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# Proposed Soil Quality Guidelines
# Cobalt
# Environmental and Human Health Effects

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## Table of Contents

**Page**

## List of Tables

**Page**



## List of Figures

## List of Appendices



## Distribution List

| | |
|---|---|
| Client | 1 copy(ies) |
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# 1.0 INTRODUCTION

Soil quality guidelines are numerical soil concentrations intended to be protective of human and environmental health for current and potential future land uses. They are frequently used for the assessment and remediation of contaminated sites.

The Canadian Council of Ministers of the Environment (CCME) published "A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines" (CCME, 2006) to provide a framework and methodology for developing risk-based soil quality guidelines protective of human health and the environment. The guidelines developed using this protocol have been published in the Canadian Soil Quality Guidelines (CCME, 1999) and also formed the basis for some provincial guidelines, including the Alberta Tier 1 Soil and Groundwater Remediation Guidelines (AEP, 2016). The current Alberta guidelines for cobalt, however, date back to the earlier Interim Canadian Environmental Quality Criteria for Contaminated Sites (CCME, 1991), which did not use modern risk-based approaches.

This report provides the basis for proposed soil quality guidelines for cobalt based on the CCME (2006) protocol. It includes a review of sources of cobalt, concentrations in the environment, fate and behaviour, and toxicological effects on soil microorganisms, plants, animals and humans. Guidelines are derived for the agricultural, residential/parkland, commercial and industrial land uses as defined by CCME (2006), as well as the natural area land use defined by AEP (2016).

# 2.0 BACKGROUND INFORMATION

## 2.1 Physical and Chemical Properties

Cobalt (Co; CAS #7440-48-4), is a hard, silvery grey ferromagnetic metal that is typically present in the environment in combination with oxygen, sulfur, and arsenic (ATSDR, 2004). Cobalt is a Group IX transition element with an atomic number of 27, an atomic weight of 58.93, a melting point of 1495°C, a boiling point of 2870°C, and a specific density of 8.9 g/cm$^3$ at 20°C (ATSDR, 2004). There is one stable isotope, cobalt-59, and there are two commercially important unstable isotopes, cobalt-57 and cobalt-60 (ATSDR, 2004). Physical and chemical properties of cobalt and some cobalt compounds are presented in Table 1.

The most common oxidations states for cobalt are 0, +2, and +3, with Co(II) being the most stable form. Other oxidation states of -1, +1, +4, and +5 are also possible but are not commonly encountered (Cotton and Wilkinson, 1980, from ATSDR, 2004). Some minerals containing cobalt include: linnaeite, carrolite, safflorite, skutterudite, erythrite, and glaucodot (ATSDR, 2004). Cobalt is an essential element and is present in vitamin $B_{12}$.

| Table 1 Physical and Chemical Properties of Cobalt and Select Cobalt Compounds | | | | | | |
|---|---|---|---|---|---|---|
| Chemical formula | Co | $CoCO_3$ | $Co_2(CO)_8$ | $CoCl_2$ | CoO | $CoSo_4$ |
| CAS Registry Number | 7440-48-4 | 513-79-10 | 10210-68-1 | 7646-79-9 | 21041-93-0 | 10124-43-3 |
| Molecular weight (g/mol) | 58.93 | 118.94 | 341.9 | 129.84 | 74.93 | 154.99 |
| Physical state at 25°C | Solid | Solid | Solid | Solid | Solid | Solid |
| Melting point (°C) | 1495 | Decomposes | 51 | 724 | 1795 | 735 |
| Boiling point (°C) | 2870 | NA | Decomposes | 1049 | - | Decomposes |
| Density ($g/cm^3$) | 8.9 (20°C) | 4.13 | 1.73 | 3.356 | 6.45 | 3.71 |
| Water solubility (g/100mL) | Insoluble | 0.18 | Insoluble | 45 | Insoluble | 3.83 |

a – values from ATSDR (2004)

### 2.2 Analytical Methods

Cobalt in environmental media is most commonly analyzed using inductively coupled plasma-optical emission spectrometry (ICP-OES) or inductively coupled plasma-mass spectrometry (ICP-MS), although atomic absorption spectrophotometry (AAS) can also be used (CCME, 2013).

The first step in both ICP-OES and IOCP-MS is conversion of an analyte solution into ions by passing it through a plasma source. ICP-OES measures the emission of light from the heated ions, which occur at a specific wavelength and which has an intensity correlated to the concentration in the original analyte solution. ICP-MS directs these ions into a magnetic field, which deflects their path based on their mass to charge ratio, and onto a detector. This allows for identification and quantification of chemical species in the original analyte solution (Harris, 2003)

AAS involves conversion of an analyte solution into a gaseous state within a flame or furnace. A light source of specified wavelength is then directed through the flame, and the concentration of the analyte is determined by the absorption of the light source (Harris, 2003).

Typical soil sample preparation techniques are intended to indicate the environmentally available concentration of cobalt, using strong acid leachate (CCME, 2013). Water samples are field-filtered and preserved in solutions with pH values less than 2 (CCME, 2013). Relevant US EPA methods for extraction of cobalt include method 3005A *Acid Digestion of Waters for Total Recoverable or Dissolved*

*Metals for Analysis by FLAA or ICP Spectroscopy* (US EPA ,1992), and method 3050B *Acid Digestion of Sediments, Sludges, and Soils* (US EPA, 1996).

The US EPA recommends analysis using either FLAA/ICP-AES or GFAA/ICP-MS using either method 6010C *Inductively Coupled Plasma-Atomic Emission Spectrometry* (US EPA, 2007a) and method 6020a *Inductively Coupled Plasma-Mass Spectrometry* (USEPA, 2007b).

CCME (1993) previously recommended method 3120B *Inductively Coupled Plasma (ICP) Method* (CCME, 1993) for determination of metals in water samples, and US EPA method 6010 for determination from metals in soils, sludges, sediments. Current CCME (2013) recommendations are for ICP-OES, ICP-MS, or AAS with no standard methodology recommended, but with a requirement that analytical standards be matrix matched to samples.

### 2.3 Production and Uses in Canada

Cobalt is typically generated as a by-product from nickel and copper ores (Harper *et al*., 2011), and is produced in Canada from nickel mining operations located in Ontario and Quebec. Total Canadian production of cobalt in 2012 was estimated at 3652 tonnes, which also includes recovered cobalt (Natural Resources Canada, 2013). The primary end users of cobalt, as of 2005, were China, Japan, and the United States (Harper *et al*., 2011), and it was estimated by Dewulf *et al*. (2010) that battery applications made up 25% of the worldwide cobalt demand. Historically, Canada has supplied a large amount of the United States demand for cobalt, and 97 tons of unrefined cobalt were exported to the US in 2010 (USGS, 2010).

Cobalt has several industrial uses, including: industrial alloys, catalysts, enamels, and pigments, (Lauwerys and Lison, 1994), and total domestic use of cobalt in Canada as of 2007 was estimated at 69.547 tonnes (Natural Resources Canada, 2008). Releases of cobalt and cobalt compounds were reported from 106 facilities in Canada during 2011, with 30 tonnes of on-site releases and 4994 tonnes of cobalt disposed of at offsite facilities (NPRI, 2012). Cobalt containing substances have been identified as priorities for action as part of the second phase of the Federal Chemicals Management Plan and end use products containing cobalt are primarily industrial in nature (ATSDR, 2004).

Cobalt alloys are utilized due to corrosion resistance, magnetic, or high strength properties (Lauwerys and Lison, 1994), and cobalt-chromium alloys are used in prosthetic devices (Gibson and Stamm, 2002). Cobalt-60 is used as a therapeutic radiation source, disinfectant for medical equipment, and to test vitamin $B_{12}$ absorption (Larrson, 2004), and is produced from elemental cobalt-59 in nuclear reactors. Nordion is the major Canadian supplier of cobalt-60 for gamma sterilization, produced primarily at the National Research Universal reactor in Ontario, which exports worldwide.

## 2.4 Sources and Concentrations in the Canadian Environment

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

Relatively high concentrations of metals can occur naturally in Canadian soils, stream sediments, and water, blurring the distinction between anthropogenic pollution versus naturally occurring geological formations and natural bodies of ore. Natural sources of cobalt include wind-blown continental dusts, weathering of rocks, seawater spray, forest fires and volcanoes (Kim et al., 2006). Areas of rich cobalt deposits in Canada include the Sudbury district, Northern Quebec and Thompson Manitoba (Environment Canada, 2011) and generally occur with nickel and copper deposits (Kim et al., 2006). Soils and sediments reflect the composition of parent material, resulting in higher metal concentrations in mineralized areas (Wilson et al., 1998). Mining districts are characterized by naturally occurring metals in soil, sediment, rock, and water at concentrations that could result in their classification as "contaminated sites" (Painter et al., 1994). Anthropogenic sources of cobalt include burning of fossil fuels, wastewater biosolids, phosphate fertilizers, mining and smelting of cobalt ores and industrial processes that use cobalt compounds (Kim et al., 2006). In the determination of anthropogenic metal contamination of soils, no single guideline concentration can adequately represent the variance in background concentrations across Canada (Painter et al., 1994; Chapman and Wang, 2000).

Data on concentrations in environmental media are summarized in Appendix A.

### 2.4.1 Atmosphere

Natural cobalt emissions to the atmosphere have been estimated to range between 690 and 11,000 tonnes per year globally (Nriagu, 1989). Atmospheric cobalt concentrations in Canada were monitored through the federal National Air Pollution Surveillance (NAPS) over a two year period from May 2004 to December 2006. Cobalt concentrations in fine particulate matter ($PM_{2.5}$) at seven sites in eastern and western Canada were less than 0.1 ng/m$^3$ (Celo and Dabek-Zlotorzynska, 2010).

The airborne dispersal of cobalt was measured near smelters in the Sudbury area (Hutchinson and Whitby, 1977). Cobalt concentration in rainfall-dustfall (up to 8.7 mg/m$^2$ in 28 days) was greater than cobalt concentration in dustfall collected during no precipitation (up to 0.6 mg/m$^2$ in 14 days). In dry periods the particulates were likely more widely dispersed over a greater area. Airborne cobalt concentrations generally decreased with distance from the smelter.

Seasonal chemical composition of fine particulate matter was measured at rural locations in Alberta (Cheng et al., 2000). Cobalt concentrations of $PM_{2.5}$ at Ester and Swan Hills showed little seasonal variation and were approximately 0.02 (Swan Hills) and 0.2 (Ester) ng/$m^3$.

### 2.4.2 Soil and Dust

Cobalt is a naturally occurring element in the terrestrial crust. Cobalt concentrations in the upper terrestrial crust range between 0.1 and 110 mg/kg (Reinmann and de Caritat, 1998). Naturally occurring cobalt exists in various mineral, oxide and salt forms. The highest concentrations of cobalt are found in ultramafic (100-200 mg/kg) and mafic (35-50 mg/kg) rocks with a substantial amount being present in organic-rich shales (14-20 mg/kg). It is present in soil as $Co^{2+}$ and $Co^{3+}$ and possibly $Co(OH)^{3-}$ (Kebata-Pendias and Pendias, 1992). The main form in solution is $Co^{2+}$ (McBride, 1994), although the total amount in solution is low. World-wide levels for soil cobalt concentrations have been reported to be between 0.1 to 70 mg/kg (Kabata-Pendias and Pendias, 1992), with a mean concentration of 8 mg/kg (Tisdale et al., 1985).

A comparison of total elemental concentrations in garden soil, house dust and street dust in the city of Ottawa, ON, reported geometric means for cobalt of 8.18 mg/kg for garden soil, 8.40 mg/kg for house dust and 8.02 mg/kg for street dust (Rasmussen et al., 2001).

Average concentrations of 18 to 34 mg/kg were reported for cobalt in soil samples from different geographic regions in Canada (McKeague and Wolynetz, 1980). The $A_e$ horizon was found to be depleted in cobalt relative to the C horizon, especially in the Appalachian region. A high correlation was noted between cobalt concentrations and clay, iron, aluminum, manganese, copper nickel and chromium.

