APPENDIX B: TOXICITY PROFILES

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1.0 TOXICITY PROFILES

1.1 Human Health Toxicity Profiles

1.1.1 Cadmium

1.1.1.1 Assessment of Carcinogenicity

Epidemiological studies demonstrate increased incidence of lung cancer in workers exposed to cadmium via the inhalation route; however, the studies did not control for factors such as smoking and simultaneous exposures to other metals so the causal relationship is somewhat controversial. Oral exposure to cadmium has not been associated with cancer in humans or animals.

The United States Environmental Protection Agency has classified cadmium as a probable human carcinogen (Group B2) when inhaled, based on limited human and sufficient animal data (US EPA 1985). Health Canada (1996a, b) has classified cadmium as a Group II carcinogen.

1.1.1.2 Susceptible Populations

Populations which may be unusually susceptible to cadmium exposure are those with a genetic predisposition to lower inducibility of metallothionein, the enzyme which sequesters cadmium. Dietary deficiencies which lead to depleted levels of calcium or iron in individuals may result in increased absorption of cadmium from the gastrointestinal tract. Infants and children may have increased uptake of cadmium via the gastrointestinal tract and higher concentrations of cadmium in the bone.

1.1.1.3 Selection of Toxicity Values

ATSDR (1998) has developed a chronic oral minimum risk level (MRL) of 0.2 µg/kg-day for cadmium. The chronic MRL is derived from a No Observed Adverse Effect Level (NOAEL) of 2.1 µg/kg-day from a study of cadmium accumulation in the kidneys of Japanese farmers living in an area of Japan with highly elevated cadmium levels. An uncertainty factor of 10 was used to account for variability in the human population.

The United States Environmental Protection Agency has developed oral reference doses for cadmium for food and water. The RfD_o for food is 1.0 µg/kg-day and for water is 0.5 µg/kg bw-day (US EPA 1994a). The highest cadmium level in the human kidney which does not produce proteinuria (excretion of low weight molecular proteins into the urine) has been determined to be 200 µg Cd/g of wet kidney cortex. A toxicokinetic model was used to determine the level of chronic oral exposure that would result in a cadmium kidney concentration of 200 µg Cd/g of wet kidney cortex. The toxicokinetic model assumes that 0.01% of the body cadmium kidney burden is eliminated daily and that absorption of

cadmium from food and water are 2.5% and 5% respectively. The NOAELs for chronic cadmium exposure were determined to be 5.0 and 10 µg/kg-day for food and water, respectively. An uncertainty factor of 10 to account for human variability was applied to the NOAELs to develop the reference doses for food and water.

The Joint Expert Committee on Food Additives (WHO 1993) proposed that the total daily intake of cadmium should not exceed 1 µg/kg bw-day. This intake was designed to keep the cadmium levels in the renal cortex below 50 µg/g, and assumed an absorption rate for dietary cadmium of 5% and a daily excretion rate of 0.005% of body burden.

Health Canada (1996a) has adopted the 1 µg/kg bw-day value as a provisional tolerable daily intake (PTDI) for both children and adults.

The US EPA (1994a) has developed an inhalation unit risk of 1.8×10^{-3} $(\mu g/m^3)^{-1}$. This unit risk is based on lung and upper respiratory tract cancers in cadmium production workers (Thun *et al.*, 1985). The air concentration at the 10^{-5} lifetime cancer risk level (1-in-100,000) is 0.006 $\mu g/m^3$.

The World Health Organization (WHO 2000) has an annual guideline value (noncancer) of 0.005 $\mu g/m^3$. Health Canada (1996b) has calculated a TC_{05} of 5.1 $\mu g/m^3$. This TC_{05} can be converted to an inhalation unit risk value of 9.8×10^{-3} $(\mu g/m^3)^{-1}$ using a target risk value of 1×10^{6}, and converted to an inhalation slope factor using an inhalation rate of 16.2 m^3/day and a body weight of 70.7 kg.

The estimate of non-cancer oral potency from Health Canada (PTDI) will be used to assess potential hazards associated with oral exposures to cadmium in this report. The Health Canada TC_{05} provides a more conservative unit risk estimate of the potency of inhaled cadmium. Both studies appear to be based on the same data. Therefore the inhalation unit risk derived from the Health Canada TC_{05} will be used to assess potential cancer risks associated with inhalation exposures to cadmium. The toxicity values used to assess the potential risks associated with ingestion and inhalation exposure to cadmium are summarized in Table B-1.

