CCME

Canadian Council of Ministers of the Environment

Le Conseil canadien des ministres de l'environnement

SCIENTIFIC CRITERIA DOCUMENT FOR CANADIAN SOIL QUALITY GUIDELINES FOR THE PROTECTION OF HUMAN HEALTH:

BERYLLIUM

PN 1535

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The Canadian Council of Ministers of the Environment (CCME) is the primary minister-led intergovernmental forum for collective action on environmental issues of national and international concern.

This scientific criteria document provides the background information and rationale for the development of Canadian Environmental Soil Quality Guidelines for beryllium. The information in this document is current as of 2014, when the document was revised and updated. For additional scientific information regarding these guidelines, please contact:

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These guidelines are included as updates in the Canadian Environmental Quality Guidelines, which was published by CCME in October of 1999. The Canadian Environmental Quality Guidelines are available online at http://ceqg-rcqe.ccme.ca/.

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.

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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 provide a healthy, *functioning* ecosystem capable of sustaining the existing and likely future uses of the site by ecological receptors and humans. Canadian soil quality guidelines can be used as the basis for consistent assessment and remediation of soils at contaminated sites in Canada.

This report was prepared by Health Canada for the CCME Soil Quality Guidelines Task Group. The Guidelines were calculated according to procedures described in *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 2006). According to this protocol, both environmental and human health soil quality guidelines are developed and the lowest value generated from the two approaches for each of the four land uses is recommended by CCME as the Canadian Soil Quality Guidelines (CCME 2006).

This scientific criteria document provides the background information and rationale for the derivation of human health soil quality guidelines for beryllium. This document contains a review of the chemical and physical properties of beryllium, the sources and emissions in Canada, the distribution and behaviour of beryllium in the environment and the toxicological effects of beryllium in humans and mammalian species. This information is used to calculate soil quality guidelines for beryllium to protect human receptors in four types of land uses: agricultural, residential/parkland, commercial, and industrial.

The human health soil quality guidelines for beryllium for each of the four land uses are: 75 mg/kg for agricultural land use, 75 mg/kg for residential/parkland land use, 110 mg/kg for commercial land use, and 1100 mg/kg (ILCR 10^{-5}) or 550 mg/kg (ILCR 10^{-6}) for industrial land use. The human health soil quality guidelines were selected from direct human health-based soil quality guidelines for soil ingestion and dermal contact for non-cancer effects, direct human health-based soil quality guidelines for particulate inhalation for cancer effects and the off-site migration check.

The Canadian Soil Quality Guidelines for the protection of environmental and human health, as recommended by the Canadian Council of Ministers of the Environment are based on the lowest of the environmental soil quality guidelines or the human health-based soil quality guidelines (CCME 2006). The soil quality guidelines presented in this document are higher than the interim (1991) criteria, therefore, the Soil Quality Guidelines for the protection of environmental and human health are based on the interim remediation criteria for soil (CCME 1991). They are: 4 mg/kg for agricultural land use, 4 mg/kg for residential/parkland land use, 8 mg/kg for commercial land use and 8 mg/kg for industrial land use.

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 un écosystème sain, capable de supporter les utilisations actuelles et probables du site par les récepteurs écologiques et humains. 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 sites contaminés de Santé Canada pour le Groupe de travail sur les recommandations pour la qualité des sols du CCME. 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). Conformément à ce protocole, les recommandations pour la qualité des sols en fonction de l'environnement et de la santé humaine sont développées et la plus petite valeur générée de ces deux approches, pour chacune des quatre utilisations des terrains, est recommandée par le CCME comme étant la recommandation canadienne pour la qualité des sols (CCME 2006).

Ce document scientifique contient l'information sur les données de base et la justification pour la détermination des recommandations pour la qualité des sols pour la santé humaine pour le béryllium. Ce document contient une revue de l'information sur les propriétés chimiques et physiques du béryllium, une revue des sources et émissions au Canada, la distribution et le comportement du béryllium dans l'environnement et les effets toxicologiques du béryllium chez les humains et les animaux de laboratoire. Cette information est utilisée pour l'élaboration des recommandations pour la qualité des sols relatives au béryllium afin de protéger la santé humaine dans quatre vocations des terrains: agricole, résidentielle/parc, commerciale et industrielle.

Les recommandations pour la qualité des sols en fonction de la santé humaine pour les quatre vocations de terrain sont : 75 mg/kg de sol pour l'utilisation résidentielle/parc et pour les terrains agricoles et 110 mg/kg de sol pour les sols à vocation commerciale. Pour les terrains à vocation industrielle, la recommandation pour la qualité des sols est 1100 mg/kg (ILCR 10^{-5}) ou 550 mg/kg (ILCR 10^{-6}). Les recommandations pour la qualité des sols visant la protection de la santé humaine ont été sélectionnées parmi les voies d'exposition humaines suivantes : l'ingestion et le contact cutané pour les effets non-cancérogènes, l'inhalation pour les effets cancérogènes ainsi que la migration hors site de sol et de poussière provenant des terrains commerciaux ou industriels.

Les recommandations canadiennes pour la qualité des sols visant la protection de l'environnement et de la santé humaine, tel que indiqué par le Conseil canadien des ministres de l'environnement sont basées sur les recommandations les plus faibles des recommandations visant la protection de l'environnement ou de la santé humaine (CCME 2006). Les recommandations pour le qualité des sols déterminés dans ce document sont plus élevés que les Critères provisoires canadiens de qualité environnementale pour les lieux contaminés (1991) par conséquent les recommandations pour la qualité des sols sont basées sur les critères provisoires canadiens de qualité environnementale pour les lieux contaminés qui sont de : 4 mg/kg pour les

terrains à vocation agricole, 4 mg/kg pour les terrains à vocation résidentielle/parc, 8 mg/kg pour les terrains à vocation commercial et 8 mg/kg pour les terrains à vocation industrielle (CCME 1991).

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1. INTRODUCTION

Canadian Environmental Quality Guidelines are intended to protect, sustain, and enhance the quality of the Canadian environment and its many beneficial uses. They are generic numerical concentrations or narrative statements that specify levels of toxic substances or other parameters in the ambient environment that are recommended to protect and maintain wildlife and/or the specified uses of water, sediment, and soil. These values are developed through the Canadian Council of Ministers of the Environment (CCME) and are recommended for toxic substances and other parameters (e.g., nutrients, pH) of concern in the ambient environment.

The development of Canadian Soil Quality Guidelines was initiated through the National Contaminated Sites Remediation Program (NCSRP) in 1991. In response to the urgent need to begin remediation of high priority "orphan" contaminated sites, an interim set of criteria was adopted from values currently in use in various jurisdictions across Canada (CCME 1991). Although the NCSRP program officially ended in March of 1995, the development of soil quality guidelines was pursued by CCME because of the continued need for soil quality guidelines for the management of soil quality with a particular focus on remediation of contaminated sites.

Canadian Soil Quality Guidelines are developed according to procedures that have been described by *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 2006). According to this protocol, both environmental and human health soil quality guidelines are developed for four land uses: agricultural, residential/parkland, commercial, and industrial. The lowest value generated by the two approaches for each of the four land uses is recommended by CCME as the Canadian Soil Quality Guideline. Guidelines for a number of substances were developed using this protocol and released in a working document entitled Recommended Canadian Soil Quality Guidelines (CCME 1997). The guidelines originally published in that document have since been revised and are now superseded by the Canadian Soil Quality Guidelines for the protection of environmental and human health published by CCME in October of 1999 and updated regularly since. The interim soil quality criteria (CCME 1991) should be used only when soil quality guidelines based on the CCME protocol have not yet been developed for a given chemical.

This scientific criteria document provides the background information and rationale for the derivation of environmental soil quality guidelines for beryllium for the protection of human health. Guidelines for the protection of the environment have not been updated in this report; therefore interim soil quality criteria (CCME 1991) remain valid for the protection of the environment. This document contains a review of information on the chemical and physical properties of beryllium, sources and emissions in Canada, the distribution and behaviour of beryllium in the environment and the toxicological effects of beryllium on the health of humans and experimental animals. In addition, various check mechanisms which consider indirect pathways of exposure (i.e., off-site migration of substances via wind and water erosion) are used to provide protection for resources and receptors not otherwise considered in the calculation of soil guidelines.

The Canadian Soil Quality Guidelines presented in this document are intended as general guidance. Site-specific conditions should be considered in the application of these values (see CCME 1996b for guidance on developing site-specific soil objectives). The reader is referred to

CCME (2006) for further generic implementation guidance pertaining to the guidelines. Soil quality guidelines are calculated to approximate a "no- to low-" effect level (or threshold level) based only on the toxicological information and other scientific data (fate, behaviour, etc.) available for the substance of concern, and they do not consider socioeconomic, technological, political factors or lifestyle choices. These non-scientific factors are to be considered by site managers at the site-specific level as part of the risk management process. Because these guidelines may be used and applied differently across provincial and territorial jurisdictions, the reader should consult the laws and regulations of the jurisdiction they are working within for applicable implementation procedures.

2. BACKGROUND INFORMATION

2.1. Physical and Chemical Properties

Beryllium (Be) CAS 7440-41-7 was historically referred to as glucinium. It is the first element of the alkaline earth metals and has a valency of two. It appears in Group IIA of the periodic table and has two common oxidation states, Be (0) and Be (+2). Beryllium is the lightest of all the solid and chemically stable elements with an atomic number of 4, a molecular weight of 9.01 and a specific gravity of 1.85 at 25°C. It has a melting point of 1287°C and a boiling point of 2468°C. Beryllium also has a high specific heat, high heat of fusion, high strength-to-weight ratio and excellent electrical and thermal conductivities. The physical and chemical properties of beryllium and some beryllium compounds are summarised in Table 1 (ATSDR 2002a; Haynes 2011).

Beryllium metal is hard, brittle and grey white in colour. It is soluble in acids (except nitric acid) and alkalis. It is resistant to oxidation at ordinary temperatures but is susceptible to oxidation at higher temperature. It has high heat capacity and thermal conductivity. Beryllium in reaction with acid and alkalis produces beryllium hydride (BeH_2) and beryllium hydroxide $Be(OH)_2$.

Beryllium compounds are formed with Al, Cu, Ni, Co and/or Fe such as: beryllium-aluminium alloy (CAS 12770-50-2), beryllium chloride (CAS 7787-47-5), beryllium fluoride (CAS 7787-49-7), beryllium hydroxide (CAS 13327-32-7), beryllium oxide (CAS 1304-56-9), beryllium phosphate (CAS 13598-15-7), beryllium sulphate (CAS 13510-49-1), beryllium sulphate tetrahydrate (CAS 7787-56-6), beryllium zinc silicate (CAS 39413-47-3) and beryl ore (CAS 1302-52-9) (ATSDR 2002a; Zorn *et al*. 1988).

^{9}Be is the only stable isotope of beryllium and natural beryllium contains 100% of this isotope (Kabata-Pendias and Pendias 2000; Newland 1982; Stonehouse *et al.* 1992). The stable isotope ^{9}Be is a trace component in all continental rocks, which is released by weathering and transferred to the ocean predominantly via rivers (Frank *et al*. 2009).

Beryllium, like aluminium has a high oxygen affinity which results in the formation of stable surface films of beryllium oxide (BeO) which protect from further oxidation, thus providing high resistance to corrosion, water and cold oxidising acids (Laul and Norman 2008).

Cationic salts of beryllium (chloride, fluoride, nitrate, phosphate and tetrahydrated sulphate) are hydrolysed in water to form acidic solutions (WHO 2001). At pH from 5 to 8 beryllium reacts to

form insoluble hydroxides or hydrated complexes and insoluble beryllates (beryllium oxides) are formed at pH greater than 8 (Reeves 1986; WHO 2001). BeO fumes are emitted when the following compounds are heated to decomposition: beryllium, beryllium-aluminium alloy, beryllium chloride, beryllium fluoride, beryllium hydroxide, beryllium oxide, beryllium sulphate and beryllium sulphate tetrahydrate. In addition, beryllium chloride emits fumes of hydrochloric acid and other chlorinated compounds. Beryllium fluoride emits fumes of hydrofluoric acid and other fluorinated compounds. Beryllium phosphate emits fumes of phosphorus oxides (PO_x). Beryllium sulphate and beryllium sulphate tetrahydrate emit fumes of sulfur oxides (SO_x) (Cooper and Harrison 2009).

Beryllium (Be) is six times harder than steel and it is also three times lighter than aluminium (Kolanz 2001; Taylor and Sauer 2002). It has other exceptional properties, such as high electric and thermal conductivity, high melting point (1278°C), good resistance to corrosion, high permeability to X-rays, the lowest neutron absorption cross-section of any metal, high modulus of elasticity and it is non-magnetic. Those unique properties make beryllium essential in many industries including: aerospace, electronics, nuclear, automotive, telecommunication, computer, jewellery and ceramics (Sharma *et al.* 2000). It is also used in dental laboratories as a component of dental amalgams and prostheses (Kotloff *et al.* 1993).

Beryllium-copper alloys are corrosion resistant, highly elastic, non-magnetic, non-sparking, electrical and thermal conductors and have extraordinary hardness which all contribute to the commercial significance of this compound (WHO 1990). Few other intermetallic compounds are industrially significant because of their brittleness and because of the low solubility of most elements in solid beryllium (WHO 1990).

Table 1: Physical and Chemical Properties of some Beryllium Compounds

Property	Beryllium metal	Beryllium fluoride	Beryllium hydroxide (alpha)	Beryllium oxide	Beryllium basic carbonate
Chemical Formula	Be	BeF_2	$Be(OH)_2$	BeO	$Be(OH)_2(CO_3)_2$
CAS no.	7440-41-7	7787-49-7	13327-32-7	1304-56-9	66104-24-3
Molecular Weight/Atomic Number	9.01[a]	47.01[a]	43.03[a]	25.01[a]	181.07[a]
Colour	Grey[a]	Colourless[a]	White[a]	White[a]	White[a]
Physical State (@25ºC)	Solid; hexagonal crystal[a]	Tetragonal crystalline or glassy; hygroscopic[a]	Powder or crystalline[a]	Hexagonal crystals[a]	Powder[a]
Melting Point (ºC)	1287[a]	552[a]	Decomposes around 200ºC[a]	2578[a]	No data
Boiling Point (ºC, 1 atm)	2468[a]	1283[a]	Not applicable[b]	3787[b]	No data
Density (g/cm^3@ 25ºC)	1.85[a]	2.1[a]	1.92[a]	3.01[a]	No data
Solubility	Insoluble in water. Soluble in acid & alkaline solutions.[a]	Very soluble in water. Slightly soluble in alcohol.[a]	Slightly soluble in water & alkaline solutions. Soluble in acid.[a]	Insoluble in water. Slightly soluble in acids & alkaline solutions.[a]	Insoluble in water. Soluble in acid & alkaline solutions.[a]

Table 1: Physical and Chemical Properties of some Beryllium Compounds -Con't

Property	Beryllium chloride	Beryllium sulphate (tetrahydrate)	Beryllium nitrate (tetrahydrate)	Beryllium phosphate (trihydrate)	Beryllium sulphate
Chemical Formula	$BeCl_2$	$BeSO_4 \cdot 4H_2O$	$Be(NO_3)_2 \cdot 4H_2O$	$Be(PO_4)_3 \cdot 3H_2O$	$BeSO_4$
CAS no.	7787-47-5	7787-56-6	13510-48-0	-	13510-49-1
Molecular Weight/ Atomic Number	79.92[a]	177.14[a]	205.08[b]	271.03[b]	105.08[a]
Colour	White/Yellowish[a]	Colourless[a]	White[b]	White[b]	Colourless[a]
Physical State (@25ºC)	Ortho-rhombic crystals, hygro-scopic[a]	Tetragonal crystals[a]	Crystals[b]	Solid[b]	Tetragonal crystals; hygroscopic[a]
Melting Point (ºC)	415[a]	~100 (decomposes)[a]	60.5[b]	100 (loses water, decomposes)[b]	1127[a]
Boiling Point (ºC, 1 atm)	482[a]	400 (loses $4H_2O$)[b]	142 (decomposes)[b]	No data	No data
Density (g/cm^3@25ºC)	1.90[a]	1.71[a]	1.557[b]	No data	2.5[a]
Solubility	71.5g/100g in water. Very soluble in alcohol, ether & pyridine. Insoluble in benzene & toluene.[a]	41.3g/100g in water Insoluble in alcohol.[a]	166g/100g in water.[b]	Soluble in water & acetic acid.[b]	41.3g/100g in water.[a]

[a] CRC Handbook (Haynes 2011)
[b] ATSDR (2002a)

2.2. Geochemical Occurrence

Beryllium was not known to ancient or medieval civilisations. It was first recognised by the French scientist, Nicholas Louis Vauquelin in 1798 as a component of the mineral beryl, and named it beryllium. Beryl is associated with the gemstones: emerald (green), aquamarine (light blue) and beryl (yellow) (ATSDR 2002a; Mineral Information Institute 2010). Metallic beryllium was not isolated until 1828, by Friederich Wohler (Kolanz 2001; Mineral Information Institute 2010).

Beryllium occurs naturally and is the 44th most abundant element in the Earth's crust (Sabey 2006). It is present in a variety of materials including rocks, coal, oil, soil and volcanic dust. Beryllium is found in earth's surface rocks at a concentration ranging from 1 to 15 mg/kg. Due to its high reactivity, beryllium is not found as a free metal in nature (ATSDR 2002a).

Over 100 mineral species with essential beryllium are known to date (Kabata-Pendias 2011). Eight of them are represented by oxides and hydroxides, four by borates, 24 by phosphates, two by arsenates and 61 by silicates (including one arsenite-silicate) (Pekov *et al.* 2008). In 2008, Pekov *et al.* reported a new mineral species - Niveolanite, the first natural carbonate of beryllium. It was found in the Poudrette pegmatite obtained from the Poudrette Quarry in Mont Saint-Hilaire, Québec (Pekov *et al*. 2008).

Of the >100 beryllium-containing minerals currently identified, only two beryllium minerals are of commercial importance for the production of beryllium. Bertrandite ($Be_4Si_2O_7(OH)_2$), contains less than 1% beryllium and is the principal beryllium mineral mined in the United States. Bertrandite is found in certain volcanic rocks derived from granite (ATSDR 2002a; Jaskula 2009). Beryl ($Be_3Al_2Si_6O_{18}$), contains about 4% beryllium and is the principal mineral mined in the rest of the world (Jaskula 2009).

Most of the world's beryllium is found in plagioclase feldspar. However, pyroxenes, micas, and clays contain substantial amounts of the element (Griffitts and Skilleter 1990). On average, common rocks and minerals contain from <1 ppm to about 10 ppm beryllium whereas ores can contain several thousand ppm (Fishbein 1981).

Beryllium is found at low levels throughout Canada in areas with igneous bedrock, mostly in northern British Columbia and the Northwest Territories (CAREX 2011). Beryllium occurrences in Canada fall into two broad classes of deposits. The main class includes pegmatites in high-temperature and pegmatitic quartz veins, and disseminations in granite. The other class is helvite-danalite occurrences in contact-metamorphic or skarn deposits (Mulligan 1960). The presence of beryllium has been documented in the Cordilleran region (British Columbia), Western Precambrian Shield (Western Ontario, Manitoba and Northwest Territories), Eastern Precambrian Shield (Eastern Ontario, Québec and Labrador) and the Appalachian Region (New Brunswick, Nova Scotia and Newfoundland and Labrador) (Mulligan 1960). Beryllium deposits are also found in Sea Lake, Labrador as barylite (Christie and Brathwaite 1999). Nepheline syenite and other nepheline-bearing rocks may contain up to 0.1% BeO (Christie and Brathwaite 1999). At a rare metals deposit at Thor Lake (near Yellowknife, Northwest Territories) five deposits have been identified as a resource of a number of metals including beryllium (Christie and Brathwaite 1999).

There are several aquamarine deposits in Canada; the most notable among them is the True Blue aquamarine showing in Yukon (Turner 2005). There are currently fourteen known beryl occurrences in New Brunswick (Beal and Lentz 2010).

Most beryl in British Columbia is found in pegmatites associated with leucocratic granites (alaskites, quartz monzonites) and in late quartz veins that cut the host intrusion or country rock (Legun 2005). There are only 10 officially-documented occurrences of beryl in Yukon (Lewis *et al.* 2003) and 40 occurrences of beryl and 3 for other beryllium minerals in British Columbia (Legun 2005).

In 2010, Nemaska Exploration of Québec City reported that its Whabouchi lithium-beryllium deposit had an "impressive" width. Results of the current drill program include 92.1 metres at 1.39% Li_2O and 133 ppm beryllium, 77.8 metres at 1.49% Li_2O and 170 ppm beryllium, and 70.3 metres at 1.39% Li_2O and 171 ppm beryllium. The property is located in the Lac des Montagnes Formation (Canadian Mining Journal 2010; Théberge 2009).

Six of the 18 shale formations analysed by McIlveen (1998) in Ontario contained beryllium concentrations which exceeded 1.2 ppm (beryllium criteria for Ontario in 2004). The most significant exceedance occurred in all Animikie-Gunflint formations where the beryllium concentrations were 2.7 ppm or greater (McIlveen1998).

2.3. Analytical Methods

Reliable and sensitive analytical procedures are required for the determination of beryllium not only in support of toxicity, bioavailability and bioaccumulation studies but also to investigate trends in human exposure and to map and control levels in tissues and body fluids, food and drinking water where the element normally occurs at the µg/kg level and below (Vaessen and Szteke 2000).