The cobalt content of Manitoba soils ranged from 2 to 37 mg/kg, with a median concentration of 12 mg/kg (Haluschak et al., 1998). The geographic distribution of cobalt in southern Manitoba soils was not strongly influenced by the lithological origin or nature of the soil parent material but appeared to be more strongly related to the texture of the soil materials. The lowest concentrations for cobalt were found in coarse textured soils (mean of 8 mg/kg). Cobalt concentrations gradually increased with increasing clay content, with mean values of 19 mg/kg in fine textured soils.

Data from soil samples from the Alberta Environmentally Sustainable Agriculture Soil Quality Resource Monitoring Program benchmark sites showed mean cobalt values for seven Canadian ecoregions ranged from 0.034 to 0.0.847 mg/kg at the 0 to 15 cm depth and from 0.019 to 0.0.419 mg/kg at the 15 to 30 cm depth (Penney, 2004). Significant differences were noted for cobalt based on slope position, with the highest values occurring in the lowest slope position.

Rencz et al. (2006) compiled geochemistry data from the published, digital data set of the Geological Survey of Canada. The data indicated a provisional natural background range of 1 to 95 mg/kg for cobalt concentrations across Canada in till samples prepared from less than 63 μm size fractions. An addendum report issued in 2012 indicated that the highest cobalt concentrations occurred in central BC and the western part of the till survey in Labrador (Grunsky, 2012). Median concentration for samples prepared using total/near total digestion techniques was 10 mg/kg with 50% of all values ranging between 6 and 15 mg/kg, as reported by the addendum report.

### 2.4.3 Water

Finished domestic water supplies usually have cobalt concentrations below detection limits, with rivers, lakes, spring water, groundwater and well water containing levels up to a few μg/L (Hamilton, 1994). Aquatic environments contaminated with effluents originating from mine sites or mineral rich areas may have much higher levels (Nagpal, 2004).

A survey of US lakes and rivers in 1970 from surface water sources of public water supplies, water courses downstream of major municipal or industrial complexes and US Geological Survey benchmark stations, found 63% of all samples had cobalt concentrations below the detection limit (Durum and Hem, 1972). The majority of detected samples were in the range of 1 to 5 μg/L.

Maximum cobalt concentrations reported for Canadian freshwater by Durum and Haffty (1961) were 1.9 μg/L in the Fraser River at Mission City in B.C., 4.0 μg/L in the St. Lawrence River in Quebec, 5.0 μg/L in the Mackenzie River in the Northwest Territories and 5.1 μg/L in the Nelson River in Manitoba.

Background concentrations of cobalt in Ontario were summarized by the Sara Group (2008). Background concentrations ranged from below the detection limit to 25 μg/L in lake water, from below the detection limit to 7.8 μg/L in river water, from below the detection limit to 8.2 μg/L in groundwater and from below the detection limit to 22 μg/L in drinking water.

AMEC Earth and Environmental assessed surface and sediment water quality in parts of Alberta and British Columbia as part of the Enbridge Northern Gateway Project (AMEC, 2010). Median concentrations measured in surface water across six hydrological zones ranged from <0.1 to 0.2 μg/L for total cobalt and from <0.05 to 0.1 μg/L for dissolved cobalt.

BC MWLAP reported mean cobalt concentrations in groundwater in British Columbia to be 21.1 μg/L, with lower and upper 95% confidence limits of 14.7 μg/L and 27.6 μg/L (Nagpal, 2004).

Median dissolved cobalt concentrations measured in the Great Lakes between 1980 and 1985 ranged between 0.0058 μg/L to 0.089 μg/L (Rossman and Barres, 1988).

### 2.4.4 Sediments

Unpolluted freshwater sediment generally contains cobalt concentrations of less than 20 μg/g (Kim et al., 2006). Cobalt concentrations up to 700 μg/g have been measured in surficial sediment near industrial plants.

Cobalt concentrations in sediment core samples from the Upper St. Lawrence River estuary ranged between 2 to 4 μg/g (Coakley et al., 1993). Metal concentrations in the sediment were independent of depth, implying the lack of significant anthropogenic releases in the area.

Surface and core sediment samples from the mouths of watercourses flowing into the Great Lakes system showed low cobalt concentrations in areas of low anthropogenic activity and ranged from 1.2 to 18.0 μg/g in Lake Superior and from 2.6 to 16.5 μg/g in Lake Huron (Fitchko and Hutchinson, 1975). Elevated levels of metals occurred in the outlets of most watercourses with urban-industrial activities, with a maximum cobalt concentration of 43.4 μg/g in the sediment of the Moira River, a tributary of Lake Ontario.

As part of the Enbridge Northern Gateway Project, AMEC Earth and Environmental performed a baseline assessment of surface and sediment water quality in parts of Alberta and British Columbia (Touchinski, et al., 2010). Cobalt concentrations measured in sediment from creeks, rivers and lakes in five hydrological zones ranged from 2 to 30 μg/g.

Three sediment samples from background areas in Grand Lake, New Brunswick and two from East River, Nova Scotia showed median cobalt concentrations of 52.6 and 11 mg/kg, respectively (Lalonde, et al., 2011).

### 2.4.5 Aquatic Organisms

A study with trout from Cayuga Lake in Ontario showed cobalt concentrations ranging from 43 to 81 μg per kg fresh weight in fish that were between one and twelve years old (Tong et al., 1974).

Lalonde et al. (2011) reported cobalt concentrations of 0.009 to 0.1 g/g (wet weight) for fish from Grand Lake, New Brunswick and 0.005 to 0.07 g/g (wet weight) for fish from East River, Nova Scotia.

Maximum cobalt concentration in the muscle of bluefin tuna caught near Newfoundland was 0.01 μg/g of dry weight (Hellou et al., 1992).

### 2.4.6 Plants

Metal and PCB concentrations in the leaves, stems and root tissue of vascular plants from remote locations in the Canadian Artic were compared to those from background areas near military radar sites (Dushenko et al., 1996). The mean cobalt concentration for plants in remote locations was 3.1

mg/kg of dry weight and did not vary significantly from the mean cobalt concentration of plants at background locations near the sites (2.8 mg/kg).

As part of an environmental monitoring program in the Sudbury region, metal concentrations were evaluated for Jack Pine needles from two background sites located north of the city (Gratton et al., 2000). Mean cobalt concentrations in the Jack Pine needles were calculated for values above the detection limit and were 0.62 and 0.18 mg/kg of dry weight for the background sites.

Heavy metal concentrations were determined in the moss *Sphagnum magellanicum* in the Maritime Provinces (Percy, 1983). Cobalt concentrations ranged from less than the detection limit (<0.20 mg/kg) to 1.90 mg/kg. A significant correlation between iron and cobalt concentrations was determined to be present.

### 2.4.7 Animals

Cobalt is an essential trace element for ruminants and horses and is required for the synthesis of vitamin $B_{12}$ from microbial action in the digestive tract. Non-ruminants lack the ability to synthesize the vitamin in significant amounts and require the intake of vitamin $B_{12}$.

Frank et al., 1986 compared cobalt levels in slaughtered bovine, porcine and avian specimens in Ontario. Mean cobalt concentrations ranged from 30 to 48 µg per kg (wet weight) in bovine muscle and from 63 to 86 µg/kg in bovine kidney. Porcine muscle and kidney contained an average cobalt concentration of 50 and 171 µg/kg, respectively. Avian muscle and liver contained an average of 21 to 25 µg/kg and 50 to 60 µg/kg of cobalt, respectively. Approximately two to three fold higher levels of cobalt were noted in the kidney compared to the muscle of the animals.

### 2.4.8 Humans

The major source of cobalt for most people is from their diet. The estimated daily intake of cobalt by Canadian adults between 20 to 59 years is 0.23 µg/kg of body weight per day (Environment Canada, 2011).

Cobalt concentration in human sera from healthy individuals was determined using adsorptive stripping voltametry and ranged from 0.07 to 0.17 µg/L, consistent with other studies (Kajic et al., 2003).

Analysis of NHANES survey data for the years 2003 to 2010 found a significant difference between cobalt levels of pregnant and non-pregnant females in urine; with a geometric mean of 0.585 µg/L in pregnant females compared to a geometric mean of 0.442 µg/L in non-pregnant females (Jain, 2013).

The geometric mean for trace element concentrations in non-smoking adults (aged 33 to 64) from the west coast of Canada was reported to be 8.69 nmol/L in human serum (Clark et al., 2007). Smokers did not have significantly different levels of cobalt compared to non-smokers (Jain, 2013).

Cobalt concentration in the blood and urine of participants aged 3 to 79 years was measured in the Canadian Health Measures Survey in Cycle 2, from 2009 to 2011 (Health Canada, 2013a). The geometric mean for cobalt concentration was 0.23 µg/L in whole blood and 0.23 µg/L in the urine of the study group

### 2.5 Existing Soil and Water Quality Criteria and Guidelines

Soil and water quality criteria and guidelines for cobalt have been developed by several agencies, and are summarized in Appendix B. The current Canadian guidelines (40 mg/kg for agricultural, 50 mg/kg for residential/parkland, 300 mg/kg for commercial and industrial) are from the CCME (1991) interim guidelines and were not derived using current risk-based approaches.

## 3.0 ENVIRONMENTAL FATE AND BEHAVIOUR IN SOIL

In the natural environment, cobalt is found primarily as a trace element in iron oxide, sulphide, arsenide, and sulfo-arsenide minerals (Hamilton, 1994), and the most common minerals incorporating cobalt directly are cobaltite (CoAsS), linnaet ($CO_3S_4$), smaltyn ($CoAs_2$) and karrolit ($CuCo_2S_4$) (Baralkiewicz and Siepak, 1999). Cobalt is not degraded in the environment, and its fate is dependent on a series of physiochemical and biological factors that influence cycling among biotic and abiotic components of the environment.

### 3.1 Atmosphere

Cobalt does not volatilize except under extreme conditions, and emissions to the atmosphere are primarily industrial in nature (Merian, 1984), such as mining and smelting activities. Other than emissions from industrial activities, cobalt is present in the atmosphere primarily from resuspension of soil particles through volcanic eruptions (Hamilton, 1994). Cobalt has a short atmospheric life cycle and rapidly ends up in aquatic and terrestrial environments (Merian, 1984).

### 3.2 Water

Between pH values of three and nine, cobalt is typically present in the dissolved phase (Hamilton, 1994), which is bioavailable (Hamilton, 1994). Cobalt is readily precipitated from solution in its +3 oxidation state (Baralkiewicz and Siepak, 1999); however, pH, complexation by organic ligands and adsorption to particles will affect cobalt speciation. Cobalt 3+ ions can replace or substitute for manganese and iron and be removed from solution (Burns, 1976).

### 3.3 Soil

Cobalt in soil is typically in the +2 oxidation state, and is often bound to manganese oxide deposits (Burns, 1976). Cobalt is more soluble and bioavailable under reductive soil conditions (Baralkiewicz and Siepak, 1999), and is an essential element for nitrogen fixation by bacteria (Collins and Kinsela, 2010). Metallic oxide materials sorb the greatest amount of cobalt, with relatively little cobalt sorbed onto clay or organic minerals (McLaren et al., 1986). Cobalt is released from soil through weathering.

### 3.4 Biota

Cobalt is an essential element and its bioavailability is influenced by pH, organic content, oxidation-reduction state, organic complexation, and co-precipitation processes (Hamilton, 1994). Poorly drained soils tend to contain the highest concentrations of extractable cobalt (Hamilton, 1994); however, the uptake of cobalt is influenced by the presence of manganese (Hamilton, 1994).

Within plants, cobalt is primarily in the +2 oxidation state (Merian, 1984), and in animals cobalt accumulates primarily in the liver, kidney, pancreas, and heart (Simonsen, 2012).

