

# FINAL REPORT

Human Health And Ecological Risk  
Assessment For The CAM-D (Simpson Lake)  
Distant Early Warning Line Site

PUBLIC WORKS AND  
GOVERNMENT SERVICES CANADA

PROJECT NO. 1003732



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## **PROJECT NO. 1003732**

**REPORT TO: Public Works and Government Services Canada  
& Department of Indian Affairs and Northern  
Development**

**FOR: Human Health and Ecological Risk Assessment for  
the CAM-D Intermediate Distant Early Warning Line  
Site, Simpson Lake, Nunavut**

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Jacques Whitford Limited  
708 – 11<sup>th</sup> Avenue SW, Suite 500  
Calgary, Alberta  
T2R 0E4

Phone: 403-263-7113  
Fax: 403-263-7116

[www.jacqueswhitford.com](http://www.jacqueswhitford.com)



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## EXECUTIVE SUMMARY

Jacques Whitford Limited (Jacques Whitford) performed a human health and ecological risk assessment (HHERA) for the CAM-D Distant Early Warning (DEW) Line site located in the middle of the Boothia Peninsula, 4.5 km south of Simpson Lake, Nunavut. The primary objective of this study was to evaluate whether known concentrations of chemicals found in on-site surface soil, water and vegetation would present a significant risk to human or ecological health based on future use of the area.

### Study Background

In the present study, a preliminary quantitative human and ecological risk assessment (HHERA) was conducted for the CAM-D site. It is supported primarily by new contaminant data, based on a phase III environmental site assessment (ESA; Earth Tech, 2005) which included analyses for hydrocarbons, PAHs, PCBs and inorganic substances in surface soils, water, and vegetation. The HHERA was also supported with previous data from the Phase II site assessment (RRC, 1994) which was combined with the recent data for statistical analysis and exposure point concentration (EPC) calculations.

### Data Compilation

All Phase II and Phase III data were assessed in this HHERA. Soil samples were screened on the basis of depth and any sample that did not intersect the surface and/or extended to a depth of greater than 0.3 m below ground surface (mbgs) was excluded. This was carried out to ensure that the data used in the HHERA were representative of surface soils, to which human and ecological receptors are exposed, and not heavily influenced by subsurface soil characteristics.

### Screening for Chemicals of Potential Concern

Chemicals of potential concern (CoPCs) included for consideration were those identified as exceeding generic soil and/or water quality guidelines (e.g., CCME). Generic CCME guidelines may be based on either ecological or human health protection and provide a means for initial protective screening of data. For the human health risk assessment (HHRA), chemicals were screened specifically against human health-based generic guidelines whereas for the ecological risk assessment they were screened specifically against ecologically-based generic guidelines. In order of preference, these guidelines were obtained from CCME (CCME, 1999), the Ontario Ministry of the Environment (OME, 1996a) or the United States Environmental Protection Agency (US EPA). A number of metals were also screened using Dew Line Cleanup (DLCU) criteria (INAC, 2005). As these guidelines are more site-specific (i.e., developed for arctic ecosystem), all exceedances of DLCU criteria were carried forward into the risk assessment process.

Based on screening using human health-based criteria, the chemicals carried forward into the HHRA were aluminum, arsenic, barium, cadmium, iron, lead, lithium, manganese, nickel, uranium, zinc, ethylbenzene, toluene, xylenes, total polychlorinated biphenyls (PCBs), naphthalene and total petroleum hydrocarbon (TPH) fractions F2 and F3.

The chemicals carried forward into the ERA included aluminum, arsenic, barium, cadmium, copper, iron, lead, lithium, manganese, nickel, silver, uranium, zinc, total PCBs, naphthalene, toluene, ethylbenzene, xylenes and TPH fractions F1, F2, F3 and F4.

## **Exposure Scenarios**

The study area was defined as the area including and surrounding the sampling locations at CAM-D. The site consisted of a main station area, five dump sites and three outlying, smaller areas of debris (i.e., Simpson Lake, Murchison River, and airstrip and freshwater lake). For purposes of the ERA, CAM-D was divided into the following six distinct segments: main station and proximity, outfall area, barrel dumps, main dump, airstrip and Simpson Lake shoreline.

Infrastructure at the site included a module train, warehouse, garage, Inuit house, POL (petroleum, oil, lubricant) tanks and pumphouse, Quonset huts, storage pads and a radar tower. Presently, only the garage and POL pumphouse are still standing. All other buildings have collapsed, or have been removed or demolished. An airstrip, located southeast of the station, was also part of the initial infrastructure and remains in good condition.

Because of the northern location of CAM-D and the probable use of the site by Inuit for traditional purposes, the conventional land use categories (residential, parkland, commercial and industrial) were expanded to incorporate a new land use, Traditional Inuit Land Use. The parameters of this land use are discussed in detail in Gartner Lee and Cantox (1998), and were developed after consultation with residents of the Eastern Arctic, the Qikiqtaaluk Corporation and DIAND.

In the current HHERA, the Traditional Inuit Land Use category was adopted as set out by Gartner Lee and Cantox (1998), with minor modifications. The original Traditional Land Use designation consists of Inuit families residing on the land (i.e., the site), in tents for periods of up to 3 months. However, the period of residency was changed from 3 months to 3 weeks as this length of time was believed to better reflect the actual site-use scenario. It was assumed that during this period of residency the Inuit engaged in traditional hunting and gathering activities. It was also assumed that all time spent on site was in the non-snow covered months, which resulted in the most extensive exposure scenario for human receptors. Detailed exposure values are presented in Section 4.3, and in the Gartner Lee and Cantox report (1998).

Based on this land use, the following conceptual models were developed:

### ***Human Health***

The conceptual model that forms the basis for calculations of potential risk was as follows:

Traditional Land Use Scenario -

- A toddler aged six months to four years is exposed to surface soil contaminated with non-carcinogenic arsenic, barium, cadmium, lead, nickel, zinc, toluene, ethylbenzene, xylenes, TPH fractions F2 and F3, naphthalene and total PCBs by inadvertent ingestion, dermal contact and dust inhalation. Additionally, exposure to these contaminants occurs by ingestion of wild game. The same toddler is also exposed to surface water contaminated with non-carcinogenic aluminum, iron, lithium, manganese and uranium by ingestion and dermal contact.

- A person visits the site yearly (i.e., 3 wks/yr) from birth to 75 years of age and is exposed throughout to surface soil contaminated with carcinogenic arsenic, cadmium and total PCBs by inadvertent ingestion, dermal contact and dust inhalation. Exposure to these contaminants also occurs via ingestion of wild game.

### ***Ecological Health***

The risks associated with exposure to contaminated surface soil and water were the focus of the ecological risk assessment (ERA). The potential means for exposure to on-site contaminants included direct ingestion, inhalation and dermal contact with surface soils and water, as well as uptake via ingestion of terrestrial plant material, terrestrial invertebrates and mammals. The major exposure pathway considered was ingestion. Inhalation and dermal absorption were also possible exposure pathways but these were considered to be relatively minor as compared to ingestion and as such were not included as direct pathways in the ERA. Soil that adheres to fur or feathers is, for the most part, ingested by preening/licking activity and was included in the estimates of direct soil ingestion.

The receptors selected for evaluation in the ERA were the ermine, arctic hare, rock ptarmigan, collared lemming, snowy owl, arctic fox and barren ground caribou. These receptors are considered to be representative of indigenous wildlife at the CAM-D site. Other valued ecosystem components (VECs) were considered (discussed in Section 5.2.4) but the above receptors were chosen as it was believed that they are protective of all VECs that may potentially be on site.

### **Risk Characterization**

The above-noted exposure scenarios were evaluated to identify potential adverse effects for human or ecological receptors, with the following outcomes:

- Exposure point concentrations (EPCs) of human health-based CoPCs generated hazard quotients (HQ) less than 0.2 and incremental excess lifetime cancer risks (IELCR) less than  $1 \times 10^{-5}$ . These results indicated that contact with the site is not expected to produce adverse health effects in human receptors under the exposure scenarios considered in this risk assessment.
- EPCs of the identified chemicals of potential concern are not anticipated to produce adverse effects in ecological receptors under the exposure scenarios considered in the risk assessment.

Site-specific target levels (SSTLs) were calculated for each of the chemicals identified as a potential risk in the ERA. The SSTLs were calculated by setting the HQ to 1.0, and determining the corresponding surface soil EPC by means of a backward calculation.

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## 1.0 INTRODUCTION

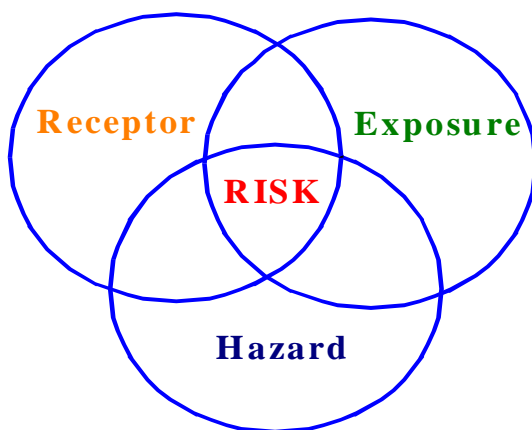
Jacques Whitford Limited (Jacques Whitford) was commissioned by Public Works and Government Services Canada (PWGSC) on behalf of the Department of Indian Affairs and Northern Development (DIAND) to complete a Human Health and Ecological Risk Assessment (HHERA) at the CAM-D Distant Early Warning (Dew) Line site. The primary objectives of the HHERA will be to evaluate the current risks associated with on-site contaminants and to develop site-specific target levels (SSTLs). The study addresses concerns regarding exposure to potentially hazardous metals and organic chemicals found in surface soil and water, and vegetation.

CAM-D is presently listed as a high priority site for mitigation and remediation of environmental impacts as part of the Federal Contaminated Sites Accelerated Action Plan (FCSAAP).

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### 1.1 Scope and Objectives

The purpose of the current study was to determine the concentrations of chemicals of potential concern (CoPCs) in surface soil and water below which no adverse human and/or ecological health effects would be expected. These SSTLs are to be used in preparation for reclamation work at the CAM-D DEW line site. To meet this objective, a widely accepted risk assessment framework was adopted wherein potential hazards, exposure pathways and receptors are evaluated to determine whether or not a risk is present, as illustrated in the diagram below:



The human health and ecological risk assessment framework comprises the following major components:

**Hazard Identification:** Identification of the environmental hazards that may pose a health risk (e.g., chemicals).

**Receptor Identification:** Identification of the receptors (i.e., human, ecological) that may be exposed to the above hazard(s).

**Toxicity Assessment:** Identification of published, scientifically reviewed toxicity values to which exposure levels can be compared.

**Exposure Assessment:** Qualitative or quantitative evaluation of the likelihood and/or degree to which the receptors will be exposed to the hazard(s).

**Risk Characterization:** Qualitative or quantitative assessment of the actual health risk each hazard poses to each receptor, based on the degree of exposure.

**Uncertainty Assessment:** Review the uncertainty associated with the risk estimation.

**SSTL Determination:** Establish the concentrations of CoPCs at the site below which no adverse health effects would be expected.

Derivation of the SSTLs presented in this report followed the general methodology outlined above. Specific tasks included:

- Review and compilation of existing data, and summarized past results;
- Qualitative risk screening to identify scenarios that are most likely to present a risk; and
- Quantitative risk analysis to develop SSTLs for those scenarios which are most likely to present a risk.

It is important to note that this report does not evaluate potential health concerns that may have existed in the past. Rather, it is designed to assess the potential risks associated with current and future exposures to contaminants at CAM-D based on present day conditions and assumed future post-reclamation conditions.

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## 1.2 Rationale for Site-Specific Risk Assessment

Generic, or Tier I, surface soil guidelines have been developed by the Canadian Council of Ministers of the Environment (CCME, 1999). These guidelines serve as conservative benchmarks for screening purposes. When soil concentrations are less than these guidelines, then the potential for human health and/or ecological effects is negligible. Alternatively, if soil concentrations exceed these guidelines it does not necessarily mean that unacceptable risks exist. The generic guidelines are purposely conservative and do not take into account regional or site-specific information (e.g., background soil conditions). Also, they may not be appropriate for every site or region of the country.

With this in mind, CCME published two documents (CCME, 1996a, b) thereby acknowledging that the generic guidelines are not definitive but may be modified in some instances if supported by sound reasoning and/or by the provision of site-specific information. In fact, to proceed with remediation without developing site-specific criteria could result in disruptive remedial action that brings little or no health benefit. Deriving SSTLs specifically for CAM-D (Simpson Lake) offers a more accurate means of assessing the potential effects of soil contaminants on the health of human and ecological receptors. Concentrations of chemicals in soil were initially evaluated using the Canadian Environmental Quality Guidelines published by the CCME in 2003.

The specific approach employed to develop the SSTLs is consistent with CCME and Health Canada protocols as referenced above, and with standard human health and ecological risk assessment methodologies.

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## 2.0 STUDY BACKGROUND

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### 2.1 Site Description

CAM-D (68°33' N, 83°19' W) is a former DEW Line site located in the middle of the Boothia Peninsula, NU approximately halfway between Shepherd Bay and Pelly Bay (Figure 1). The main station area is situated on a hill 370 meters above sea level and 4.5 km south of Simpson Lake from which the site derives its name (Figure 2). Simpson Lake was reserved by the Department of National Defence (DND) in 1956 and CAM-D was constructed in 1957 as an intermediate DEW Line site. The station was removed from service in 1963 and in 1965 responsibility for the site was assumed by DIAND. The terrain at CAM-D is characterized by rolling grassy hills cut by rock outcrops, and several lakes and rivers.

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### 2.2 Study Areas

The study area was defined as the area including and surrounding the sampling locations at CAM-D. The site consisted of a main station area, five dump sites and three outlying, smaller areas of debris (i.e., Simpson Lake, Murchison River, and airstrip and freshwater lake).

Infrastructure at the site included a module train, warehouse, garage, Inuit house, POL (petroleum, oil, lubricant) tanks and pumphouse, Quonset huts, storage pads and a radar tower. Presently, only the garage, one POL tank and POL pumphouse are still standing. All other buildings have collapsed, or have been removed or demolished. An airstrip, located southeast of the station, was also part of the initial infrastructure and remains in good condition.

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### 2.3 Previous Reports

During the summer of 1985, the Environmental Protection Branch of Environment Canada and the DND assisted DIAND in the removal of hazardous materials from CAM-D (Holtz et al., 1986). The objective of the cleanup was to remove surface contaminants such as PCBs and POLs, and to identify areas of buried materials which could pose future environmental risks. Various pieces of PCB-containing equipment were removed from the site including capacitors from the de-icing motors found in some electrical cabinets. The soil directly under these motors was tested and PCB contamination was identified at a range of concentrations. No mention is made of any soil remediation.

In 1994, the Environmental Sciences Group of Royal Roads Military College (RMC) completed a detailed surface soil, water and vegetation sampling program at CAM-D (RMC, 1994). Soil contamination exceeding Tier I and/or Tier II DLCU criteria was identified at various locations throughout the CAM-D site. Contaminants exceeding the DLCU criteria included PCBs, arsenic, cadmium, copper and zinc. Soil samples were also analyzed for polycyclic aromatic hydrocarbons (PAHs); although detectable, no PAHs exceeded CCME criteria. PCB amended paint was not identified at CAM-D, however, insulation samples collected from the garage and warehouse contained chrysotile asbestos. The 1994 investigation did not include assessment of hydrocarbon impacts, a potentially significant source of contamination at the site.



**Figure 1: Location of CAM-D Simpson Lake DEW line site**

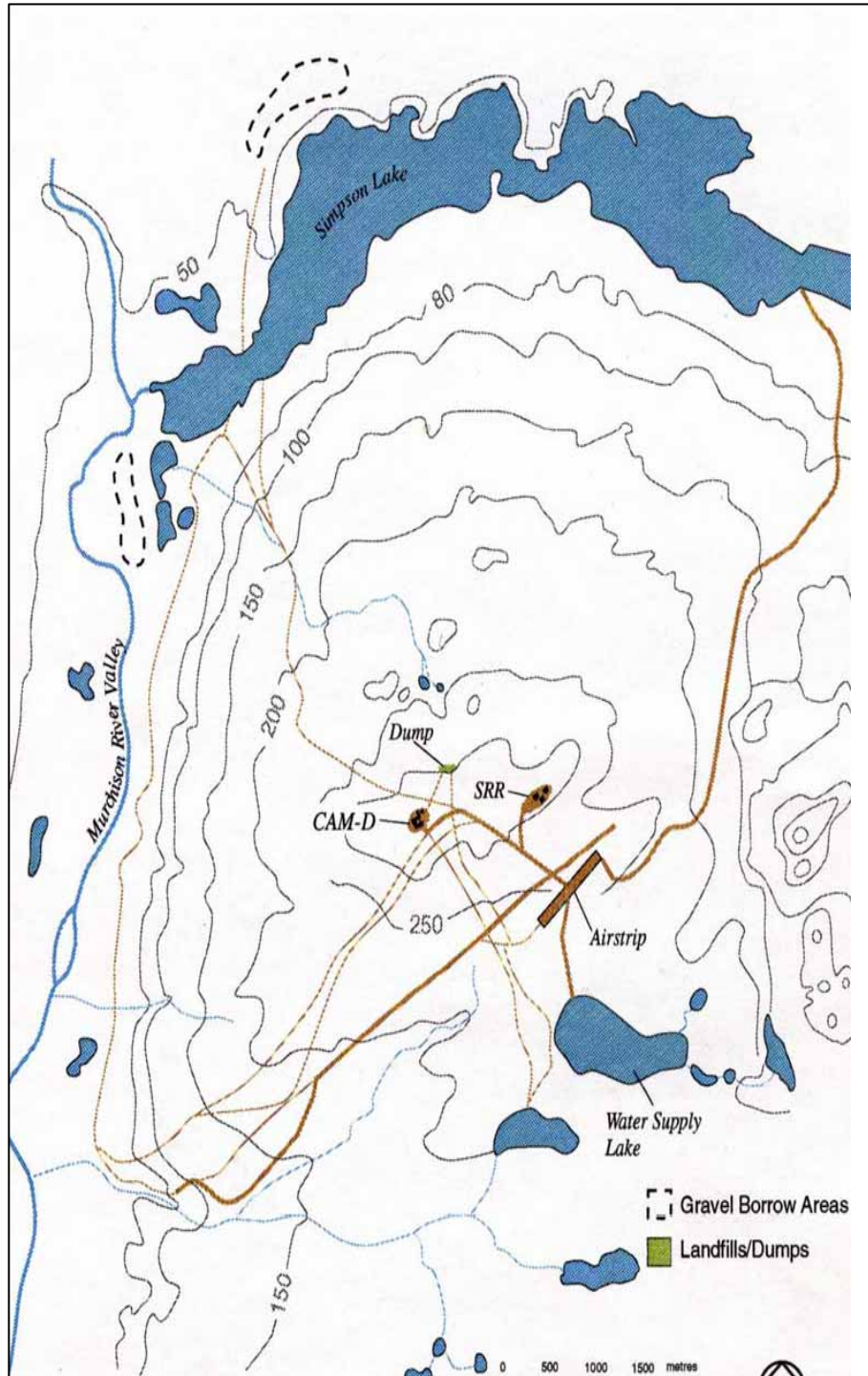


Figure 2: CAM-D site layout



In September of 2003, SENES Consultants Ltd. was retained by DIAND to conduct a screening level risk assessment (SLRA) to evaluate the potential impacts on human and ecological health from exposure to contaminants at CAM-D. SENES identified zinc as a chemical of potential concern (CoPC) with respect to human health, and arsenic, copper, nickel, zinc and total PCBs as CoPCs for ecological health. It was considered that all CoPCs identified at CAM-D posed a low risk to human and/or ecological health.

In the current study, a quantitative human and ecological risk assessment was performed for the CAM-D Intermediate Dew Line site. It is supported by new contaminant data, acquired via a Phase III ESA conducted by Earth Tech (2005), which included analyses for hydrocarbons, PAHs, PCBs and inorganic elements in surface soils, vegetation and water. In addition, a number of background soil samples were collected. The ERA considers a broad range of ecological receptors and incorporates the new data while retaining the previously collected data for CAM-D and surrounding areas. The HHRA also evaluates both Phase II and the newly generated Phase III data supplied by Earth Tech (2005).

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## **2.4 Inuit Traditional Land Use**

In 1998, Gartner Lee Limited and Cantox Inc. conducted a risk assessment for the FOX-C DEW Line site and contended that due to its northern location and probable use by Inuit for traditional purposes the conventional land use categories (residential, parkland, commercial and industrial) should be expanded to incorporate an additional land use, which they termed "Traditional Land Use". The parameters of this land use were developed after consultation with residents of the Eastern Arctic, the Qikiqtaaluk Corporation and DIAND.

In the current HHERA, the Traditional Inuit Land Use category was adopted as set out by Gartner Lee and Cantox (1998), with minor modifications. The original Traditional Land Use designation consists of Inuit families residing on the land (i.e., the site), in tents for periods of up to 3 months. However, based on extensive professional experience in dealing with remote northern locations, the period of residency was changed from 3 months to 3 weeks as this length of time was believed to better reflect actual Inuit behavior. It was assumed that during this period of residency the Inuit engaged in traditional hunting, fishing and gathering activities. It was also assumed that all time spent on site was in the non-snow covered months, which resulted in the most extensive exposure scenario for human receptors. Detailed exposure values are discussed in Section 4.3, and in the Gartner Lee and Cantox report (1998).

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## **3.0 DATA COMPILATION**

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### **3.1 Sources**

The data used in the present HHERA was primarily supplied by Earth Tech Environmental Inc. who, on behalf of PWGSC and DIAND, conducted a field investigation and sampling program at CAM-D in August of 2005. Earth Tech collected soil, surface water and vegetation samples from CAM-D and surrounding areas. A detailed list of samples and sampling locations is presented in a separate report (Earth Tech, 2005). In addition to the data collected in 2005, analyses for inorganic parameters and

PCBs in soil, water and vegetation were conducted and presented by the Environmental Sciences Group at RMC in a 1994 report. A detailed list of samples and sampling locations can be found in the 1994 report.

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## **3.2 Selection of Data**

The soil data obtained via the Phase II and Phase III sampling programs were screened for use in this risk assessment. For the purposes of human health and ecological risk assessment, only surface soil samples (i.e., ground surface to 0.3 meters below ground surface) are relevant for potential exposures. Thus, Phase II and Phase III data were screened on the basis of depth and any sample that did not intersect the surface and/or extended to a depth of greater than 0.3 meters below ground surface (mbgs) was excluded. This was carried out to ensure that the data used in the HHERA were representative of surface soils and not heavily influenced by subsurface soil characteristics.

All surface water samples were considered to be valid inputs for the risk assessment.

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### **3.2.1 Phase II Data**

The RMC Environmental Sciences Group presented phase II data relating to soil and water sampling at CAM-D in a 1994 report (RRMC, 1994).

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### **3.2.2 Phase III Data**

Phase III data was supplied and described by Earth Tech (2005). Soil and water samples were collected and analyzed in August of 2005.

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## **3.3 Description and Statistical Analyses of Data**

The maximum concentrations of all chemicals were screened against generic guidelines for either human or ecological health. If a chemical exceeded a guideline, then phase II and/or phase III data were combined and statistical analyses were performed.

Environmental data are often log-normally distributed, and consequently skewed. In the present study, the distribution of data points for the majority of contaminants closely resembled a log normal distribution. However, most individual data sets failed the Shapiro Wilk's Test for log normal distribution confirmation. The distribution of the data sets is likely the result of non-random sample selection where "hot spots", or areas where substantial contamination is known to exist, were selectively sampled. This method of sampling causes many outliers, samples with very high concentrations of contaminants as compared to other samples collected on-site. Accordingly, the data is more representative of a log normal distribution than a normal distribution, and the data were log transformed to calculate the appropriate exposure point concentrations (EPCs) – 95% upper confidence limit (UCL) of the geometric mean.



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## **4.0 HUMAN HEALTH RISK ASSESSMENT**

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### **4.1 Risk Assessment Framework**

To guide the human health and ecological risk assessments a common framework was developed (Figure 3). Each step in this flowchart is briefly described below:

#### **Box 1 Compare maximum contaminant concentrations to guidelines**

Maximum concentrations identified on site were compared to generic CCME and Ontario Ministry of the Environment (OME) soil quality guidelines for residential/parkland land use, and to DLCU criteria.

#### **Box 2 Determine if maximum concentration is greater than guideline value**

If the maximum concentration of a particular contaminant was less than the appropriate generic guideline (e.g., CCME, OME) then the chemical was not carried forward into the quantitative risk assessment.

#### **Box 3 Calculate the EPC**

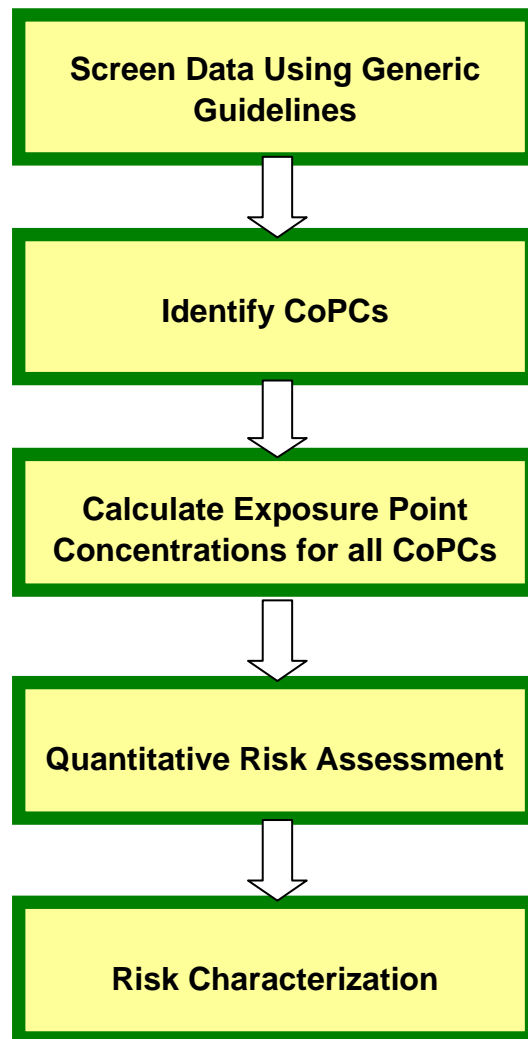
The 95% upper confidence limit of the mean was adopted as the EPC for all CoPCs in both the human health and ecological risk assessments.

#### **Box 4 Conduct quantitative risk assessment**

CoPCs that exceeded guideline values were carried forward into the risk assessment. The risk assessment process was conducted separately for human health and ecological health, and independently for each CoPC. In this way, the chemicals subjected to human health risk assessment were not necessarily included in the ecological risk assessment.

#### **Box 5 Do hazard quotients exceed target hazard quotient values?**

When a calculated hazard quotient (HQ) exceeds the target HQ value (0.2 for HHRA, 1.0 for ERA), an inherent risk may exist on site. Consequently, site-specific target levels (SSTLs) would be derived and remedial action could be taken to attain these concentrations on site.



**Figure 3: Risk Assessment Framework**

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## 4.2 Chemical Screening

The maximum concentration of all chemicals (phase II and III) was screened against generic CCME and OME guidelines for either human or ecological health. A variety of CoPCs were identified at CAM-D including metals, toluene, ethylbenzene, xylenes, total petroleum hydrocarbon (TPH) fractions, naphthalene and total polychlorinated biphenyls (PCBs).

Generic CCME and OME soil quality guidelines may be based on either ecological or human health protection, and are generally used in the initial protective screening of site data. For the human health risk assessment, the identified CoPCs are then screened specifically against human health based generic guidelines. Because the HHRA for CAM-D includes an exposure scenario for Inuit camping on site, residential/parkland values have been adopted for the screening. In order of preference, guidelines from the following organizations were used for screening: CCME (2001, 2003), OME (2004) or the US EPA (2005). The maximum concentration of CoPCs was also screened using the INAC Dew Line Cleanup (DLCU) criteria (INAC, 2005). These criteria were developed by Defence Construction Canada (DCC) for the DND for use at the main Dew Line sites, and are considered to be protective of the arctic ecosystem. In instances where DLCU criteria were less than CCME guideline values, DLCU criteria were used in place of CCME guidelines.

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### 4.2.1 Identification of Chemicals of Potential Concern

The results of human health soil screening for CAM-D (Simpson Lake) are presented in Table 1.

#### Soils

As indicated in Table 1, the maximum soil concentration of total PCBs exceeded Tier I DLCU criteria while cadmium exceeded Tier II DLCU criteria. As such, both contaminants were carried forward into the human health risk assessment. It can be noted that total PCBs also exceeded CCME human health based criterion whereas cadmium was less than these guidelines.

The maximum soil concentrations of arsenic, barium, lead, nickel, zinc, ethylbenzene, toluene, xylenes and naphthalene were greater than the CCME human health based guidelines. Even though many of these contaminants were less than the corresponding OME human health based criterion they were all carried forward as CoPCs into the HHRA.

The maximum soil concentration of total petroleum hydrocarbon (TPH) fractions F2 and F3 exceeded the Canada Wide Standard (CWS) criterion, and these fractions were carried forward into the HHRA. TPH fractions F1 and F4 were below CWS criteria and eliminated from the assessment.

Maximum soil concentrations of all other metals and PAHs were less than their respective human health based criteria, and were therefore not carried forward.

The soil CoPCs from CAM-D carried forward into the HHRA included:

- *arsenic*
- *barium*

- *cadmium*
- *lead*
- *nickel*
- *zinc*
- *ethylbenzene*
- *toluene*
- *xylenes*
- *TPH fraction F2*
- *TPH fraction F3*
- *Naphthalene*
- *Total PCBs*

All substances identified as CoPCs based on human health soil screening were quantitatively assessed in surface water, providing they were measured above the level of detection in this media. This ensures that derived total hazard quotients (i.e., risk estimates) take into consideration all potential exposure pathways.

### **Surface Water**

In August of 2005, surface water samples were collected from Simpson Lake, a nearby freshwater lake and from a variety of locations around the site, including the sewage outfall area, the pallet line area, the POL tank area, the vehicle dump and the main dump. The results of human health surface water screening for CAM-D are presented in Table 2. The maximum concentration of uranium in surface water from the sewage outfall area exceeded CCME drinking water guidelines. Similarly, the maximum concentrations of aluminum, iron, lithium and manganese were greater than US EPA Region III freshwater screening benchmarks. As such, *aluminum, iron, lithium, manganese, and uranium* were carried forward as CoPCs into the HHRA.

A number of parameters, including bismuth, silicon, sulfur and titanium, do not have published health-based drinking water criteria. Therefore, there is no benchmark against which to evaluate the concentrations of these contaminants found in surface water. Because neither the CCME, OME or US EPA has derived drinking water criteria for these parameters, they are assumed to have low inherent toxicity and not to pose a risk to human health.

Maximum surface water concentrations of all other metals, BTEX, TPH fractions, PAHs and PCBs were less than their respective human health based criteria.

### **Wild Game**

Additional exposure to on-site CoPCs could occur through ingestion of contaminated wild game. It is necessary to consider this source of CoPCs as CAM-D may be used by Inuit visitors for recreational purposes (e.g., camping) or could serve as a basecamp for hunting and gathering activities.

While concentrations of CoPCs can be, and have been, measured in surface water, soil and plants, it is necessary to estimate the concentrations of CoPCs in wild game. The procedure for estimating plant or animal uptake of CoPCs from soil is explained in detail in Section 5.4.2.

**Table 1. Human Health Soil Screening for CAM-D**

Soil CoPCs	Phase II <sup>1</sup> Maximum Concentration	Phase III Maximum Concentration	Total No. of Samples	CCME <sup>2</sup>	OME <sup>3</sup>	CCME CWS <sup>4</sup>	DLCU <sup>5</sup> Criteria Tier I	DLCU <sup>5</sup> Criteria Tier II	No. Of Samples Exceeding Guidelines
				Human Health	Human Health	Human Health			
Inorganics									
Arsenic	60	2.8	93	12	25	-	NC	30	1
Barium	na	1720	93	500	1000	-	NC	NC	1
Cadmium	9.3	5.13	93	14	12	-	NC	5	2
Lead	183	92.3	93	140	200	-	200	500	2
Nickel	53	18	93	50	200	-	NC	100	1
Zinc	1370	1570	93	200	800	-	NC	500	4
Organics									
Ethylbenzene	na	4.29	52	1.2	500	-	-	-	2
Toluene	na	4.77	52	0.8	150	-	-	-	1
Xylenes (total)	na	39.4	52	5	210	-	-	-	3
F2	na	16700	52	NC	NC	8000	-	-	1
F3	na	28300	52	NC	NC	18000	-	-	2
Naphthalene	na	37	42	0.6	40	-	-	-	5
PCBs (total)	1.5	0.6	80	1.3	5	-	1	5	1

**Notes:**

All units are in mg/kg

Applicable guideline

na - not assessed

NC - No Criteria

1 - RRM, 1994

2 - Canadian Council of Ministers of the Environment (CCME) Canadian Environmental Quality Guidelines: Soil Quality Guidelines (1991, Updated 1997, 1999 & 2003)

3 - Ontario Ministry of the Environment (OME) Soil, Groundwater and Sediment Standards for use under Part XV.1 of the Environmental Protection Act – Full Depth Site Condition Standards in a Non-Potable Ground Water Condition. (March, 2004).

4 - Canadian Council of Ministers of the Environment "Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil" (May 2001) for fine-grained surface soils – Residential land use (soil ingestion)

5 - INAC, 2005. Abandoned Military Site Remediation Protocol. Appendix B, DEW Line Cleanup (DLCU) Soil Criteria.

**Table 2. Human Health Surface Water Screening**

Surface Water CoPCs	Phase III maximum concentration	Total no. of samples	CCME <sup>1</sup> Human Health		OME <sup>2</sup> Drinking Water	EPA <sup>3</sup> Region III Freshwater Screening	No. of samples exceeding guidelines
			MAC	IMAC			
Inorganics							
Aluminum	9.3	7	NC	NC	NC	0.087	6
Iron	8.2	7	NC	NC	NC	0.3	3
Lithium	0.016	7	NC	NC	NC	0.014	1
Manganese	0.14	7	NC	NC	NC	0.12	1
Uranium	0.02	7	NC	0.02	NC	NC	1

**Note:**

All units are in mg/L

Applicable guideline

NC - No Criteria

1 - Canadian Council of Ministers of the Environment (CCME) Canadian Environmental Quality Guidelines: Community Water Supplies (1999, Updated 2003)

2 - Ontario Ministry of the Environment (OME) Soil, Groundwater and Sediment Standards for use under Part XV.1 of the Environmental Protection Act - Full Depth Site Condition Standards in a Potable Ground Water Condition. (March, 2004)

3 - US Environmental Protection Agency. Region III Freshwater Screening Benchmarks (2005)



## Exposure Point Concentrations

Exposure point concentrations (EPCs) were calculated for each CoPC in surface soil, water and vegetation (Table 3). The EPC is intended to be a conservative (i.e., guarded, but not necessarily worst-case) estimate of the average on-site concentration to which wildlife may be exposed. For purposes of this HHRA, EPCs were estimated as the 95% upper confidence limit of the mean concentration.

A summary of the calculated concentrations of CoPCs in wild game meat is also presented in Table 3. Detailed calculations showing the derivation of contaminant concentrations in wild game meat are summarized in Appendix C.

**Table 3. Summary of CoPCs and Exposure Point Concentrations Used in the CAM-D HHRA**

CoPCs	Surface Soil EPC (mg/kg)	Surface Water EPC (mg/L)	Vegetation EPC (mg/kg)	Wild Game EPC (mg/kg)
<b>Inorganics</b>				
Aluminum	na	4.7	2980	na
Arsenic	3.0	8.53E-04	1.70E-01	3.00E-03
Barium	233	6.96E-02	49	1.00E-01
Cadmium	8.20E-01	1.62E-04	0.53	1.40E-01
Iron	na	4.2	1500	na
Lead	20.3	3.69E-03	7.3	9.40E-01
Lithium	na	9.80E-03	3	7.70E-01
Manganese	na	1.02E-01	338	6.56E-15
Nickel	8.4	5.10E-03	14	6.50E-01
Uranium	na	1.25E-02	na	9.63E-05
Zinc	129	6.87E-02	296	37.4
<b>Organics</b>				
Ethylbenzene	3.20E-01	ND	na	1.32E-06
Toluene	3.00E-01	ND	na	4.07E-07
Xylenes	2.8	ND	na	3.81E-06
TPH F2	1808	ND	na	27.1
TPH F3	3560	ND	na	32.5
Naphthalene	4.1	ND	na	5.70E-04
Total PCBs	3.49E-02	ND	na	9.60E-03

**Notes:**

na - not assessed

ND - not detected

EPC = 95% UCL of the geometric mean

### 4.2.2 Receptor Identification

For any given site, existing and intended land uses are important factors in evaluating potential exposures and estimating risk. This risk assessment assumes that the future land uses at CAM-D are as follows:

- Intermittent use of the general area for recreational and/or hunting purposes.

Therefore, the potential human “receptors”, or those that may be most affected by the potential on-site hazards, are individuals camping and/or hunting on the land. For this risk assessment, the human

receptor is characterized as an adult or child with no extreme sensitivities. Carcinogenic and non-carcinogenic chemicals are evaluated differently as shown below:

	RESIDENTIAL EXPOSURE
<b>NON-CARCINOGENIC CHEMICALS</b>	Most sensitive receptor modeled as a toddler aged six months to four years old.
<b>CARCINOGENIC CHEMICALS</b>	It is assumed that the receptor visits the site yearly from birth to 75 years of age. Exposures are averaged over five age groups: (0 to 6 months) + (6 months to 4 yrs) + (5 to 11 yrs) + (12 to 19 yrs) + (20 to 75yrs).

The above assumptions pertaining to human receptors represent the most protective approaches for the intended land uses. Important receptor characteristics (e.g., body weight, soil ingestion rate) that are considered in the analysis are presented in Section 4.3.

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#### 4.2.3 Exposure Pathway Assessment

In the exposure assessment, the likelihood that human receptors would come into contact with the potential hazards was evaluated. The likelihood of exposure was determined through consideration of the chemical properties of individual hazards that control mobility, and the various pathways through which the hazard could move to contact the receptor or through which the receptor could move to contact the hazard. The exposure analysis also considers the possible mechanisms whereby a hazard could be introduced to a human receptor (e.g., ingestion, dermal contact, inhalation).

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#### 4.2.4 Potential Transport Pathways

The principal pathways by which environmental hazards can contact human receptors include:

- direct contact (e.g., soil, dust, liquid phase product, water);
- transport of liquid phase contaminants;
- transport in groundwater;
- transport in surface water;
- airborne transport (i.e., dust); and
- transport as a vapour.

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#### 4.2.5 Potential Exposure Mechanisms

The mechanisms by which receptors typically become exposed to hazards include:

- inhalation;
- ingestion;
- dermal contact; and
- uptake by plants and/or animals, followed by ingestion.

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#### 4.2.6 Human Receptor Exposure Scenarios

The exposure scenarios that have been considered for human receptors include:

- ingestion/dermal contact with soil;
- inhalation/ingestion/dermal contact with dust;
- ingestion of vegetation and/or garden produce grown in contaminated soil or irrigated with contaminated water;
- ingestion of wild game acquired by hunting in the CAM-D area;
- ingestion/dermal contact with surface water;
- ingestion/dermal contact with groundwater; and
- inhalation of vapours.

Jacques Whitford qualitatively evaluated the likelihood that human receptors could be exposed to the identified hazards through the various exposure scenarios outlined above. The likelihood of exposure was considered and evaluated in terms of the definitions presented in Table 4.

The relevant exposure pathways are summarized in Table 5, which includes a qualitative evaluation for each pathway and a justification for the likelihood of exposure assigned. The likelihood of exposure was determined by considering the duration and frequency of contact with each potential hazard, and the relative concentrations to which receptors are likely to be exposed. Those hazard-exposure-receptor combinations considered most likely to contribute to a human health risk are carried forward for further quantitative analysis.

**Table 4: Exposure Definitions**

Likelihood of Exposure	Definition
Very Unlikely	Level of exposure that could result in adverse effects is not expected.
Unlikely	Level of exposure that could result in adverse effects is not likely.
Possible	Level of exposure that could result in adverse effects might be expected.
Likely	Level of exposure that could result in adverse effects is expected.

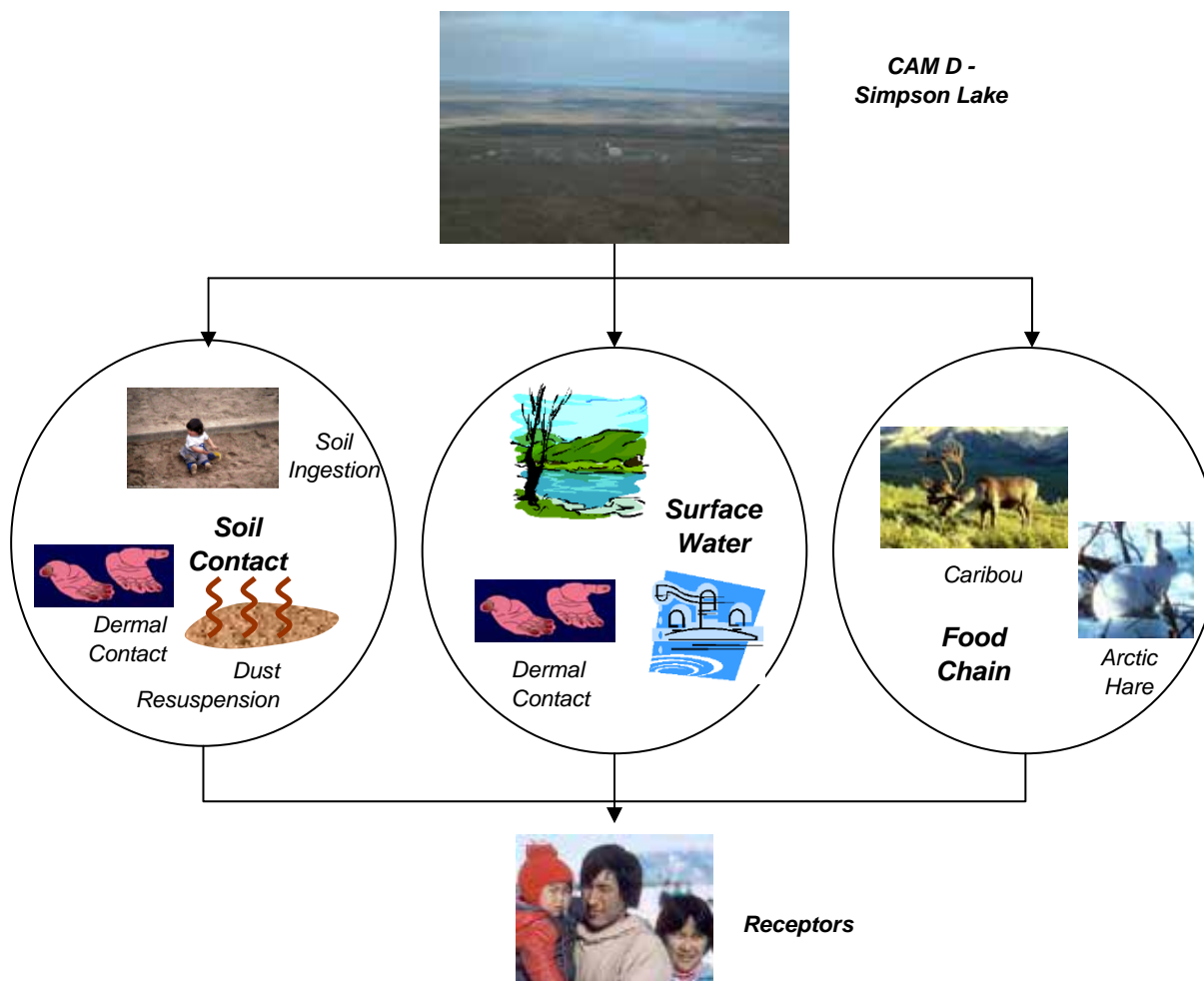


**Table 5: Potential Exposure Scenarios – Inuit Visitor**

Exposure Pathway Description	Likelihood of Exposure	Carried Forward for Quantitative Analysis	Justification
Ingestion of soil	Likely	Yes	Surface soil at CAM-D is impacted by a number of contaminants exceeding human health guidelines, including arsenic, barium, cadmium, lead, nickel, zinc, ethylbenzene, toluene, xylenes, TPH fractions F2 and F3, naphthalene and total PCBs. See section 4.2.1.
Dermal contact with soil			
Inhalation of soil particles			
Inhalation of soil vapours	Unlikely	No	Petroleum hydrocarbons and sparsely chlorinated PCBs can volatilize from surface soil. However, it is unlikely that human receptors would be exposed to these vapours as the dilution potential for soil vapours released to outdoor air is high.
Ingestion of sediment	Unlikely	No	It is unlikely that receptors would come into direct contact with sediment considering the cold water temperatures.
Dermal contact with sediment			
Inhalation of sediment particles/vapours			
Ingestion of surface water	Unlikely	Yes	A number of inorganic CoPCs were identified in surface water at CAM-D and nearby lakes. Thus, surface water was deemed a potential exposure pathway. However, this pathway is unlikely as concentrations of CoPCs in surface water are low.
Dermal contact with surface water			
Inhalation of surface water vapours	Unlikely	No	No volatile CoPCs were identified in surface water, therefore surface water vapour inhalation was not considered a valid pathway.
Ingestion of ground water	Very Unlikely	No	It is not anticipated that groundwater will be used as a source for either drinking or showering and therefore was not considered a valid pathway.
Dermal contact with ground water			
Inhalation of ground water particles/vapours			
Ingestion of vegetation	Very Unlikely	No	Because of the climate, substantial harvest vegetation was not expected.
Ingestion of wild game	Likely	Yes	It is expected that on-site human receptors would hunt and consume wild game on the site.

Based on the qualitative risk screening presented above, the following conceptual model (Figure 4) was developed and formed the basis for calculations of potential risk:

- A toddler aged six months to four years is exposed to non-carcinogenic CoPCs in surface soil by ingestion, inhalation and dermal contact. The toddler is also exposed via ingestion of wild game, and ingestion and dermal contact with surface water.
- A person visits the site yearly from birth to 75 years of age and is exposed throughout to surface soil contaminated with carcinogenic CoPCs via inadvertent ingestion, dermal contact and dust inhalation. Exposure to CoPCs also occurs by ingestion of wild game, and ingestion and dermal contact with surface water.



**Figure 4: Conceptual Site Model**

### 4.3 Receptor Characteristics

It is important that the most protective assumptions are made when assessing human exposure to CoPCs at CAM-D. To evaluate the risks associated with exposure to non-carcinogenic CoPCs, a toddler aged 6 months to 4 years was selected as the most sensitive receptor. Additionally, a composite receptor was included to assess the carcinogenic CoPCs. A traditional land use scenario was adopted wherein it was assumed that receptors will be present at CAM-D for 21 days per year, 24 hours per day. It was also conservatively assumed that human receptors are exposed to the most highly contaminated soil found on site. This assumption is conservative as the most contaminated spots are localized to small areas of the site and receptors are not expected to spend all their time on these "hot spots". Nevertheless, to be protective of human health, exposure to maximum concentrations was assumed. Receptor characteristics for the toddler and composite receptor are presented below (Table 6).