Currently, atomic spectrometry techniques are the preferred analytical methods (Vaessen and Szteke 2000). For the spectrophotometric and both flame atomic absorption spectrometry (AAS) methods, the detection limit for beryllium is 5 µg/L (test solution). For the flameless AAS-technique (ET-AAS) the detection limit is 0.2 µg/L and for both inductively coupled plasma (ICP) procedures it is 0.3 µg/L (Vaessen and Szteke 2000).

The US EPA sample preparation methods for extraction of beryllium from environmental media samples include:

- EPA Method 3050B *Acid Digestion of Sediments, Sludges, and Soils* (hydrochloric acid digestion) (US EPA 1996)
- EPA Method 3005A *Acid Digestion of Waters for Total Recoverable or Dissolved Metals for Analysis for FLAA or ICP Spectroscopy (nitric acid digestion) for extraction from surface and groundwater samples* (US EPA 1992)
- EPA Method 3015 *Microwave Assisted Acid Digestion of Aqueous Samples and Extracts* (US EPA 2007a). The estimated instrumental detection limit (DL) is 0.3 µg/L soil digested in 100 mL water.

The US EPA analytical method 6020 *Inductively Coupled Plasma with Mass Spectrometry* is a more sensitive technique, with an estimated instrumental detection limit (DL) of <0.01 µg/L (US EPA 2007b). This method is applicable to groundwater, aqueous samples, industrial wastes, soils, sludges, sediments, other solid wastes and any other samples requiring preliminary treatment by acid digestion to determine total beryllium content. Water samples must be filtered and acid-preserved prior to analysis to determine dissolved beryllium content (US EPA 2007b).

The analytical methods recommended by CCME (1993) for the determination of beryllium in water and wastewater samples include:

- Method 3111D *Direct Nitrous Oxide-Acetylene Flame Method, for the Determination of Metals*. This method is not as sensitive for beryllium as either the electrothermal atomic absorption method (SM-3113B) or the plasma emission methods (SM-3120B). The detection limit is 5 µg/L.
- Method SM 3113B *Electrothermal Atomic Absorption Spectrometric of Water and Wastewater.* The detection limit is 2 µg/L.

- Method SM 3120B *Inductively Coupled Plasma (ICP) Method, for the Determination of Metals*. The detection limit is 0.3 µg/L.

For more information concerning the analytical methods for determining beryllium in environmental samples, refer to Table 7.2 in the ASTDR 2002a document.

2.4. Production and Uses in Canada

World production of beryllium was estimated to be approximately 180 metric tons in 2008 (USGS 2009). World resources in known deposits of beryllium have been estimated to be more than 80 000 tons. About 65% of these resources come from the United States (USGS 2009), which is one of only three countries known to process beryl ores and beryllium concentrates into beryllium products (NMAB 2008).

A number of beryllium-bearing areas in Canada have been prospected and examined for commercial production (IARC 1993); however, beryllium production levels for Canada were not available at the time of writing this document. There is little documentation regarding mining or commercial extraction of beryllium in Canada (Buffa 1994 *pers. com*.; EMRC 1990; Holliday *et al.* 1987; Soja and Sabin 1986).

In 2008, Canada imported 58 kg beryllium from the United States and in 2010 exported to 3485 kg to the United States in the form of alloys unwrought, waste or scrap/powders based on the harmonised system code HS811211 (UN 2010). The value of exported beryllium has shown a notable decrease (-31.6%) in 2007 (CMY 2007).

Although beryllium was discovered more than 200 years ago, the commercial value of beryllium became recognised in 1926, when beryllium-copper-nickel alloy was patented. Beryllium was initially used in military equipment and then in products such as fluorescent lights and neon signs.

Beryllium and beryllium alloys have been used in consumer products since the middle of the 20th century. The first major use for beryllium was in phosphors for fluorescent lights but this application was abandoned because of the potential health hazard (Kriebel *et al.* 1988a; Venugopal and Luckey 1978).

Due to their strength, light weight, good thermal and electrical conductive properties, neutron-moderating properties, beryllium and beryllium alloys were used in experimental nuclear reactors and the production of nuclear weapons, radar and in other defence applications during World War II and the subsequent Cold War. The demand for beryllium has declined since the end of the Cold War because of the decrease in production of nuclear weapons (Kolanz 2001).

Beryllium alloys, predominantly beryllium-copper are extensively used in the electronics field and in the manufacture of bushings, bearings, welding electrodes, non-sparking tools, precision instruments, submarine cable housings, automobiles, computers, golf club heads, camera shutters, air bag sensors, electro-magnetic shielding, fire control sprinkler heads, aircraft landing gear bushings and moulds in the plastic industry (ATSDR 2002a; Hoover 2005; US EPA 1987b; WHO 1990; Zorn *et al.* 1988). In Canada, beryllium-copper alloys are used in the manufacture and production of electrical springs, welding electrodes, fuse clips, contacts, connectors, fastenings, resistance welding to manufacture of automobiles and large household appliances and in the melting production of moulds and castings (Holliday *et al.* 1987). Beryllium-nickel alloys are used in diamond drill bit matrixes, watch balance wheels and aircraft/spacecraft parts (Reeves 1986;

Venugopal and Luckey 1978; WHO 1990). Beryllium-aluminium alloys are used in light aircraft construction. In Canada, beryllium-nickel-chromium alloys are found in dental prostheses and cement used to repair crowns and bridges (Bertolini 1989; Covington *et al.* 1985; EC 1974; Holliday *et al.* 1987; WHO 1990). In dentistry, a nickel alloy is used because it is an economical alternative to precious metals. The nickel alloy typically contains 1 to 2% beryllium (Haberman *et al*. 1993).

Beryllium oxide (BeO) is currently used in the manufacture of high-technology ceramics, crucibles, electronic heat sinks, electrical insulators, gyroscopes, military vehicle armour, microwave ovens, laser tubes, rocket nozzles and thermocouple tubing (ATSDR 2002a; Luttrell 2008; Reeves 1986; US EPA 1987b; Zorn *et al.* 1988). Beryllium oxide is also used as a component of gas mantles and acetylene lamps (Griggs 1973). In Canada, BeO is used mainly in the production of heat sink and insulator ceramics, laser bores and tubes and hybrid microcircuits (EC 1974; Hoover 2005; US Patent 1991).

Since 1999, there has been a steady decline in the consumption of beryllium in the United States because of concerns about the health and environmental problems of beryllium oxide (NMAB 2008). Import data suggest a similar declining trend in Canada (UN 2010). The decreased use, in conjunction with the health and environmental issues, is driving up the cost of producing beryllium and stimulating interest in the development of substitute materials.

IARC (1993) and ATSDR (2002a) reclassification of beryllium as an inhalation carcinogen in both animals and humans may lead to a further decreases in use.

2.5. Sources and Concentrations in the Canadian Environment

Major sources of beryllium emissions into the environment are anthropogenic; however, natural sources of airborne beryllium are windblown dust and volcanic particles. It is estimated that wind-blown dust contributes 5 metric tons of beryllium per year and volcanic particles contribute 0.2 metric tons per year to the atmosphere (NAP 2007). Deposition of atmospheric beryllium also adds to water concentrations however, the relative contributions of these sources cannot be assessed. Beryllium can also enter the water through the weathering of rocks and soils (ATSDR 2002a).

Virtually every industry (e.g., electric power stations, heating and industrial plants, gasoline combustion, non-ferrous metal smelters, kiln operations in cement plants and refuse incineration) will emit heavy metals via high temperature processes into the atmospheric, aquatic, and terrestrial ecosystems (Wilson *et al.* 1998). In 1970, Canadian beryllium emissions from coal, coke and heavy oil combustion were estimated at 7.1×10^3 kg (EC 1974). Increased emissions from the combustion of fossil fuels have been projected as a result of an expected increase in the use of these fuels for power generation and heating (Fishbein 1981). Beryllium is not included in the National Pollutant Release Inventory (EC 2014). Recent emission data are not available.

The most important source of exposure to beryllium in the general environment is the burning of coal (IARC 1993). In the 1980s, estimated beryllium emissions in the United States from the combustion of coal, fuel oil and petroleum-based products accounted for approximately 93% (1.87×10^5 kg per year) of total beryllium emissions (US EPA 1987b; WHO 1990). However, emission values are only broad estimates since not all facilities are required to report (ATSDR 2002a) and there is variation reported in the beryllium content of the combustible materials.

The concentration of beryllium in Canadian feed-coals varies between 0.8 and 1.2 mg/kg (ppm). (Goodarzi 2002). Fuel oil contains approximately 0.08 ppm beryllium (Fishbein 1981). U.S. coal generally contains less than 0.4 to 8 ppm (mean value 1.5 ppm) beryllium and occasionally up to 100 ppm in the organic matter (Merian 1984). According to Fishbein (1981) world coals contain 0.1 to 1000 ppm. Beryllium has been recognised as concentrating in coals, with an average of 46 ppm in coal ash (Christie and Brathwaite 1999).

Atmospheric releases following emission treatment are not well documented (US EPA 1987b). Reports regarding releases from coal combustion range from less than 4% (Gladney and Owens 1976) to 84% (Phillips 1973). Ito *et al.* (2006) analysed trace elements in the coal used in modern coal-fired power plants in Japan. The mean concentration in coal was 2.07 mg/kg (n=131, dry weight, obtained from 86 brands of coal collected from 21 plants at 13 stations; note: all coal imported from Australia, China, Indonesia, U.S.A, Canada and South Africa) and the mean emission rate was 2.75 mg/kWh (n=25).

Beryllium emissions are also produced by foundries, machine shops, propellant and ceramic plants, incinerators, municipal waste combustors and open-burning waste disposal sites (Cleverly *et al.* 1987; Hallenbeck *et al.* 1993; US EPA 1987a; WHO 1990). Emissions from ore production and post-ore production metallurgical processes are localised and the overall contribution is very small to negligible (US EPA 1987b; WHO 1990). In Canada, it was estimated that less than 0.5 kg of beryllium was released by manufacturing processes in 1970 (EC 1974).

Beryllium waste disposal problems do not exist (Stonehouse *et al.* 1992). Beryllium is expensive to produce (Stonehouse *et al.* 1992; US EPA 1987b) and most dusts and solid and liquid wastes from production processes and pollution control devices are recycled into production or sold for recovery of beryllium (ATSDR 2002a; WHO 1990). Wastes with low beryllium content are discarded or in the case of Cu-Be alloys, salvaged for the copper content (WHO 1990). 98.5% of non-recyclable beryllium wastes were disposed on-land in the United States in 2002 (ATSDR 2002a). 1.4% was released to the air and 0.1% was released into surface waters (ATSDR 2002a).

Soils and sediments reflect the composition of parent material, resulting in higher metal concentrations in mineralised areas that could result in their classification as "contaminated sites" (Grunsky 2010). Shale deposits have been reported to contain concentrations above regional background levels; which may be reflected in beryllium concentration in surface soils or soil parent material immediately above shale deposits (McIlveen 1998).

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

Data representing environmental concentrations of beryllium in Canada are presented hereafter for air, dust, soil, surface water, groundwater, drinking water, sediment, biota used as human food, commercial foods and human breast milk. In some cases, the data reported came from published sources; however, some of the information was also provided as unpublished (raw) data. The monitoring data provided is dynamic and should be considered representative of the best available information for the specified time period.

In the case of beryllium, many of the reported concentrations were at or below analytical detection (MDL). In order to calculate environmental concentrations, non-detect concentrations were assumed to be equal to ½ MDL. Details regarding the portion of data for each media requiring conversion to one-half the detection limit are discussed individually in the sections below.

2.5.1. Ambient Air

The National Air Pollution Surveillance (NAPS) Network is a joint program of the federal and provincial governments which monitors and assesses the quality of the ambient air in Canadian urban centres. Particulate matter with aerodynamic diameters less than 10 μm (PM_{10}) and less than 2.5 μm ($PM_{2.5}$) are measured and sample filters are analysed for 50 elements (including beryllium), 14 inorganic and organic anions and 11 inorganic cations (EC 2011).

The mean beryllium concentration in ambient air is 0.013 ng/m^3 (arithmetic mean, SD=0.011, n=3054, range=0-0.087 ng/m^3) (HC 2011). This mean is based on NAPS data ($PM_{2.5}$ fraction analysed by ICP-MS following acid digestion) collected from 2003 to 2009 from British Columbia, Ontario, Québec and New Brunswick from urban and rural centres. ICP-MS data are not currently available for the remaining provinces and territories. Ninety-five percent of the data was reported as less than detection limit but only 53% was assumed to be equal to one-half the detection limit to determine the concentration of beryllium in ambient air (those not flagged as below the detection limit were not converted to ½ MDL). These data were used to develop the beryllium estimated daily intake (EDI) values for inhalation of ambient air used in the soil quality guideline (SQG) calculation. The EDI methodology is further discussed in Section 5.2 of this document.

A summary of available environmental concentrations for ambient air is provided in Appendix 1.

2.5.2. Indoor Air

Due to technical difficulties associated with monitoring very low concentrations of metals in air, there are a limited number of studies pertaining to beryllium concentrations in indoor air. Additionally, there is no Canadian database for indoor air concentrations (Rasmussen *et al.* 2006).

Beryllium concentrations in indoor air samples (n=47) collected in the Windsor Air Quality Study were all less than the detection limit of 0.25 ng/m^3 (Bell *et al.* 1994). These data were used to develop the beryllium EDI values for inhalation of indoor air used in the SQG calculation. The EDI methodology is further discussed in Section 5.2 of this document.

A summary of available environmental concentrations for indoor air is provided in Appendix 1.

2.5.3. Indoor Dust

Similar to indoor air, no database exists for concentrations of beryllium in indoor settled dust in Canada. One study is available which provides measured values for samples collected within Ottawa, Ontario (Rasmussen *et al.* 2001). 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 Ottawa. Metal content was determined by ICP-MS; the beryllium detection limit was 0.02 mg/kg. In house dust (n=48), beryllium concentrations ranged from 0.28-1.0 mg/kg with a mean of 0.56 mg/kg (dry weight) (Rasmussen *et al.* 2001). No values were reported as less than the detection limit. These

data were used to develop the beryllium EDI values for exposure to indoor dust used in the SQG calculation. The EDI methodology is further discussed in Section 5.2 of this document.

A summary of available environmental concentrations in indoor dust is provided in Appendix 1.

2.5.4. Soil

While beryllium concentrations in soil throughout Canada vary based on geology and anthropogenic inputs; a single background soil concentration is required to develop soil quality guideline for use throughout Canada. Data from geological surveys conducted by both the Geological Survey of Canada (GSC) and the New Brunswick Department of Natural Resources (NBDNR) were used to develop the concentrations distribution of beryllium in Canadian soil (HC 2011). Based on till data (<63µm) from this data set, the mean beryllium concentration in soil was determined to be 0.75 mg/kg (arithmetic mean, SD=0.99, n=9876, range = 0.25-16 mg/kg) (HC 2011). Samples were analysed by AAS/ICP-ES following aqua-regia digestion (partial digestion by HCl and HNO_3). 66.8% of the data were reported as less than the detection limited and assumed to be equal to ½ MDL to calculate the concentration of beryllium in soil. These data were used to develop the beryllium EDI values for exposure to soil used in the SQG calculation. The EDI methodology is further discussed in Section 5.2 of this document. Worldwide ranges of 4-6 mg/kg are commonly cited (Kabata-Pendias 2011).

It is recognised that till may not be representative in terms of the geochemical composition of surficial soils to which people are generally exposed to since there is limited exposure to till. The background concentration of beryllium in till was used to represent background concentrations of beryllium in Canada without anthropogenic input. For site-specific assessments, local background concentrations of beryllium in surface soils may be preferable, both to estimate the background soil concentration (BSC) and estimated daily intake (EDI) for beryllium.

Localised Canadian soil concentrations for beryllium in soils are summarised in Appendix 1 and discussed below. The reported concentrations may not be directly comparable, as methods of sample digestion will influence the degree to which beryllium is leached from the soil matrix as discussed in previous section regarding analytical methods.

The Ontario Ministry of Environment and Energy derived Ontario Typical Range (OTR) values for concentrations of beryllium in urban and rural parkland, which were approximately 0.52 mg/kg and 0.47 mg/kg, respectively (OMEE 1993). These values are slightly lower than the mean value obtained from the GSC and NBDNR for partial analysis and fall between the 50th and 75th percentiles of the GSC and NBDNR data. Thus, the average beryllium concentrations in Ontario soil appear to be similar to those from the rest of Canada (HC 2011).

Beryllium concentrations may be elevated in soil from locations influenced by coal combustion, nuclear weapons test sites, or beryllium industries (Kabata-Pendias 2011).

2.5.5. Surface Water

Releases of beryllium to surface water can occur through runoff from slag and ash dumps, coal piles and beryllium containing waste sites (ATSDR 2002a; Kubiznakova 1987; Saha and Roy 1992).

Surface water data were available for rivers, streams, and lakes in British Columbia, Alberta,

Saskatchewan, Manitoba, Ontario and Newfoundland and Labrador. In the majority of collected samples, total beryllium concentrations in surface waters were below analytical detection. Reported detection limits (DLs) varied according to province, but may be used to represent environmental beryllium concentrations for surface waters in British Columbia (DL=0.05 µg/L), Alberta (DL=0.2 µg/L), Saskatchewan and Manitoba (DL=1 µg/L) and Ontario (DL=0.1 µg/L) (Anderson 2004 *pers. comm*; Boyd 2004 *pers. comm.*; Cheug 2004 *pers. comm.*; Regnier and Ryan 1998; Yee 2004 *pers. comm.*).

A summary of concentrations in surface water is provided in Appendix 1. An average concentration of beryllium in Canadian surface waters was not determined for the purposes of setting human health based soil quality guidelines. Surface water used as a source for drinking water is addressed in the drinking water portion of this section.

Radioactive Beryllium

According to Frank *et al.* (2009), the deep Canada Basin shows elevated concentrations of ^{10}Be of up to 900 atoms/g in the uppermost 25 m followed by pronounced minima of between 200 and 300 atoms/g at 35-150 m depth, again followed by an increase to concentrations of 300-500 atoms/g below 400 m depth.

On the other hand, surface waters in the Canada Basin show low ^{9}Be (stable isotope) concentrations of around 10 pmol/kg in the uppermost 50 m, followed by an increase to values between 17 and 31 pmol/kg at 150 m depth underlain by again low concentrations of 9-15 pmol/kg to depths of 3700 m (Frank *et al*. 2009).

2.5.6. Groundwater

Water samples were collected from two natural artesian flows in Simcoe County, Ontario and analysed using inductively coupled plasma-sector field mass spectrometry (ICP-SMS). The mean value for the Johnson site (n=11) was 0.55± 0.06 ng/L and 0.56± 0.11 ng/L for the Parnell site (n=12) (Shotyk *et al.* 2010).

Groundwater monitoring data for beryllium were provided for deep (n=100) and shallow (n=111) wells located across Alberta (Holt-Oduro 2004 *pers. comm.)*. Collectively, all the beryllium concentrations analysed in groundwater (EPA 200.8, APHA 3210B or SW 6010) were below analytical detection limits (i.e.<0.054 µg/L, <1 µg/L, or <2 µg/L, respectively).

A summary of beryllium concentrations in groundwater is provided in Appendix 1. An average concentration of beryllium in Canadian groundwater was not determined for the purposes of setting human health based soil quality guidelines. Groundwater used as a source for drinking water is addressed in the drinking water portion of this section.

2.5.7. Drinking Water

The background beryllium concentration in Canadian drinking water is estimated to be 0.04 µg/L (arithmetic mean, SD=0.07, n=3841) based on data obtained from the Ontario Ministry of Environment (1998-2007) and Saskatchewan Ministry of Environment (2000-2009) (HC 2011). 91% of the data were below the detection limit and was assumed to be equal to one-half the detection limit to calculate the concentration of beryllium in drinking water. Data from other provinces were not available to include in estimating the national background value. These data

were used to develop the beryllium EDI values for exposure to drinking water used in the SQG calculation. The EDI methodology is further discussed in Section 5.2 of this document.

The mean beryllium concentration in Ontario treated drinking water is estimated to be 0.03 µg/L (SD=0.05, n=3777, range=0.025–1.85 µg/L) based on data from the Drinking Water Surveillance Program (DWSP) for years 1998 through to 2007 (OMOE 2010). Data were not available from the DWSP program for years beyond 2007.

The mean beryllium concentration in Saskatchewan drinking water is estimated to be 0.44 µg/L (SD=0.15, n=64, range=0.05–0.5 µg/L) based on data from the Government of Saskatchewan's (GS) Ministry of Saskatchewan Environment (MSE) for years 2000 through 2009 (GS 2008).

A summary of beryllium concentrations in drinking water is provided in Appendix 1.

2.5.8. Sediments

Beryllium concentrations in lake sediments of the Carswell Structure in Northwestern Saskatchewan ranged from less than 0.1 to 2.8 mg/kg with a mean of 0.5 mg/kg (Dunn 1980). Beryllium concentrations in sediments from 14 sites in Tadanac Lake near Parry Sound, Ontario, an undisturbed Precambrian Shield lake, ranged from 1.3 to 2.1 mg/kg with a mean of 1.7 mg/kg (Wren *et al.* 1983).

Beryllium concentrations determined in 83 samples of marine sediments, taken from various regions of the Bay of Fundy, Nova Scotia ranged from 0.8 to 2.9 mg/kg with an average of 1.6 mg/kg (Loring 1979). In argillaceous sediment, beryllium can reach concentrations of 6 mg/kg (Kabata-Pendias 2011). Zhu and Olsen (2009) measured ^{7}Be sediment concentrations in the Neponset River estuary, Massachusetts ranging from 48 to 546 mBq/cm^2.