## 4.0 BEHAVIOUR AND EFFECTS IN TERRESTRIAL BIOTA

The available information on the toxicological effects of cobalt on soil microbial processes, terrestrial plants, invertebrates, as well as mammals and birds have been reviewed and summarized below. Detailed data are provided in Appendix C.

Plants and animals may accumulate contaminants over time if the amount to which they are directly exposed is greater than the amount they can eliminate through excretion and metabolic processes. The process by which contaminants are directly taken up by an organism from the exposure medium (e.g. soil) is referred to as bioconcentration. The process of contaminant uptake occurring through direct uptake as well as ingestion at a rate faster than it is metabolized or excreted is referred to as bioaccumulation (CCME 2006).

### 4.1 Terrestrial Plants

#### 4.1.1 Uptake, Metabolism, and Elimination

Cobalt is a required element for nitrogen-fixing microorganisms that are associated with legumes; however, it had not been demonstrated to be essential for higher plants. Gad (2012) showed cobalt concentrations of 8 mg/kg had a positive effect on root nodulation and plant growth when accompanied by nitrogen treatments, compared to nitrogen treatment alone. Cobalt concentrations up to 50 mg/kg have shown positive effects on the growth of soybeans (Jayakumar et al., 2009). Numerous soil factors have been noted to influence the uptake of cobalt by plants, including soil pH, other soil chemical parameters (*e.g.* manganese and organic carbon), the form and concentration of cobalt in the soil and variations in microbial populations. Although significant correlations have been

reported between these factors and plant uptake of cobalt, they do not appear to be universally applicable (Collins and Kinsela, 2011). Generally, an increase in aqueous cobalt concentration is expected to result in increased plant uptake of cobalt; however, this may be further confounded by cobalt complexation to plant- or microbially-derived organic ligands. Cobalt uptake by plants appears to be by active transport processes, with the majority of cobalt being retained in the roots (Palit et al., 1994). Excess cobalt in the soil increased its translocation from the roots to shoots of cauliflower (Chatterjee and Chatterjee, 2000).

### 4.1.2 Bioaccumulation

Plants can generally accumulate small amounts of cobalt from the soil, with the uptake and distribution being species specific and may be controlled by different mechanisms (Nagajyoti, et al., 2010). Specimens of *Haumaniastrum robertii* collected from heavy metal mineralized areas of Zaire showed elevated levels of cobalt in their leaves, with a mean concentration of 4303 µg/g of dry weight (Brooks, 1977). Approximately 2/3 of $^{60}CO^{2+}$ ions taken up from enriched Hoagland's nutrient solution accumulated in tobacco roots, with 1/3 being transported to the shoots. Autoradiography showed that cobalt was preferentially accumulated in the younger leaves of the plants (Vrtoch et al., 2007). Soybean plants showed a significant accumulation of cobalt in all parts of the plant at soil concentrations of 100 to 200 mg/kg (Jayakumar and Jaleel, 2009).

### 4.1.3 Toxicity

Although low concentrations of cobalt has a positive effect on plant growth, in non-vascular and higher plants, the uptake of excess cobalt can result in plant toxicity (Palit et al., 1994). High concentrations of cobalt in the roots and leaves may disrupt a range of metabolic processes due to competitive processes with essential micronutrient cations (Liu et al., 2000).

Excess supply of cobalt caused its accumulation in barley shoots and, especially, in roots and resulted in a decrease in dry matter yield (Agarwala, et al., 1976). Excess cobalt reduced iron absorption by plants, reduced iron translocation to shoots, induced chlorosis and decreased the activity of catalase, an iron containing enzyme, in young leaves.

Excess $Co^{2+}$ also caused chlorosis in sugar beets, tomato, potato, oat, kale, bush beans and cabbage and caused a depression of growth in these plants (Hewitt, 1953; Wallace et al., 1977; Pandey et al., 2002).

Elevated concentrations of cobalt in the growth medium caused the tissue concentrations of mung bean plants to increase and decreased concentrations of chlorophylls and carotenoids (Tewari et al., 2002). An increase in the activities of anti-oxidative enzymes was also observed suggesting induction of oxidative stress due to excess cobalt concentrations in the growth medium. A decrease in the activity of catalase was observed above 50 uM concentrations of $CoSO_4$.

$CoSO_4$ treatment of cauliflower grown in refined sand decreased plant biomass, iron, chlorophyll a and b concentrations, catalase protein and activity in leaves (Chatterjee and Chatterjee, 2000).

Physiological changes in unifoliate white bean leaves in response to excess cobalt included a decrease in dry matter yield, abnormal starch accumulation, a shift towards relatively non-polar phenolic compounds and elicitation and maintenance of altered leaf orientations (Rauser, 1978).

Upper critical plant tissue concentrations of cobalt were examined through a survey of published work (Macnicol and Beckett, 1985). The upper critical levels for barley and ryegrass in soil were reported as 20 to 25 and 40 mg/kg dry matter, respectively. The upper critical levels for barley, cabbage, bush bean and ryegrass in solution were reported to range from 6 to 40 mg/kg dry matter.

The effective concentration of $CoCl_2$ causing a 50% inhibition ($EC_{50}$) to barley root growth ranged from 45 to 863 mg/kg for different soil types (Mico et al., 2008). Soil effective cation exchange capacity and exchangeable calcium were the most consistent predictors of the $EC_{50}$ values for excess cobalt added to the soil. This suggests that sorption of cobalt to the negatively charged sites on the soil has a large influence of cobalt toxicity to barley root growth.

Li et al. (2009) evaluated the phytotoxicity and bioavailability of $CoCl_2$ for barley, oilseed rape and tomato shoot growth in ten soil types. Soil properties greatly influenced the expression of cobalt toxicity by plants. The $EC_{50}$ values ranged from 40 to 1708 mg/kg for barley, from 7 to 966 mg/kg for oilseed rape and from 7 to 733 mg/kg for tomato. The variation was substantially reduced and was between 4 to 15-fold when the toxicity thresholds were based on cobalt concentrations in the soil solution. The $EC_{10}$ and $EC_{50}$ values were predicted most consistently using the regression models based on exchangeable calcium. The study concluded that solubility of cobalt is the key factor influencing its toxicity to plants and cobalt toxicity threshold values for plants should be normalized using soil exchangeable calcium.

Phytotoxicity tests to set ecological screening levels using two soil matrices and three plant species determined a geometric mean for the $EC_{20}$ of 30.6 mg/kg for $CoCl_2$, excluding emergence, mortality and nodule numbers (Kapustka et al., 2006).

Environment Canada (2010) reported the phytotoxicty of $CoSO_4$ for northern wheatgrass, radish, red clover and tomato in terms of emergence, shoot length and weight and root length and weight in 14 or 21 day exposure studies. The emergence $EC_{10}$ and $EC_{50}$ values for red clover were 287 and 865 mg Co/kg dw soil, respectively, and greater than 1210 mg Co/kg dw for both $EC_{10}$ and $EC_{50}$ for the other three plant species. The reaming results can be summarized as follows:

- Shoot length: $IC_{10}$ and $IC_{50}$ values ranging between 98 and 189 mg Co/kg dw soil and 488 and 1364 mg Co/kg dw soil, respectively, for the four test species;
- Root length: $IC_{10}$ and $IC_{50}$ values ranging between 22.2 and 267mg Co/kg dw soil and 163 and 561 mg Co/kg dw soil, respectively, for the four test species;

- Shoot weight: $IC_{10}$ and $IC_{50}$ values ranging between 426 and 524 mg Co/kg dw soil and 783 and 2213 mg Co/kg dw soil, respectively, for the four test species;

- Root weight: $IC_{10}$ and $IC_{50}$ values ranging between 11.9 and 407 mg Co/kg dw soil and 69.5 and 741 mg Co/kg dw soil, respectively, for the four test species;

Oorts et al. (2011) acknowledged soil properties strongly affect the toxicity of cobalt to soil organisms and was best correlated with the effective cation exchange capacity of the soil. The authors report plant $EC_{10}$ values ranging between 2.9 and 617 mg Co/kg soil based on a cumulative seven species and 72 reliable data end points. Data is noted to be a compilation of multiple studies. When corrected for differences in biovailabilty between lab and field soil (leaching – ageing) and considering a background cobalt concentration the authors calculated mean normalized plant $EC_{10}$ values (using CEC slopes) to range between 25.1 and 166 mg Co/kg soil.

## 4.2 Terrestrial Invertebrates

### 4.2.1 Uptake, Metabolism, and Elimination

$^{60}Co$ added to the food source of the earthworm, *Eisenia foetida* was retained with a half-life of 387 days (Neuhauser et al., 1984). After 172 days, higher cobalt concentrations were measured in the gut than in the body wall. $^{60}Co$ was not transmitted from adults to cocoons. Earthworms fed $CoCl_2$ concentrations of up to 25.9 μg cobalt per gram in a food source showed increased maximum weights and produced more cocoons compared to controls.

### 4.2.2 Bioaccumulation

$CoCl_2$ concentrations of 1000 to 5000 μg/g added to a food source of the woodlice, *Porcellio scaber*, showed the accumulation of $Co^{2+}$ ions in the hepatopancreas (Novak et al., 2013).

### 4.2.3 Toxicity

Fecal production used as an indicator of food consumption was reduced in woodlice exposed to 2500 μg of cobalt per gram of food. The decreased consumption rates may have been due to feeding deterrence rather than toxicity as none of the organisms died (Drobne and Hopkin, 1994).

The 28-day $EC_{50}$ for the reproduction of springtail (*Folsomia candida*) was determined to be 1480 mg of cobalt per kg of dry weight in standard artificial soil and 409 mg of cobalt per kg of dry weight in standard field soil (Lock et al., 2004). The lower cobalt toxicity in artificial soil was correlated to decreased bioavailability due to higher pH and cation exchange capacity. The 28-day $EC_{50}$ values were similar when expressed as pore water concentrations and were 159 mg of cobalt per litre for artificial soil and 174 mg cobalt per litre for field soil.

He et al. (2015) demonstrated that body concentrations are a better indicator of toxicity than free ion activity. In their study toxicity increased with time as a measure of free ion activity whereas the

toxicity of cobalt to *Enchytraeus Crypticus* measured as a body concentration remained constant after 7 days of exposure. Median effect concentrations for exposures measured as free ion activity ($Co^{2+}$) and body metal concentration ranged from 370 to 534 µM and 1718 to 4091 µM/kg, respectively.

The ability of cations to compete for cobalt binding sites and reduce cobalt toxicity was investigated by Lock et al. (2006). Higher activities of $Ca^{2+}$, $Mg^{2+}$ and $H^{+}$ ions linearly increased the 14 day $LC_{50}$ in potworms, but $Na^{+}$ did not.

Cobalt exposure was found to cause multi-biological defects in *C. elegans* and affected life span, development, reproduction and chemotaxis plasticity at concentrations of 2.5 µM and higher (Wang et al., 2007). Many of the defects could be transferred from the parents to the F1 and F2 generations.

Oorts et al. (2011) report invertebrate $EC_{10}$ values ranging between 14.3 and 1091 mg Co/kg soil based on a cumulative four species and 27 reliable data end points. Data is noted to be a compilation of multiple studies. When corrected for differences in lab and field soil (leaching – ageing) and considering a background cobalt concentration the authors calculated mean normalized invertebrate $EC_{10}$ values (considering a soil CEC of 8.6 cmol/kg) to range between 105 and 417 mg Co/kg soil.

Environment Canada (2010) reported $LC_{50}$ values greater than 1210 mg Co/kg dw soil for adult survival of both earthworms and springtail based on 28 or 35 day exposure durations. An $IC_{50}$ of 16.4 mg Co/kg dw soil based on a 63 day exposure was reported for earthworm juvenile production. An $IC_{50}$ of 432 mg Co/kg dw soil based on 28 day exposures was determined for springtail juvenile production.