**Table 6: Summary of Receptor Characteristics**

Characteristics		Toddler (6 mos – 4 yrs)	Composite (75 yrs)	Reference
<b>Averaging Times and Constant Values</b>				
AT <sub>c</sub>	Averaging Time – Carcinogen (days)	--	27375	Health Canada (2004)
AT <sub>nc</sub>	Averaging Time – Non-carcinogen (days)	1642.5	--	Health Canada (2004)
ED	Exposure Duration (yr)	4.5	75	Health Canada (2004)
EF	Exposure Frequency – (days/yr)	21	21	Conservative site-specific assumption
ET	Exposure Time (hr/d)	24	24	Based on full-time exposure to the site
BW	Body weight (kg)	16.5	63.27	Health Canada (2004)
TSP	Total Suspended Particulate (kg/m <sup>3</sup> )	2.50E-07	2.50E-07	Health Canada (2004)
<b>Ingestion of Surface Soil</b>				
IR <sub>soil</sub>	Ingestion Rate of Surface Soil (mg/day)	80	23.87	Health Canada (2004)
<b>Dermal Contact with Surface Soil</b>				
SA <sub>body</sub>	Exposed surface area - body (cm <sup>2</sup> )	3009	8650	Richardson (1997)
SA <sub>hand</sub>	Exposed surface area - hand (cm <sup>2</sup> )	430	833	Richardson (1997)
SAF <sub>body</sub>	Soil adherence factor – body (mg/cm <sup>2</sup> -d)	0.01	0.01	Health Canada (2004)
SAF <sub>hand</sub>	Soil adherence factor – hand (mg/cm <sup>2</sup> -d)	0.10	0.10	Health Canada (2004)
<b>Inhalation of Soil Particles</b>				
IR <sub>air</sub>	Inhalation rate (m <sup>3</sup> /hr)	0.39	0.64	Richardson (1997)
FR <sub>soil</sub>	Fraction of dust from soil – outdoor (unitless)	1	1	assumed
<b>Ingestion of Surface Water</b>				
IR <sub>water</sub>	Ingestion of surface water (L/d)	0.6	1.34	Richardson (1997)
<b>Dermal Contact with Surface Water</b>				
SA <sub>water</sub>	Exposed surface area dermal water (cm <sup>2</sup> )	430	833	Richardson (1997)
t <sub>event</sub>	Event Duration (hr/event)	0.5	0.5	assumed
<b>Ingestion of Wild Game</b>				
IR <sub>game</sub>	Ingestion rate of wild game (Kg/d)	0.09	0.24	Health Canada (2004)
F <sub>site</sub>	Fraction of wild game that is from site (unitless)	1.00	1.00	Conservative site-specific assumption

For non-threshold chemicals (i.e., carcinogens), where any level of exposure may produce adverse health effects, exposures are not calculated for specific age groups but are averaged over a lifetime. In accordance with the rational maximum exposure approach, it is assumed that all visitors to CAM-D grow up in Nunavut from birth to 75 years of age. For risk characterization, exposures are averaged over five age groups: (0 to 0.5 years) + (0.5 to 4 years) + (5 to 11 years) + (12 to 19 years) + (20 to 75 years). Receptor characteristics for each age group are presented in Table 7.

**Table 7: Summary of Receptor Characteristics for Individual Age Groups**

Characteristic	Receptor Values						Reference
	Infant	Toddler	Child	Teen	Adult	Composite	
Age	0 – 6 mo.	7 mo. – 4 yr	5 – 11 yr	12 – 19 yr	20 - 75 yr	over 75 year lifetime	Health Canada (2004)
AT (years)	0.5	4.5	7	8	56	75	Health Canada (2004)
BW (kg)	8.2	16.5	32.9	59.7	70.7	62.33	Health Canada (2004)
IR <sub>soil</sub> (mg/h)	0.83	3.33	0.83	0.83	0.83	0.98	Health Canada (2004)
IR <sub>air</sub> (m <sup>3</sup> /hr)	0.0875	0.4	0.6	0.7	0.7	0.6	Health Canada (2004)
IR <sub>water</sub> (L/d)	0.3	0.6	0.8	1	1.5	1.319	Health Canada (2004)
SA <sub>hand</sub> (cm <sup>2</sup> )	320	430	590	800	890	821	Richardson, 1997
SA <sub>body</sub> (cm <sup>2</sup> )	1780	3010	5140	8000	9110	8206	Richardson, 1997
IR <sub>game</sub> (mg/d)	0	85	125	175	270	233.43	Health Canada (2004)

Note: all acronyms defined in Table 8.

## 4.4 Toxicity Assessment

The potential hazards associated with exposure to non-carcinogenic (threshold) substances are assessed differently than those for carcinogenic (non-threshold) substances. For threshold substances, it is assumed that there is a dose (or concentration) that does not produce any adverse effects. The tolerable daily intake (TDI) is an estimate of a chemical intake that is unlikely to cause increased incidence of deleterious health effects during a lifetime of exposure. TDIs are specifically developed to be protective of chronic chemical exposures. For purposes of deriving HQs and site-specific threshold levels, a chronic daily intake (CDI) is calculated for exposed individuals and weighed against the TDI. If  $CDI/TDI > 0.2$ , then adverse health effects are considered possible and further assessment would be required.

For contaminants whose critical effect is assumed to have no threshold (i.e., carcinogens), any level of exposure can potentially cause adverse health effects (CCME, 1996). A linear dose-response relationship has been developed that converts estimated daily intakes averaged over a lifetime of exposure directly to the incremental risk of an individual developing cancer. For purposes of deriving site-specific soil quality guidelines, Health Canada considers that a single additional case of cancer in an exposed population of 100,000 merits action (Health Canada, 2003). As such, a target risk (TR) of one in one hundred thousand (or  $10^{-5}$ ) is used in this risk assessment for carcinogenic effects.

## 4.5 Selection of Toxicity Reference Values

An important part of the risk assessment process is the identification of appropriate toxicity reference values (TRVs), such as TDIs and cancer slope factors. This is typically accomplished by a comprehensive review of published risk assessment guidance documents and/or by internet searches (e.g., Integrated Risk Information System - IRIS).

Toxicity values have been established by a number of agencies, including Health Canada and the United States Environmental Protection Agency (US EPA). However, preference has been given to Health Canada TRVs as per federal guidance (Health Canada, 2003). In the event that a Health Canada TRV is not available, IRIS, the Risk Assessment Information System (RAIS), US EPA and/or the US EPA's National Center for Environmental Assessment have been consulted. Toxicity reference

values selected for inclusion in this risk assessment are presented in Tables 8 and 9, and rationales for each of the toxicity values are supplied in Appendix B.

**Table 8: Selected Cancer Toxicity Values**

CoPC	Route of Exposure	Exposure Limit (mg/kg)/d	Toxicological Basis	Source Agency
<b>Inorganics</b>				
Arsenic	Ingestion	2.8	Not Specified	Health Canada (2004)
	Inhalation	28	Not Specified	Health Canada (2004)
Cadmium	Ingestion	na	na	na
	Inhalation	42.9	Not Specified	Health Canada (2004)
<b>Organics</b>				
PCBs	Ingestion	2.0	Liver hepatocellular adenomas, carcinomas and cholangiomas	IRIS (2004)
	Inhalation	0.4	Not Specified	IRIS (2004)

**Table 9: Selected Non-Carcinogenic Toxicity Values**

CoPC	Route of Exposure	Exposure Limit (mg/kg)/d	Toxicological Basis	Source Agency
<b>Inorganics</b>				
Aluminum	Ingestion	1	Not Specified	RAIS (2005)
	Inhalation	1.4E-03	Not Specified	RAIS (2005)
Arsenic	Ingestion	3.0E-04	Hyperpigmentation, keratosis and vascular complications	IRIS (2005)
	Inhalation	na	na	na
Barium	Ingestion	1.6E-02	Not Specified	Health Canada (2004)
	Inhalation	1.4E-04	Not Specified	RAIS (2005)
Cadmium	Ingestion	8.00E-04	Not Specified	Health Canada (2004)
	Inhalation	5.7E-05	Not Specified	NCEA (2005)
Iron	Ingestion	0.3	Not Specified	RAIS (2005)
	Inhalation	na	na	na
Lead	Ingestion	3.6E-03	Not Specified	Health Canada (2004)
	Inhalation	na	na	na
Lithium	Ingestion	0.02	Not Specified	NCEA (2005)
	Inhalation	na	na	na
Manganese	Ingestion	0.14	Neurological Effects	IRIS (2005)
	Inhalation (mg/m <sup>3</sup> )	5.0E-05	Impaired neurobehavioral function	IRIS (2005)
Nickel	Ingestion	2.0E-02	Decreased body and organ weights	IRIS (2005)
	Inhalation (mg/m <sup>3</sup> )	1.8E-05	Not Specified	Health Canada (1996)
Uranium	Ingestion	6.0E-04	Not Specified	RAIS (2005)
	Inhalation	na	na	na
Zinc	Ingestion	0.3	Decline in human erythrocyte superoxide dismutase activity	IRIS (2005)
	Inhalation	na	na	na

**Table 9: Selected Non-Carcinogenic Toxicity Values**

CoPC		Route of Exposure	Exposure Limit (mg/kg)/d	Toxicological Basis	Source Agency
Organics					
Ethylbenzene		Ingestion	0.1	Liver and kidney toxicity	IRIS (2005)
		Inhalation (mg/m <sup>3</sup> )	1	Developmental toxicity	IRIS (2005)
Toluene		Ingestion	0.22	Not Specified	Health Canada (1996)
		Inhalation (mg/m <sup>3</sup> )	3.8	Not Specified	Health Canada (1996)
Xylenes		Ingestion	1.5	Not Specified	Health Canada (1996)
		Inhalation (mg/m <sup>3</sup> )	0.18	Not Specified	Health Canada (1996)
Naphthalene		Ingestion	0.02	Decreased weight in males	IRIS (2005)
		Inhalation (mg/m <sup>3</sup> )	3.0E-03	Nasal effects	IRIS (2005)
PCBs		Ingestion	0.001	Not Specified	Health Canada (2004)
		Inhalation	na	na	na
Petroleum Hydrocarbons CWS Fractions					
F2	Aliph>C10-C12	Ingestion	0.1	Hepatic and hematological alterations	CCME (2000)
		Inhalation	na	na	na
	Aliph>C12-C16	Ingestion	0.1	Hepatic and hematological alterations	CCME (2000)
		Inhalation	na	na	na
	Arom>C10-C12	Ingestion	0.04	Decreased body weight	CCME (2000)
		Inhalation	na	na	na
	Arom>C12-C16	Ingestion	0.04	Decreased body weight	CCME (2000)
		Inhalation	na	na	na
F3	Aliph>C16-C21	Ingestion	2	Hepatic granuloma	CCME (2000)
		Inhalation	na	na	na
	Aliph>C21-C34	Ingestion	2	Hepatic granuloma	CCME (2000)
		Inhalation	na	na	na
	Arom>C16-C21	Ingestion	0.03	Nephrotoxicity	CCME (2000)
		Inhalation	na	na	na
	Arom>C21-C34	Ingestion	0.03	Nephrotoxicity	CCME (2000)
		Inhalation	na	na	na

na – not available. When a separate inhalation TRV is not available, the inhalation dose is compared to the oral TRV.

## 4.5.1 Bioavailability

Bioavailability refers to “the fraction of a substance to which the body is exposed (i.e., ingestion, inhalation, dermal contact) that reaches the systemic circulation”. For CoPCs where multiple exposure pathways will be summed for comparison to a single TRV, it is necessary to apply relative bioavailability factors (RBFs) in exposure calculations. This is required as the bioavailability of a substance generally differs from one route of exposure to another (i.e., ingestion vs. inhalation). It should always be assumed that oral exposures have a relative bioavailability of 100% (i.e., RBF = 1). Where inhalation exposures are being summed with oral exposures, the inhalation RBF will generally default to 1 unless there is good evidence that respiratory absorption is considerably less than 100%. Table 10 summarizes the relative bioavailability factors selected for use in this HHRA. RBFs were obtained from Health Canada and the Risk Assessment Information System.

**Table 10: Relative Bioavailability Factors**

CoPC	Oral	Dermal	Inhalation
Aluminum	1	0.001	1
Arsenic	1	0.03	1
Barium	1	0.1	1
Cadmium	1	0.14	1
Iron	1	0.001	1
Lead	1	0.006	1
Lithium	1	0.001	1
Manganese	1	0.001	1
Nickel	1	0.35	1
Uranium	1	0.001	1
Zinc	1	0.02	1
Ethylbenzene	1	0.2	1
Toluene	1	0.12	1
Xylenes	1	0.12	1
Naphthalene	1	0.1	1
Total PCBs	1	0.1	1
TPH fraction F2	1	0.2	1
TPH fraction F3	1	0.2	1

## 4.6 Risk Characterization

The potential health effects associated with exposure to non-carcinogenic chemicals are assessed differently than those for carcinogenic chemicals as these two groups of substances generally have different etiologies and act via unrelated mechanisms.

### Approach and Methodology for Non-Carcinogenic CoPCs

Non-carcinogenic chemicals are generally active through a threshold mechanism where it is assumed that there is a level of exposure (dose) below which no health effects are expected. As the level of exposure (dose) increases to a point where the body can no longer process or excrete the substance, an adverse effect may occur. This juncture is termed the threshold and is unique for every chemical.

For risk characterization of non-carcinogenic CoPCs, hazard quotients (HQs) were calculated for each CoPC by deriving the CDIs of exposed individuals and weighing these against the respective TDIs. Equations showing the derivation of TDIs are presented in Appendix C. Estimated daily intakes (EDIs) were not available for the CoPCs evaluated in this assessment and thus could not be subtracted from the TDI. Therefore, the HQs were calculated as follows:

$$HQ = \frac{CDI}{TDI}$$



where:

CDI     Chronic Daily Intake (mg/kg)/d  
         = sum of all site-specific intake pathways  
         = soil/dust ingestion + soil/dust dermal contact + soil particulate/dust inhalation + ingestion and dermal  
             contact with surface water + ingestion of wild game  
TDI     Tolerable Daily Intake (mg/kg)/d

Because this risk assessment did not address all potential exposure pathways, the target HQ was set to 0.2. By using a HQ benchmark of 0.2, 80% of an individual's intake of CoPCs is assumed to come from off-site exposures, such as ingestion of contaminants via supermarket goods. If the calculated total HQ is less than 0.2, then intake of CoPCs from site exposures does not exceed the tolerable level and no adverse health effects are expected.

### Approach and Methodology for Carcinogenic CoPCs

Three CoPCs identified at CAM-D (i.e., arsenic, cadmium and total PCBs) have both carcinogenic and non-carcinogenic potential, and as such, these substances are assessed as both carcinogens and non-carcinogens.

To derive a conservative estimate of the total incremental lifetime cancer risk (ILCR) associated with exposure to arsenic, cadmium or total PCBs at CAM-D, the estimated exposure is multiplied by the appropriate slope factor or unit risk as shown below:

$$\text{ILCR} = \text{LADD} \times \text{CSF}$$

where:

ILCR     Incremental Lifetime Cancer Risk  
LADD     Lifetime Averaged Daily Dose (mg/kg)/day  
CSF     Cancer Slope Factor (mg/kg)/day

The ILCR estimates the incremental probability that an individual will develop cancer as a result of lifetime exposure to a substance (e.g., arsenic at CAM-D). The incremental lifetime cancer risk is in addition to the probability of developing cancer due to ambient exposures. Given the conservatism associated with the derivation of cancer slope factors and unit risks, Health Canada has recommended a benchmark cancer risk level of 1-in-100,000 ( $1 \times 10^{-5}$ ) for the purposes of assessing and managing federal sites contaminated with carcinogenic substances. Accordingly, cancer risks are deemed negligible when the estimated ILCR is  $\leq 1\text{-in-}100,000$  ( $1 \times 10^{-5}$ ). Calculations of lifetime average daily dose (LADD) are based on methods presented by the US EPA (1989), CCME (1996) and OME (1996). Detailed ILCR derivations and parameter values used in the analyses are provided in Appendix B.

In general, exposure pathways and intake values were unchanged from those used in the development of non-carcinogenic HQs. However, exposures were averaged over a lifetime rather than a specific age group.



#### 4.6.1 Non-Carcinogenic Risk Estimates

HQs for aluminum, arsenic, barium, cadmium, iron, lead, lithium, manganese, nickel, uranium, zinc, ethylbenzene, toluene, xylenes, naphthalene, total PCBs, and TPH fractions F2 and F3, which were derived using the 95% UCL of the geometric mean as EPC, are presented in Table 11.

Using the traditional land use scenario, the calculated total hazard quotients for all CoPCs at CAM-D are less than 0.2. Thus, exposure to all on-site CoPCs results in a negligible risk to human receptors.

It should be noted that soil data was unavailable for aluminum, iron, lithium, manganese and uranium. Thus, HQs were calculated for these CoPCs using only the ingestion and dermal contact with surface water pathways. Additionally, although ethylbenzene, toluene, xylenes, naphthalene, total PCBs and TPH fractions F2 and F3 were identified as CoPCs based on human health soil screening, they were not quantitatively assessed in surface water as they were not measured above the level of detection in this media.

Table 12 summarizes the contribution of each pathway to the total HQs.

**Table 11: Total Hazard Quotients for Non-Carcinogenic CoPC at CAM-D**

CoPC	EPC Soil (mg/kg)	EPC Water (mg/L)	Total HQ	Target HQ	Exceeds Target HQ
Aluminum	–	4.7	1.68E-02	2.0E-01	No
Arsenic	3.0	8.53E-04	1.61E-02	2.0E-01	No
Barium	2.33E+02	6.96E-02	3.55E-02	2.0E-01	No
Cadmium	8.17E-01	1.62E-04	5.15E-02	2.0E-01	No
Iron	–	4.2	5.05E-02	2.0E-01	No
Lead	2.03E+01	3.69E-03	8.19E-02	2.0E-01	No
Lithium	–	9.80E-03	1.32E-02	2.0E-01	No
Manganese	–	1.02E-01	2.63E-03	2.0E-01	No
Nickel	8.4	5.10E-03	1.71E-02	2.0E-01	No
Uranium	–	1.25E-02	7.48E-02	2.0E-01	No
Zinc	1.29E+02	6.87E-02	3.77E-02	2.0E-01	No
Ethylbenzene	3.20E-01	ND	1.07E-06	2.0E-01	No
Toluene	3.04E-01	ND	4.29E-07	2.0E-01	No
Xylenes	2.8	ND	8.04E-07	2.0E-01	No
Naphthalene	4.1	ND	9.08E-05	2.0E-01	No
Total PCBs	3.49E-02	ND	2.85E-03	2.0E-01	No
TPH F2 Fraction	1.81E+03	ND	8.82E-02	2.0E-01	No
TPH F3 Fraction	3.56E+03	ND	1.54E-02	2.0E-01	No

Note: –, data unavailable; ND, not detected.

**Table 12: Contribution of Individual Pathway HQs to Total HQs**

CoPC	Soil Ingestion HQ	Soil Dermal HQ	Soil/Dust Inhalation HQ	Surface Water Ingestion HQ	Surface Water Dermal HQ	Wild Game Ingestion HQ
Aluminum	–	–	–	9.79E-03	7.02E-03	–
Arsenic	2.79E-03	7.71E-05	8.12E-05	5.95E-03	4.26E-03	2.92E-03
Barium	4.07E-03	3.75E-04	1.35E-02	9.10E-03	6.52E-03	1.92E-03
Cadmium	2.85E-04	3.67E-05	1.16E-04	4.24E-04	3.04E-04	5.03E-02
Iron	–	–	–	2.94E-02	2.11E-02	–
Lead	1.58E-03	8.71E-06	4.59E-05	2.14E-03	5.26E-04	7.76E-02
Lithium	–	–	–	1.03E-03	7.35E-04	1.14E-02
Manganese	–	–	–	1.53E-03	1.10E-03	1.39E-17
Nickel	1.18E-04	3.79E-05	6.74E-03	5.33E-04	7.65E-05	9.59E-03
Uranium	–	–	–	4.36E-02	3.12E-02	4.76E-05
Zinc	1.20E-04	2.21E-06	3.49E-06	4.79E-04	2.06E-04	3.69E-02
Ethylbenzene	8.93E-07	1.64E-07	4.60E-09	ND	ND	3.92E-09
Toluene	3.85E-07	4.26E-08	1.15E-09	ND	ND	5.49E-10
Xylenes	5.22E-07	5.77E-08	2.24E-07	ND	ND	7.52E-10
Naphthalene	5.74E-05	5.29E-06	1.97E-05	ND	ND	8.39E-06
Total PCBs	9.74E-06	8.97E-07	1.01E-06	ND	ND	2.84E-03
TPH F2 Fraction	6.56E-03	1.21E-03	4.68E-05	ND	ND	8.04E-02
TPH F3 Fraction	7.02E-03	1.29E-03	2.04E-04	ND	ND	6.90E-03

Note: –, data unavailable; ND, not detected.

#### 4.6.2 Carcinogenic Risk Estimates

Total ILCRs were calculated for arsenic, cadmium and total PCBs and are presented in Table 13.

The total ILCRs for arsenic, cadmium and total PCBs were less than the acceptable Health Canada benchmark of  $1 \times 10^{-5}$ . Therefore, the carcinogenic risks associated with exposures to these CoPCs at CAM-D are expected to be negligible for human receptors.

Table 14 summarizes the contribution of each pathway considered to the total ILCRs. Since only an inhalation slope factor was available for cadmium (i.e., sole pathway by which cadmium is carcinogenic), only this pathway was assessed and the total ILCR can be attributed to inhalation of soil particles. Additionally, total PCBs were not detected in surface water (i.e.,  $<0.0001$ ) and as such the surface water ingestion and dermal contact pathways were not considered in calculation of the total ILCR.

**Table 13: Incremental Lifetime Cancer Risks for Carcinogenic CoPCs at CAM-D**

CoPC	EPC (mg/kg)	Total ILCR	Target HQ	Exceeds Target HQ?
Arsenic	3.0	5.21E-06	1.00E-05	No
Cadmium	0.82	1.23E-07	1.00E-05	No
Total PCBs	0.125	2.69E-06	1.00E-05	No

**Table 14: Contribution of Individual Pathway ILCRs to Total ILCRs**

CoPC	Soil Ingestion ILCR	Soil Dermal ILCR	Soil/Dust Inhalation ILCR	Surface Water Ingestion ILCR	Surface Water Dermal ILCR	Wild Game Ingestion ILCR
Arsenic	1.82E-07	3.92E-08	2.94E-07	2.91E-06	3.51E-09	1.78E-06
Cadmium	–	–	1.23E-07	–	–	–
Total PCBs	5.43E-09	3.89E-09	1.80E-10	ND	ND	2.68E-06

– unable to calculate as ingestion and dermal contact slope factors are unavailable.

ND – Not Derived.

#### 4.6.3 Summary of Risks on Site

Using the traditional land use scenario for CAM-D, the HQs for all modeled CoPCs were less than 0.2. Thus, the risks associated with on-site exposures to these CoPCs are expected to be negligible for human receptors.

The ILCRs for all carcinogenic CoPCs were less than the acceptable Health Canada benchmark of  $1 \times 10^{-5}$ . Therefore, the risks associated with on-site exposures to these CoPCs are expected to be negligible for human receptors.

It should be noted that this assessment is based on numerous conservative assumptions, such as the assumption that receptors visit the site for their lifetime of exposure, all metals are 100% bioavailable, exposure is to the 95% UCL concentration for the duration of site visit, etc. This risk assessment also does not incorporate any potential remediation and the likely reduction in CoPC site concentrations. Thus, the calculated risks are inherently conservative and likely over-estimate the actual risks associated with exposure to CoPCs at CAM-D.

#### 4.7 Human Health Site-Specific Target Levels

As no substances exceeded human health benchmarks, it was unnecessary to generate site-specific target levels (SSTLs). SSTLs represent concentrations above which adverse health effects are possible and risk management may be required.

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## 4.8 Uncertainty Analysis

Risk estimates normally include an element of uncertainty, and generally these uncertainties are addressed by incorporating conservative assumptions in the analysis. As a result, risk assessments tend to overstate the actual risk. Although many factors are considered in preparation of a risk analysis, the results are generally only sensitive to very few of these factors. The uncertainty analysis is included to demonstrate that assumptions used are conservative, or that the analysis result is not sensitive to the key assumptions.

A risk assessment containing a high degree of confidence will be based on:

- conditions where the problem is defined with a high level of certainty based on data and physical observations;
- an acceptable and reasonable level of conservatism in assumptions that will ensure that risks are overstated; or
- an appreciation of the bounds and limitations of the final solution.

The exposure assessment performed as part of this assessment was based on:

- available data to describe existing surface soil conditions and CoPC distributions;
- sound conservative assumptions for certain parameters, as required; and
- well-understood and generally accepted methods for risk prediction.

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### 4.8.1 Uncertainties in Toxicological Information

There is a very limited amount of toxicological information on the effects associated with human exposures to low levels of chemicals in the environment. What human information is available is generally based on epidemiological studies of occupationally exposed workers. These studies are generally limited in scope and provide results that may not be applicable to chronic or continuous exposures to low levels of chemicals. Because human toxicological information is limited, reference doses and cancer potency estimates for many compounds are based on the results of dose-response assessment studies using animals.

The use of experimental animal data to estimate potential biological effects in humans introduces uncertainties into the evaluation of potential human health effects. These estimations require that a number of assumptions be made:

- The toxicological effect reported in animals is relevant and could occur in humans.
- The assumption that extrapolation from high-dose studies to low-dose environmental exposures adequately represents the shape of the dose-response curve in the low-dose exposure range.
- Short-term exposures used in animal studies can be extrapolated to chronic or long-term exposures in humans.
- The uptake of a compound from a test vehicle (drinking water, food, etc.) in animals will be the same as the uptake of the chemical from environmental media (soil, sediment, air-borne particulate matter) in humans.
- The pharmacokinetic processes that occur in the test animals also occur in humans.

There are clearly a number of uncertainties associated with extrapolating from experimental animal data to humans. To address these weaknesses, regulatory agencies, such as the Health Canada and the US EPA, incorporate a large number of conservative assumptions to try and account for the uncertainties associated with this process. The uncertainties are accounted for by the use of uncertainty factors that serve to lower the reference dose well below the level at which adverse health effects have been reported in the test species. Uncertainty factors are generally applied by factors of 10 and are used to account for the following types of uncertainties:

- Variation within the population (protection of sensitive members of the population).
- Differences between humans and the test species.
- Differences in using short or medium-term studies to estimate the health effects associated with long-term or chronic exposures.
- Limitations in the available toxicological information.

The magnitude of the uncertainty factors applied by the various regulatory agencies provides an indication of the level of confidence that should be placed in the reference value. Uncertainty factors typically range between 100 and 10,000, although some can be lower than 10. The latter values are found for a few chemicals where sound and substantial human toxicological information is available to enable the setting of toxicological end-point solely on the basis of human epidemiological information.

The application of uncertainty factors is intended to introduce a high degree of conservatism into the risk assessment process and to ensure, as far as possible, that limited exposures that exceed the reference concentrations will not result in adverse human health effects. Because risk assessments that use these regulatory limits incorporate the conservatism used in the development of the toxicological information, the results can generally be viewed as being extremely conservative.

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#### **4.8.2 Summation of Hazards for a Single Compound**

For some CoPCs, the toxicity values for inhalation and oral exposures are based on different biological end-points. In this case, the summation of exposures or hazard indices is not a sound toxicological practice and will not provide adequate assessments of either the inhalation or ingestion hazard. Therefore, it is necessary to assess the biological end-points separately. The estimate of overall risk would be based on the greater of the two risks. Inhalation and ingestion exposure hazard have been assessed independently and the greater of the two hazards selected as the representative hazard. However, for the CoPCs considered in this risk assessment where the biological end-point is the same for both routes of exposure, estimates of hazard were based on estimates of total exposure.

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#### **4.8.3 Summation of Hazards of Multiple Compounds**

The summation of hazards between compounds that do not have the same biological end-point or mechanism of action has little practical meaning.

The summation of hazard indices across compounds is only supportable when the individual compounds affect the same target organ and have similar mechanisms of action. In these cases, the summation of hazard indices may provide a better estimate of total risk than evaluations based on

exposures to single chemicals. For this risk assessment, toxicity values for the assessed metals are based on different biological end-points, hence, hazard indices have not been summed to provide an estimate of the overall hazard associated with these exposures.

#### 4.8.4 Modeling Assumptions

Table 15 summarizes the assumptions used in this risk analysis, and provides an evaluation of each assumption and an opinion as to whether the assumption is acceptable.

**Table 15: Modeling Assumptions**

Risk Assessment Study Factor/Assumption	Justification	Analysis Likely to Over/Under Estimate Risk?	Acceptable Assumption?
<b>Hazard Identification</b>			
1. Screening CoPCs against generic human health based guidelines (i.e., CCME, OME, US EPA).	Generic guidelines are inherently conservative as they are derived without consideration of site-specific information. As such, they can be reliably applied under most circumstances. Substances present at concentrations less than generic guidelines are unlikely to be of concern.	Neutral	Yes
2. 95% upper confidence limit of the mean serving as exposure point concentration (EPC).	Using the 95% UCL of the mean as the EPC will overestimate risks posed by on-site CoPCs. Because sampling is biased towards highly contaminated areas, the 95% UCL of the mean are likely to be much greater than the true mean concentrations of CoPCs on site.	Over-estimate.	Yes
<b>Receptor Characteristics</b>			
1. For analyses of non-carcinogenic exposures, a toddler (0.5 – 4 years old) was selected as the receptor.	Young children represent the most sensitive age group for assessing non-carcinogenic effects. Resultant risks are generally over protective for an adult population. This approach is in accordance with standard practice (i.e., Health Canada and US EPA).	Neutral for young children but will over-estimate risks to adults.	Yes
2. For risk analyses of carcinogenic substances, it is assumed that the receptor visits CAM-D yearly from birth to 75 years of age, and a lifetime average exposure is calculated.	For carcinogenic chemicals, this approach is the most protective. In contrast, CCME models adult exposure over 50 years (20-75 years of age) while the US EPA models exposure for 25 years (0-25 years of age) averaged over a lifetime.	Approach likely to over-estimate risk.	Yes
3. For the traditional land use scenario, it is assumed that both potential receptors (toddler and composite lifetime) are on site 21 days/year for 24 hours/day. It is also assumed that human receptors will be hunting on the site.	These values provide a reasonable maximum exposure estimate for the toddler but likely overestimate lifetime exposure.	Neutral to over-estimate.	Yes

**Table 15: Modeling Assumptions**

<b>Risk Assessment Study Factor/Assumption</b>	<b>Justification</b>	<b>Analysis Likely to Over/Under Estimate Risk?</b>	<b>Acceptable Assumption?</b>
<b>Toxicological Information</b>			
1. Used most current toxicological values available (i.e., Health Canada, US EPA Integrated Risk Information System),	This approach is in accordance with standard practice, and provides the most current scientific basis with which to conduct a risk assessment.	Neutral	Yes
2. Potential antagonistic, additive and synergistic effects of chemical mixtures were not quantitatively assessed.	The summation of hazards for compounds that do not have the same biological end-point or mechanism of action has little practical meaning. Summation of hazard indices is only supportable when the individual compounds affect the same target organ and have similar mechanisms of action.	Neutral	Yes
<b>Risk Characterization</b>			
1. Exposure was modeled for four potential pathways: soil/dust ingestion, inhalation and dermal contact; inhalation of soil vapours; ingestion of wild game; and ingestion and dermal contact with surface water.	CCME generic guidelines are based solely on soil ingestion. Therefore, a multi-pathway approach is more protective of human and ecological health.	Neutral to over-estimate	Yes
2. Use of default CCME soil ingestion rate of 80 mg/day.	CCME adopted a soil ingestion rate of 80 mg/day for toddlers when developing the 1999 soil quality guidelines. In Nunavut, the climate restricts outdoor exposure to soil to a limited period of time each year. Nevertheless, during the winter months, residents may still be exposed to soil-derived household dust.	Neutral	Yes

**Table 15: Modeling Assumptions**

Risk Assessment Study Factor/Assumption	Justification	Analysis Likely to Over/Under Estimate Risk?	Acceptable Assumption?
3. Evaluation of the current nutritional status of local receptors was not included in the scope of work.	The nutritional status of Inuits in the Canadian Arctic is commonly different than that of southern populations due to dissimilar diets. Micronutrient deficiencies (e.g., Ca, Fe, Vitamin D) have been reported, and may affect the absorption of other chemicals into the body (e.g., increased absorption of lead). However, quantitative data is not available to determine the scope or magnitude of this effect. Furthermore, information is not available regarding the current health status of residents in the vicinity of CAM-D who generally have a mixed diet of supermarket foods and country foods (e.g., wild game). Evaluation of current health status, past exposures and lifestyle factors (e.g., smoking) are beyond the scope of this risk assessment, which is a forward-looking process that considers current and future exposures based on post-reclamation site conditions.	Neutral	Yes
4. ILCR set to 1 in 100,000 ( $10^{-5}$ ) for evaluating exposures to carcinogenic CoPCs at CAM-D.	This value has been adopted by CCME to represent an "acceptable" target risk. Similarly, Health Canada uses target risks in the range of $10^{-5}$ to $10^{-6}$ .	Neutral	Yes
5. For evaluating exposures to CoPCs at CAM-D, target HQ = 0.2.	Because this risk assessment did not address all potential exposure pathways, the target HQ was set to 0.2 as per CCME guidance. By using a HQ benchmark of 0.2, 80% of an individual's intake of CoPCs is assumed to come from off-site exposures, such as ingestion of contaminants via supermarket goods	Neutral	Yes



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## 5.0 ECOLOGICAL RISK ASSESSMENT

Jacques Whitford Limited (Jacques Whitford) was commissioned by PWGSC on behalf of DIAND to conduct a qualitative and quantitative ecological risk assessment (ERA) at the CAM-D Intermediate Dew Line site (Figures 1 and 2). The primary objectives of the ERA will be to evaluate the risks for adverse effects to non-domesticated fauna based on current conditions and habitats, and to develop site-specific target levels (SSTLs). The study addresses concerns regarding exposure to potentially hazardous metals and organic chemicals found in surface soil and water, vegetation and various biological specimens

Ecological risk assessment is the process that evaluates the likelihood that adverse environmental effects may occur, or are occurring, as a result of exposure to one or more stressors (Suter, 1993). The potential hazards, or chemicals of potential concern (CoPCs), identified within the study area are substances in environmental media (soil, surface water and terrestrial vegetation) that originated from sources associated with past operations at CAM-D. Therefore, the purpose of this ERA was to qualitatively and quantitatively assess the likelihood and potential magnitude of adverse effects to valued environmental components (VECs), or ecological receptors, from exposure to contaminants at Cam-D.

To perform the ERA at CAM-D, the site was divided into five geographic areas based on geographic boundaries, previous assessment activities and analytical results. The main purpose of this division was to ensure that "hot spots" in one geographic area do not unduly influence, or bias, the assessment of risks in another. For instance, excessive TPH contamination at the airstrip may pose a risk to ecological receptors in this area; however, this contamination is negligible when evaluating the risks from TPH exposure at the Simpson Lake shoreline. CAM-D was divided into the following geographic regions:

- Area A: Main Station and Proximity - the main station and immediate surrounding areas including the garage, warehouse and module train remnants, POL tank and pumphouse, electrical cabinets, shack remains, and the diesel tanks on the road towards the airstrip;
- Area B: Outfall Area - sewage outfall area northwest of the module train remnants;
- Area C: Barrel Dumps - the main barrel dump southeast of the garage as well as the barrel dump by the pallet line area;
- Area D: Main Station Dump - the large pile of debris northeast of the station beyond the pallet line area;
- Area E: Airstrip - including the remains at the junction of the airstrip and access road to the station; and
- Area F: Simpson Lake Shoreline - including the debris along the southeast shore of Simpson Lake.

Risk characterization was performed individually for each VEC in each area to determine whether CoPCs in these discrete locations posed a health risk to ecological receptors.

## Approach and Objectives

This ERA was conducted according to established health risk assessment protocols endorsed by Health Canada (1994), CCME (1996) and the US EPA (1996). The specific objectives of the ERA are to:

- Qualitatively characterize the potential ecological receptors that have been observed or could be present in terrestrial habitats on, or adjacent to, the site.
- Evaluate the potential exposures of ecological receptors to CoPCs in various environmental media within terrestrial habitats under current conditions.
- Quantify the risks associated with exposure of ecological receptors to CoPCs in various environmental media under current conditions.
- If an unacceptable risk is identified, determine acceptable concentrations of contaminants (site-specific target levels, SSTLs) that would allow re-establishment of the habitat and would not pose ongoing risks.

For this ERA, a general framework similar in concept to that used for the human health risk assessment was adopted but is distinguished by the following features:

- The ecological risk assessment does not consider effects on individuals of a single species, rather it is concerned with potential effects on populations, communities or ecosystems. In order to achieve this goal, the toxicity reference values (TRVs) that are used for evaluation of potential adverse effects are based on lowest-observed adverse effect level (LOAEL) data from the ecotoxicological literature, with a focus on sub-lethal reproductive or developmental endpoints.
- There is no single, generalized set of ecological receptors that can be applied to every site, thus the selection of VECs and exposure pathways for the ERA process is site-specific.
- If appropriate, the ecological risk assessment may consider non-chemical, as well as chemical, stressors.

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### 5.1 Ecological Risk Assessment Framework

Conceptually, ecological risk assessments are comprised of three main components:

- **Problem Formulation** – A review is conducted of all available physical and biological data for the site and receptor habitats that may be affected by releases of chemicals to environmental media. This serves to: i) identify potential ecological receptors (i.e., biological communities, populations, individuals or habitats potentially at risk), ii) determine contaminants of potential concern and other ecological stressors, iii) identify operating exposure pathways and iv) establish appropriate assessment and measurement endpoints for the ecological risk assessment. Each of these elements is integrated into a conceptual model that is specific to the site.
- **Exposure-Effects Assessment** - Qualitative or quantitative evaluation of the likelihood and/or degree to which the receptors will be exposed to the hazard(s) (i.e., CoPCs). Here, exposure-response standards are identified based on the concentrations of CoPCs in various environmental media.

- **Risk Characterization** - The nature and magnitude of potential ecological risks are evaluated by comparing exposure estimates for various media, exposure-response standards for the ecological receptors and results of site-specific surveys. Exposures may be based directly on the concentration of a CoPC in an environmental media, or on an estimated dose or intake rate for the CoPC. In general, exposure-response standards are either biological responses associated with the measured or estimated concentration of a CoPC, such as mortality or impairment, or toxicological responses to the estimated dose, such as impaired reproduction or development. Risk characterization also includes a discussion of the uncertainties in the analysis, an evaluation of the necessity for remedial action and may involve estimating the maximum chemical concentrations, or site-specific threshold levels (SSTLs), consistent with an acceptable level of risk.

The organization of this ERA is in accordance with the above elements. The hazard identification process is described in Section 5.2.1, ecological receptors (VECs) are identified in Section 5.2.3 and exposure pathways by which the VECs may come into contact with CoPCs are outlined in Section 5.3.1. Information in these three sections is brought together in Section 5.3.2, which describes the conceptual site model. Risk characterization for the CAM-D DEW Line site is presented in Section 5.4. For those substances that may pose a risk to one or more ecological receptors, site-specific threshold levels are calculated and presented in Section 5.5. Finally, an uncertainty analysis for the ERA is presented in Section 5.6.

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## 5.2 Chemical Screening

The ERA dealt exclusively with substances present in environmental media that are accessible to wildlife. To this end, CAM-D data were screened such that only surface soils (0 to 30 cm depth), water and vegetation were considered. Subsurface soils were not included as they were deemed inaccessible to wildlife. The data used in the present ERA was primarily that supplied by Earth Tech Environmental Inc. who, on behalf of PWGSC and DIAND, conducted a field investigation and sampling program at CAM-D in August of 2005. A detailed list of samples and sampling locations is presented in a separate report (Earth Tech, 2005). In addition to the data collected in 2005, analyses for inorganic parameters and PCBs in soil, water and vegetation were conducted and presented by the Environmental Sciences Group at RMC in a 1994 report. A detailed list of samples and sampling locations can be found in the 1994 report. Data from both reports were compiled and included in the present ecological risk assessment.

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### 5.2.1 Identification of Chemicals of Potential Concern

This component of the ERA involved identifying chemical substances at CAM-D that have the potential to adversely impact the health of ecological receptors. These substances, CoPCs, were identified by screening the maximum surface soil and water concentrations of all chemicals (phase II and III) against generic CCME guidelines for ecological health (CCME, 1999). Maximum surface soil concentrations were also screened using INACs Dew Line Cleanup (DLCU) criteria (INAC, 2005). These criteria were developed by Defence Construction Canada (DCC) for the Department of National Defence for use at the main Dew Line sites, and are considered to be protective of the arctic ecosystem. In instances where DLCU criteria were less than CCME guideline values, DLCU criteria were used in place of CCME guidelines.

## Surface Soil

The results of ecological health soil screening for CAM-D are presented in Table 16. The table lists those substances deemed to pose a risk to ecological health, the maximum measured soil concentration of each and the relevant guideline. All substances presented in Table 16 were carried forward as CoPCs into the quantitative ERA.

As indicated in Table 16, the maximum soil concentration of arsenic, barium, copper, nickel and zinc was greater than the corresponding CCME ecological health-based guideline and these substances were carried forward into the ERA. Similarly, the maximum soil concentration of cadmium exceeded Tier II DLCU criteria and cadmium was also carried forward. The maximum soil concentration of lead was less than the applicable guidelines but lead was carried forward into the ERA to be consistent with the human health risk assessment.

For organic compounds, the maximum soil concentration of ethylbenzene, toluene, xylenes, TPH fractions F1, F2, F3 and F4, naphthalene and total PCBs was greater than the corresponding CCME ecological health-based guideline. Therefore, these substances were also carried forward as CoPCs into the ERA.

All substances identified as CoPCs based on ecological health soil screening were quantitatively assessed in surface water, providing they were measured above the level of detection in this media. This ensures that derived total hazard quotients (i.e., risk estimates) take into consideration all potential exposure pathways.

## Surface Water

Because surface water may serve as a drinking water source for terrestrial wildlife, it was necessary to assess this environmental medium for CoPCs. Maximum concentrations of all substances measured in surface water were screened against generic CCME guidelines developed for the protection of freshwater aquatic life (CCME, 1999). The results of ecological health surface water screening for CAM-D are presented in Table 17.

The only new CoPC identified via ecological health surface water screening was silver. All other surface water substances carried forward into the ERA had previously been identified as CoPCs through screening of soil data (i.e., cadmium, copper, lead and zinc) or were carried forward to be consistent with the human health risk assessment (i.e., aluminum, iron, lithium, manganese and uranium).

**Table 16: Ecological Health Soil Screening for CAM-D**

Soil CoPCs	Phase II <sup>1</sup> Maximum Concentration	Phase III Maximum Concentration	Total No. Of Samples	CCME <sup>2</sup>	CCME CWS <sup>3</sup>	DLCU <sup>4</sup> Criteria Tier I	DLCU <sup>4</sup> Criteria Tier II	No. Of Samples Exceeding Guidelines
				Ecological Health	Ecological Health			
Inorganics								
Arsenic	60	2.8	93	17	-	NC	30	1
Barium	na	1720	93	500	-	NC	NC	1
Cadmium	9.3	5.13	93	10	-	NC	5	2
Copper	131	107	93	63	-	NC	100	2
Lead	183	92.3	93	300	-	200	500	0
Nickel	53	18	93	50	-	NC	100	1
Zinc	1370	1570	93	200	-	NC	500	4
Organics								
Ethylbenzene	na	4.29	52	1.2	-	-	-	2
Toluene	na	4.77	52	1.4	-	-	-	1
Xylenes (total)	na	39.4	52	1	-	-	-	4
F1	na	1300	52	NC	260	-	-	4
F2	na	16700	52	NC	900	-	-	12
F3	na	28300	52	NC	800	-	-	12
F4	na	13300	52	NC	5600	-	-	4
Naphthalene	na	37	42	0.6	-	-	-	5
PCBs (total)	1.5	0.6	80	1.3	-	-	-	1

**Notes:**

All units are in mg/kg

Applicable guideline

na - not assessed

NC - No Criteria

1 - RMC, 1994

2 - Canadian Council of Ministers of the Environment (CCME) Canadian Environmental Quality Guidelines: Soil Quality Guidelines - Residential/Parkland, Environmental Health (1991, Updated 1997, 1999 & 2003)

3 - Canadian Council of Ministers of the Environment "Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil" (May 2001) fine-grained surface soils – Residential land use (eco soil contact)

4 - INAC, 2005. Abandoned Military Site Remediation Protocol. Appendix B, DEW Line Cleanup (DLCU) Soil Criteria.

**Table 17: Ecological Health Surface Water Screening**

Surface Water CoPCs	Phase III Maximum Concentration	Total No. Of Samples	CCME <sup>1</sup> FAL Guideline	No. Of Samples Exceeding Guideline	% Of Samples Exceeding Guideline
<b>Inorganics</b>					
Aluminum	9.3	7	0.1	5	71.4
Cadmium	0.00028	7	0.000017	4	57.1
Copper	0.01	7	0.002	4	57.1
Iron	8.2	7	0.3	3	42.9
Lead	0.0072	7	0.002	1	14.3
Lithium	0.016	7	NC	-	-
Manganese	0.14	7	NC	-	-
Silver	0.0002	7	0.0001	5	71.4
Uranium	0.02	7	NC	-	-
Zinc	0.122	7	0.03	2	28.6

**Note:**

All units are in mg/L

NC - No Criteria

1 - Canadian Council of Ministers of the Environment (CCME) Canadian Environmental Quality Guidelines: Freshwater Aquatic Life (1999, Updated 2003)



## Exposure Point Concentrations

Exposure point concentrations (EPCs) were calculated for each CoPC identified in surface soil, for all six individual areas of CAM-D and are summarized in Table 18. Only one set of surface water EPCs was generated (Table 19) and used to derive HQs for each distinct site, as it was assumed that VECs in each area obtain water from the same sources. The EPC is intended to be a conservative (i.e., guarded, but not necessarily worst-case) estimate of the average on-site concentration to which wildlife may be exposed. Where CoPCs were measured above the level of detection and  $n \geq 6$ , the EPC is estimated as the 95% upper confidence limit of the mean concentration. Alternatively, if  $n < 6$  the EPC is set to the maximum measured concentration. Where CoPCs were undetected, the EPC was estimated as half the detection limit.

**Table 18: Exposure Point Concentrations for Surface Soil at Individual Areas of CAM-D**

	Main Station	Outfall	Barrel	Main Station		Simpson
CoPCs	and Proximity	Area	Dumps	Dump	Airstrip	Lake Shoreline
<b>Inorganics</b>						
Aluminum	na	na	na	na	na	na
Arsenic	1.2	12.9	1	1.6	1.6	1
Barium	109	81	94	92	94	28
Cadmium	0.42	0.83	0.59	0.61	0.94	0.5
Copper	9.3	71	7.5	15.1	16.7	17.5
Iron	na	na	na	na	na	na
Lead	9.7	12.2	7.8	30.4	36.3	179
Lithium	na	na	na	na	na	na
Manganese	na	na	na	na	na	na
Nickel	7.7	27	5.3	11.6	9.2	5.4
Silver	0.067	0.1	0.05	0.1	0.1	0.05
Uranium	na	na	na	na	na	na
Zinc	50	406	48	103	124	540
<b>Organics</b>						
Ethylbenzene	0.026	na	0.01	na	0.01	na
Toluene	0.022	na	0.013	na	0.02	na
Xylenes	0.069	na	0.01	na	0.01	na
TPH F1	10.2	na	1.9	na	0.5	na
TPH F2	346	na	77	na	830	na
TPH F3	362	na	835	na	25500	na
TPH F4	92	na	470	na	4560	na
Naphthalene	0.2	na	0.025	na	0.13	na
Total PCBs	0.067	0.64	0.019	0.055	0.043	0.098

**Notes:**

All units are in mg/kg

na - not assessed

EPC = 95% UCL of the geometric mean

Where  $n \leq 6$ , EPC = maximum measured concentration

Where CoPCs are below the level of detection, EPC = half of detection limit.