Table B-1 Selected Toxicity Values for Cadmium

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Ingestion – toddler and adult	1 µg/kg bw-day	Kidney damage in humans	Health Canada, 1996a
Inhalation	NA		
Cancer Effects			
Ingestion	NA		
Inhalation	9.8×10^{-3} $(\mu g/m^3)^{-1}$	Lung cancer in cadmium workers	Health Canada, 1996b

The biological end-point of greatest concern differs between oral and inhalation exposures. Therefore, the potential hazards associated with oral exposures and the potential cancer risks associated with inhalation exposures to cadmium will be assessed separately.

1.1.1.4 Bioavailability of Cadmium

Oral Route of Exposure

Oral absorption of cadmium in humans generally is reported to be very low (1 to 7 percent) (ATSDR 1997). The total retention of cadmium in the bodies of humans has been measured following ingestion of radioactive cadmium. About 25% of a dose of cadmium administered mixed with food to 5 healthy adults was retained after 3-5 days, but retention decreased to about 6% after about 20 days (Rahola *et al.* 1973). Similar results were obtained with 14 healthy adults, with an average of 4.6% of cadmium chloride in water taken with a meal retained in the body l-2 weeks after a simultaneously administered fecal marker (trivalent chromium) had been completely excreted (McLellan *et al.* 1978). The influence of chemical complexation of cadmium on human absorption was evaluated in seven volunteers who ingested brown crab meat (hepatopancreas) that had been labeled with radioactive cadmium chloride by prior feeding of the crabs (Newton *et al.* 1984). Whole-body counting was used to evaluate uptake. Whole-body retention in the volunteers ranged from 1.2 to 7.6% with a mean of 2.7% (Newton *et al.* 1984), only slightly lower than the values of 4.6-6% obtained using dissolved cadmium ion (McLellan *et al.* 1978; Rahola *et al.* 1973). These results indicated that, in general, cadmium absorption from food is not dependent on chemical complexation.

Evidence that the bioavailability of cadmium in soil may be reduced compared to the bioavailability of soluble cadmium forms is available from a limited number of studies. Several studies have reported reduced oral bioavailability of a soluble cadmium form, cadmium chloride, mixed with soil (Griffin *et al.* 1990; Schilderman *et al.* 1997). For cadmium in weathered soil, data are available for soil from a single site (the site of a former zinc smelter) that has been evaluated in vivo in rats (Schoof and Freeman 1995; PTI 1994). A relative cadmium bioavailability estimate of 33 percent was obtained based on comparison of liver and kidney tissue concentrations in animals fed rodent chow mixed with soil, versus those fed rodent chow mixed with cadmium chloride.

Dermal Route of Exposure

An in vitro study of dermal absorption in human cadaver skin of cadmium chloride mixed with soil yielded an estimate of 0.02 to 0.07 percent absorption based on cadmium in receptor fluid (Wester *et al.* 1992). An additional 0.06 to 0.13 percent of the dose was retained in the skin. The US EPA default value of 1.0 percent for dermal absorption of cadmium compounds from soil is more than 10 times higher than the maximum percent of the cadmium chloride dose reaching the receptor fluid and 5 times

higher than the maximum combined percent dose in receptor fluid and skin. Dermal absorption of cadmium from weathered soils may be even lower.

Selected Values

Based on the above review, oral bioavailability is set at 10% to ensure a protective assessment. Dermal bioavailability is set at the US EPA default of 1% which is likely to overestimate dermal absorption.

1.1.2 Lead

1.1.2.1 Assessment of Carcinogenicity

Epidemiological studies of occupationally exposed adults were not able to demonstrate an increase in cancers among an exposed population compared to a control group. The International Agency for Research on Cancer considers the overall evidence of lead carcinogenicity in humans to be inadequate. The US EPA (1993) has classified lead as a probable human carcinogen based on sufficient animal evidence but did not recommend derivation of a quantitative estimate of oral carcinogenic risk due to a lack of understanding of the toxicological and pharmacokinetic characteristics of lead. Neurobehavioural effects of lead in children were considered to be the most relevant endpoints in determining a toxicity value.

CCME (1992) classified lead as Group IIIB – possibly carcinogenic to man (inadequate data in humans, limited evidence in animals) according to the classification scheme of the Environmental Health Directorate of Health and Welfare Canada. Chemicals classified in Group IIIB are treated as non-carcinogens and are evaluated against a Tolerable Daily Intake (TDI), based on a NOAEL.

1.1.2.2 Susceptible Populations

There is a very large database that documents the effects of acute and chronic lead exposure in adults and children. Extensive summaries of the human health effects of lead are available from a number of sources including Health Canada (1996c), the US EPA Integrated Risk Information System (IRIS) database and the Association for Toxic Substances and Disease Registry (ATSDR). These reviews show that infants, young children up to the age of six, and pregnant women (developing fetuses) are the most susceptible.