## 5.0 BEHAVIOUR AND EFFECTS IN LIVESTOCK AND TERRESTRIAL WILDLIFE SPECIES

### 5.1 Uptake, Metabolism and Elimination

Along with a role in vitamin $B_{12}$ production in ruminants, cobalt may also play a role in rumen fermentation by increasing fiber digestion from low quality forages (Zelenak et al., 1992). Generally, ruminants fed forages with low cobalt concentrations (<0.08 mg/kg) develop signs of vitamin $B_{12}$ deficiency (McDowell, 1997), which include loss of appetite, reduced growth rate and anaemia (Underwood and Suttle, 1999). Cattle have the highest sensitivity to cobalt deficiency, followed by sheep and goats (McDowell, 2003).

Studies by Wehner et al. (1977) showed that hamster lungs absorb approximately 30% of an inhaled dose of cobalt oxide. Cobalt particles in the respiratory tract can be absorbed into the blood or transferred into the gastrointestinal tract after swallowing (Bailey et al., 1989). Studies on rats show that 13 to 34% of soluble cobalt chloride is absorbed in the gastrointestinal tract, compared to only 3% of insoluble cobalt oxides (Kim et al., 2006).

Animal elimination data indicate that soluble cobalt compounds such as cobalt(II) oxide are cleared at a faster rate from the lungs than less soluble compounds such as cobalt(II, III) oxide (Barnes et al., 1976; Kreyling, 1984). Urinary excretion rates appear to correlate with the translocation of cobalt from the lungs to blood and faecal excretion rates appear to correlate with mechanical clearance rates of cobalt from the lungs to the gastrointestinal tract (Kim et al., 2006).

The primary route of elimination following oral exposure to cobalt appears to be through faecal excretion of the unabsorbed fraction in animals (Kim et al., 2006). Absorbed cobalt is excreted via the kidney and also via milk (EFSA, 2009).

### 5.2 Bioaccumlation

Chronic exposure to cobalt chloride, with a maximum dose of 15 mg cobalt per kg of body weight, caused an approximately 100-fold and 20-fold increase of the trace element in sheep liver and kidney, respectively (Corrier et al., 1986). Prolonged exposure to cobalt at the administered doses was tolerated without any pathological manifestations. These results differ from previous reports indicating that daily doses between 4 to 11 mg cobalt per kg of body weight caused anemia, severe anorexia, weight loss, acute toxicosis and mortality (Becker and Smith, 1951). In animals capable of synthesizing cobalamin, ingested cobalt(II) chloride is also deposited in the form of vitamin $B_{12}$.

### 5.3 Toxicity

Cobalt tolerance in animals may vary considerably and may be affected by dietary factors such as protein, sulphhydryl-compounds, iron, selenium, vitamin E and age (NRC, 2005). Ruminants have a high tolerance to cobalt, greatly in excess of their dietary requirements. The maximum tolerable level of cobalt was established as 25 mg/kg for cattle, poultry, sheep and horses and 100 mg/kg for swine (NRC, 2005). Current EU legislation recommends that the feed for all animal species should not exceed 2 mg total cobalt per kg of complete feed (EFSA, 2012).

Chronic cobalt toxicity in most species results in reduced feed intake, reduced body weight, emaciation, anaemia, hyperchromaemia, debility and increased liver cobalt concentrations (NRC, 1980). Cattle experiencing cobalt toxicity may show excessive urination, defecation and salivation, shortness of breath and increased red blood cell count (NRC, 1996).

Cobalt dichloride and cobalt sulphate are considered to be of low acute toxicity but are classified as respiratory and skin sensitizers (EFSA, 2012). Cobalt(II) cations are considered genotoxic under *in vivo* and *in vitro* conditions, with carcinogenic, mutagenic and reproduction toxicant properties. Data are not available for the potential carcinogenicity of oral exposure to cobalt(II) in animals. The EU Panel on additives and products used in animal feed (FEEDAP) has developed a health-based guidance value for oral cobalt exposure of 0.0016 mg/kg of body weight per day (EFSA, 2012).

The U.S. EPA has conducted an in-depth review of literature pertaining to cobalt toxicity in birds and mammals (US EPA, 2005). Twenty studies (38 end points) involving mammals and 11 studies (24 end

points) involving birds were used in the selection of oral toxicity reference values (TRVs) for risk-based ecological soil screening levels (Eco-SSLs).

## 6.0 BEHAVIOUR AND EFFECTS IN HUMANS AND EXPERIMENTAL ANIMALS

### 6.1 Overview

As a component of cyanocobalamin (vitamin $B_{12}$), cobalt is essential in the body. Cobalt has been identified in most tissues of the body, with the highest concentrations found in the liver.

### 6.2 Pharmacokinetics

#### 6.2.1 Absorption

Inhaled cobalt particles are deposited in the upper or lower respiratory tract, depending on particle size (WHO, 2006). Studies conducted in hamsters (Wehner et al., 1977) indicate approximately 30% of cobalt oxide is absorbed after inhalation.

Gastrointestinal absorption is affected by the species, dose and nutritional status, and has varied from 18% to as high as 97% in human studies (WHO, 2006). Rat studies have shown cobalt chloride absorption between 13 and 34%, compared to cobalt oxide absorption in the range of 1 to 3% (WHO, 2006). Water-soluble forms are generally absorbed more readily than insoluble forms, and iron deficiency increases absorption while increasing dose decreases absorption; there are also indications that younger animals may absorb cobalt more readily than older animals based on rat studies (WHO, 2006).

Health Canada (2010) has not specified a dermal relative absorption factor for cobalt but recommends a default value of 0.01 for inorganics with insufficient data for a chemical-specific value.

#### 6.2.2 Distribution

In most humans, cobalt is found distributed throughout the body and in most tissues, with the highest levels found in the liver, kidney and bones (ATSDR, 2004). Occupationally exposed workers have been found to have elevated cobalt concentrations in the lungs; metal workers were also found to have increased levels in lymph nodes, liver, spleen and kidneys (Kim et al., 2006). Histological analysis of animals exposed to cobalt through inhalation indicated that cobalt particles were found in macrophages within the bronchial wall or interstitium near the terminal bronchiole (Brune et al, 1980, cited by Kim et al., 2006).

After oral exposure, animal studies have indicated cobalt is distributed mainly to the liver, although it has also been found in the kidneys, heart, stomach and intestines; in pregnant rats, oral exposure also lead to increased concentrations in foetal blood and amniotic fluid (Kim et al., 2006).

### 6.2.3 Metabolism

Cobalt is essential in the body because it is a component of vitamin $B_{12}$ (Vouk, 1986 as reported by ATSDR, 2004). Vitamin $B_{12}$ is a cofactor in the synthesis of methionine and metabolism of folates and purines (ATSDR, 2004; Allen, 2012; Environment Canada, 2014 (draft)). No other essential function of cobalt has been reported. The recommended daily allowance of vitamin B12 in adults is 2.4 µg/day and the cobalt content in this dose is about 0.1 µg/day (ATSDR, 2004).

### 6.2.4 Elimination

After inhalation, elimination occurs in three phases. Initially, particles in the tracheobronchial region are removed through mucociliary clearance with a half-life of 2 to 44 hours. The second phase involves cobalt particles being removed from the lungs through macrophage-mediated clearance, with a half-life of 10 to 78 days. The final phase is long-term lung clearance with a half-life of several years (Kim et al., 2006).

A human aerosol exposure study found that 17% of the initial lung burden was eliminated during the first week, primarily through mechanical clearance to the gastrointestinal tract and subsequent excretion in faeces. After 6 months, a total of approximately 60% of the initial lung burden had been eliminated, with approximately half that in urine and half in faeces (Foster et al., 1989 cited in Kim et al., 2006). Generally large particles are more likely to be cleared to the gastrointestinal tract. Animal studies have shown that more soluble cobalt compounds are cleared from the lungs more quickly (ATSDR, 2004).

Following oral exposure, excretion is primarily through faeces, particularly for low-solubility compounds where absorption may be low and excretion rapid. For more soluble compounds that are absorbed, urine may also be a significant method of excretion (ATSDR, 2004).

### 6.2.5 Physiologically-based Pharmacokinetic Models

The International Commission on Radiological Protection (ICRP, 1994) has developed pharmacokinetic models for inhalation and ingestion exposures to radionuclides; while they are not specific to cobalt they are expected to be applicable for cobalt (Kim et al., 2006). These models are primarily designed for estimating radiation doses, however.

An updated biokinetic model was developed by Leggett (2008) that updates the ICRP model but is also intended to apply to environmental or medical exposures. This model was validated by Unice et al. (2012) and also applied by Finley et al. (2012a) to relate oral exposures to blood concentrations.

### 6.3 Essentiality

Cobalt is a component of vitamin $B_{12}$. However, cobalt deficiency is normally only encountered in ruminant animal species and there is no evidence that dietary cobalt is limiting in humans (NRC, 1989).

### 6.4 Acute and Sub-Chronic Exposure

#### 6.4.1 Human

*Oral*

A post-mortem autopsy of a 19-month-old male who died following ingestion of approximately 30 mL of $CoCl_2$ identified coagulative necrosis of the stomach mucosa edema of the brain. The child's liver, kidneys and spleen were collectively noted to contain 89.4 mg Co, two orders of magnitude greater than a typical human body content (Jacobziner and Raybin, 1961; Paustenbach et al., 2013).

Patients injected with a single diagnostic dose of radioactive iodine, and then treated 48 hours later with 1 mg cobalt/kg/day as cobalt chloride for 2 weeks, resulted in a greatly reduced uptake of radioactive iodine by the thyroid in 1 week, with uptake nearing 0 by the second week (Roche and Layrisse, 1956 as cited in ASTDR, 2004). When the cobalt treatment ended, the uptake values returned to normal.

A study by Davis & Fields (1958) demonstrated that six normal men aged 20–47 exposed to a daily oral dose of cobalt chloride (150 mg/day) for up to 22 days experienced polycythaemia. Red blood cell numbers were noted to increase approximately 16–20% increase over pretreatment levels. Haemoglobin levels were also increased, by 6–11% over pretreatment levels (WHO, 2006). Increase levels of red blood cells were also found following oral treatment of anephric patients (with resulting anemia) with 0.16–1.0 mg cobalt/kg/day daily as cobalt chloride for 3–32 weeks (Duckham and Lee 1976b; Taylor et al. 1977 as cited by ASTDR, 2004). Alternatively, Jaimet & Thode (1955) dosed 15 young children with similar and higher doses (0.45, 0.90 or 1.8 mg Co/kg-d) for 10 weeks with no clinically significant increase in blood hemoglobin levels at any applied dose.

*Inhalation*

Human acute exposure studies to cobalt are limited. In a study of 15 healthy young men Kusaka et al. (1986) noted a significant decrease in forced vital capacity (FVC) occurred after exposure to hard metal dust for six hours with a mean cobalt concentration of 38 $\mu g/m^3$. The ventilatory change was noted to likely be the result of an irritant effect on the large bronchi.

### 6.4.2 Animal

*Oral*

Acute oral toxicity is affected by the cobalt compound and animal species tested; reported $LD_{50}$ values are as low as 42.4 mg/kg for cobalt chloride to 3672 mg/kg for insoluble tri-cobalt tetraoxide in rats; mouse $LD_{50}$s include 89.3 mg/kg cobalt chloride and 123 mg/kg cobalt sulphate (WHO, 2006).

Oral exposure to cobalt chloride at 50 mg/kg-bw/d (12.4 mg/kg-bw/d as cobalt) exhibited cardiac damage after 3 weeks (Morvai et al., 1993 cited in WHO, 2006).

Longer exposures (2-3 months) have resulted in increased heart weight and degenerative heart lesions in rats exposed to cobalt at 26 mg/kg-bw/d, while dietary exposure at 8.4 mg/kg-bw/d resulted in reduced cardiac enzyme activity levels after 24 weeks, and 10 mg/kg-bw/d for 4-5 months led to renal injury (WHO, 2006).