A summary of beryllium concentrations in sediments is provided in Appendix 1. An average concentration of beryllium in Canadian sediments was not determined for the purposes of setting human health based soil quality guidelines.

2.5.9. Biota Uses as Human Food

The British Columbia Ministry of Environment determined the metal concentrations in fish tissue from 54 uncontaminated lakes in the province (Rieberger 1992). For liver tissues samples rainbow trout had the highest mean concentration of beryllium (0.31 µg/L, n=110), cutthroat trout samples contained 0.28 µg/L (n=75), lake trout had a mean concentration of 0.28 µg/L (n=19); Dolly Varden had a mean concentration of 0.26 µg/L (n=49), Mountain whitefish had a mean concentration of 0.25 µg/L (n=24); and arctic grayling had the lowest mean concentration of beryllium of 0.22 µg/L (n=13) (Rieberger 1992).

As part of a contaminant monitoring program in the Northwest Territories, the livers and kidneys of 20 barren ground caribou (Beverly herd) were analysed for metals, beryllium concentrations were generally below analytical detection limits (not reported) (Elkin 2001).

A summary of available concentrations in biota used as human food is provided in Appendix 1. Beryllium concentrations in biota used as food are included in the dataset used to generate the estimated daily intake of beryllium resulting from food ingestion as discussed in the next section.

2.5.10. Commercial Foods

Health Canada's Food Directorate has provided estimated daily intakes of beryllium from food for Canadians from the Health Canada Total Diet Study (HC TDS) (Dabeka *et al*. 2010). Intake rates were provided for various age groups from 2000 to 2007 (inclusive) as presented in Table 2. These data were considered to be the most appropriate data to represent the Canadian population since they were developed recently by Health Canada in a methodical manner and have therefore been used to develop the beryllium EDI values for exposure to food used in the SQG calculation. The EDI methodology is further discussed in Section 5.2 of this document (HC 2011). The details regarding beryllium intake through food ingestion are presented in Table 2 below and Appendix 2.

Table 2. Beryllium intake via food ingestion by age group 2000-2007

Beryllium concentration (µg/kg-d)	0-6 months	7mo-4 yrs	5-11 yrs	12-19 yrs	20+ yrs
	M & F	M & F	M & F	M & F	M & F
Arithmetic Mean	0.059	0.042	0.028	0.018	0.016
Standard Deviation	0.077	0.052	0.035	0.021	0.019

A summary of beryllium intake via food ingestion from additional sources, including international data, is included in Appendix 1.

2.5.11. Human Breast Milk

In estimating the average concentration of beryllium in human breast milk, studies from other developed countries were considered because Canadian and U.S. studies were not available. Based on Austrian studies (Krachler *et al*. 1998; 1999) and an Emirati study (Abdulrazzaq *et al.* 2008), the mean beryllium concentration in human breast milk was determined to be 0.13 µg/L (arithmetic mean, SD=0.30, n=287) (HC 2011). This concentration includes human breast milk at various stages/maturity. None of the data were reported as less than the detection limit. These data were used to develop the beryllium EDI values for exposure to breast milk (applicable to breastfed infants only). The EDI methodology is further discussed in Section 5.2 of this document.

2.5.12. Consumer Products

The rising cost of gold and silver contributed to the introduction of beryllium alloys into the dental prosthetics industry. Beryllium was added to improve hardness, strength, corrosion resistance and castability of crowns, bridges and dentures. Beryllium is also used to reduce the fusion temperature in Ni-Cr alloys in the construction of partial denture frameworks and in ceramic-metal dental restorations. The alloys employed typically contain 0.5 to 2% beryllium (Kotloff *et al.* 1993). The potential risk of beryllium use in the dental industry has been recognised for some time. Dental laboratory technicians are at risk of exposure to unsafe levels of beryllium while working on dental alloys if proper precautions are not taken (OSHA 2002). It has been demonstrated that dental technicians exposed to beryllium are at high risk of developing chronic beryllium disease and other lung diseases (Lockey *et al.* 1983; Lu *et al.* 2009).

Studies have shown that nickel and beryllium can react synergistically, an effect which potentiates the dissolution of both metals in human saliva (Covington *et al.* 1985). Although the extent of ingestion and transport into the body has not been elucidated, it has been suggested that such ingestion may have a cumulative effect in terms of chronic exposure (Covington *et al.* 1985).

Release of beryllium has been reported during the ignition of some camping lantern mantles (Fishbein 1981). The mantle of a gas lantern contains about 600 µg beryllium metal added as a hardener (400 µg is volatilised within the first minutes of use) (Griggs 1973).

Tobacco smoke may contain significant amounts of beryllium (ATSDR 2002a). In three brands of cigarettes, beryllium concentrations were 0.47, 0.68 and 0.74 µg per cigarette, with 4.5, 1.6 and 10.0% of the beryllium content escaping into the smoke during smoking (Petzow and Zorn 1974). An average of 35 ng beryllium can be inhaled per cigarette (US EPA 1987b). An analysis of filterless cigarettes showed that value increases to 120 ng beryllium per cigarette (Stiefel *et al*. 1980).

Although many electronics contain beryllium alloys, exposure to beryllium via this route is highly unlikely as the beryllium-containing alloys are restricted to the inner structure of electronic devices or the metal or alloy is enclosed in a protective case (ATSDR 2002b).

An average concentration of beryllium in consumer products was not determined for the purposes of setting human health based soil quality guidelines.

2.5.13. Snow

Snow samples were collected from hand-dug pits at two sites in Simcoe County, Ontario by Shotyk *et al.* (2010). Two snow pits were dug at two sites on the Old Johnson Farm and at one site on the Parnell Farm. Beryllium mean concentration were 1.4 ± 0.5 ng/L (n=3); 2.9 ± 3.5 ng/L (n=3) and 2.3 ± 0.7 ng/L (n=5) respectively (Shotyk *et al.* 2010).

A summary of available environmental concentrations in snow is provided in Appendix 1. An average concentration of beryllium in Canadian snow was not determined for the purposes of setting human health based soil quality guidelines.

2.6. Existing Criteria, Standards and Guidelines

Existing Canadian guidelines, standards and criteria for beryllium in soil and groundwater are presented in Table 3.

The U.S. Department of Labour's Occupational Safety and Health Administration (OSHA) has set an occupational permissible exposure limit (PEL) of 2 µg/m^3 beryllium (8-hour time-weighted average) (OSHA 2006).

Table 3. Existing criteria and guidelines for beryllium in Canadian jurisdictions

Jurisdiction	Category	Criterion/Guideline	Reference
Ontario	Full Depth Generic Site Conditions Standards. Agricultural/Other Residential/Parkland/Institutional Industrial/Commercial/Community Stratified Site Condition Standards in potable and non-potable groundwater situations Surface Soil Residential/Parkland/Institutional Industrial/Commercial/Community Subsurface soil (applies to both coarse and medium/fine textured soils). Residential/Parkland/Institutional Industrial/Commercial/Community Potable Groundwater Criteria (all land uses) Non-potable Groundwater Criteria (all land uses)	 (5) 4 mg/kg[1,2] (5) 4 mg/kg[2] (10) 8 mg/kg[2] (5) 4 mg/kg[2] (10) 8 mg/kg[2] 60 mg/kg 60 mg/kg 4 µg/L 67 µg/L	Soil, Ground Water and Sediment Standards for Use Under Part XV.1 of the *Environmental Protection Act*, OMOE, 2009.
Yukon	Generic Numerical Soil Standards (Schedule 1) Agricultural Parkland/Residential Commercial/Industrial Generic Numerical Water Standards FAQL MAQL DW LW	 4 mg/kg 4 mg/kg 8 mg/kg 53 µg/L 1000 µg/L 100 µg/L 100 µg/L	Environment Act, Yukon Government, Department of Environment, Schedule 1 (2002) Environment Act, Yukon Government, Department of Environment, Schedule 3 (2002)
Alberta	Tier 1 Soil Remediation Guidelines (Fine and Coarse Soil) Agricultural Residential/Parkland Commercial Industrial	 5 mg/kg 5 mg/kg 8 mg/kg 8 mg/kg	Alberta Environment Tier 1 Soil Remediation Guidelines (2010)
British Columbia	Generic Numerical Soil Standards Agricultural Residential/Parkland Commercial Industrial Generic Numerical Water Standards FAQL – under review MAQL (minimal risk) – under review MAQL (hazard) – under review DW LW (tentative maximum) IR (maximum continuous use, all soils) – under review IR (maximum up to 20 yrs on fine-textured, neutral to alkaline soils) – under review	 5 mg/kg 5 mg/kg 8 mg/kg 8 mg/kg 5.3 µg/L 100 µg/L 1500 µg/L 4 µg/L 100 µg/L 100 µg/L 500 µg/L	BCMWLAP 2010, Schedule 4 2006 Compendium of Working Water Quality Guidelines for British Columbia and BCMWLAP 2010, Schedule 6
Canada (federal, provinces and territories not reported above, and CCME)	Soil Quality Guidelines[3] Agricultural Residential/Parkland Industrial/Commercial Canadian Water Quality Guidelines for the Protection of Agricultural Water Resources Irrigation Water Livestock Water	 4 mg/kg 4 mg/kg 8 mg/kg 100 µg/L 100 µg/L	CCME 1999 CCME 1999

[1] Guideline value is not provided for agricultural land use under non-potable groundwater scenario.
[2] Bracketed value applies to medium/fine textured soil, unbracketed value applies to coarse soil.
[3] Adopted by Northwest Territories, Alberta, Saskatchewan, Manitoba, New Brunswick, Nova Scotia, Prince Edward Island, Newfoundland and Labrador.
FAQL: freshwater aquatic life, MAQL: marine (and/or estuarine) aquatic life
DW: groundwater used as drinking water
SW: groundwater seeping into surface water or infiltrating sewers
IR: Irrigation Water
LW: livestock water use
NG: No guideline value

3. ENVIRONMENTAL FATE AND BEHAVIOUR

3.1. Atmosphere

The majority of beryllium released to the atmosphere is through thermal processes and thus beryllium appears to be present predominantly as BeO (ATSDR 2002a; Fishbein 1981; WHO 1990). It is not known whether BeO reacts with sulphur or nitrogen oxides to produce beryllium sulphate and/or nitrate. The conversion of beryllium to these water-soluble compounds would enhance wet deposition and removal from the atmosphere (WHO 2001). Beryllium appears to be predominantly associated with the organic fraction, which suggests it would be discharged in small particles probably as high fired refractory oxides (Skilleter 1987). Stack emission studies showed that beryllium is bound to the small particle fraction of emissions (Gladney and Owens 1976). Specifically, beryllium binds to particles smaller than 1 µm with a deposition time of 10 days (US EPA 1987b). Particulate loading studies have shown beryllium deposited on particulate matter greater than 0.3 µm throughout the United States (Chambers *et al.* 1955).

Other known isotopes, namely ^{6}Be, ^{7}Be ^{8}Be and ^{10}Be, are produced by nuclear reactions initiated by cosmic rays in the upper atmosphere or by accelerator mass spectrometers (Kabata-Pendias and Pendias 2000). Radioactive half-lives of 53.3 days (^{7}Be) and 1.51×10^{6} years (^{10}Be) have been reported (ATSDR 2002a; Yoshimori 2005). Once deposited, ^{7}Be rapidly adsorbs to above ground vegetation and soils in terrestrial systems and onto suspended particles in river-estuarine systems (Zhu *et al*. 2009).

From the atmosphere, beryllium is wet and dry deposited to terrestrial and aquatic surfaces (US EPA 1987b). The deposition of beryllium has not been measured according to the US EPA (1987b); however, Meehan and Smythe (1967) found beryllium concentrations between 0.05 and 0.08 µg/L in Australian rainwater. Beryllium was detected but not quantified in rainwater from Fresno, California, which suggests that transport of beryllium from the atmosphere to terrestrial and aquatic surfaces occurs by wet deposition (Salo *et al.* 1986). Based on particle size, wind speed and surface roughness, Kwapulinski and Pastuszka (1983) calculated that aerosol deposition probably occurs in a similar manner to other metals and on particles of comparable size (US EPA 1987b).

Beryllium-7 deposition

Beryllium-7 (^{7}Be) is a naturally occurring radionuclide of cosmogenic origin. Beryllium-7 has a relatively short-lived half-life of 53.6 days (478 keV gamma-ray photons) (Papastefanou 2009; Pöschl *et al.* 2010; UNSCEAR 2008). In estimating lifetimes of ^{7}Be-aerosols in ambient air, a mean residence time of about 8 days averaged for atmospheric aerosols of 0.90 µm AMAD (activity median aerodynamic diameter) was determined by Papastefanou (2009). The residence time of ^{7}Be in the stratosphere is about 1 year and in the troposphere is about 10 days (Yoshimori 2005). Once ^{7}Be is formed in the troposphere it rapidly associates, primarily with submicrometer aerosol particles. In these fine aerosols, ^{7}Be may subsequently enter the aquatic and terrestrial environments via dry and wet deposition processes and is easily found in soil and plants (Papastefanou 2009; Pöschl *et al*. 2010). The environmental concentration of ^{7}Be in the temperate zone is about 3 mBq/m^3 in ambient air and 700 Bq/m^3 in rainwater (UNSCEAR 2008).

Zhu and Olsen (2009) measured the monthly ^{7}Be atmospheric fluxes in Boston, Massachusetts

between September 2000 and August 2007. Fluxes exhibited seasonality and correlated with precipitation and solar activity.

3.2. Water

With most of the atmospheric beryllium coming from combustion, beryllium is most likely deposited in surface waters as the insoluble BeO and would remain in this form in waters in the normal environmental pH range (Callahan *et al.* 1979; US EPA 1987b). However, beryllium can be deposited as a soluble salt, which can be hydrolysed to form beryllium hydroxide, which possesses limited solubility in the pH range of natural waters (i.e., aquifers, lakes and rivers) or would be complexed with hydroxide ions (Callahan *et al.* 1979; WHO 1990). However, this latter reaction generally occurs at higher pH values than those normally found in natural waters and even when high concentrations of complexing agents are found (i.e., polluted rivers) very little dissolved beryllium is present in water (Callahan *et al.* 1979).

In both lakes and rivers, beryllium is predominantly present in the sorbed state, on suspended particles or in sediment (ATSDR 2002a; Callahan *et al.* 1979; Popp 1980; Rossmann and Barres 1988; WHO 2001). Veselý *et al*. (1989) observed that in alkaline surface waters (pH >6), beryllium was bound to suspended and colloidal particles with high iron content. Greater than 95% beryllium is dissolved in acidic surface water (pH <6) (Veselý *et al*. 1989).

Mobilisation of beryllium from the surface horizons of lake sediments by acidification has been suggested (Veselý *et al.* 1989). Veselý *et al*. (1989) have shown that beryllium concentrations in water are pH dependent and that the concentration of beryllium increased exponentially with decreasing pH. At pH 7.4 to 8.0, beryllium concentrations were low and often below the detection limit (20 ng/L). At the pH 4.0 range, beryllium concentrations were 100 times higher. Furthermore, it was found that in acidic surface waters, beryllium is present primarily as Be^{2+} and low-molecular beryllium fulvates. However, the range of beryllium concentrations in acid waters with similar pH values was large, which indicates factors other than just pH affect the beryllium concentration. *In situ* dialysis and membrane filtration studies suggest that mobilisation in acid waters results from the formation of the BeF^{+} fluoro complex and fulvic acid complexes (Veselý *et al.* 1989). Griffitts *et al.* (1973) also indicated that beryllium found in solution is likely complexed with fluoride, organic complexes and chlorocarbonate.

The content in ocean water is about 0.0002 ppb in the form of $Be(OH)^{+}$ and $Be(OH)_2$, with an estimated residence time of a few hundred years before it is removed by sedimentation or other removal processes (Bowen 1979; Griffitts and Skilleter 1990; Merrill *et al.* 1960).

Due to the physical and chemical properties of beryllium and fate factors, there is a low probability of having toxic levels of beryllium in natural waters (i.e., aquifers) (Irwin *et al*. 1997).

3.3. Sediment

In sediment, beryllium is usually associated with the clay fraction and is adsorbed to clay minerals, hydroxides and organic matter (Dunn 1980; WHO 1990). Lum and Gammon (1985) showed a positive correlation between beryllium and aluminium in studies of the Detroit River and western Lake Erie sediment. They also reported that HCl-extractable beryllium (the potentially bioavailable portion) formed only a small part of the total beryllium concentrations in the top 3 to 5 cm of sediments. Loring (1979) studied Bay of Fundy sediment and reported that the beryllium

concentration increased with decreasing grain size.

3.4. Soil

Beryllium occurs in soils primarily in the oxide forms $(BeO_2)^{2-}$, $(Be_2O_3)^{2-}$, $(BeO_4)^{6-}$ or $(Be_2O)^{2+}$ and in calcareous soils as complex anions of $Be(OH)CO_3^-$ and $Be(CO_3)_2^{2-}$ (Kabata-Pendias and Pendias 2000; Kabata-Pendias 2011). The chemistry of beryllium in soils is believed to be similar to that of aluminium (Fishbein 1981). At low pHs, beryllium will most likely be tightly adsorbed onto clay surfaces, displacing divalent cations that share common sorption sites (Fishbein 1981). At high pH values, beryllium may remain precipitated in insoluble complexes or form soluble polynuclear hydroxide complexes (Callahan *et al.* 1979).

Beryllium readily complexes with organic compounds such as humic substances and accumulation in organic soil horizons has been reported (ATSDR 2002a; Kabata-Pendias and Pendias 2000; Newland 1982). Beryllium will covalently bond with these organics resulting in organo-beryllium compounds, including dimethylberyllium ($(CH_3)_2Be$) and many other complexes (WHO 2001, Kabata-Pendias and Pendias 2000). There are many stable chelate compounds of beryllium such as beryllium oxalate and stable organic salts (Lee 1965; Venugopal and Luckey 1978). Possible reactions of beryllium in soil are hydrolysis of soluble salts, anion exchange reaction by which one salt is converted to another, and reactions with ligands present in the soil (such as humic substances). Beryllium mobility is reduced in the presence of dissolved aluminium and organic carbon (Veselý et al. 2002) and is more strongly bonded to clay minerals than organic matter (Kabata-Pendias and Mukherjee 2007). Beryllium salts (e.g., $BeCl_2$ and $BeSO_4$) are readily soluble and may, therefore, be potentially toxic to plants (Kabata-Pendias 2011). Reactions of beryllium in soil are likely to be responsive to pH (Irwin 1997). In acidic forest soils (pH 3.4) beryllium is highly mobile. Furthermore, distributions in some soil profiles show an accumulation of beryllium in subsoil layers, indicating possible leaching (Anderson *et al*. 1990; Asami and Fukazawa 1985; Kabata-Pendias 2011).

3.5. Indoor Dust

Rasmussen *et al.* (2008) determined that indoor dust and soil are geochemically distinct. Indoor dust has approximately five times more organic matter than soil (Rasmussen *et al.* 2008). Organic carbon is a key factor controlling metal partitioning and bioavailability. The higher metal concentrations in indoor dust compared to soils may be explained by the affinity some metals have for organic matter, in addition to the smaller particle size of dust. Consequently, the use of outdoor soil metals data to predict indoor dust concentrations can result in the underestimation of indoor dust exposures (Rasmussen 2004). In areas where spatial correlations do exist between indoor dust and exterior soil concentrations, they suggest an elevated external source (i.e., mining or other sources of industrial contamination). In these cases, house dust may contain lower metal concentrations than garden soil.

In studies of house dust, street dust and residential garden soils in Ottawa, Ontario, Rasmussen *et al.* (2001; 2008) showed that house dust can contribute significantly to metals exposure in residential urban environments. Rasmussen *et al.* (2001) determined the arithmetic mean concentration of beryllium in house dust to be 0.56 mg/kg (n=48). Dust samples were analysed by ICP-MS following a nitric acid/hydrofluoric acid digestion.

3.6. Biota

The ATSDR (2002a) indicates that the bioconcentration of beryllium in plants is very low and the vast majority of beryllium taken up by plants is retained in the roots (Kabata-Pendias 2011). Soluble forms of beryllium are required for plant uptake to occur. There is no evidence of biomagnification of beryllium within the food chain of humans (Fishbein 1981). Therefore, for the purpose of calculating the Soil Quality Guideline for the Protection of Human Health, beryllium was considered to not bioaccumulate.

4. BEHAVIOUR AND EFFECTS IN HUMANS AND NON-HUMAN MAMMALIAN SPECIES

4.1. Overview

The behaviour and effects of beryllium in humans and mammalian species have been reviewed by several international health agencies including the Agency for Toxic Substances and Disease Registry (ATSDR 2002a), World Health Organization (WHO 2001), and US Environmental Protection Agency (US EPA 1998). It is not the role or the intention of this document to comprehensively re-evaluate the toxicology of beryllium. This report focuses on the studies most relevant to the development of toxicological reference values (TRVs) for beryllium, for use in development of soil quality guidelines.

Beryllium easily forms covalent compounds (ATSDR 2002a; Griffitts and Skilleter 1990). Beryllium toxicity might be due to its capacity for forming strong covalent bonds, thus displacing other divalent cations. In support of this hypothesis, the example of *in vitro* inhibition of the magnesium-activated alkaline phosphatase by very low (10^{-6} mol/L) concentrations of beryllium is cited (Vouk 1986). Also, it is possible that soluble beryllium binds to other cells (such as lymphocytes) or is taken up in small amounts through fluid pinocytosis mechanisms, thus broadening the toxic influences of the metal (Skilleter 1987). Be^{2+} binding is highly specific and it does not bind indiscriminately with biological macromolecules (Venugopal and Luckey 1978).