1.1.2.3 Selection of Toxicity Values

The WHO (1986) established a provisional tolerable weekly intake (PTWI) for children of 25 μg/kg bw, equivalent to approximately 3.57 μg/kg bw-day from all sources. This PTWI was established based on metabolic studies in infants (Ryu *et al.* 1983; Ziegler *et al.* 1978) showing that a mean daily intake of 3-

4 µg Pb/kg body weight was a NOAEL and was not associated with an increase in blood lead levels or in the body burden of lead. WHO more recently extended this PTWI to all age groups to protect other sensitive population groups, such as women of child-bearing age.

The Ontario Ministry of Environment and Energy (MOEE 1994) derived a lead intake of concern (IOC) for a toddler of 3.7 µg/kg bw-day based on a blood lead level of concern of 10 µg/dL, in close agreement with the values derived by the WHO. The OMOE applied an uncertainty factor of 2 to derive an IOC for populations of 1.85 µg/kg bw-day, intended to represent a maximum intake not to be exceeded. As noted by MOEE, there was no scientific rationale for the size of the uncertainty factor, only judgement based on the quality and quantity of data.

Health Canada (1996c) adopted the WHO toxicity value for the derivation of the CCME Soil Quality Guidelines. The toxicity values used to assess the potential risks associated with ingestion and inhalation exposure to lead are summarized in Table B-2.

Table B-2 Selected Toxicity Values for Lead

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Ingestion	3.57 µg/kg bw-day	Blood lead level in young children	Health Canada (1996c)
Inhalation	3.57 µg/kg bw-day	Blood lead level in young children	Health Canada (1996c)
Cancer Effects			
Ingestion	NA		
Inhalation	NA		

1.1.2.4 Bioavailability of Lead

Oral Route of Exposure

Gastrointestinal absorption of lead varies with the age, diet, and nutritional status of the subject, as well as with the chemical species and the particle size of lead that is administered (ATSDR 1993). Age is a well-established determinant of lead absorption; adults typically absorb 7 to 15 percent of lead ingested from dietary sources, while estimates of lead absorption from dietary sources in infants and children range from 40 to 53 percent. Estimates derived from dietary balance studies conducted in infants and children (ages 2 weeks to 8 years) indicate absorption of approximately 40–50% of ingested lead (Alexander *et al.* 1974; Ziegler *et al.* 1978).

Soil lead absorption has been studied in rats, swine, and humans. The swine model has been used to test soils from numerous sites. A physiologically based extraction method is also well developed (Ruby *et al.* 1993, 1996; Medlin 1997) and is undergoing detailed validation studies. The studies in rats and swine have indicated that absorption of lead from soil will vary with the source of the lead, ranging from

near zero to greater than 50 percent absolute bioavailability (*i.e.*, relative bioavailability of 1.0, or more compared to soluble lead forms) (Casteel *et al.* 1997; Dieter *et al.* 1993; Freeman *et al.* 1992, 1996; Schoof *et al.* 1995; US EPA 1996b-e, 1998a-e). On average, the results of these studies support the use of a default assumption that 30 percent of an oral lead dose is absorbed from soil (*i.e.*, relative bioavailability of 0.6). A study in adult humans indicates that absolute lead bioavailability from a mining-area soil varies from approximately 3 to 26 percent, depending on how recently the test subject had eaten (Maddaloni *et al.* 1998).

The absorption of lead in soil is less than that of dissolved lead. In US EPA's childhood lead model, it is assumed that 50 percent of an oral lead dose is absorbed from food and water, while 30 percent of a soil lead dose is assumed to be absorbed. Thus, the default assumption for lead is that the relative bioavailability of soil lead compared to soluble lead forms is 0.6 (*i.e.*, 30 percent divided by 50 percent) (US EPA 1994b). Adult subjects who ingested soil (particle size less than 250 μm) from the Bunker Hill NPL site absorbed 26% of the resulting 250 μg/70 kg body weight lead dose when the soil was ingested in the fasted state and 2.5% when the same soil lead dose was ingested with a meal (Maddaloni *et al.* 1998). Additional evidence for a lower absorption of soil-borne lead compared to dissolved lead is provided from studies in laboratory animal models. In immature swine that received oral doses of soil from one of four NPL sites (75 or 225 μg Pb/kg body weight), bioavailability of soil-borne lead ranged from 50% to 82% of that of a similar dose of highly water soluble lead acetate (Table 2-5) (Casteel *et al.* 1997; US EPA 1996a, 1996b, 1996c). If the relative bioavailability of soil-borne lead (soil/acetate) in immature swine is indicative of the relative bioavailability in human children, and if the absolute bioavailability of water soluble lead in humans children is 50%, as the Alexander *et al.* (1974) and Ziegler *et al.* (1978) studies would suggest, then the absolute bioavailability of soil-borne lead in human children predicted from the swine studies would range from 25 to 41%.