*Inhalation*

Various inhalation studies have been conducted using rats and mice. A cobalt sulphate heptahydrate concentration of 19 mg/m$^3$ was found to cause inflammation of the respiratory tract epithelium, thymus necrosis and testicular atrophy after 16 days in rats; mice exposed at 1.9 mg/m$^3$ for 16 days also experienced the respiratory effects (NTP, 1991). Ultrafine cobalt particles caused focal hypertrophy, macrophage damage, intracellular oedema, interstitial oedema and proliferation of type II alveolar epithelium after 4 days at 2.12 mg/m$^3$ for 5 hours/day (Kyono et al., 1992 cited in WHO, 2006).

Palmes et al. (1959, cited in WHO, 2006) exposed rats, guinea pigs and beagles to cobalt hydrocarbonyl at 9 mg/m$^3$ for 6 hours/day, 5 days/week over 3 months; foam cell aggregates were evident in all 3 species but were no longer present 3 months or 6 months after exposure. Rabbits exposed to cobalt chloride at 0.4 to 2 mg/m$^3$ for 1 to 4 months had lesions in the alveolar region.

NTP (1991) conducted a 13-week (6 hours/day, 5 days/week) inhalation study using F344/N rats and B6C3F1 mice using cobalt sulphate heptahydrate at concentrations of 0, 0.3, 1, 3, 10 and 30 mg/m$^3$. Rats exhibited squamous metaplasia in the larynx at 0.3 mg/m$^3$ and higher; chronic inflammation of the larynx and hystiocytic infiltrates in the lung were observed at 3 mg/m$^3$. At higher concentrations other respiratory tract effects including epithelial degeneration and hyperplasia and larynx necrosis were observed. Similarly in mice squamous metaplasia of the larynx and, in males, histiocytic infiltrates in the lung were observed at 0.3 mg/m$^3$ with other respiratory tract effects becoming evident at higher concentrations. Sperm motility was also affected in mice at 3 mg/m$^3$ and higher and

the estrous cycle was increased at 30 mg/m$^3$; these effects were not assessed in the lower exposure concentrations.

### 6.5 Chronic Exposure

#### 6.5.1 Human

#### 6.5.2 Oral Toxicity

Several studies have reported lethal cardiomyopathy in people who consumed large quantities of beer over a period of years (approximately 8 to 30 pints per day) with added cobalt sulfate (Morin & Daniel, 1967; Kesteloot et al., 1968; Alexander, 1969, 1972; Bonenfant et al., 1969; Sullivan et al., 1969; Morin et al., 1971 as cited by WHO, 2006). The practice of adding cobalt sulfate to beer as a foam stabilizer occurred during the early-to-mid 1960's in breweries in the United States, Canada and Europe. The cobalt exposures leading to death were determined to range between 0.04 to 0.14 mg/kg bw/d (ASTDR, 2004). Approximately 40–50% of the patients admitted to the hospital for cardiomyopathy died within several years of diagnosis (Alexander, 1972). Potential compounding factors contributing to the cardiomyopathy have been noted including protein-poor diets of the beer-drinkers and potential prior cardiac damage from alcohol abuse (ASTDR, 2004; WHO, 2006).

Morin et al. (1971) noted in 27 of 50 patients with beer-cobalt cardiomyopathy that pulmonary rales were heard and pulmonary oedema presented in 9 of 50 patients. Patients also presented central hepatic necrosis accompanied by increased levels of serum bilirubin and serum enzymes. Initial symptoms generally manifested as nausea, vomiting and diarrhoea. Potential compounding factors from a high rate of alcohol ingestion over a prolonged period (years) could not be ruled out.

Paustenbach et al. (2013) summarized several studies (Chamberlain et al., 1961; Roche and Layrisse, 1956; Paley et al., 1958) that report oral cobalt doses ranging between 0.11 and 10 mg Co/kg-d decreased iodine uptake by the thyroid. This sometimes resulted in the development of a goiter and signs of hypothyroidism. It was noted that the thyroid effects were generally reversed when treatment was ceased.

Veinen et al. (1987) described allergic dermatitis in cobalt-sensitized patients challenged orally with 0.014 mg Co/kg/d (as cited in ASTDR, 2004).

#### 6.5.3 Inhalation Toxicity

In a cross-sectional study of 82 workers employed at a cobalt refinery and exposed for 8 years on average Swennen et al. (1993) determined those exposed to airborne cobalt metal, salts, or oxides

(mean concentration 0.125 mg/$m^3$) showed an increased prevalence of dyspnea and wheezing and had significantly more skin lesions (eczema, erythema) than control workers. A slight interference with thyroid metabolism (decreased T3, T4, and increased TSH), a slight reduction of some erythropoietic variables (red blood cells, haemoglobin, packed cell volume) and increased white cell count were found in the exposed workers. Prescott et al. (1992) showed significantly elevated levels of T4 and free thyroxine, but no change in T3 levels for female workers occupationally exposed to cobalt-zinc silicate (estimated concentration of 0.05 mg Co/$m^3$).

Nemery et al. (1992) conducted a cross-sectional study of 194 diamond polishers exposed to airborne cobalt from polishing discs and compared with 59 control workers. The workers were separated into three exposure groups: control (mean of 0.0004 mg Co/$m^3$), low (mean of 0.0053 mg Co/$m^3$) and high (mean of 0.0151 mg Co/$m^3$). The high exposure group was more likely to complain about respiratory symptoms and had significantly higher prevalence of eye, nose, and throat irritation and cough. The prevalence of some symptoms (e.g. cough, phlegm) was elevated in the low exposure group compared with the control group, but they were not significantly elevated. Lung function, as measured by FVC, $FEV_1$, MMEF (forced expiratory flow between 25% and 75% of the FVC) and mean PEFR was significantly reduced in the high exposure group versus the low exposure and control groups. Lung function was not decreased in the low exposure group compared with the control group and a NOAEC was determined based on the low exposure group (0.0053 mg Co/$m^3$).

In an occupational exposure study Gennart and Lauwerys (1990) examined the ventilatory function of 48 diamond polishing workers relative to control workers. Exposure to cobalt was at mean concentrations of 0.0152 or 0.1355 mg cobalt/$m^3$. Significant decreases in ventilatory function were found in the exposed workers relative to the control workers. Duration of exposure played a significant factor, with no significant differences in workers who had been exposed for ≤5 years; reported decreases in ventilatory function were noted in workers exposed for > 5 years (as cited by ASTDR, 2002).

Eight workers in a hard metal plant were found to have occupational asthma due to cobalt reactivity based on the findings of positive broncho-provocation with $CoCl_2$. The sensitization phenomenon includes the production of IgE antibodies to cobalt. Exposure levels ranged from 0.07 to 0.893 mg Co/$m^3$. The development of hard metal induced asthma in exposed workers to cobalt was supported by Davison et al. (1983).

Occupational exposure of humans to cobalt-containing dust, either as cobalt metal or as hard metal, has been shown to result in cardiomyopathy, characterized by functional effects on the ventricles (Horowitz et al. 1988) and/or enlargement of the heart (Barborik and Dusek 1972; Jarvis et al. 1992),

but the exposure levels associated with cardiac effects of inhaled cobalt in humans have not been determined (as cited in ASTDR, 2004).

### 6.5.4 Animal

### 6.5.5 Oral Toxicity

A review of available literature did not return any chronic oral toxicity studies with animals as test subjects.

### 6.5.6 Inhalation Toxicity

Inhalation of cobalt metal has been found to cause increased incidence of hyperplasia in the alveolar and bronchiolar epithelia in rats and mice (NTP, 2014); cobalt sulphate inhalation was found to result in hyperplasia in alveoloar epithelia of rats but not mice (NTP, 1998). Both cobalt compounds induced non-neoplastic lesions in the nose of rats and mice (Behl et al., 2015). NTP (2014) also observed adrenal medullary hyperplasia in female rats exposed to cobalt metal at concentrations of 1.25 or 2.5 $mg/m^3$, as well as increased incidence of infarct of the testes in male rats exposed to 5 $mg/m^3$ and an exposure-related increase in germinal epithelium degeneration in mice (NTP, 2014; Behl et al., 2015).

## 6.6 Carcinogenicity and Genotoxicity

### 6.6.1 Human Data

Mortality studies of the hard metal industry, in which cobalt is used, suggest an increase in lung cancer mortality; however, exposure to other compounds with potential additive effects is noted (WHO, 2006). Hogstedt and Alexandersson (1990) reported on 3,163 male employees of hard metal manufacturing plants in Sweden with at least one year of occupational exposure during 1940 to 1982. Of the entire worker sample population, the authors reported a lung cancer standardized mortality ratio (SMR) of 2.78 for workers with 10 years of employment and more than 20 years since first exposure. Smoking habits of workers were noted to be similar to those of the male Swedish population (as cited in WHO 2006).

Lasfargues et al. (1994) reported on the mortality of a cohort of 709 male workers in a French hard metal plant. The overall mortality relative to national rates did not differ from expected though a significant increase in mortality due to cancer of the trachea, bronchus, and lung (SMR=2.13) was noted. Smoking alone did not account for the lung cancer excesses, although the influence of smoking on the observed mortality could not be entirely ruled out (as cited by ASTDR, 2004).

Moulin et al. (2000) examined the lung cancer mortality of 4,288 male and 609 female workers employed in the production of stainless and alloyed steel from 1968 to 1992. No significant changes in

mortality rate from lung cancer were seen among exposed workers (SMR=1.19), and a concurrent case control study identified no correlation between lung cancer excess and exposure to cobalt (as cited in ASTDR, 2004).

The correlation between cancer mortality and trace metals in water supplies (10 basins) throughout the United States was investigated by Berg and Burbank (1972). No correlation was found between cancer mortality and the level of cobalt in the water. Cobalt levels of 1–19 µg/L, with resulting human intakes ranging from 0.03 to 0.54 µg/kg/day, were reported (ASTDR, 2004).

### 6.6.2 Animal Data

NTP (1998; 2014) inhalation studies found evidence of alveolar/bronchiolar adenomas and carcinomas in rats and mice from both cobalt sulphate and cobalt metal; cobalt metal was also associated with cystic keratinizing epitheliomas in the lungs of rats. Pheochromocytomas in the adrenal gland were also associated with both cobalt sulphate and cobalt metal (NTP, 1998; 2014). Cobalt metal (but not cobalt sulphate) also led to increased incidences of pancreatic isled adenoma/carcinoma in rats, mononuclear cell leukemia in rats, and renal tubule adenoma/carcinoma in male rats compared to controls; however these increases were either not statistically significant or did not demonstrate clear relationships with exposure concentration (NTP, 2014; Behl et al., 2015). Behl et al. (2015) concluded that, contrary to expectations, cobalt metal was more toxic/carcinogenic at similar cobalt concentrations compared to soluble cobalt sulphate.

### 6.6.3 Genotoxicity/Mutagenicity

Kirkland et al. (2015) conducted genotoxicity studies using soluble and poorly soluble cobalt compounds. They found evidence of chromosomal damage *in vitro* but no biologically significant mutagenic responses and negative results for chromosomal aberrations and micronuclei *in vivo*. They concluded that low solubility cobalt compounds do not appear to be genotoxic, and that chromosome damage caused by soluble cobalt compounds appeared to be related to oxidative stress, and that overall cobalt did not appear to have genetic toxicity to humans. Behl et al. (2015) also hypothesized generation of reactive oxygen species as a potential mechanism of cobalt-induced carcinogenicity.

### 6.6.4 Carcinogenic Classification

Cobalt metal with tungsten carbide has been classified as probably carcinogenic to humans (Group 2A) based on limited evidence in humans (IARC, 2006). Cobalt metal without tungsten carbide, cobalt sulfate and other soluble cobalt (II) salts have been classified as possibly carcinogenic to humans (Group 2B) based on inadequate evidence in humans for carcinogenicity of cobalt metal, sufficient evidence in animals for carcinogenicity of cobalt sulfate and cobalt-metal powder, and limited evidence in animals for carcinogenicity of metal alloys containing cobalt (IARC, 2006).