**Table 19: Exposure Point Concentrations of CoPCs in Surface Water at CAM-D**

CoPCs	Surface Water
	Exposure Point Concentration (mg/L)
<b>Inorganics</b>	
Aluminum	4.70E+00
Arsenic	8.53E-04
Barium	6.95E-02
Cadmium	1.62E-04
Copper	7.85E-03
Iron	4.20E+00
Lead	3.69E-03
Lithium	9.83E-03
Manganese	1.02E-01
Nickel	5.10E-03
Silver	1.46E-04
Uranium	1.25E-02
Zinc	6.87E-02
<b>Organics</b>	
Ethylbenzene	ND
Toluene	ND
Xylenes	ND
TPH F1	ND
TPH F2	ND
TPH F3	ND
TPH F4	na
Naphthalene	ND
Total PCBs	ND

**Notes:**

na - not assessed

ND – not detected

EPC = 95% UCL of the geometric mean

## 5.2.2 Receptor Identification

Receptor selection was based on fundamental ecological considerations, but was also guided by observations made during a site visit and information solicited from members of the local community. The following criteria were considered in selecting receptors for use in this ERA:

- keystone species known to be central to ecosystem function;
- exposed to surface soil and water at the site;
- representative of higher and lower trophic feeding levels (i.e., herbivorous and carnivorous animals);
- present on or near the site for some or most of the year;
- of substantial cultural and/or economic significance; and
- endangered or sensitive species.



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### 5.2.3 Valued Environmental Components – Wildlife Receptors

Valued environmental components (VECs) are defined as resources or environmental features important to human populations. VECs may have economic and/or social value, and are generally of intrinsic ecological significance. These components also provide a baseline from which the impacts of development can be evaluated, including changes in management and/or regulatory policies.

Based on the above criteria, and on observations made during a site visit, the following ecological receptors were selected for inclusion in the quantitative ERA for CAM-D:

- collared lemming (*Dicrostonyx torquatus*),
- ermine (*Mustela erminea*),
- arctic hare (*Lepus arcticus*),
- arctic fox (*Alopex lagopus*),
- caribou (*Rangifer tarandus groenlandicus*),
- rock ptarmigan (*Lagopus mutus*), and
- snowy owl (*Nyctea scandiaca*).

All of these wildlife species have been observed on or near the site. Receptors were selected to be typical and representative of those likely to be found at CAM-D, including birds and mammals, and herbivores and predators. This approach was based on the premise that if highly exposed components of the ecosystem are protected then populations of other exposed biota will also be adequately protected. Although this methodology is considered reasonable by CCME (1996a, b), it is recognized that the protection of selected ecological receptors for particular endpoints (e.g., reproduction) may not always effectively protect all endpoints for all ecological receptors. The choice of representative receptors was made, in part, using a trophic level approach in that they were selected to represent lower and higher trophic levels. As a result, representative species were not selected because of their sensitivity (information on sensitivity of arctic receptors is lacking), but because of their ecological significance, place of habitat and trophic level. The following sections provide more detailed descriptions of the VECs listed above.

The collared lemming (*Dicrostonyx torquatus*) is a small rodent, weighing approximately 0.09 kg, which lives on the tundra throughout the high arctic (CWS & CWF, 2005). It is a key species in arctic ecosystems, as it is an integral food source for many arctic species (e.g., ermine, arctic fox, snowy owl, gyrfalcon). Lemmings have roughly four-year cycles of drastically fluctuating population density (CWS & CWF, 2005). Average home range sizes are approximately 0.35 ha for females and 2.40 ha for males (Predavec and Krebs, 2000). The lemming is herbivorous, feeding on whatever vegetation exists within its habitat (e.g., willow, cranberries) (CWS & CWF, 2005). In the winter, lemmings do not hibernate; rather, they forage in the space that forms between the snow and soil. On average, collared lemmings consume 0.02 kg of wet-weight food per day and 0.01 L of water or its equivalent per day (Appendix B).

Ermine (*Mustela erminea*), or short-tailed weasel, weigh on average 0.09 kg (USEPA, 1993) and are found in the north temperate regions of Eurasia and North America in riparian woodlands, marshes, shrubby fencerows and open areas adjacent to forests or shrub borders (Loso, 1999). Ermine home ranges vary from approximately 10 ha to 20 ha, and home ranges of males are usually twice the size of

that for females. Although fierce and aggressive, potential predators include foxes, martens, badgers, raptors and domestic cats (Loso, 1999). Ermine are ferocious hunters that specialize in small mammals (USEPA, 1993; Loso, 1999), preferably of rabbit size and smaller, and play an important role in the small mammal communities in which they live (Loso, 1999). When mammalian prey is scarce, ermine may eat birds, eggs, frogs, fish and insects. On average, an ermine will consume approximately 0.04 kg of wet-weight food per day and will consume 0.01 L of water or its equivalent per day (Appendix B).

The Arctic hare (*Lepus arcticus*) weighs approximately 4.31 kg and inhabits the tundra regions of Canada from Newfoundland to the Mackenzie Delta and north to the tip of Ellesmere Island (Gorog, 2003). The Arctic hare resides in mountainous and lowland areas and requires broken country with areas of shelter. Snowshoe hares, which have similar diets to arctic hares, have home ranges from 3 ha to 7 ha (Shefferly, 1999). Immature arctic hares are hunted by arctic foxes, gyrfalcons, snowy owls and ermine; however, as adults they have few enemies save for wolves and humans (Canadian Museum of Nature, 2000; Gorog, 2003). They are primarily herbivorous and consume willow twigs and roots, bark, shoots, leaves, grasses, herbs and berries, but have been observed eating meat from hunters' traps (Gorog, 2003). An adult arctic hare will consume approximately 0.68 kg of wet-weight food per day and 0.37 L of water or its equivalent per day (Appendix B). Native peoples consume the meat and use the hides of arctic hare for clothing and bandages (Gorog, 2003).

The arctic fox (*Alopex lagopus*) is a relatively small canid mammal, weighing approximately 4.95 kg (USEPA, 1993), that is widely distributed throughout the Arctic (CWS & CWF, 2005). Breeding dens are built in the surface soil and may be used for many generations. Arctic foxes have few enemies other than humans, although wolves will eat them if they are able to catch one (CWS & CWF, 2005). The Arctic fox is active year-round and is predominantly carnivorous, feeding mainly on lemmings throughout the year in the continental tundra region, and consuming birds, eggs, ground squirrels and berries during the summer. They cache food in the summer and will also eat meat cached by Inuit hunters, and scavenge from wolf kills (CWS & CWF, 2005). Those that inhabit coastal regions also hunt for small marine mammals, fish and carrion along shorelines. When lemmings are abundant, foraging home ranges are approximately 250 ha to 500 ha in size (CWS & CWF, 2005). These animals consume approximately 0.80 kg of wet-weight food per day and 0.42 L of water or its equivalent per day (Appendix B). The Arctic fox is highly valued for its fur and is an important source of income for Native peoples (CWS & CWF, 2005).

The Canadian variety of barren-ground caribou (*Rangifer tarandus groenlandicus*), which weighs approximately 117.50 kg, is the most common caribou in Canada and a mainstay of many Native and Inuit peoples (CWS & CWF, 2005). The typically migratory barren-ground caribou spends much or all of the year on the tundra, and migrates seasonally (hundreds of kilometers) from the tundra to the taiga, returning north in the springtime to their small calving grounds and summer range on the tundra. Near-stationary populations of barren-ground caribou have also been noted (CWS & CWF, 2005). Woodland caribou, which have similar diets and life cycles to barren-ground caribou, have home ranges of approximately 6,000 ha and 26,000 ha for calving grounds and early winter home ranges, respectively (Ferguson and Elkie, 2004). The caribou diet is dependant on seasonal availability, but lichens are the caribou's primary food source for much of the year (CWS & CWF, 2005). They will also feed on flowers, grasses and leaves of shrubs. On average, a barren-ground caribou will consume approximately 6.59 kg of wet-weight food per day and 7.22 L of water or its equivalent per day (Appendix B). The Dolphin and Union population of barren-ground caribou is designated as "Special

Concern” under COSEWIC and is being considered for addition to Schedule 1 under SARA (Government of Canada, 2005).

The rock ptarmigan (*Lagopus mutus*), weighing approximately 0.49 kg, is a member of the grouse family and lives in arctic and alpine habitats (CWS & CWF, 2005). In the summer, it resides at high elevations and latitudes selecting habitat that is dry and supportive of sparse, very low vegetation. While in the fall, it moves down slope or southward into dense shrubs and forested areas. Winter home ranges of white-tailed ptarmigan (*Lagopus leucurus*), which inhabit the Cascade and Rocky Mountains of North America, are approximately 24 ha to 390 ha in size (Giesen and Braun, 1992). Ptarmigan spend most of their lives on the ground and are mainly herbivorous, feeding on willow seeds, buds and twigs, and any other vegetation that might be available (CWS & CWF, 2005). Ptarmigan, especially chicks, will also feed on insects when they are accessible. Rock ptarmigan are estimated to consume approximately 0.19 kg of wet-weight food per day and 0.04 L of water or its equivalent per day (Appendix C). They are valued for their meat and hunted by local residents (CWS & CWF, 2005).

The snowy owl (*Nyctea scandiaca*), which weighs on average 2.05 kg, is one of the chief predators of the arctic (CWS & CWF, 2005). Snowy owls breed on the arctic tundra and may migrate southerly for the winter (as far as the northern United States), although many remain in the arctic year-round. Breeding territories range from approximately 100 ha to 200 ha (CWS & CWF, 2005). The diet of the snowy owl consists primarily of small mammals (e.g., lemmings, arctic hare, meadow vole, mice) and birds (e.g., ptarmigan, seabirds), and they will occasionally feed on fish opportunistically (Atkinson and Kirschbaum, 2002; CWS & CWF, 2005). On average, snowy owls will consume 0.29 kg of wet-weight food per day and 0.10 L of water or its equivalent per day (Appendix B).

Lemming, arctic hare and rock ptarmigan were included in the ERA to represent “highly exposed” herbivorous wildlife as they are expected to be in close contact with potentially contaminated surface soil and water year-round in a relatively limited area. Ermine, arctic fox and snowy owl were selected to represent higher trophic level species that might be more likely to be exposed to contaminants via prey. Finally, Caribou were selected for inclusion as they are representative of a large ungulate that is likely to be found on site

#### 5.2.4 Rare, Threatened or Endangered Species and Species of Special Concern

Four species at risk are found in the region of CAM-D (Table 20).

**Table 20: Species at Risk in the CAM-D Area**

Common Name	Scientific Name	Status under Species at Risk Act (SARA)
Peregrine Falcon, tundrius subspecies	<i>Falco peregrinus</i>	Species of Special Concern in Schedule 3
Eskimo curlew	<i>Numenius borealis</i>	Endangered Species in Schedule 1
Wolverine, western population	<i>Gulo gulo</i>	Species of Special Concern in Schedule 3, pending public consultation for addition to Schedule 1
Polar Bear	<i>Ursus maritimus</i>	Species of Special Concern in Schedule 3

The above four species were not selected as representative receptors because the snowy owl, ermine and arctic fox are exposed to contaminants via similar pathways but have smaller home ranges and consequently greater exposure to contaminants. Thus, if contaminant levels at CAM-D are below toxic thresholds for these species, it can be deduced that concentrations are also safe for the peregrine falcon, Eskimo curlew, wolverine and polar bear.

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## 5.3 Exposure Assessment

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### 5.3.1 Potential Exposure Pathways

In order for a substance to induce an adverse effect, it must gain access to an organism, or receptor. The route by which this occurs is referred to as an exposure pathway, and is dependent on the nature of both the chemical and receptor. A complete exposure pathway is one that meets all of the following criteria (USEPA, 1989):

- a source of CoPC must be present;
- release and transport mechanisms, and media must be available to move the CoPC from the source to the ecological receptor(s);
- the ecological receptor(s) must have an opportunity to contact the affected media; and
- a mechanism must exist for the CoPC to be taken up by the ecological receptor, such as ingestion, inhalation, or dermal contact.

The sources of CoPCs at CAM-D were surface soil and surface water. Subsurface soil was not considered a potential source for contaminant exposure of wildlife as there is no direct exposure pathway for this environmental media. Furthermore, transport of contaminants from subsurface soils to surface soil and water was expected to be negligible.

The mechanisms by which receptors, including mammals and birds, typically become exposed to environmental contaminants from surface soil and water include:

- incidental ingestion of soil (e.g., feeding or grooming);
- ingestion of plants and/or prey species that have accumulated chemicals from the soil;
- inhalation of volatile soil contaminants; and
- ingestion and dermal contact with surface water.

In an ambient air scenario, the inhalation pathway is typically negligible for wildlife receptors as the dilution potential for soil vapours released to outdoor air is very high. Furthermore, the CoPCs carried forward into this ERA have low or negligible vapour pressures. Therefore, inhalation is not considered a significant exposure pathway at CAM-D and will not be evaluated further. The assessment of dermal contact with soil is included with that of incidental ingestion of soil.

Choice of site-specific exposure pathways is dependent on the nature of the contaminants, their source environmental media and the characteristics of the VECs selected for consideration in the ERA. The following sections elaborate on exposure pathway selection for CAM-D.

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### 5.3.2 Conceptual Model

A conceptual model was developed for CAM-D and is presented below (Figure 5). The figure schematically depicts the interactions between VECs and CoPCs, via the exposure pathways identified. The relevant exposure pathways are denoted by arrows leading from the contaminant source media to each VEC. A pathway is considered to be complete (i.e., functioning) when the exposure pathway box is marked with an X. The model demonstrates how the pathways corresponding to soil and water ingestion, and ingestion of food items (i.e., terrestrial plants, small mammal prey items) have been conceptualized and implemented for each VEC.

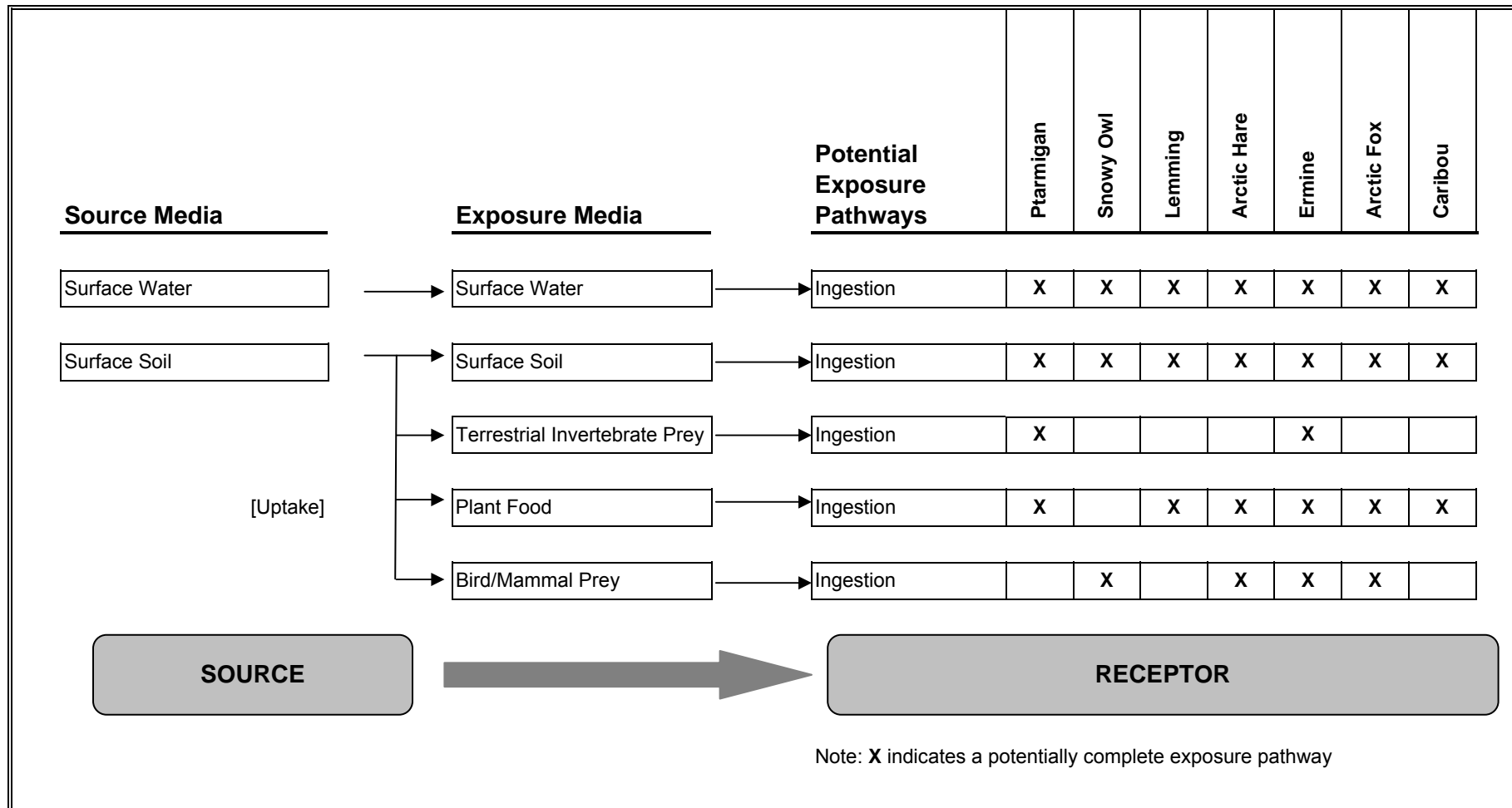


Figure 5: Conceptual Model for Ecological Risk Assessment at CAM-D

### 5.3.3 Selection of Assessment and Measurement Endpoints

Assessment endpoints are explicit expressions of environmental values or characteristics, and reflect societal and ecological values (Suter, 1993). Societal values address the need to protect species that are endangered, threatened, of special interest, important as game or commercial species, or widely recognized as having aesthetic value. Ecological relevance refers to the importance of the species to the function of the ecosystem. In environmental risk assessment, evaluation of the potential for adverse effects at the population level is used to infer potential for adverse effects at higher levels of organization, such as communities and ecosystems. For birds and mammals inhabiting CAM-D, assessment endpoints focus on maintenance and protection of their populations, such that contaminants in surface soil and water would not significantly impact species abundance and/or diversity through increased mortality or decreased reproduction.

The information needed to contend directly with an assessment endpoint is difficult to generate and rarely available, thus measurement endpoints are used to bridge the gap. Measurement endpoints are quantifiable responses to stressors related to assessment endpoints, and are intended to provide a basis for evaluating the potential risk for the assessment endpoint. They may be defined in terms of an unacceptable degree of impact to ecological receptors, such as a certain relative percent decrease in survival, growth or reproduction of ecological populations (Suter, 1993). As part of a weight-of-evidence approach, one or more measurement endpoints may be used for each assessment endpoint. Choice of measurement endpoints for each VEC-CoPC interaction is typically limited by the availability of toxicity data. Those most commonly used to quantify survival, growth or reproduction of receptors include the LC<sub>50</sub> and LD<sub>50</sub> (concentration or dose that will be lethal to 50% of exposed organisms, over a defined period of exposure), the EC<sub>50</sub> and ED<sub>50</sub> (concentration or dose that elicits a specific response or effect over a defined period of time), the lowest observable adverse effect level (LOAEL) and the no-observable adverse effect level (NOAEL). Although the dose-response relationships derived from these measurement endpoints are characteristic of test species exposed under controlled conditions, appropriate safety factors are incorporated in order to consider the response of species in the natural environment.

Measurement endpoints are used to resolve whether or not observed concentrations of chemicals in soil or water are likely to result in doses to birds or mammals that are greater than those known to increase mortality or decrease reproduction following chronic exposure.

Consequently, the key components of this ecological risk assessment are:

- characterization of relationships between the dose resulting from exposure to surface soils and the threshold dose for adverse effects; and
- characterization of relationships between the concentration of a chemical present in surface water and the threshold for adverse effects.

The dose-response relationships incorporated into this ERA are based upon LOAELs corresponding to the survival or reproduction of birds and mammals after chronic exposure to the CoPCs. These relationships are expressed in terms of the daily ingested dose, normalized to the body weight of the test organism (i.e., the reference toxicity dose expressed as mg substance ingested / kg body weight-day). Where such data were not available, LOAEL values were estimated from other endpoints including the NOAEL or the LD<sub>50</sub>. Standard conversion factors were implemented including division by 5 to convert an acute dose to a chronic dose, division by 6 to convert an LD<sub>50</sub> value to a LOAEL



value, or multiplication by 5 to convert a NOAEL to a LOAEL. These conversion factors are cumulative, thus an acute LD<sub>50</sub> value would be converted to a chronic LOAEL value through division by 30.

If appropriate data for a specific representative mammalian receptor was not available, a body-size scaling factor was used for extrapolation of available data between species (Sample and Arenal, 1999). The body-size scaling factor is calculated as:

$$\text{Mammal Body Weight SF} = (\text{BWt}/\text{BWr})^{0.06}$$

where:

SF = scaling factor

BWt = mean body weight of test species

BWr = mean body weight of receptor species

Similarly, if appropriate data for a specific representative avian receptor was not available, a body-size scaling factor was used for extrapolation of available data between species. The body-size scaling factor is calculated as:

$$\text{Bird Body Weight SF} = (\text{BWt}/\text{BWr})^{-0.20}$$

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## 5.4 Risk Characterization

Risk characterization is the final component of an ecological risk assessment. It includes quantification of the nature and magnitude of potential adverse effects that may occur to ecological receptors as a result of CoPCs at the site. In this stage, characterization of exposure and of ecological effects for each CoPC is integrated into a quantitative estimate (i.e., hazard quotients, HQs) of the potential for adverse effects to receptors.

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### 5.4.1 Approach

Ecological hazard quotients were calculated for all CoPCs in each individual area of CAM-D by dividing the exposure (i.e., EPC or total ingested dose) for each receptor by the appropriate reference toxicity dose (RTD) as follows:

$$\text{HQ} = (\text{Exposure}) / (\text{Reference Toxicity Dose})$$

For birds and mammals, the exposure value represents the total ingested dose (mg/kg-day) derived by summing all exposure pathways.

A HQ value of less than 1.0 indicates that the exposure concentration is less than the threshold for adverse effects, and a low probability exists that these effects might occur. Given the overall tendency to introduce conservatism into risk assessments through the use of data or assumptions that are likely to overstate rather than understate risk, it is expected that no adverse effect would occur. Alternatively, a HQ value of >1.0 does not necessarily indicate that there is an unacceptable level of risk. In these cases, the conservative approach reduces the likelihood of this conclusion and dictates the need for more careful review of both predicted exposure levels and exposure limit derivations. Therefore, HQ values greater than 1.0 should be scrutinized carefully, and a more focused investigation may be required to reduce conservatism and provide a more realistic evaluation of the actual level of risk. If it



is ultimately determined that the HQ value is indeed greater than 1.0, then site management and/or remedial activities may be appropriate in order to reduce the risks to ecological receptors.

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#### 5.4.2 Derivation of Media to Biota Uptake Factors

The concentrations of substances evaluated in this ERA were measured empirically in soil, water and plants from CAM-D. However, in order to complete the exposure assessment for the selected VECs, it is necessary to estimate the concentration of each CoPC in a variety of biological compartments. This task is accomplished generically using uptake factors (UF) that relate concentrations in various types of biota (e.g., invertebrates, small mammals) to concentrations in soil or water.

Substances carried forward into the ERA as CoPCs included ethylbenzene, toluene, xylenes, naphthalene, TPH fractions, total PCBs and a variety of metals. The approaches used to estimate uptake factors for each of these substances, for each biological food group, are described below. The specific uptake factors used in this assessment can be found in the ERA model printouts located in Appendix C.

##### Soil to Plant Uptake Factors

For organic substances, soil to plant uptake factors were generally calculated using the following equation (Travis and Arms, 1988):

$$\log(UF_{SP}) = (1.588 - 0.578 \log(K_{ow})) \times 0.19,$$

where  $UF_{SP}$  is the uptake factor for soil to plant (mg/kg dry plant / mg/kg dry soil),  $K_{ow}$  is the octanol-water partition coefficient for the organic substance under consideration and 0.19 is a conversion factor to adjust dry weight plant tissue concentrations to wet weight values.

For some inorganic substances (Cd, Cu, Pb, Zn) and total PCBs, data for plant tissue metal concentrations were collected from the site by the Environmental Sciences Group of the Royal Roads Military College (RMC, 1994). EPC values were calculated by deriving the 95% upper confidence limit of the geometric mean and multiplying this value by a conversion factor representing the average dry solids fraction (0.40) to obtain wet-weight tissue concentrations.

For other inorganic substances, a variety of approaches were used, although the equations of Efroymson et al. (2001) were preferred as they adjusted the soil to plant uptake factor according to the CoPC concentration in soil.

##### Soil to Animal Uptake Factors

For organic substances, soil to animal uptake factors were generally calculated using the following equation (Travis and Arms, 1988):

$$\log(Ba_{p,s,w}) = -7.6 + \log(K_{ow}),$$

where  $Ba_{p,s,w}$  is the transfer factor for soil to beef (day/kg), which is assumed to also be applicable to caribou meat. To estimate the CoPC concentration in meat, transfer factors are multiplied by the CoPC concentration in soil, feed and drinking water (mg/kg or mg/L), and by the ingestion rates of soil, feed and drinking water (kg/day or L/day). For TPH substances, which are not as readily absorbed but are

more efficiently metabolized than the pesticide compounds that form the basis of the Travis and Arms equation, a bioavailability factor (from 0 to 1) is also applied.

For concentrations of inorganic substances in meat, a similar approach was employed except that the transfer factors to meat ( $Ba_{p,s,w}$ ) were obtained from the compilation of Baes et al. (1984).

For small animal prey, including lemming, ptarmigan and arctic hare, a variety of approaches and data sources were used to attain uptake factors. For organic substances, the approach of Travis and Arms (1988) was followed. For inorganics, data (n=1) for lemming tissue concentrations were collected from the site by the Environmental Sciences Group of the Royal Roads Military College (RMC, 1994).

### Soil to Soil Invertebrate Uptake Factors

For soil to soil invertebrate uptake factors (UPSI), a conservative default uptake factor of 0.1 was assumed for ethylbenzene, toluene, xylenes, naphthalene and TPH fractions. For inorganic substances, the equations of Sample et al. (1998) were preferred.

### 5.4.3 VEC Home Ranges and Periods of Residency

In calculating VEC exposures to CoPCs at CAM-D it was necessary to make a number of assumptions regarding residency times at the site.

For exposure modeling, it was conservatively assumed that the collared lemming and rock ptarmigan were present at each individual area of CAM-D for 100% of their lifetime. Residency time for the barren ground caribou, arctic hare, arctic fox, ermine and snowy owl was divided between the main station and proximity, main dump, barrel dumps and background areas (Table 21). The barren ground caribou, arctic fox and snowy owl all have large home ranges as compared to the areas of the individual sites of CAM-D, and as such, spend most of their time in background areas. In calculating periods of residency using the actual home ranges of these VECs, time spent at CAM-D was less than 10%. However, it was conservatively assumed that each of these receptors would spend 10% of their time between the main station and proximity, main dump and barrel dumps, and 90% of their time in background areas.

Because of the small size of the outfall and Simpson Lake shoreline areas, the only VECs modelled at these sites were the collared lemming and rock ptarmigan. Furthermore, the airstrip area was not considered in the risk characterization section as contamination here was localized to one “hot spot”, and it was assumed that this spot would be removed as part of the planned remediation program.

**Table 21: Home Ranges and Residency Times of VECs for the ERA**

Location	Area of Site (hectare)	Caribou	Caribou Assumed	Arctic Hare	Arctic Fox	Arctic Fox Assumed	Ermine	Snowy Owl	Snowy Owl Assumed
Home Range	–	6000 ha	6000 ha	7 ha	250 ha	250 ha	10 ha	100 ha	100 ha
Main Station	4.54	0.08%	6.55 %	64.86%	1.82%	6.55%	45.40%	4.54%	6.55%
Main Dump	0.79	0.01%	1.15 %	11.38%	0.32%	1.15%	7.97%	0.80%	1.15%
Barrel Dump	1.59	0.03%	2.3 %	22.81%	0.64%	2.3%	15.97%	1.60%	2.3%
Percentage Time on Site	–	0.12%	10.00 %	99.04%	2.77%	10.00%	69.33%	6.93%	10.00%
Background	–	99.88 %	90.00 %	1.00 %	97.23 %	90.00 %	30.67 %	93.07%	90.00%

## 5.4.4 Reference Toxicity Doses

Reference toxicity doses (RTDs) for the terrestrial receptors are included in the risk assessment model results, and are presented in Appendix C. The RTD values are unique for each CoPC.

## 5.4.5 Risk Estimation

Tables showing the derivation of risk estimates for avian and mammalian receptors can be found in Appendix C. Tables 22 to 28 present the total HQ values for each VEC at each individual area of CAM-D. Risk estimates are summarized and analyzed for all VECs and areas in the text that follows.

### 5.4.5.1 Risk Estimates for Rock Ptarmigan

For rock ptarmigan, intake pathways included ingestion of surface soil and water, and ingestion of plant materials and soil invertebrates. The ptarmigan feeds primarily on vegetation such as leaves, flowers, seeds, berries, and buds and twigs of willow and birch, and will also consume insects.

Risks (HQ values) for the rock ptarmigan were less than 1 for all CoPCs in each area (Table 22). However, all individual areas of CAM-D had HQ values in the range of 0.1 to 1.0. These areas and CoPCs are as follows: main station and proximity (barium, lead, lithium, zinc, TPH fractions F2, F3 and F4), main dump (zinc), barrel dumps (TPH fractions F3 and F4), outfall area (zinc) and Simpson Lake shoreline (lead, zinc). All other substances evaluated had HQ values below 0.1 in each area. For organic substances at the main station and proximity, elevated hazard quotients were dominated by the surface soil ingestion pathway and to a lesser extent by the terrestrial plant and soil invertebrate ingestion pathways. For inorganic substances at the main dump, outfall area and Simpson Lake shoreline, elevated HQs were governed by the soil, plant and invertebrate ingestion pathways. Finally, for organic substances at the barrel dumps, elevated HQs were dominated by the soil and soil invertebrate ingestion pathways and to a lesser extent by the plant ingestion pathway. For all areas and CoPCs, risks due to ingestion of surface water are anticipated to be negligible.

**Table 22: Hazard Quotients for Rock Ptarmigan**

	Main Station Area HQ	Main Dump HQ	Barrel Dumps HQ	Outfall Area HQ	Simpson Lake HQ
<b>Organics</b>					
Ethylbenzene	8.92E-05	na	2.79E-06	na	na
Toluene	8.39E-05	na	3.56E-06	na	na
Xylenes	7.51E-05	na	2.68E-07	na	na
F1 - Total	7.74E-02	na	1.19E-03	na	na
F2 - Total	9.85E-01	na	4.17E-02	na	na
F3 - Total	9.77E-01	na	2.29E-01	na	na
F4 - Total	9.27E-01	na	2.67E-01	na	na
Naphthalene	3.25E-02	na	1.97E-04	na	na
Total PCBs	4.78E-03	1.83E-03	5.47E-04	3.43E-02	3.67E-03
<b>Inorganics</b>					
Arsenic	9.04E-03	3.26E-03	2.22E-03	2.13E-02	2.19E-03
Barium	1.63E-01	1.65E-02	1.69E-02	1.46E-02	5.06E-03
Cadmium	1.57E-02	6.62E-03	6.48E-03	8.44E-03	5.72E-03
Copper	4.85E-02	7.79E-03	5.35E-03	2.14E-02	8.48E-03
Lead	1.77E-01	3.50E-02	1.51E-02	2.22E-02	2.55E-01

**Table 22: Hazard Quotients for Rock Ptarmigan**

	Main Station Area HQ	Main Dump HQ	Barrel Dumps HQ	Outfall Area HQ	Simpson Lake HQ
Lithium	2.17E-01	1.82E-04	1.82E-04	1.82E-04	1.82E-04
Manganese	1.36E-02	1.07E-06	1.07E-06	1.07E-06	1.07E-06
Nickel	4.82E-02	5.62E-03	5.27E-03	7.63E-03	5.26E-03
Silver	9.65E-03	1.09E-03	5.44E-04	1.09E-03	1.09E-03
Uranium	7.05E-06	7.05E-06	7.05E-06	7.05E-06	7.05E-06
Zinc	9.27E-01	1.01E-01	7.10E-02	2.10E-01	2.48E-01

HQ values in the range of 0.1 to 1.0

#### 5.4.5.2 Risk Estimates for Snowy Owl

For snowy owl, intake pathways included ingestion of surface soil and water, and ingestion of terrestrial mammalian prey. The Snowy Owl feeds primarily on small mammals.

Risks (total HQ values) for the snowy owl were less than 0.1 for all CoPCs (Table 23). Thus, exposure of snowy owl to CoPCs at CAM-D is not expected to result in adverse health effects.

**Table 23: Total Hazard Quotients for Snowy Owl**

	Total Hazard Quotients	Main Station Area HQ	Main Dump HQ	Barrel Dumps HQ	Background HQ
<b>Organics</b>					
Ethylbenzene	2.24E-07	2.22E-07	na	2.43E-09	na
Toluene	2.04E-07	2.01E-07	na	3.00E-09	na
Xylenes	1.88E-07	1.88E-07	na	2.35E-10	na
F1 - Total	1.53E-04	1.52E-04	na	8.21E-07	na
F2 - Total	1.70E-02	1.67E-02	na	2.49E-04	na
F3 - Total	2.97E-02	2.74E-02	na	2.26E-03	na
F4 - Total	2.45E-02	2.23E-02	na	2.25E-03	na
Naphthalene	4.63E-06	4.62E-06	na	9.85E-09	na
Total PCBs	9.34E-05	8.26E-05	6.38E-06	4.41E-06	na
<b>Inorganics</b>					
Arsenic	2.45E-05	2.00E-05	1.92E-06	2.52E-06	1.58E-04
Barium	1.70E-04	1.39E-04	1.00E-05	2.04E-05	6.68E-04
Cadmium	1.08E-04	7.45E-05	1.12E-05	2.22E-05	2.86E-04
Copper	4.55E-04	3.06E-04	5.37E-05	9.55E-05	3.97E-03
Lead	9.59E-04	6.88E-04	1.21E-04	1.50E-04	5.57E-03
Lithium	1.28E-03	1.27E-03	2.06E-06	4.12E-06	na
Manganese	4.95E-08	3.24E-08	5.70E-09	1.14E-08	na
Nickel	7.95E-05	5.35E-05	1.11E-05	1.48E-05	8.95E-04
Silver	1.24E-06	8.32E-07	1.99E-07	2.09E-07	1.12E-05
Uranium	3.35E-07	2.20E-07	3.86E-08	7.71E-08	na
Zinc	4.00E-03	2.68E-03	4.61E-04	8.61E-04	3.34E-02

### 5.4.5.3 Risk Estimates for Collared Lemming

Intake pathways for the collared lemming included ingestion of surface soil and water, and ingestion of plant materials. The lemming feeds on vegetation such grasses and shrubs, and bark and twigs of willow and birch.

Risks (HQ values) for the collared lemming were less than 1 for all CoPCs in each area (Table 24). However, HQ values in the range of 0.1 to 1.0 were identified at the main station and proximity. For organic substances at the main station area, elevated hazard quotients were dominated by the surface soil and terrestrial plant ingestion pathways. For inorganic substances, elevated HQs were dominated by the plant ingestion pathway and to a lesser extent by the surface soil ingestion pathway. For all CoPCs, risks due to ingestion of surface water are anticipated to be negligible.

**Table 24: Hazard Quotients for Collared Lemming**

	Main Station Area HQ	Main Dump HQ	Barrel Dumps HQ	Outfall Area HQ	Simpson Lake HQ
<b>Organics</b>					
Ethylbenzene	9.17E-06	na	2.87E-07	na	na
Toluene	1.65E-05	na	6.99E-07	na	na
Xylenes	1.46E-05	na	5.19E-08	na	na
F1 - Total	2.26E-02	na	3.48E-04	na	na
F2 - Total	2.90E-01	na	1.23E-02	na	na
F3 - Total	1.86E-01	na	4.36E-02	na	na
F4 - Total	1.19E-02	na	3.42E-03	na	na
Naphthalene	9.56E-03	na	5.80E-05	na	na
Total PCBs	3.52E-03	1.55E-03	5.35E-04	1.80E-02	2.82E-03
<b>Inorganics</b>					
Arsenic	4.77E-02	1.29E-02	9.00E-03	7.83E-02	8.87E-03
Barium	4.64E-01	2.89E-02	2.95E-02	2.54E-02	9.01E-03
Cadmium	1.18E-02	1.84E-03	1.81E-03	2.25E-03	1.64E-03
Copper	1.71E-01	1.62E-02	1.11E-02	4.35E-02	1.77E-02
Lead	2.10E-02	1.89E-03	8.44E-04	1.22E-03	1.34E-02
Lithium	3.48E-02	6.20E-05	6.20E-05	6.20E-05	6.20E-05
Manganese	2.57E-01	4.27E-05	4.27E-05	4.27E-05	4.27E-05
Nickel	3.80E-02	1.02E-03	5.01E-04	2.25E-03	5.08E-04
Silver	1.40E-02	7.07E-04	3.56E-04	7.07E-04	7.07E-04
Uranium	2.81E-04	2.81E-04	2.81E-04	2.81E-04	2.81E-04
Zinc	2.02E-01	8.09E-03	5.00E-03	2.02E-02	2.47E-02

HQ values in the range of 0.1 to 1.0

#### 5.4.5.4 Risk Estimates for Arctic Hare

For arctic hare, intake pathways included ingestion of surface soil and water, plants and small mammals. The arctic hare is primarily herbivorous but will also feed on carrion.

Risks (total HQ values) for the arctic hare were less than 1 for all CoPCs (Table 25). However, total HQ values were identified in the range of 0.1 to 1.0 for a number of organic and inorganic substances (i.e., TPH fractions F2 and F3, barium, manganese, zinc). High total HQ values were the direct result of elevated main station area HQs. For organic substances at the main station area, elevated hazard quotients were dominated by the surface soil and terrestrial plant ingestion pathways and to a lesser extent by the terrestrial mammal ingestion pathway. For inorganic substances, elevated HQs were dominated by the plant ingestion pathway, while the ingestion of surface soil and terrestrial mammal pathways were negligible. For all CoPCs, risks due to ingestion of surface water are anticipated to be negligible.

**Table 25: Total Hazard Quotients for Arctic Hare**

	Total Hazard Quotient	Main Station Area HQ	Main Dump HQ	Barrel Dumps HQ	Background HQ
<b>Organics</b>					
Ethylbenzene	4.93E-06	4.87E-06	na	5.36E-08	na
Toluene	8.88E-06	8.75E-06	na	1.31E-07	na
Xylenes	7.76E-06	7.75E-06	na	9.70E-09	na
F1 - Total	1.20E-02	1.20E-02	na	6.49E-05	na
F2 - Total	1.65E-01	1.62E-01	na	2.41E-03	na
F3 - Total	1.18E-01	1.09E-01	na	9.00E-03	na
F4 - Total	8.57E-03	7.78E-03	na	7.88E-04	na
Naphthalene	5.00E-03	4.99E-03	na	1.06E-05	na
Total PCBs	2.32E-03	2.05E-03	1.58E-04	1.09E-04	na
<b>Inorganics</b>					
Arsenic	2.81E-02	2.52E-02	1.21E-03	1.69E-03	1.12E-04
Barium	2.52E-01	2.43E-01	2.72E-03	5.57E-03	2.06E-04
Cadmium	6.83E-03	6.28E-03	1.87E-04	3.68E-04	4.34E-06
Copper	9.46E-02	9.04E-02	1.74E-03	2.48E-03	1.30E-04
Lead	1.14E-02	1.11E-02	1.95E-04	1.81E-04	7.27E-06
Lithium	1.84E-02	1.84E-02	6.11E-06	1.23E-05	na
Manganese	1.34E-01	1.34E-01	4.07E-06	8.15E-06	na
Nickel	2.01E-02	1.99E-02	1.07E-04	1.09E-04	9.83E-06
Silver	7.41E-03	7.28E-03	6.54E-05	6.61E-05	4.28E-06
Uranium	2.33E-04	1.53E-04	2.68E-05	5.37E-05	na
Zinc	1.08E-01	1.06E-01	8.75E-04	1.17E-03	4.86E-05

HQ values in the range of 0.1 to 1.0

### 5.4.5.5 Risk Estimates for Ermine

For ermine, intake pathways included ingestion of surface soil and water, and ingestion of plants, small mammals and invertebrate. The ermine feeds primarily on small mammals, but also consumes minor amounts of invertebrates and plant materials.

Risks (total HQ values) for the ermine were less than 1 for all CoPCs (Table 26). However, total HQ values were identified in the range of 0.1 to 1.0 for TPH fractions F2 and F3 as a direct result of elevated main station area HQs. For TPH fractions F2 and F3 at the main station area, elevated hazard quotients were dominated by the surface soil and terrestrial mammal ingestion pathways and to a lesser extent by the terrestrial invertebrate ingestion pathway. Risks due to ingestion of plant materials and surface water are anticipated to be negligible.

**Table 26: Total Hazard Quotients for Ermine**

	Total Hazard Quotients	Main Station Area HQ	Main Dump HQ	Barrel Dumps HQ	Background HQ
<b>Organics</b>					
Ethylbenzene	2.10E-06	2.09E-06	na	1.07E-08	na
Toluene	3.64E-06	3.62E-06	na	2.50E-08	na
Xylenes	3.37E-06	3.37E-06	na	1.96E-09	na
F1 - Total	4.58E-03	4.57E-03	na	1.32E-05	na
F2 - Total	2.84E-01	2.81E-01	na	3.58E-03	na
F3 - Total	3.31E-01	3.09E-01	na	2.22E-02	na
F4 - Total	4.67E-02	4.26E-02	na	4.01E-03	na
Naphthalene	8.18E-05	8.17E-05	na	1.01E-07	na
Total PCBs	6.43E-03	5.73E-03	4.14E-04	2.83E-04	na
<b>Inorganics</b>					
Arsenic	9.51E-03	8.48E-03	4.42E-04	5.87E-04	1.78E-03
Barium	2.86E-02	2.58E-02	9.15E-04	1.87E-03	3.00E-03
Cadmium	5.18E-03	3.62E-03	5.24E-04	1.03E-03	6.18E-04
Copper	5.63E-02	3.83E-02	6.48E-03	1.16E-02	2.36E-02
Lead	4.46E-03	3.30E-03	5.15E-04	6.42E-04	1.17E-03
Lithium	6.48E-03	6.45E-03	1.02E-05	2.05E-05	na
Manganese	2.84E-05	1.86E-05	3.26E-06	6.53E-06	na
Nickel	2.59E-03	1.78E-03	3.30E-04	4.77E-04	1.30E-03
Silver	1.31E-04	9.85E-05	1.58E-05	1.63E-05	4.44E-05
Uranium	1.91E-04	1.25E-04	2.19E-05	4.40E-05	na
Zinc	3.18E-02	2.15E-02	3.59E-03	6.69E-03	1.27E-02

HQ values in the range of 0.1 to 1.0



#### 5.4.5.6 Risk Estimates for Arctic Fox

For arctic fox, intake pathways included ingestion of surface soil and water, plants and small mammals. The arctic fox feeds primarily on small mammals, but also consumes minor amounts of plant materials.

Risks (total HQ values) for the arctic fox were less than 0.1 for all CoPCs (Table 27). Thus, exposure of arctic fox to CoPCs at CAM-D is not expected to result in adverse health effects.

**Table 27: Total Hazard Quotients for Arctic Fox**

	Total Hazard Quotient	Main Station Area HQ	Main Dump HQ	Barrel Dumps HQ	Background HQ
<b>Organics</b>					
Ethylbenzene	9.60E-08	9.50E-08	na	1.04E-09	na
Toluene	1.67E-07	1.64E-07	na	2.45E-09	na
Xylenes	1.53E-07	1.53E-07	na	1.91E-10	na
F1 - Total	2.06E-04	2.05E-04	na	1.11E-06	na
F2 - Total	1.91E-02	1.88E-02	na	2.80E-04	na
F3 - Total	2.27E-02	2.10E-02	na	1.73E-03	na
F4 - Total	3.27E-03	2.97E-03	na	3.01E-04	na
Naphthalene	1.70E-05	1.70E-05	na	3.62E-08	na
Total PCBs	4.60E-04	4.06E-04	3.14E-05	2.17E-05	na
<b>Inorganics</b>					
Arsenic	5.16E-04	4.28E-04	3.80E-05	5.03E-05	3.09E-03
Barium	1.97E-03	1.72E-03	8.36E-05	1.71E-04	5.51E-03
Cadmium	3.61E-04	2.53E-04	3.61E-05	7.13E-05	9.18E-04
Copper	4.47E-03	3.08E-03	5.01E-04	8.91E-04	3.71E-02
Lead	3.44E-04	2.54E-04	3.99E-05	4.95E-05	1.83E-03
Lithium	5.53E-04	5.50E-04	1.02E-06	2.04E-06	na
Manganese	3.72E-04	3.71E-04	4.09E-07	8.18E-07	na
Nickel	2.23E-04	1.68E-04	2.37E-05	3.17E-05	1.91E-03
Silver	2.49E-05	2.31E-05	9.00E-07	9.53E-07	4.97E-05
Uranium	2.37E-05	1.55E-05	2.73E-06	5.46E-06	na
Zinc	2.62E-03	1.85E-03	2.69E-04	5.03E-04	1.95E-02

#### 5.4.5.7 Risk Estimates for Caribou

Intake pathways for caribou included ingestion of surface soil and water, and plant materials. Caribou feed on vegetation such as low-growing shrubs and lichens.

Risks (total HQ values) for caribou were less than 0.1 for all CoPCs (Table 28). Thus, exposure of caribou to CoPCs at CAM-D is not expected to result in adverse health effects.