Dermal Route of Exposure

Limited information is available regarding absorption after dermal exposure in humans. Dermal absorption of inorganic lead compounds is reported to be much less significant than absorption by inhalation or oral routes of exposure, because of the greatly reduced dermal absorption rate (US EPA 1986). Following skin application of ^{203}Pb-labeled lead acetate in cosmetic preparations (0.1 mL of a lotion containing 6 mmol lead acetate/L or 0.1 g of a cream containing 9 mmol lead acetate/kg) to 8 male volunteers for 12 hours, absorption was ~0.3%, but expected to be 0.06% during normal use of such preparations (Moore *et al.* 1980). Most of the absorption took place by 12 hours of exposure. The US EPA default value of for dermal absorption of lead compounds from soil is 1%.

Selected Values

Based on the above review, absolute oral bioavailability for lead through food ingestion is set at 50% to ensure a protective assessment for young children. In accordance with the US EPA recommendations and the above review, the relative bioavailability of lead from soil is 60%, yielding an absolute soil bioavailability of 30%. Dermal bioavailability is set at the US EPA default of 1% which is likely to overestimate dermal absorption.

1.1.3 Zinc

1.1.3.1 Assessment of Carcinogenicity

Epidemiological studies of workers exposed to zinc have not shown a relationship between zinc exposure and the development of cancer (ATSDR 1994). Animal studies have also not shown a link between inhalation, oral or dermal exposure to zinc and an increase in the incidence of cancers (ATSDR 1994). Based on inadequate evidence in humans and animals, US EPA classified zinc as Class D; not classifiable as to human carcinogenicity (US EPA 1992). Health Canada (1996b) and CCME (1987) do not consider zinc as a human carcinogen.

1.1.3.2 Susceptible Populations

There is no specific information regarding the existence of human subpopulations that are sensitive to the toxic effects of zinc; however there is limited indication that people who are malnourished or have marginal copper status may be more susceptible to excessive zinc exposures than the average population (ATSDR 1994).

1.1.3.3 Selection of Toxicity Values

CCME considers zinc to be an essential nutrient and has not developed an oral TDI to account for environmental exposures to zinc (CCME 1987).

The US EPA has developed an RfD_o of 300 µg/kg bw-day based on a clinical study of females taking zinc as a dietary supplement (US EPA 1992). The study reported a decrease in the level of erythrocyte superoxide dismutase in study participants. An uncertainty factor of 3 was applied to the NOEL from the study to account for sensitive members of the population (US EPA 1992).

Neither the US EPA nor Health Canada has developed reference concentration values for inhalation exposures to zinc. The oral reference dose developed by the US EPA has been selected for assessing the potential hazards associated with oral exposures to zinc. The toxicity values used to assess the potential risks associated with ingestion and inhalation exposure to zinc are summarized in Table B-3.

Table B-3 Selected Toxicity Values for Zinc

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Ingestion	300 μg/kg bw-day	Decreased erythrocyte SOD	US EPA (1992)
Inhalation	NA	NA	NA
Cancer Effects			
Ingestion	NA		
Inhalation	NA		

1.1.3.4 Bioavailability of Zinc

Oral Route of Exposure

Several studies have measured oral absorption rates of zinc in humans. Absorption ranged from 8% to 81% following short-term exposures to zinc supplements in the diet; differences in absorption are probably due to the type of diet (amount of zinc ingested, amount and kind of food eaten) (Aamodt *et al.* 1983; Hunt *et al.* 1991; Istfan *et al.* 1983; Reinhold 1991; Sandstrom and Abrahamson 1989; Sandstrom and Cederblad 1980; Sandstrom and Sandberg 1992). The body's natural homeostatic mechanisms control zinc absorption from the gastrointestinal tract (Davies 1980). Persons with adequate nutritional levels of zinc absorb approximately 20-30% of all ingested zinc. Those who are zinc-deficient absorb greater proportions of administered zinc (Johnson *et al.* 1988; Spencer *et al.* 1985).

Dermal Route of Exposure

Dermal absorption of zinc occurs, but its mechanism is not clearly defined. Studies are very limited regarding the absorption of zinc through the skin. Historically, zinc oxide has been used clinically to promote the healing of burns and wounds (Gordon *et al.* 1981). Absorption has been observed in burn patients treated with gauze dressings containing zinc oxide (Hallmans 1977).