## 6.7 Toxicological Limits Developed by Health Canada

Health Canada (2010) has not published TRVs for cobalt. A screening assessment conducted for the Chemicals Management Plan (Environment Canada and Health Canada, 2011) concluded that 0.04 mg/kg-bw/d would be a conservative oral LOAEL for cobalt, but noted that there is evidence of therapeutic use at higher doses. A LOAEC in humans of 0.0151 mg/m$^3$ was identified based on a cross-sectional study of diamond-polishers (Nemery et al., 1992). TRVs were not specifically derived from these values.

An updated screening assessment (Environment Canada and Health Canada, 2014) used a biokinetic model to establish a whole blood cobalt concentration of 26 µg/L as being equivalent to the LOAEL of 0.04 mg/kg-bw/d. It was also noted that this endpoint is specific to an atypical sensitive sub-population (people with severely deteriorated health who consume large amounts of alcohol chronically), while a point of departure of 0.45 mg/kg-bw/d may be more appropriate for the general population (equivalent to a blood concentration of 290 µg/L).

## 6.8 Toxicological Limits Developed by Others

*United States Environmental Protection Agency*

US EPA has not published TRVs for cobalt in the Integrated Risk Information System (IRIS). However, a Provisional Peer Reviewed Toxicity Value has been published (US EPA, 2008). A chronic provisional RfD of 0.0003 mg/kg-bw/d was derived based on thyroid toxicity. A LOAEL of 1 mg/kg-bw/day (Kriss et al., 1955) for decreased iodine uptake in the thyroid was adjusted by uncertainty factors of 10 for use of a LOAEL, 10 for the protection of sensitive populations, 10 for use of a subchronic study, and 3 to account for the lack of a multi-generation toxicity study (3000 total). A subchronic provisional RfD of 0.003 mg/kg-bw/d was also proposed using the same study but without the uncertainty factor for use of a subchronic (2 week) study. US EPA (2008) considered confidence in the principal study to be low to medium, and confidence in the database to be low to medium, resulting in low confidence in the provisional RfD. It should be noted that the chronic RfD is within the range of typical dietary exposures (Finley et al., 2012b)

Provisional chronic and subchronic RfCs were also derived by US EPA (2008) of 0.000006 mg/m$^3$ and 0.00002 mg/m$^3$, respectively. A NOAEL of 5.3 µg/m$^3$ and LOAEL of 15.1 µg/m$^3$ for effects on pulmonary function in workers (Nemery et al., 1992) were used as the basis. The NOAEL was adjusted for continuous exposure (1.9 µg/m$^3$) and uncertainty factors of 10 each for database insufficiencies and inter-individual variability were applied. For the chronic RfC, an additional uncertainty factor of 3 (for a total uncertainty factor of 300) was applied; data on worker exposure duration were not available from the critical study and therefore subchronic exposure was assumed. It was noted that this RfC may not be protective for people with hypersensitivity to cobalt.

Confidence in the key study was considered low, and confidence in the database was considered medium, for an overall low to medium confidence in the provisional RfC.

A provisional inhalation unit risk was also developed (US EPA, 2008) based on alveolar/bronchiolar neoplasms observed in a 2-year rat and mouse carcinogenicity study (NTP, 1998; Bucher et al., 1999). The study exposure concentrations were adjusted for continuous exposure and human-equivalent concentrations. Benchmark dose modelling was then used to derive a provisional unit risk of 9 $(mg/m^3)^{-1}$. No appropriate data were identified for the derivation of an oral slope factor.

*Agency for Toxic Substances and Disease Registry*

The Agency for Toxic Substances and Disease Registry (ATSDR, 2004) published a chronic inhalation minimal risk level (MRL) for cobalt of 0.0001 $mg/m^3$. This value was derived from a cross-sectional study of diamond polishers (Nemery et al., 1992), where a NOAEL of 0.0053 $mg/m^3$ was established based on respiratory effects (eye, nose and throat irritation, cough, and reduced lung function) in workers with high cobalt exposure. This value was adjusted for continuous exposure (0.0013 $mg/m^3$) and an uncertainty factor of 10 for human variability was applied.

ATSDR (2004) also published an intermediate oral MRL of 0.01 mg/kg-bw/d based on the development of polycythemia in humans administered 150 mg/day cobalt chloride (~1 mg/kg-bw/day cobalt) in water or milk for up to 22 days; the effects were found to be reversible (Davis and Fields, 1958). This dose was considered to be a LOAEL; an uncertainty factor of 100 (10 for use of a LOAEL and 10 for human variability) was applied.

*Netherlands RIVM*

The Netherlands National Institute for Public Health and the Environment (RIVM, 2001) has established an oral tolerable daily intake (TDI) for cobalt of 1.4 μg/kg-bw/d (0.0014 mg/kg-bw/d) based on cardiomyopathy in humans (Vermeire et al., 1991). A LOAEL of 0.04 mg/kg-bw/d after intermediate oral exposure was identified in humans. Since this study reflected a small population where adverse effects due to intake of alcohol were a possibility, an intra-human uncertainty factor of 3 was applied, along with a factor of 10 for use of a LOAEL.

A tolerable concentration (TC) of 0.0005 $mg/m^3$ (Sprince et al., 1998) was also derived based on interstitial lung disease in humans (ATSDR, 1992; RIVM, 2001). This value was based on a LOAEL of 0.05 $mg/m^3$ with uncertainty factors of 10 for extrapolation from a LOAEL and 10 for intrahuman variability.

*Finley et al., 2012b*

Finley et al. (2012b) proposed an oral RfD for non-cancer effects of 0.03 mg/kg-bw/d using methodologies based on US EPA guidance. A thyroid function test (Jaimet and Thode, 1955) conducted on 18 children aged 5 to 9 over 10 weeks was selected as the critical study. A NOAEL of 1.8 mg/kg-bw/d was identified based on the absence of endocrine or hematological effects; however, Finley et al. (2012b) used the next lowest dose, 0.9 mg/kg-bw/d, due to a lower reported LOAEL in another study that was considered too unreliable to itself be used for RfD derivation.

An uncertainty factor of 1 was applied for extrapolation to a chronic duration based on the approach US EPA used for perchlorate, which is believed to have a similar mode of action. An uncertainty factor of 10 was applied for sensitive subpopulations, though it was noted that the study population (children) was expected to be a sensitive group. An uncertainty factor of 3 was applied for database limitations.

### 6.9 Selection of Toxicity Reference Values

The oral tolerable daily intake value of 0.0014 mg/kg-bw/d specified by the Netherlands National Institute for Public Health and the Environment (RIVM, 2001) is adopted herein. The oral TDI is based upon the lowest LOAEL value reported from human studies with uncertainty applied, which is also the value identified as a conservative LOAEL by Health Canada (Environment Canada and Health Canada, 2011). The resulting TDI is higher than the US EPA (2008) RfD value; however, confidence in the principal study used to develop the US EPA RfD value is low which resulted in a high degree of uncertainty and a very large uncertainty factor. It is also noted that the US EPA provisional RfD is lower than estimated background exposures.

US EPA (2008) and ATSDR (2004) derived chronic inhalation TRVs using the same study but different uncertainty factors. The US EPA (2008) value of 0.000006 $mg/m^3$ is more conservative and reflects some of the uncertainties in the database, and is therefore applied. The US EPA (2008) provisional unit risk of 9 $(mg/m^3)^{-1}$ is also applied to evaluate potential inhalation carcinogenicity.

## 7.0 DERIVATION OF ENVIRONMENTAL AND HUMAN HEALTH SOIL QUALITY GUIDELINES

### 7.1 Environmental Soil Quality Guidelines

Canadian Soil Quality Guidelines are derived for four different land uses: agricultural, residential/parkland, commercial and industrial. Alberta also adds guidelines for the natural area land use.

All data for use in the following derivations have been screened for ecological relevance and are presented in the preceding sections. For the soil contact pathway, data were not selected from soils that are outside the typical conditions found in Canada (e.g. pH <4), or from studies that did not use

soil or artificial soil, did not record soil texture and pH, did not use appropriate statistical analyses, did not use controls, or involved sewage sludge or mixtures of toxicants.

### 7.1.1 Soil Quality Guidelines for Soil Contact

Soil quality guidelines for soil contact ($SQG_{SC}$) are based on toxicological data for plants and soil invertebrates. The preferred approach is to use a weight of evidence method using $EC_{25}$ or similar values; if the data do not meet the requirements for this method, then additional approaches using other data points can be applied, such as effects/ no effects data, lowest observed effects concentrations, and median effects.

The data requirements for the preferred weight of evidence approach include:

- At least 10 discrete data points from at least 3 studies.
- A minimum of 2 soil invertebrate and 2 crop/plant data points.

The plant and invertebrate data available for cobalt does not meet the minimum requirements for the weight of evidence method using an $EC_{25}$ distribution; however, the available data does meet the minimum requirements for the weight-of evidence method using an effects/ no effects data distribution approach. This approach has been applied herein.

In some cases it is prudent to combine data points to eliminate redundancy by calculating the geometric mean of individual data points (CCME, 2006). For example, data points representing the same type of response in the same species under highly similar exposure conditions, or different responses that are known to be directly, causally connected should be combined. Consideration can also be given to combining data for different soil types – in general variations in toxicity due to exposure conditions such as soil type are considered to be a valid part of the sensitivity distribution, but in some cases it may be appropriate to combine data points to prevent a significant bias of the sensitivity distribution to a single species.

Plant toxicity data from Mico et al. (2008), Li et al. (2009) and Environment Canada (2010) met the minimum requirements. Mico et al. (2008) provided NOEC and $EC_{50}$ data for the effect of cobalt on root growth of one plant species (barley) grown in 10 field-collected soil types. Li et al. (2009) provided NOEC, LOEC and $EC_{50}$ data for the effect of cobalt on shoot growth of three plant species (oilseed rape, tomato and barley) grown in 10 field-collected soil types. Environment Canada (2010) provided $EC_{50}$ and $IC_{50}$ data for the effect of cobalt on emergence, shoot and root length and shoot and root dry weight for four plant species (red clover, radish, tomato and wheat grass) grown in a field-collected soil.

Invertebrate toxicity data from Lock et al. (2004, 2006) and Environment Canada (2010) also met the minimum requirements. Lock et al. (2004) included NOEC, LOEC and $EC_{50}$ data for the effect of cobalt on *F.candida* (springtail) reproduction in both an artificial and field-collected soil. Lock et al. (2006) provided $LC_{50}$ data for the effect of cobalt on *E.albidus* (potworm) survival in both an artificial and field-collected soil. Environment Canada (2010) provided $LC_{50}$ and $IC_{50}$ data for the effect of cobalt on *F.candida* (springtail) and *E.andrei* (earthworm) survival and reproduction in a field soil.

A total of 119 plant data points and 13 invertebrate endpoints were retained.

As specified by CCME (2006), the selected data were ranked and rank percentiles determined for each data point. The protocol allows plant and invertebrate data to be either combined or treated separately; both approaches have been examined herein. The resulting species sensitivity distributions were found to be similar, and therefore the combined data set was used for greater statistical power.

The resulting effects/no-effects distributions are presented below (Figure 1); the complete data set is summarized in Appendix C.