Both cancer and non-cancer endpoints are significant in the toxicological evaluation of beryllium. IARC (1993) has classified beryllium and beryllium compounds as “carcinogenic to humans” (Group 1) based on evidence of carcinogenicity following inhalation exposure in humans and animals. The US EPA (1998) has classified beryllium as a probable human carcinogen (B1) following inhalation exposure, based on sufficient evidence in animals and limited evidence in humans.

There is inadequate information available to assess the carcinogenic potential of beryllium by the oral route. Substances deemed unclassifiable with respect to carcinogenicity in humans are generally assessed by Health Canada as threshold toxicants (HC 1994). For oral exposure, beryllium should therefore be considered a threshold toxicant.

4.2. Toxicokinetics

The solubility of a beryllium compound influences its toxicokinetics; more soluble compounds undergo greater systemic absorption, distribution and urinary elimination (NAP 2007).

4.2.1. Absorption

Beryllium and beryllium compounds are poorly absorbed after oral exposure. Most of the data indicate that ingested beryllium compounds pass through the gastrointestinal tract unabsorbed and are eliminated in the faeces. Soluble beryllium compounds, such as beryllium sulphate, may also precipitate as insoluble beryllium phosphate in the higher pH environment of the intestine (Reeves 1965). For consideration in human health risk assessments, a default gastrointestinal absorption factor of 1% has been recommended based on a study by Furchner *et al.* (1973) in which beryllium chloride was administered orally and parenterally to mice, rats, monkeys and beagles.

The level of absorption from the gut depends on the intrinsic nature of the compounds administered. Beryllium sulphate is better absorbed by the gastrointestinal tract than beryllium oxide or beryllium metal (Watanabe *et al*. 1985). In rats, beryllium oxide was more readily absorbed than beryllium hydroxide, and beryllium fluoride was more readily absorbed than beryllium chloride, sulphate, nitrate and hydroxide (Bugryshev *et al.* 1984).

The deposition and clearance of inhaled beryllium particles are affected by dose, particle size and the solubility of particles (Kolanz 2001; Muller *et al.* 2010; Stefaniak *et al*. 2007). Small amounts of soluble beryllium compounds are absorbed into the bloodstream, while insoluble beryllium compounds can be retained in the lung for long periods of time (Léonard and Lauwerys 1987).

Calcining temperature has been shown to affect beryllium absorption. Beryllium oxide particles calcinated at 1000°C were shown to be retained in the lungs of dogs for a longer period of time than beryllium oxide calcinated at 500°C (Finch *et al*. 1990). This is because the solubility of beryllium oxide decreases as the temperature at which it is calcinated increases (ATSDR 2002a). Beryllium oxide calcinated at 500°C is absorbed and translocated more rapidly to extrapulmonary tissues, and excreted in greater amounts than beryllium oxide calcinated at 1000°C (Finch *et al*. 1990).

There are few studies that demonstrate dermal absorption of beryllium. Petzow and Zorn (1974) demonstrated that small amounts of beryllium were absorbed through the tail skin of rats after exposure to an aqueous solution of beryllium chloride. In an *in vitro* study using guinea pig epidermis, Belman (1969) demonstrated that beryllium can bind to alkaline phosphatase and nucleic acids in the skin. This binding is thought to result in inefficient transfer of beryllium to the blood and thus, poor absorption via the dermal route.

Particle size may be a key factor for dermal absorption. A recent study by Tinkle *et al*. (2003) demonstrated that relatively insoluble particles <1 µm in diameter may be transported through the skin. Emond *et al*. (2007) calculated dermal and inhalation exposure at a recycling industry processing spent pot linings from aluminium smelters. In this study, dermal daily dose, on the head and hands, was negligible compared to the inhalation daily dose. In this particular situation, the fraction of particles ≤1 µm in diameter represented only 5 percent of the total beryllium. The

authors of the study caution that this exposure scenario is highly specific to this industry and emphasise the importance of particle size and chemical form in identifying safe exposure levels.

4.2.2. Distribution

Once beryllium is absorbed into the systemic circulation it is distributed primarily to the skeleton (Finch *et al*. 1990; Morgareidge *et al.* 1977; Reeves 1965). It is also distributed to the liver, tracheobronchial lymph nodes and other soft tissues (ATSDR 2002a; Muller *et al*. 2010; NAP 2007).

Administration of beryllium sulphate in the diet of rats produced a dose-dependent accumulation of beryllium in the skeleton (Morgareidge *et al.* 1977). Reeves (1965) also demonstrated accumulation of beryllium in the skeleton of rats following administration of a daily dose of beryllium sulphate, via drinking water, for 24 weeks. In rabbits, Shima *et al*. (1983) observed that orally administered beryllium sulphate and beryllium oxide (firing temperature unknown) at doses of 200 mg every other day for 2 months were largely retained and distributed in the liver and large intestine, respectively. Watanabe *et al*. (1985) administered beryllium sulphate, beryllium oxide, or beryllium metal to hamsters, in the diet, for a period of 3 to 12 months. Administration of beryllium sulphate resulted in beryllium predominately in the liver, large and small intestine, kidney, lung, stomach and spleen. Administration of beryllium metal and beryllium oxide resulted in distribution of beryllium mainly in the large and small intestines.

Distribution of beryllium has also shown to be affected by the calcination temperature, with more beryllium being translocated to the skeleton and liver at 180 days after exposure to beryllium oxide prepared at 500°C than at 1000°C (Finch *et al.* 1990).

Meehan and Smythe (1967) measured beryllium concentrations in various human organs and found the highest levels in the lungs, with lower levels in the brain, kidneys and spleen. Beryllium was also detected in liver, muscle, vertebrae, heart, bone and hair. In occupationally exposed individuals, the highest beryllium concentrations were found in lungs and bone with lower concentrations in liver and kidneys (Tepper *et al.* 1961 in ATSDR 1988).

Krachler *et al.* (1999) studied the transfer of essential and trace elements including beryllium across the placenta and into breast milk of humans. Beryllium concentrations in umbilical cord serum and colostrum were higher than in maternal serum. Bencko *et al.* (1979) demonstrated the ability of beryllium to cross the placenta following intravenous administration of a single dose of beryllium chloride to mice. Beryllium chloride was administered at different times during pregnancy (i.e., prior to copulation, on day 7 of gestation, or on day 14 of gestation) and, while some beryllium circulated in the bloodstream long enough to penetrate the foetuses, placental permeability was slight.

4.2.3. Metabolism

Beryllium and beryllium compounds are not metabolised in the body, but soluble beryllium salts, such as beryllium sulphate, may be partially converted to more insoluble forms in the lung

(Reeves and Vorwald 1967). Soluble beryllium compounds may also be converted to insoluble beryllium phosphate in the gastrointestinal tract (Reeves 1965).

4.2.4. Elimination

Most beryllium administered orally or entering the gastrointestinal tract *via* mucociliary clearance is not absorbed but is excreted in the faeces (Finch *et al.* 1990; Hart *et al.* 1980). Any beryllium that is absorbed typically is excreted via the urine (Furchner *et al.* 1973).

In rats administered daily doses of beryllium sulphate in drinking water for 24 weeks, 60% to 90% of the total administered dose appeared in the faeces while less than 1% was excreted in the urine (Reeves 1965). Furchner *et al*. (1973) studied excretion of orally administered radioactive beryllium chloride in four mammalian species (rats, monkeys, mice and dogs), and determined that more than 98% of the dose was excreted in the faeces. Shima *et al.* (1983) reported that oral doses of beryllium sulphate and beryllium oxide were mainly excreted in the faeces of rabbits.

Inhaled soluble compounds tend to be cleared more rapidly than insoluble compounds (i.e., solution versus particulate) because dissolved substances would more readily be diffused through bronchoalveolar tissues to lymph channels or the blood stream (Reeves and Vorwald 1967). Clearance of insoluble beryllium compounds from the lung was reported to be biphasic (Rhoads and Sanders 1985). The initial phase is rapid and involves mucociliary transport of particles deposited in the upper respiratory tract to the gastrointestinal tract (Finch *et al.* 1990). The slower, second phase involves clearance by translocation to tracheobronchial lymph nodes, uptake by alveolar macrophages and dissolution of beryllium (Camner *et al.* 1977; Delic 1992; Finch *et al.* 1990; HSE 1994; Sanders *et al.* 1978). Typically, the rapid phase has a half-time of days (up to 60 days in rats), whereas the half-time of the slower phase is in the order of years (up to 2.3 years in rats) depending on the solubility of the beryllium compound (NCRP 1997; Reeves *et al*. 1967; Reeves and Vorwald 1967; Rhoads and Sanders 1985; Schlesinger 1995; Zorn *et al*. 1977). Insoluble beryllium compounds have been found in the lungs of occupationally-exposed workers many years after cessation of exposure (Schepers 1962).

One hundred eighty (180) days after exposure, dogs inhaling aerosol beryllium oxide calcinated at 500°C excreted significantly more beryllium than dogs inhaling aerosol beryllium oxide calcinated at 1000°C (Finch *et al*. 1990). Beryllium oxide calcinated at 500°C is more soluble than beryllium oxide calcinated at 1000°C and cleared the lungs more rapidly, yet was excreted only slightly more via the urine than beryllium oxide calcinated at 1000°C. The authors postulated that this might be indicative of beryllium's tendency to bind preferentially to extrapulmonary tissues (e.g., blood, liver, skeleton) following solubilisation in the lung. In this study, beryllium excretion was predominantly via the faeces (45-68% versus 33-55% of total excretion) until 59 (beryllium oxide calcinated at 500°C) and 130 (beryllium oxide calcinated at 1000°C) days following exposure, after which (180 days after exposure) urinary excretion was similar to faecal excretion (46-53% versus 47-54% of total excretion).

Urinary excretion of beryllium in humans appears to be inconsistent (e.g., levels did not correlate with exposure and reaction severity) and not useful for biological monitoring (DeNardi *et al.* 1953; Klemperer *et al.* 1951; Reeves 1986; Stoeckle *et al.* 1969). In an inhalation exposure study by Muller *et al*. (2010), exposure of mice to fine particles (250 $\mu g/m^3$) of one of three different

beryllium species (beryllium metal, beryllium oxide and beryllium aluminium) for 3 weeks resulted in urine concentrations differing between beryllium species.

4.2.5. Human Tissues and Body Fluids

The body burden estimate of beryllium for a 70 kg person is 36 µg or approximately 0.5 µg/kg body weight (Bowen 1979). A similar estimate of 30 µg of beryllium for total bodily content was previously made by Schroeder (1971). According to Venugopal and Luckey (1978), 75% of the beryllium found in the human body is found in the bone.

Data regarding beryllium concentrations in human tissues and fluids are summarised in Appendix 1.

4.3. Acute Toxicity

4.3.1. Oral Exposure

No human data were identified regarding the oral toxicity of beryllium or beryllium compounds. Oral LD_{50} (lethal dose, 50% mortality rate) values are dependent on the solubility of the compound. Reported oral LD_{50} values for soluble beryllium compounds include beryllium sulphate (120 mg/kg in rats and 140 mg/kg in mice), and beryllium chloride (200 mg/kg in rats) (Ashby *et al*. 1990; Kimmerle 1966; Lanchow University 1978). Other reports of oral LD_{50} values include 18-20 mg/kg for beryllium fluoride in mice and 18.3 mg/kg for beryllium oxyfluoride in rats (Kimmerle 1966; Lanchow University 1978). The lower LD_{50} value observed for beryllium fluoride and beryllium oxyfluoride has been attributed to the additional toxicity of the fluoride ion. In a recent study of beryllium metal powder in rats, a gavage dose of 2000 mg/kg bw in polyethylene glycol 300 did not result any signs of toxicity (Strupp 2011). This is thought to be due to the low solubility of beryllium metal.

4.3.2. Inhalation Exposure

The NAP (2007), ATSDR (2002) and US EPA (1998) have comprehensively reviewed effects following beryllium inhalation exposure in humans (occupational exposures) and animals (laboratory studies). There is extensive evidence that the lung is the primary target following inhalation exposure to beryllium.

Short-term exposure to high levels of soluble and slightly soluble beryllium compounds can result in acute beryllium disease (ABD) (ATSDR 2002a; WHO 2001). ABD is a type of pneumonitis generally thought to be due to the direct toxicity of beryllium, although immune and hypersensitivity responses have also been suggested (Cummings *et al*. 2009). Onset of respiratory symptoms (ranging from mild nasopharyngitis to tracheobronchitis and severe chemical pneumonitis) usually occurs over several weeks (ATSDR 2002a). ABD is often reversible upon removal from exposure and initiation of supportive respiratory care (ATSDR 2002a; NAP 2007). Recovery times for survivors are typically less than a year (Wambach and Laul 2008). ABD is generally associated with exposure to concentrations of beryllium above 1000 µg/m^3 (Eisenbud *et al*. 1948), and has rarely been reported since the implementation of exposure limits in the 1950s and subsequent decreases in respiratory exposure to beryllium

(ATSDR 2002a; NAP 2007). Chemical pneumonitis induced by exposures to beryllium of less than 1000 µg/m^3 has been reported to be reversible (Delic 1992; HSE 1994).

4.3.3. Dermal Exposure

Dermal exposure to beryllium compounds can cause irritant contact dermatitis, allergic contact dermatitis, chemical ulcers, ulcerating granulomas and allergic dermal granulomas (Berlin *et al.* 2003). Dermal exposure appears to play a role in beryllium sensitisation and chronic beryllium disease (CBD) and may be a significant route of exposure to beryllium (Cummings *et al.* 2007; Curtis 1951; Day *et al.* 2007; Henneberger *et al*. 2001; Kreiss *et al.* 2007).

Skin granulomas have been observed after a few weeks to several months in boars following subcutaneous implantation of approximately 5 mg beryllium oxide, phosphor containing beryllium oxide, or beryllium metal (Dutra 1951). Granulomas were found mostly in the subcutaneous fat but also in the dermis. Samples of powdered beryllium oxide and metallic beryllium resulted in the production of foreign body giant cells and eventually non-specific granulomatous inflammation. Beryllium oxide calcinated at a lower temperature (1200˚C for three hours) demonstrated a more pronounced reaction, lasting longer than beryllium oxide calcinated at a higher temperature (1370˚C for five hours) or beryllium metal. Phosphor containing beryllium oxide produced granulomas of loosely arranged collagenous tissue, lymphocytes, phagocytes and occasional giant cells of Langhan's type.

Topical application of 100 µL 0.5M beryllium sulphate in dibutyl phthalate or beryllium oxide in petrolatum was sufficient to cause beryllium sensitisation in mice (Tinkle *et al*. 2003). This was demonstrated when animals were subsequently challenged by dermal application of 25 µL 0.5M beryllium sulphate 7 days after the initial exposure. Application of beryllium sulphate on the ear resulted in a 25-30% increase in ear thickness at 24 and 48 hours after the challenge and beryllium oxide resulted in a 30% increase in ear thickness at 24 and 48 hours.

Application of 0.5 g of beryllium metal, moistened by 0.5 mL of water to adhere to the skin, did not produce irritating or corrosive dermal effects in rabbits (Strupp 2011). There was no skin sensitising potential of beryllium metal in guinea pigs tested using the Magnussen and Kligman maximisation method (Strupp 2011).

4.4. Subchronic and Chronic Toxicity

4.4.1. Oral Exposure

The addition of 0.125-3% beryllium carbonate to the diet (calculated to be 13-300 mg/kg bw/day (US EPA 1986)) for 24-28 days, produced bone lesions characteristic of rickets in young rats (Guyatt *et al.* 1933). Similar results were seen by Kay and Skill (1934) who demonstrated severe rickets and decreased blood inorganic phosphorus, erythrocyte phosphorus, and liver and kidney phosphatase levels in rats fed 0.5% (50 mg/kg bw/day) (US EPA 1986) beryllium carbonate in the diet for 21-22 days. Kay and Skill (1934) suggested that rickets is primarily a "phosphate deficiency disease" rather than a direct toxic effect of beryllium. Phosphorus levels in the body decrease with the formation of insoluble beryllium phosphate in the gastrointestinal tract

resulting in decreased phosphorus absorption. Supplementing beryllium-exposed rats with glycerophosphate reduced the severity of rickets (Kay and Skill 1934).

Rats receiving doses of 20 mg of beryllium nitrate through the diet every third day for 2.5 months (40 doses) exhibited pathologic lesions in bronchioles, alveoli and arterioles (Goel *et al.* 1980). Morphologically, the lungs of exposed rats were harder and more opaque than in controls. Biochemical analysis of tissues showed increased activity of pulmonary alkaline phosphatase, acid phosphatase and 5'-ribonucleotide phosphohydrolase. As the respiratory system does not appear to be a target of oral exposure to beryllium compounds it is possible that the observed pulmonary effects may have been caused by aspiration of beryllium nitrate during feeding (ATSDR 2002a; US EPA 1998).

The gastrointestinal tract appears to be the most sensitive target for oral exposure to beryllium. In a chronic toxicity study by Morgareidge *et al.* (1976), male and female beagle dogs (5 per sex per dose group) were fed a diet containing 1, 5, 50 or 500 ppm beryllium as beryllium sulphate for up to 172 weeks. Using estimated time weighted average (TWA) body weights and the reported average food intake, these concentrations correspond to doses of 0.023, 0.12, 1.1 and 12.2 mg/kg bw/day for males and 0.029, 0.15, 1.3 and 17.4 mg/kg bw/day for females (US EPA 1998). Dogs exposed to 500 ppm had extensive ulcerative and inflammatory lesions in the small intestine (predominately), stomach and large intestine. Bone marrow hypoplasia was also observed in the high dose group. In the 50 ppm, two dogs exhibited similar but less severe gastrointestinal lesions.

Chronic oral exposure to beryllium sulphate did not result in gastrointestinal effects in rats (Morgareidge *et al*. 1977). Male and female Wistar albino rats were fed diets containing 5, 50 or 500 ppm beryllium sulphate tetrahydrate from four weeks through maturation, mating, gestation and lactation. Using estimated TWA body weights and the reported average food intake, these concentrations correspond to doses of 0.36, 3.6 and 37 mg/kg bw/day for males and 0.42, 4.2 and 43 mg/kg bw/day for females (US EPA 1998). No effects were observed on mortality, gross abnormalities, histology, or tumour incidence. Slight decreases (10%) in growth were observed toward the end of the study but were not statistically significant. Relative kidney weight was increased in male rats exposed to 50 ppm. Relative testes weights were decreased in male rats exposed to 5 and 50 ppm. Relative kidney and adrenal weights were decreased in female rats exposed to 500 ppm. Results were not provided for the F_0 generation.

In a study by Schroeder and Mitchener (1975a), rats of both sexes were exposed to 0 or 5 ppm beryllium sulphate (equivalent to 0.63 and 0.71 mg/kg bw/day for males and females respectively (US EPA 1998)) in drinking water from weaning until natural death. Body weights of male and female rats were significantly greater than controls at 30 days, but were significantly less than control animals at 60, 90, 120 and 180 days in male rats. No significant changes in body weight were noted at 150, 360 or 540 days. Beryllium exposure also resulted in altered urine and serum glucose and cholesterol although these changes were not considered large enough to suggest impaired organ function (US EPA 1998). No effects were observed on mortality or longevity and tumour incidence was not significantly different between treated and control groups. Histology results were not reported.

In a second study by Schroeder and Mitchener (1975b), mice of both sexes were exposed for their lifetime to 0 or 5 ppm of beryllium as beryllium sulphate in drinking water (equivalent to

1.2 mg/kg bw/day for male and female mice (US EPA 1998)). Body weight was significantly increased in male rats exposed to beryllium at 30 days and significantly decreased in female rats at 90 and 360 days. No effects were seen on mortality, longevity or tumour incidence.

4.4.2. Inhalation Exposure

While ABD is generally considered to be an irritant response to high exposure levels, chronic beryllium disease (CBD) is considered a hypersensitivity response to lower exposure levels. Symptoms of CBD include dyspnea, cough, fatigue, anorexia, weight loss and chest pain, although some cases are asymptomatic (Aronchik 1992; Hasan and Kazemi 1974; Kriebel *et al.* 1988b; Meyer 1994; NAP 2007; Sterner and Eisenbud 1951; Williams 1993). CBD is a pulmonary granulomatous disease characterised by a beryllium-specific immune response and the formation of noncaseating granulomas (NAP 2007; US EPA 1998). In the lung, granulomas interfere with lung function eventually leading to fibrosis, respiratory failure and cor pulmonale (Sood 2009; Wambach and Laul 2008). Interstitial granulomas occur primarily in the lungs but noncaseating granulomas have been reported in the skin, liver, spleen, lymph nodes, myocardium, skeletal muscles, kidney, bone and salivary glands (Freiman *et al*. 1970).

CBD is preceded by beryllium sensitisation, although not all individuals who are sensitised develop CBD (Newman *et al*. 2005). CBD is known to have a genetic susceptibility component. Studies have shown that an allele of the HLA-DP gene containing glutamic acid (HLA-DPβGlu69) is associated with an increased risk of developing beryllium sensitisation and CBD (Kreiss et al. 2007; NAP 2007; Tarantino-Hutchison *et al*. 2009). However, CBD is assumed to be a multigenetic disease and it is likely that other genes also play a role in its development (NAP 2007; Sawyer and Maier 2011).

Dose-response evaluations have been difficult for CBD because it is an immune disease and can have a long latency period (ATSDR 2002a; US EPA 1998). Progression from beryllium sensitisation to CBD can occur from several months to decades after the initial beryllium exposure (NAP 2007; Sawyer and Maier 2011). It has been suggested that both exposure and genetics may affect the development of CBD after first exposure (Mroz *et al*. 2009). Beryllium sensitisation is detected by the beryllium lymphocyte proliferation test (BeLPT), which has enabled the detection of subclinical beryllium disease and beryllium sensitisation in asymptomatic workers (ATSDR 2002a; Day *et al*. 2006; NAP 2007).