**Table 28: Total Hazard Quotients for Caribou**

	Total Hazard Quotients	Main Station Area HQ	Main Dump HQ	Barrel Dumps HQ	Background HQ
<b>Organics</b>					
Ethylbenzene	2.69E-07	2.66E-07	na	2.92E-09	na
Toluene	4.83E-07	4.75E-07	na	7.08E-09	na
Xylenes	4.25E-07	4.24E-07	na	5.31E-10	na
F1 - Total	6.34E-04	6.31E-04	na	3.41E-06	na
F2 - Total	8.76E-03	8.63E-03	na	1.28E-04	na
F3 - Total	6.60E-03	6.10E-03	na	5.02E-04	na
F4 - Total	4.52E-04	4.11E-04	na	4.16E-05	na
Naphthalene	2.32E-04	2.32E-04	na	4.94E-07	na
Total PCBs	1.10E-04	9.72E-05	7.51E-06	5.19E-06	na
<b>Inorganics</b>					
Arsenic	1.48E-03	1.31E-03	6.98E-05	9.60E-05	5.71E-03
Barium	1.22E-02	1.17E-02	1.60E-04	3.27E-04	1.07E-02
Cadmium	3.14E-04	2.89E-04	8.42E-06	1.65E-05	1.63E-04
Copper	4.34E-03	4.16E-03	7.82E-05	1.03E-04	5.01E-03
Lead	5.39E-04	5.20E-04	1.06E-05	9.18E-06	3.24E-04
Lithium	8.40E-04	8.39E-04	5.22E-07	1.04E-06	na
Manganese	6.20E-03	6.20E-03	3.60E-07	7.20E-07	na
Nickel	9.33E-04	9.22E-04	5.84E-06	5.66E-06	4.78E-04
Silver	3.44E-04	3.38E-04	3.34E-06	3.39E-06	1.93E-04
Uranium	2.06E-05	1.35E-05	2.37E-06	4.74E-06	na
Zinc	4.98E-03	4.90E-03	3.76E-05	4.56E-05	1.66E-03

## 5.5 Ecological Site-Specific Target Levels

At each individual area of CAM-D, risks (total HQ values) for all VECs were less than 1 for each CoPCs (Tables 22 to 28). However, a number of VECs had HQ values in the range of 0.1 to 1.0, and as a result site-specific target levels (SSTLs) were calculated. SSTLs are risk-based values for CoPCs that are protective of human and/or ecological health for specific exposures and exposure pathways which are developed for a particular site. For this ERA, SSTLs were derived for surface soils at the main station and proximity. This area was selected as it represents the most conservative of those at CAM-D for calculation of SSTLs due to its large size which allows species to spend their entire life cycles within it. It was only necessary to calculate SSTLs for the collared lemming and rock ptarmigan since these species were the most sensitive of those evaluated (i.e., lemming is a small mammal in constant contact with surface soil while the ptarmigan is exposed to CoPCs via numerous pathways). Furthermore, because of the size of their home ranges, collared lemming and rock ptarmigan could potentially be exposed to CoPCs throughout their lifetimes.

SSTLs were calculated for surface soils at the main station area by setting the total HQ to 1.0, and determining the corresponding surface soil EPC by means of a backward calculation. The SSTLs, once derived, were applied to all individual areas of CAM-D. The maximum concentrations of CoPCs in

surface soil at all areas of CAM-D, corresponding CCME guidelines and SSTLs for the collared lemming and rock ptarmigan are presented in Table 29.

As demonstrated in Table 29, a number of maximum soil concentrations exceeded surface soil SSTLs. For instance, maximum concentrations of zinc in soils from the outfall area, airstrip and Simpson Lake shoreline exceeded the SSTL for rock ptarmigan. Here, the rock ptarmigan SSTL is much lower than that for the collared lemming as ptarmigan consume plant materials that contain high levels of zinc. In addition, maximum concentrations of TPH fractions in soils from the main station and proximity, barrel dumps and airstrip exceeded the SSTLs for both collared lemming and rock ptarmigan. Collectively, these results indicate that a number of “hot spots” exist at specific areas of CAM-D that will require clean-up in order to ensure protection of ecological receptors.

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## 5.6 Uncertainty Analysis

Uncertainties are inherent in every aspect of the ERA process. The most effective way to reduce uncertainty is to collect site-specific data and to be conservative when making assumptions. Application of site-specific information assists in reducing uncertainty by eliminating the need for generic uptake or transfer factors that are typically calculated in a conservative manner. For CAM-D, site-specific data has been collected for concentrations of CoPCs in surface soil and surface water.

Despite inclusion of a considerable amount of site-specific data, this ERA contains many assumptions, and incorporates simplifications and uncertainty with respect to characteristics of the receptors, exposure pathways and CoPC concentrations in the environment. In the sections that follow, some important aspects of uncertainty inherent in this risk assessment are qualitatively discussed.

### Data Limitations

The quality of a risk assessment often depends on the extent and quality of the supporting data. In addition to incorporating existing site data, many surface soil and water samples were collected for the purposes of this risk assessment. The time available for data collection precluded consideration of fluctuations in measured concentrations due to daily or seasonal influences. Because data were statistically summarized, including calculation of the 95% UCL as the EPC, the derived values represent conservative estimates of the true concentrations to which wildlife species would be exposed.

Key limitations in the ERA included insufficient background surface water samples. It is possible that some of the substances carried forward as CoPCs due to concentrations in surface water were not elevated as a result of human activities, but reflect natural background levels. Additionally, measurement of substances in wildlife meat would have provided more accurate estimates of soil to plant and soil to animal uptake factors. These factors allow for more precise evaluation of risks to ecological and human receptors on site.

**Table 29: Site-Specific Target Levels for Surface Soil at Main Station and Proximity of CAM-D**

CoPCs	Max. Soil Conc.	Max. Soil Conc.	Max. Soil Conc.	Max. Soil Conc.	Max. Soil Conc.	Max. Soil Conc.	CCME <sup>1,2</sup> Guideline	Surface Soil SSTL	Surface Soil SSTL
	Main Station	Outfall Area	Barrel Dump	Main Dump	Airstrip	Simpson Lake	Ecological Health	collared lemming	rock ptarmigan
<b>Inorganics</b>									
Arsenic	2.4	60	1.6	1.9	2.8	1	17	206	730
Barium	1,720	81	94	92	94	28	500	2,900	6,000
Cadmium	9.3	1.0	0.5	0.5	3.63	0.5	10	1,940	320
Copper	43	131	9.9	16.8	107	17.5	63	2,640	5,300
Lead	56	14.7	13.6	183	92.3	179	300	15,350	675
Nickel	16.1	53	9.5	14	15.9	5.4	50	15,100	6,075
Silver	0.1	0.1	0.05	0.1	0.1	0.05	20	143	93
Zinc	274	1,370	136	183	1,570	540	200	50,000	500
<b>Organics</b>									
Ethylbenzene	4.3	na	0.01	na	0.01	na	1.2	35,000	3,600
Toluene	4.8	na	0.02	na	0.02	na	1.4	18,500	3,640
Xylenes	39.4	na	0.01	na	0.01	na	1	193,000	37,500
TPH F1	1,300	na	19	na	0.5	na	260	133	29.6
TPH F2	16,700	na	3,340	na	830	na	900	4,498	1003
TPH F3	11,300	na	28,300	na	25,500	na	800	4,703	1049
TPH F4	13,300	na	7,590	na	4,560	na	5,600	1,192	266
Naphthalene	37	na	0.025	na	0.13	na	0.6	432	127
Total PCBs	0.6	1.5	0.28	0.081	0.05	.098	1.3	36	9.2

**Notes:**

All units are in mg/kg

na - not assessed

Maximum soil concentration exceeds an SSTL

1 - Canadian Council of Ministers of the Environment (CCME) Canadian Environmental Quality Guidelines: Soil Quality Guidelines - Residential/Parkland, Environmental Health (1991, Updated 1997, 1999 & 2003)

2 - Canadian Council of Ministers of the Environment "Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil" (May 2001) for fine-grained surface soils – Residential land use (eco soil contact)

## Selection of Chemicals of Potential Concern

Chemicals of potential concern were selected independently for each media evaluated. If a substance exceeded screening criteria for any one media, the analysis was completed to include all media (i.e., surface soil, water and exposed biota). For each media, there are knowledge gaps with respect to understanding the toxicology of the CoPCs, and the physical and chemical properties of these substances. The approach for selecting CoPCs involved comparison of maximum concentrations of each detected chemical to values that are believed to be protective of most North American species, in most ecosystems. Because empirical data do not exist for all CoPCs and media, it is possible that relevant test species, and sometimes even the same environmental media, have not been evaluated in the proper context for comparison.

## **Chemical Speciation**

For many inorganic and organic substances, chemical and toxicological fate, and food chain interactions depend to a large extent upon chemical speciation and the context in which they are introduced to biological specimens. As such, conservative assumptions regarding chemical form, bioavailability and absorption across the gut were applied in the risk assessment, and consequently the potential for toxicity is likely to be overestimated. For example, it was assumed that 100% of ingested soil and water CoPCs are absorbed across the gut and available to the organism. This may be reasonable for some CoPCs, but will highly overestimate the bioavailability of others.

## **Food Chain Interactions**

Very limited empirical data exists to allow quantification of the relationship between a chemical in an environmental medium and chemical transfer through the food chain. Numerous chemicals appear to be magnified through the food chain, including methyl mercury, and some PCBs, chlorinated pesticides (e.g., DDT) and PCDD/PCDF compounds. These chemicals all have the tendency to partition into fatty tissue rather than water (i.e., hydrophobic), and are also resistant to natural degradation processes by metabolic enzymes. TPH fractions and PAHs are also hydrophobic environmental contaminants, but they are only moderately absorbed following ingestion and are also metabolized and/or excreted by some invertebrates and most vertebrates. For these reasons, food chain magnification does not tend to occur with TPH or PAH substances. The extent of food chain magnification is another uncertainty that is generally treated in a conservative manner. Collection and analyses of wildlife tissues for chemical concentrations would have reduced uncertainties associated with food chain magnification; however, these activities were beyond the scope of the ecological field program.

## **Wildlife Exposure Factors**

Many of the factors incorporated into dose calculations for wildlife species possess site-specific components. Consequently, validating each factor requires consideration of its site-specific nature. In the absence of site-specific validation, exposure factors are incorporated based on validations performed elsewhere, under different circumstances and sometimes for other species. Parameters such as food and water ingestion rates, incidental soil ingestion rates, dietary composition, home range and time spent at the site were collected from the scientific literature based on other sites and circumstances.

It has been assumed that each receptor organism spends its entire life cycle at, or near, CAM-D. Based on this assumption, VECs are modeled as being exposed to the 95% UCL of the geometric mean concentration of each CoPC. Therefore, it is likely that the level of wildlife exposure to CoPCs has been substantially overestimated.

## **Habitat Survey and Selection of Valued Environmental Components**

For this risk assessment, much effort was invested into consideration of existing ecological habitats and the species that reside within them. Terrestrial habitats were evaluated to identify relevant species, and to support appropriate VEC selection. Thus, the VECs that were chosen for inclusion in this ERA are known to be present, or can reasonably be expected to be present, at CAM-D. VECs were also selected to be typical and representative of those likely to be found on site. Use of site-specific receptors decreases the overall uncertainty of the ERA as local species are considered rather than highly sensitive non-native species.

## Receptor-Specific Toxicity Data

For the majority of VECs selected for inclusion in this ERA, toxicity data was sparse or unavailable. Thus, the applied toxicity values were not necessarily specific to the VEC species of this ERA, or to a reproductive or population-level endpoint. As a result, there is uncertainty associated with extrapolation of toxicity data from a laboratory test species to a receptor species in the wild. However, the conversion factors that were used for purposes of extrapolation are scientifically based and were applied in a manner that is believed to be conservative.

In some cases, there was a lack of chemical toxicity data. Typically, when this was the case, an RTD value was obtained for a small mammal test species and was conservatively translated into an RTD value by incorporating a safety factor of 5.

## Measurement Endpoints from the Toxicity Data

The paucity of toxicity data for many of the identified CoPCs limited the measurement endpoints that were available. Where LOAEL values were not available, it was necessary to extrapolate from NOAEL values. The correction factors used in this process are relatively conservative and tend to underestimate the LOAEL value. As this approach is conservative, if observed chemical concentrations are lower than RTD values there is little potential for adverse effects at the population level. This method is more conservative than that suggested by Suter (1993), where a 20% effect level (e.g., 20% reduction in survival) was treated as a conservative approximation of the threshold for regulatory concern. Therefore, use of extrapolated reference toxicity doses may overestimate the potential for adverse effects to ecological receptors.

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### 5.6.1 Summary of Uncertainty Analysis

On account of thorough literature reviews and scientific investigations, and adherence to applicable risk assessment guidance documents, it is believed that the results of this ERA present a reasonable yet conservative evaluation of the risks to ecological receptors present at CAM-D. Where uncertainty or lack of knowledge were encountered in the development of risk estimates, reasonable yet conservative assumptions were made, or data selected, in order to ensure that risks were not underestimated.

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## 6.0 REFERENCES

- Atkinson R and K Kirschbaum. 2002. *Nyctea scandiaca*. Animal Diversity Web. Accessed October, 2005 at [http://animaldiversity.ummz.edu/site/accounts/information/Nyctea\\_scandiaca.html](http://animaldiversity.ummz.edu/site/accounts/information/Nyctea_scandiaca.html).
- ATSDR (Agency for Toxic Substances and Disease Registry). 2002. Draft Interaction Profile for: Arsenic, cadmium, chromium and lead. Division of Toxicology, ATSDR, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- Baes CF, RD Sharp, AL Sjoreen and RW Shor. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture. Oak Ridge National Laboratory Report ORNL-5786.
- Beyer WN, EE Connor and S Gerould. 1994. Estimates of soil ingestion by wildlife. *J Wildl Manage* 58:375-382.
- Canadian Museum of Nature. 2000. Natural History Notebooks. Accessed October, 2005 at <http://www.nature.ca/notebooks/english/mon2.htm>.
- CCME (Canadian Council of Ministers of the Environment). 1999, revised 2004. Canadian soil quality guidelines for the protection of environmental and human health. *In*: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.
- CCME (Canadian Council of Ministers of the Environment). 1996a. Guidance Manual for Developing Site-Specific Soil Quality Objectives for Contaminated Sites in Canada, Winnipeg.
- CCME (Canadian Council of Ministers of the Environment). 1996b. A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines, Winnipeg.
- CCME (Canadian Council of Ministers of the Environment). 1991. Interim Canadian environmental quality criteria for contaminated sites, Winnipeg.
- CCME (Canadian Council of Ministers of the Environment). 1996. A Framework for Ecological Risk Assessment: General Guidance. CCME Subcommittee on Environmental Quality Criteria for Contaminated Sites. March, 1996.
- CCME (Canadian Council of Ministers of the Environment). 1997. A Framework for Ecological Risk Assessment: Technical Appendices. CCME Subcommittee on Environmental Quality Criteria for Contaminated Sites. March, 1997.
- CCME (Canadian Council of Ministers of the Environment). 1999. Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment, Winnipeg. Originally published in 1999, updated in 2001.
- CCME (Canadian Council of Ministers of the Environment). 2001. Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil.



- CWS & CWF (Canadian Wildlife Service & Canadian Wildlife Federation). 2005. Hinterland Who's Who. Accessed October, 2005 at <http://www.hww.ca>.
- Efroymson RA, ME Will and GW Suter. 1997. Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Soil and Litter Invertebrates and Heterotrophic Process: 1997 Revision. Prepared by Oak Ridge National Laboratory for the U.S. Department of Energy, Office of Environmental Management. ES/ER/TM-126/R2.
- Efroymson RA, BE Sample and GW Suter. 2001. Uptake of inorganic chemicals from soil by plant leaves: Regressions of field data. *Environ Toxicol Chem* 20:2561-2571.
- Ferguson SH and PC Elkie. 2004. Seasonal movement patterns of woodland caribou (*Rangifer tarandus caribou*). *J Zool Lond* 262:125-134. Accessed October, 2005 at <http://www.umanitoba.ca/faculties/science/zoology/faculty/ferguson/Ferg%20&%20Elkie%20CARMOVE.pdf>.
- Gartner Lee Ltd. and Cantox Inc. 1998. Site Risk Assessment. Final Report prepared for Qikiqtaaluk Corporation. June, 1998.
- Giesen KM and CE Braun. 1992. Winter home range and habitat characteristics of White-tailed Ptarmigan in Colorado. *The Wilson Bulletin* 104(2):263-272. Accessed October, 2005 at <http://elibrary.unm.edu/sora/Wilson/v104n02/p0263-p0272.pdf>.
- Gorog A. 2003. *Lepus arcticus*. Animal Diversity Web. Accessed October, 2005 at [http://animaldiversity.ummz.edu/site/accounts/information/Lepus\\_arcticus.html](http://animaldiversity.ummz.edu/site/accounts/information/Lepus_arcticus.html).
- Government of Canada. 2005. Species at Risk Act Public Registry. Accessed October, 2005 at <http://www.sararegistry.gc.ca>.
- Health Canada. 1994. Canadian Environmental Protection Act. Human Health Risk Assessment for Priority Substances. Ottawa. Cat. No. En40-215/41E. pp 36.
- Health Canada. 1996. Canadian Soil Quality Guidelines for Contaminated Sites, Human Health Effects: Inorganic Lead. Final Report. March 1996.
- Health Canada. 2003. Federal Contaminated Site Risk Assessment in Canada Part I: Guidance on Human Health Screening Level Risk Assessment (SLRA), Version 1.1, October 2003.
- Health Canada. 2003a. Federal Contaminated Site Risk Assessment in Canada Part II: Health Canada Toxicological Reference Values (TRVs), Version 1.0, October 2003.
- INAC (Indian and Northern Affairs Canada). 2005. Abandoned Military Site Remediation Protocol. Appendix B, DEW Line Cleanup Soil Criteria.
- Loso H. 1999. *Mustela erminea*. Animal Diversity Web. Accessed October, 2005 at [http://animaldiversity.ummz.edu/site/accounts/information/Mustela\\_erminea.html](http://animaldiversity.ummz.edu/site/accounts/information/Mustela_erminea.html).

- Ontario Ministry of the Environment (OME). 1996a, revised 1997. Guidelines for use at Contaminated Sites in Ontario.
- Ontario Ministry of the Environment (OME). 1996b. Rationale for the Development and Application of Generic Soil, Groundwater and Sediment Criteria for use at Contaminated Sites in Ontario.
- Ontario Ministry of Environment and Energy (OMEE). 1993. Ontario Typical Range of Chemical Parameters in Soil, Vegetation, Moss Bags and Snow. Originally published in December 1993, updated in 1999.
- OME. 1997. Guideline for Use at Contaminated Sites in Ontario. Revised February 1997.
- Predavec M and CJ Krebs. 2000. Microhabitat utilization, home ranges, and movement patterns of the collared lemming (*Dicrostonyx groenlandicus*) in the central Canadian Arctic. Can J Zool 78:1885-1890. Accessed October, 2005 at [http://article.pubs.nrc-cnrc.gc.ca/ppv/RPViewDoc?\\_handler=HandleInitialGet&journal=cjz&volume=78&calyLang=eng&articleFile=z00-135.pdf](http://article.pubs.nrc-cnrc.gc.ca/ppv/RPViewDoc?_handler=HandleInitialGet&journal=cjz&volume=78&calyLang=eng&articleFile=z00-135.pdf).
- Richardson GM. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment. O'Connor Associates Environmental Inc., Ottawa, ON.
- RMC (Royal Roads Military College). 1994. Environmental Study of Abandoned DEW Line Sites: II. Six Intermediate Sites in the Eastern Arctic. Indian and Northern Affairs Canada; Environment Canada, Environmental Protection; Environmental Sciences Group, RMC.
- Sample BE, DM Opresko and GW Suter. 1996. Toxicological benchmarks for wildlife: 1996 revision. Prepared by Oak Ridge National Laboratory for U.S. Department of Energy, Office of Environmental Management. ES/ER/TM-86/R3. pp 43.
- Sample BE, JJ Beauchamp, RA Efroymsen and GW Suter. 1998. Development and validation of bioaccumulation models for small mammals. Prepared by Oak Ridge National Laboratory for the U.S. Department of Energy, Office of Environmental Management. ES/ER/TM-219. pp 40.
- Sample BE, JJ Beauchamp, RA Efroymsen, GW Suter and TL Ashwood. 1998b. Development and Validation of Bioaccumulation Models for Earthworms. Prepared by Oak Ridge National Laboratory for the U.S. Department of Energy, Office of Environmental Management. ES/ER/TM-220. pp 45.
- SENES Consultants Limited. 2003. Ecological Risk Evaluation for CAM-D Simpson Lake Former Military Site. Level I Custodial Input Section. Prepared for Department of Indian Affairs and Northern Development. November, 2003.
- SENES Consultants Limited. 2003. Human Health Screening Level Risk Assessment for CAM-D Simpson Lake Former Military Site. Prepared for Department of Indian Affairs and Northern Development. November, 2003.
- Shefferly N. 1999. *Lepus americanus*. Animal Diversity Web. Accessed October, 2005 at [http://animaldiversity.ummz.edu/site/accounts/information/Lepus\\_americanus.html](http://animaldiversity.ummz.edu/site/accounts/information/Lepus_americanus.html).

Suter GW. 1993. Ecological Risk Assessment. Lewis Publishers, Boca Raton, Florida.

Travis CC and AD Arms. 1988. Bioconcentration of organics in beef, milk and vegetation. Environmental Science and Technology 22:271-173.

United States Environmental Protection Agency (US EPA). 2002. Region 3: Mid-Atlantic Region Hazardous Site Cleanup Division Risk Based Concentration Tables. <http://www.epa.gov/reg3hwmd/risk/rbc1002.pdf> October 9, 2002.

United States Environmental Protection Agency (US EPA). 1993. Wildlife Exposure Factors Handbook. EPA/600/R-93/187.

United States Environmental Protection Agency (US EPA). 1989. Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A) Interim Final. EPA/540/1-89/002.

United States Environmental Protection Agency (US EPA). 1986. Guidelines for the health risk assessment of chemical mixtures. Fed. Reg. 51: 34014-34025.

# Appendix A

## **Toxicological Profiles**

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## GLOSSARY AND ACRONYMS

Absolute bioavailability	Absolute bioavailability is the fraction or percentage of an administered dose that reaches systemic circulation (blood) irrespective of via the gastrointestinal tract, skin or lungs
Ah	Aryl hydrocarbon
ATSDR	Agency for Toxic Substances and Disease Registry
Bioavailability	The degree to which a substance becomes available to the target tissue after administration or exposure.
CEPA	Canadian Environmental Protection Act
COPC	Contaminants of Potential Concern
ESOD	Erythrocyte Superoxide Dismutase
FAO	Food and Agriculture Organization. An organization of the United Nations.
IARC	International Agency for Research on Cancer. An organization of the WHO.
IOC	Intake of concern
IOM	Institute of Medicine
IPCS	International Programme on Chemical Safety
IRIS	Integrated Risk Information System. A database maintained by the US EPA.
LOAEL	Lowest-observed-effects-level. A term that describes the benchmark on a threshold dose-response curve at which the lowest dose results in observed adverse health effects. May be used in place of a NOAEL where a NOAEL cannot be determined.
MAC	Maximum Allowable Concentration
MADEP	Massachusetts Department of Environmental Protection
MOE	Ontario Ministry of the Environment
MRL	Minimal Risk Level. A term used by the ATSDR to describe an estimate of daily human exposure to a hazardous substance that is likely to be without an appreciable risk of adverse noncancer health effects over a specified route and duration of exposure.

## GLOSSARY AND ACRONYMS

NATO	North Atlantic Treaty Organization
NCEA	National Center for Environmental Assessment
NIOSH	National Institute for Occupational Safety and Health
NOAEL	No-observed-effects-level. A term that describes the benchmark on a threshold dose-response curve at which the highest dose does not result in adverse effects.
NRC	National Research Council
OEHHA	Office of Environmental Health Hazard Assessment
ORD	Office of Research and Development
PCB	Polychlorinated biphenyls
PCDD	Polychlorinated dibenzo-p-dioxins
PCDF	Polychlorinated dibenzofurans
PTWI	Provisional Tolerable Weekly Intake
RAF	Relative absorption factor
RDA	Recommended Dietary Allowance
REL	Reference Exposure Level is a NIOSH time-weighted average concentration for up to a 10-hour workday during a 40-hour work week.
Relative bioavailability	A comparative fraction which predicts bioavailability in one medium or form in relation to the medium for which the TRV was derived.
RfC	Reference Concentration. The RfC is an estimate of lifetime daily exposure to a non-carcinogen in air for the general human population that appears to be without appreciable risk of deleterious effects expressed in mg chemical/kg body weight-day.
RfD	Reference Dose. The RfD is an estimate of lifetime daily exposure to a non-carcinogen for the general human population that appears to be without appreciable risk of deleterious effects expressed in mg chemical/kg body weight-day.

## GLOSSARY AND ACRONYMS

SF	Slope factor. The SF is a plausible upper bound estimate of the probability of a response per unit intake of a chemical over a lifetime expressed as (mg chemical/kg body weight-day) <sup>-1</sup> and is used to express carcinogenic effects.
STSC	Superfund Health Risk Technical Support Center
TC	Tolerable Concentration. A term used by Health Canada to describe concentrations in air that a person may be continuously exposed to over a lifetime without adverse effects. The TC is used to derive the TDI.
TC <sub>05</sub>	Tumorigenic concentration that will induce a 5% increase in the incidence of tumors or deaths due to tumors following exposure to that chemical in air.
TD	Tumorigenic Dose. A term used to describe a dose that will induce an increase in the incidence of tumors or deaths due to tumours as calculated from a non-threshold dose-response curve.
TD <sub>05</sub>	Tumorigenic Dose that will induce a 5% increase in the incidence of tumors or deaths due to tumors.
TDI	Tolerable Daily Intake. A term used by Health Canada in place of RfD.
TEF	Toxic Equivalency Factor
TEQ	Toxic Equivalent
TRV	Toxicity Reference Value
UF	Uncertainty Factor. A factor that is applied to NOAELs or LOAELs to yield a RfC or RfD. For example, the UF can be used to account for intra-species and inter-species extrapolations.
UL	Tolerable upper intake level. A term used by the IOM to describe the highest daily nutrient intake that will not result in adverse health effects.
Unit Risk	Units risks estimate the upper bound probability of an individual developing cancer following exposure to a particular level (usually as 1 µg/L in water or 1 µg/m <sup>3</sup> ) of a potential carcinogen. For example, if the unit risk is 1.2 x 10 <sup>-6</sup> µg/L then it is expected that 1.2 excess tumours are expected to occur per 1,000,000 people exposed to 1 µg of that chemical in 1 L of drinking water.
US EPA	United States Environmental Protection Agency
WHO	World Health Organization

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## 1.0 INTRODUCTION

For the purpose of this assessment, toxicity reference values (TRVs) were obtained for each of the identified chemicals of potential concern (CoPC). Toxicological information was obtained, as necessary, from various sources including Health Canada, the US EPA Integrated Risk Information System (IRIS) database, and the Agency for Toxic Substances and Disease Registry (ATSDR).

TRVs are values used to describe maximum acceptable doses of chemicals that will not result in the development of adverse health effects. TRVs can be used to describe non-carcinogenic and carcinogenic effects and can express effects in different terms based on magnitude of the dose, length of exposure and route of exposure.

---

### 1.1 Non-Carcinogenic TRVs

Non-carcinogenic chemicals exhibit threshold effects following exposure. Threshold effects are defined by the observation of adverse effects at a given dose or concentration. Given these threshold effects, two measures of interest can describe the dose-response curve: the no-adverse-effects-level (NOAEL) and lowest-adverse-effects-level (LOAEL). The NOAEL is the benchmark at which the highest dose does not result in observed adverse effects. The LOAEL may be used when a NOAEL is not available and is the lowest dose at which adverse effects are observed.

The reference dose (RfD) is used for the assessment of non-carcinogenic endpoints. The RfD is the estimate of lifetime daily exposure to a non-carcinogenic substance for the general human population that appears to be without appreciable risk of deleterious effects. It is expressed as mg chemical/kg body weight/day (i.e., mg/kg-day). The RfD is derived from either the NOAEL or the LOAEL determined in a laboratory study. Uncertainty factors (UF) are applied to the NOAEL or LOAEL to account for interspecies variability and interspecies variability (i.e., sensitive sub-populations). Additionally, uncertainty factors are applied to extrapolate from subchronic exposure to chronic exposure or where there is a paucity of data available for a chemical (i.e., no data regarding effects on reproduction).

Other regulatory agencies have substituted the term RfD to be reflective of objectives and toxicological endpoints. Health Canada replaces the term RfD with tolerable daily intake (TDI), also expressed in mg/kg-day. Health Canada also uses a tolerable concentration (TC) to express concentrations in air that a person can be continuously exposed to over their lifetime without adverse effects. The Institute of Medicine (IOM) uses the tolerable upper intake level (UL) expressed as mg chemical/day to describe the highest daily nutrient intake that will not result in adverse health effects. The ATSDR uses a minimal risk level (MRL) similar to the IOM's UL that estimates daily human exposure to a substance that, over a specified duration, will not cause an appreciable risk of adverse effects.

The reference concentration (RfC) is also used as a non-carcinogenic endpoint specific to inhalation exposure. The RfC is typically reported as a concentration in air which can be converted to an RfD for inhaled dose expressed as mg/kg-day.



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## 1.2 Carcinogenic TRVs

Carcinogenic chemicals exhibit non-threshold effects following exposure. Non-threshold effects are defined by the observation of adverse effects regardless of concentration and length of exposure. Primarily, two TRVs are used to describe carcinogenic effects: the slope factor and unit risk.

A slope factor (SF) is used for assessment of carcinogenic effects of a chemical. The SF is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime, expressed as  $(\text{mg/kg body weight/day})^{-1}$ . It is used to estimate an upper bound probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen.

Unit risks are used to estimate an upper bound probability of an individual developing cancer as a result of exposure to a particular level (usually as  $1 \mu\text{g/L}$  in water, or  $1 \mu\text{g/m}^3$  in air) of a potential carcinogen. Unit risks are calculated by dividing the SF by body weight and multiplying that product by the inhalation or drinking rate as applicable.

Health Canada uses tumorigenic doses and concentrations for substances that are considered to have non-threshold or carcinogenic effects. The potency is expressed as a dose or concentration that will induce a 5% increase in the incidence of tumours or deaths due to tumours as calculated from a dose-response curve. The TRVs that defined the 5% increased are tumorigenic concentration 05 ( $\text{TC}_{05}$ ) primarily used as a benchmark for exposure to a certain chemical in air or tumorigenic dose 05 ( $\text{TD}_{05}$ ).

---

## 1.3 Bioavailability

The definition of bioavailability varies with the source and context in which the term is used. The simplest and broadest definition of bioavailability describes the extent or rate that a chemical enters a receptor or is made available at the target site (i.e., blood). The importance of bioavailability in risk assessment is illustrated by comparison TRVs as toxicity measures that are usually defined by laboratory studies. The fraction of a dose which is absorbed during an animal study may differ from the fraction that is available to a receptor in the environment due to several factors including weathering.

There are two specific types of bioavailability that are applicable to risk assessment: absolute and relative bioavailability. Absolute bioavailability is the fraction or percentage of an administered dose that reaches systemic circulation (blood) irrespective of via the gastrointestinal tract, skin or lungs. Relative bioavailability is the absolute bioavailability in one medium divided by the absolute bioavailability of the chemical under the conditions used to derive the TRV. Therefore, the relative bioavailability is a comparative fraction which predicts bioavailability in one medium or form in relation to the medium for which the TRV was derived. Relative bioavailability can be expressed as a relative absorption fraction (RAF).

In the following toxicity profiles, both absolute and relative bioavailabilities have been provided, where applicable, with the relative bioavailability selected for use in the assessment.

---

## 2.0 ALUMINUM

Aluminum (Al) is a trivalent cation found in its ionic form in most animal and plant tissues, and in natural waters. It is the third most prevalent element and the most abundant metal in the earth's crust (i.e., 8.1%). Dietary aluminum is ubiquitous, but in such small quantities that it is not a significant source of concern in persons with normal elimination capacity. The average person consumes between three and ten milligrams of aluminum a day. Urban water supplies may contain greater concentrations of aluminum as it is generally treated with the element before becoming part of the supply. Health Canada states that no consistent, convincing evidence exists that aluminum in drinking water causes adverse health effects in human receptors (HC, 1998). Furthermore, no acute pathogenic effects in the general population have been described after exposure to aluminum (WHO, 1997).

---

### 2.1 Assessment of Carcinogenicity

There is insufficient information to allow for classification of the cancer risk from human exposures to aluminum and its compounds (WHO, 1997). Occupational exposure within the aluminum production industry has been linked to elevated cancer rates in humans, however, causative agents are thought to be pitch volatiles (IARC, 1987).

---

### 2.2 Selection of Toxicity Values

Provisional or calculated toxicity reference values (TRVs) have been obtained from the Risk Assessment Information System (RAIS, 2005) (Table 1).

**Table 1: Selected Toxicity Reference Values for Aluminum**

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	1.0 mg/kg-day	RfD	RAIS, 2005
Inhalation	1.43E-03 mg/kg-day	RfD	RAIS, 2005
	5.00E-03 mg/m <sup>3</sup>	RfC	RAIS, 2005
Dermal	1.00E-01 mg/kg-day	RfD	RAIS, 2005

---

### 2.3 Bioavailability

The following section describes the bioavailability of aluminum.

---

#### 2.3.1 Oral Bioavailability

The relative oral absorption factor for aluminum has been conservatively assumed to be 1.0.

---

#### 2.3.2 Inhalation Bioavailability

The relative inhalation absorption factor for aluminum has been conservatively assumed to be 1.0.

---

### 2.3.3 Dermal Bioavailability

The United States Environmental Protection Agency (US EPA, 1995) recommends a relative dermal absorption factor of 0.001 for aluminum.

The bioavailability of aluminum is summarized in the following table:

Table 2: Selected Bioavailabilities for Aluminum		
Route of Exposure	Relative Bioavailability	Reference
Ingestion	1.0	Assumed
Inhalation	1.0	Assumed
Dermal	0.001	US EPA, 1995

---

## 2.4 References

Health Canada. 1998. Guidelines for Canadian Drinking Water Quality – Supporting Documents. “Aluminum”. November, 1998.

IARC (International Agency for Research on Cancer) (1987) Summaries & Evaluations – Aluminum. Supplement 7.

Risk Assessment Information System (2005) Chemical-specific toxicity values: Aluminum. Web page accessed November, 2005: [http://risk.lsd.ornl.gov/cgi-bin/tox/TOX\\_select?select=nrاد](http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nrاد).

United States Environmental Protection Agency (1995) Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment (Interim Guidance). Waste Management Division, Office of Health Assessment.

World Health Organization (WHO)/International Programme on Chemical Safety (1997) Environmental Health Criteria 194. Aluminium. pp 1-13.

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## 3.0 ARSENIC

Arsenic is a natural, ubiquitous element found in soils and minerals. Arsenic can occur in both organic and inorganic forms in the environment with substantially different toxicological effects. Inorganic arsenic is considered to be more toxic than arsenic in its organic form. The most common form of inorganic arsenic in air is arsenic trioxide ( $\text{As}_2\text{O}_3$ ), while a variety of arsenites (the trivalent form, As III) and arsenates (the pentavalent form, As V) occur in water, soil and food (ATSDR, 2000). Organic arsenic tends to be less extensively metabolized and more rapidly eliminated in both humans and laboratory animals. In addition, no conclusive evidence has been found on the carcinogenicity of organic arsenic (ATSDR, 2000; EHC, 1981; EHC, 2001). Most cases of human toxicity from arsenic have been associated with exposure to inorganic arsenic; therefore for the purposes of this assessment the total concentrations of arsenic are believed to be in the inorganic form.

---

### 3.1 Assessment of Carcinogenicity

Exposure to high levels of arsenic has been shown to cause both carcinogenic and non-carcinogenic effects in humans. Inorganic arsenic is a known human carcinogen (Environment Canada and Health Canada, 1993; US EPA, 1998; US EPA, 2002). Arsenic is listed as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC, 1987).

---

### 3.2 Susceptible Populations

No studies were located regarding unusual susceptibility of any human subpopulation to arsenic; however, since the degree of arsenic toxicity may be influenced by the rate and extent of methylation in the liver, it is likely that members of the population with lower than normal methylating capacity might be more susceptible (ATSDR, 2000).

---

### 3.3 Selection of Toxicity Values

The following section describes various studies conducted to establish arsenic toxicity values via ingestion, inhalation and dermal routes of exposure.

---

#### 3.3.1 Oral Non-Cancer Toxicity Reference Values

Chronic oral exposure to inorganic arsenic in humans has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyper pigmentation, gangrene of the extremities, vascular lesions, and liver or kidney damage (ATSDR, 2000).

The United States Environmental Protection Agency (US EPA, 1993) provides an oral RfD for non-carcinogenic effects from inorganic arsenic of  $3 \times 10^{-4}$  mg/kg-day. This value is based on the extensive data set of both non-cancerous and carcinogenic health effects of Taiwan residents that were exposed to inorganic arsenic (predominately as arsenate ( $\text{As}[\text{V}]$ ) in their drinking water. Tseng (1977) studied the prevalence blackfoot disease in 40,421 inhabitants of an area on the Southwest coast of Taiwan where well water with a high concentration of arsenic was used for over 60 years. The rates of blackfoot disease were recorded for three ranges of arsenic concentrations in well water. The low

range (<0.3 ppm arsenic) from the Tseng (1977) study was taken as a LOAEL of 0.17 mg/L (converted to 0.014 mg/kg-day) (Tseng *et al.*, 1968; US EPA, 1993).

In an earlier study (Tseng *et al.*, 1968), prevalence of hyper pigmentation, keratosis, skin cancer and blackfoot disease were observed. A control population of 7,500 individuals was also examined. In the control population, 4,978 persons used water with non-detectable levels of arsenic and 2,522 persons used water with 0.001 to 0.017 ppm of arsenic. Not a single case of keratosis, hyper pigmentation or skin cancer was observed in these populations. The US EPA (1993) adopted a NOAEL of 0.009 mg/L based on this study (converted to 0.0008 mg/kg-day).

The RfD was developed based on the NOAEL of 0.8 µg/kg-day of arsenic divided by an uncertainty factor of 3. The uncertainty factor of 3 was to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals; therefore, this RfD is appropriate for comparison to exposures averaged over an entire lifetime (US EPA, 2003). The US EPA (1993) weights the selected study as medium given the poor characterization of doses, the presence of other contaminants despite the large sample population.

The US EPA RfD of  $3 \times 10^{-4}$  mg/kg-day is used for non-carcinogenic effects from inorganic arsenic as Health Canada does not provide an RfD.

---

### 3.3.2 Oral Cancer Toxicity Reference Values

The US EPA (1998) provides an oral cancer SF of  $1.5 \text{ (mg/kg-day)}^{-1}$ . The slope factor was based on data provided by the US EPA (2002) from increased incidence of skin cancer in Taiwanese populations orally exposed to arsenic in drinking water (Tseng, 1977; Tseng *et al.*, 1968). These studies did not examine rates of internal cancers (i.e., bladder and lung cancer) and are thus considered to underestimate total carcinogenic risks from arsenic. Arsenic is being reassessed under the Integrated Risk Information System (IRIS) program (US EPA, 1998).

Based on the same data set Health Canada (2003) recommends an oral SF of  $2.8 \text{ (mg/kg-day)}^{-1}$  based on a TD<sub>05</sub>.

In this assessment, the Health Canada SF of  $2.8 \text{ (mg/kg-day)}^{-1}$  will be used.

---

### 3.3.3 Non-Cancer Inhalation Toxicity Reference Values

Chronic inhalation exposure to inorganic arsenic in humans is associated with irritation of the skin and mucous membranes (dermatitis, conjunctivitis, pharyngitis, and rhinitis) (ATSDR, 2000). Health Canada nor the US EPA have not established TRVs for inorganic arsenic (US EPA, 2002).

A non-cancer inhalation TRV has not been selected for this assessment due to the lack of sufficient data.

---

### 3.3.4 Cancer Inhalation Toxicity Reference Values

Health Canada (1996) made TD<sub>05</sub> estimates for inhalation carcinogenic risk for the Anaconda, Tacoma and Ronnskar (Sweden) cohorts of 7.83, 10.2, and 50.5 µg/m<sup>3</sup>, respectively. These equate to unit risks of 6.4 x10<sup>-3</sup> per µg/m<sup>3</sup>, of 4.9 x10<sup>-3</sup> per µg/m<sup>3</sup>, and of 0.99x10<sup>-3</sup> per µg/m<sup>3</sup> for the Anaconda, Tacoma and Ronnskar cohorts, respectively. Health Canada reviewed only one follow-up study for the Anaconda cohort. The Health Canada TD<sub>05</sub> is based on only the Anaconda smelter data as being the most conservative. Recently, Health Canada (2003) has recommended an inhalation SF of 28.0 (mg/kg-day)<sup>-1</sup> based on a TC<sub>05</sub> of 7.8 µg/m<sup>3</sup> for arsenic and its inorganic compounds (Health Canada, 1996).

The Health Canada (2003) inhalation SF of 28.0 (mg/kg-day)<sup>-1</sup> is used in this assessment.

---

## 3.4 Bioavailability

Distribution of arsenic within the body is affected by the route through which exposure occurs. Arsenic tends to be evenly distributed amongst tissues within the body (Environment Canada and Health Canada, 1993). The interaction of arsenic with various tissues is dependent on the chemical form of the arsenic. The primary pathway of elimination of inorganic arsenic is excretion within the urine (ATSDR, 2000).

---

### 3.4.1 Oral Bioavailability

For this assessment, the oral bioavailability factor for soil can be conservatively assumed to be 1.0, in accordance with guidance from Health Canada (2003).

---

### 3.4.2 Inhalation Bioavailability

For this assessment, the inhalation bioavailability factor for soil was conservatively assumed to be 1.0, in accordance with guidance from Health Canada (2003).

---

### 3.4.3 Dermal Bioavailability

Arsenic is not very bioavailable when it is on the skin in a soil-bound matrix. Wester *et al.* (1993) administered arsenic in water topically to rhesus monkeys in vivo over 24 hours. The amount recovered in urine was 6.4% at the low dose and 2.0% at the high dose. Using human skin in vitro, 1.9% was recovered after 24 hour administration (combined receptor fluid accumulation of 0.93% and skin concentration of 0.98%).

Dutkiewicz (1977) applied pentavalent inorganic arsenic dermally to rats. Wistar rats tails were submerged in solution for one hour. An absorption rate of 1.14 to 33.1 µg/cm<sup>2</sup>-hour was recorded for concentrations ranging from 0.01 to 0.2M.

Soil was also applied to human skin. The in vitro application to human skin yielded 0.43% receptor fluid accumulation and 0.33% skin concentration for a total absorption of 0.8%. The in vivo application of soil to rhesus monkeys yielded absorption estimates of 3.2% to 4.5%. Based on this study, the US

EPA (2001) recommends a dermal absorption fraction for soil of 3%. This value was used to assess the bioavailable fraction of arsenic that would be absorbed through exposure to the skin.

Health Canada (2003) recommends the application of a relative dermal absorption fraction (RAF) of 0.03 to the estimation of daily dose.

The dermal bioavailability factor of 0.03 as an adjustment from dermal absorption to oral used in this assessment is based on the recommendations of Health Canada (2003).

---

### 3.5 Conclusion

Table 3 summarizes the selected toxicity reference values and Table 4 summarizes the selected relative bioavailabilities.

**Table 3: Selected Toxicity Reference Values for Arsenic**

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	$3 \times 10^{-4}$ mg/kg-day	RfD	US EPA, 1993
Inhalation	NA	NA	NA
<b>Cancer Effects</b>			
Ingestion	$2.8 \text{ (mg/kg-day)}^{-1}$	SF	Health Canada, 2003
Inhalation	$28.0 \text{ (mg/kg-day)}^{-1}$	SF	Health Canada, 2003
<b>Notes:</b> NA: Not Applicable			

**Table 4: Selected Relative Bioavailabilities for Arsenic**

Route of Exposure	Relative Bioavailability	Reference
Ingestion	1.0	Assumed
Inhalation	1.0	Assumed
Dermal	0.03	Health Canada, 2003

---

### 3.6 References

ATSDR (Agency for Toxic Substances and Disease Registry), 2000. *Toxicological Profile for Arsenic*. September 2000.

Dutkiewicz T, 1977. Experimental studies on arsenic absorption routes in rats. *Environmental Health Perspectives* 19:173-177.

Environment Canada and Health Canada, 1993. Priority substances list assessment report, Arsenic and its compounds. Canadian Environmental Protection Act. Government of Canada, Ottawa, Ontario.

EHC (Environmental Health Criteria 18). 1981. Arsenic. IPCS International Programme on Chemical Safety. World Health Organization, Geneva.



EHC (Environmental Health Criteria 224). 2001. Arsenic and Arsenic Compounds. IPCS International Programme on Chemical Safety. World Health Organization, Geneva.

Health Canada. 1996. Health based Tolerable daily intakes/concentrations and tumorigenic doses/concentrations for priority substances. Minister of Supply and Services Canada, Ottawa.

Health Canada, 2003. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs). October 3, 2003.

IARC (International Agency for Research on Cancer), 1987. Summaries & Evaluations – Arsenic. Supplement 7.

Tseng WP. 1977. Effects and dose-response relationships of skin cancer and Blackfoot disease with arsenic. *Environmental Health Perspectives*. 19:109-119.

Tseng WP, Chu HM, How SW, Fong JM, Lin CS and Yeh S. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. *J. Natl. Cancer Inst.* 40: 453-463.

US EPA (United States Environmental Protection Agency), 1993. Integrated Risk Information System (IRIS) Database: Arsenic, inorganic (Oral RfD Assessment). Last revised 02/01/1993. Available on-line at: <http://www.epa.gov/iris/>.

US EPA (United States Environmental Protection Agency), 1998. Integrated Risk Information System (IRIS) Database: Arsenic, inorganic (Carcinogenicity Assessment). Last revised 04/10/1998. Available on-line at: <http://www.epa.gov/iris/>.

US EPA (United States Environmental Protection Agency), 2001. Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Office of Emergency and Remedial Response, EPA/540/R/99/005, Interim, Review Draft, September. United States Environmental Protection Agency.

US EPA (United States Environmental Protection Agency), 2002. *Implementation Guidance for the Arsenic Rule, Drinking Water Regulations for Arsenic and Clarification to Compliance and New Source Contaminants Monitoring*. August 2002. United States Environmental Protection Agency.

US EPA (United States Environmental Protection Agency), 2003. Integrated Risk Information System (IRIS) Glossary. Revised September 2003. Available <http://www.epa.gov/iris/gloss8.htm>.

Wester RC, Maibach HI, Sedik L, Melendres J, and Wade M. 1993. In vivo and in vitro percutaneous absorption and skin decontamination of arsenic from water and soil. *Fundamental and Applied Toxicology* 20: 336-340.

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## **4.0 BARIUM**

Barium is a silvery-white metal found in nature. Health effects of barium compounds depend on how well the compound dissolves in water. Compounds that do not dissolve well in water are not generally harmful. However, barium compounds that dissolve well in water may cause harmful effects in people. Ingestion of barium compounds that readily dissolve in water can lead to difficulties in breathing, increased blood pressure, changes in heart rhythm, stomach irritation, brain swelling, muscle weakness and damage to the liver, kidney, heart and spleen. (ATSDR, 1992).

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### **4.1 Assessment of Carcinogenicity**

The oral database of information suggests that barium is unlikely to be carcinogenic to humans, and the inhalation database is inadequate to assess carcinogenicity (US EPA, 1998). Barium would be classified as Group D - not classifiable as to human carcinogenicity, under the United States Environmental Protection Agency's (US EPA) 1986 Guidelines for Carcinogen Risk Assessment. Under the Proposed Guidelines for Carcinogenic Risk Assessment (US EPA, 1999), barium is considered not likely to be carcinogenic to humans following oral exposure and its carcinogenic potential cannot be determined for inhalation exposure.

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### **4.2 Susceptible Populations**

Gastrointestinal absorption data suggest that barium absorption may be higher in children than in adults (US EPA, 1998). No other studies were located regarding unusual susceptibility of any human sub-population to barium (ATSDR, 1992).

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### **4.3 Selection of Toxicity Values**

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#### **4.3.1 Non-Cancer Oral Toxicity Reference Values**

An oral reference dose (RfD) of 0.07 mg/kg/day was provided for barium by the U.S. EPA (1999) based on several different studies. The main endpoint of concern was increased kidney weights. The U.S. EPA (1998) reported an adjusted no observable adverse effects level (NOAEL) of 0.21 mg/kg-day, and applied an uncertainty factor of 3 to the NOAEL to derive the RfD.

Health Canada (2003) provides a tolerable daily intake (TDI) of 0.016 mg/kg-day, which was used in this assessment.

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#### **4.3.2 Cancer Oral Toxicity Reference Values**

The lack of suitable positive carcinogenic data precludes the derivation of an oral slope factor or unit risk for barium.