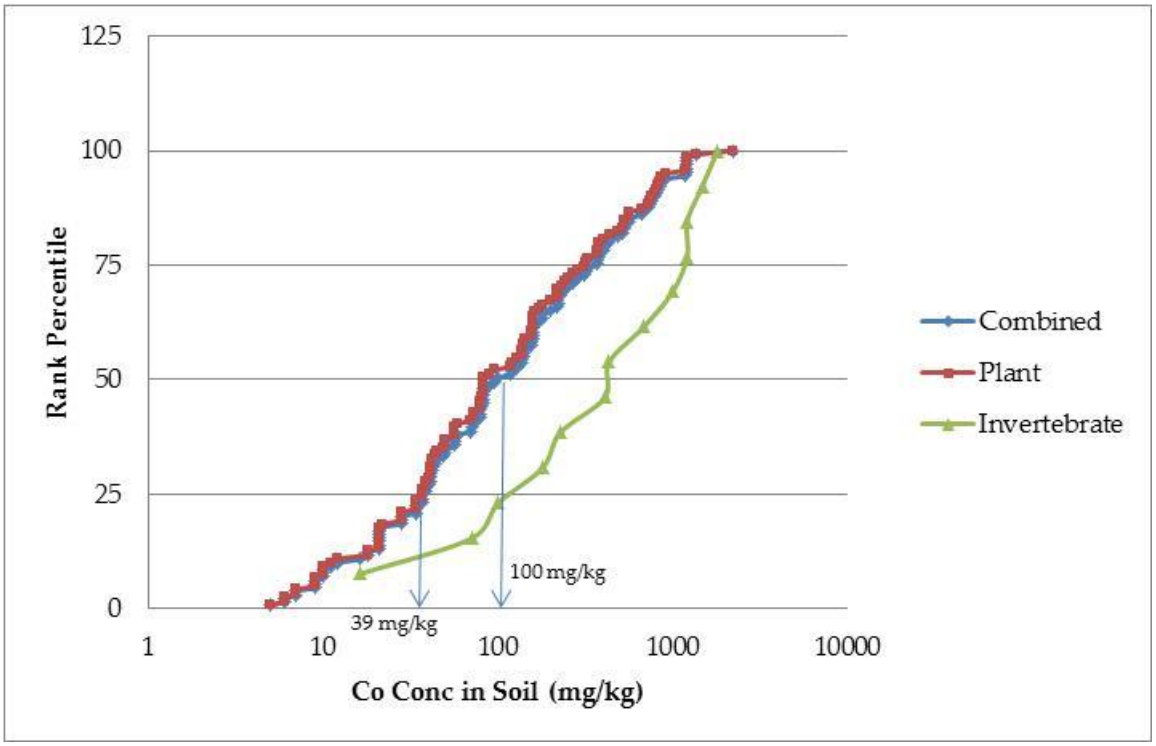


**Figure 1: Species Sensitivity Distribution for Ecological Soil Contact**

#### 7.1.1.1 Guidelines for the Agricultural, Residential/Parkland and Natural Area Land Uses

The soil contact guidelines are calculated from the 25th percentile of the estimated species sensitivity distribution ($ESSD_{25}$). The $ESSD_{25}$ has been calculated at 39 mg/kg.

The threshold effects concentration is then calculated as:

$$TEC = ESSD_{25}/UF$$

Where:

| | | |
|---|---|---|
| TEC | = | threshold effects concentration (mg/kg) |
| $ESSD_{25}$ | = | estimated species distribution – 25th percentile (mg/kg) |
| UF | = | uncertainty factor (if needed) |

An uncertainty factor is only applied if the data are borderline, such as if only the minimum number of studies is available, fewer than three taxonomic groups are represented, greater than 50% of either the plant or invertebrate toxicity data are in the lower 25th percentile of the combined distribution, short-term toxicity studies were used, or more than 50% of the data reflect low bioavailability conditions (CCME, 2006). None of these conditions apply, and therefore an uncertainty factor is not considered to be warranted.

The $SQG_{SC}$ for the agricultural, residential/parkland and natural area land uses is set at the TEC, or 39 mg/kg.

#### 7.1.1.2 Guidelines for the Commercial and Industrial Land Uses

The soil contact guidelines are calculated from the 50th percentile of the estimated species sensitivity distribution ($ESSD_{50}$). The $ESSD_{50}$ has been calculated at 100 mg/kg.

The effects concentration - low is then calculated as:

$$ECL = ESSD_{50}$$

Where:

| | | |
|---|---|---|
| ECL | = | threshold effects concentration (mg/kg) |
| $ESSD_{50}$ | = | estimated species distribution – 50th percentile (mg/kg) |

An uncertainty factor is not normally applied to the ECL. The $SQG_{SC}$ for the commercial and industrial land uses is set at the ECL, or 100 mg/kg.

#### 7.1.1.3 Confidence Ranking for the Soil Contact Guideline

CCME (2006) uses a ranking system to indicate the confidence in the guideline, based on the method used and whether there were enough data to evaluate plants and invertebrates separately.

For cobalt, the weight of evidence approach using effects/no effects data was used and the plant and invertebrate data were combined. Therefore, a confidence ranking of 'D' is assigned.

### 7.1.2 Nutrient and Energy Cycling

The nutrient and energy cycling guideline ($SQG_{NEC}$) is used to evaluate biological processes in the soil that are expected to affect the overall soil ecosystem performance. Professional judgment is applied as to whether this guideline should be used in the overall soil quality guideline calculation (CCME, 2006).

The preferred data for the calculation of guidelines for this pathway are nitrification and nitrogen-fixation data. In the absence of sufficient nitrification and nitrogen fixation values, the data set can be supplemented with decomposition, respiration and nitrogen mineralization data. Studies specifically addressing these issues were not identified; therefore the data are not considered adequate for guideline calculation.

### 7.1.3 Soil Quality Guidelines for Soil and Food Ingestion

Soil and food ingestion guidelines ($SQG_I$) are calculated for the agricultural and natural area land uses to protect domestic animals and wildlife.

The CCME (2006) process normally evaluates grazing herbivores on agricultural lands, although other species can be considered if identified as being particularly sensitive to the contamination. The first step is to identify the species most at threat based on oral toxicological data for grazing/foraging species. The minimum data requirements include at least two oral mammalian studies, only one of which can be a laboratory rodent study and at least one of which should reflect a grazing herbivore, and one oral avian study.

As discussed in Section 5.3, several wildlife studies have been identified, along with a smaller number of avian studies.

#### 7.1.3.1 Development of the Daily Threshold Effect Dose (DTED)

Several toxicity studies have been conducted using avian, wildlife and livestock species, including chickens, ducks, cattle, mice, rats, pigs and guinea pigs. In nearly all cases the studies resulted in unbounded NOAELs and LOAELs due to the small number of treatment groups. Furthermore most of these studies involved very small numbers of animals in each treatment group. US EPA (2005) compiled the available studies and arrived at an avian TRV of 7.61 mg/kg-bw/d and a mammalian

TRV of 7.33 mg/kg-bw/d, in both cases based on the geometric mean of NOAEL values for biologically relevant endpoints. These values are also slightly lower than the lowest bounded LOAEL values and would be conservative compared to the CCME (2006) approach of selecting the lowest effects concentration. Therefore the lower of these TRVs, the mammalian TRV of 7.33 mg/kg-bw/d, is applied as a DTED.

#### 7.1.3.2 Receptor Parameters

A cattle body weight of 550 kg and soil ingestion rate of 0.747 kg/d have previously been used for soil quality guideline derivation (CCME, 2008; AEP, 2016). A typical cattle food ingestion rate is 2.5% of body weight per day or 13.75 kg/d dry weight. This value is slightly higher than what would be calculated using the allometric equation recommended by CCME (2006).

For Alberta natural area land use calculations, a vole with a body weight of 0.017 kg and soil ingestion rate of 0.000058 kg/d is used (AEP, 2016). A food ingestion rate of 0.00241 kg/d is calculated using the CCME (2006) allometric equation.

#### 7.1.3.3 Bioavailability

There is no information on the relative bioavailability of cobalt in natural food/soil compared to bioavailability in the critical toxicity studies. A bioavailability factor of 1 is therefore assumed.

#### 7.1.3.4 Bioconcentration Factors

In general the bioconcentration of cobalt in plants appears to be low. Many of the available studies where plant uptake do not contain sufficient information to calculate a meaningful bioconcentration factor. US EPA have previously endorsed a literature-derived bioconcentration factor of 0.02 which has also been previously proposed by Oak Ridge National Laboratory (Baes et al. 1984); since this is the only BCF that was identified it is adopted herein.

#### 7.1.3.5 Calculation of the Soil Quality Guideline for Ingestion

The guideline for soil and food ingestion can be calculated for a primary consumer using the following equation (CCME 2006):

$$SQG_I = \frac{0.75 \times DTED \times BW}{(SIR \times BF) + (FIR \times BCF)}$$

Where,

SQGI = soil quality guideline for food and soil ingestion (mg/kg)

DTED = daily threshold effects dose (mg/kg-bw/d)

| | | |
|---|---|---|
| BW | = | body weight (kg) |
| SIR | = | soil ingestion rate (kg/d) |
| FIR | = | food ingestion rate (kg/d dry weight) |
| BF | = | bioavailability factor |
| BCF | = | bioconcentration factor (mg/kg plant per mg/kg soil) |

The resulting $SQG_I$ for the agricultural land use is 2,950 mg/kg. For the Alberta natural area land use, the wildlife SQGI is 880 mg/kg.

### 7.1.4 Guidelines for the Protection of Groundwater

No guidelines for protection of groundwater (freshwater life, livestock water and irrigation water) were derived for cobalt due to restrictions on the mathematical model when applied to metals (CCME 2006).

### 7.1.5 Off-site Migration Guidelines for Commercial and Industrial Land Uses

The guideline for offsite migration ($SQG_{OM-E}$) is calculated for the commercial and industrial land uses to protect against transfer of contaminated soil to a more sensitive nearby property through processes such as wind and water erosion. CCME (2006) derived the following equation to evaluate this pathway, based on the Universal Soil Loss Equation and Wind Erosion Equation:

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

Where,

| | | |
|---|---|---|
| $SQG_{OM-E}$ | = | environmental soil quality guideline for off-site migration (mg/kg) |
| $SQG_A$ | = | soil quality guideline for agricultural land use (37 mg/kg) |
| BSC | = | background concentration of chemical in receiving soil (10 mg/kg) |

A background cobalt concentration in soil of 10 mg/kg was selected based on the Geological Survey of Canada soil and till surveys (Grunsky et al., 2012). The resulting SQGOM-E for commercial and industrial land uses is 400 mg/kg.

### 7.1.6 Summary and Selection of the $SQG_E$

For all land uses, the direct ecological soil contact guideline is lowest. All mandatory pathways have been evaluated and therefore a $SQG_E$ can be established.

### 7.1.7 Data Gaps in the Derivation of Environmental Soil Quality Guidelines

The data set for the direct soil contact pathway is considered to be robust, with a variety of plant and invertebrate species evaluated in multiple soil types using standard test protocols.

The nutrient and energy cycling guideline could not be calculated reliably, but it is assumed that the guideline for this pathway would likely be higher than the direct contact guideline.

Uncertainty in the soil and food ingestion pathway arises in part from limited information on bioaccumulation of cobalt in plants. Therefore, when cobalt contamination is present within topsoil on agricultural lands potentially used for grazing, site-specific evaluation of cobalt concentrations in plants/feed may be warranted.

## 7.2 Human Health Soil Quality Guidelines

### 7.2.1 Estimated Daily Intakes

A background cobalt concentration in soil of 10 mg/kg was selected based on the Geological Survey of Canada soil and till surveys (Grunsky et al., 2012). These data represent a Canada-wide survey with extensive prairies soil data. Areas with high background cobalt concentration should be evaluated on a site-specific basis.

Data on cobalt concentrations in drinking water indicated that concentrations are typically below laboratory detection limits as discussed in Section 2.4.3. Due to the variability in detection limits and differences in the ranges of detected concentrations in the available studies, no estimate of background cobalt concentration in drinking water was made, but exposures from water are expected to be negligible.

Data on cobalt in air published by Celo and Dabek-Zlotorzynska (2010) and Cheng et al., (2000) are considered to be representative of typical ambient air concentrations in Canada. The values at the study locations were typically below 0.1 ng/m$^3$, which was applied as the outdoor air background concentration. The same value is applied for indoor air due to the absence of actual indoor air data.

Cobalt was included in the Canadian Total Diet Study between 1993 and 2007. Food and beverage ingestion rates are based on the most recent (2007) information available (Health Canada, 2013b). Concentrations are summarized in Appendix D.