Selected Values

Based on the above review, oral bioavailability is set at 80% to ensure a protective assessment. Dermal bioavailability is set at the US EPA default of 1% which is likely to overestimate dermal absorption.

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1.2 Ecological Health Toxicity Profiles

1.2.1 Cadmium

Cadmium (Cd) is a eavy metal that does not appear to be biologically essential to wildlife. Background concentrations of cadmium in Canadian soils range from not detectable to as high as 8.1 mg/kg (CCME 1999). In Ontario, for surface soils from rural parklands remote from pollution point-sources, the 98th percentile for cadmium was 0.71 mg/kg (MOEE 1993).

Cadmium is a known teratogen, carcinogen, and a probable mutagen in some terrestrial and aquatic biota (Hoffman *et al.* 1995). Exposure to low concentrations of cadmium may result in adverse effects in a wide range of ecological receptors. Although biomagnification of cadmium in the food chain has not been demonstrated, cadmium does bioaccumulate in invertebrate and vertebrate species, microorganisms, and plants.

The bioavailability of cadmium is dependent on its chemical form as well as other environmental conditions such as pH, clay content, particle size, hardness, and organic carbon. The free ionic form of cadmium is more toxic to aquatic biota than cadmium that is complexed with dissolved organic matter or with soluble particulate matter or colloidal matter. Soils and sediments tend to attract cadmium, due to the low solubility and volatility of cadmium. Based on its persistence in the environment and its toxicity to wildlife, cadmium contamination may present a significant threat to ecosystems at all levels of biological organization.

Cadmium can be present in soil as free cadmium compounds or in solution as the Cd^{2+} ion dissolved in interstitial water (ATSDR 1993). The speciation of cadmium in soils is dependent on the soil geochemistry. For soil types ranging from sand to silty clay loam, the adsorption of Cd^{2+} is highly

dependent on pH. As pH increases, so too does cadmium sorption. Under acidic conditions, cadmium is more likely to move within the soil matrix and into other media. Cadmium tends to sorb more strongly to organic matter than to metal oxides, such as iron, aluminum and manganese oxides. Movement of cadmium within soils is also affected by wind transport, leaching and uptake by terrestrial organisms. In acidic soils, the release of Cd^{2+} and its uptake by plants is favored (ATSDR 1993).

In the aqueous environment, cadmium is relatively mobile, and can exist as the hydrated ion, as inorganic complexes, and as organic complexes. Cadmium tends to complex with dissolved organic carbon and also in association with colloidal and particulate matter.

Terrestrial plants and animals have been shown to be adversely affected by exposure to cadmium. In terrestrial plants, uptake of cadmium has been demonstrated, although uptake rates and translocation of cadmium are variable (CCME 1999). The lowest soil cadmium concentrations at which toxicity to plants have been observed are 2.5 and 4 mg/kg (CCME 1999).

Adverse effects of cadmium ingestion have been observed in various mammals and bird species, and include reduction of food intake and growth rate, impaired reproduction and mortality (Environment Canada 1996). Animals have a limited ability to eliminate cadmium from their bodies, and cadmium has been shown to accumulate primarily in the liver and renal cortex.

Many studies have demonstrated uptake of cadmium by earthworms in contaminated soils, and adverse effects to chronic earthworm endpoints were indicated by cadmium concentrations ranging from 25 mg/kg to 108 mg/kg soil (summarized in Sample *et al.* 1997). The bioconcentration factor from soil to earthworms has been shown to be negatively correlated with soil pH, presumably because the lowering of pH leads to increased desorption of metal cations, such as cadmium, thereby increasing bioavailability.

In laboratory rats, reduced growth has been observed from a dietary exposure of 7.15 mg/kg (Weigel at al. 1984 in Sample *et al.* 1997). Reproductive effects on rats have been associated with drinking water exposures as low as 1 mg/kg $CdCl_2$, and in mice from drinking water exposures of 10 mg/L Cd (as $CdCl_2$)(summarized in Sample *et al.* 1997). For dietary exposures, reproductive effects on rats have been observed from exposures as low as 0.125 mg/kg $CdCl_2$ over four generations (Wills *et al.* 1981 in Sample *et al.* 1997). For exposure via oral gavage, the lowest observed adverse effects level for reproduction in rats was reported to be 10 mg/kg body weight per day (Sutoe *et al.* 1980 in Sample *et al.* 1996). Reproductive effects in avian species have also been reported. Mallard duck hens orally exposed to cadmium at 19 mg/kg-day for 90 days exhibited suppressed egg production (White and Finley 1978).