Despite reductions in respiratory exposure to beryllium new cases of CBD are still being identified (ATSDR 2002a). The increasing use of beryllium compounds in a wide range of applications has also resulted in new groups of occupationally-exposed workers including dental technicians and jewellers that work with beryllium alloys, workers at nuclear sites and in industries that recycle electronics, computers and scrap alloys (NAP 2007; Sawyer and Maier 2011). CBD has also been reported outside of the beryllium industry. Since the 1940s, cases of CBD have been recognised in individuals residing near beryllium facilities and in family members of beryllium workers who may have had contact with beryllium-contaminated clothing (ATSDR 2002a; Eisenbud *et al*. 1949; Kreiss *et al.* 2007; Maier *et al*. 2008; NAP 2007).

Epidemiological studies of CBD have been thoroughly reviewed by NAP (2007) and ATSDR (2002a). The most important studies for the development of the toxicological reference value for beryllium are discussed below and in Section 4.7.

A cross-sectional study by Kreiss *et al*. (1996) of 136 workers exposed to beryllium oxide at a beryllia ceramics plant reported eight cases of beryllium sensitisation. Five of these workers were also diagnosed with CBD based on consistently abnormal blood BeLPT results (performed by two different laboratories) and the observation of granulomas upon lung biopsy. Two workers had abnormal blood BeLPT results from one of the two laboratories but had no evidence of granulomas upon initial investigation. One of these workers developed symptoms of CBD within two years. CBD was also diagnosed in a worker who initially had normal BeLPT results but subsequently developed a nonhealing granulomatous response to a beryllium-contaminated skin wound. Abnormal blood BeLPT results and the development of lung granulomas, after several months, confirmed the diagnosis. Of the eight beryllium sensitised workers, seven had worked as machinists. The rate of beryllium sensitisation among machinists was 14.3% compared to 1.2% for all other workers. Average beryllium exposures for the eight beryllium-sensitised workers ranged from 0.2 to 1.1 μg Be/m^3, with a median concentration of 0.55 μg Be/m^3 and cumulative exposure ranged from 92.6 to 1945 μg/m^3-days. The lowest-observed-adverse-effect level (LOAEL) for beryllium sensitisation and chronic beryllium disease in this study is considered to be 0.55 μg/m^3.

In a more recent study by Schuler *et al*. (2012), the lowest exposure at which CBD was observed for total mass concentration was 0.20, 0.38 and 0.22 for average (μg/m^3), cumulative (μg/m^3-year), and highest job worked (μg/m^3), respectively. The population examined was from a primary beryllium manufacturing plant and consisted of workers (n=264) with ≥6 years' work history.

Eisenbud *et al*. (1949) conducted an investigation of community-associated cases of CBD in 10 000 residents living within one mile of a beryllium production plant. CBD was diagnosed based on radiological and clinical findings. Ten cases were identified within 0.75 miles of the plant including three more cases that were detected in a follow-up study in 1951 (Sterner and Eisenbud 1951). Estimates of beryllium concentrations at 0.75 miles from the facility were estimated to be between 0.01 and 0.1 μg Be/m^3 which is considered the NOAEL for CBD.

Mammalian (Non-human Studies)

Numerous studies have investigated the effects of inhalation exposure to beryllium in laboratory animals. The respiratory tract is the primary target for inhalation exposure to beryllium in laboratory animals as well as humans. Inhalation exposure to beryllium has resulted in inflammation, emphysema, granuloma formation, fibrosis and the development of lung tumours in several laboratory animal species (ATSDR 2002a; US EPA 1998; WHO 2001). Effects in other systems (cardiovascular, haematological, hepatic and renal) have also been reported in a detailed review of beryllium toxicology completed by the ATSDR (2002a), but effects are generally only observed at high doses and are often secondary to severe lung disease (NAP 2007). While several laboratory animal species (rats, mice, guinea pigs, dogs, monkeys) have been used in inhalation exposure studies, no one animal model has been able to demonstrate all the features of CBD (development of immune granulomas, a beryllium specific-immune response and a disease progression that mimics human CBD) (NAP 2007; US EPA 1998). As

human health toxicological information is available for inhalation exposure to beryllium compounds and toxicity reference values for inhalation exposure are based on human studies, laboratory animal studies have not been summarised here.

4.5. Reproductive and Developmental Toxicity

No human data were located regarding the potential reproductive and/or developmental effects of beryllium following oral exposure.

The effect of beryllium on reproductive and/or developmental effects has not been adequately assessed. Reviews by the ATSDR (2002a) and US EPA (1998) include only one study on the reproductive and developmental toxicity of beryllium following oral exposure (Morgareidge *et al.* 1976) and no studies on the reproductive and developmental toxicity of inhaled beryllium. No multi-generation or male reproductive studies have been carried out. It has also been noted that many of the animal studies may have been conducted at doses that result in maternal toxicity (NAP 2007; US EPA 1998).

No adverse reproductive or developmental effects were reported in a chronic feeding study by Morgareidge *et al.* (1976). In this study, beryllium sulphate tetrahydrate was fed at dietary concentrations of 1, 5 or 50 ppm (equivalent to 0.23, 0.12 or 1.1 mg/kg bw/day for males and 0.29, 0.15 or 1.3 mg/kg bw/day for females) to dogs from before mating through weaning at 6 weeks of age (US EPA 1998).

In a case-control study using the National Natality and National Fetal Mortality Survey data, paternal occupational exposure to beryllium showed no effects on the risk of stillbirth, preterm delivery or small-for-gestational-age infants (Savitz *et al*. 1989).

Clary *et al*. (1975) studied the reproductive effects of a single intratracheal administration of 0.2 mg radiolabelled beryllium/kg as beryllium oxide calcinated at 500 or 960 ºC in male and female Sprague-Dawley rats. No reproductive effects were reported in beryllium-exposed rats.

A single intravenous beryllium dose of 0.316 mg/kg ($1/10^{th}$ of LD_{50}) as beryllium nitrate was injected into pregnant Sprague Dawley rats (n=5-8/group) on gestation day 1, 11, 12, 13, 15 or 17 (Mathur *et al.* 1987). Rats were laparotomised on gestation day 10 and again on gestation day 20 for examination of implantation sites (day 10) and number of foetuses (day 20). After the surgeries, the dams were allowed to deliver their pups. All the pups died within 2-3 days of delivery except for pups from the group injected on gestation day 11, which were resorbed. WHO (2001) suggested that the effects on the pups may have been related to the multiple surgeries.

In a Russian study (Selivanova and Savinova 1986) described by ATSDR (2002a), rats treated by intratracheal injection with 50 mg Be/kg body weight as beryllium chloride and beryllium oxide (calcination temperature not specified) on gestation days 3, 5, 8 or 20. Increased foetal mortality was reported for rats treated on day 5 with beryllium chloride and on days 3 and 5 with beryllium oxide. Decreased foetal weight was reported for animals treated on day 3 for either compound and an increase in the percentage of pups with internal abnormalities was reported for rats treated on days 3 and 5 for beryllium chloride and on days 3, 5 and 8 for beryllium oxide. No differences were observed in the number of live births per dam or in foetal length.

4.6. Carcinogenicity and Genotoxicity

No evidence of beryllium carcinogenicity via the oral route has been identified (ATSDR 2002a; US EPA 1998; WHO 2001). There are no epidemiological studies regarding the oral carcinogenicity of beryllium in humans. Chronic oral exposure studies in dogs, rats and mice did not find significant increases in tumour incidences (Morgareidge *et al*. 1975; 1976; 1977; Schroeder and Mitchener 1975a; b; US EPA 1998).

A large number of epidemiological studies have evaluated the carcinogenicity of inhaled beryllium. These studies have been thoroughly reviewed by US EPA (2008 draft; 1998) and ATSDR (2002a). There is general agreement among organisations that beryllium should be classified as a probable or known human carcinogen based on the results of human epidemiological studies and laboratory animal carcinogenicity studies (ACGIH 2001; ATSDR 2002a; IARC 1993; NTP 2005; US EPA 1998; WHO 2001). The US EPA (1998; 1987b) and ATSDR (2002a) calculated a unit risk value for beryllium of 2.4×10^{-3} µg Be/m^3 via the inhalation route, based on a cohort study by Wagoner *et al*. (1980).

The US EPA (1998; 1987b) and ATSDR (2002a) cite the cohort study of Wagoner *et al.* (1980) as providing evidence that inhalation exposure to beryllium has resulted in increases in lung cancer-related worker mortality. Wagoner *et al*. (1980) evaluated the carcinogenic potency of inhaled beryllium in a large cohort (n=3055) of exposed workers at a major beryllium production facility in Pennsylvania from 1942 to 1967, with follow-up until 1975. This study revealed a statistically significant increase in lung cancer mortality among workers, in particular among workers with a latency of more than 25 years. There was also a statistically significant increase in the number of lung cancer deaths among workers hired before 1950. The US EPA (1987b) adjusted the lung cancer standardised mortality ratios (SMRs) from this study to account for the use of older vital statistics and a lack of information on smoking. Following this adjustment, the SMRs for a latency of 25 years remained elevated but were no longer statistically significant. Despite these limitations, the available weight of epidemiologic evidence suggests an association between beryllium and lung cancer.

Updated reviews of epidemiologic studies attempt to address some of the limitations of previous studies, such as the lack of information on individual exposures and/or job-history data, and adequate smoking history information (Boffetta *et al*. 2012; Schubauer-Berigan *et al.* 2011a; b; 2008). Schubauer-Berigan *et al.* (2008) re-analysed data from a published case-control study by Sanderson *et al.* (2001). This re-analysis adjusted for the effects of birth cohort, to account for differences in smoking rates by birth year. Adjustment for birth cohort did attenuate the effect of cumulative beryllium exposure on lung cancer; however, the log of average and maximum exposure remained significantly associated with lung cancer mortality.

Schubauer-Berigan *et al.* (2011a) also demonstrated an association between exposure to beryllium and lung cancer. This follow-up of a cohort of 9199 workers from seven beryllium processing plants, previously studied by Ward *et al.* (1992), used quantitative exposure data and job exposure matrices for improved exposure estimates. This study found that mortality in the cohort was elevated for lung cancer (SMR=1.17; 95% CI=1.08-1.28) compared to the general US population. In the subcohort of workers with quantitative exposure estimates, lung cancer SMRs were significantly elevated for workers whose maximum beryllium exposure was ≥10

µg/m^3. A significant trend for lung cancer with increasing cumulative exposure was observed when workers employed for less than one year were excluded.

In another follow-up using a cohort of 5436 workers from 3 of the 7 beryllium processing plants, Schubauer-Berigan *et al.* (2011b) evaluated the exposure-response associations between various exposure metrics and lung cancer while considering confounding factors such as race, duration of exposure, occupational site and exposure to other pulmonary carcinogens. There were statistically significant positive associations between lung cancer and mean and maximum exposure (adjusted for age, birth cohort and plant) and between lung cancer and cumulative exposure (adjusted for age, birth cohort, plant, short-term work status and exposure to asbestos). The authors concluded that there was evidence for increased risk of lung cancer at the U.S. Occupational Safety and Health Administration limit of 2.0 µg/m^3 daily weighted average (DWA) for workers.

The review by Boffetta *et al*. (2012) also considered the cohort previously studied by Ward *et al.* (1992) and Schubauer-Berigan *et al.* (2011a) plus other previously published epidemiological studies, such as the case-control study by Sanderson *et al.* (2001) and patients in the Beryllium Disease Registry. Boffetta *et al*. (2012) concluded that, once confounding factors (e.g., smoking, urban residence, year of birth, year of hire) and lack of correlation with duration of employment and cumulative exposure were taken into consideration, the available evidence did not provide sufficient evidence for a causal association between occupational exposure to beryllium and the risk of cancer.

The carcinogenicity of inhaled beryllium in experimental animals has been extensively reviewed by the US EPA (2008 draft; 1998). Inhalation exposure of beryllium has resulted in lung cancer in rats and monkeys (Nickell-Brady *et al.* 1994; Reeves and Vorwald 1967; Wagner *et al.* 1969). Intravenous and intramedullary injection of beryllium has also induced osteosarcomas in rabbits and possibly mice, although these are some of the earliest studies of beryllium in experimental animals and are not of sufficient quality to evaluate the carcinogenicity of beryllium (Hollins *et al*. 2009; US EPA 2008 draft).

Mutation and chromosomal aberration assays have yielded mixed results for beryllium compounds. While the results of bacterial assays have been largely negative, mutations, chromosomal aberrations and cell transformations have been seen in mammalian test systems exposed to beryllium compounds (Gordon and Bowser 2003; NAP 2007).

4.7. Toxicological Reference Values

Since there is not enough information to support a separate toxicity reference value (TRV) for dermal exposure, the oral TRV will be used in place for both oral and dermal exposure pathways. The TRVs selected for oral and inhalation exposure pathways are discussed below.

4.7.1. Oral Exposure

For evaluation of oral exposures, the TRVs for beryllium from various agencies (ATSDR, US EPA, WHO) are in agreement. No specific tolerable daily intake has been previously provided by Health Canada. For the evaluation of oral exposures, the beryllium reference dose of 2 µg/kg bw/day recommended by the US EPA (1998) was used. In deriving a reference dose, the US EPA (1998) concluded that the Morgareidge *et al.* (1976) dog dietary study was the most appropriate

study. Based on this study, a benchmark dose level resulting in a 10% increase in small intestinal lesions was estimated by US EPA (1998) to be 0.46 mg Be/kg bw/day. A 300-fold uncertainty factor was then applied to estimate a reference dose (i.e., 10-fold for extrapolation for interspecies differences, 10-fold for consideration of intraspecies variation and 3-fold for database deficiencies [i.e., lack of human oral exposure data and inadequate assessment of reproductive/developmental and immunotoxicologic endpoints in animals]). Thus, the US EPA (1998) estimated an oral reference dose of 2 µg/kg bw/day for beryllium. This reference dose has also previously been adopted by WHO (2001) and ATSDR (2002a) and is considered to be appropriate as the tolerable daily intake for estimating soil quality guidelines for beryllium and. Beryllium is not considered to be carcinogenic via the oral route and, thus, the TDI of 2 µg beryllium/kg bw/day is considered to be protective of all endpoints.

4.7.2. Inhalation Exposure – Non-Cancer Effects

For the evaluation of inhalation exposures, the TRVs for beryllium from various agencies (US EPA, ATSDR, WHO) are in agreement. No specific tolerable concentration has been previously provided by Health Canada for the protection of non-cancer effects. For the evaluation of inhalation exposures, the reference concentration of 0.02 µg Be/m^3 recommended by the US EPA (1998) was used to protect for non-cancer effects. In deriving a reference dose, the US EPA (1998) chose the Kreiss *et al*. (1996) occupational exposure study, which identified a $LOAEL_{(HEC)}$ (LOAEL Human Equivalent Concentration) of 0.20 $\mu g/m^3$ and the Eisenbud *et al*. (1949) community monitoring study, which identified a $NOAEL_{(HEC)}$ of 0.01-0.1 $\mu g/m^3$, as the 2 pivotal studies. Other occupational studies were examined for the selection of the LOAEL, but lacked exposure monitoring data or were not selected because CBD was not well defined (Cotes *et al*. 1983; Cullen *et al*. 1987; US EPA 1998). The Eisenbud *et al*. (1949) study evaluated the incidence of CBD at different distances from the plant (hence at different estimated exposure levels) and as such, was the only study that was able to identify a NOAEL for CBD. No uncertainty factor was applied to account for human variability as those developing CBD are the most sensitive subpopulation, nor was an uncertainty factor applied to adjust for the less than chronic duration of the Kreiss *et al*. (1996) study since evidence indicates that effects are not related to exposure duration. The occupational study by Kreiss *et al*. (1996) used more sensitive screening methods to detect CBD than the Eisenbud *et al*.(1949) study, so an uncertainty factor of 3 was applied to account for the sensitive nature of the subclinical endpoint (beryllium sensitisation). A database uncertainty factor of 3 was applied to account for the poor quality of exposure monitoring in the co-principal studies as well as other studies of beryllium sensitisation and CBD. Thus, the US EPA (1998) estimated a reference concentration of 0.02 $\mu g/m^3$. This reference concentration has also been previously adopted by WHO (2001) and ATSDR (2002a) and is considered appropriate as the tolerable concentration for estimating soil quality guidelines for beryllium.

4.7.3. Inhalation Exposure – Cancer Effects

For the evaluation of inhalation exposures, the TRVs for beryllium from various agencies (ATSDR, US EPA, WHO) are in agreement. No specific tolerable concentration has been previously provided by Health Canada for the protection of cancer effects. For the evaluation of inhalation exposures, the inhalation unit risk value of 2.4×10^{-3} $(\mu g\ Be/m^3)^{-1}$ recommended by the US EPA (1998; 1987b) was used for the protection of cancer effects. In deriving an inhalation

unit risk, the US EPA chose the Wagoner *et al*. (1980) study. Study results indicated a statistically significant increase in lung cancer mortality among workers, particularly workers with a latency of more than 25 years. Lung cancer SMRs (standard mortality ratios) were then adjusted by the US EPA (1987b) to account for the use of older vital statistics and the lack of information on smoking. Following this adjustment, lung cancer deaths for the subcohort followed for at least 25 years ranged from 13.91 to 14.67, in comparison with 20 observed, resulting in relative risks of 1.44 to 1.36, respectively. While these risk estimates were no longer statistically significant ($p > 0.05$), they have been used to calculate an upper limit of lung cancer risk (US EPA 1998). The US EPA calculated a range of cancer potency estimates using various assumptions about relative risk estimates and level of exposures. Median beryllium exposure concentrations of 100 to 1000 $\mu g/m^3$ were estimated using the NIOSH (1972) and Eisenbud and Lisson (1983) studies. To calculate the lifetime cancer risk, the median level of beryllium exposure was converted to an 'effective" dose by multiplying by a factor of (8/24) × (240/365) × (f/L) (f/L=ratio of years of exposure to years at risk). This calculation reflects that workers were exposed to beryllium 8 hours/day, 240 days/year, for f years out of a period of L years at risk (i.e. from the onset of employment to the termination of follow-up). The ratio of exposure duration to duration of risk was assumed to range from a minimum of 0.25 to 1.0. The geometric mean of the of potency estimates is 2.4×10^{-3} $\mu g/m^3$. For the purposes of SQG development, a unit risk value of 2.4×10^{-3} $(\mu g\ Be/m^3)^{-1}$ was assumed for beryllium to protect for cancer effects. This unit risk value corresponds to a risk specific concentration of 0.004 $\mu g/m^3$ for an incremental lifetime cancer risk of 1×10^{-5} and 0.0004 $\mu g/m^3$ for an incremental lifetime cancer risk of 1×10^{-6}. Consequently, these values are more stringent than the tolerable concentration of 0.02 μg beryllium/m^3 for protection of non-cancer effects (see above).

The beryllium inhalation unit risk value of $2.4x10^{-3}$ $(\mu g\ Be/m^3)^{-1}$ is based on the US EPA (1998; 1987b) value, as described above. It is noted that new epidemiological data are available for the inhalation effects of beryllium in an occupational setting (as described in Section 4.6); however the inhalation unit risk has not been updated at this time.

5. DERIVATION OF HUMAN HEALTH SOIL QUALITY GUIDELINES

The *Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 2006) can be used to establish guidelines for agricultural, residential/parkland, commercial and industrial land uses. This guidance document was used to calculate the soil quality guidelines for beryllium presented in this document.

5.1. Protocol

For compounds for which the critical effect is believed to have a threshold of exposure, two key factors are considered in the setting of soil guidelines in Canada (CCME 2006). First, it is recognised that, exclusive of hazardous waste sites or any other point source of pollution, everyone is exposed to a "background" level of contamination that cannot be avoided. For beryllium, this background exposure arises primarily from food. In setting soil guidelines for beryllium, the background estimated daily intake (EDI) is subtracted from the Tolerable Daily Intake (TDI) as part of the guideline derivation process.

Secondly, a multimedia approach to guidelines development has evolved whereby guidelines for one medium are established recognising that guidelines for other media may also be required. Guidelines must be established in a manner such that total simultaneous exposure at the guideline levels for all media will not result in exposure which exceeds the TDI. Therefore, in order to set soil guidelines for threshold substances, some portion of the residual tolerable daily intake (TDI - EDI) must be attributed to each medium. With five primary media to which people are exposed (i.e. air, water, soil, food, and consumer products), 20% of the residual tolerable daily intake for threshold (non-carcinogenic) substances is apportioned to each of these media. In cases in which the mechanism of toxicity varies by exposure route, it is possible to calculate SQG using TDIs for each exposure route (i.e. soil ingestion only, dermal contact only or particulate inhalation only). The final direct contact SQG is then the lowest of the calculated values for each direct exposure pathway.

In order to calculate a quantitative guideline, it is necessary to define one or more scenarios by which exposure will occur. As over 80% of Canadians live in cities (Statistics Canada 2001), an urban exposure scenario is the most common situation expected.