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### 4.3.3 Non-Cancer Inhalation Toxicity Reference Values

Inhalation toxicity values for barium have not been developed by the US EPA or Health Canada and therefore, due to insufficient data, a non-cancer inhalation TRV has not been selected for this assessment.

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### 4.3.4 Cancer Inhalation Toxicity Reference Values

The lack of suitable positive carcinogenic data precludes the derivation of an inhalation slope factor or unit risk for barium.

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## 4.4 Bioavailability

The following section describes the bioavailabilities of barium.

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### 4.4.1 Oral Bioavailability

Like other metals, barium is poorly absorbed from the gastrointestinal tract, with studies suggesting that absorption in humans is less than 5% (ATSDR, 1992). The range of reported oral absorption factors for all animal studies was 0.7 to 85% (US EPA, 1998). The oral bioavailability of barium was reported to be 0.07 by the Oak Ridge National Laboratory (ORNL, 2004). For the purpose of this assessment, the relative oral bioavailability from soil exposure was assumed to be 100% or 1.0.

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### 4.4.2 Inhalation Bioavailability

No data are available on inhalation absorption of barium in humans; however, animal studies provide evidence that barium compounds are absorbed from the respiratory tract (US EPA, 1998). The inhalation bioavailability factor used in this assessment was 1.0.

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### 4.4.3 Dermal Bioavailability

Health Canada (2003) also recommends a relative dermal absorption factor of 0.1 for barium. Therefore, a relative dermal bioavailability of 0.1 was adopted for this assessment.

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## 4.5 Conclusion

The following tables present the TRV and bioavailability summaries for barium.

**Table 5: Selected Toxicity Reference Values for Barium**

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	$1.60 \times 10^{-2}$ mg/kg-day	TDI	Health Canada, 2003
Inhalation	NA	NA	NA
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	NA	NA	NA

NA – Not Applicable

**Table 6: Selected Bioavailabilities for Barium**

Route of Exposure	Relative Bioavailability	Reference
Ingestion	1.0	Assumed
Inhalation	1.0	Assumed
Dermal	0.1	Health Canada, 2003

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## 4.6 References

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. Toxicological Profile for Barium. Available on-line at: <http://www.atsdr.cdc.gov/toxprofiles/>

Health Canada, 2003. Federal Contaminated Site Risk Assessment in Canada, Part I: Guidance on Human Health Screening Level Risk Assessment (SLRA). October 3, 2003.

ORNL (Oak Ridge National Laboratory). 2004. Risk Assessment Program. Risk Assessment Information System. Toxicity and Chemical-specific Factors Data Base Search. Available on-line at: <http://risk.lsd.ornl.gov/>

US EPA (Environmental Protection Agency). 1998. Toxicological Review of Barium and Compounds (CAS No. 7440-39-3). In support of Summary Information on the Integrated Risk Information System (IRIS). March 1998. Available on-line at: <http://www.epa.gov/iris/toxreviews/>

US EPA (Environmental Protection Agency). 1999. Integrated Risk Information System (IRIS) Database – Barium and compounds. Confirmed current as of December 2004. Available on-line at: <http://www.epa.gov/iris/>

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## **5.0 CADMIUM**

Cadmium is a naturally occurring element that is commonly found as a mineral combined with other elements. Cadmium has many uses in industry and consumer products, mainly in batteries, pigments, metal coatings, plastics, and some metal alloys (ATSDR, 1999).

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### **5.1 Assessment of Carcinogenicity**

Several occupational studies have reported an excess risk of lung cancer in humans from exposure to inhaled cadmium; however, the evidence is limited rather than conclusive due to confounding factors (US EPA, 1994; ATSDR, 1999). Animal studies have reported cancer resulting from inhalation exposure to several forms of cadmium, while animal ingestion studies have not demonstrated carcinogenicity (US EPA, 1994; ATSDR, 1999). The US EPA (1994) considers cadmium to be a probable human carcinogen and has classified it as Group B1. Health Canada (Environment Canada and Health Canada, 1994) has classified cadmium as a Group II carcinogen – probably carcinogenic to humans.

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### **5.2 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 (ATSDR, 1999). Dietary deficiencies which lead to depleted levels of calcium or iron in individuals may result in increased absorption of cadmium from the gastrointestinal tract (ATSDR, 1999). Infants and children may have increased uptake of cadmium via the gastrointestinal tract and higher concentrations of cadmium in the bone (ATSDR, 1999).

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### **5.3 Selection of Toxicity Values**

Chronic inhalation and oral exposure of humans to cadmium results in a build-up of cadmium in the kidneys that can cause kidney disease, including proteinuria, a decrease in glomerular filtration rate, and an increased frequency of kidney stone formation (ATSDR, 1999). The following section describes various studies conducted to establish cadmium toxicity values via ingestion, inhalation and dermal routes of exposure.

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#### **5.3.1 Non-Cancer Oral Toxicity Reference Values**

Health Canada (2003a) has adopted the value of 0.0008 mg/kg-day as a tolerable daily intake (TDI). The Health Canada TDI is based upon the Canadian Guidelines for Drinking Water Quality, Supporting Documentation (2003b). The Canadian drinking water maximum allowable concentration (MAC) of 0.005 mg/L was calculated based on the joint FAO/WHO expert committee's proposed upper limit provisional tolerable weekly intake (PTWI) of 0.5 mg for adults (WHO, 1992; Health Canada, 2003b). The PTWI was based on the estimation that a daily intake of 0.05 mg would lead to 0.1% of the population reaching the "critical" concentration of 0.2 mg/g of cadmium in the renal cortex after 50 years. The WHO (1992) derived a PTWI range of 0.4 to 0.5 mg for cadmium. The kidney is believed to be the target organ of cadmium, specifically affecting the renal cortex.

The US EPA (1994) has developed oral RfDs for cadmium in food and water also based on kidney effects. The RfD for food is  $1.0 \times 10^{-3}$  mg/kg-day and for water is  $5.0 \times 10^{-4}$  mg/kg-day (US EPA, 1994). Both RfDs are based on significant proteinuria in humans with an assumed 2.5% absorption of cadmium from food and 5% from water. 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 US EPA rates the information database and corresponding RfD with high confidence.

The Health Canada TDI of  $8.0 \times 10^{-4}$  mg/kg-day was selected to assess non-carcinogenic effects from oral cadmium exposure.

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### 5.3.2 Cancer Oral Toxicity Reference Values

The lack of suitable positive carcinogenic data precludes the derivation of an oral slope factor or unit risk for cadmium..

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### 5.3.3 Non-Cancer Inhalation Reference Toxicity Values

A non-cancer inhalation TRV has not been selected for this assessment due to the lack of sufficient data.

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### 5.3.4 Cancer Inhalation Reference Toxicity Values

The US EPA (1994) has developed an inhalation unit risk of  $1.8 (\text{mg}/\text{m}^3)^{-1}$  to be used only if the air concentration does not exceed  $6 \mu\text{g}/\text{m}^3$ . This unit risk is based on lung and upper respiratory tract cancers in cadmium production workers (Thun *et al.*, 1985) and was selected over another study that yielded a more conservative unit risk because it was based on human data which involved a large cohort and took into consideration the effects of arsenic and smoking.

Health Canada (2003a) has calculated an inhalation unit risk of  $9.8 (\text{mg}/\text{m}^3)^{-1}$  which is equivalent to an inhalation slope factor of  $4.29\text{E}+01 (\text{mg}/\text{kg}\cdot\text{day})^{-1}$ , which was based on a  $\text{TC}_{05}$  of  $5.1 \mu\text{g}/\text{m}^3$  (Health Canada, 1996). The estimated  $\text{TD}_{05}$  for cadmium chloride based on multistage model of lung tumour incidences observed in rats by Takenaka *et al.* (1983). The  $\text{TD}_{05}$  of  $2.9 \mu\text{g}$  of cadmium/ $\text{m}^3$  was amortized to be constant over the entire life of the rat, adjusted for longer than lifetime duration of the experiment and converted to an equivalent concentration for humans using standard breathing rates and body weights which yielded a  $\text{TC}_{05}$  of  $5.1 \mu\text{g}/\text{m}^3$  (Environment Canada and Health Canada, 1994). The Health Canada  $\text{TC}_{05}$  provides a more conservative unit risk estimate of the potency of inhaled cadmium.

Therefore, an inhalation slope factor of  $4.29\text{E}+01 (\text{mg}/\text{kg}\cdot\text{day})^{-1}$  was adopted for this assessment.

---

## 5.4 Bioavailability

Cadmium compounds have varying degrees of solubility ranging from very soluble to nearly insoluble. The solubility affects their absorption and toxicity. Exposure to cadmium and cadmium compounds may occur in both occupational and environmental settings, the latter primarily via the diet and drinking water (ATSDR, 1999).

---

### 5.4.1 Oral Bioavailability

Cadmium bound in a soil matrix is expected to be less bioavailable than cadmium in drinking water, as in the study from which the oral RfD was derived. Other studies have reported the oral absorption of cadmium to range from 0.027 (Newton *et al.*, 1984) to 0.06 (Rahola *et al.*, 1975).

The selected oral RfD for cadmium is based on kidney effects following water consumption. Water consumption was assumed to be 5% absorbed in humans (US EPA, 1994). Falling within the range of the above studies. For this assessment, an oral relative bioavailability of 1.0 in soil was used.

---

### 5.4.2 Inhalation Bioavailability

Cadmium in air exists primarily as fine suspended particulate matter. When inhaled, some fraction of the larger particles (i.e., greater than 10 microns in diameter) is deposited in the airways or lungs, and the rest is exhaled. Finer particles tend to penetrate into the alveoli. While some soluble cadmium compounds may be absorbed from the airways or lungs, the major site of absorption is the alveoli (ATSDR, 1999). Comprehensive modelling of the kinetics of cadmium in the respiratory tree indicates that 5 to 50% of particles will be deposited, and that 50 to 100% of cadmium deposited in the alveoli will be absorbed (Nordberg *et al.*, 1985). An inhalation relative bioavailability factor of 1.0 was used in this assessment.

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### 5.4.3 Dermal Bioavailability

Health Canada (2003a) recommends a  $RAF_{\text{dermal}}$  of 0.14. Therefore, a dermal relative bioavailability of 0.14 for cadmium has been adopted in this assessment.

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## 5.5 Conclusion

The following tables present the TRV and bioavailability summaries for cadmium.

**Table 7: Selected Toxicity Reference Values for Cadmium**

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	$8.0 \times 10^{-4}$ mg/kg-day	TDI	Health Canada, 2003a
Inhalation	NA	NA	NA
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	$4.29\text{E}+01$ (mg/kg-day) <sup>-1</sup>	Unit Risk	Health Canada, 2003a

NA – Not Applicable

**Table 8: Selected Relative Bioavailabilities for Cadmium**

Route of Exposure	Relative Bioavailability	Reference
Ingestion	1.0	Assumed
Inhalation	1.0	Assumed
Dermal	0.14	Health Canada, 2003a



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## 5.6 References

- ATSDR (Agency for Toxic Substances and Disease Registry), 1999. Toxicological Profile for Cadmium. July 1999.
- Environment Canada and Health Canada, 1994. Priority Substances List Assessment Report, Cadmium and its Compounds. Canadian Environmental Protection Act. Government of Canada, Ottawa, Ontario.
- Health Canada. 1996. Health based Tolerable daily intakes/concentrations and tumorigenic doses/concentrations for priority substances. Minister of Supply and Services Canada, Ottawa.
- Health Canada, 2003a. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs). October 3, 2003.
- Health Canada, 2003b. Cadmium in Guidelines for Canadian Drinking Water Quality – Supporting Documents. Confirmed current as of December, 2004. Available online at: <http://www.hc-sc.gc.ca/hecs-sesc/water/dwgsup.htm>.
- Newton D, Johnson P, Lally AE, *et al.* 1984. The uptake by man of cadmium ingested in crab meat. *Hum Toxicol* 3:23-28. Cited In: ATSDR, 1999.
- Nordberg GF, Kjellstrom T, and Nordberg M, 1985. Kinetics and Metabolism. In: Friberg *et al.*, eds. Cadmium and health: A toxicological and epidemiological appraisal. Volume I: Exposure, dose, and metabolism. Boca Roton, FL: CRC Press, 103-178. Cited In: ATSDR, 1999.
- Rahola T, Aaran RK, and Miettinen JK, 1973. Retention and Elimination of Cd in Man. In: Health Physics Problems of International Contamination, Bujdoso, Ed. Akademiai Kiado Budapest, 1973, 213pp. Cited In: ATSDR, 1999.
- Takenaka S, Oldiges H, Konig H, *et al.*, 1983. Carcinogenicity of cadmium chloride aerosols in Wistar rats. *Journal of the National Cancer Institute* 70:367-373. Cited In: ATSDR, 1999.
- Thun MJ, Schnorr TM, Smith AB, and Halperin WE, 1985. Mortality Among a Cohort of US Cadmium Production Workers: An update. *J. Natl. Cancer Inst.* 74(2): 325-333. Cited In: US EPA, 1994.
- US EPA (United States Environmental Protection Agency). 1994. Integrated Risk Information System (IRIS) Database – Cadmium. Confirmed current as of December 2004. Available: <http://www.epa.gov/iris/>.
- WHO (World Health Organization). 1992. Toxicological Evaluation of Certain Food Additives and Contaminants – Food Additives Series 24. Prepared by the 33rd meeting of the Joint FAO/WHO Expert Committee on Food Additives, March 21-30, 1989, Geneva. Cambridge University Press, New York. pp. 163-219. Cited In: Health Canada, 1996.

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## 6.0 IRON

Iron (Fe) is an essential nutrient and a primary constituent of the earth's crust (approximately 5%). The Institute of Medicine (IOM; 2000) has developed upper limits for iron intake based on gastrointestinal effects (i.e., vomiting and diarrhea) associated with high levels of ingestion of iron supplements. For infants and children, an upper limit of 40 mg/day is recommended, while for teens and adults, an upper limit of 45 mg/day is recommended. Ingestions exceeding 60 mg/kg may be lethal. The risks associated with iron intake have not been quantitatively evaluated as iron is an essential nutrient and very high intakes are required to produce adverse human health effects.

Iron is an important constituent of haemoglobin, a molecule that binds with oxygen in red blood cells (WHO, 1992; IOM, 2000). Anemia is a deficiency in red blood cells or blood haemoglobin that can result from insufficient Fe intake and can cause adverse health effects such as decreased cognitive performance and growth in children (WHO, 2001).

---

### 6.1 Assessment of Carcinogenicity

Iron is not classifiable as a human carcinogen (ACGIH, 2005).

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### 6.2 Toxicity Reference Values

Provisional or calculated toxicity reference values (TRVs) have been obtained from the Risk Assessment Information System (RAIS, 2005) (Table 1).

**Table 9: Selected Toxicity Reference Values for Iron**

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	3.00E-01 mg/kg-day	RfD	RAIS, 2005
Dermal	4.50E-02 mg/kg-day	RfD	RAIS, 2005

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### 6.3 Bioavailability

The following section describes the oral, inhalation and dermal bioavailability of iron.

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#### 6.3.1 Oral Bioavailability

The relative oral absorption factor for iron has been conservatively assumed to be 1.0.

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#### 6.3.2 Inhalation Bioavailability

The relative inhalation absorption factor for iron has been conservatively assumed to be 1.0.

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#### 6.3.3 Dermal Bioavailability

The United States Environmental Protection Agency (US EPA, 1995) recommends a relative dermal absorption factor of 0.001 for iron.

The bioavailability of iron is summarized in the following table:

**Table10: Selected Bioavailabilities for Iron**

Route of Exposure	Relative Bioavailability	Reference
Ingestion	1.0	Assumed
Inhalation	1.0	Assumed
Dermal	0.001	US EPA, 1995

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## 6.4 References

American Conference of Governmental Industrial Hygienists (ACGIH) (2005) TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices, pp 34. Cincinnati, OH.

IOM (Institute of Medicine) (2000). Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Panel on Micronutrients, Institute of Medicine, Food and Nutrition Board. Washington, D.C., National Academy Press.

Risk Assessment Information System (2005) Chemical-specific toxicity values: Aluminum. Web page accessed November, 2005: [http://risk.lsd.ornl.gov/cgi-bin/tox/TOX\\_select?select=nr4d](http://risk.lsd.ornl.gov/cgi-bin/tox/TOX_select?select=nr4d).

United States Environmental Protection Agency (1995) Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment (Interim Guidance). Waste Management Division, Office of Health Assessment.

WHO. (2001). Iron deficiency anaemia: assessment, prevention and control. A guide for programme managers. Geneva, World Health Organization. Document WHO/NHD/01.3. Available at: [http://www.who.int/nut/documents/ida\\_assessment\\_prevention\\_control.pdf](http://www.who.int/nut/documents/ida_assessment_prevention_control.pdf).

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## 7.0 LEAD

Lead is a naturally occurring element found in the earth's crust. Exposure to lead can lead to effects to the central nervous system. In adults, exposure can result in decreased performance, weakness and anemia. Kidney damage and brain damage may also occur at high exposures. In children exposed to lead, central nervous system effects occur at blood lead levels so low as to indicate that there is no threshold level below which effects do not occur (ATSDR, 1999).

---

### 7.1 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 US EPA (2004) lists lead as a Group 2B, 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.

Health Canada (1996) 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 no observed adverse effects level (NOAEL).

The International Agency for Research on Cancer (IARC) (1987) lists lead and inorganic lead compounds as Group 2B, possibly carcinogenic to humans. The IARC states that there is inadequate evidence of carcinogenicity in humans.

---

### 7.2 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 (1996) and the Agency for Toxic Substances and Disease Registry (ATSDR, 1999). These reviews show that infants, young children up to the age of six, and pregnant women (developing fetuses) are the most susceptible.

---

### 7.3 Selection of Toxicity Values

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#### 7.3.1 Non-Cancer Oral Toxicity Reference Values

The oral reference dose (RfD) for lead used by Health Canada (1996), is the same as the provisional tolerable weekly intake (PTWI) for children of 25 µg/kg, equivalent to approximately 3.57 µg/kg/day from all sources, established by the World Health Organization (WHO) (1986). The PTWI is considered sufficiently low to protect against effects on the central nervous system and blood (i.e., neurobehavioural effects and anemia). This PTWI was based on the results of metabolic studies in infants and was used to establish Canadian drinking water standards for lead (CCME, 1987).

WHO (1993) has more recently extended this PTWI to all age groups to protect other sensitive population groups, such as women of child-bearing age. The PTWI of 0.025 mg/kg was maintained at the fifty-third meeting of the Joint FAO/WHO Expert Committee on Food Additives (WHO, 1999).

The MOE (1994) developed an intake of concern ( $\text{IOC}_{\text{pop}}$ ) of 1.85  $\mu\text{g/kg-day}$  based on a lowest observed adverse effects level (LOAEL) of 10  $\mu\text{g/dL}$  blood lead level. A transfer factor of 0.21  $\mu\text{g}$  lead per dL blood level per  $\mu\text{g/day}$  was applied for a 13kg child aged 6 months to 4 years. An uncertainty factor of 2 was applied. The LOAEL is based on a convergence of data on blood levels of 10 to 15  $\mu\text{g/dL}$  as the level of concern for impairment of neurological behaviour.

An oral RfD of  $3.57 \times 10^{-3}$  mg/kg-day has been adopted in this assessment based on Health Canada's recommended oral RfD.

---

### 7.3.2 Cancer Oral Toxicity Reference Values

The lack of suitable positive carcinogenic data precludes the derivation of an oral slope factor or unit risk for lead.

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### 7.3.3 Non-Cancer Inhalation Toxicity Reference Values

Inhalation toxicity values for lead have not been developed by the US EPA or Health Canada and therefore, due to insufficient data, a non-cancer inhalation TRV has not been selected for this assessment.

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### 7.3.4 Cancer Inhalation Toxicity Reference Values

The lack of suitable positive carcinogenic data precludes the derivation of an inhalation slope factor or unit risk for lead.

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## 7.4 Bioavailability

The following section describes the bioavailabilities of lead.

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### 7.4.1 Oral Bioavailability

Adult humans absorb 10-15% of ingested lead; however, children absorb up to 50% of ingested lead (ORNL, 1994). Gastrointestinal absorption may vary depending on dietary factors and the chemical form of the lead. Lead is more readily absorbed in fasting individuals (up to 45% for adults) than when ingested with food. Absorption is also increased in children suffering from iron or calcium deficiencies. Gastrointestinal absorption in children may be only 30% for lead present in dust and dirt and 17% for lead in paint chips, compared with 50% for lead in food and beverages (US EPA, 2004).

Oral bioavailability for lead assuming normal feeding habits are 42 to 53% in children (Hrudey *et al.*, 1996) and 4 to 13% in adults (CCME, 1996; Hrudey *et al.*, 1996). Other studies for estimating lead oral bioavailability assuming normal feeding habits are 40 to 50% in children (Alexander *et al.*, 1974; Ziegler *et al.*, 1978) and 4 to 13% in adults (Harrison *et al.*, 1969;

Rabinowitz *et al.*, 1980; Blake *et al.*, 1983; Chamberlain, 1985). The oral bioavailability is assumed to be 53% since the RfD is based on oral exposure that is protective of children. The absorption of lead in soil and dust by children has been estimated at 30% (CCME, 1996). For the purpose of this assessment, the relative oral bioavailability from soil exposure was assumed to be 100%.

---

## 7.4.2 Inhalation Bioavailability

The relative inhalation absorption factor for lead has been conservatively assumed to be 1.0.

---

## 7.4.3 Dermal Bioavailability

The dermal bioavailability factor of 0.01 is recommended by US EPA Region III (1995). Health Canada (2003) recommends a relative dermal absorption factor of 0.006 for lead. Therefore, a relative dermal bioavailability of 0.006 was adopted for this assessment.

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## 7.5 Conclusion

The following tables present the TRV and bioavailability summaries for lead.

**Table 11: Selected Toxicity Reference Values for Lead**

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	$3.57 \times 10^{-3}$ mg/kg-day	RfD	Health Canada, 1996
Inhalation	NA	NA	NA
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	NA	NA	NA
NA – Not Applicable			

**Table 12: Selected Bioavailabilities for Lead**

Route of Exposure	Relative Bioavailability	Reference
Ingestion	1.0	Assumed
Inhalation	1.0	Assumed
Dermal	0.006	Health Canada, 2003

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## 7.6 References

Alexander YW, Clayton BE, and Delves HT, 1974. Mineral and Trace-Metal Balances in Children Receiving Normal and Synthetic Diets. *Quart. J. Med.* 43: 89. Cited In: ATSDR, 1999.

ATSDR (Agency for Toxic Substances and Disease Registry), 1999. Toxicological Profile for Lead. July 1999. Available on-line at: <http://www.atsdr.cdc.gov/toxpro2.html>.

Blake KHC, Barbezat GO and Mann M, 1983. Effect of Dietary Constituents on the Gastrointestinal Absorption of  $^{203}\text{Pb}$  in Man. *Environ. Res* 30:182. Cited In: ATSDR, 1999.

CCME (Canadian Council of Ministers of the Environment), 1987. Guidelines for Canadian Drinking Water Quality. Supporting Documentation. Health and Welfare Canada.

- CCME (Canadian Council of Ministers of the Environment), 1996. Canadian soil quality Guidelines For contaminated Sites. Human Health Effects: Inorganic Lead. Final report. The National Contaminated Sites Remediation Program. Canadian Council of Ministers of the Environment.
- Chamberlain AC, 1985. Prediction of Response of Blood Lead to Airborne and Dietary Lead from Volunteer Experiments with Lead Isotopes. *Proc R Soc Lond B*224:149. Cited In: ATSDR, 1999.
- Harrison GE, Carr TEF, Sutton A and Humphreys ER, 1969. Effect of Alginate on the Absorption of Lead in Man. *Nature* 224:1115. Cited In: ATSDR, 1999.
- Health Canada. 1996. Health based Tolerable daily intakes/concentrations and tumorigenic doses/concentrations for priority substances. Minister of Supply and Services Canada, Ottawa.
- Health Canada, 2003. Federal Contaminated Site Risk Assessment in Canada, Part I: Guidance on Human Health Screening Level Risk Assessment (SLRA). October 3, 2003.
- Hrudey SE, Chen W and Rousseaux CG, 1996. Bioavailability in Environmental Risk Assessment. CRC Press, Lewis Publishers, Boca Raton.
- IARC (International Agency for Research on Cancer), 1987. Lead and Lead Compounds. *Monographs*. Supplement 7: p. 230. World Health Organization.
- MOE (Ontario Ministry of Environment), 1994. Scientific Criteria Document for Multimedia Environmental Standards Development – Lead.
- ORNL (Oak Ridges National Laboratory), 1994. Toxicity Summary for Lead (Inorganic). December, 1994.
- Rabinowitz MB, Kopple JD and Wetherill GW. 1980. Effect of food intake and fasting on gastrointestinal lead absorption in humans. *Am J Clin Nu.* 33:1784. Cited In: ATSDR, 1999.
- US EPA (United States Environmental Protection Agency) Region III, 1995. Preliminary Remediation Goals Database.
- US EPA (United States Environmental Protection Agency), 2004. Integrated Risk Information System (IRIS) Database. Lead and compounds (inorganic). Confirmed current as of December 2004. Available on-line at: <http://www.epa.gov/iris>.
- WHO (World Health Organization), 1986. Evaluation of Certain Food Additives and Contaminants. Thirtieth report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report Series 751. The World Health Organization, Geneva, 1986.
- WHO (World Health Organization). 1993. Evaluation of Certain Food Additives and Contaminants. Forty-first report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report Series 837. The WHO, Geneva, 1993.
- Ziegler EE, Edwards BB, Jensen RL, Mahaffey KR, and Fomon SJ, 1978. Absorption and retention of lead by infants. *Pediat Res* 12:29. Cited In: ATSDR, 1999



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## **8.0 LITHIUM**

Lithium (Li), a group 1 alkali metal, is the thirty-first most abundant element and is found in the Earth's crust at a concentration of 65 parts per million (ppm) (Stwertka, 2002). Lithium is widely distributed but does not occur in nature in its free form. Because of its reactivity, it is always found bound with one or more other elements or compounds. Lithium forms a minor part of almost all igneous rocks and is also found in natural brines, waters and some foods. The average dietary intake is estimated to be about 2 mg per day (Beliles, 1994). Commercially, it is primarily used in heat transfer alloys and batteries.

Lithium is also commonly used in the treatment of depressive and bipolar affective disorders (Ellenhorn and Barceloux, 1988). As such, it is used in a population at relatively high risk for overdose. Because it has a comparatively narrow therapeutic index, lithium intoxication is also a frequent complication of chronic lithium therapy. Symptoms of lithium toxicity include abdominal pain, ataxia, slurred speech, tremors, vomiting and diarrhea (Arena, 1986).

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### **8.1 Assessment of Carcinogenicity**

The carcinogenic potential of lithium has not been established in humans or animals.

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### **8.2 Toxicity Reference Values**

Toxicity reference values (TRVs) have not been established by Health Canada, the United States Environmental Protection Agency or the Risk Assessment Information System. However, the oral toxicity of most lithium compounds is relatively low, with oral LD50 values for several compounds and animal species ranging from 422 to 1165 mg/kg (RAIS, 2005).

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### **8.3 Bioavailability**

The following section describes the oral, inhalation and dermal bioavailability of lithium.

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#### **8.3.1 Oral Bioavailability**

The relative oral absorption factor for lithium has been conservatively assumed to be 1.0.

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#### **8.3.2 Inhalation Bioavailability**

The relative inhalation absorption factor for lithium has been conservatively assumed to be 1.0.

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#### **8.3.3 Dermal Bioavailability**

The United States Environmental Protection Agency (US EPA, 1995) recommends a relative dermal absorption factor of 0.001 for lithium.

The bioavailability of lithium is summarized in the following table:

**Table 13: Selected Bioavailabilities for Lithium**

Route of Exposure	Relative Bioavailability	Reference
Ingestion	1.0	Assumed
Inhalation	1.0	Assumed
Dermal	0.001	US EPA, 1995

## **8.4 References**

Arena JM (ed) (1986) Poisoning: Toxicology, Symptoms, Treatments. Charles C Thomas, Springfield, IL.

Beliles RP (1994) Lithium, in Patty's Industrial Hygiene and Toxicology. 4th ed., vol II, Part C. GD Clayton and FE Clayton (eds), John Wiley and Sons Inc., New York, NY.

Ellenhorn MJ and DG Barceloux (1988) Medical Toxicology: Diagnosis and Treatment of Human Poisoning. Elsevier, New York, NY.

Risk Assessment Information System (RAIS; 1995) Toxicity profiles: Lithium. Web page accessed November, 2005: <http://risk.lsd.ornl.gov/tox/profiles/lith.shtml>.

Stwertka A (2002) A guide to the elements. Oxford University Press, New York, NY.

United States Environmental Protection Agency (1995) Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment (Interim Guidance). Waste Management Division, Office of Health Assessment.

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## 9.0 MANGANESE

Manganese (Mn) is an essential trace element for humans and can be found in several food items, including grains and cereals. Exposure to elevated concentrations of Mn has been associated with adverse effects on human health and it is subject to regulation (USEPA, 1996; WHO, 1973).

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### 9.1 Assessment of Carcinogenicity

The ATSDR (2001) states that there is no human cancer data available for manganese. Exposure to high levels of manganese in food resulted in a slightly increased incidence of pancreatic tumors in male rats and thyroid tumors in male and female mice.

The EPA has determined that manganese is not classifiable as to human carcinogenicity.

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### 9.2 Susceptible Populations

Some individuals exposed to very high levels of manganese for long periods of time in their work developed mental and emotional disturbances and slow and clumsy body movements, a condition referred to as "manganism" (ATSDR, 2001). Manganism is symptomatic of brain injury resulting from exposure to elevated manganese over a time span of months or years. Exposure to high levels of the metal may also cause respiratory problems and sexual dysfunction (ATSDR, 2001).

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### 9.3 Selection of Toxicity Values

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#### 9.3.1 Non-Cancer Oral Toxicity Reference Values

The United States Environmental Protection Agency (US EPA, 1996) provides an oral RfD for non-carcinogenic effects from manganese of  $1.4 \times 10^{-1}$  mg/kg-day (last updated 18/11/2004). According to the US EPA, the Food and Nutrition Board of the National Research Council (NRC, 1989) determined an "estimated safe and adequate daily dietary intake" of manganese to be 2-5 mg/day for adults. The lower end of this range was based on a study by McLeod and Robinson (1972), who reported equilibrium or positive balances at intakes of 2.5 mg Mn/day or higher. An "extra margin of safety" was included in the ESADDI from the level of 10 mg/day, which the NRC considered to be safe for an occasional intake.

After a review of several investigations of adult diets, the World Health Organization (WHO, 1973) reported the average daily consumption of manganese to range from 2.0-8.8 mg Mn/day. Higher manganese intakes are associated with diets high in whole-grain cereals, nuts, green leafy vegetables, and tea. From manganese balance studies, the WHO concluded that 2-3 mg/day is adequate for adults and 8-9 mg/day is "perfectly safe."

The US EPA therefore concludes, based on this information taken together, that an appropriate reference dose for manganese is 10 mg/day (0.14 mg/kg-day). In applying the reference dose for manganese to a risk assessment, it is important that the assessor consider the ubiquitous nature of manganese, specifically that most individuals will be consuming about 2-5 mg Mn/day in their diet

(USEPA 1996). The US EPA states that this is particularly important when one is using the reference dose to determine acceptable concentrations of manganese in water and soils.

Kondakis et al (1989) performed an epidemiological study of manganese in drinking water. Because of the uncertainty in the amount of manganese in the diet and the amount of water consumed, it is impossible to estimate the total oral intake of manganese in this study (US EPA, 1996). These limitations preclude the use of this study to determine a quantitative dose-response relationship for the toxicity of manganese in humans (US EPA, 1996). This study, nevertheless, raises significant concerns about possible adverse neurological effects at doses not far from the range of essentially.

Because of this concern, the US EPA recommends that a modifying factor of 3 be applied when assessing risk from manganese in drinking water or soil.

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### 9.3.2 Non-Cancer Inhalation Toxicity Reference Values

The US EPA has derived a reference concentration (RfC) of  $5 \times 10^{-5} \text{ mg/m}^3$  (last revised 12/01/1993) based on impairment of neuro-behavioural function. The RfC was derived from a study of occupational workers exposed to Mn in the air.

Health Canada has not derived an inhalation TRV for Mn.

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## 9.4 Bioavailability

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### 9.4.1 Oral Bioavailability

The gastrointestinal absorption of manganese in humans typically averages about 3-5% (Davidsson *et al.* 1988, 1989; Mena *et al.* 1969, cited in ATSDR, 2000). No marked differences in manganese retention between food ingestion and water ingestion routes were found, but manganese absorption as manganese (II) chloride in demineralized water has been shown to be higher than manganese absorption from food, even though the biological half-lives of manganese absorbed from the two media are the same.

Roels *et al.* (1997, cited in ATSDR, 2000) conducted a gavage study on 3-month-old male rats, similar to their study described above, and the results suggested that the more soluble forms of manganese are more readily absorbed through the gastrointestinal tract.

---

### 9.4.2 Inhalation Bioavailability

No studies were located by the ATSDR (2000) regarding absolute amounts of Mn absorbed after human or animal inhalation exposure to Mn dusts. Two studies on rats, however, were cited by the ATSDR as lending support to the notion of manganese absorption through the olfactory mucosa leading to increased brain manganese deposition in rats.

Tjälve *et al.* (1996, cited in ATSDR, 2000) administered  $4 \mu\text{g } ^{54}\text{Mn/kg}$  intranasally to weanling male Sprague-Dawley rats, and conducted whole body autoradiography at different time points, up to 12 weeks, to determine how much Mn was absorbed, and how it was distributed in the rats' bodies.

Their results indicated that the olfactory bulb contained most of the measured Mn; 90% of the initial dose was present in the olfactory bulb 1 day after dosing, decreasing to 16% after 12 weeks.

A secondary study was also conducted by Roels *et al* (1997, cited in ATSDR, 2000) using the same manganese compounds. In this study, 1.22 mg Mn/kg was administered intratracheally once weekly for four weeks to 3-month-old rats. At the end of the study period, the rats dosed with the chloride showed a 68% increase in blood manganese, and the rats dosed with the oxide showed a 41% increase in blood manganese, suggesting that as far as the inhalation route is concerned, the more soluble forms of manganese tend to be more readily absorbed. The blood manganese results were further supported by analysis of manganese levels in the rats' brains, which were significantly increased in the rats exposed to the manganese compounds over control levels.

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### 9.4.3 Dermal Bioavailability

Health Canada (2003) does not provide a relative dermal absorption fraction (RAF) for manganese, however, the United States Environmental Protection Agency (US EPA, 1995) recommends a RAF of 0.001.

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## 9.5 References

- ATSDR (Agency for Toxic Substances and Disease Registry), 2000. *Toxicological Profile for Manganese*. February 2001.
- Health Canada, 2003. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs). October 3, 2003.
- Kondakis, X.G., N. Makris, M. Leotsinidis, M. Prinou and T. Papapetropoulos. 1989. Possible health effects of high manganese concentration in drinking water. *Arch. Environ. Health*. 44(3): 175-178.
- NRC (National Research Council). 1989. Recommended Dietary Allowances, 10th ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington, DC. p. 230-235.
- United States Environmental Protection Agency. 1995. Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment (Interim Guidance). Waste Management Division, Office of Health Assessment.
- US EPA (United States Environmental Protection Agency), 1996. Integrated Risk Information System (IRIS) Database: Manganese, inorganic (Oral RfD Assessment). Last revised 18/11/2004. Available on-line at: <http://www.epa.gov/iris/>.
- WHO (World Health Organization). 1973. Trace Elements in Human Nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland. p. 34-36.

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## 10.0 NICKEL

Nickel (Ni) is a naturally occurring metal existing in various mineral forms, and may be found throughout the environment including rivers, lakes, oceans, soil, air, drinking water, plants and animals. Soil and sediment are the primary receptacles for nickel, but mobilization may occur depending on the physico-chemical characteristics of the soil (ATSDR, 1988). The average worldwide concentration of nickel in soil is 8 parts per million (ppm), however, areas can naturally contain much higher concentrations. Nickel is used in a wide variety of metallurgical processes such as electroplating and alloy production, as well as in nickel-cadmium batteries. Some evidence suggests that nickel may be an essential trace element for mammals (Goyer, 1991). As for most metals, the toxicity of nickel is dependent on the route of exposure and the solubility of the nickel compound (Coogan et al., 1989).

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### 10.1 Assessment of Carcinogenicity

Nickel is considered to be carcinogenic to humans and is listed as a Group 1 carcinogen by IARC. The US EPA (1991) considers nickel refinery dust to be a human carcinogen via inhalation exposure. The carcinogenic activity of nickel is dependent upon the specific species of nickel present. Compounds such as nickel sulphide and nickel subsulphide, both present in nickel refinery dusts, have been shown to be carcinogenic in humans (CEPA, 1994; US EPA, 1991).

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### 10.2 Susceptible Populations

Sensitized individuals may be unusually susceptible because exposure to nickel by any route may trigger an allergic response (ATSDR, 1997). Persons with kidney dysfunction are also likely to be more susceptible to nickel as the primary route of nickel elimination is via the urine. Increased nickel serum concentrations have been observed in dialysis patients (Hopfer et al., 1989).

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### 10.3 Selection of Toxicity Values

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#### 10.3.1 Non-Cancer Oral Toxicity Reference Value

The oral RfD developed by the US EPA (1996) for nickel (soluble salts) is 2.0E-2 mg/kg-day. The RfD was based on decreased body weight and organ weights in rats exposed to nickel in food for two years (Ambrose et. al., 1976).

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#### 10.3.2 Cancer Inhalation Toxicity Reference Value

The US EPA (1991) has developed a unit risk value of 2.4E-1 (mg/m<sup>3</sup>)<sup>-1</sup>. The inhalation slope factor of 1.1 (mg/kg/day)<sup>-1</sup> was calculated from the inhalation unit risk by multiplying the inhalation unit risk by a body weight of 69.4 kg and dividing by an inhalation rate of 15.33 m<sup>3</sup>/day.

**Table 14: Selected Toxicity Values for Nickel**

Route of Exposure	TRV	Toxicological Basis	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	2.0E-02 mg/kg-day	Decreased body and organ weights in rats	US EPA
Inhalation	N/A	N/A	N/A
<b>Cancer Effects</b>			
Ingestion	N/A	N/A	N/A
Inhalation	1.1 (mg/kg/day) <sup>-1</sup>	Lung cancer	US EPA

N/A Not Available

## 10.4 Bioavailability

### 10.4.1 Oral Bioavailability

An oral absorption fraction was reported to be 0.2 for nickel refinery dust and nickel subsulphide (US EPA, 2000). CEPA (1994) reported that 1 to 10% of ingested nickel is absorbed. The oral bioavailability factor for this assessment was 1.0.

### 10.4.2 Inhalation Bioavailability

There was no data available for inhalation bioavailability, therefore, a inhalation bioavailability factor of 1.0 was used.

### 10.4.3 Dermal Bioavailability

The dermal bioavailability factor of 0.01 used in this assessment is based on the recommendations of the US EPA Region III (1995).

## 10.5 References

- Ambrose, A.M., Larson, D.S. Borzelleca, J.R. and Hennigar, G.R. 1976. Long term toxicologic assessment of nickel in rats and dogs. J Food Sci Technol 13:181-87. Cited In: US EPA, 1996.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1997. Draft Toxicological Profile for Nickel. Available on-line at: <http://www.atsdr.cdc.gov/toxprofiles/>
- ATSDR (Agency for Toxic Substances and Disease Registry). 1988. Toxicological Profile for Nickel, ATSDR/U.S. Public Health Service, ATSDR/TP-88/19.
- CEPA. 1994. Nickel and its Compounds. Canadian Environmental Protection Act. Priority Substances List Assessment Report.
- Coogan, T. P., D. M. Latta, E. T. Snow, and M. Costa. 1989. Toxicity and carcinogenicity of nickel compounds, In: Critical Reviews in Toxicology, Vol 19. McClellan, R.O., ed., CRC Press, Boca Raton, FL. pp. 341-384.



- Goyer. R. 1991. Toxic effects of metals, In: Casarett and Doull's Toxicology, 4th ed. Amdur, M.O., J.D. Doull and C.D. Klaassen, eds., Pergamon Press, New York. pp.623-680.
- Hopfer SM, Fay WP, Sunderman FW Jr. 1989. Serum nickel concentrations in hemodialysis patients with environmental exposure. Ann Clin Lab Sci 19:161-167. Cited In: ATSDR, 1997.
- ORNL (Oak Ridge National Laboratory). 2003. Risk Assessment Program. Risk Assessment Information System. Toxicity and Chemical-specific Factors Data Base Search. [http://risk.lsd.ornl.gov/tox/tox\\_values.shtml](http://risk.lsd.ornl.gov/tox/tox_values.shtml)
- OME (Ontario Ministry of the Environment). 2001. Soil Investigation and Human Health Risk Assessment for the Rodney Street Community: Port Colborne (2001). Standards Development Branch, Report No. SDB-101-3511-2001. March 2001.
- US EPA (Environmental Protection Agency). 1996. Integrated Risk Information System (IRIS) Database – Nickel – refinery dust. Confirmed current as of May 2003. Available on-line at: <http://www.epa.gov/iris/>
- US EPA (Environmental Protection Agency). 1991. Integrated Risk Information System (IRIS) Database – Nickel – soluble salts. Confirmed current as of May 2003. Available on-line at: <http://www.epa.gov/iris/>
- U.S. EPA Region III. 1995. Risk Assessment: Technical Guidance Manual. Assessing Dermal Contact with Soil. <http://www.epa.gov/reg3hwmd/risk/solabsg2.htm>
- US EPA. 2000. Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment Bulletins. EPA Region 4, originally published November 1995, Website version last updated May 2000: <http://www.epa.gov/region4/waste/ots/healthbul.htm> Cited in: ORNL, 2003.

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## 11.0 NATURAL URANIUM

Uranium is a naturally occurring chemical substance that is mildly radioactive. Everyone is exposed to low amounts of uranium through food, air, and water. Exposure to high levels of uranium can cause kidney disease. It is not known to cause cancer, but can decay into other radioactive materials that may cause cancer (ATSDR, 1999).

Uranium is a normal part of rocks, soil, air, and water, and it occurs in nature in the form of minerals - but never as a metal. Uranium metal is silver-colored with a grey surface and is almost as strong as steel. Natural uranium is a combination of three types or isotopes called U-234 ( $^{234}\text{U}$ ), U-235 ( $^{235}\text{U}$ ), and U-238 ( $^{238}\text{U}$ ). All three isotopes are the same chemical, but they have different radioactive properties (ATSDR, 1999).

---

### 11.1 Assessment of Carcinogenicity

Humans and animals exposed to high levels of uranium did not have higher cancer rates (ATSDR, 1999). The Committee on the Biological Effects of Ionizing Radiation (BEIR IV) reported that eating food or drinking water that has normal amounts of uranium will most likely not cause cancer.

Uranium can decay into other radioactive substances, such as radium, which can cause cancer if you are exposed to enough of them for a long enough period of time. Studies have reported lung and other cancers in uranium miners; however, the miners also smoked and were exposed to other substances that cause cancer, such as radon and silica dust (ATSDR, 1999).

---

### 11.2 Susceptible Populations

It is not known if exposure to uranium can affect the developing human fetus. In laboratory animals, high doses of uranium in drinking water resulted in birth defects and an increase in fetal deaths (ATSDR, 1999). Measurements of uranium have not been made in pregnant women, so it is not known if uranium can cross the placenta and enter the fetus. In an experiment with pregnant animals, only a small amount of the injected uranium reached the fetus. Furthermore, populations susceptible to uranium toxicosis would include people with impaired renal function. People with stomach ulcers are thought to have elevated absorption of some toxic metals and might be unusually susceptible to uranium toxicity (ATSDR, 1999).

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### 11.3 Selection of Toxicity Values

The following section describes various studies conducted to establish uranium soluble salts toxicity values via ingestion and dermal routes of exposure.

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#### 11.3.1 Oral Non-Cancer Toxicity Reference Values

The United States Environmental Protection Agency (US EPA, 1996) provides an oral RfD of 3.0  $\mu\text{g}/\text{kg}\cdot\text{day}$  for non-carcinogenic effects from uranium soluble salts (last updated 18/11/2004). They do not provide an assessment for natural uranium.

In a study by Maynard and Hodge (1949), rabbits, rats and dogs were administered uranium compounds in the diet for 30 days. Of the three test species, rabbits showed greater sensitivity to the toxic effects of uranium. Based on this study, the lowest dose tested in rabbits (2.8 mg/kg-day) to cause lowered body weight and renal damage was judged to be the LOAEL. An uncertainty factor of 1000 reflects 10 for both interspecies and interspecies variability to the toxicity of the chemical in lieu of specific data, and 10 for use with a LOAEL from an animal study. The US EPA rates confidence in the oral RfD as medium given the small number of test animals and adequate number of studies.

Health Canada (2001) proposed a tolerable daily intake (TDI) for uranium of 0.6 µg/kg-day based on a LOAEL of 60 µg/kg-day derived from a 91-day (sub-chronic) study on rats administered uranium in drinking water. The LOAEL was based on degenerative lesions on the kidneys and applied an uncertainty factor of 100 (10 for interspecies and 10 for interspecies).

The Health Canada TDI of 0.6 µg/kg-day is used for non-carcinogenic effects from uranium. This value is based on a more recent study that the review conducted by the US EPA, which provides a lower LOAEL and is therefore the more appropriate selection.

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### **11.3.2 Dermal Non-Cancer Toxicity Reference Values**

According to the ATSDR (1999), no studies were located regarding the health effects in humans after dermal exposure to uranium.

According to the ATSDR (1999), there is a lack of information regarding the health effects in humans from dermal exposures; only two studies were located in this respect. Deaths have occurred in animals after dermal exposure to uranium compounds from both single and repeated exposures. Generally, the more water-soluble uranium compounds were the most toxic and the rabbit was the most sensitive species (ATSDR, 1999). Deaths were due to renal failure.

Experiments with other uranium compounds in rabbits using a lanolin vehicle showed that water-soluble compounds (uranyl fluoride, uranium tetrachloride, uranium pentachloride) were the most toxic; the somewhat soluble compounds (uranium trioxide, sodium diuranate, ammonium diuranate) had intermediate toxicity; and the water insoluble compounds (uranium tetrafluoride, uranium dioxide, uranium peroxide, triuranium octoxide) caused no deaths (Orcutt 1949).

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## **11.4 Bioavailability**

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### **11.4.1 Dermal Bioavailability**

According to the ATSDR (1999), absorption of uranium through the skin has not been characterized in humans, and Health Canada (2003) does not provide a relative dermal absorption fraction (RAF) for uranium. However, the United States Environmental Protection Agency (US EPA, 1995) recommends a RAF of 0.001.

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## 11.5 References

- ATSDR (Agency for Toxic Substances and Disease Registry), 2000. *Toxicological Profile for Uranium*. September 1999.
- Health Canada, 2003. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs). October 3, 2003.
- Health Canada, 2001. Water Quality and Health: Uranium. Available at: <http://www.hc-sc.gc.ca/hecs-sesc/water/publications/uranium/toc.htm>.
- Maynard, E.A. and H.C. Hodge. 1949. Studies of the toxicity of various uranium compounds when fed to experimental animals. In: *The Pharmacology and Toxicology of Uranium Compounds*. Nations Nuclear Energy Service. Division VI, Vol. I, C. Voegtlin and H.C. Hodge, Ed. McGraw Hill, New York, NY. p. 309-376.
- Orcutt JA. 1949. The toxicology of compounds of uranium following application to the skin. In: Voegtlin C, Hodge HC, eds. *Pharmacology and toxicology of uranium compounds*. Vols 3 and 4. New York, NY: McGraw Hill Book Co.
- US EPA (United States Environmental Protection Agency), 1989. Integrated Risk Information System (IRIS) Database: Uranium, soluble salts (Oral RfD Assessment). Last revised 10/01/1989. Available on-line at: <http://www.epa.gov/iris/>.
- United States Environmental Protection Agency. 1995. Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment (Interim Guidance). Waste Management Division, Office of Health Assessment.