The soil quality guideline for cadmium for the protection of ecological health has been determined to be 10 mg/kg (based on soil contact) by CCME (1999).

1.2.2 Copper

Copper (Cu) is a metal that occurs naturally in rock, soil, water, sediment, air, and biota. Copper can occur in four oxidation states (Cu, Cu^{+}, Cu^{2+} and Cu^{3+}), although Cu^{2+} is the most common state, particularly in oxidizing conditions. Copper is an essential nutrient for all living organisms; however, long-term exposure to elevated or deficient levels of copper may result in adverse effects in plants and animals (CCME 1999).

Copper is used widely in the manufacturing industry and occurs in a wide range of mineral deposits as both a primary and secondary mineral. Most copper occurs in the form of sulphide minerals. The average copper concentration in Canadian soil is estimated to be 20 mg/kg, with a range from 2 to 100 mg/kg (BCMELP 1992). In surface soils from rural parkland remote from pollution point-sources, the 98^{th} percentile was 41 mg/kg for rural parkland (MOEE 1993).

In soils, most copper is in mineral form or strongly bound to particles, and therefore has low bioavailability. However, copper may be solubilized and thereby made more available for uptake by plants and animals. Factors that influence the availability of copper in soils are pH, cation exchange capacity (CEC), organic matter content, reduction-oxidation potential and the presence of metal oxides (e.g., as iron, manganese and aluminum oxides). Copper is more mobile in acidic soils. In general, copper will be immobilized in soils with high CEC and organic matter content. The CEC of soils is influenced by pH and clay and organic matter content. Copper is specifically adsorbed by iron, manganese and aluminum oxides, and binds more strongly that most other metals. However, under reducing conditions, copper bound to these metal oxides will be solubilized.

Copper has been demonstrated to be potentially toxic to plants and animals. Toxicity to terrestrial plants has been shown to occur at concentrations ranging from 100 mg/kg to 200 mg/kg copper in soil (summarized in Sample *et al.* 1997). Copper bioaccumulates in plants to a small degree.

Animals have the ability to regulate internal concentrations of copper. Nevertheless, adverse reactions by wildlife have been shown to occur in association with high copper and deficient copper concentrations.

Studies have shown that earthworms are adversely affected by copper concentrations in soil as low as 68 mg/kg, and also show that the organic content of the soil is a strong determinant of copper availability and toxicity to litter invertebrates (summarized in Sample *et al.* 1997). In mink, the lowest observed adverse effect level was estimated to be 15 mg/kg-day, based on an increased mortality rate in offspring of mink fed a diet supplemented with copper (Sample *et al.* 1996). In chicks, dietary copper levels from 588 to 1176 mg/kg for 10 days resulted in growth effects, with 500 mg/kg estimated to be the minimum toxic level, corresponding to a lowest observed adverse effect level of 61.7 mg/kg-day (Mehring *et al.* 1960 in Sample *et al.* 1996).

The soil quality guideline for copper for the protection of ecological health has been determined to be 63 mg/kg (based on soil contact) by CCME (1999).

1.2.3 Lead

Lead is a non-essential and highly toxic heavy metal. Natural environments rarely contain lead in its elemental form. However, lead is present in low concentrations throughout the environment as a result anthropogenic inputs (e.g., from animal wastes, coal residues, incineration of municipal wastes, waste waters and automobile emissions) and natural sources (e.g., volcanoes, forest fires, sea salt, geologic weathering). In Canada, mean background levels of lead in soils remote from ore bodies have been estimated to range from 12 to 25 mg/kg (CCME 1999). In surface soils from rural parklands remote from pollution point-sources, the 98th percentile was 45 mg/kg (MOEE 1993). Near lead emissions sources such as smelters, lead concentrations in excess of 700 mg/kg in soil have been found (CCME 1999).

In soils, lead can pose a threat if it is mobile and moves to surface or groundwaters, or into biota. Lead form and mobility in soils is influenced by pH, soil texture and organic matter. The dissolved form of lead in soils is Pb^{2+}, and therefore the cation exchange capacity (CEC) of the soil can decrease its mobility in the short term (CCME 1999). Soil erosion by wind or water is a mechanism by which lead can be mobilized from soil to contaminate the surrounding environment.

The most toxic forms of lead are considered to be the organolead compounds, the most important of which are tetramethyl and tetraethyl lead. Most of the lead that is ingested by biota is rapidly egested (Eisler 1988). By contrast, inhaled lead is quickly absorbed into the bloodstream (ATSDR 1993). Lead has been shown to bioconcentrate and, as a result, older organisms tend to have the highest body burdens. However, lead does not appear to biomagnify in the food chain (Eisler 1988).