Receptor characteristics used for the Estimated Daily Intake (EDI) calculations are summarized in Table 2 below. The calculated EDI for cobalt is summarized in Table 3. Background exposure to cobalt is primarily dietary, but soil ingestion is also significant.

| Table 2 Receptor Characteristics for EDI Calculation[a] | | | | | | |
|---|---|---|---|---|---|---|
| Age Group / Characteristic | 0-6 months | 0.5 – 4 yr | 5 – 11 yr | 12 – 19 yr | 20 – 59 yr | 60+ yr |
| Inhalation Rate (m3/day) | 2.1 | 9.3 | 14.5 | 15.8 | 15.8 | 15.8 |
| Water Ingestion Rate (L/day) | 0.3 | 0.6 | 0.8 | 1.0 | 1.5 | 1.5 |
| Soil & Dust Ingestion Rate (mg/day) | 20 | 80 | 20 | 20 | 20 | 20 |
| Body Weight (kg) | 8.2 | 16.5 | 32.9 | 59.7 | 70.7 | 70.7 |
| Time Spent Outdoors (day/day) | 0.125 | 0.125 | 0.125 | 0.125 | 0.125 | 0.125 |
| Time Spent Indoors (day/day) | 0.875 | 0.875 | 0.875 | 0.875 | 0.875 | 0.875 |

a – values provided by CCME (2006) for EDI unless otherwise stated.

| Table 3 Estimated Intake (µg/kg-bw/d) of Cobalt | | | | | | |
|---|---|---|---|---|---|---|
| Age Group / Route of Exposure | 0-6 months | 0.5 – 4 yr | 5 – 11 yr | 12 – 19 yr | 20 – 64 yr | 65+ yr |
| Ambient Air | $3.2x10^{-6}$ | $7.1x10^{-6}$ | $5.5x10^{-6}$ | $3.3x10^{-6}$ | $2.8x10^{-6}$ | $42.8x10^{-6}$ |
| Indoor Air | $2.2x10^{-5}$ | $4.9x10^{-5}$ | $3.9x10^{-5}$ | $2.3x10^{-5}$ | $2.0x10^{-5}$ | $2.0x10^{-5}$ |
| Drinking Water | 0 | 0 | 0 | 0 | 0 | 0 |
| Food and Beverages[a] | 0.60 | 0.56 | 0.41 | 0.29 | 0.21 | 0.16 |
| Soil | 0.024 | 0.049 | 0.0061 | 0.0034 | 0.0028 | 0.0028 |
| Total Intake | 0.62 | 0.61 | 0.42 | 0.29 | 0.21 | 0.16 |

a –values provided by Health Canada (2013b) (Appendix D)

### 7.2.2 Soil Guideline for Direct Contact with Soil

Direct human contact with soil is calculated for the agricultural, residential/parkland, commercial and industrial land uses. Cobalt is treated as both a threshold and non-threshold substance. For non-carcinogenic exposure a toddler is considered to be the most sensitive human receptor, except for the industrial land use where only adults are assumed to spend significant amounts of time (CCME, 2006). For non-threshold exposure an adult is considered to be the most sensitive receptor for all land uses.

The direct contact guideline includes 3 separate exposure pathways: incidental soil ingestion, dermal contact with soil, and soil particulate inhalation. Since cobalt is treated as a threshold substance for oral/dermal exposures and non-threshold for inhalation with separate TRVs, the inhalation pathway is removed from the oral and dermal direct contact:

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

Since cobalt has demonstrated inhalation carcinogenicity, a separate inhalation-only calculation is also performed for an adult:

$$SQG_{HH-PI} = \frac{RSC}{(AF_L \times Pa) \times ET_1 \times ET_2}$$

Where:

| | | |
|---|---|---|
| $SQG_{DH}$ | = | direct contact human health soil quality guideline (mg/kg) |
| TDI | = | tolerable daily intake (mg/kg-bw/d) |
| EDI | = | estimated daily intake (mg/kg-d) |
| RSC | = | risk-specific concentration ($mg/m^3$) = 0.00001/unit risk |
| SAF | = | soil allocation factor (dimensionless) |
| BW | = | body weight (kg) |
| BSC | = | background soil concentration (mg/kg) |
| $AF_G$ | = | relative absorption factor for the gut (dimensionless) |
| $AF_L$ | = | relative absorption factor for the lung (dimensionless) |
| $AF_S$ | = | relative absorption factor for the skin (dimensionless) |
| SIR | = | soil ingestion rate (kg/d) |
| Pa | = | particulate concentration in air ($kg/m^3$) = $7.6x10^{-10}$ |
| $IR_S$ | = | soil inhalation rate (kg/d) |
| SR | = | soil dermal contact rate (kg/d) |
| | = | (hand surface area x hand soil adherence factor) + (arm/leg surface area x arm/leg adherence factor) x events/day |
| $ET_1$ | = | exposure term 1 (dimensionless) – days per week/7 x weeks per year/52 |
| $ET_2$ | = | exposure term 2 (dimensionless) – hours per day/24 |

Where RSC = risk-specific concentration ($mg/m^3$) = 0.00001/Unit Risk

Input values are summarized in Table 4 below for each land use, along with the calculated guidelines.

**Table 4 $SQG_{DH}$ Input Values for Each Land Use**

| Parameter | Agricultural | Residential / Parkland | Commercial | Industrial |
|---|---|---|---|---|
| TDI (mg/kg-bw/d) | 0.0014 | 0.0014 | 0.0014 | 0.0014 |
| EDI (mg/kg-d) | 0.00063 | 0.00063 | 0.00063 | 0.00021 |
| Unit Risk $(mg/m^3)^{-1}$ | 9 | 9 | 9 | 9 |
| SAF | 0.2 | 0.2 | 0.2 | 0.2 |
| BW (kg) | 16.5 | 16.5 | 16.5 | 70.7 |
| BSC (mg/kg) | 10 | 10 | 10 | 10 |
| $AF_G$ | 1 | 1 | 1 | 1 |
| $AF_L$ | 1 | 1 | 1 | 1 |
| $AF_S$ | 0.01 | 0.01 | 0.01 | 0.01 |
| SIR (kg/d) | 0.00008 | 0.00008 | 0.00008 | 0.00002 |
| IRS (kg/d) | $7.07x10^{-9}$ | $7.07x10^{-9}$ | $7.07x10^{-9}$ | $1.20x10^{-8}$ |
| SR (kg/d) | 0.000069 | 0.000069 | 0.000069 | 0.00011 |
| $ET_1$ | 1 | 1 | 0.66 | 0.66 |
| $ET_2$ | 1 | 1 | 0.42 | 0.42 |
| **$SQG_{DH}$** | **42** | **42** | **58** | **1,200** |
| **$SQG_{HH-PI}$** | **14,000** | **14,000** | **50,000** | **50,000** |

### 7.2.3 Guideline for the Protection of Potable Groundwater

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

### 7.2.4 Guideline for the Protection of Indoor Air Quality

Cobalt is not a volatile chemical and therefore a guideline for the protection of indoor air quality is not required.

### 7.2.5 Produce, Meat and Milk Ingestion Check

The produce, meat and milk ingestion check is not normally calculated for inorganics (CCME, 2006). Bioaccumulation of inorganics in food is highly affected by soil chemistry as well as specific plant species and can vary significantly between sites.

This pathway should be evaluated on a site-specific basis if food crops are grown in cobalt-contaminated soils.

### 7.2.6 Off-site Migration Guidelines for Commercial and Industrial Land Uses

The guideline for offsite migration ($SQG_{OM-HH}$) is calculated for the commercial and industrial land uses to protect against transfer of contaminated soil to a more sensitive nearby property through processes such as wind and water erosion. CCME (2006) derived the following equation to evaluate this pathway, based on the Universal Soil Loss Equation and Wind Erosion Equation:

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

Where,

| | | |
|---|---|---|
| $SQG_{OM-HH}$ | = | human health soil quality guideline for off-site migration (mg/kg) |
| $SQG_A$ | = | soil quality guideline for agricultural land use (42 mg/kg) |
| BSC | = | background concentration of chemical in receiving soil (10 mg/kg) |

The resulting $SQG_{OM-HH}$ for commercial and industrial land uses is 470 mg/kg.

### 7.2.7 Discussion of Uncertainties Associates with the Human Health Soil Quality Guidelines

There is considered to be uncertainty in the oral TDI due to limited oral toxicity data; however the selected TDI was based on a conservative LOAEL and is expected to be protective.

Some components of the EDI are relatively uncertain due to the limited data on cobalt concentrations in the environment. However, the EDI is dominated by food ingestion, which has been characterized as part of Canada's Total Diet Study; exposure from other environmental media appears to be negligible, with the exception of soil ingestion which is well characterized. The EDI is also significantly lower than the toxicity benchmarks. Therefore the uncertainty in the EDI is not expected to affect the resulting soil quality guidelines.

Soil guidelines have not been calculated based on uptake by plants and subsequent ingestion by humans. This pathway is not normally quantitatively evaluated for inorganics (CCME 2006), but can be a significant source of exposure if crops are grown directly in contaminated soils. If food crops are

grown directly in soils with cobalt contamination in the rooting zone, then this pathway may need to be evaluated on a site-specific basis.

## 8.0 DERIVATION OF THE FINAL SOIL QUALITY GUIDELINE

The final soil quality guideline ($SQG_F$) considers both environmental and human health.

### 8.1 Considerations Other than Toxicity

If there is evidence that a contaminant may cause significant adverse effects other than toxicity to human and ecological receptors, this evidence may be used to derive a soil quality guideline for management considerations ($SQG_M$). This may include aesthetic concerns, damage to infrastructure, explosive hazards, or mobile free-phase liquid formation.

There is no indication that significant adverse effects other than toxicity are of concern for cobalt, and no $SQG_M$ is proposed.

### 8.2 Evaluation Against Geochemical Background and Practical Quantitation Limits

The proposed guidelines are above geochemical background at the majority of locations within Canada, and also above practical quantitation limits; therefore no adjustment of the guidelines for these factors is necessary.

### 8.3 Final Soil Quality Guidelines

The guidelines are summarized in Table 5 below.

| **Table 5 Soil Quality Guidelines for Cobalt** | | | | | |
|---|---|---|---|---|---|
| **Pathway** | **Natural Area** | **Agricultural** | **Residential/ Parkland** | **Commercial** | **Industrial** |
| **Guideline ($SQG_F$)** | **39** | **39** | **39** | **58** | **100** |
| *Human health guidelines* | | | | | |
| ***$SQG_{HH}$*** | ***NA*** | ***42*** | ***42*** | ***58*** | ***390*** |
| Direct Contact ($SQG_{DH}$) | NA | 42 | 42 | 58 | 390 |
| Protection of Indoor Air Quality ($SQG_{IAQ}$) | NA | NA | NA | NA | NA |
| Protection of Potable Water ($SQG_{PW}$) | NA | NA | NA | NA | NA |
| Off-site migration check ($SQG_{OM-HH}$) | NA | NA | NA | 470 | 470 |
| Produce, meat & milk check ($SQG_{FI}$) | NC | NC | NC | NC | NC |
| *Environmental health guidelines* | | | | | |
| ***$SQG_E$*** | **39** | **39** | **39** | **100** | **100** |
| Soil contact ($SQG_{SC}$) | 39 | 39 | 39 | 100 | 100 |
| Soil and food ingestion ($SQG_I$) | 880 | 2950 | NA | NA | NA |
| Protection of freshwater life ($SQG_{FL}$) | NA | NA | NA | NA | NA |
| Livestock watering ($SQG_{LW}$) | NA | NA | NA | NA | NA |
| Irrigation water ($SQG_{IR}$) | NA | NA | NA | NA | NA |
| Nutrient and energy cycling ($SQG_{NEC}$) | NC | NC | NC | NC | NC |
| Off-site migration check ($SQG_{OM-E}$) | NA | NA | NA | 470 | 470 |
| $SQG_M$ (non-toxicity considerations) | NA | NA | NA | NA | NA |

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