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## 12.0 ZINC

Zinc is the 23<sup>rd</sup> most abundant element in the earth's crust and is found in air, soil, water and all foods. It has many commercial uses such as in coatings to prevent rust, in dry cell batteries, and mixed with other metals to make alloys like brass and bronze (ATSDR, 1994). Zinc is an essential element, necessary for sustaining all life. It stimulates the activity of approximately 100 enzymes, supports a healthy immune system, is needed for wound healing, helps maintain the sense of taste and smell, and is needed for DNA synthesis. Zinc also supports normal growth and development during pregnancy, childhood and adolescence. The recommended daily allowance of zinc is 15 mg for adult males, 12 mg for adult females, 10 mg for children older than 1 year, and 5 mg for infants 0-12 months old (NRC, 1989).

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### 12.1 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). Additionally, animal studies have 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, the US EPA classified zinc as a Class D substance; not classifiable as to human carcinogenicity (US EPA, 1992).

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### 12.2 Susceptible Populations

There is no specific information regarding the existence of human subpopulations that are sensitive to the toxic effects of zinc (ATSDR, 1994).

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### 12.3 Selection of Toxicity Values

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#### 12.3.1 Non-Cancer Oral Toxicity Reference Value

The U.S. EPA (1992) suggested an oral RfD of 0.3 mg/kg-day based on decreased blood enzyme levels (i.e., superoxide dismutase) in females in a diet supplement study (Yadrick *et al.*, 1989).

No toxicity reference values were available in the literature for the inhalation and dermal routes of exposure.

**Table 15: Selected Toxicity Values for Zinc**

Route of Exposure	TRV	Toxicological Basis	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	3.0E-01 mg/kg-day	Decreased erythrocyte SOD	US EPA, 1992
Inhalation	NA	NA	NA
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	NA	NA	NA

NA - Not Available

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## **12.4 Bioavailability**

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### **12.4.1 Oral Bioavailability**

Several studies have measured oral absorption rates of zinc in humans. Absorption ranged from 8% to 81% following short-term exposures to zinc supplement in the diet (ATSDR, 1994). The relative oral absorption factor for zinc has been conservatively assumed to be 1.0.

---

### **12.4.2 Inhalation Bioavailability**

Quantitative studies regarding absorption of zinc and zinc compounds after inhalation exposure in humans are limited. The absorption of inhaled zinc depends on the particle size and solubility (ATSDR, 1994). Elevated levels of zinc have been found in the blood and urine of workers exposed to zinc oxide fume (Hamdi, 1969). In this assessment, the relative inhalation absorption factor for zinc has been conservatively assumed to be 1.0.

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### **12.4.3 Dermal Bioavailability**

Dermal absorption of zinc occurs, but its mechanism is not clearly defined. Studies are very limited regarding the absorption of zinc through the skin (ATSDR, 1994). Nonetheless, the United States Environmental Protection Agency (US EPA, 1995) recommends a relative dermal absorption factor of 0.001 for zinc.

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### **12.4.4 References**

- ATSDR (Agency for Toxic Substances and Disease Registry). 1994. Draft Toxicological Profile for Zinc. Available on-line at: <http://www.atsdr.cdc.gov/toxprofiles/>.
- Hamdi, EA. 1969. Chronic exposure to zinc of furnace operators in a brass foundry. *Br J Ind Med* 26:126-134.
- NRC (National Research Council). 1989. Recommended Dietary Allowances. 10<sup>th</sup> ed National Academy Press, Washington, DC.
- US EPA (Environmental Protection Agency). 1992. Integrated Risk Information System (IRIS) Database – Zinc. Confirmed current as of November, 2005. Available on-line at: <http://www.epa.gov/iris/>.
- United States Environmental Protection Agency. 1995. Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment (Interim Guidance). Waste Management Division, Office of Health Assessment.
- Yadrick, M.K., Kenney, M.A. and Winterfeldt, E.A. 1989. Iron, copper, and zinc status: Response to supplementation with zinc or zinc and iron in adult females. *Am J Clin Nutr* 49:145-150.

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## 13.0 ETHYLBENZENE

Ethylbenzene, an aromatic hydrocarbon, is a colorless, flammable liquid that has an odor akin to gasoline (Cavender, 1994). It is found in many natural products including coal tar and petroleum (ATSDR, 1998). Commercially, ethylbenzene is used as a solvent, chemical intermediate (especially in the manufacture of styrene and synthetic rubber) and as an additive to some automotive and aviation fuels. Exposure to ethylbenzene most often occurs through inhalation of vapors and mists (NTP, 1992). Occupational exposures may occur during production and conversion to polystyrene, and during production and use of mixed xylenes (Fishbein, 1985). The general public can be exposed to ethylbenzene in ambient air as a result of releases from vehicle exhaust and cigarette smoke (Fishbein, 1985).

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### 13.1 Assessment of Carcinogenicity

The only available human cancer study monitored the conditions of workers exposed to ethylbenzene for 10 years, with no tumors reported; however, no firm conclusions can be made from this study because exposure information was not provided, and 10 years is insufficient for detecting long latency tumors in humans (ATSDR, 1999). In a study by the National Toxicology Program (NTP, 1999), exposure to ethylbenzene by inhalation resulted in a clearly increased incidence of kidney and testicular tumors in male rats, and a suggestive increase in kidney tumors in female rats, lung tumors in male mice, and liver tumors in female mice. The US EPA has classified ethylbenzene as a Group D substance; not classifiable as to human carcinogenicity (US EPA, 1998).

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### 13.2 Susceptible Populations

Groups that may be more susceptible to the toxic effects of ethylbenzene are individuals with diseases of the respiratory system, liver, kidney, or skin; young children, fetuses, pregnant women, and individuals taking certain medications such as hepatotoxic medications or drugs (ATSDR, 1999).

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### 13.3 Selection of Toxicity Values

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#### 13.3.1 Non-Cancer Oral Toxicity Reference Value

The US EPA (1998) has calculated an oral reference dose (RfD) for ethylbenzene of 0.1 mg/kg-day based on liver and kidney toxicity in rats.

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#### 13.3.2 Non-Cancer Inhalation Toxicity Reference Values

In humans, chronic exposure to ethylbenzene by inhalation has shown conflicting results regarding its effects on the blood. In one study of workers occupationally exposed to ethylbenzene, adverse effects on the blood were noted, while in another study no adverse effects were observed (ATSDR, 1999). In a 20-year study of humans occupationally exposed to ethylbenzene, no liver toxicity was noted (ATSDR, 1999). Animal studies have reported effects on the blood, liver, and kidneys from chronic inhalation exposure to ethylbenzene (ATSDR, 1999).



A reference concentration (RfC) of 1.0 mg/m<sup>3</sup> was calculated by the US EPA (1998) for ethylbenzene. This concentration is based on developmental toxicity in rats and rabbits. An inhalation reference dose of 2.2E-01 mg/kg-day was calculated from the reference concentration by multiplying the RfC by an inhalation rate of 15.33 m<sup>3</sup>/day, and dividing by a body weight of 69.4 kg.

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### 13.3.3 Non-Cancer Dermal Toxicity Reference Value

The ORNL (2005) has calculated a dermal reference dose (RfD) for ethylbenzene of 0.1 mg/kg-day.

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### 13.3.4 Cancer Inhalation Toxicity Reference Values

The ORNL (2005) has calculated a provisional inhalation slope factor (SF) for ethylbenzene of 3.85E-03 mg/kg-day. The SF was calculated from the provisional inhalation unit risk of 1.1E-03.

**Table 16: Selected Toxicity Values for Ethylbenzene**

Route of Exposure	TRV	Toxicological Basis	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	1.0E-01 mg/kg-day	Liver and kidney effects	US EPA
Inhalation	2.2E-01 mg/kg-day	Developmental toxicity	US EPA
Dermal	1.0E-01 mg/kg-day	-	ORNL - Calculated
<b>Cancer Effects</b>			
Ingestion	N/A	N/A	N/A
Inhalation	3.85E-03 mg/kg-day	-	ORNL - Provisional
N/A – Not Available			

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## 13.4 Bioavailability

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### 13.4.1 Oral Bioavailability

No studies were located regarding the absorption of ethylbenzene in humans following oral exposure; however, studies in animals indicate that ethylbenzene is quickly and efficiently absorbed via this route (ATSDR, 1998). These studies indicated that 72 to 92% of administered doses were absorbed. Thus, the relative oral absorption factor for ethylbenzene has been conservatively assumed to be 1.0.

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### 13.4.2 Inhalation Bioavailability

Ethylbenzene is rapidly and efficiently absorbed via inhalation, with human studies indicating that 49 to 64% of inhaled ethylbenzene is absorbed (ATSDR, 1998). Inhalation studies in animals showed similar results, with one study indicating an estimated absorption rate of 44% (ATSDR, 1998). The relative inhalation absorption factor for ethylbenzene has been conservatively assumed to be 1.0.

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### 13.4.3 Dermal Bioavailability

Studies in humans dermally exposed to liquid ethylbenzene demonstrated rapid absorption through the skin, but absorption of ethylbenzene vapours through the skin appears to be minimal. Total dermal absorption in a study on mice indicated 3.4% of ethylbenzene was absorbed (ATSDR, 1998). The

United States Environmental Protection Agency (US EPA, 1995) recommends a relative dermal absorption factor of 0.01 for ethylbenzene, and this value was adopted in the risk assessment.

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### 13.5 References

- ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological Profile for Ethylbenzene. Available on-line at: <http://www.atsdr.cdc.gov/>
- Cavender F. 1994. Ethylbenzene, in Patty's Industrial Hygiene and Toxicology, 4th rev ed, vol II, part B, GD Clayton and FE Clayton (eds) Wiley Interscience, New York. pp. 1342-1346.
- Fishbein L. 1985. An overview of environmental and toxicological aspects of aromatic hydrocarbons. IV. Ethylbenzene. Sci Total Environ 44:269-287.
- NTP (National Toxicology Program). 1999. Toxicology and Carcinogenesis Studies of Ethylbenzene (CAS No. 100-41-4) in F344/N Rats and B6C3F1 Mice (Inhalation Studies). TR No. 466. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, Bethesda, MD.
- NTP (National Toxicology Program). 1992. NTP Management Status Report, 1995. National Toxicology Program, Research Triangle Park, North Carolina.
- ORNL (Oak Ridge National Laboratory). 2003. Risk Assessment Program. Risk Assessment Information System. Toxicity and Chemical-specific Factors Data Base Search. [http://risk/lsd.ornl.gov/cgi-bin/tox/TOX\\_9801](http://risk/lsd.ornl.gov/cgi-bin/tox/TOX_9801)
- United States Environmental Protection Agency. 1995. Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment (Interim Guidance). Waste Management Division, Office of Health Assessment.
- US EPA (Environmental Protection Agency). 1998. Integrated Risk Information System (IRIS) Database – Ethylbenzene. Confirmed current as of May 2003. Available on-line at: <http://www.epa.gov/iris/>
- U.S. EPA Region III. 1995. Risk Assessment: Technical Guidance Manual. Assessing Dermal Contact with Soil. <http://www.epa.gov/reg3hwmd/risk/solabsg2.htm>

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## 14.0 TOLUENE

Toluene, or methylbenzene, is a clear, colorless liquid with a sweet pungent odor (HSDB, 1992). It occurs naturally in crude oil and in the tolu tree, but is also produced in the process of making gasoline and other fuels from crude oil, and in making coke from coal (ATSDR, 2000). Commercially, toluene is mainly used in the production of benzene and for backblending into gasoline to increase octane ratings (US EPA, 1990). Toluene is also used as raw material in the production of benzyl chloride, benzoic acid, phenol, cresols, vinyl toluene, TNT and toluene diisocyanate (US Air Force, 1989); as a solvent for paints and coatings; and in adhesives, inks and pharmaceuticals (US EPA, 1990).

For both the general population and occupationally exposed individuals, inhalation is the primary route of exposure to toluene. Evaporation of gasoline and automobile exhaust is the largest source of toluene in the environment, while industries that use toluene as a solvent are the second largest source (EPA, 1984). Toluene is also a common indoor contaminant due to releases from common household products and from cigarette smoke (ATSDR, 2000). Non-atmospheric releases of toluene are relatively small (i.e., to water and soil) and are estimated to comprise less than 1% of total toluene releases (ATSDR, 2000). Because of volatilization and biodegradation, toluene levels in the environment are not expected to increase over time (EPA, 1990; ATSDR, 2000).

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### 14.1 Assessment of Carcinogenicity

Two epidemiological studies did not detect a statistically significant increase in the cancer risk due to inhalation exposure to toluene; however, these studies were limited due to the size of the study population and lack of historical monitoring data (ATSDR, 2000). Chronic inhalation exposure of rats did not produce an increased incidence of treatment-related neoplastic lesions (ATSDR, 2000; US EPA, 1992). The US EPA (1992) has classified toluene as a Group D substance; not classifiable as to human carcinogenicity. Toluene has been classified as Group IV-C – probably not carcinogenic to man under the classification scheme developed by the Bureau of Chemical Hazards (Environment Canada and Health Canada, 1992).

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### 14.2 Susceptible Populations

Environmental or genetic factors that decrease the capacity for metabolic detoxification of toluene are likely to increase susceptibility (ATSDR, 2000). Nutritional status may also affect susceptibility to toluene. Finally, individuals with pre-existing medical conditions, such as defects in heart rhythm, asthma or other respiratory difficulties, may be more susceptible to the toxic effects of toluene (ASTDR, 2000).

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### 14.3 Selection of Toxicity Values

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#### 14.3.1 Non-Cancer Oral Toxicity Reference Values

The US EPA (2005) provides an oral RfD of 0.08 mg/kg-day for toluene based on changes in kidney weight in studies on rats (NTP, 1990). The NOAEL was reported to be 223 mg/kg-day, while the LOAEL was 446 mg/kg-day. A benchmark dose level (BMDL) of 238 mg/kg-day was derived using

EPA's (US EPA, 2001) benchmark dose software (BMDS, Version 1.3), and used as the point of departure. The BMDL corresponds to the lower bound of the dose associated with a 10% increase in individuals having a kidney weight greater than the 98<sup>th</sup> percentile of kidney weights in the control group. An uncertainty factor of 3000 was applied to the BMDL to account for inter- and interspecies extrapolations, for subchronic-to-chronic extrapolation and for database insufficiencies and contradictions.

Health Canada (1996) has recommended an oral tolerable daily intake (TDI) of 0.22 mg/kg-day for toluene. The US EPA RfD was selected for use in this assessment as it is the value recommended by the Atlantic Partners in RBCA Implementation (PIRI) Committee for use in Atlantic Canada (Atlantic PIRI, 2003).

### 14.3.2 Non-Cancer Inhalation Toxicity Reference Values

An inhalation RfC of 5.0 mg/m<sup>3</sup> was suggested by the US EPA (2005) based on neurological effects in humans exposed to toluene occupationally. As no single study stood-out as the best study on which to characterize neurological effects or to specify a single critical effect, results from ten individual studies were combined and analyzed. The average NOAEL was 34 ppm (128 mg/m<sup>3</sup>), while the adjusted NOAEL was 46 mg/m<sup>3</sup>. The NOAEL was adjusted from an occupational exposure scenario to reflect continuous exposure conditions. A total uncertainty factor of 10 was applied to the adjusted average NOAEL for consideration of interspecies variation.

An inhalation reference dose of 1.43 mg/kg-day was calculated for toluene by multiplying the RfC by an inhalation rate of 20 m<sup>3</sup>/day, and dividing by a body weight of 70 kg.

Health Canada (Environment Canada and Health Canada, 1992) derived an inhalation TDI of 1.25 mg/kg-day based on animal studies that indicated decreased body weight in mice (Huff, 1990). The NOEL identified in this study was 0.375 mg/m<sup>3</sup>. Health Canada used an uncertainty factor of 100 (i.e., x 10 for both inter- and interspecies variation).

### 14.3.3 Non-Cancer Dermal Toxicity Reference Value

The ORNL (2005) presents a dermal toxicity reference dose of 6.40E-02 mg/kg-day.

**Table 17: Selected Toxicity Values for Toluene**

Route of Exposure	TRV	Toxicological Basis	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	8.0E-02 mg/kg-day	Increased kidney weight	US EPA, 2005
Inhalation	8.8E-02 mg/kg-day	Neurological effects	US EPA, Atlantic PIRI
Dermal	6.4E-02 mg/kg-day	Calculated	ORNL, 2005
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	NA	NA	NA

NA – Not Available

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## 14.4 Bioavailability

Studies indicate that toluene is readily absorbed from the respiratory and gastrointestinal tracts and, to a lesser extent, through the skin.

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### 14.4.1 Oral Bioavailability

In human and animal studies, complete gastrointestinal absorption of toluene was observed following oral administrations (ATSDR, 2000). The binding of toluene to soil does not prevent absorption, although it can slow down the rate of absorption (ATSDR, 2000).

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### 14.4.2 Inhalation Bioavailability

In human studies where volunteers were exposed to toluene concentrations of 50 to 80 ppm, average retention rates ranged from 50 to 83% (ATSDR, 2000).

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### 14.4.3 Dermal Bioavailability

Human and animal studies indicate that toluene is absorbed through the skin (ATSDR, 2000). Health Canada (2004) recommends a relative dermal absorption factor of 0.12 for toluene while the United States Environmental Protection Agency (US EPA, 1995) recommends a RAF of 0.01.

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## 14.5 References

- ATSDR (Agency for Toxic Substances and Disease Registry). 2000. Toxicological Profile for Toluene. ATSDR/U.S. Public Health Service.
- Environment Canada and Health Canada. 1992. Canadian Environmental Protection Act, Priority Substances List Assessment Report, Toluene. Government of Canada, Environment Canada, Health Canada.
- Health Canada (2004) Contaminated Sites Program, Federal Contaminated Site Risk Assessment in Canada, Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment. Prepared by Environmental Health Assessment Services Safe Environments Program.
- HSDB (Hazardous Substances Data Bank). 1992. Toluene, computer printout. National Library of Medicine, Bethesda, MD.
- NTP (National Toxicology Program). 1990. Toxicology and carcinogenesis studies of toluene in F344/N rats and B6C3F1 mice. Technical Report Series No. 371. Research Triangle Park, NC.
- ORNL. 2005. Oak Ridge National Laboratory. Risk Assessment Program. Risk Assessment Information System. Toxicity and Chemical-specific Factors Data Base Search. [http://risk/lcd.ornl.gov/cgi-bin/tox/TOX\\_9801](http://risk/lcd.ornl.gov/cgi-bin/tox/TOX_9801)

- US Air Force. 1989. "Toluene." The Air Force Installation Restoration Program Toxicology Guide, Vol. 2. Wright-Patterson Air Force Base, OH, pp. 19-1 to 19-37.
- US EPA (US Environmental Protection Agency). 1984. Health Assessment Document for Toluene. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/540/1-86-033.
- US EPA (US Environmental Protection Agency). 1990. Drinking Water Criteria Document for Toluene. Prepared by the Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Cincinnati, OH, for the Office of Drinking Water. ECAO-CIN-408.
- U.S. EPA (Environmental Protection Agency). 1992. Integrated Risk Information System (IRIS) Database - Toluene. Confirmed current as of May 2003. Available on-line at: <http://www.epa.gov/iris/>
- U.S. EPA Region III. 1995. Risk Assessment: Technical Guidance Manual. Assessing Dermal Contact with Soil. <http://www.epa.gov/reg3hwmd/risk/solabsg2.htm>
- U.S. EPA. (2001) Benchmark dose software (BMDS) version 1.3. Available from: <http://www.epa.gov/ncea/bmds.htm> (last modified March 22, 2001).

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## 15.0 XYLENES

Xylenes (dimethylbenzenes) are volatile solvents widely used in chemical syntheses, consumer products and agricultural chemicals. Xylenes occur naturally in petroleum and coal tar and are formed during forest fires; chemical industries produce xylenes from petroleum (ATSDR, 1995). They are also present as constituents in gasoline (Ransley, 1984). The commercial technical product "mixed xylenes" generally contains about 40% m-xylene and 20% each of o-xylene, p-xylene and ethylbenzene, as well as small quantities of toluene (Fishbein, 1985). In this summary, xylene or xylenes, refers to mixed xylenes unless the individual isomer is specified.

Because of its volatility, most exposures to xylene occur by inhalation (ATSDR, 1995). Following inhalation, xylene is readily absorbed from the lungs and systemic toxicity may ensue. Symptoms of xylene poisoning include CNS effects, ventricular arrhythmias, acute pulmonary edema, respiratory depression, nausea, vomiting and reversible hepatic impairment. Xylenes have been measured in the ambient air and drinking water of industrialized cities.

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### 15.1 Assessment of Carcinogenicity

No information is available on the carcinogenic effects of mixed xylenes in humans (ATSDR, 1995). The US EPA has classified mixed xylenes as a Group D substance; not classifiable as to human carcinogenicity (US EPA, 2003a).

Toluene has been classified as a Group IV-C substance – probably not carcinogenic to man – under the classification scheme developed by the Bureau of Chemical Hazards (Environment Canada and Health Canada, 1993).

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### 15.2 Susceptible Populations

Available data indicate that pregnant women, fetuses and very young children may be at greater risk of adverse health effects from xylenes than the general population (ATSDR, 1995). People with sub-clinical and clinical epilepsy are at increased risk of seizures if exposed to xylene because of its excitatory effects on the central nervous system (ATSDR, 1995). Individuals with renal, hepatic or cardiac disease may also be more susceptible to the toxic effects of xylene (ATSDR, 1995).

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### 15.3 Selection of Toxicity Values

Chronic exposure of humans to mixed xylenes, such as in occupational settings, has resulted primarily in neurological effects including headache, dizziness, fatigue, tremors, un-coordination, anxiety, impaired short-term memory and inability to concentrate (ATSDR, 1995). Labored breathing, impaired pulmonary function, increased heart palpitation, severe chest pain, abnormal EKG and possible effects on the kidneys have also been reported (ATSDR, 1995). Mixed xylenes have not been extensively tested for chronic effects, although animal studies show effects on the liver and central nervous system from inhalation and oral exposures, and effects on the kidney from oral exposure to mixed xylenes (ATSDR, 1995).



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### 15.3.1 Non-Cancer Oral Toxicity Reference Values

Health Canada (1996) recommended an oral tolerable daily intake of 1.5 mg/kg-day. The GSI Atlantic RBCA (Risk-Based Corrective Action) Toolkit, version 2.0, uses an oral TDI of 2 mg/kg-day. This TDI was recommended by the Atlantic PIRI (Partners in RBCA Implementation) Committee for use in Atlantic Canada (Atlantic PIRI, 2003) and was based on the reference dose (RfD) provided by the US EPA. However, the US EPA (2003a) has recently revised the RfD for mixed xylenes to 0.2 mg/kg-day based on hyperactivity, decreased body weight and increased mortality in rats. The previous and new RfD values are based on the same principal study (NTP, 1986). A data base uncertainty factor was not considered in the derivation of the previous RfD.

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### 15.3.2 Non-Cancer Inhalation Toxicity Reference Values

The US EPA (2003a) has established a reference concentration (RfC) of 0.1 mg/m<sup>3</sup> for mixed xylenes based on impaired motor coordination in rats (Korsak et al., 1994). An inhalation reference dose of 2.86E-02 mg/kg-day was calculated for xylenes by multiplying the RfC by an inhalation rate of 20 m<sup>3</sup>/day, and dividing by a body weight of 70 kg. ATSDR (1995) has calculated a chronic inhalation minimal risk level (MRL) of 0.4 mg/m<sup>3</sup> for mixed xylenes based on neurological effects in occupationally exposed workers.

Health Canada (1996) recommends a RfC of 180 ug/m<sup>3</sup> for individual xylene isomers. The RfC was based on a teratogenic study wherein rats were exposed to xylenes via inhalation. This reference concentration is recommended by the Atlantic PIRI (Partners in RBCA Implementation) Committee for use in Atlantic Canada (Atlantic PIRI, 2003).

**Table 18: Selected Toxicity Values for Xylenes**

Route of Exposure	TRV	Toxicological Basis	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	2.0E-01 mg/kg/day	Hyperactivity, body weight, and mortality	US EPA, 2003
Inhalation	2.2E-02 mg/kg/day	Impaired motor coordination	US EPA, 2003
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	NA	NA	NA

NA – Not Available

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## 15.4 Bioavailability

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### 15.4.1 Oral Bioavailability

Oral absorption was reported by ATSDR (1995) to range from 87 to 92%. Toxicokinetic studies using xylenes adsorbed to soil have shown that the bioavailability of xylenes in females that have ingested soil contaminated with xylenes is greater than when xylenes are ingested alone (ATSDR, 1995). As a result, female toddlers that ingest soil contaminated with xylenes may have an increased risk of adverse health effects. Thus, for the purposes of this assessment, the oral bioavailability factor was conservatively assumed to be 1.0.

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### 15.4.2 Inhalation Bioavailability

Studies in animals and humans have shown that xylenes are well absorbed by the inhalation route of exposure (ATSDR, 1995). In experimental studies with human subjects, retention of xylene isomers consistently remained around 60%, regardless of the inhalation exposure scenario (US EPA, 2003b).

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### 15.4.3 Dermal Bioavailability

Studies in animals and humans have demonstrated that xylenes are well absorbed by the dermal route of exposure. Furthermore, it has been shown that the dermal bioavailability of xylene adsorbed to clay is greater than the bioavailability of xylene alone (ATSDR, 1995). Health Canada (2004) recommends a relative dermal absorption factor of 0.12 for xylenes while the United States Environmental Protection Agency (US EPA, 1995) recommends a RAF of 0.01.

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## 15.5 References

- Atlantic PIRI (Partners in RBCA Implementation). 2003. Atlantic RBCA (Risk-Based Corrective Action) Reference Documentation For Petroleum Impacted Sites, Version 2.0.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Toxicological Profile for Total Xylenes. ATSDR/US Public Health Service.
- Condie LW, Hill JR and Borzelleca JF. 1988. Oral toxicology studies with xylene isomers and mixed xylenes. *Drug Chem Toxicol* 11(4):329-354.
- Environment Canada and Health Canada. 1993. Canadian Environmental Protection Act, Priority Substances List Assessment Report, Xylenes. Government of Canada, Environment Canada, Health Canada.
- Fishbein L. 1985. An overview of environmental and toxicological aspects of aromatic hydrocarbons III. Xylene. *Sci Total Environ* 43:165-183.
- Health Canada. 1996. Health-Based Tolerable Daily Intakes/Concentrations and Tumourigenic Doses/Concentrations for Priority Substances. Canadian Environmental Protection Act. Priority Substances Supporting Documentation.
- Korsak Z, Wisniewska-Knypl J and Swiercz R. 1994. Toxic effects of subchronic combined exposure to n-butyl alcohol and m-xylene in rats. *Int J Occup Med Environ Health* 7:155-166.
- NTP (National Toxicology Program). 1986. NTP technical report on the toxicology and carcinogenesis of xylenes (mixed) (60% m-xylene, 13.6% p-xylene, 17.0% ethylbenzene, and 9.1% o-xylene) in F344/N rats and B6C3F1 mice (gavage studies). Research Triangle Park, NC. NTP TR 327, NIH Publ. No. 86-2583.

- ORNL (Oak Ridge National Laboratory). 2003. Risk Assessment Program. Risk Assessment Information System. Toxicity and Chemical-specific Factors Data Base Search. [http://risk/lcd.ornl.gov/cgi-bin/tox/TOX\\_9801](http://risk/lcd.ornl.gov/cgi-bin/tox/TOX_9801)
- Ransley DL. 1984. Xylenes and ethylbenzene, *in* Kirk-Othmer Encyclopedia of chemical Technology, vol 24, 3<sup>rd</sup> ed. M Grayson and D Eckroth (eds), John Wiley, New York, NY pp. 709-744.
- Ungavary G and E Tatrai. 1985. On the embryotoxic effects of benzene and its alkyl derivatives in mice, rats and rabbits. Arch Toxicol Suppl 8:425-430.
- US EPA (Environmental Protection Agency). 2003a. Integrated Risk Information System (IRIS) Database - Xylenes. Confirmed current as of November 2005. Available on-line at: <http://www.epa.gov/iris/>
- US EPA (Environmental Protection Agency). 2003b. Toxicological review of xylenes (CAS No. 1330-20-7). In support of summary on the integrated risk information system. National Center for Environmental Assessment, Washington, DC. Available online at: <http://www.epa.gov/iris>
- US EPA Region III. 1995. Risk Assessment: Technical Guidance Manual. Assessing Dermal Contact with Soil: <http://www.epa.gov/reg3hwmd/risk/solabsg2.htm>

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## 16.0 PETROLEUM HYDROCARBON CWS FRACTIONS F2 AND F3

Petroleum hydrocarbons (PHCs) are mixtures of organic compounds that are derived from naturally occurring geological formations such as coal or oil. Common mixtures include gasoline, diesel and jet fuels. The lighter range petroleum hydrocarbons ( $C_6$ - $C_{16}$ ) tend to float on water, forming sheens or slicks, and tend to be relatively volatile, mobile and toxic (TPHCWG, 1997). These compounds may evaporate from the water surface, dissolve and disperse, and are readily degraded by natural weathering or microbial processes. Heavier oils have lower volatility and tend to be denser, and may clump or sink, becoming incorporated into sediments. Heavier PHCs ( $C_{>16}$ - $C_{>34}$ ) pose problems with their persistence in the environment and are less volatile (TPHCWG, 1997).

Fractionation of petroleum hydrocarbon mixtures is based upon the number of carbon atoms (i.e., lighter vs. heavier molecular weight chains), volatility, as well as aromatic or aliphatic structural comparisons. In general, petroleum hydrocarbons pose aesthetic problems such as unpleasant taste and odour. Some health effects that are associated with PHC exposure include neuropathy and degeneration of neural axons.

The quantification of petroleum hydrocarbon mixtures has historically been accomplished by a variety of methods where the petroleum mixture is extracted using solvents such as hexane or cyclohexane. Fractionation of the total petroleum hydrocarbon (TPH) compounds according to their volatility, number of carbon atoms, and whether they are aromatic or aliphatic compounds, is common. These aliphatic and aromatic sub-fractions play an important role in toxicity to humans and ecological receptors. In general, aromatics are more water soluble and less volatile than aliphatics. Of these fractions, the aromatic  $C_5$ - $C_8$  fraction contains the indicator compounds benzene, toluene, ethylbenzene and xylenes, collectively referred to as BTEX. Total petroleum hydrocarbons (excluding the BTEX compounds) are generally evaluated as a group due to similar toxicity end points. The BTEX compounds are generally evaluated individually (CCME, 2000).

The toxicity of petroleum hydrocarbon compounds varies widely as a consequence of the variability in the chemical composition of mixtures. The light compounds (particularly aromatics) tend to be most toxic because these compounds are most likely to penetrate and disrupt cell membranes (TPHCWG, 1997). Thus, gasoline and light fuel oils are considerably more toxic than heavy oils (i.e., Bunker C) or crude oils. The potency estimates for chemicals of concern are based on the values reported by Health Canada, the US Environmental Protection Agency (US EPA) or the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG).

The TPH model characterizes the toxicity of TPH by dividing these grouped chemicals into separate fractions based upon molecular structure and carbon size, as per TPHCWG (1997) and CCME (2000) methodology. These fractions are treated as threshold toxicants. The toxicity values employed for each of the fractions were chosen from representative indicator compounds in the specific fraction, upon which toxicity data and studies were available. The toxicity data were chosen to be representative of that fraction, whether by arbitrary reference dose (RfD) selection, or by RfD weighting, etc. The specific toxicity data employed in the assessment are detailed in the sections that follow.

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## 16.1 Assessment of Carcinogenicity

TPHCWG (1997) and CCME (2000) do not consider TPH fractions F2 and F3 carcinogenic.

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## 16.2 Susceptible Populations

There is no information readily available on susceptible populations.

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## 16.3 Selection of Toxicity Reference Values

Calculations for individual petroleum hydrocarbon sub-fractions are combined to form CWS fractions F2 and F3, on a weight percent basis. The composition of CWS fractions F2 and F3 are presented below.

**Table 19: Composition of Petroleum Hydrocarbon CWS Fractions F2 and F3**

TPH Sub-fraction	Percentage (%)	Reference
Aliphatics C <sub>&gt;10</sub> -C <sub>12</sub>	36	CCME, 2000
Aliphatics C <sub>&gt;12</sub> -C <sub>16</sub>	44	CCME, 2000
Aromatics C <sub>&gt;10</sub> -C <sub>12</sub>	9	CCME, 2000
Aromatics C <sub>&gt;12</sub> -C <sub>16</sub>	11	CCME, 2000
<b>F2 Total</b>	<b>100</b>	
Aliphatics C <sub>&gt;16</sub> -C <sub>21</sub>	56	CCME, 2000
Aliphatics C <sub>&gt;21</sub> -C <sub>34</sub>	24	CCME, 2000
Aromatics C <sub>&gt;16</sub> -C <sub>21</sub>	14	CCME, 2000
Aromatics C <sub>&gt;21</sub> -C <sub>34</sub>	6	CCME, 2000
<b>F3 Total</b>	<b>100</b>	

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### 16.3.1 Non-Cancer Oral Toxicity Reference Value

#### 16.3.1.1 Aromatic Fractions >C10-C12 and >C12-C16

The TPHCWG (1997) derived an oral reference dose (RfD) for the C<sub>9</sub>-C<sub>16</sub> aromatic hydrocarbon range by considering toxicity data for isopropylbenzene (C<sub>9</sub>), naphthalene (C<sub>10</sub>), acenaphthene (C<sub>12</sub>), biphenyl (C<sub>12</sub>), fluorene (C<sub>13</sub>), anthracene (C<sub>14</sub>), fluoranthene (C<sub>16</sub>) and pyrene (C<sub>16</sub>). The TPHCWG determined that an oral RfD of 0.04 mg/kg-day would be appropriate for this carbon range. Of the eight identified compounds within this range, four have an oral RfD of 0.04 mg/kg-day (i.e., isopropylbenzene, naphthalene, fluorene and fluoranthene). The remaining four compounds had the following oral RfDs: acenaphthene 0.06 mg/kg-day, biphenyl 0.05 mg/kg-day, anthracene 0.04 mg/kg-day and pyrene 0.03 mg/kg-day. Since this aromatic fraction is a mixture, it was believed that the oral RfD of 0.04 mg/kg-day is appropriate.

#### 16.3.1.2 Aromatic Fractions >C16-C21 and >C21-C34

The TPHCWG (1997) found that RfDs for aromatic chemicals in the C<sub>16</sub>-C<sub>35</sub> range had not been previously developed. It was also established that there was insufficient data available to develop an

RfD. After reviewing all existing information, the TPHCWG adopted the oral RfD for pyrene (C<sub>16</sub>) (0.03 mg/kg-day) as a surrogate for this fraction. This was considered a conservative approach as pyrene has a lower carbon number than any of the compounds in this aromatic fraction.

#### **16.3.1.3 Aliphatic Fractions >C10-C12 and >C12-C16**

The TPHCWG (1997) found very little information on individual compounds within the C<sub>9</sub>-C<sub>16</sub> aliphatic range. Studies on JP-8 fuel streams and dearomatized petroleum streams were utilized to produce an RfD for these fractions. An oral RfD of 0.1 mg/kg-day was derived using oral gavage data for dearomatized aliphatics. This RfD is expected to be protective of systemic toxicity as well as developmental and reproductive endpoints.

#### **16.3.1.4 Aliphatic Fractions >C16-C21 and >C21-C34**

To calculate an oral RfD value for these fractions, the TPHCWG (1997) evaluated subchronic oral studies with F/344 rats administered white mineral oils. A no-observable adverse effects level (NOAEL) of 200 mg/kg-day was observed. A safety factor of 100 was applied to the NOAEL (i.e., 3 for animal to human extrapolation, 10 for individual susceptibility and 3 for subchronic to chronic extrapolation) to derive an RfD of 2.0 mg/kg-day.

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### **16.3.2 Cancer Oral Toxicity Reference Value**

The lack of suitable positive carcinogenic data precludes the derivation of slope factors or unit risks for oral exposures.

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### **16.3.3 Non-Cancer Inhalation Toxicity Reference Value**

#### **16.3.3.1 Aromatic Fractions >C10-C12 and >C12-C16**

The TPHCWG (1997) determined that an inhalation reference concentration (RfC) of 0.2 mg/m<sup>3</sup> would be appropriate for these fractions. This was based on inhalation RfD studies with C<sub>9</sub> aromatic mixtures. The RfC was converted to an inhalation RfD of 0.04 mg/kg-day by multiplying by a breathing rate of 15.8 m<sup>3</sup>/day and dividing by 70.7 kg (i.e., average weight of receptor).

#### **16.3.3.2 Aromatic Fractions >C16-C21 and >C21-C34**

TPHCWG (1997) and CCME (2000) have not established inhalation toxicity reference values for these fractions.

#### **16.3.3.3 Aliphatic Fractions >C10-C12 and >C12-C16**

The TPHCWG (1997) determined that an RfC of 1.0 mg/m<sup>3</sup> would be appropriate for these fractions based on studies with JP-8 (C<sub>9</sub>-C<sub>16</sub>) jet fuel. The RfC was converted to an inhalation RfD of 0.2 mg/kg-day by multiplying by a breathing rate of 15.8 m<sup>3</sup>/day and dividing by 70.7 kg (i.e., average weight of receptor).

#### 16.3.3.4 Aliphatic Fractions >C16-C21 and >C21-C34

TPHCWG (1997) and CCME (2000) have not established inhalation toxicity reference values for these fractions.

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#### 16.3.4 Cancer Inhalation Toxicity Reference Value

The lack of suitable positive carcinogenic data precludes the derivation of slope factors or unit risks for inhalation exposures.

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### 16.4 Bioavailability

The following section describes the bioavailability of Petroleum Hydrocarbon CWS Fractions F2 and F3.

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#### 16.4.1 Oral Bioavailability

The Canada Wide Standards for Petroleum Hydrocarbons in Soil: Scientific Rationale (CCME, 2000) employs an Oral Absorption Factor of 1.0.

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#### 16.4.2 Inhalation Bioavailability

The Canada Wide Standards for Petroleum Hydrocarbons in Soil: Scientific Rationale (CCME, 2000) employs an Inhalation Absorption Factor of 1.0.

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#### 16.4.3 Dermal Bioavailability

The Canada Wide Standards for Petroleum Hydrocarbons in Soil: Scientific Rationale (CCME, 2000) employs a Dermal Absorption Factor of 0.2.

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### 16.5 Conclusion

The following tables present the TRV and bioavailability summaries for Petroleum Hydrocarbon CWS Fractions F2, F3 and F4.

**Table 20: Selected TRVs for Petroleum Hydrocarbon CWS Fractions F2 and F3**

TPH Sub-fraction	Route of Exposure	Toxicity Reference Value	TRV Type	Source Agency
<b>Non-Cancer Effects</b>				
Aliphatic, C <sub>&gt;10</sub> -C <sub>12</sub> , C <sub>&gt;12</sub> -C <sub>16</sub>	Ingestion	0.1 mg/kg-day	RfD	CCME, 2000
Aliphatic, C <sub>&gt;16</sub> -C <sub>21</sub> , C <sub>&gt;21</sub> -C <sub>34</sub>	Ingestion	2.0 mg/kg-day	RfD	CCME, 2000
Aromatic, C <sub>&gt;10</sub> -C <sub>12</sub> , C <sub>&gt;12</sub> -C <sub>16</sub>	Ingestion	0.04 mg/kg-day	RfD	CCME, 2000
Aromatic, C <sub>&gt;16</sub> -C <sub>21</sub> , C <sub>&gt;21</sub> -C <sub>34</sub>	Ingestion	0.03 mg/kg-day	RfD	CCME, 2000
Aliphatic, C <sub>&gt;10</sub> -C <sub>12</sub> , C <sub>&gt;12</sub> -C <sub>16</sub>	Inhalation	0.2 mg/kg-day	RfD	CCME, 2000
Aliphatic, C <sub>&gt;16</sub> -C <sub>21</sub> , C <sub>&gt;21</sub> -C <sub>34</sub>	Inhalation	NA	RfD	NA
Aromatic, C <sub>&gt;10</sub> -C <sub>12</sub> , C <sub>&gt;12</sub> -C <sub>16</sub>	Inhalation	0.04 mg/kg-day	RfD	CCME, 2000
Aromatic, C <sub>&gt;16</sub> -C <sub>21</sub> , C <sub>&gt;21</sub> -C <sub>34</sub>	Inhalation	NA	RfD	NA



**Table 20: Selected TRVs for Petroleum Hydrocarbon CWS Fractions F2 and F3**

TPH Sub-fraction	Route of Exposure	Toxicity Reference Value	TRV Type	Source Agency
<b>Cancer Effects</b>				
Aliphatic, C <sub>&gt;10</sub> -C <sub>12</sub> , C <sub>&gt;12</sub> -C <sub>16</sub>	Ingestion	NA	NA	NA
Aliphatic, C <sub>&gt;16</sub> -C <sub>21</sub> , C <sub>&gt;21</sub> -C <sub>34</sub>	Ingestion	NA	NA	NA
Aromatic, C <sub>&gt;10</sub> -C <sub>12</sub> , C <sub>&gt;12</sub> -C <sub>16</sub>	Ingestion	NA	NA	NA
Aromatic, C <sub>&gt;16</sub> -C <sub>21</sub> , C <sub>&gt;21</sub> -C <sub>34</sub>	Ingestion	NA	NA	NA
Aliphatic, C <sub>&gt;10</sub> -C <sub>12</sub> , C <sub>&gt;12</sub> -C <sub>16</sub>	Inhalation	NA	NA	NA
Aliphatic, C <sub>&gt;16</sub> -C <sub>21</sub> , C <sub>&gt;21</sub> -C <sub>34</sub>	Inhalation	NA	NA	NA
Aromatic, C <sub>&gt;10</sub> -C <sub>12</sub> , C <sub>&gt;12</sub> -C <sub>16</sub>	Inhalation	NA	NA	NA
Aromatic, C <sub>&gt;16</sub> -C <sub>21</sub> , C <sub>&gt;21</sub> -C <sub>34</sub>	Inhalation	NA	NA	NA

NA – Not Available

**Table 21: Selected Bioavailabilities for Petroleum Hydrocarbon CWS Fractions F2 and F3**

Route of Exposure	Relative Bioavailability	Reference
Oral	1.0	CCME, 2000
Dermal	0.2	CCME, 2000
Inhalation	1.0	CCME, 2000

## 16.6 References

CCME, 2000. Canada Wide Standards for Petroleum Hydrocarbons in Soil: Scientific Rationale. Canadian Council for Ministers of the Environment.

TPHCWG. 1997. Total Petroleum Hydrocarbon Criteria Working Group. Development of Fraction Specific Reference Doses (RfDs) and Reference Concentrations (RfCs) for Total Petroleum Hydrocarbons (TPH), Volume 4. Amherst Scientific.

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## 17.0 POLYCHLORINATED BIPHENYLS (PCBS)

Polychlorinated biphenyls (PCBs) were previously manufactured for use as dielectric and heat-exchange fluids, as well as various other applications (IPCS, 1993). PCBs have been produced as mixtures under various trade names including Aroclor, Pyranol, Pyroclor, Phenoclor, Pyralene, Clophen, Elaol, Kanechlor, Santotherm, Fenchlor, Apirolol and Sovol (WHO, 2003).

Although no longer manufactured (since 1977), PCBs are ubiquitous and persistent in the environment with food being the primary route of exposure for the general population (IPCS, 1993; ATSDR, 2000). Studies have demonstrated the carcinogenic potential of PCBs and furthermore the potential for PCBs to promote the carcinogenicity of other chemicals (IPCS, 1993). Commercial PCBs may contain polychlorinated dibenzofurans (PCDFs) as impurities but do not contain polychlorinated dibenzo-p-dioxins (PCDDs) (IPCS, 1993).

There are potentially 209 PCB congeners however only 130 have been identified in commercial products (IPCS, 1993; WHO, 2000). Congeners with the same number of chlorines are referred to as isomers. The number and position of chlorine atoms predicts the environmental fate and toxicity of individual congeners. In general, PCBs with a higher degree of chlorination are more lipophilic, less volatile, less readily absorbed and less water-soluble (WHO, 2000).

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### 17.1 Assessment of Carcinogenicity

Human studies provide inconclusive, yet suggestive, evidence of an association between exposure to PCBs and liver cancer; however, the studies are inconclusive due to confounding exposures and lack of exposure quantification (US EPA, 1997; ATSDR, 2000). Oral exposure studies in animals show an increase in liver tumors in rats and mice, as well as thyroid tumours in male rats (US EPA, 1997; ATSDR, 2000). No animal inhalation studies are available on the health effects of PCBs; however, PCBs are absorbed through inhalation indicating that there may be a concern for this exposure route (ATSDR, 2000).

The US EPA (1997) has classified PCBs as a group B2 substance; probable human carcinogen. The International Agency for Research on Cancer (IARC, 1987) has classified PCBs as a Group 2A substance; probably carcinogenic to humans.

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### 17.2 Susceptible Populations

Two susceptible populations were identified by the Agency for Toxic Substances and Disease Registry (ATSDR, 2000). The first was populations with incompletely developed conjugation mechanisms such as those with Gilbert's syndrome, a congenital liver disorder which occurs in approximately 3 to 7% of the adult population. These individuals are considered susceptible because of their diminished capacity to detoxify and excrete PCBs. Others with decreased hepatic activity, including individuals with hepatitis B or liver cirrhosis, may also be susceptible to PCB toxicity (ATSDR, 2000).

The second susceptible population identified by ATSDR was children, as there is strong evidence that PCBs may be transferred across the placenta of pregnant women. This together with transfer in breast

milk, and the more common routes of exposure such as consumption of contaminated foods, may potentially contribute to altered development, specifically neurobehavioral alterations (ATSDR, 2000).

## 17.3 Selection of Toxicity Reference Values

Since PCBs usually occur as mixtures of congeners with varying degrees of chlorination, toxicity data must be based on PCB mixtures to predict potential health effects. Information on PCB exposure, however, is primarily from occupational studies and accidental exposures that may be associated with exposure to other chemicals.

The most documented cases of human exposure to PCDFs are the Yusho (Japan, 1968) and Yucheng (Taiwan, 1979) incidents where people were accidentally exposed to PCDF and PCB contaminated food supply (IARC, 1978; IARC, 1987). These two incidences produced conflicting human health effects. At Yusho, Japan, an increase in liver cancer was observed in Japanese men while no excess liver mortality was observed in the affected Yucheng, Taiwan population (IARC, 1978; IARC, 1987).

### 17.3.1 Non-Cancer Oral Toxicity Reference Values

The US EPA provides toxicity reference values for PCB mixtures such as Aroclor 1254 and 1016. The US EPA (1996a) established an oral reference dose (RfD) for Aroclor 1254 of 2.0E-05 mg/kg-day based on immunological effects in monkeys. The RfD was calculated from a lowest observable adverse effect level (LOAEL) of 0.005 mg/kg-day. The US EPA (1996b) has also developed an RfD for Aroclor 1016 of 7.0E-05 mg/kg-day based on a no observable adverse effect level (NOAEL) of 0.007 mg/kg-day and LOAEL of 0.028 mg/kg-day. Aroclor 1016 is a commercial PCB mixture that is devoid of chlorinated dibenzofurans (US EPA, 1996b). The oral RfDs and effects are summarized below.