In plants, significant adverse effects are generally only seen at relatively high lead levels. For example, root and shoot growth was reduced in red spruce when exposed to 150 mg/kg lead in soil (Seiler and Paganelli 1987). Uptake and accumulation rates in plants are related to soil pH and vary among and within species. The decline of some European spruce forests has been attributed to excessive concentrations of atmospheric lead. Reported effects include inhibition of plant growth, and reductions in germination, seed viability, and rates of photosynthesis and transpiration (Hoffman *et al.* 1995).

In animals and birds, lead acts to inhibit enzymes necessary for normal biological function. In mammals, lead toxicity may affect the hematological system, the brain and nervous system, learning and behavior, and reproduction (Hoffman *et al.* 1995).

In rats, oral doses of 80 mg/kg-day have been estimated to be the lowest observed level associated with reproductive impairment (Azar *et al.* 1973 in Sample *et al.* 1996). In birds, reproductive and

developmental effects include decreases in egg hatching success at an estimated lowest observed adverse effect concentration of 11.3 mg/kg-day via oral exposures in Japanese quail (Edens *et al.* 1976 in Sample *et al.* 1996). Earthworms accumulate lead, but total lead concentrations in earthworms are almost always well below that in soil (CCME 1999). Earthworms, like all soil invertebrates, cycle soil through their bodies, but assimilate low net amounts of lead compared with other trace elements, such as cadmium (CCME 1999).

The soil quality guideline for lead for the protection of ecological health has been determined to be 300 mg/kg (based on soil contact) by CCME (1999).

1.2.4 Silver

Silver is a naturally occurring element in the earth's crust at an average concentration of approximately 0.1 mg/kg, and is generally present at low concentrations (Purcell and Peters 1998). Although there are natural deposits, silver is primarily found on land where it has been deposited because of human activity, in the atmosphere as a result of smelting and coal burning activities, and in aquatic systems as a result of discharges from mining, industry or sewage treatment plants. In Canada, the average silver content in soils remote from mineralized zones has been reported to be 0.30 mg/kg (Boyle 1968 in ATSDR 1990).

Silver occurs in four oxidation states: Ag, Ag^{+}, Ag^{2+} and Ag^{3+}, with Ag and Ag^{+} being the most common. Most silver minerals are compounds of silver with sulfur or the homologues and neighbours on the periodic chart. In soils, silver is primarily found in sulfide form in association with iron, lead or tellurides. In surface waters, silver can be found in a number of forms: monovalent ion, sulfide, bicarbonate, sulfate salts, or adsorbed onto organic or inorganic materials. Many of these forms are insoluble and as a result, their bioavailability is not great. The potential for effects in the environment is dependent on the chemical form of silver.

The mobility of silver in the environment depends on its physical and chemical form, as well as physico-chemical conditions in surrounding media (e.g., soil, sediment, and water). The majority (>94%) of silver released into the environment is expected to remain in the soil or wastewater sludge at the emission site (Ratte 1999). The remainder will be transported via air and water. In water, silver is found as a free monovalent ion, as part of a chloride or sulfide compound, or adsorbed onto particulate matter. The bioavailability of silver in soils is largely dependent on environmental factors such as drainage, oxidation-reduction potential, pH, and organic matter content. Silver partitions preferentially into the particulate phase and forms a variety of organic-inorganic solids. The bioaccumulation of silver in soil appears to be low (Ratte 1999).

Toxicity of silver occurs mainly in the aqueous phase and depends on the concentration of free Ag^{+} ions. Studies on silver toxicity are almost all based on the free ion, but this does not represent the form most likely to be encountered in the environment. Most recent studies of silver compounds conclude that they

are much less toxic than silver ions (Purcell and Peters 1998). In soil, the toxicity of silver, even at high total concentrations, is very low (Ratte 1999).

In higher plants and fungi, silver is only expected to accumulate in contaminated areas, such as mine tailings and soils amended with sewage sludge that contains silver. The sensitivity of terrestrial plant species to silver exposure varies, and includes effects on growth and germination to sensitive species at a concentration of 7.5 mg/L silver nitrate (Ratte 1999).