5.2. Estimated Daily Intakes

Estimated daily intakes (EDIs) for the Canadian population have been calculated on the basis of the environmental concentration of beryllium in uncontaminated environmental media (see section 2.5). In general, the EDI is an estimate of the typical total concurrent background exposure from all known or suspected sources via a multimedia exposure assessment for the average Canadian. It does not include exposures that may occur from a contaminated site or activities that may result in increased exposure of substances that are not considered background exposure. The EDI calculation is illustrated in the following equation (CCME 2006).

$$EDI = \sum_{i=1}^{n} ED_i$$

The EDIs are expressed in units of µg/kg bw/day and they are intended to represent the average exposure that the Canadian population may receive from beryllium. The general population was subdivided into five age classes: infants (birth to 6 months - breast fed and non-breast fed infants considered separately), toddlers (7 months to 4 years), school age children (5 to 11 years), teenagers (12 to 19 years) and adults (20 years and older). The following media were considered in calculating the EDI: ambient air, indoor air, indoor dust, soil, drinking water, food and breast milk (applicable to breast fed infants only). Consumer products were not included in the EDI estimation because there are limited data in this area. The equation below illustrates the media and pathway-specific EDI calculation (CCME 2006).

$$ED_i = \frac{C \times CR \times BF \times EF}{BW}$$

where,

EDi = exposure dose from pathway i (mg/kg-day)
C = substance concentration in medium (e.g., mg/L)

CR = media specific contact rate (e.g., L/day)
BF = bioavailability factor (unitless)
EF = exposure factor which is the product of the exposure frequency (events/year) and exposure duration (years/lifetime) and is unitless
BW = body weight (kg)

Concentrations of beryllium in environmental media were obtained from government databases, scientific literature and grey literature as summarised in section 2.5 and presented in Appendix 3. Data were selected for EDI determination based on a scoring system to ensure a minimum data quality (HC 2011) or provided directly from Canadian government databases when available, and treated as probability density functions (PDFs) using Crystal Ball® (version 11.1.1.0.00).

Receptor characteristics and intake rates for each age class were treated as probability distribution functions (PDFs) as described in Health Canada (2011). PDFs were assumed to be lognormal except for human breast milk intake and time spent outdoors. A triangular distribution was used for human breast milk intake because of limited availability of data.

PDFs were generated for concentrations in environmental media, receptor characteristics and intake rates. These were used to generate EDI distributions by age group for each media and a combined total EDI from all media and exposure routes using Crystal Ball® (10 000 iterations). Therefore, the total EDI is generated separately and is not simply the sum of the separate EDIs generated for each media per age group. Receptor characteristics and intake rate distributions are presented in Appendices 4 and 5. Appendix 6 summarises the daily intake estimates for beryllium via all media for five age groups of the Canadian general population (HC 2011).

The total beryllium EDIs for adults, teenagers, children and toddlers are 0.011, 0.012, 0.019 and 0.032 µg/kg bw/day (median values). Depending on whether infants are exclusively formula fed, exclusively breast fed or fed a mixture of breastmilk, infant formula and table food, the EDI for infants can be 0.0078 µg/kg bw/day (exclusively breastfed) or 0.046 µg/kg bw/day (non-breastfed). For the purpose of soil quality guidelines derivation, the EDI for toddlers and adults were used.

Certain Canadian subpopulations may be exposed to higher levels of beryllium. For example, people living near industrial areas associated with beryllium emissions could be exposed to higher concentrations of beryllium via inhalation of ambient air. Due to insufficient data, it is not possible, at this time, to perform an exposure assessment for those groups. However, the current analysis does suggest that, next to the consumption of food, the direct contact pathways for soil (incidental ingestion, inhalation and dermal contact) are small contributors to total beryllium exposure.

5.3. Exposure Limits for Human Receptors

The toxicological reference values (TRVs) selected for the derivation of the direct human health-based soil quality guideline (SQG_{DH}) for beryllium are summarised below. More details are available in Section 4.7.

Oral and Dermal Pathway TRV = 2 μg/kg body weight/day (US EPA 1998)

Inhalation Pathway (non carcinogen) TRV = 0.02 $\mu g/m^3$ (US EPA 1998)

Inhalation Pathway (carcinogen) Unit Risk = 2.4×10^{-3} $(\mu g/m^3)^{-1}$ (US EPA 1998)

5.4. 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 substance by the body in the two different media. This can be done according to the following formula:

$$Relative\ dermal\ absorption\ factor = \frac{Absolute\ dermal\ absorption\ rate}{Absolute\ oral\ absorption\ rate\ in\ TRV\ study}$$

Beryllium sulphate was administered to dogs in their diet in the critical study (Morgareidge *et al.* 1976) used to develop the TRV for the oral and dermal pathways. The bioavailability of beryllium in soil via the oral and inhalation routes will vary significantly depending on factors such as soil conditions, the form of beryllium present in the soil and gastrointestinal conditions. Furthermore, there is insufficient information to relate the bioavailability of beryllium in soils to that in the medium administered in the critical study. Therefore, a relative absorption factor of 1 (100%) was selected for exposure via inhalation and ingestion.

OMOE (2008), as referenced in Health Canada's PQRA Part II 2010, recommends a dermal relative absorption factor of 0.1 as a generic default assumption for metals that is not specific to beryllium (HC 2010b). Quantitative data are insufficient to estimate the dermal absorption factor for beryllium; therefore, the OMOE selected the default RAF of 0.1 using an order-of-magnitude approach (default absolute dermal absorption of 1% is approximately an order of magnitude lower than the estimated absolute oral absorption). The absolute dermal absorption value was estimated using a comparison approach of data-derived estimates recommended by international organisations (i.e. US EPA and CalEPA) for various inorganics (OMOE 2011).

For the purposes of this assessment, a relative absorption factor of 0.1 (10%) was used for dermal exposure.

5.5. Ingestion and Dermal Pathways

5.5.1. Agricultural and Residential/Parkland Land Uses

Agricultural lands are characterised by the presence of a farm with a family, including children, where residents consume the produce, meat and milk produced on-site, and groundwater may be used as potable water (50% of produce may be consumed from the agricultural site as well as 50% of meat and 100% of milk). In a residential/parkland setting, the most sensitive receptor may

have access to a backyard, and it is assumed that up to 10% of produce is grown on-site. In both cases, the most sensitive receptor is the toddler, as this age category has the largest exposure to body weight ratio.

Using the above assumptions, a soil quality guideline for direct exposure to soils that applies to agricultural and residential/parkland land uses can be determined using the equation below from the CCME (2006) protocol:

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

where,

SQG_{DH} = direct human health-based soil quality guideline (mg/kg)
TDI = tolerable daily intake by oral route of exposure = $2x10^{-3}$ mg/kg bw/day (US EPA 1998);
EDI = estimated daily intake for a toddler = 3.2×10^{-5} mg/kg bw/day (Appendix 6);
SAF = soil allocation factor (20% by default) = 0.2 (CCME 2006);
BW = body weight for a toddler (16.5 kg) (CCME 2006);
AF_G = relative absorption factor for beryllium across the gut (100%) = 1.0 (CCME 2006);
SIR = soil ingestion rate for a toddler (8×10^{-5} kg/d) (CCME 2006);
AF_S = relative absorption factor for beryllium across the skin (10%) = 0.1 (section 5.4);
SR = soil dermal contact rate for toddler = 6.9×10^{-5} kg/d [hands surface area of 0.043 m^2 × soil adherence factor of 0.001 kg/m^2/d + arms/legs surface area of 0.26 m^2 × soil adherence factor of 0.0001 kg/m^2/d (CCME 2006)];
ET_1 = exposure term 1 (unitless) = 1.0 [7 days per week /7 × 52 weeks per year /52 assumed at the site (CCME 2006)]
BSC = background soil concentration = 0.75 mg/kg (Appendix 3)

therefore,

SQG_{DH} = **75 mg/kg** beryllium for soil ingestion and dermal contact at agricultural and residential/parkland sites.

5.5.2. Commercial Land Use

Commercial sites include such places as shopping malls and places of business. Access to the site is not restricted and since some commercial properties may include daycare facilities, the critical receptor is the toddler. Commercial sites do not include any areas where manufacturing takes place, or areas where individuals may reside.

Since access to commercial sites is not assumed to be 24 hours, exposure assumptions are appropriately less than for residential land use. Discretion should be used in employing the commercial land use classification. In scenarios where unrestricted 24-hour access by children or toddlers, or residential occupancy by any individual is possible, the residential/parkland classification may be more appropriate.

The toddler is the most sensitive receptor at commercial sites. 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.

The direct human health-based soil quality guideline (SQG_{DH}) for soil ingestion and dermal contact for beryllium on commercial lands was calculated as **110 mg/kg**.

5.5.3. Industrial Land Use

Industrial lands typically have limited or restricted access to the public so that adult, occupational exposure will predominate. The most common exposure scenario is expected to be unintentional soil ingestion by an adult. The potential for off-site migration of substances (i.e., via soil and dust) is evaluated for industrial land use scenarios.

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 land use is a manufacturing plant.

Using the above assumptions, a soil quality guideline that applies to industrial land use can be determined:

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

where,

SQG_{DH} = direct human health-based soil quality guideline (mg/kg)
TDI = tolerable daily intake by oral route of exposure = 2×10^{-3} mg/kg bw/day (US EPA 1998);
EDI = estimated daily intake for an adult = 1.1×10^{-5} mg/kg bw/day (Appendix 6);
SAF = soil allocation factor (20% by default) = 0.2 (CCME 2006);
BW = body weight for an adult = 70.7 kg (CCME 2006);
AF_G = relative absorption factor for beryllium across the gut (100%) = 1.0 (CCME 2006);
SIR = soil ingestion rate for an adult = 2×10^{-5} kg/d (CCME 2006);
AF_S = relative absorption factor for beryllium across the skin (10%) = 0.1 (section 5.4);
SR = soil dermal contact rate for an adult = 1.14×10^{-4} kg/d [hands surface area of 0.089 m^2 × soil adherence factor of 0.001 kg/m^2/d + arms surface area of 0.25 m^2 × soil adherence factor of 0.0001 kg/m^2/d (CCME 2006)];
ET_1 = exposure term 1 (unitless) = 0.66 [5 days per week/7 x 48 weeks per year/52 at the site (CCME 2006)];
BSC = background soil concentration = 0.75 mg/kg (Appendix 3).

therefore,

SQG_{DH} = **1400 mg/kg** beryllium for soil ingestion and dermal contact for industrial sites.

5.6. Inhalation Pathway

Beryllium is considered a potential carcinogen via the inhalation pathway. Threshold-based health effects are also associated with exposure to beryllium via the inhalation pathway. Beryllium direct human health-based soil quality guidelines for particulate inhalation ($SQG_{DH\text{-}PI}$) are developed by adapting the dose-based TRV equations in the soil protocol (CCME 2006; threshold p. 108; non-threshold p. 109) to identify the concentration-based TRVs selected in this

document (section 5.3). Threshold and non-threshold $SQG_{DH\text{-}PI}$ were adapted by 1) rearranging equations on page 111 of the soil protocol to solve for TDI and RSD and substituting this into the soil protocol $SQG_{DH\text{-}PI}$ equations [note RSC = ILCR/UR; Health Canada 2010] and, 2) substituting IRs in the soil protocol $SQG_{DH\text{-}PI}$ with DC (dust concentration from resuspension of soil in kg/m^3) x IR (air inhalation rate in m^3/d). The substitutions and cancellation of terms produces the $SQG_{DH\text{-}PI}$ equations presented in this document. Adults are considered the most appropriate receptor for evaluating life-time cancer risk for non-threshold substances. Given that the exposure period is greater than the likely latency period for most carcinogens, the CCME (2006) default exposure term for all land uses is one. The non-threshold soil quality guideline for beryllium based on inhalation of soil particles is calculated as follows:

$$SQG_{DH-PI} = \frac{TILCR}{(DC \times UR \times AF_l) \times ET} + BSC$$

This is essentially a mathematical re-arrangement of the CCME equation for estimation of soil quality guidelines for carcinogens when the cancer potency factor is expressed as a unit risk factor where,

$SQG_{DH\text{-}PI}$ = direct human health-based soil quality guideline for particulate inhalation – non threshold effects (mg/kg);
TILCR = Target Incremental Lifetime Cancer Risk (1×10^{-6} or 1×10^{-5});
UR = unit risk = 2.4×10^{-3} $(\mu g/m^3)^{-1}$ (US EPA 1998);
AF_l = absorption factor for beryllium across the lung (100%) = 1.0 (CCME 2006);
DC = dust concentration from resuspension of soil = 7.6×10^{-10} kg/m^3 (CCME 2006);
ET = exposure term (unitless) = 1 (i.e., continuous lifetime exposure for an individual);
BSC = background soil concentration = 0.75 mg/kg (Appendix 3).

Derivations are provided based on incremental lifetime cancer risks (ILCR) of both 1×10^{-6} and 1×10^{-5}.

The $SQG_{DH\text{-}PI}$ for inhalation of soil particulates is **5500 mg/kg** for an excess cancer risk of 1×10^{-5}, and **550 mg/kg** for an excess cancer risk of 1×10^{-6}.

For threshold health effects, toddlers were considered the most appropriate receptor for residential and commercial lands, while adults were considered the most appropriate receptor for industrial lands. The threshold soil quality guidelines for beryllium based on inhalation of soil particles are calculated as follows:

$$SQG_{DH-PI} = \frac{TC \times SAF}{(DC \times AF_l \times ET_1 \times ET_2)} + BSC$$

This is essentially a mathematical re-arrangement of the CCME equation for estimation of soil quality guidelines for non-carcinogens when the toxicity reference value is expressed as a tolerable concentration where,

$SQG_{DH\text{-}PI}$ = direct human health-based soil quality guideline for particulate inhalation – threshold effects (mg/kg);
TC = tolerable concentration in air = 2.0×10^{-2} $\mu g/m^3$ (US EPA 1998);
SAF = soil allocation factor (20% by default) = 0.2 (CCME 2006);
AF_l = absorption factor for beryllium across the lung = 1.0 (CCME 2006);

DC = dust concentration from resuspension of soil = 7.6×10^{-10} kg/m^3 (CCME 2006);
BSC = background soil concentration = 0.75 mg/kg (Appendix 3);
ET_1 = exposure term 1 (unitless) = 1.0 for residential land use (i.e., 24 hours/day); 0.66 (unitless) for commercial and industrial land use (i.e., 5/7 days per week x 48/52 weeks per year at the site) (CCME 2006);
ET_2 = exposure term 2 (unitless) = 1.0 for residential land use; 0.42 for commercial and industrial land use (10/24 hours per day at the site) (CCME 2006).

The SQG_{DH-PI} for inhalation of soil particulates for protection of non-cancer risks are **5300 mg/kg** for agricultural and residential/park lands, and **19 000 mg/kg** for commercial/industrial lands.

A summary of SQGs for soil inhalation is provided below in Table 4 along with the overall soil inhalation SQG for each land use category.

Table 4. Soil inhalation pathway soil quality guidelines for beryllium.

Land Use-Specific Soil Inhalation SQG_{DH-PI} (mg/kg)				
	Agricultural	Residential/ Parkland	Commercial	Industrial
Non-Threshold (Target risk of 1×10^{-5})	5500	5500	5500	5500
Non-Threshold (Target risk of 1×10^{-6})	550	550	550	550
Threshold	5300	5300	19 000	19 000
Overall Land Use-Specific Soil Inhalation SQG_{DH-PI} (mg/kg)				
1×10^{-5} ILCR	5300[a]	5300[a]	5500	5500
1×10^{-6}ILCR	550	550	550	550

[a] The guideline values is set at the lowest of the guideline values. For the soil inhalation pathway, the threshold guideline value is lower than the non-threshold value for an incremental lifetime cancer risk of 10^{-5} and is therefore, set as the guideline value for this land use.

5.7. Protection of Groundwater Used as a Source of Raw Water

The guideline for protection of groundwater used as a source of raw water for drinking water was not calculated for beryllium due to constraints of the mathematical model when applied to inorganic compounds (CCME 2006).

5.8. Guideline for Consumption of Produce, Meat and Milk

The check mechanism for consumption of produce, meat and milk was not required because beryllium is not a substance that bioaccumulates as discussed in Section 3.6. It is recognised that the CCME (2006) protocol assumes the consumption of produce, meat and milk for agricultural land use and the consumption of produce for residential land use.

5.9. Guideline for Off-site Migration for Commercial and Industrial Land Uses

Soil Quality Guidelines for Commercial and Industrial sites account for on-site exposures only. Transfers of contaminated soil, from one property to another are possible by environmental occurrences such as wind and water erosion (CCME 2006).

The human health soil quality guideline for off-site migration (SQG_{OM-HH}) refers to the concentration in soil eroded from the site that will raise the contaminant concentration in the receiving soil to the level of the agricultural guideline within a specific time frame. If the guidelines for commercial or industrial sites are found to be above SQG_{OM-HH}, then the adjacent, more sensitive land (e.g., agricultural or residential) could potentially become unacceptably contaminated from off-site deposition (CCME 2006). The following equation has been derived to allow the calculation of SQG_{OM-HH}.

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

where,

SQG_{OM-HH} = human health soil quality guideline for off-site migration [i.e., the concentration of substance in eroded soil (mg/kg)]
SQG_{A-HH} = Human health soil quality guideline for agricultural land uses = 75 mg/kg
BSC = background concentration of substance in the receiving soil = 0.75 mg/kg

The SQG_{OM-HH} for commercial and for industrial land uses was determined to be **1100 mg/kg**, which is more than the SQG_{DH} for commercial (110 mg/kg) but less than the industrial (1400 mg/kg) land uses. Therefore, only the industrial SQG_{HH} needs to be modified to protect against off-site migration.

5.10. Final Human Health Soil Quality Guidelines

Based on the CCME (2006) protocol, three types of exposure pathways are evaluated: required pathways, applicable pathways and check mechanisms. The SQGs for each of the pathways calculated are listed in Table 5 (next section).

Human health soil quality guidelines were calculated for beryllium at contaminated sites, based on ingestion, inhalation and dermal contact with contaminated soil. Beryllium was assumed to behave as a threshold substance via the ingestion, inhalation and dermal contact pathways and as a non-threshold substance via the inhalation pathway. The proposed human health soil quality guidelines for agricultural and residential/parkland land uses is 75 mg/kg soil, 110 mg/kg soil for commercial land use, based on direct contact via ingestion and dermal exposures and 1100 mg/kg (ILCR 10^{-5}), based on the offsite migration check or 550 mg/kg (ILCR 10^{-6}) soil for industrial land use based on inhalation exposures.

While the SQG_{HH} provided above are considered to be protective at most sites not all exposure pathways have been evaluated such as beryllium levels in garden produce, meat, milk consumption or beryllium leaching from soils to groundwater or surface water used for drinking water. A site-specific risk assessment and sampling of additional media may be warranted at sites where these media may be impacted by elevated levels of beryllium in soil.

With the above in mind, the SQG_{HH} are considered to be protective of human health at most sites.

6. RECOMMENDED CANADIAN SOIL QUALITY GUIDELINES

According to the soil protocol (CCME 2006), both environmental and human health soil quality guidelines are developed for four land uses: agricultural, residential/parkland, commercial and industrial. The lowest value generated by the two approaches for each of the four land uses is recommended by CCME as the final Canadian Soil Quality Guideline. If either the SQG_{HH} or the SQG_{E} cannot be calculated, and the final soil quality guideline is higher than the interim (1991) criterion, then the final soil quality guideline will be set at the 1991 criterion. The environmental soil quality guidelines for beryllium were not developed, and the SQG_{HH} are higher than the 1991 criterion for beryllium, therefore, the final Soil Quality Guidelines for the protection of environmental and human health is based on the 1991 criteria. The recommended Canadian Soil Quality Guidelines for the protection human health are presented below in Table 5.

Table 5. Soil quality guidelines and check values for beryllium (mg/kg).

	Land use			
	Agricultural	**Residential/ parkland**	**Commercial**	**Industrial**
Guideline	**4**[a]	**4**[a]	**8**[a]	**8**[a]
Human health guidelines/check values				
SQG_{HH} (ILCR 10^{-6})	75[b]	75[b]	110[b]	550[b]
SQG_{HH} (ILCR 10^{-5})	75[b]	75[b]	110[b]	1100[b]
Direct contact guideline (Ingestion + Dermal)	75	75	110	1400
Direction contact guideline (Inhalation)				
Non-threshold (ILCR 10^{-6})	550	550	550	550
Non-threshold (ILCR 10^{-5})	5500	5500	5500	5500
Threshold	5300	5300	19 000	19 000
Inhalation of indoor air check	NC[c]	NC[c]	NC[c]	NC[c]
Off-site migration check	—	—	1100	1100
Groundwater check (drinking water)	NC[d]	NC[d]	NC[d]	NC[d]
Produce, meat, and milk check	NC[e]	NC[e]	—	—
Environmental health guidelines/check values				
SQG_E	NC[f]	NC[f]	NC[f]	NC[f]
Soil contact guideline	NC	NC	NC	NC
Soil and food ingestion guideline	NC	—	—	—
Nutrient and energy cycling check	NC	NC	NC	NC
Off-site migration check	—	—	NC	NC
Groundwater check (aquatic life)	NC[d]	NC[d]	NC[d]	NC[d]
Interim soil quality criterion (CCME 1991)	4	4	8	8

Notes: NC = not calculated; ND = not determined; SQG_E = soil quality guideline for environmental health; SQG_{HH} = soil quality guideline for human health. The dash indicates a guideline/check value that is not part of the exposure scenario for this land use and therefore is not calculated.

[a] Data are sufficient and adequate to calculate only SQG_{HH}. They are greater than the corresponding interim soil quality criteria (CCME 1991). Therefore the interim soil quality criteria are retained as the soil quality guideline for this land use.

[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 metal substances.

[d] Applies to organic compounds and is not calculated for metal substances. Concerns about metal substances should be addressed on a site specific basis.

[e] This check is intended to protect against chemicals that may bioconcentrate in human food. Beryllium is not expected to exhibit this behaviour, therefore this pathway was not evaluated.