**Table 22: Oral Reference Dose for PCB Mixtures**

Congener	TRV	TRV Type	Agency	Effects
Aroclor 1254	2.0E-05 mg/kg-day	RfD	IRIS, US EPA	Ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased IgG and IgM response to sheep erythrocytes.
Aroclor 1016	7 x 10 <sup>-5</sup> mg/kg-day	RfD	IRIS, US EPA	Reduced birth weights

The ATSDR (2000) provides oral minimal risk levels (MRLs) for intermediate and chronic exposures to PCBs. These MRLs were derived to reflect exposure to PCB mixtures and are based on studies that involved Aroclor 1254 (Table 17).

**Table 23: Minimal Risk Levels for Oral Exposure to PCBs**

Exposure	TRV	Basis	Effects
Intermediate (15-364 days)	0.03 µg/kg-day	LOAEL (0.0075 mg/kg-day)	Neurobehavioral alterations in infant monkeys that were exposed to a PCB congener mixture representing 80% of the congeners typically found in human breast milk
Chronic (365 days or more)	0.02 µg/kg-day	LOAEL (0.005 mg/kg-day)	Immunological effects in adult monkeys that were evaluated after 23 and 55 months of exposure to Aroclor 1254

The chronic MRL calculated by the ATSDR is similar to the US EPA RfD for Aroclor 1254. Health Canada (2003) provides a TDI of 0.001 mg/kg-day.

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### **17.3.2 Cancer Oral Toxicity Reference Value**

The US EPA (1997) established oral slope factors for PCB mixtures using a tiered approach based on the quantity of information available. Slope factors for high risk and persistence are considered appropriate for food chain exposure, sediment and soil ingestion, inhalation of dust or aerosol, dermal exposure (if an absorption factor has been applied) and all early life exposure. Slope factors for low risk and persistence are considered appropriate for inhalation of evaporated congeners. Central and upper-bound estimates are provided; central estimates describe a typical individual's risk, while upper bounds provide assurance that this risk is not likely to be underestimated. Based on the above, the upper-bound slope factor of 2.0 mg/kg-day for high risk and persistence is used to assess the potential for carcinogenic effects via oral exposure pathways.

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### **17.3.3 Non-Cancer Inhalation Toxicity Reference Value**

Chronic inhalation exposure of workers to PCBs has been reported to result in respiratory tract symptoms (ATSDR, 2000). Despite these observed effects, non-cancer inhalation TRVs were not found.

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### **17.3.4 Cancer Inhalation Toxicity Reference Values**

The US EPA (1997) established inhalation slope factors for PCB mixtures using a tiered approach based on the quantity and quality of information available. Slope factors for high risk and persistence are considered appropriate for food chain exposure, sediment and soil ingestion, inhalation of dust or aerosol, dermal exposure (if an absorption factor has been applied) and all early life exposure. Slope factors for low risk and persistence are considered appropriate for inhalation of evaporated congeners. Central and upper-bound estimates are provided; central estimates describe a typical individual's risk, while upper bounds provide assurance that this risk is not likely to be underestimated. Based on the above, the upper-bound slope factor of 0.4 mg/kg-day is used to assess the potential for carcinogenic effects via inhalation exposure.

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## **17.4 Bioavailability**

PCBs are well absorbed following oral, inhalation or dermal exposure, and transported similarly to the systemic circulation (US EPA, 1997; ATSDR, 2000). Initially, absorbed PCBs are transported to the liver and muscle, however, soon after they are stored in fat and skin (US EPA, 1996c).

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### **17.4.1 Oral Bioavailability**

Studies with animals have shown that PCBs are readily absorbed from the gastrointestinal tract with the degree of absorption ranging from 66 to 96% (ATSDR, 2000; WHO, 2000).

Specific information concerning absorption of Aroclor 1254 is limited. Pregnant ferrets administered a single oral dose of 0.06 mg/kg Aroclor 1254 absorbed 85% of the administered dose (Bleavins *et al.*, 1984). Rats, mice and monkeys absorb between 75 to >90% of orally administered doses of PCBs (US EPA, 1996a). Oral exposure through consumption of contaminated food (including breast milk) is the major route of exposure to PCBs for the general population.

The oral relative bioavailability factor for PCBs used in this assessment was 1.0.

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#### 17.4.2 Inhalation Bioavailability

Inhalation is considered a major occupational route of exposure to PCBs; however, quantitative data concerning inhalation exposure is scarce (ATSDR, 2000). In rats, absorption and distribution of PCBs is similar following inhalation or oral administration (WHO, 2000; US EPA, 1997). Furthermore, PCB mixtures are readily absorbed after administration via aerosol with 50% of the maximum applied concentration measured in the liver 2 h later (IPCS, 1993).

The ATSDR summarized a study by Wolff (1985) wherein it was suggested that approximately 80% of PCB levels in adipose tissue of exposed capacitor workers may have been absorbed by the inhalation route. A maximum of 20% would have been derived from dermal and/or oral exposures (ATSDR, 2000).

The relative inhalation bioavailability factor used in this assessment was 1.0.

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#### 17.4.3 Dermal Bioavailability

In experimental animals, dermal absorption has been observed ranging from 20 to 60% (WHO, 2000). This is consistent with Wolff (1985), where approximately 20% of PCB levels in adipose tissue was attributed to oral and/or dermal exposures. The US EPA Region III (1995) recommends a dermal bioavailability factor of 0.06 based on the dermal absorption of 3,3',4,4'-tetrachlorobiphenyl.

The US EPA (2001) recommends a dermal absorption factor of 0.14 based on *in vitro* human and monkey studies, while the World Health Organization (WHO) recommends a conservative relative dermal bioavailability factor of 0.6 based on a number of animal studies.

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### 17.5 Conclusion

The following tables summarize the selected TRVs and relative bioavailabilities for PCBs.

**Table 24: Selected Toxicity Reference Values for PCBs**

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	0.01 mg/kg-day	TDI	Health Canada, 2003
Inhalation	NA	NA	NA
<b>Cancer Effects</b>			
Ingestion	2.0 mg/kg-day	Slope Factor	US EPA, 1997
Inhalation	0.4 mg/kg-day	Slope Factor	US EPA, 1997

Notes: NA - Not Available

**Table 25: Selected Bioavailabilities for PCBs**

Route of Exposure	Relative Bioavailability	Reference
Ingestion	1.0	Assumed
Inhalation	1.0	Assumed
Dermal	0.6	WHO, 2000

## 17.6 References

- ATSDR (Agency for Toxic Substances and Disease Registry), 2000. Toxicological Profile for Polychlorinated Biphenyls (PCBs). November, 2000.
- Bleavins MR, Breslin WJ, Aulrich RJ, *et al.*, 1984. Placental and Mammary Transfer of a Polychlorinated Biphenyl Mixture (Aroclor 1254) in the European Ferret (*mustela putoris furo*). *Environ Toxicol Chem* 3:637-644.
- IARC (International Agency for Research on Cancer), 1978. Environmental Monographs Volume 18: Polychlorinated Biphenyls. Available: <http://193.51.164.11/htdocs/monographs/vol18/polychlorinatedbiphenyls.html>.
- IARC (International Agency for Research on Cancer), 1987. Environmental Monographs Supplement 7: Polychlorinated Biphenyls. Available: <http://193.51.164.11/htdocs/monographs/suppl17/polychlorinatedbiphenyls.html>.
- IPCS (International Programme on Chemical Safety), 1993. Environmental Health Criteria 140: Polychlorinated Biphenyls and Terphenyls, 2<sup>nd</sup> ed. Available at <http://www.inchem.org/documents/ehc/ehc/ehc140.htm#p5.0>
- US EPA (United States Environmental Protection Agency). 1996a. Integrated Risk Information System (IRIS) Database – Aroclor 1254. Confirmed current as of November, 2005. Available on-line at: <http://www.epa.gov/iris>
- US EPA (United States Environmental Protection Agency). 1996b. Integrated Risk Information System (IRIS) Database – Aroclor 1016. Confirmed current as of November, 2005. Available on-line at: <http://www.epa.gov/iris>
- US EPA (United States Environmental Protection Agency). 1996c. PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures. September 1996. Available at: <http://www.epa.gov/opptintr/pcb/pcb.pdf>
- US EPA (United States Environmental Protection Agency). 1997. Integrated Risk Information System (IRIS) Database - Polychlorinated Biphenyls (PCBs). Confirmed current as of November, 2005. Available on-line at: <http://www.epa.gov/iris>
- US EPA (United States Environmental Protection Agency) Region III. 1995. Risk Assessment: Technical Guidance Manual. Assessing Dermal Contact with Soil. <http://www.epa.gov/reg3hwmd/risk/solabsg2.htm>.

Wolff MS. 1985. Occupational exposure to polychlorinated biphenyls (PCBs). *Environ Health Perspect* 60:133-138.

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe: Second Edition. WHO Regional Publications, European Series, No. 91.

WHO (World Health Organization). 2003. Concise International Chemical Assessment Document 55, Polychlorinated Biphenyls: Human Health Aspects.



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## 18.0 NAPHTHALENE

Naphthalene, a linear polycyclic aromatic hydrocarbon (PAH), is a white solid with a strong odour (ATSDR, 2003). It is also referred to as white tar or tar camphor, and has been used in mothballs and moth flakes. Naphthalene is a natural component of fossil fuels such as petroleum and coal, and is also formed when natural products such as wood or tobacco are burned (USEPA, 1986).

Commercially, naphthalene is primarily used as an intermediate in the production of phthalic anhydride, which serves as an intermediate in the production of phthalate plasticizers, resins, phthaleins, dyes, pharmaceuticals, insect repellents and other materials (Sandmeyer, 1981; US EPA, 1986; ATSDR, 2003). Other products manufactured from naphthalene include moth repellents, in the form of mothballs or crystals, and toilet and diaper pail deodorant blocks. Naphthalene is also used for making leather tanning agents and the insecticide carbaryl.

Most naphthalene entering the environment is released directly to ambient air from sources such as burning of fossil fuels and use of naphthalene-containing mothballs (ATSDR, 2003). Other sources include urban air pollution and cigarette smoke. Small amounts of naphthalene are released to the aqueous environment as a result of discharges from coal tar production and distillation processes. Occupational exposure to naphthalene may occur through inhalation and dermal contact with this compound at workplaces where naphthalene is produced and/or used (ATSDR, 2003). The most likely pathway by which the general public is exposed to naphthalene is by inhalation due to the release of this substance from combustion fuels, moth repellents and cigarette smoke (HSDB, 2005; ATSDR, 2003). Monitoring data also indicate that the general population may be exposed to naphthalene via ingestion of food and drinking water, although these pathways are considered minor when compared to inhalation.

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### 18.1 Assessment of Carcinogenicity

Using criteria of the 1986 Guidelines for Carcinogen Risk Assessment, naphthalene is classified as a Group C substance; a possible human carcinogen (US EPA, 2005). This is based on inadequate data of carcinogenicity in humans exposed to naphthalene via the oral and inhalation routes, and limited evidence of carcinogenicity in animals via the inhalation route.

According to IARC (2002), there is inadequate evidence in humans for the carcinogenicity of naphthalene, while there is sufficient evidence in experimental animals for the carcinogenicity of naphthalene. Thus, IARC considers naphthalene possibly carcinogenic to humans (Group 2B).

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### 18.2 Susceptible Populations

The hemolytic response to naphthalene is enhanced by the presence of inherited erythrocyte glucose-6-phosphate dehydrogenase deficiency (ATSDR, 2003). Although any human may develop acute hemolysis if exposed to a sufficiently high dose of naphthalene, this enzyme deficiency may cause some persons to be unusually sensitive. The incidence of the deficiency amongst Caucasians of European origin is relatively low, while there is a higher incidence among certain groups of Asians and Middle Eastern populations. Newborns and infants are thought to be more susceptible to the adverse

health effects of naphthalene (i.e., hemolytic anemia from acute exposure) because hepatic enzyme systems involved in conjugation and excretion of naphthalene metabolites are not well developed shortly after birth (EPA, 1987). No studies were located, however, that specifically examined the influence of age on naphthalene toxicokinetic capabilities in humans. Finally, individuals exposed to naphthalene in conjunction with particles from tobacco smoke, fossil fuel combustion and coal fly ash may also be at an elevated risk of developing toxic effects (ATSDR, 2003).

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## **18.3 Selection of Toxicity Reference Values**

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### **18.3.1 Non-Cancer Oral Toxicity Reference Value**

The US EPA (2005) provides an oral reference dose (RfD) for naphthalene of 0.02 mg/kg-day, based on decreased mean terminal body weight in male rats (BCL, 1980). The subchronic study established a no observable adverse effects level (NOAEL) of 71 mg/kg-day and a lowest observable adverse effects level (LOAEL) of 143 mg/kg-day. An uncertainty factor of 3000 was applied to the NOAEL (i.e., x10 for interspecies extrapolation, x10 for interspecies extrapolation, x10 for use of a subchronic exposure study and x3 for database deficiencies including lack of chronic oral exposure studies and 2-generation reproductive toxicity studies).

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### **18.3.2 Cancer Oral Toxicity Reference Value**

Data are insufficient to derive a cancer oral toxicity reference value for naphthalene (US EPA, 2005).

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### **18.3.3 Non-Cancer Inhalation Toxicity Reference Value**

The US EPA (2005) provides an inhalation reference concentration (RfC) for naphthalene of 0.003 mg/m<sup>3</sup>. This value is based on a human equivalent LOAEL of 9.3 mg/m<sup>3</sup> derived from a chronic mouse inhalation study (NTP, 1992). No NOAEL was established. Effects at the LOAEL included metaplasia of the nasal olfactory epithelium and hyperplasia of the nasal respiratory epithelium. An uncertainty factor of 3000 was applied to the LOAEL (i.e., x10 for interspecies extrapolation, x10 for interspecies extrapolation, x10 for use of a LOAEL and x3 for database deficiencies including lack of a 2-generation reproductive toxicity study and chronic inhalation data for other animal species).

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### **18.3.4 Cancer Inhalation Toxicity Reference Value**

Data are insufficient to derive a cancer inhalation toxicity reference value for naphthalene (US EPA, 2005).

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## **18.4 Bioavailability**

Absorption of naphthalene by the oral, inhalation or dermal route can be inferred in humans from the systemic toxic effects of the compound. However, the rate and extent of absorption is not known (ATSDR, 2003).

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### 18.4.1 Oral Bioavailability

Several case reports indicate that naphthalene ingested by humans can be absorbed in quantities sufficient to elicit toxicity (ATSDR, 2003). However, no studies have been located that report the rate or extent of absorption. Absorption of naphthalene presumably occurs by passive diffusion through the lipophilic matrix of the intestinal membrane.

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### 18.4.2 Inhalation Bioavailability

Clinical reports suggest that prolonged exposure to naphthalene vapors can cause adverse health effects in humans (ATSDR, 2003). Unfortunately, the rate and extent of naphthalene absorption have not been determined. Presumably naphthalene traverses the alveolar membrane by passive diffusion through the lipophilic matrix.

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### 18.4.3 Dermal Bioavailability

Several cases of naphthalene toxicity in neonates have been reported in which the proposed route of exposure was dermal (Dawson et al., 1958; Schafer, 1951; ATSDR, 2003). Each case involved the use of diapers which had been stored in contact with naphthalene (i.e., mothballs or naphthalene flakes). The authors proposed that naphthalene was absorbed through the skin, causing hemolytic anemia. It was suggested that this absorption may have been enhanced by the presence of oils which had been applied to the babies' skin (Schafer, 1951). Inhalation of vapors from the treated diapers probably contributed to the total exposure.

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## 18.5 References

- ATSDR (Agency for Toxic Substances and Disease Registry). 2003. Toxicological Profile for Naphthalene, 1-Methylnaphthalene and 2-Methylnaphthalene. September, 2003.
- Battelle's Columbus Laboratories (BCL). 1980. Unpublished subchronic toxicity study: Naphthalene (C52904), Fischer 344 rats. Prepared by Battelle Laboratories under NTP Subcontract No. 76-34-106002. Available from the Center for Environmental Research Information. (202)566-1676.
- Dawson JP, Thayer WW and Desforges JF. 1958. Acute hemolytic anemia in the newborn infant due to naphthalene poisoning: Report of two cases, with investigations into the mechanism of the disease. Blood 13:1113-1125.
- HSDB (Hazardous Substances Data Bank). 2005. Naphthalene, computer printout. National Library of Medicine, Bethesda, MD.
- IARC (International Agency for Research on Cancer). 2002. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, pp. V82 418.
- National Toxicology Program (NTP). 1992. Technical Report on the Toxicology and Carcinogenesis Studies of Naphthalene (CAS No. 91-20-3) in B6C3F1 Mice. (Inhalation Studies). DHHS, PHS, NIH, Rockville, MD.

- Sandmeyer EE. 1981. Aromatic hydrocarbons, *in* Clayton GD and FE Clayton (eds) Patty's Industrial Hygiene and Toxicology, 3<sup>rd</sup> ed, vol 2B. John Wiley and Sons, New York, NY, pp 3333-3343.
- Schafer WB. 1951. Acute hemolytic anemia related to naphthalene: Report of a case in a newborn infant. *Pediatrics* 7:172-174.
- US EPA. 1986. Health and Environmental Effects Profile for Naphthalene. Prepared by the Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, US Environmental Protection Agency, Cincinnati, OH. EPA-CIN-P192
- US EPA. 2005. Naphthalene, last updated September, 2002. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Available from: <http://www.epa.gov/ngispgm3/iris>



# Appendix B

## HHRA Model Equations, Inputs and Outputs

## B-1.0 EXPOSURE ASSESSMENT ASSUMPTIONS FOR THE HUMAN HEALTH QUANTITATIVE RISK ASSESSMENT

Receptor characteristics were selected based on site-specific assumptions for a Traditional Inuit Land Use scenario at CAM-D as well as information from other sources. These include CCME (1996), Richardson (1997) and Health Canada (2004). The relevant on-site receptors were a toddler (6 months – 4 years) for assessment of non-carcinogenic exposures and a composite receptor (75 years) for assessment of carcinogenic exposures.

The receptor characteristics for each of the relevant parameters used in this QRA are shown in Table B-1 below.

**Table B-1 – Receptor Characteristics**

Characteristics		Toddler (6 mos – 4 yrs)	Composite (75 yrs)	Reference
<b>Averaging Times and Constant Values</b>				
AT <sub>c</sub>	Averaging Time – Carcinogen (days)	--	27375	Health Canada (2004)
AT <sub>nc</sub>	Averaging Time – Non-carcinogen (days)	1642.5	--	Health Canada (2004)
ED	Exposure Duration (yr)	4.5	75	Health Canada (2004)
EF	Exposure Frequency – (days/yr)	21	21	Conservative site-specific assumption
ET	Exposure Time (hr/d)	24	24	Based on full-time exposure to the site
BW	Body weight (kg)	16.5	63.27	Health Canada (2004)
TSP	Total Suspended Particulate (kg/m <sup>3</sup> )	2.50E-07	2.50E-07	Health Canada (2004)
<b>Ingestion of Surface Soil</b>				
IR <sub>soil</sub>	Ingestion Rate of Surface Soil (mg/day)	80	23.87	Health Canada (2004)
<b>Dermal Contact with Surface Soil</b>				
SA <sub>body</sub>	Exposed surface area - body (cm <sup>2</sup> )	3009	8650	Richardson (1997)
SA <sub>hand</sub>	Exposed surface area - hand (cm <sup>2</sup> )	430	833	Richardson (1997)
SAF <sub>body</sub>	Soil adherence factor – body (mg/cm <sup>2</sup> -d)	0.01	0.01	Health Canada (2004)
SAF <sub>hand</sub>	Soil adherence factor – hand (mg/cm <sup>2</sup> -d)	0.10	0.10	Health Canada (2004)
<b>Inhalation of Soil Particles</b>				
IR <sub>air</sub>	Inhalation rate (m <sup>3</sup> /hr)	0.39	0.64	Richardson (1997)
FR <sub>soil</sub>	Fraction of dust from soil – outdoor (unitless)	1	1	assumed
<b>Ingestion of Surface Water</b>				
IR <sub>water</sub>	Ingestion of surface water (L/d)	0.6	1.34	Richardson (1997)
<b>Dermal Contact with Surface Water</b>				
SA <sub>water</sub>	Exposed surface area dermal water (cm <sup>2</sup> )	430	833	Richardson (1997)
t <sub>event</sub>	Event Duration (hr/event)	0.5	0.5	assumed
<b>Ingestion of Wild Game</b>				
IR <sub>game</sub>	Ingestion rate of wild game (Kg/d)	0.09	0.24	Health Canada (2004)
F <sub>site</sub>	Fraction of wild game that is from site (unitless)	1.00	1.00	Conservative site-specific assumption

**Table B-2 – Summary of Receptor Characteristics for Individual Age Groups**

Characteristic	Receptor Values						Reference
	Infant	Toddler	Child	Teen	Adult	Composite	
Age	0 – 6 mo.	7 mo. – 4 yr	5 – 11 yr	12 – 19 yr	20 - 75 yr	over 75 year lifetime	Health Canada (2004)
AT (years)	0.5	4.5	7	8	56	75	Health Canada (2004)
BW (kg)	8.2	16.5	32.9	59.7	70.7	62.33	Health Canada (2004)
IR <sub>soil</sub> (mg/h)	0.83	3.33	0.83	0.83	0.83	0.98	Health Canada (2004)
IR <sub>air</sub> (m <sup>3</sup> /hr)	0.0875	0.4	0.6	0.7	0.7	0.6	Health Canada (2004)
IR <sub>water</sub> (L/d)	0.3	0.6	0.8	1	1.5	1.319	Health Canada (2004)
SA <sub>hand</sub> (cm <sup>2</sup> )	320	430	590	800	890	821	Richardson, 1997
SA <sub>body</sub> (cm <sup>2</sup> )	1780	3010	5140	8000	9110	8206	Richardson, 1997
IR <sub>game</sub> (mg/d)	0	85	125	175	270	233.43	Health Canada (2004)

**Table B-3 – Summary Toxicological Reference Values and Relative Absorption Factors**

Compound	Non-carcinogenic		Carcinogenic		SAF	RAF <sub>ing</sub>	RAF <sub>inh</sub>	RAF <sub>derm</sub>
	TDI (oral) mg/kg-d	TDI (inhal.) mg/kg-d	SFo (oral) (mg/kg-d) <sup>-1</sup>	SFi (inhal) (mg/kg-d) <sup>-1</sup>				
Inorganics								
Aluminum	1.0	0.0014	--	--	0.2	1	1	0.001
Arsenic	0.0003	--	2.8	28	0.2	1	1	0.03
Barium	0.016	0.00014	--	--	0.2	1	1	0.1
Cadmium	0.0008	0.000057	--	42.9	0.2	1	1	0.14
Iron	0.3	--	--	--	0.2	1	1	0.001
Lead	0.0036	--	--	--	0.2	1	1	0.006
Lithium	0.02	--	--	--	0.2	1	1	0.001
Manganese	0.14	0.00005	--	--	0.2	1	1	0.001
Nickel	0.02	0.000018	--	--	0.2	1	1	0.35
Uranium	0.0006	--	--	--	0.2	1	1	0.001
Zinc	0.3	--	--	--	0.2	1	1	0.02
TPH - CCME CWS								
Aliph>C06-C08 - F1	5.0	--	--	--	0.2	1	1	0.2
Aliph>C08-C10 -F1	0.1	--	--	--	0.2	1	1	0.2
Arom>C08-C10 -F1	0.04	--	--	--	0.2	1	1	0.2
F1 - Total								
Aliph>C10-C12 -F2	0.1	0.223	--	--	0.2	1	1	0.2
Aliph>C12-C16 -F2	0.1	0.223	--	--	0.2	1	1	0.2
Arom>C10-C12 -F2	0.04	0.04	--	--	0.2	1	1	0.2
Arom>C12-C16 -F2	0.04	0.04	--	--	0.2	1	1	0.2
F2 - Total								
Aliph>C16-C21-F3	2.0	--	--	--	0.2	1	1	0.2
Aliph>C21-C34 -F3	2.0	--	--	--	0.2	1	1	0.2
Arom>C16-C21 -F3	0.03	--	--	--	0.2	1	1	0.2
Arom>C21-C34 -F3	0.03	--	--	--	0.2	1	1	0.2
F3 - Total								



**Table B-3 – Summary Toxicological Reference Values and Relative Absorption Factors**

Compound	Non-carcinogenic		Carcinogenic		SAF	RAF <sub>ing</sub>	RAF <sub>inh</sub>	RAF <sub>derm</sub>
	TDI (oral) mg/kg-d	TDI (inhal.) mg/kg-d	SFo (oral) (mg/kg-d) <sup>-1</sup>	SFi (inhal) (mg/kg-d) <sup>-1</sup>				
Organics								
Ethylbenzene	0.1	1	--	--	0.2	1	1	0.2
Naphthalene	0.02	0.003	--	--	0.2	1	1	0.1
PCBs – Total	0.001	--	2.0	0.4	0.2	1	1	0.1
Toluene	0.22	3.8	--	--	0.2	1	1	0.12
Xylenes	0.001	1.5	--	--	0.2	1	1	0.12

-- not available:

**Table B-4 – Dermal Permeability Coefficient (Kp) Values**

Compound	Kp (cm/hr)	Reference
Inorganics		
Aluminium	1.0E-03	US EPA (2004)
Arsenic	1.0E-03	US EPA (2004)
Barium	1.0E-03	US EPA (2004)
Cadmium	1.0E-03	US EPA (2004)
Iron	1.0E-03	US EPA (2004)
Lead	3.42E-04	US EPA (2004)
Lithium	1.0E-03	US EPA (2004)
Manganese	1.0E-03	US EPA (2004)
Nickel	2.0E-04	US EPA (2004)
Uranium	1.0E-03	US EPA (2004)
Zinc	6.0E-04	US EPA (2004)

**Table B-5 – CoPC Concentrations Used in HHRA**

Compound	C <sub>soil</sub> (mg/kg)	C <sub>wildgame</sub> (mg/kg)	C <sub>water</sub> (mg/L)
Inorganics			
Aluminium	--	--	4.68
Arsenic	3.00	0.00296	0.000853
Barium	234.00	0.103	0.0696
Cadmium	0.817	0.136	0.000162
Iron	--	--	4.22
Lead	20.3	0.943	0.00369
Lithium	--	0.502	0.0098
Manganese	--	6.56E-15	0.102
Nickel	8.43	0.647	0.0051
Uranium	--	0.0000626	0.0125
Zinc	129	37.4	0.0687

**Table B-5 – CoPC Concentrations Used in HHRA**

<b>Compound</b>	<b>C<sub>soil</sub> (mg/kg)</b>	<b>C<sub>wildgame</sub> (mg/kg)</b>	<b>C<sub>water</sub> (mg/L)</b>
TPH – CCME CWS			
Aliph>C10-C12 -F2	651	1.78	--
Aliph>C12-C16 -F2	798	25.3	--
Arom>C10-C12 -F2	163	0.00878	--
Arom>C12-C16 -F2	199	0.0195	--
F2 - Total	1810	27.1	--
Aliph>C16-C21-F3	1990	31.0	--
Aliph>C21-C34 -F3	855	1.33	--
Arom>C16-C21 -F3	499	0.0539	--
Arom>C21-C34 -F3	214	0.161	--
F3 - Total	3560	32.5	--
Organics			
Ethylbenzene	0.32	0.00000132	--
Naphthalene	4.12	0.000566	--
PCBs – Total	0.125	0.00958	--
Toluene	0.304	0.000000407	--
Xylenes	2.81	0.00000381	--

-- = Parameter not evaluated for this pathway

## B-2.0 EQUATIONS FOR THE EXPOSURE ASSESSMENT

Human receptors may be exposed to CoPCs in surface soil at the site by inadvertent ingestion, inhalation and dermal contact. Humans can also be exposed to CoPCs via ingestion of wild game, and ingestion and dermal contact with surface water. Receptor characteristics and model parameters for these pathways are provided in Section B-1 of this Appendix. The potential for CoPCs to produce adverse health effects increases with increasing exposure.

### HUMAN HEALTH INTAKE EQUATIONS

Model values have been presented to provide an example illustrating how daily intakes and human health risk estimates were calculated by the model. These equations were used to evaluate all receptors, scenarios and chemicals of potential concern.

#### B-2.1 Inhalation

##### B-2.1.1 Inhalation of Resuspended Soil/Dust – Summer - Outdoor

$$\text{Intake}_{\text{INHS/DSO}} = \frac{\text{IR}_{\text{inh}} \times \text{AF}_{\text{inh}} \times \text{TSP} \times \text{FR}_{\text{soil}} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

	<u>Units</u>
$\text{Intake}_{\text{INHS/DSO}}$ = intake factor from inhalation of resuspended soil/dust – summer outdoor	kg/kg day
$\text{IR}_{\text{inh}}$ = air inhalation rate	m <sup>3</sup> /hour
$\text{AF}_{\text{inh}}$ = inhalation absorption factor	unitless
$\text{TSP}$ = Total Suspended Particulate	kg/m <sup>3</sup>
$\text{FR}_{\text{soil}}$ = Fraction of dust from soil - outdoor	unitless
$\text{ET}$ = exposure time	hours/day
$\text{EF}$ = exposure frequency	days/year
$\text{ED}$ = exposure duration	years
$\text{BW}$ = body weight of receptor	kg
$\text{AT}_{\text{c}}$ = averaging time carcinogen = (365 days/year) x (75 years)	days
$\text{AT}_{\text{nc}}$ = averaging time non-carcinogen = (exposure frequency) x (exposure duration)	days

$$\text{LADD/CDI}_{\text{INHS/DSO}} = \text{Intake}_{\text{INHS/DSO}} \times \text{C}_{\text{soil}}$$

Where:

	<u>Units</u>
$\text{CDI}_{\text{INHS/DSO}}$ = chronic daily intake from inhalation of resuspended soil/dust – summer outdoor	mg/kg-day
$\text{LADD}_{\text{INHS/DSO}}$ = lifetime average daily dose from inhalation of soil/dust – summer outdoor	mg/kg-day
$\text{Intake}_{\text{INHS/DSO}}$ = intake factor from inhalation of resuspended soil/dust – summer outdoor	kg/kg day
$\text{C}_{\text{soil}}$ = concentration of chemical in soil	mg/kg

## B-2.2 Oral/Dermal Exposure

### B-2.2.1 Soil/Dust Ingestion – Summer – Outdoor

$$\text{Intake}_{\text{SDINGSO}} = \frac{\text{IR}_{\text{soil}} \times \text{AF}_{\text{oral}} \times \text{ET} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

Where:

$\text{Intake}_{\text{SDINGSO}}$  = daily intake from ingestion of soil/dust – summer outdoor

$\text{IR}_{\text{soil}}$  = ingestion rate of soil

$\text{AF}_{\text{oral}}$  = oral absorption factor

$\text{ET}$  = exposure time

$\text{EF}$  = exposure frequency

$\text{ED}$  = exposure duration

$\text{CF}$  = conversion factor

$\text{BW}$  = body weight of receptor

$\text{AT}_{\text{c}}$  = averaging time carcinogen = (365 days/year) x (75 years)

$\text{AT}_{\text{nc}}$  = averaging time non-carcinogen = (exposure frequency)

x (exposure duration)

#### Units

kg/kg-day

mg/hour

unitless

hours/day

days/year

years

kg/mg

kg

days

days

$$\text{LADD/CDI}_{\text{SDINGSO}} = \text{Intake}_{\text{SDINGSO}} \times \text{C}_{\text{soil}}$$

Where:

$\text{CDI}_{\text{SDINGSO}}$  = chronic daily intake from ingestion of soil/dust – summer outdoor

$\text{LADD}_{\text{SDINGSO}}$  = lifetime average daily dose from ingestion of soil/dust – summer outdoor

$\text{Intake}_{\text{SDINGSO}}$  = daily intake from ingestion of soil/dust – summer outdoor

$\text{C}_{\text{soil}}$  = concentration of chemical in soil

#### Units

mg/kg-day

mg/kg-day

kg/kg-day

mg/kg

## B-2.2.2 Soil/Dust Dermal – Summer – Outdoor

$$\text{Intake}_{\text{SDERMSO}} = \frac{((\text{SA}_{\text{body}} \times \text{SAF}_{\text{body}}) + (\text{SA}_{\text{hand}} \times \text{SAF}_{\text{hand}})) \times \text{AF}_{\text{dermal}} \times \text{ET} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

Where:

		<u>Units</u>
$\text{Intake}_{\text{SDERMSO}}$	= daily intake from dermal contact with soil/dust – summer outdoor	mg/kg-day
$\text{SA}_{\text{body}}$	= exposed surface area in summer - body	cm <sup>2</sup>
$\text{SAF}_{\text{body}}$	= soil adherence factor - body	mg/cm <sup>2</sup> -day
$\text{SA}_{\text{hand}}$	= exposed surface area - hand	cm <sup>2</sup>
$\text{SAF}_{\text{hand}}$	= soil adherence factor - hand	mg/cm <sup>2</sup> -day
$\text{AF}_{\text{dermal}}$	= dermal absorption factor	unitless
ET	= exposure time	hours/day
EF	= exposure frequency	days/year
ED	= exposure duration	years
CF	= conversion factor	days/hour
BW	= body weight of receptor	kg
$\text{AT}_{\text{c}}$	= averaging time carcinogen = (365 days/year) x (75 years)	days
$\text{AT}_{\text{nc}}$	= averaging time non-carcinogen = (exposure frequency) x (exposure duration)	days

$$\text{LADD/CDI}_{\text{SDERMSO}} = \text{Intake}_{\text{SDERMSO}} \times \text{C}_{\text{soil}} \times \text{CF}$$

Where:

		<u>Units</u>
$\text{CDI}_{\text{SDERMSO}}$	= chronic daily intake from ingestion of soil/dust – summer outdoor	mg/kg-day
$\text{LADD}_{\text{SDERMSO}}$	= lifetime average daily dose from ingestion of soil/dust – summer outdoor	mg/kg-day
$\text{Intake}_{\text{SDERMSO}}$	= daily intake from dermal contact with soil/dust – summer outdoor	mg/kg-day
$\text{C}_{\text{soil}}$	= concentration of chemical in soil	mg/kg
CF	= conversion factor	kg/mg

### B-2.2.3 Drinking Water Ingestion

$$\text{Intake}_{\text{DWATER}} = \frac{C_{\text{dwater}} \times \text{IR}_{\text{dwater}} \times F_{\text{dwater}} \times \text{AF}_{\text{oral}} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

	<u>Units</u>
$\text{Intake}_{\text{DWATER}}$ = daily intake from the ingestion of drinking water	mg/kg-day
$C_{\text{dwater}}$ = concentration in drinking water	mg/L
$\text{IR}_{\text{dwater}}$ = ingestion rate of drinking water	L/day
$F_{\text{dwater}}$ = fraction of drinking water consumed from site	unitless
$\text{AF}_{\text{oral}}$ = oral absorption factor	unitless
$\text{EF}$ = exposure frequency	days/year
$\text{ED}$ = exposure duration	years
$\text{BW}$ = body weight of receptor	kg
$\text{AT}_{\text{c}}$ = averaging time carcinogen = (365 days/year) x (75 years)	days
$\text{AT}_{\text{nc}}$ = averaging time non-carcinogen = (365 days/year) x (exposure duration)	days

$$\text{CDI}_{\text{AHARE}} = \text{Intake}_{\text{AHARE}} \times C_{\text{AHARE}}$$

Where:

	<u>Units</u>
$\text{CDI}_{\text{AHARE}}$ = chronic daily intake from ingestion of arctic hare	mg/kg-day
$\text{Intake}_{\text{AHARE}}$ = daily intake from ingestion of arctic hare	kg/kg-day
$C_{\text{ahare}}$ = concentration of chemical in arctic hare	mg/kg

## B-2.2.4 Dermal Exposure from Surface Water

$$\text{Intake}_{\text{dermwater}} = \frac{\text{DA}_{\text{event}} \times \text{SA}_{\text{water}} \times \text{ET}_{\text{dwater}} \times \text{EF}_{\text{dwater}} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

	<u>Units</u>
$\text{Intake}_{\text{dermwater}}$ = daily intake from the dermal contact with surface water	mg/kg-day
$\text{SA}_{\text{water}}$ = exposed surface area – dermal water	cm <sup>2</sup>
$\text{ET}_{\text{dwater}}$ = exposure time – dermal water	event/day
$\text{EF}_{\text{dwater}}$ = exposure frequency – dermal water	days/year
$\text{ED}$ = exposure duration	years
$\text{BW}$ = body weight of receptor	kg
$\text{AT}_{\text{c}}$ = averaging time carcinogen = (365 days/year) x (75 years)	days
$\text{AT}_{\text{nc}}$ = averaging time non-carcinogen = (365 days/year) x (exposure duration)	days

$$\text{LADD/CDI}_{\text{DERMWATER}} = \text{Intake}_{\text{DERMWATER}} \times \text{DA}_{\text{event}}$$

Where:

	<u>Units</u>
$\text{CDI}_{\text{DERMWATER}}$ = chronic daily intake from dermal contact with water	mg/kg-day
$\text{LADD}_{\text{DERMWATER}}$ = lifetime average daily dose from dermal contact with water	mg/kg-day
$\text{Intake}_{\text{DERMWATER}}$ = daily intake from dermal contact with water	mg/kg-day
$\text{DA}_{\text{event}}$ = absorbed dose per event	mg/cm <sup>2</sup> -event

$$\text{DA}_{\text{event}} = \text{K}_{\text{p}} \times \text{C}_{\text{water}} \times \text{t}_{\text{event}}$$

Where:

	<u>Units</u>
$\text{DA}_{\text{event}}$ = absorbed dose per event	mg/cm <sup>2</sup> -event
$\text{K}_{\text{p}}$ = dermal permeability coefficient of compound in water	cm/hr
$\text{C}_{\text{water}}$ = concentration of chemical in water	mg/cm <sup>3</sup>
$\text{t}_{\text{event}}$ = event duration	hour/event



## B-2.3 Food Ingestion

### B-2.3.1 Wild Game Ingestion

$$\text{Intake}_{\text{wildgame}} = \frac{\text{IR}_{\text{wgame}} \times \text{AF}_{\text{oral}} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

	<u>Units</u>
$\text{Intake}_{\text{wildgame}}$ = daily intake from the ingestion of wild game	kg/kg-day
$\text{IR}_{\text{wgame}}$ = ingestion rate of wild game	kg/day
$\text{AF}_{\text{oral}}$ = oral absorption factor	unitless
$\text{EF}$ = exposure frequency	days/year
$\text{ED}$ = exposure duration	years
$\text{BW}$ = body weight of receptor	kg
$\text{AT}_{\text{c}}$ = averaging time carcinogen = (365 days/year) x (75 years)	days
$\text{AT}_{\text{nc}}$ = averaging time non-carcinogen = (365 days/year) x (exposure duration)	days

$$\text{LADD/CDI}_{\text{wildgame}} = \text{Intake}_{\text{wildgame}} \times \text{C}_{\text{wildgame}}$$

Where:

	<u>Units</u>
$\text{CDI}_{\text{wildgame}}$ = chronic daily intake from ingestion of wild game	mg/kg-day
$\text{LADD}_{\text{wildgame}}$ = lifetime average daily dose from ingestion of wild game	mg/kg-day
$\text{Intake}_{\text{wildgame}}$ = daily intake from ingestion of wild game	kg/kg-day
$\text{C}_{\text{wildgame}}$ = concentration of chemical in wild game	mg/kg

## B-2.4 Risk Characterization

After the various intakes are derived, the final step is calculation of the incremental lifetime cancer risks (ILCR) and non-carcinogenic hazard quotient (HQ) values for each of the pathways and receptors identified. ILCRs and HQs are then summed for individual receptors, across all applicable exposure pathways to obtain an estimate of the total individual ILCRs and HQs for specific receptors.

### B-2.4.1 Carcinogenic Chemicals

For carcinogenic chemicals, risk estimates represent the incremental probability that an individual will develop cancer over a lifetime as a result of a specific exposure to a particular chemical (US EPA, 1998a). Since carcinogenic risk estimates are over a lifetime of exposure, a composite receptor comprising five separate lifestages (i.e., infant, toddler, child, teen, adult) was used to evaluate carcinogenic intakes. A lifetime average daily dose was derived for each CoPC and each pathway. These values were then summed to get a pathway specific cancer intake.

$$ILCR_x = LADD_x \times \frac{CSF_x}{AF_{CSF}}$$

Where:

		<u>Units</u>
$ILCR_x$	= incremental lifetime cancer risk for pathway x	unitless
$LADD_x$	= chemical specific lifetime average daily dose for pathway x	mg/kg-day
$CSF_x$	= chemical specific cancer slope factor for pathway x	$(\text{mg/kg-day})^{-1}$
$AF_{CSF}$	= cancer slope factor absorption factor	unitless

$$ILCR_{TOTAL} = ILCR_{INH} + ILCR_{O/D}$$

Where:

		<u>Units</u>
$ILCR_{TOTAL}$	= total incremental lifetime cancer risk	unitless
$ILCR_{INH}$	= incremental lifetime cancer risk for inhalation exposure	unitless
$ILCR_{O/D}$	= incremental lifetime cancer risk for oral/dermal exposure	unitless

## B-2.4.2 Non-carcinogenic Chemicals

The potential for non-carcinogenic health effects resulting from exposure to a chemical is generally assessed by comparing an exposure estimate to a reference dose (RfD). A RfD is defined as a daily oral intake rate that is estimated to pose no appreciable risk of adverse health effects, even to sensitive populations (US EPA, 1998a).

$$HQ_x = \frac{CDI_x}{RfD_x \times AF_{RfD}}$$

Where:

		<u>Units</u>
HQ <sub>x</sub>	= hazard quotient for pathway x	unitless
CDI <sub>x</sub>	= chemical specific chronic daily intake for pathway x	mg/kg-day
RfD <sub>x</sub>	= chemical specific reference dose for pathway x	mg/kg-day
AF <sub>RfD</sub>	= reference dose absorption factor	unitless

The total non-carcinogenic hazard attributable to exposure to all chemicals through a single exposure pathway is known as a hazard index (HI) (US EPA, 1998a). The HI is calculated as follows:

$$HI = \sum_i HQ_x$$

Where:

		<u>Units</u>
HI	= hazard index for a specific exposure pathway	unitless
HQ <sub>x</sub>	= hazard quotient for chemical x	unitless

A receptor's total hazard is considered to be the sum of all the HI values for each of the specific exposure pathways.

$$\text{Total HI} = \sum HI$$

Where:

		<u>Units</u>
Total HI	= total hazard index from multiple exposure pathways	unitless
HI	= hazard index for a specific exposure pathway	unitless

### **B-3.0 REFERENCES**

- CCME (Canadian Council of Ministers of the Environment). 1996. A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines.
- Environment Canada. 2004. Canadian Climate Normals or Averages 1971-2000. Rocky Mountain House Weather Station, February, 2004.
- Health Canada. 2004. Federal Contaminated Site Risk Assessment in Canada. Part 1: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA), September, 2004.
- Richardson M. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment.
- US EPA (United States Environmental Protection Agency). 2004. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540/R/99/005.

