases toxicity of both chemicals	Chang 1983; Hansen 1988; Skerfving 1978
methionine	-antagonistic relationship with selenium; decreases selenium toxicity; believed to detoxify selenium by either forming methylated derivatives which are readily excreted, or through a protein synthesis mechanism	Lombeck *et al.* 1987; Tarantal *et al.* 1991;
vitamin E	-antagonistic relationship with selenium; complex interactions with selenium compounds in repairing oxidative damage to cell membranes	Levander and Morris 1970
silver	-antagonist of selenium; selenium has been shown to protect against the toxic effects of silver in rats; antagonism believed to be due to formation of silver selenides which are of low solubility and toxic potency	Ebyl *et al.* 1992
sulfate	-does not appear to protect against selenium-induced liver damage but does appear to reduce growth inhibition that results from oral exposure to high doses of selenates or selenites	Halverson and Monty 1960
antimony, germanium, bismuth	-apparent antagonistic relationship	Paul *et al.* 1989
vitamin C	-may increase absorption and toxicity of selenium in humans	HSDB 1993; Lombeck *et al.* 1987; Mack 1990

Table 16. Soil Quality Guidelines and Check Values for Selenium (µg/g)

	Land use			
	Agricultural	**Residential/ parkland**	**Commercial**	**Industrial**
Guideline (SQG_F)	**1[a]**	**1[a]**	**2.9[a]**	**2.9[a]**
Human health guidelines/check values				
SQG_{HH}	80[b]	80[b]	125[b]	1135[b]
Direct contact (SQG_{DH})	80	80	125	4050
Protection of indoor air quality - basement (SQG_{IAQ})	NC[c]	NC[c]	NC[c]	NC[c]
Protection of indoor air quality - slab-on-grade (SQG_{IAQ})	NC[c]	NC[c]	NC[c]	NC[c]
Off-site migration check (SQG_{OM-HH})	—	—	1135	1135
Protection of potable water (SQG_{PW})	NC[d]	NC[d]	NC[d]	NC[d]
Produce, meat and milk check (SQG_{FI})	NC[e]	NC[e]	—	—
Environmental health guidelines/check values				
SQG_E	1[f]	1[f]	2.9[f]	2.9[f]
Soil contact (SQG_{SC})	1	1	2.9	2.9
Soil contact confidence rank[h]	F	F	F	F
Soil and food ingestion (SQG_I)	4.5	—	—	—
Protection of freshwater life (SQG_{FL})	NC[d]	NC[d]	NC[d]	NC[d]
Livestock watering (SQG_{LW})	NC[d]	NC[d]	NC[d]	NC[d]
Irrigation water (SQG_{IR})	NC[d]	NC[d]	NC[d]	NC[d]
Nutrient and energy cycling check (SQG_{NEC})	NC[g]	NC[g]	NC[g]	NC[g]
Off-site migration check (SQG_{OM-E})	—	—	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

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

[a] 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.

[b] The SQG_{HH} is the lowest of the human health guidelines and check values.

[c] The inhalation of indoor air check applies to volatile organic compounds and is not calculated for inorganic contaminants.

[d] 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.

[e] The produce, metal 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.

[f] The SQG_E is the lowest of the environmental health guidelines and check values.

[g] Data are insufficient/inadequate to calculate these environmental guidelines/check values.

[h] For an explanation of the soil contact confidence rank, refer to CCME (2006).