[f] Data are insufficient/inadequate to calculate an SQG_E or provisional SQG_E for this land use (no attempt was made to derive environmental soil quality guidelines for beryllium).

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APPENDICES

Appendix 1. Summary Tables of Beryllium Concentrations in Environmental Media

Ambient Air

Location	Year	Concentration mean ng/m^3	Range ng/m^3	Comments	Reference
Canada					
British Columbia, Ontario, Quebec and New Brunswick from urban and rural centres.	2003-2009	0.013	0-0.087	SD=0.011, n=3054. Mean based on NAPS data ($PM_{2.5}$ fraction analysed by ICP-MS following acid digestion).	EC 2011.
Canada	2003-2006	0.01	-	n=3528 from 13 monitoring stations across Canada. ICP-MS analysis. Be levels <DL in>99% of samples.	Dann 2007 *pers. comm*.
Quebec					
Shawinigan			<DL	n=27, Analysed by ICP-AES. DL=0.2 ng/m^3.	
Quebec					
Rouyn Noranda			0.2 to 0.4	n=242, 93% < analytical detection limit of 0.2 ng/m^3.	
Quebec					
Murdochville	1999-2002	-	0.2 to 2.3	n=242, 73% < analytical detection limit of 0.2 ng/m^3.	Bisson 2004 *pers. comm*.
Ontario					
Burnt Island					
Egbert					
Point Petre	1997-2001	<DL	-	n=355, DL=0.02ng/m^3.	Dryfhout-Clark 2004 *pers. comm*.
Ontario				n=27; Samples collected outdoors at homes and offices.	
Winsor	1991-1992	<DL	-	Analysed by ICP-MS. DL=0.25ng/m^3.	Bell *et al*. 1994.
Alberta		<0.1in $PM_{2.5}$ and		Samples from 4 Edmonton locations adjacent to the	
Edmonton	2001-2002	TSP (median)	-	Inland Cement Facility (upwind and downwind).	Alberta Environment 2002
NWT					
Alert	1997-2001	<DL		n=259. DL=0.01 ng/m^3	Gong 2004 *pers. comm.*
BC				n=16, maximum (22 ng/m^3) occurred during weeks of low	
Burnaby, Vancouver and New Westminster	1995	2.4	0.4 to 22	precipitation. DL=0.5 ng/m^3. Samples analysed by ICP-AES.	Brewer and Belzer 2001.
International					
United States		-	0.03-6.71		
Germany, Berlin		-	0.22-0.33		
Germany, Frankfurt		-	0.02-0.06		
Japan		0.042	-		
Japan, Tokyo	2001	0.22	-	-	Thorat *et al.* 2001.

Indoor Air

Location	Year	Concentration mean ng/m^3	Range ng/m^3	Comments	Reference
Ontario					
Windsor	1991-1992	<DL	-	19 indoor (10 home and 9 office) samples analysed by ICP-MS. DL=0.25 ng/m^3.	Bell *et al*. 1994.

Location	Year	Concentration mean ng/m^3	Range ng/m^3	Comments	Reference
United States		Personal			
New York City		0.002 ± 0.0008			
		Home indoor			
		0.015			
		Home outdoor			
		0.028			
Los Angeles		Personal			
		0.002 ± 0.0008			
		Home indoor			
		0.018		Collection of personal, home indoor and home	
		Home outdoor		outdoor samples from 46 high school students from	
	1999 and 2000	0.018	-	NYC and 41 from LA.	Sax *et al*. 2006.

Indoor Dust

Location	Year	Concentration mean mg/kg	Range mg/kg	Comments	Reference
Ontario				n=48. Samples analysed by ICP-MS. DL=0.02	
Ottawa	2001	0.56	0.28-1	mg/kg.	Rasmussen *et al*. 2001.

Soil

Location	Year	Soil type	Sample Depth	Concentration mean mg/kg	Range mg/kg	Comments	Reference
Canada	Multiple surveys	<63 µm size fraction	Till (C-horizon)	0.75	0.25-16	n=9876. Aqua regia digestion, CPMS or AAS analysis. Data from GSC database available from NRCan website.	Grunsky 2010 *in* HC 2011.
Ecoregions across Canada	-	Overburden Soils	-	60% below DL.	-	DL=0.5 mg/kg. Analysis by INA.	Garrett 2007.
New Brunswick							
Fredericton (rural garden)				1.5 ± 0.1 (n=2)			
East Saint John (urban garden)				1 ± 0.1 (n=18)			
West Saint John (urban garden)	1992	Garden Soils	-	1.4 ± 0.1 (n=4)	-	Analysis by HNO_3/ H_2O_2/HCl ICP-MS.	Pilgrim and Schroeder 1997.
Ontario							
Essex County	1970s	Clay Loam	-	0.7	0.4-1.0	-	Kemp and Dell 1976.
Ontario							
All soil Agricultural belt				0.35±0.16	0.10-0.89		
Clay soil				0.48 ±0.16	0.20-0.89	n=228, 62 and 70 respectively. Soil	
Sandy soil	Late 1970	Surface Soils	15 cm	0.25 ± 0.08	0.10-0.44	analysed by AAS.	Frank *et al.* 1979.
		Old urban parkland		0.6	0.2-1.12	n=60 and 101 respectively. Analysis by HNO_3/HCl/ICP/AA	
Ontario	1994	Rural Parkland	-	0.4	0.2-1.2	DL=0.5 mg/kg.	OMEE 1994.
			Field 1: 0-20cm	1.57	1.5-1.7		
			Field 2: 0-20cm				
Ontario		Agricultural Clay	Field 1:50-65cm	1.96	1.8–2.1		
Ottawa	-	Loam	Field 2: 50-65cm			Analysis by $HClO_4$/HNO_3, ICP/AE.	Wang 1994 *pers. com.*
Ontario		Street dust		0.97	0.31-1.25	Samples analysed by ICP-MS.	Rasmussen *et al.*
Ottawa	2001	Garden soil	-	1.4	1-1.77	DL=0.02 mg/kg.	2001.
Manitoba			**North**				
North: (*JenPeg, Snow*			0-5 cm (organic)	33	<0.1-0.19		
Lake, FlinFlon, Gillam,			5-10 cm (clay/silt)	43	<0.1-2.0		
James Lake, Snow Lake,			10-15 cm (clay/silt)	38	<0.1-2.0		
Thompson - Orr Lake),			15-30 cm (clay/silt)	8	<0.1-2.0		
			Central				
Central: *(Minitonas,*			0-12 cm (organic)	19	0.30		
Swan River, The Pas)			12-25 cm (clay/silt)	16	0.35		
			South			Northern Manitoba (n=33), 86% <DL.	
South: *(Mediation Lake,*			0-2 cm (organic)	37	<0.1–0.4	Be detected in all central Manitoba	
Manitoba Escarpment,	1995 to		2-15 cm (clay/silt	38	<0.1–0.9	surface samples. In surface soils from	
Pine Falls)	2003		sand)			the south (n=37), Be was frequently	Jones 2004 *pers.*
		-	17-22 cm (sand)	10	<0.1	(61%) <DL (DL=0.1mg/kg).	*comm.*

Location	Year	Soil type	Sample Depth	Concentration mean mg/kg	Range mg/kg	Comments	Reference
North America			0-5cm	1.2			
(Manitoba is the only			A-horizon	1.2			
Canadian data included)			C-horizon	1.3			
	2005	-	O-horizon	0.7	-	Analysis by ICP-MS.	USGS 2005.
British Columbia		Sandbox soils		0.34±0.09	0.2-0.5		
Trail		(n=35)					
		Park soils (n=41)		0.35±0.08	0.2-0.5		
		Residential soils				Analysis by $HNO_3/HClO_4$/HCl	
	1989	(n=122)	-	0.43±0.08	0.3-0.7	ultrasonic nebulisation-ICP.	Kelly *et al*. 1991.
British Columbia	Before						
Greater Vancouver	Incinerator:						
	1987			0.1			
	After incinerator:						
	1988			0.3			
	1989			0.1		Analysis by $HNO_3/HClO_4$/ICP.	
	1990	-	-	0.2	-	DL=0.1 mg/kg	Soilcon 1987-1990.
British Columbia							
Vancouver Island				1.5			
Lower Mainland				1.5			
Greater Vancouver				1			
Southern Interior				2			
Kootenay				2			
Cariboo		Regional		2			
Skeena		Background Soil	Depths ranging from	1.5			
Omineca Peace	-	Quality Estimates	0 cm to 60 cm.	2	-	Aqua-Regia digestion used.	BCMWLAP 2010.
British Columbia		Nitric perchloric					
Across BC excluding		digestion.		0.6	0.1 to 2.7	n=408	
Vancouver		Aqua regia	depths ranging from				Harris 2004 *pers.*
	-	digestion.	0 cm to 60 cm.	0.8	0.1 to 2.7	n=140	*comm.*

Notes:

- AA-Atomic Absorption; AE- Atomic Emission; ICP-Inductively Coupled Plasma; INAA-Instrumental Neutron Activation Analysis; IPAA- Instrumental Photon Activation Analysis; NR-Not Reported

- The method of digestion strongly influences the results of analysis of beryllium in soils. While *aqua regia* (HNO_3/HCl) releases the "biologically-relevant" beryllium contained in soluble salts and organic matter, the beryllium adsorbed onto soil particles as well as the beryllium present in some weak silicates, hydrofluoric acid (HF) also leaches beryllium from most silicates and stable mineral matrices (R. Garrett, NRCan, *pers. comm*.). For example, Fox (1990) obtained recoveries of 17%, 30% and 80-98% beryllium from leaches of HNO_3/HCl, $HNO_3/HClO_4$ and HF/HCl/$HNO_3/HClO_4$, respectively. OMEE laboratories recovered an average of 20% (4-34%) of the beryllium contained in Canadian reference samples after HNO_3/HCl digestion of samples (L. Pastorek, OMEE, *pers. comm*.).

Surface Water

Location	Year	Concentration mean µg/L	Range µg/L	Comments	Reference
Newfoundland	1985-2000	-	0.006-0.94	-	NLDEC 1985-2000.
Ontario			Average	Water collected at water treatment plant locations	
Lakes (n=84)			concentration	under Ontario Drinking Water Surveillance	
Rivers (n=47)	1990-2002		0.01-0.06	Program	Cheung 2004 *pers. comm*.
Ontario	1994-2003	< 0.1	20.2-50.3	Analysed using methods E33080A and E3391A.	Boyd 2004 *pers. comm.*
Great Lakes					
Dissolved beryllium			0.0037-0.032	Analyses using graphite-furnace atomic absorption	
Beryllium in particulate fraction			0.00009-0.1	spectrophotometry (GFAA) on 10 to 22 samples	
Total beryllium	1988	-	0.0039-0.12	from each lake.	Rossmann and Barres 1988.
Saskatchewan					
Rivers and streams (n=63)					
Lakes (n=103)					
Rural Northern Manitoba (n=5)	1998-2001	<DL		ICP-AES (Method 1728)	Hase 2004 *pers. comm*.
Alberta				DL = 0.2 µg/L	
Bow River Basin			<0.2-.2.5	n=168; 93% <0.2	
Athabasca River Basin			<0.2-.5	n=185: 84% <0.2	
North Saskatchewan River Basir			<0.2-.14	n=78: 81% <0.2	
Oldman River Basin			<0.2-.2.4	n=110: 86% <0.2	Anderson 2004 *pers.*
Red Deer River Basin	1997-2003	-	<0.2-.2.5	n=36: 89% <0.2	*comm.*
NTW Nahanni Park					
Dissolved beryllium	1988-1991		0.0168 - 0.0266		
Total beryllium	1992-1997	-	0.025 - 0.1218	-	Halliwell and Catto 2003.
British Columbia					
Quinsam River near the Mouth	1986-2004		<0.05-.18		BC and EC, 2007g
Thompson River at Spences Bridge	1984-2004		<0.05-0.09		BC and EC, 2007e
Nechako River at Prince George	1985-2004		<0.05-0.15		BC and EC, 2007f
Flathead River at the U.S. border	1980-2004		<0.05-0.10		BC and EC, 2005
Columbia river at Birchbank	1990-2005		<0.05		BC and EC, 2008b
Columbia river at Waneta	1979-2005		<0.05-0.07		BC and EC, 2008a
Kootenay River at Fenwick	1984-2005		<0.05-0.125		BC and EC, 2007a
Kootenay River at Creston	1979-2005		<0.05-0.18		BC and EC, 2007b
Elk River at Highway 93 near Elko	1984-2005		<0.05-0.2		BC and EC, 2007d
Salmon River at Salmon Arm	1985-2004		<0.05-0.3	A background surface water concentration of 0.05	BC and EC, 2007h
Columbia River at Nicholson	2003-2006	-	0-0.10	µg/L would be representative of these sites	BC and EC, 2007c
International Data					NTP 1999. Merrill *et al*. 1960.
			0.000035- 0.00022		Venugopal and Luckey 1978.
Ocean water					Measures and Edmond 1982.
	-	-	0.0006 – 0.04	-	Léonard and Lauwerys 1987.

Groundwater

Location	Year	Concentration mean µg/L	Range µg/L	Comments	Reference
Newfoundland & Labrador					
Duffins Creek-Rouge River basin	1997	5	-	3 wells across the province	NLDEC 1997.
Ontario	1990-2002	<DL	-	DL=0.01-0.03. 48 groundwater supply wells sampled under the Ontario Drinking Water Surveillance Program (n=2615)	Cheung 2004 *pers. comm*.
Ontario Simcoe County		0.00055±			
Johnson site (n=11)	2004-2006-	0.00006		Beryllium determined in water using inductively coupled	
Parnell site (n= 12	2004-2006	0.00056± 0.00011	-	plasma-sector field mass spectrometry (ICP-SMS).	-
Alberta				Deep wells (n=100) and shallow wells (n=111) analysed by various methods. Be concentrations <DL in all samples	
Deep Wells		N/A	<DL	(DL=0.054 µg/L for EPA 200.8; <1µg/L for APHA 3210B	
Shallow Wells	-	N/A	<DL	and <2 µg/L for SW6010).	Holt-Oduro 2004 *pers. comm*.
British Columbia					
Wasa aquifer	2003-2004	0.02	-	5 wells sampled. n=29	BC MWLAP 2004.
Yukon	1995-2001	<DL	-	DL=0.2 & 0.6 µg/L. n=32.	Beckerton 2004 *pers. comm*.

Drinking Water

Location	Year	Concentration mean µg/L	Range µg/L	Comments	Reference
	1992	1.5	-	80 cities across Canada. Analysed by inductively coupled argon plasma emission spectrometry.	Meringur and Lo 1992.
Canada	2000	0.95	-	500mL tap water samples collected as part of the 2000 Canadian Total Diet Study.	Dabeka 2004 pers. com.
Ontario	1998-2007	0.03	0.025-1.85	Data from the Drinking Water Surveillance Program. n=3777, SD=0.05.	OMOE 2010.
Ontario					
Groundwater				Groundwater n=996 (48 supply wells). Lake water	
Lakes			DL (<0.01 -	n=2874 (84 treatment plants) and River water	
Rivers	1990-2002	<DL	0.03)-	n=2219(47 treatment plants).	Cheung 2004 *pers. comm.*
Manitoba					
Rural Northern groundwater wells				Well water n=8 and treatment plant water n=3 Beryllium concentrations in water collected from	
treatment plants	-	-	<0.1-6	treatment plants were below detection (<1 µg/L).	Yee 2004 *pers. comm*.
Saskatchewan	2000-2009	0.44	0.05-0.5	n=64, SD=0.15.	GS 2008.
Yukon	1999-2003	<DL	<0.01-0.6	n=9. All below analytical detection limits.	Bergsam 2004 *pers. comm.*

Snow

Location	Year	Concentration mean ng/L	Range ng/L	Comments	Reference
Ontario					
Simcoe County		1.4 ± 0.5 (n=3)			
Old Johnson Farm (2 sites)	2009	2.9 ± 3.5	-	-	
Parnell Farm		2.3 ± 0.7			Shotyk *et al*. 2010.

Sediment

Location	Year	Concentration mean mg/kg dry weight	Range mg/kg dry weight	Comments	Reference
Ontario					
Lake Ontario		1.2	0.9-1.7		
Lake Erie	1976	1.5	1.2-1.9	-	Kemp and Dell 1976.
Ontario				n=14. Sample digestates analysed using inductively coupled plasma atomic emission	
Tadenac Lake	1979	1.7	1.3-2.1	spectrometer.	Wren *et al.* 1983
Ontario					
Detroit River and western Lake Erie					
Total beryllium			0.1-3.8		
Extractable beryllium	1982	-	<0.02-0.8	n=20.	Lum and Gammon 1985.
				Samples analysed for dry weight concentrations of total beryllium (method E3062A).	
Ontario	1994-2003	-	0.6-1.5	67% of samples ≤analytical DL (0.5 mg/kg).	Boyd 2004 *pers com*.
Nova Scotia					
Bay Fundy	1979	1.6	0.8-2.9	n=83.	Loring 1979.
Manitoba					
Northern lakes and rivers	-	-	0.06-0.17	n=5.	Yee 2004 *pers. com.*
Northwestern Saskatchewan					
Carswell Structure	-	mean of 0.5	<0.1-2.8	-	Dunn 1980.

Biota Used as Human Food

Location and Type of Food	Year	Concentration mean mg/kg dw	Range mg/kg dw	Comment	Reference
Newfoundland					
Crab tissue (male Atlantic snowcrab, queen crab and spider crab)					
Inner Avalon			<0.004-0.016 (n=23)		
Bay St. George			<0.004-0.037 (n=30)		
Outer Avalon			<0.004-0.049 (n=36)		
Bonavista		-	<0.004-0.017 (n=24)		
White Bay			<0.004-0.056 (n=24)		
Conception Bay			<0.004-0.067 (n=36)	Tissue analyses conducted by ICP/MS. Maximum concentration consistently occurred in crab gills.	
Shoal Patch			<0.004-0.026 (n=24)		
Port au Choix			<0.004-0.041 (n=29)	There was a greater frequency of occurrence of lower range concentrations (i.e., non-detect)	
Clam tissue (Arctic surfclam) sampled at Banquereau Bank	1996		<0.004-0.037 (n=19)	compared to high concentrations in both crabs and clams. DL=0.004 mg/kg.	Fancey 2004 *pers comm.*
Ontario					
Tadenac Lake			0.10-0.28		
Clam (soft tissues)		0.19			
Fish Muscle (minnow, smelt, bass, pike and char)	1983	<0.10		n=14. Samples digested and analysed using inductively coupled plasma atomic emission spectrometry.	Wren *et al.* 1983.
Northwest Territories				Livers and kidneys of 20 barren ground caribou (Beverly herd) analysed for metals as part of a NWT contaminant monitoring program. Concentrations	
Barren ground caribou (Beverly herd)	2001	<DL	-	generally <DL (DL=0.01).	Gamberg 2002.
British Columbia – 54 uncontaminated lakes		Liver Tissue	Muscle Tissue		
Rainbow trout		0.31 ± 0.21 (n=110)	0.23 ± 0.03 (n=112)		
Cutthroat trout		0.28 ± 0.21 (n=75)	0.22 ± 0.02 (n=54)		
Lake trout	-	0.28 ± 0.12 (n=19)	0.23 ± 0.04 (n=25)		
Dolly Varden		0.26± 0.12 (n=49)	0.23 ± 0.05 (n=51)		
Mountain Whitefish		0.25 (n=24)	0.24 ± 0.03 (n=20)		
Arctic Grayling		0.22 ± 0.06 (n=24)	0.20 ± 0.02 (n=22)	- Concentrations are expressed as wet weight.	Rieberger 1992.
United States					
Commencement Bay, Tacoma Washington					
English Sole	-	0.006	-		Nicola *et al.* 1987.

Commercial Foods

Food Type	Year	Concentration mean	Range	Comment	Reference
Bottled Water					
Mineral Water		0.93 µg/L (n=42)			
Soda		0.23 µg/L (n=19)		Water analyses conducted by	
Distilled Water	1995-	0.23 µg/L (n=25)		inductively coupled plasma mass	Dabeka *et al*.
Spring	1996	0.23 µg/L (n=102)		spectrometry (ICP-MS),	2002.
	-		Levels ranged from less than detection limits for foods such as bananas and beans	Be concentrations in food from peer reviewed literature up to 1998. 65 foods sampled (e.g., beans and peas). Accuracy of analytical methods varied greatly, which, when corrected for, suggest concentrations of <1 to 20 µg/kg Be fresh weight.	
Kidney beans		2,2 mg/kg			Vaessen and
Garden Peas		0.109 mg/kg			Szteke 2000.
Cane Sugar					
Barbados brown sugar		0.03 mg/kg			
Demerara sugar		0.006 mg/kg	-	-	
Refined sugar	1972-	0.002 mg/kg			Hamilton and
granulated sugar	1973	0.0002 mg/kg			Minsky 1972-1973.
Spain					
Tarragona County (Catalonia) Meat, fish and seafood; pulses; cereals, vegetables; tubers; fruit; whole milk, dairy products; eggs, sugar.	2006	<0.05 µg/kg fresh weight		DL=0.05. All samples analysed were <DL. Food samples were divided into 11 groups. n=360.	Martí-Cid *et al*. 2009.
United States					
Carrot	-	<1 µg/g dw	-	DL=1	Wolnik *et al.* 1984.
United States				DL=1. n=8 for fruits. n=9 for	
Fruit				vegetables. From 11 areas of	
Vegetables	-	<1 µg/g	-	commercial food production.	Shacklette 1980.
Germany					
Lettuce		0.0165 µg/g WW (0.330 µg/g DW)			
Tomatoes		0.012 µg/g WW (0.240 µg/g DW)	-		
Rice		0.066 µg/g WW (0.080 µg/g DW)			
Potatoes		0.039 µg/g WW (0.170 µg/g DW)		WW=wet weight. DW=dry weight.	Zorn and Diem 1974.
Egyptian crops					
Eggplant		0.370 µg/g DW			
Potatoes		0.300 µg/g DW	-		
Green Pepper		0.400 µg/g DW		Analysis by Inductively coupled	
Kidney Bean		2.500 µg/g DW		plasma-atomic emission	Awadallah *et al.*
Garden Pea	1986	0.430 µg/g DW		spectrometry.	1986.