Silver toxicity in terrestrial animals has been investigated mainly in laboratory bioassays. Oral exposure to high doses of silver in drinking water killed rats and dermal exposures resulted in reduced weight gain in guinea pigs. The lowest observed adverse effects level for rats was 362 mg/kg-day (death; two week exposure period)(Walker 1971 in ATSDR 1990) and for guinea pigs was 137.13 mg/kg-day (reduced weight gain; eight week exposure period)(Wahlberg 1965 in ATSDR 1990). There is no substantial potential for bioaccumulation in mammals (Ratte 1999). Birds have been shown to bioaccumulate silver in their livers (Eisler 1996 in Ratte 1999). Silver toxicity to terrestrial invertebrates has been investigated mainly using earthworms. Neither carcinogenic nor mutagenic effects are suggested from silver exposure. No studies demonstrating adverse chronic effects to reproductive and developmental endpoints in mammals have been identified (ATSDR 1990; Ratte 1999).

Although no Canadian soil quality guideline has been developed to date for silver for the protection of ecological health, the Ontario Ministry of Environment and Energy has developed a guideline of 20 mg/kg in soil (MOEE 1993).

1.2.5 Zinc

Zinc is a naturally-occurring common metallic element. In Canada, the mean level of zinc in soils is reported to be 74 mg/kg, with average concentrations in various regions ranging from 54 to 81 mg/kg (CCME 1999). In surface soils from rural parklands remote from pollution point-sources, the 98th percentile was 120 mg/kg (MOEE 1993). The continental crustal zinc content is reported to be 52 mg/kg in the upper layer (Wedepohl 1995). Zinc is primarily used for galvanizing and in the manufacture of brass and bronze and for heating and cooling system components (CCME 1999).

In the environment, zinc occurs primarily in the Zn^{2+} oxidation state. Zinc tends to strongly react with organic and inorganic compounds and forms stable combinations with many organic substances and a wide range of biochemical compounds. Zinc is an essential element and is naturally used in many biochemical compounds. In soils, zinc is highly reactive and forms both soluble and insoluble organic complexes. Zinc can also be adsorbed to clay or metallic oxides and may also be part of the parent soil material. Soil pH has been shown to be a main factor in controlling zinc mobility and sorption in soils; as pH decreases, zinc becomes more soluble and bioavailable to organisms. However, there are other factors that contribute to zinc mobility in soils, such as plant uptake, leaching, moisture content,

reduction-oxidation potential, and mineralization of organic matter (CCME 1999). In anaerobic soils, zinc sulfide controls the mobility of zinc (ATSDR 1994). Zinc sulfide is insoluble, and as a result, zinc mobility and bioavailability in anaerobic soils is low.

In unpolluted waters, zinc exists mostly in the hydrated divalent cationic form, and in polluted waters zinc often forms organic and inorganic complexes (ATSDR 1994). The hydrated divalent cationic form of zinc is much more toxic to aquatic biota than zinc that is complexed with dissolved organic matter or with particulate or colloidal matter. Bioconcentration of zinc in aquatic organisms is relatively high, though it is much lower in terrestrial organisms, and biomagnification does not occur in either terrestrial or aquatic food chains (ATSDR 1994).

Because zinc is an essential element, organisms can regulate internal concentrations and therefore tend to be relatively tolerant of high exposure concentrations. Adverse effects of zinc on terrestrial plants have been observed at 50 mg zinc/kg dry soil (reduction in seed yield in turnips; in CCME 1999).

Mammals have been shown to be tolerant tolerant to intake rates of zinc up to levels 100 times greater than the minimum recommended daily requirement (Eisler 1993). The primary toxic effects of zinc are on zinc-dependent enzymes that regulate RNA and DNA. Eisler (1993) reported a number of adverse reproductive, developmental and survival effects on animals resulting from chronic dietary exposures to zinc ranging from 500 to over 6000 mg/kg for rats and mice and at 3000 mg/kg for Mallard duck. Schlicker and Cox (1986, in Sample *et al.* 1996) demonstrated reproductive effects in rats at a lowest observed adverse effect level of 320 mg/kg-day zinc in diet. In birds, Stahl *et al.* (1990, in Sample *et al.* 1996) demonstrated reproductive effects to white leghorn hens from a dietary dose of 131 mg/kg-day (lowest observed adverse effects level). The lowest effect concentration reported for domestic mammals was a dietary exposure of 750 mg zinc/kg for Cheviot sheep, resulting in a 64% reduction in the number of viable offspring (in CCME 1999). For earthworms, an LC50 of 80 mg zinc/kg dry soil has been reported, however, the lethal concentration has been shown to be highly dependent upon soil pH (CCME 1999).

The soil quality guideline for zinc for the protection of ecological health has been determined to be 200 mg/kg (based on soil contact) by CCME (1999).

1.2.6 References for Ecological Health Toxicity Profiles

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