Food Type	Year	Concentration mean	Range	Comment	Reference
Vegetable Marrow		0.400 µg/g DW			
Pear		0.400 µg/g DW			
Lettuce		0.600 µg/g DW			
Dill		0.42 µg/g DW			
Parsley		0.40 µg/g DW			
Coriander		0.47 µg/g DW			
Formula Milk					
USA milk based		0.0001 ± 0.0002 µg/mL	-	-	
USA soy-based		0.00007 ± 0.00011 µg/mL			
UK milk-based liquid	2002	0.0011 ± 0.0009 µg/mL			Ikem *et al*. 2002.

Human Tissues and Biological Fluids

Tissue/Fluid	Year	Concentration mean	Range	Comment	Reference
Blood Serum	2008	0.63±0.08 µg/L 0.43±0.03 µg/L	0.48-0.74 µg/L 0.40-0.49 µg/L	n=10 non-exposed individuals. Concentration determined using graphite furnace atomic absorption spectrometry	Stephan *et al.* 2008.
Blood Serum	2005	0.42±0.19 µg/L 0.21±0.12 µg/L		n=110. Non-exposed adults aged 20-61 yrs (mean 42±10) from urban Rome. Analysis performed by inductively-coupled plasma mass spectrometry (ICP-MS).	Alimonti *et al*. 2005.
Serum	1990	0.15±0.006 µg/L	<0.08-0.36 µg/L	Analyses of serum from unexposed Italian subjects (n=398) living in Lombardy, Italy. Analysis performed by flame atomic absorption analysis (AAS), neutron activation analysis-electrothermal atomic absorption analysis (ETA-AAS), or inductively-coupled plasma atomic emission spectrometry (ICP-AES).	Minoia *et al*. 1990.
Blood	1998	Less than 0.004 µg/mL (DL)	-	72 men, 72 women (>16 years) living in Tarragona, Spain and surroundings. Analysis performed by inductively coupled plasma-mass spectrometry (ICP-MS).	Llobet *et al.* 1998b.
	1980	1.0±0.4 ng/g	-	n=10 non-occupationally exposed German subjects Bowen (1979) reported Be levels of <0.01 µg/L in whole blood. Analysis performed by flameless atomic absorption spectrometry (AAS) method.	Stiefel *et al.* 1980.
	1979	<0.01 µg/L	-		Bowen 1979.
Plasma	2008	0.02±0.032 µg/L	0-0.294 µg/L	Sera samples (n=206) from lactating mothers. Analysed by inductively-coupled plasma mass spectrometry (ICP-MS).	Abdulrazzaq *et al.* 2008.

Tissue/Fluid	Year	Concentration mean	Range	Comment	Reference
Urine	1980	0.9±0.5 ng/g	-	n=10 non-occupationally exposed Germans. Analysis performed by flameless atomic absorption spectrometry (AAS).	Stiefel *et al.* 1980,
	1980	2 ng/g	-	Urine of smokers. 120 ng Be per cigarette. Analysis performed by flameless atomic absorption spectrometry (AAS).	Stiefel *et al*. 1980.
	1990	0.4±0.09 µg/L	<0.02-0.82 µg/L	Analyses of serum from unexposed subjects (n=579) living in Lombardy, Italy. Analysis performed by flame atomic absorption analysis (AAS), neutron activation analysis-electrothermal atomic absorption analysis (ETA-AAS) or inductively-coupled plasma atomic emission spectrometry (ICP-AES)	Minoia *et al.* 1990.
	1989	0.24±0.16 µg/L	-	Italian population not occupationally exposed. Analysis performed by electrothermal atomization atomic absorption spectrometry (ETA-AAS).	Apostoli *et al.* 1989.
	1989	0.31±0.17 µg/L 0.20±0.14 µg/L	-	Heavy smokers Non-smokers	Apostoli *et al*. 1989.
	2002	0.83±0.46 µg/L	0.41–1.79 µg/L	Healthy control subjects. Urine samples digested by microwave. Graphite furnace. hydrid atomic absorption used for quantitative Be measurements. Normal urine beryllium level is <2 µg/L	Horng *et al*. 2002.
	2001	<0.03 µg/L (DL)	-	Control group for occupational exposure study (n=30). Analysed by graphite furnace atomic absorption spectrometry (GFAAS) or inductively-coupled plasma mass spectrometry (ICP-MS)..	Apostoli and Schaller 2001.
	1985	0.6±0.2 µg/L		n=56 non-occupationally exposed Italian subjects. Analysis performed by electrothermal atomization atomic absorption spectrometry (ETA-AAS).	Minoia *et al.* 1985.
	1986	0.126 µg/L	-	Testing of analysis procedure. Freeze-dried urine samples from 2 lyophilized urine pools with “normal” and elevated Be levels and in vitro spiked urine samples (source of urine not stated). Analysis performed by electrothermal atomization atomic absorption spectrometry (ETA-AAS).	Paschal and Bailey 1986
	1977	0.9±0.4 µg/L	0.40-0.90 µg/L	“Normal” subjects (n= 120). Analysed by	Grewal and Kearns 1977.

Tissue/Fluid	Year	Concentration mean	Range	Comment	Reference
				flameless atomic absorption.	
	1998	0.28 µg/L	-	US residents (n=496). Analysed by inductively-coupled plasma mass spectrometry (ICP-MS) and graphite furnace atomic absorption spectrometry (AAS).	Paschal and Bailey 1998.
Colostrum	1998	0.4 µg/L	<0.05-0.6 µg/L	Samples (n=13) from day 1-3 post partum from 46 healthy mothers from Graz, Austria. Analysed by inductively-coupled plasma mass spectrometry (ICP-MS). DL=0.05 µg/L.	Krachler *et al*. 1998.
	1999	0.9±0.5 µg/L	<0.05-1.6 µg/L	Samples (n=27) from day 1-3 post partum from healthy mothers from Graz, Austria. Analysed by inductively-coupled plasma mass spectrometry (ICP-MS). DL=0.05 µg/L.	Krachler *et al.* 1999
Human Breast Milk	2008	0.008±0.003 µg/L	0.003-0.045 µg/L	n=205. Mature milk (mothers with children aged 4 to 80 wks) from United Arab Emirates. Analysed by inductively-coupled plasma mass spectrometry (ICP-MS).	Abdulrazzaq *et al.* 2008.
	1998	0.2 µg/L	<0.05-0.9 µg/L	Samples (n=55) from days 1 to 293 post partum from 46 healthy mothers from Graz, Austria. Analysed by inductively-coupled plasma mass spectrometry (ICP-MS). DL=0.05 µg/L. Concentration higher in colostrum (0.4 µg/L) than mature milk (0.1-0.2 µg/L).	Krachler *et al*. 1998.
Kidney: Liver: Muscle Bone Hair Nail	-	0.2 µg/kg 1.6 µg/kg 0.75 µg/kg 3.0 µg/kg 6.0-20.0 µg/g <10 µg/kg	-	-	Bowen 1979.
Dry lung tissue -Not exposed to any particular dust -Exposed to particular dust	1986	- 0.068 µg/g	0.002-0.03 µg/g -	Samples from thoracic surgery were digested with HNO_3 and analysed by graphite furnace AAS.	Baumgardt *et al*. 1986.
Lung	2008	<0.001 µg/g (dry wet; DL)	-	Samples from lung biopsies of 8 healthy control subjects. Analysed by inductively-coupled plasma mass spectrometry (ICP-MS)	De Palma *et al*. 2008.
Lung tissue	1988	0.005 µg/g (wet	0.002-0.008 µg/g	Samples taken from unexposed, non-	Caroli *et al*. 1988.

Tissue/Fluid	Year	Concentration mean	Range	Comment	Reference
		weight)	(wet weight)	smoking, middle-aged, Italian subjects (n=12) who had died from causes other than respiratory problems. Analysed by inductively-coupled plasma atomic emission spectrometry (ICP-AES)	
Tarragona (Spain) Brain, bone, kidney, liver, and lung	1998	<0.02 µg/g (DL) for all tissues.	-	Samples (n=20; 15 men, 5 women) taken at autopsy from individuals with no metal exposure history. Analysed by inductively-coupled plasma mass spectrometry (ICP-MS). DL=0.02 µg/g.	Llobet *et al*. 1998a.
Hair	1998	<0.04 µg/g (DL)	-	Samples (n=124) from 11-13 yr children from Tarragona, Spain near a hazardous waste incinerator under construction. Analysed by inductively-coupled plasma mass spectrometry (ICP-MS).	Granero *et al.* 1998.
India Teeth Non-carious Carious	-	 12.45±5.92 µg/g 10.90±3.37 µg/g	-	Samples from extracted sound (n=15) and carious (n=15) molars and premolars. Analysed by inductively-coupled plasma mass spectrometry (ICP-MS)	Riyat and Sharma 2009.

Appendix 2. Average Intake Rates of Beryllium via Food Ingestion by Year (µg/kg-d)$^{-1}$

Beryllium (Be)	0-6mo M&F	7mo-4yrs M&F	5-11yrs M&F	12-19yrs M&F	20+yrs M&F
2000	0.12	0.10	0.069	0.045	0.044
2001	0.10	0.075	0.050	0.030	0.026
2002	0.21	0.13	0.087	0.053	0.044
2003	0.015	0.010	0.0059	0.0033	0.0029
2004	0.0077	0.0066	0.0046	0.0028	0.0024
2005	0.0078	0.0046	0.0037	0.0025	0.0021
2006	0.0049	0.0043	0.0039	0.0028	0.0023
2007	0.0047	0.0039	0.0033	0.0022	0.0019
Mean	0.059	0.042	0.028	0.018	0.016
Std. Dev.	0.077	0.052	0.035	0.021	0.019

Notes:
- Reference: Dabeka *et al*. (2010). - Above values were applied to non-breast-fed infants for the purposes of calculating EDIs for infants (birth to 6 months). Breast milk concentrations were used to calculate the EDI for breast-fed infants.

Appendix 3. Typical Environmental Concentrations Used in EDI Calculations

Media	Units	Distribution	Statistics	Beryllium
Drinking Water[1]	µg/L	Lognormal	Arithmetic Mean	0.037
			Standard Deviation	0.074
			z	3
			Minimum	0
			Maximum	1
Outdoor Air[2]	ng/m^3	Lognormal	Arithmetic Mean	0.013
			Standard Deviation	0.011
			z	3
			Minimum	0
			Maximum	0
Indoor Air[3]	ng/m^3	Uniform	Arithmetic Mean	-
			Standard Deviation	-
			z	-
			Minimum	0
			Maximum	0.25 (MDL)
Soil[4]	mg/kg	Lognormal	Arithmetic Mean	0.75
			Standard Deviation	0.99
			z	3
			Minimum	0
			Maximum	9
Settled Dust[5]	mg/kg	Lognormal	Arithmetic Mean	0.56
			Standard Deviation	0.56
			z	3
			Minimum	0
			Maximum	5
Breast Milk[6]	µg/L	Triangular	Arithmetic Mean	0.129
			Standard Deviation	0.304
			z	3
			Minimum	0
			Maximum	3

[1] Based on average beryllium concentrations of drinking water from Ontario (1998-2007) and Saskatchewan (2000-2009) (HC 2011).
[2] Outdoor air $PM_{2.5}$ concentrations NAPS data collected from 2003 to 2009 from British Columbia, Ontario, Quebec and New Brunswick from urban and rural centres (HC 2011).
[3] Indoor air concentrations based on $PM_{2.5}$ from Bell *et al.* (1994). Note all values were below the MDL (0.25 ng/m^3) (HC 2011).
[4] Based on Geological Survey of Canada and New Brunswick Department of Natural Resources data (HC 2011).
[5] Based on arithmetic mean of total beryllium in indoor settled dust from Rasmussen *et al.* 2001 (HC 2011).
[6] Based on beryllium concentration in breastmilk for various literature sources: Krachler *et al*. (1998), Krachler *et al.* (1999) and Abdulrazzaq *et al*. (2008) (HC 2011).

Appendix 4. Receptor Characteristics of the Canadian General Population[1]

	Statistic	Breast fed Infant (0 to 6 mo.)	Non-Breast fed Infant (0 to 6 mo.)	Toddler (7 mo. to 4 yr)	Child (5 to 11 yr)	Teen (12 to 19 yr)	Adult (20+ yr)
Body Weight (kg)	Minimum	2.8	2.8	7.1	14.2	30.0	38.1
	Maximum	21.5	21.5	35.9	71.5	112.2	126.5
	Mean	8.2	8.2	16.5	32.9	59.7	70.7
	Std. dev.	2.9	2.9	4.5	8.9	13.5	14.5
	Distribution	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal
Skin Surface Area Hands (cm^2)	Minimum	242	242	299	396	556	614
	Maximum	416	416	614	863	1142	1262
	Mean	320	320	430	590	800	70.7
	Std. dev.	30	30	50	80	100	14.5
	Distribution	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal
Skin Surface Area Arms (cm^2)	Minimum	200	200	396	797	1409	1588
	Maximum	1367	1367	1882	2645	3465	3906
	Mean	550	550	890	1480	2230	2510
	Std. dev.	180	180	240	300	340	360
	Distribution	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal
Skin Surface Area Legs (cm^2)	Minimum	539	539	907	1604	3042	3753
	Maximum	1496	1496	3012	5655	7945	8694
	Mean	910	910	1690	3070	4970	5720
	Std. dev.	160	160	340	660	810	760
	Distribution	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal
Soil Loading to exposed skin[2] Hands Surfaces other than hands (kg/cm^2/event)	Mean	1.0×10^{-7} 1.0×10^{-8}	1.0×10^{-7} 1.0×10^{-8}	1.0×10^{-7} 1.0×10^{-8}	1.0×10^{-7} 1.0×10^{-8}	1.0×10^{-7} 1.0×10^{-8}	1.0×10^{-7} 1.0×10^{-8}
Time spent[3] outdoors (hr/d)	Minimum	0.000	0.000	0.000	0.000	0.000	0.000
	Maximum	3	3	3	4	9.45	10.76
	Mean/Mode	1.25	1.25	1.25	2.2	1.42	1.43
	Std. dev.	N/A	N/A	N/A	N/A	1.17	1.28
	Distribution	Triangular	Triangular	Triangular	Triangular	Lognormal	Lognormal

[1]Mean receptor characteristics from Richardson (1997) and CCME (2006) unless otherwise stated.
[2]Soil loadings from Kissel *et al.* (1996; 1998) as referenced in CCME (2006).
[3]Time spent outdoors by infant, toddler or child is assumed to be equivalent to that of an adult if child or infant is assumed to be accompanied by an adult.

Appendix 5. Typical Values for Intakes of Air, Water and Soil by the Canadian General Population[1]

Intake rates[1]	*Statistic*	*Breast fed Infant (0 to 6 mo.)*	*Non-Breast fed Infant (0 to 6 mo.)*	*Toddler (7 mo. to 4 yr)*	*Child (5 to 11 yr)*	*Teen (12 to 19 yr)*	Adult (20+ yr)
Air inhalation (m^3/d)	Minimum	1.1	1.1	4.6	8.3	9	9.5
	Maximum	4.4	4.4	15.6	25	28.9	33
	Mean	2.18	2.18	8.31	14.52	15.57	16.57
	Std. dev.	0.59	0.59	2.19	3.38	4.00	4.05
	Distribution	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal
Water Ingestion[2] (L/d)	Minimum	N/A	0.1	0.2	0.2	0.2	0.2
	Maximum	N/A	0.7	0.9	1.1	2	2.7
	Mean	N/A	0.3	0.6	0.8	1	1.5
	Std. dev.	N/A	0.2	0.4	0.4	0.6	0.8
	Distribution	N/A	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal
Soil Ingestion[3] (kg/d)		2.0×10^{-5}	2.0×10^{-5}	8.0×10^{-5}	2.0×10^{-5}	2.0×10^{-5}	2.0×10^{-5}
Soil Inhalation[4] (m^3/d)		1.66×10^{-9}	1.66×10^{-9}	6.32×10^{-9}	1.10×10^{-8}	1.10×10^{-8}	1.26×10^{-8}
Indoor Settled Dust Ingestion (kg/d)	Minimum	8.0×10^{-8}	8.0×10^{-8}	0.00	0.00	0.00	0.00
	Maximum	1.77×10^{-3}	1.77×10^{-3}	9.4×10^{-4}	8.33×10^{-4}	3.39×10^{-5}	6.20×10^{-5}
	Mean	3.74×10^{-5}	3.74×10^{-5}	4.06×10^{-5}	3.17×10^{-5}	2.07×10^{-6}	2.51×10^{-6}
	Std. dev.	8.33×10^{-5}	8.33×10^{-5}	5.22×10^{-5}	4.58×10^{-5}	2.32×10^{-6}	3.06×10^{-6}
	Distribution	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal
Food[5]	Minimum	0.5	5.590	0.000	0.000	0.000	0.000
	Maximum	1	19.475	23.981	17.744	10.667	8.323
	Mean/Mode	0.7	12.533	11.142	8.148	4.956	3.945
	Std. dev.	N/A	2.314	4.280	3.199	3.945	1.459
	Distribution	Triangular	Lognormal	Lognormal	Lognormal	Lognormal	Lognormal

[1] Probability distribution function curves for receptor intake rates from Health Canada (2011) unless otherwise stated.
[2] Breast fed infants are assumed to be exclusively breastfed for 6 months and are not given drinking water.
Infants that are not breastfed are assumed to consume 0.3L of drinking water based on HC (2004).
[3] Soil ingestion rates from CCME (2006)
[4] Soil inhalation rates based on Allan *et al*. (2008) and a PM10 concentration of 0.76 µg/m3 (CCME 2006)
[5] Breastfed infants are assumed to be exclusively breast fed for 6 months and non-breastfed infants are assumed to be fed a mixture of milk, formula and table food.

Appendix 6. Estimated Total Daily Beryllium Intake by Age Class for the Canadian General Population[1]

Medium of exposure	Daily Beryllium Intake (μg/kg bw/day)					
	BF-Infant (0-6 mo)	NBF-Infant (0-6 mo)	Toddler (7mo- 4yr)	Child (5-11 yr)	Teen (12-19 yr)	Adult (20 yr+)
AIR						
Ambient Air (Inhalation)	0.00000014	0.00000014	0.00000026	0.00000035	0.000000011	0.00000010
Indoor Air (Inhalation)	0.000030	0.000030	0.000056	0.000048	0.000029	0.000026
DRINKING WATER						
Drinking Water (Ingestion)	NA	0.00051	0.00045	0.00032	0.00023	0.00029
INDOOR SETTLED DUST						
Settled Dust (Ingestion)	0.00078	0.00078	0.00063	0.00022	0.0000095	0.0000090
Settled Dust (Dermal)	0.00024	0.00024	0.00017	0.00013	0.000069	0.000065
SOIL						
Soil (Ingestion)	0.0012	0.0012	0.0023	0.00029	0.00016	0.00013
Soil (Inhalation)	0.0000000050	0.0000000050	0.0000000092	0.000000012	0.0000000041	0.0000000036
Soil (Dermal)	0.00027	0.00027	0.00019	0.00015	0.000079	0.000074
FOOD						
Food (Ingestion)	0.0047	0.042	0.026	0.018	0.011	0.0098
TOTAL						
TOTAL EDI*	0.0078	0.046	0.032	0.019	0.012	0.011

[1] Median value of estimated daily intake values for each age class were modelled based on receptor characteristics details listed in Appendix 4 and 5 and the probability distribution functions of typical concentrations of air (indoor and outdoor), drinking water, indoor settled dust, soil and food based on details in Appendix 3. The probabilistic modelling of the EDI was completed as described in Health Canada (2011). The median value (50th percentile) was chosen to represent the EDI values for the Canadian population.

* Since a probabilistic method was used to develop the EDIs, the total EDI is not the sum of all sub-EDIs for each age category. The total EDI and each sub-EDI have individual probability distribution functions. The 50th percentile (median) for each distribution is displayed in the above table.

Appendix 7. Typical Values for Average Body Weights and Intakes of Air, Water and Soil by the Canadian General Population used in SQG Calculation

Age (years)	Body weight[1] (kg)	Air intake[2] (m^3/d)	Water intake[1] (L/d)	Soil intake[1] (g/d)	Soil inhalation[3,4] (g/d)	Settled indoor dust ingestion[5] (g/d)
0-6 months	8.2	2.2	0.3	0.02	0.0000017	0.037
7 months – 4 yrs	16.5	8.3	0.6	0.08	0.0000063	0.041
5-11	32.9	14.5	0.8	0.02	0.000011	0.032
12-19	59.7	15.6	1.0	0.02	0.000012	0.0021
20+	70.7	16.6	1.5	0.02	0.000013	0.0025

[1] Health Canada (2010a) and CCME (2006).
[2] Allan *et al.* (2008; 2009).
[3] Health Canada (2010a).
[4] Air intake (m^3/d) x average airborne concentration of respirable particulate (0.00076 g/m^3)]
[5] Wilson *et al.* (2012).