# Appendix C

## **Species Inventory**

# Intake Parameters - Ermine (*Mustela erminea*)

<b>Body Weight<sup>a</sup></b>	8.85E+01	grams
	8.85E-02	kilograms
<b>Food Intake Rate<sup>b</sup></b>	9.36E+00	g dry-wt/day
	9.36E-03	kg dry-wt/day
	3.50E-02	kg wet-wt/day
<b>Water Intake Rate<sup>c</sup></b>	1.12E-02	L/day

Dietary Composition <sup>d</sup>	Fraction of Diet <sup>d</sup> (by weight)	Water Content of Dietary Item <sup>e</sup>	Dry Solid Content of Dietary Item	Fraction of Dietary Composition that is Dry Solid	Food Intake Rate (kg wet-wt/day)	Total Soil/Sediment Fraction of Diet <sup>f</sup>	Fraction of Diet that is Soil	Fraction of Diet that is Sediment
Terrestrial (soils):								
plants								
invertebrates	2.50E-02	6.50E-01	3.50E-01	8.75E-03	8.75E-04			
mammals/birds/herps	9.50E-01	7.35E-01	2.65E-01	2.52E-01	3.32E-02			
Aquatic (sediments):								
plants								
invertebrates								
fish	2.50E-02	7.15E-01	2.85E-01	7.13E-03	8.75E-04			
<b>Total</b>	<b>1.00E+00</b>			<b>2.68E-01</b>	<b>3.50E-02</b>	<b>2.80E-02</b>	<b>2.73E-02</b>	<b>7.00E-04</b>

References:

<sup>a</sup>USEPA 1993: average of average male and average female body weights

<sup>b</sup>USEPA 1993: equation 3-7 (all mammals)

<sup>c</sup>USEPA 1993: equation 3-17 (all mammals)

<sup>d</sup>USEPA 1993; Loso 1999

<sup>e</sup>USEPA 1993: Table 4-1 (grasshoppers, crickets, beetles; mammals, birds, amphibians; aquatic vertebrates)

<sup>f</sup>Table 3.1; Beyer et al. 1994 (red fox)

# Intake Parameters - Arctic Hare (*Lepus arcticus*)

<b>Body Weight<sup>a</sup></b>	4.31E+03	grams
	4.31E+00	kilograms
<b>Food Intake Rate<sup>b</sup></b>	2.53E+02	g dry-wt/day
	2.53E-01	kg dry-wt/day
	6.78E-01	kg wet-wt/day
<b>Water Intake Rate<sup>c</sup></b>	3.69E-01	L/day

Dietary Composition <sup>d</sup>	Fraction of Diet <sup>d</sup> (by weight)	Water Content of Dietary Item <sup>e</sup>	Dry Solid Content of Dietary Item	Fraction of Dietary Composition that is Dry Solid	Food Intake Rate (kg wet-wt/day)	Total Soil/Sediment Fraction of Diet <sup>f</sup>	Fraction of Diet that is Soil	Fraction of Diet that is Sediment
Terrestrial (soils):								
plants	9.50E-01	6.24E-01	3.76E-01	3.57E-01	6.44E-01			
invertebrates								
mammals/birds/herps	5.00E-02	6.80E-01	3.20E-01	1.60E-02	3.39E-02			
Aquatic (sediments):								
plants								
invertebrates								
fish								
<b>Total</b>	<b>1.00E+00</b>			<b>3.73E-01</b>	<b>6.78E-01</b>	<b>6.30E-02</b>	<b>6.30E-02</b>	<b>0.00E+00</b>

References:

<sup>a</sup>Gorog 2003

<sup>b</sup>USEPA 1993: equation 3-9 (herbivores)

<sup>c</sup>USEPA 1993: equation 3-17 (all mammals)

<sup>d</sup>Gorog 2003

<sup>e</sup>USEPA 1993: Tables 4-1 (mammals) and Table 4-2 (monocots, dicot leaves, fruit)

<sup>f</sup>Table 3.1; USEPA 1993: Table 4-5 (jackrabbit)

# Intake Parameters - Arctic Fox (*Alopex lagopus*)

<b>Body Weight<sup>a</sup></b>	4.95E+03	grams
	4.95E+00	kilograms
<b>Food Intake Rate<sup>b</sup></b>	2.56E+02	g dry-wt/day
	2.56E-01	kg dry-wt/day
	7.99E-01	kg wet-wt/day
<b>Water Intake Rate<sup>c</sup></b>	4.18E-01	L/day

Dietary Composition <sup>d</sup>	Fraction of Diet <sup>d</sup> (by weight)	Water Content of Dietary Item <sup>e</sup>	Dry Solid Content of Dietary Item	Fraction of Dietary Composition that is Dry Solid	Food Intake Rate (kg wet-wt/day)	Total Soil/Sediment Fraction of Diet <sup>f</sup>	Fraction of Diet that is Soil	Fraction of Diet that is Sediment
Terrestrial (soils):								
plants	2.50E-02	7.70E-01	2.30E-01	5.75E-03	2.00E-02			
invertebrates								
mammals/birds/herps	9.75E-01	6.78E-01	3.23E-01	3.14E-01	7.79E-01			
Aquatic (sediments):								
plants								
invertebrates								
fish								
<b>Total</b>	<b>1.00E+00</b>			<b>3.20E-01</b>	<b>7.99E-01</b>	<b>2.80E-02</b>	<b>2.80E-02</b>	<b>0.00E+00</b>

References:

<sup>a</sup>USEPA 1993

<sup>b</sup>USEPA 1993: equation 3-7 (all mammals)

<sup>c</sup>USEPA 1993: equation 3-17 (all mammals)

<sup>d</sup>CWS & CWF 2005: continental tundra region (i.e., not coastal areas)

<sup>e</sup>USEPA 1993: Tables 4-1 (mammals and birds) and 4-2 (fruit)

<sup>f</sup>Table 3.1; Beyer et al. 1994 (red fox)



**Intake Parameters - Barren-ground Caribou (*Rangifer tarandus groenlandicus*)**

<b>Body Weight<sup>a</sup></b>	1.18E+05	grams
	1.18E+02	kilograms
<b>Food Intake Rate<sup>b</sup></b>	2.80E+03	g dry-wt/day
	2.80E+00	kg dry-wt/day
	6.59E+00	kg wet-wt/day
<b>Water Intake Rate<sup>c</sup></b>	7.22E+00	L/day

Dietary Composition <sup>d</sup>	Fraction of Diet <sup>d</sup> (by weight)	Water Content of Dietary Item <sup>e</sup>	Dry Solid Content of Dietary Item	Fraction of Dietary Composition that is Dry Solid	Food Intake Rate (kg wet-wt/day)	Total Soil/Sediment Fraction of Diet <sup>f</sup>	Fraction of Diet that is Soil	Fraction of Diet that is Sediment
Terrestrial (soils):								
plants	1.00E+00	5.75E-01	4.25E-01	4.25E-01	6.59E+00			
invertebrates								
mammals/birds/herps								
Aquatic (sediments):								
plants								
invertebrates								
fish								
<b>Total</b>	<b>1.00E+00</b>			<b>4.25E-01</b>	<b>6.59E+00</b>	<b>8.20E-02</b>	<b>8.20E-02</b>	<b>0.00E+00</b>

References:

<sup>a</sup>CWS & CWF 2005

<sup>b</sup>USEPA 1993: equation 3-9 (herbivores)

<sup>c</sup>USEPA 1993: equation 3-17 (all mammals)

<sup>d</sup>CWS & CWF 2005

<sup>e</sup>USEPA 1993: Table 4-2 (monocots, dicot leaves)

<sup>f</sup>Table 3.1; Beyer et al. 1994 (Canada Goose)

# Intake Parameters - Rock Ptarmigan (*Lagopus mutus*)

<b>Body Weight<sup>a</sup></b>	4.88E+02	grams
	4.88E-01	kilograms
<b>Food Intake Rate<sup>b</sup></b>	7.67E+01	g dry-wt/day
	7.67E-02	kg dry-wt/day
	1.87E-01	kg wet-wt/day
<b>Water Intake Rate<sup>c</sup></b>	3.65E-02	L/day

Dietary Composition <sup>d</sup>	Fraction of Diet <sup>d</sup> (by weight)	Water Content of Dietary Item <sup>e</sup>	Dry Solid Content of Dietary Item	Fraction of Dietary Composition that is Dry Solid	Food Intake Rate (kg wet-wt/day)	Total Soil/Sediment Fraction of Diet <sup>f</sup>	Fraction of Diet that is Soil	Fraction of Diet that is Sediment
Terrestrial (soils):								
plants	7.50E-01	5.71E-01	4.29E-01	3.22E-01	1.41E-01			
invertebrates	2.50E-01	6.50E-01	3.50E-01	8.75E-02	4.68E-02			
mammals/birds/herps								
Aquatic (sediments):								
plants								
invertebrates								
fish								
<b>Total</b>	<b>1.00E+00</b>			<b>4.09E-01</b>	<b>1.87E-01</b>	<b>9.30E-02</b>	<b>9.30E-02</b>	<b>0.00E+00</b>

References:

<sup>a</sup>CWS & CWF 2005: average of willow and white-tailed ptarmigan body weights (rock ptarmigans are intermediate in size)

<sup>b</sup>USEPA 1993: equation 3-4 (passerines); although ptarmigan are not passerines, they eat similar low quality diets

<sup>c</sup>USEPA 1993: equation 3-15 (all birds)

<sup>d</sup>CWS & CWF 2005

<sup>e</sup>USEPA 1993: Tables 4-1 (grasshoppers, crickets, beetles) and 4-2 (dicot leaves and seeds, fruit)

<sup>f</sup>Table 3.1; Beyer et al. 1994 (Wild Turkey)

# Intake Parameters - Snowy Owl (*Nyctea scandiaca*)

<b>Body Weight<sup>a</sup></b>	2.05E+03	grams
	2.05E+00	kilograms
<b>Food Intake Rate<sup>b</sup></b>	9.28E+01	g dry-wt/day
	9.28E-02	kg dry-wt/day
	2.89E-01	kg wet-wt/day
<b>Water Intake Rate<sup>c</sup></b>	9.54E-02	L/day

Dietary Composition <sup>d</sup>	Fraction of Diet <sup>d</sup> (by weight)	Water Content of Dietary Item <sup>e</sup>	Dry Solid Content of Dietary Item	Fraction of Dietary Composition that is Dry Solid	Food Intake Rate (kg wet-wt/day)	Total Soil/Sediment Fraction of Diet <sup>f</sup>	Fraction of Diet that is Soil	Fraction of Diet that is Sediment
Terrestrial (soils):								
plants								
invertebrates								
mammals/birds/herps	9.50E-01	6.78E-01	3.23E-01	3.06E-01	2.75E-01			
Aquatic (sediments):								
plants								
invertebrates								
fish	5.00E-02	7.15E-01	2.85E-01	1.43E-02	1.45E-02			
<b>Total</b>	<b>1.00E+00</b>			<b>3.21E-01</b>	<b>2.89E-01</b>	<b>2.80E-02</b>	<b>2.66E-02</b>	<b>1.40E-03</b>

## References:

<sup>a</sup>CWS & CWF 2005: average of average adult body weights

<sup>b</sup>USEPA 1993: equation 3-3 (all birds)

<sup>c</sup>USEPA 1993: equation 3-15 (all birds)

<sup>d</sup>CWS & CWF 2005; Atkinson and Kirschbaum 2002

<sup>e</sup>USEPA 1993: Table 4-1 (mammals, birds; aquatic vertebrates)

<sup>f</sup>Table 3.1; Beyer et al. 1994 (red fox)

# Appendix D

## ERA Model Equations, Inputs and Outputs

**Jacques Whitford's Ecological Risk Assessment Model (Version 2.1)**

**Intake Parameters for the Snowy Owl**

<b>Receptor Name</b>	Snowy Owl	
<b>Name of Area</b>	CAM-D	
<b>Receptor Type</b>	1	(1-Bird, 2-Mammal)
<b>Small Mammal Type</b>	1	(1-General, 2-Herbivore, 3-Insectivore) Default value should be 1
<b>Benthic Invertebrates, Fish and Aquatic Plants based on Sediment or Surface Water Uptake</b>	1	(1-Sediment, 2-Surface Water) Default value should be 1
<b>General Parameters</b>		
Body weight	2.05	kg
Food intake rate	0.289	kg wet-wt/day
Water intake rate	0.095	L/day
<b>Ingestion of Soil</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction diet that is dry solid	0.321	
Fraction of food intake rate	0.027	
Ingestion rate	0.002504763	kg dry-wt/day
Fraction from site	0.0655	
Intake factor (IFing-sl)	8.00302E-05	kg/kg-day
<b>Ingestion of Terrestrial Plants</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-tp)	0	kg/kg-day
<b>Ingestion of Terrestrial Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-ti)	0	kg/kg-day
<b>Ingestion of Terrestrial Mammals/Birds</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.95	
Ingestion rate	0.27455	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-tm)	0.008772207	kg/kg-day
<b>Ingestion of Surface Water</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Ingestion rate	0.095	L/day
Fraction from site	0.0655	
Intake factor (IFing-sw)	0.003035366	L/kg-day
<b>Ingestion of Sediment</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction diet that is dry solid	0.321	
Fraction of food intake rate	0.001	
Ingestion rate	0.000092769	kg dry-wt/day
Fraction from site	0.0655	
Intake factor (IFing-sed)	2.96408E-06	kg/kg-day
<b>Ingestion of Aquatic Plants</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-ap)	0	kg/kg-day
<b>Ingestion of Benthic Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-ai)	0	kg/kg-day
<b>Ingestion of Fish</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.05	
Ingestion rate	0.01445	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-fsh)	0.000461695	kg/kg-day

**Jacques Whitford's Ecological Risk Assessment Model (Version 2.1)**

**Intake Parameters for the Rock Ptarmigan**

<b>Receptor Name</b>	Rock Ptarmigan	
<b>Name of Area</b>	CAM-D	
<b>Receptor Type</b>	1	(1-Bird, 2-Mammal)
<b>Small Mammal Type</b>	1	(1-General, 2-Herbivore, 3-Insectivore) Default value should be 1
<b>Benthic Invertebrates, Fish and Aquatic Plants based on Sediment or Surface Water Uptake</b>	1	(1-Sediment, 2-Surface Water) Default value should be 1
<b>General Parameters</b>		
Body weight	0.4875	kg
Food intake rate	0.187358246	kg wet-wt/day
Water intake rate	0.036458182	L/day
<b>Ingestion of Soil</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction diet that is dry solid	0.40925	
Fraction of food intake rate	0.093	
Ingestion rate	0.007130902	kg dry-wt/day
Fraction from site	1	
Intake factor (IFing-sl)	0.014627491	kg/kg-day
<b>Ingestion of Terrestrial Plants</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.75	
Ingestion rate	0.140518684	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-tp)	0.288243455	kg/kg-day
<b>Ingestion of Terrestrial Invertebrates</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.25	
Ingestion rate	0.046839561	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-ti)	0.096081152	kg/kg-day
<b>Ingestion of Terrestrial Mammals/Birds</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-tm)	0	kg/kg-day
<b>Ingestion of Surface Water</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Ingestion rate	0.036458182	L/day
Fraction from site	1	
Intake factor (IFing-sw)	0.074786014	L/kg-day
<b>Ingestion of Sediment</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction diet that is dry solid	0	
Fraction of food intake rate	0	
Ingestion rate	0	kg dry-wt/day
Fraction from site	1	
Intake factor (IFing-sed)	0	kg/kg-day
<b>Ingestion of Aquatic Plants</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-ap)	0	kg/kg-day
<b>Ingestion of Benthic Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-ai)	0	kg/kg-day
<b>Ingestion of Fish</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-fsh)	0	kg/kg-day

Jacques Whitford's Ecological Risk Assessment Model (Version 2.1)

**Intake Parameters for the Collared Lemming**

<b>Receptor Name</b>	Collared Lemming	
<b>Name of Area</b>	CAM-D	
<b>Receptor Type</b>	2	(1-Bird, 2-Mammal)
<b>Small Mammal Type</b>	1	(1-General, 2-Herbivore, 3-Insectivore) Default value should be 1
<b>Benthic Invertebrates, Fish and Aquatic Plants based on Sediment or Surface Water Uptake</b>	1	(1-Sediment, 2-Surface Water) Default value should be 1
<b>General Parameters</b>		
Body weight	0.085	kg
Food intake rate	0.02	kg wet-wt/day
Water intake rate	0.011	L/day
<b>Ingestion of Soil</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction diet that is dry solid	0.376	
Fraction of food intake rate	0.063	
Ingestion rate	0.00047376	kg dry-wt/day
Fraction from site	1	
Intake factor (IFing-sl)	0.005573647	kg/kg-day
<b>Ingestion of Terrestrial Plants</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	1	
Ingestion rate	0.02	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-tp)	0.235294118	kg/kg-day
<b>Ingestion of Terrestrial Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-ti)	0	kg/kg-day
<b>Ingestion of Terrestrial Mammals/Birds</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-tm)	0	kg/kg-day
<b>Ingestion of Surface Water</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Ingestion rate	0.011	L/day
Fraction from site	1	
Intake factor (IFing-sw)	0.129411765	L/kg-day
<b>Ingestion of Sediment</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction diet that is dry solid	0	
Fraction of food intake rate	0	
Ingestion rate	0	kg dry-wt/day
Fraction from site	1	
Intake factor (IFing-sed)	0	kg/kg-day
<b>Ingestion of Aquatic Plants</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-ap)	0	kg/kg-day
<b>Ingestion of Benthic Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-ai)	0	kg/kg-day
<b>Ingestion of Fish</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-fsh)	0	kg/kg-day

**Jacques Whitford's Ecological Risk Assessment Model (Version 2.1)**

**Intake Parameters for the Ermine**

<b>Receptor Name</b>	Ermine	
<b>Name of Area</b>	CAM-D	
<b>Receptor Type</b>	2	(1-Bird, 2-Mammal)
<b>Small Mammal Type</b>	1	(1-General, 2-Herbivore, 3-Insectivore) Default value should be 1
<b>Benthic Invertebrates, Fish and Aquatic Plants based on Sediment or Surface Water Uptake</b>	1	(1-Sediment, 2-Surface Water) Default value should be 1
<b>General Parameters</b>		
Body weight	0.089	kg
Food intake rate	0.035	kg wet-wt/day
Water intake rate	0.011	L/day
<b>Ingestion of Soil</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction diet that is dry solid	0.268	
Fraction of food intake rate	0.027	
Ingestion rate	0.00025326	kg dry-wt/day
Fraction from site	1	
Intake factor (IFing-sl)	0.002845618	kg/kg-day
<b>Ingestion of Terrestrial Plants</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	1	
Intake factor (IFing-tp)	0	kg/kg-day
<b>Ingestion of Terrestrial Invertebrates</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.025	
Ingestion rate	0.000875	kg wet-wt/day
Fraction from site	0.454	
Intake factor (IFing-ti)	0.004463483	kg/kg-day
<b>Ingestion of Terrestrial Mammals/Birds</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.95	
Ingestion rate	0.03325	kg wet-wt/day
Fraction from site	0.454	
Intake factor (IFing-tm)	0.16961236	kg/kg-day
<b>Ingestion of Surface Water</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Ingestion rate	0.011	L/day
Fraction from site	0.454	
Intake factor (IFing-sw)	0.05611236	L/kg-day
<b>Ingestion of Sediment</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction diet that is dry solid	0	
Fraction of food intake rate	0	
Ingestion rate	0	kg dry-wt/day
Fraction from site	0.454	
Intake factor (IFing-sed)	0	kg/kg-day
<b>Ingestion of Aquatic Plants</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.454	
Intake factor (IFing-ap)	0	kg/kg-day
<b>Ingestion of Benthic Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.454	
Intake factor (IFing-ai)	0	kg/kg-day
<b>Ingestion of Fish</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.025	
Ingestion rate	0.000875	kg wet-wt/day
Fraction from site	0.454	
Intake factor (IFing-fsh)	0.004463483	kg/kg-day



**Jacques Whitford's Ecological Risk Assessment Model (Version 2.1)**

**Intake Parameters for the Arctic Fox**

<b>Receptor Name</b>	Arctic Fox	
<b>Name of Area</b>	CAM-D	
<b>Receptor Type</b>	2	(1-Bird, 2-Mammal)
<b>Small Mammal Type</b>	1	(1-General, 2-Herbivore, 3-Insectivore) Default value should be 1
<b>Benthic Invertebrates, Fish and Aquatic Plants based on Sediment or Surface Water Uptake</b>	1	(1-Sediment, 2-Surface Water) Default value should be 1
<b>General Parameters</b>		
Body weight	4.95	kg
Food intake rate	0.799	kg wet-wt/day
Water intake rate	0.418	L/day
<b>Ingestion of Soil</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction diet that is dry solid	0.32	
Fraction of food intake rate	0.028	
Ingestion rate	0.00715904	kg dry-wt/day
Fraction from site	0.0655	
Intake factor (IFing-sl)	9.47307E-05	kg/kg-day
<b>Ingestion of Terrestrial Plants</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.025	
Ingestion rate	0.019975	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-tp)	0.000264316	kg/kg-day
<b>Ingestion of Terrestrial Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-ti)	0	kg/kg-day
<b>Ingestion of Terrestrial Mammals/Birds</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.975	
Ingestion rate	0.779025	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-tm)	0.010308311	kg/kg-day
<b>Ingestion of Surface Water</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Ingestion rate	0.418	L/day
Fraction from site	0.0655	
Intake factor (IFing-sw)	0.005531111	L/kg-day
<b>Ingestion of Sediment</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction diet that is dry solid	0	
Fraction of food intake rate	0	
Ingestion rate	0	kg dry-wt/day
Fraction from site	0.0655	
Intake factor (IFing-sed)	0	kg/kg-day
<b>Ingestion of Aquatic Plants</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-ap)	0	kg/kg-day
<b>Ingestion of Benthic Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-ai)	0	kg/kg-day
<b>Ingestion of Fish</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-fsh)	0	kg/kg-day

**Jacques Whitford's Ecological Risk Assessment Model (Version 2.1)**

**Intake Parameters for the Arctic Hare**

<b>Receptor Name</b>	Arctic Hare	
<b>Name of Area</b>	CAM-D	
<b>Receptor Type</b>	2	(1-Bird, 2-Mammal)
<b>Small Mammal Type</b>	1	(1-General, 2-Herbivore, 3-Insectivore) Default value should be 1
<b>Benthic Invertebrates, Fish and Aquatic Plants based on Sediment or Surface Water Uptake</b>	1	(1-Sediment, 2-Surface Water) Default value should be 1
<b>General Parameters</b>		
Body weight	4.31	kg
Food intake rate	0.678	kg wet-wt/day
Water intake rate	0.369	L/day
<b>Ingestion of Soil</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction diet that is dry solid	0.373	
Fraction of food intake rate	0.063	
Ingestion rate	0.015932322	kg dry-wt/day
Fraction from site	0.6486	
Intake factor (IFing-sl)	0.002397611	kg/kg-day
<b>Ingestion of Terrestrial Plants</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.95	
Ingestion rate	0.6441	kg wet-wt/day
Fraction from site	0.6486	
Intake factor (IFing-tp)	0.096928831	kg/kg-day
<b>Ingestion of Terrestrial Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.6486	
Intake factor (IFing-ti)	0	kg/kg-day
<b>Ingestion of Terrestrial Mammals/Birds</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	0.05	
Ingestion rate	0.0339	kg wet-wt/day
Fraction from site	0.6486	
Intake factor (IFing-tm)	0.005101517	kg/kg-day
<b>Ingestion of Surface Water</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Ingestion rate	0.369	L/day
Fraction from site	0.6486	
Intake factor (IFing-sw)	0.055529791	L/kg-day
<b>Ingestion of Sediment</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction diet that is dry solid	0	
Fraction of food intake rate	0	
Ingestion rate	0	kg dry-wt/day
Fraction from site	0.6486	
Intake factor (IFing-sed)	0	kg/kg-day
<b>Ingestion of Aquatic Plants</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.6486	
Intake factor (IFing-ap)	0	kg/kg-day
<b>Ingestion of Benthic Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.6486	
Intake factor (IFing-ai)	0	kg/kg-day
<b>Ingestion of Fish</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.6486	
Intake factor (IFing-fsh)	0	kg/kg-day

**Jacques Whitford's Ecological Risk Assessment Model (Version 2.1)**  
**Intake Parameters for the Barren Ground Caribou**

<b>Receptor Name</b>	Barren-ground Caribou	
<b>Name of Area</b>	CAM-D	
<b>Receptor Type</b>	2	(1-Bird, 2-Mammal)
<b>Small Mammal Type</b>	1	(1-General, 2-Herbivore, 3-Insectivore) Default value should be 1
<b>Benthic Invertebrates, Fish and Aquatic Plants based on Sediment or Surface Water Uptake</b>	1	(1-Sediment, 2-Surface Water) Default value should be 1
<b>General Parameters</b>		
Body weight	117.5	kg
Food intake rate	6.587	kg wet-wt/day
Water intake rate	7.222	L/day
<b>Ingestion of Soil</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction diet that is dry solid	0.425	
Fraction of food intake rate	0.082	
Ingestion rate	0.22955695	kg dry-wt/day
Fraction from site	0.0655	
Intake factor (IFing-sl)	0.000127966	kg/kg-day
<b>Ingestion of Terrestrial Plants</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Fraction of food intake rate	1	
Ingestion rate	6.587	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-tp)	0.003671902	kg/kg-day
<b>Ingestion of Terrestrial Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-ti)	0	kg/kg-day
<b>Ingestion of Terrestrial Mammals/Birds</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-tm)	0	kg/kg-day
<b>Ingestion of Surface Water</b>		
Applicable pathway?	1	(0 = no, 1 = yes)
Ingestion rate	7.222	L/day
Fraction from site	0.0655	
Intake factor (IFing-sw)	0.004025881	L/kg-day
<b>Ingestion of Sediment</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction diet that is dry solid	0	
Fraction of food intake rate	0	
Ingestion rate	0	kg dry-wt/day

Fraction from site	0.0655	
Intake factor (IFing-sed)	0	kg/kg-day
<b>Ingestion of Aquatic Plants</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-ap)	0	kg/kg-day
<b>Ingestion of Benthic Invertebrates</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-ai)	0	kg/kg-day
<b>Ingestion of Fish</b>		
Applicable pathway?	0	(0 = no, 1 = yes)
Fraction of food intake rate	0	
Ingestion rate	0	kg wet-wt/day
Fraction from site	0.0655	
Intake factor (IFing-fsh)	0	kg/kg-day

Reference Toxicity Doses for Bird Species Exposed to Constituents fo Concern from Radio Island

Constituent	Test Species	Test Species Body Weight (kg wet)	Body Weight Reference	Effect	Reference	Endpoint	Daily Dose (mg/kg-day)	Total Uncertainty Factor (a)	Chronic LOAEL- Test Species (b) (mg/kg-day)	Receptor Species	Body Weight Scaling Factor	Reference Toxicity Dose (mg/kg-day)
TPH - CCME CWS												
Aliph>C06-C08 - F1	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	75	1	7.50E+01	Snowy Owl	1.154385259	8.66E+01
Aliph>C08-C10 - F1	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	75	1	7.50E+01	Snowy Owl	1.154385259	8.66E+01
Arom>C08-C10 - F1	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	25	1	2.50E+01	Snowy Owl	1.154385259	2.89E+01
F1 - Total							--		--			--
Aliph>C10-C12 - F2	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	75	1	7.50E+01	Snowy Owl	1.154385259	8.66E+01
Aliph>C12-C16 - F2	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	75	1	7.50E+01	Snowy Owl	1.154385259	8.66E+01
Arom>C10-C12 - F2	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	25	1	2.50E+01	Snowy Owl	1.154385259	2.89E+01
Arom>C12-C16 - F2	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	25	1	2.50E+01	Snowy Owl	1.154385259	2.89E+01
F2 - Total							--		--			--
Aliph>C16-C21 - F3	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	100	1	1.00E+02	Snowy Owl	1.154385259	1.15E+02
Aliph>C21-C34 - F3	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	100	1	1.00E+02	Snowy Owl	1.154385259	1.15E+02
Arom>C16-C21 - F3	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	50	1	5.00E+01	Snowy Owl	1.154385259	5.77E+01
Arom>C21-C34 - F3	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	50	1	5.00E+01	Snowy Owl	1.154385259	5.77E+01
F3 - Total							--		--			--
Aliph>C34-C50 - F4	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	150	1	1.50E+02	Snowy Owl	1.154385259	1.73E+02
Arom>C34-C50 - F4	Mallard Duck	1	Heinz et al. 1989	Liver hypertrophy and splenic atrophy	Adapted from Szaro et al. (1978)	chronic LOAEL	20	1	2.00E+01	Snowy Owl	1.154385259	2.31E+01
F4 - Total							--		--			--
PCBs												
Aroclor 1254 (Total PCBs)	Ring-necked Pheasant	1	USEPA (1993e)	reproduction	Dahlgren et al. (1972), Sample et al. (1996)	chronic LOAEL	1.80E+00	1	1.80E+00	Snowy Owl	1.154385259	2.08E+00
Inorganics												
Antimony	Mouse	0.03	USEPA (1988a)	lifespan, longevity	Schroeder et al. (1968), (Sample et al. (1996)	chronic LOAEL	1.25E+00	5	2.50E-01	Snowy Owl	2.327697399	5.82E-01
Arsenic	Mallard duck	1	Heinz et al. (1989)	mortality	USFWS (1964), Sample et al. (1996)	chronic LOAEL	12.84	1	12.84	Snowy Owl	1.154385259	1.48E+01
Barium	Chicken (chicks)	0.121	EPA (1988a)	mortality	Johnson et al. 1960, Sample et al. (1996)	subchronic LOAEL	4.17E+02	5	8.34E+01	Snowy Owl	1.761139258	1.47E+02
Beryllium	Rat	0.35	USEPA (1988a)	longevity, weight loss (sub lethal)	Schroeder and Mitchner (1975), Sample et al. (1996)	chronic LOAEL	1.32	1	1.32E+00	Snowy Owl	1.424089103	1.88E+00
Boron	Mallard duck	1	Heinz et al. (1989)	reproduction	Smith & Anders (1989), Sample et al. (1996)	chronic LOAEL	100	1	100	Snowy Owl	1.154385259	1.15E+02
Cadmium	Mallard duck	1	Heinz et al. (1989)	reproduction	White & Finley (1978), Sample et al. (1996)	chronic LOAEL	20	1	20	Snowy Owl	1.154385259	2.31E+01
Chromium (Total)	Black duck	1.25	Dunning (1984)	reproduction	Haseltine et al. (unpubl. data), Sample et al. (1996)	chronic LOAEL	5	1	5	Snowy Owl	1.103999228	5.52E+00
Cobalt	Rat	0.35	USEPA (1988a)	reproduction	Mollenhauer et al. (1985), ATSDR (2004)	chronic NOAEL	13.25	5	2.65	Snowy Owl	1.424089103	3.77E+00
Copper	Chicken (chicks)	0.121	EPA (1988a)	growth, mortality	Mehring et al. (1960), Sample et al. (1996)	chronic LOAEL	61.7	1	61.7	Snowy Owl	1.761139258	1.09E+02
Lead	Japanese quail	0.15	Vos et al.(1971)	reproduction	Edens et al. (1976), Sample et al. (1996)	chronic LOAEL	11.3	1	11.3	Snowy Owl	1.687067738	1.91E+01
Mercury - Inorganic	Japanese quail	0.15	Vos et al. (1971)	reproduction	Hill & Schaffner (1976), Sample et al. (1996)	chronic LOAEL	0.9	1	0.9	Snowy Owl	1.687067738	1.52E+00
Nickel	Mallard duck	1	Heinz et al. (1989)	mortality, growth, behavior	Cain & Pafford (1981), Sample et al. (1996)	chronic LOAEL	107	1	107	Snowy Owl	1.154385259	1.24E+02
Selenium	Mallard Duck	1	Heinz et al. (1989)	reproduction	Heinz et al. (1987), Sample et al. (1996)	chronic LOAEL	1.00E+00	1	1.00E+00	Snowy Owl	1.154385259	1.15E+00
Silver	Mouse	0.03	USEPA (1988a)	reproduction, hypoactivity	Rungby and Danscher (1984), ATSDR (1990)	chronic LOAEL	1.81E+01	5	3.62E+00	Snowy Owl	2.327697399	8.43E+00
Tin	Japanese quail	0.15	Vos et al. (1971)	reproduction	Schlatterer et al. (1993), Sample et al. (1996)	chronic LOAEL	1.69E+01	1	1.69E+01	Snowy Owl	1.687067738	2.85E+01
Vanadium	Mallard duck	1	Heinz et al. (1989)	mortality, body weight, blood chemistry	White & Dieter (1978), Sample et al. (1996)	chronic NOAEL	11.4	0.2	57	Snowy Owl	1.154385259	6.58E+01
Zinc	White Leghorn hen	1.766	Sample et al. (1996)	reproduction	Stahl et al. (1990), Sample et al. (1996)	chronic LOAEL	131	1	131	Snowy Owl	1.030273744	1.35E+02

Notes:  
(a) The following uncertainty factors are used: 5 for subchronic to chronic; 0.2 for NOAEL to LOAEL (5 for LOAEL to NOAEL); 6 for LD<sub>50</sub> or LD<sub>Lo</sub> to LOAEL; 5 for mammal to bird.  
(b) The chronic LOAEL is calculated as the Daily Dose divided by the Total Uncertainty Factor.  
NA - Not Available

Reference Toxicity Doses for Test Organisms - Mammals - Exposed to Constituents fo Concern from Radio Island

Constituent	Test Species	Test Species Body Weight (kg wet)	Body Weight Reference	Effect	Reference	Endpoint	Daily Dose (mg/kg-day)	Total Uncertainty Factor (a)	Chronic LOAEL - Test Species (b) (mg/kg-day)	Receptor Species	Body Weight Scaling Factor	Reference Toxicity Dose (mg/kg-day)
TPH - CCME CWS												
Aliph>C06-C08 - F1	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	70	1	NA	NA	NA	NA
Aliph>C08-C10 - F1	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	70	1	NA	NA	NA	NA
Arom>C08-C10 - F1	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	15	1	NA	NA	NA	NA
F1 - Total							--		NA	NA	NA	NA
Aliph>C10-C12 - F2	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	70	1	NA	NA	NA	NA
Aliph>C12-C16 - F2	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	70	1	NA	NA	NA	NA
Arom>C10-C12 - F2	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	15	1	NA	NA	NA	NA
Arom>C12-C16 - F2	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	15	1	NA	NA	NA	NA
F2 - Total							--		NA	NA	NA	NA
Aliph>C16-C21 - F3	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	140	1	NA	NA	NA	NA
Aliph>C21-C34 - F3	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	140	1	NA	NA	NA	NA
Arom>C16-C21 - F3	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	30	1	NA	NA	NA	NA
Arom>C21-C34 - F3	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	30	1	NA	NA	NA	NA
F3 - Total							--		NA	NA	NA	NA
Aliph>C34-C50 - F4	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	1300	1	NA	NA	NA	NA
Arom>C34-C50 - F4	Cow	755	Ng (2001)	Various	Weight of Evidence from ATSDR (200X), TPHCWG (1997), Hutcheson et al. (1996) and CWS (2001)	chronic LOAEL	135	1	NA	NA	NA	NA
F4 - Total							--		NA	NA	NA	NA
PCBs												
Aroclor 1254 (Total PCBs)	Mouse	0.03	USEPA (1988a)	reproduction	McCoy et al (1995), Sample (1996)	chronic LOAEL	0.68	1	NA	NA	NA	NA
Inorganics												
Antimony	Mouse	0.03	USEPA (1988a)	lifespan, longevity	Schroeder et al. (1968b), Sample et al. (1996)	chronic LOAEL	1.25	1	NA	NA	NA	NA
Arsenic	Mouse	0.03	USEPA (1988a)	reproduction	Schroeder & Mitchner (1971), Sample et al. (1996)	chronic LOAEL	1.26	1	NA	NA	NA	NA
Barium	Rat	0.35	USEPA (1988a)	growth, hypertension	Perry et al. (1983), Sample et al. (1996)	chronic NOAEL	5.1	0.2	NA	NA	NA	NA
Beryllium	Rat	0.35	USEPA (1988a)	longevity, weight loss	Schroeder & Mitchner (1975), Sample et al. (1996)	chronic LOAEL	6.6	1	NA	NA	NA	NA
Boron	Rat	0.35	USEPA (1988a)	reproduction	Weir & Fisher (1972), Sample et al. (1996)	chronic LOAEL	93.6	1	NA	NA	NA	NA
Cadmium	Rat	0.35	USEPA (1988a)	reproduction	Sutou et al. (1980b), Sample et al. (1996)	chronic LOAEL	10	1	NA	NA	NA	NA
Chromium (Total)	Mouse	0.03	USEPA (1988a)	reproduction	Elbetieha & Al-Hamood (1997), Zahid et al (1990), (2000)	chronic LOAEL	5	1	NA	NA	NA	NA
Cobalt	Rat	0.35	USEPA (1988a)	reproduction	Mollenhauer et al. (1985), ATSDR (2004)	chronic LOAEL	13.25	1	NA	NA	NA	NA
Copper	Mink	1	USEPA (1993e)	reproduction	Aulerich et al. (1982), Sample et al. (1996)	chronic LOAEL	15.14	1	NA	NA	NA	NA
Lead	Rat	0.35	USEPA (1988a)	reproduction	Azar et al. (1973), Sample et al. (1996)	chronic LOAEL	80	1	NA	NA	NA	NA
Mercury - Inorganic	Mink	1	USEPA (1993e)	reproduction	Aulerich et al. (1974), Sample (1996)	chronic NOAEL	1	0.2	NA	NA	NA	NA
Nickel	Rat	0.35	USEPA (1988a)	reproduction	Ambrose et al. (1976), Sample et al. (1996)	chronic LOAEL	80	1	NA	NA	NA	NA
Selenium	Rat	0.35	USEPA (1988a)	reproduction	Rosenfeld & Beath (1954), Sample et al. (1996)	chronic LOAEL	0.33	1	NA	NA	NA	NA
Silver	Mouse	0.03	USEPA (1988a)	hypoactivity	Rungby and Danscher (1984), ATSDR (1990)	subchronic LOAEL	18.1	5	NA	NA	NA	NA
Tin	Mouse	0.03	USEPA (1988a)	reproduction	Davis et al. (1987), Sample et al. (1996)	chronic LOAEL	35	1	NA	NA	NA	NA
Vanadium	Rat	0.35	USEPA (1988a)	reproduction	Domingo et al. (1986), Sample et al. (1996)	chronic LOAEL	2.1	1	NA	NA	NA	NA
Zinc	Rat	0.35	USEPA (1988a)	reproduction	Schlicker & Cox (1968), Sample et al. (1996)	chronic LOAEL	320	1	NA	NA	NA	NA

Notes:

(a) The following uncertainty factors are used: 5 for subchronic to chronic; 0.2 for NOAEL to LOAEL (5 for LOAEL to NOAEL); 6 for LD50 or LDLO to LOAEL.

(b) The chronic LOAEL is calculated as the Daily Dose divided by the Total Uncertainty Factor.

NA - Not Available

## Final Exposure Point Concentrations for the CAM-D Main Station

Constituent	CAS-RN	Soil Conc. (mg/kg)	Terrestrial Plant Conc. (mg/Kg)	Terrestrial Invertebrate Conc. (mg/Kg)	Terrestrial Mammal Conc. (mg/Kg)	Surface Water Conc. (mg/L)
<b>BTEX</b>						
Ethylbenzene	100414	3.20E-01	9.73E-03	3.06E-03	1.64E-05	--
Toluene	108883	3.04E-01	9.90E-03	3.32E-03	5.72E-06	--
Xylenes	1330207	2.81E+00	8.36E-02	2.65E-02	1.56E-04	--
<b>TPH - CCME CWS</b>						
	% Composition					
Aliph>C06-C08 - F1	0.55	6.79E+01	3.29E+00	5.02E+00	3.26E-02	--
Aliph>C08-C10 - F1	0.36	4.45E+01	1.34E+00	2.51E+00	1.29E-01	--
Arom>C08-C10 - F1	0.09	1.11E+01	6.64E-01	9.27E-01	2.44E-03	--
F1 - Total	1.00					
Aliph>C10-C12 - F2	0.36	6.51E+02	4.88E+00	2.81E+01	9.29E+00	--
Aliph>C12-C16 - F2	0.44	7.96E+02	3.00E+00	1.86E+01	1.32E+02	--
Arom>C10-C12 - F2	0.09	1.63E+02	7.00E+00	1.28E+01	4.59E-02	--
Arom>C12-C16 - F2	0.11	1.99E+02	7.30E+00	1.43E+01	1.02E-01	--
F2 - Total	1.00					
Aliph>C16-C21 - F3	0.56	1.99E+03	2.33E+00	6.22E+01	3.05E+02	--
Aliph>C21-C34 - F3	0.24	8.55E+02	9.97E-01	1.52E+01	1.31E+01	--
Arom>C16-C21 - F3	0.14	4.99E+02	7.03E+00	2.46E+01	5.30E-01	--
Arom>C21-C34 - F3	0.06	2.14E+02	1.87E+00	2.02E+01	1.58E+00	--
F3 - Total	1.00					
Aliph>C34-C50 - F4	0.80	1.30E+03	3.04E-01	9.75E+00	1.93E+02	--
Arom>C34-C50 - F4	0.20	3.26E+02	7.80E-01	8.72E+01	2.83E+01	--
F4 - Total	1.00					
<b>PAHs</b>						
Naphthalene		4.12E+00	7.54E+00	3.66E-01	1.15E-02	--
<b>PCBs</b>						
Aroclor 1254 (Total PCBs)	11097691	1.25E-01	6.60E-03	3.87E-02	1.84E-02	--
<b>Inorganics</b>						
Aluminum		--	--	--	--	--
Arsenic	7440382	3.00E+00	1.68E-01	8.39E-02	6.18E-03	8.53E-04
Barium	7440393	2.34E+02	4.92E+01	3.40E+00	1.77E-01	6.96E-02
Cadmium	7440439	8.17E-01	5.27E-01	1.13E+00	1.89E-01	1.62E-04
Copper	7440508	1.51E+01	1.24E+01	1.75E+00	3.65E+00	--
Iron		--	--	--	--	--
Lead	7439921	2.03E+01	7.27E+00	1.46E+00	1.31E+00	3.69E-03
Lithium		--	3.02E+00	--	7.72E-01	9.80E-03
Manganese		1.00E-15	3.38E+02	4.20E-12	6.56E-18	1.02E-01
Nickel	7440020	8.43E+00	1.39E+01	3.63E+00	6.75E-01	5.10E-03
Silver	7440224	7.20E-02	2.00E-01	2.36E-02	9.22E-05	1.46E-04
Uranium		--	--	--	9.63E-05	1.25E-02
Zinc	7440666	1.29E+02	2.97E+02	6.74E+01	4.01E+01	6.87E-02

## Final Exposure Point Concentrations for the CAM-D Barrel Dump

Constituent	CAS-RN	Soil Conc. (mg/kg)	Terrestrial Plant Conc. (mg/Kg)	Terrestrial Invertebrate Conc. (mg/Kg)	Terrestrial Mammal Conc. (mg/Kg)	Surface Water Conc. (mg/L)
<b>BTEX</b>						
Ethylbenzene	100414	1.00E-02	3.04E-04	9.56E-05	5.12E-07	--
Toluene	108883	1.29E-02	4.20E-04	1.41E-04	2.43E-07	--
Xylenes	1330207	1.00E-02	2.98E-04	9.44E-05	5.55E-07	--
<b>TPH - CCME CWS</b>	% Composition					
Aliph>C06-C08 - F1	0.55	1.05E+00	5.06E-02	7.73E-02	5.02E-04	--
Aliph>C08-C10 - F1	0.36	6.85E-01	2.06E-02	3.87E-02	1.99E-03	--
Arom>C08-C10 - F1	0.09	1.71E-01	1.02E-02	1.43E-02	3.75E-05	--
F1 - Total	1.00					
Aliph>C10-C12 - F2	0.36	2.76E+01	2.07E-01	1.19E+00	3.93E-01	--
Aliph>C12-C16 - F2	0.44	3.37E+01	1.27E-01	7.88E-01	5.60E+00	--
Arom>C10-C12 - F2	0.09	6.89E+00	2.96E-01	5.41E-01	1.94E-03	--
Arom>C12-C16 - F2	0.11	8.42E+00	3.09E-01	6.05E-01	4.32E-03	--
F2 - Total	1.00					
Aliph>C16-C21 - F3	0.56	4.68E+02	5.45E-01	1.46E+01	7.14E+01	--
Aliph>C21-C34 - F3	0.24	2.00E+02	2.34E-01	3.55E+00	3.06E+00	--
Arom>C16-C21 - F3	0.14	1.17E+02	1.65E+00	5.78E+00	1.24E-01	--
Arom>C21-C34 - F3	0.06	5.01E+01	4.40E-01	4.73E+00	3.71E-01	--
F3 - Total	1.00					
Aliph>C34-C50 - F4	0.80	3.76E+02	8.76E-02	2.81E+00	5.56E+01	--
Arom>C34-C50 - F4	0.20	9.39E+01	2.25E-01	2.51E+01	8.16E+00	--
F4 - Total	1.00					
<b>PAHs</b>						
Naphthalene		2.50E-02	4.58E-02	2.22E-03	6.97E-05	--
<b>PCBs</b>						
Aroclor 1254 (Total PCBs)	11097691	1.90E-02	1.00E-03	2.98E-03	2.80E-03	--
<b>Inorganics</b>						
Aluminum		--	--	--	--	--
Arsenic	7440382	1.02E+00	2.07E-02	3.91E-02	2.55E-03	8.53E-04
Barium	7440393	9.40E+01	1.21E+00	1.37E+00	9.34E-02	6.96E-02
Cadmium	7440439	5.88E-01	6.97E-02	8.69E-01	1.61E-01	1.62E-04
Copper	7440508	7.50E+00	6.48E-01	1.45E+00	3.30E+00	--
Iron		--	--	--	--	--
Lead	7439921	7.80E+00	1.26E-01	6.75E-01	8.56E-01	3.69E-03
Lithium		--	--	--	3.78E-03	9.80E-03
Manganese		1.00E-15	2.55E-16	4.20E-12	6.56E-18	1.02E-01
Nickel	7440020	5.32E+00	5.66E-02	4.10E+00	5.45E-01	5.10E-03
Silver	7440224	5.00E-02	3.89E-03	1.64E-02	6.40E-05	1.46E-04
Uranium		--	--	--	9.63E-05	1.25E-02
Zinc	7440666	4.82E+01	6.23E+00	4.88E+01	3.73E+01	6.87E-02



## Final Exposure Point Concentrations for the CAM-D Main Dump

Constituent	CAS-RN	Soil Conc. (mg/kg)	Terrestrial Plant Conc. (mg/Kg)	Terrestrial Invertebrate Conc. (mg/Kg)	Terrestrial Mammal Conc. (mg/Kg)	Surface Water Conc. (mg/L)
<b>PCBs</b>						
Aroclor 1254 (Total PCBs)	11097691	5.50E-02	2.90E-03	1.27E-02	8.10E-03	--
<b>Inorganics</b>						
Arsenic	7440382	1.59E+00	2.66E-02	5.37E-02	3.68E-03	8.53E-04
Barium	7440393	9.20E+01	1.19E+00	1.34E+00	9.20E-02	6.96E-02
Cadmium	7440439	6.05E-01	7.08E-02	8.89E-01	1.63E-01	1.62E-04
Copper	7440508	1.51E+01	8.53E-01	1.75E+00	3.65E+00	--
Lead	7439921	2.03E+01	2.15E-01	1.46E+00	1.31E+00	3.69E-03
Lithium		--	--	--	3.78E-03	9.80E-03
Manganese		1.00E-15	2.55E-16	4.20E-12	6.56E-18	1.02E-01
Nickel	7440020	1.16E+01	1.01E-01	3.34E+00	7.84E-01	5.10E-03
Silver	7440224	1.00E-01	7.77E-03	3.28E-02	1.28E-04	1.46E-04
Uranium		--	--	--	9.63E-05	1.25E-02
Zinc	7440666	1.03E+02	9.50E+00	6.26E+01	3.94E+01	6.87E-02

## Final Exposure Point Concentrations for the CAM-D Outfall Area

Constituent	CAS-RN	Soil Conc. (mg/kg)	Terrestrial Plant Conc. (mg/Kg)	Terrestrial Invertebrate Conc. (mg/Kg)	Terrestrial Mammal Conc. (mg/Kg)	Surface Water Conc. (mg/L)
<b>PCBs</b>						
Aroclor 1254 (Total PCBs)	11097691	6.40E-01	3.38E-02	3.57E-01	9.43E-02	--
<b>Inorganics</b>						
Arsenic	7440382	1.29E+01	8.67E-02	2.36E-01	2.04E-02	8.53E-04
Barium	7440393	8.10E+01	1.04E+00	1.18E+00	8.41E-02	6.96E-02
Cadmium	7440439	8.30E-01	8.42E-02	1.14E+00	1.90E-01	1.62E-04
Copper	7440508	7.08E+01	1.57E+00	2.63E+00	4.56E+00	--
Lead	7439921	1.22E+01	1.62E-01	9.68E-01	1.04E+00	3.69E-03
Lithium		--	--	--	3.78E-03	9.80E-03
Manganese		1.00E-15	2.55E-16	4.20E-12	6.56E-18	1.02E-01
Nickel	7440020	2.69E+01	1.91E-01	2.69E+00	1.16E+00	5.10E-03
Silver	7440224	1.00E-01	7.77E-03	3.28E-02	1.28E-04	1.46E-04
Uranium		--	--	--	9.63E-05	1.25E-02
Zinc	7440666	4.06E+02	2.03E+01	9.82E+01	4.36E+01	6.87E-02

## Final Exposure Point Concentrations for the CAM-D Simpson Lake Shoreline Area

Constituent	CAS-RN	Soil Conc. (mg/kg)	Terrestrial Plant Conc. (mg/Kg)	Terrestrial Invertebrate Conc. (mg/Kg)	Terrestrial Mammal Conc. (mg/Kg)	Surface Water Conc. (mg/L)
<b>PCBs</b>						
Aroclor 1254 (Total PCBs)	11097691	1.00E-01	5.28E-03	2.85E-02	1.47E-02	--
<b>Inorganics</b>						
Arsenic	7440382	1.00E+00	2.05E-02	3.86E-02	2.51E-03	8.53E-04
Barium	7440393	2.80E+01	3.61E-01	4.08E-01	4.00E-02	6.96E-02
Cadmium	7440439	5.00E-01	6.38E-02	7.64E-01	1.48E-01	1.62E-04
Copper	7440508	1.75E+01	9.04E-01	1.82E+00	3.73E+00	--
Lead	7439921	1.79E+02	7.30E-01	8.46E+00	3.42E+00	3.69E-03
Lithium		--	--	--	3.78E-03	9.80E-03
Manganese		1.00E-15	2.55E-16	4.20E-12	6.56E-18	1.02E-01
Nickel	7440020	5.40E+00	5.73E-02	4.08E+00	5.49E-01	5.10E-03
Silver	7440224	1.00E-01	7.77E-03	3.28E-02	1.28E-04	1.46E-04
Uranium		--	--	--	9.63E-05	1.25E-02
Zinc	7440666	5.40E+02	2.38E+01	1.08E+02	4.45E+01	6.87E-02

## Final Exposure Point Concentrations for the CAM-D Background Area

Constituent	CAS-RN	Soil Conc. (mg/kg)	Terrestrial Plant Conc. (mg/Kg)	Terrestrial Invertebrate Conc. (mg/Kg)	Terrestrial Mammal Conc. (mg/Kg)
<b>Inorganics</b>					
Arsenic	7440382	1.70E+00	2.76E-02	5.62E-02	3.88E-03
Barium	7440393	8.01E+01	1.03E+00	1.17E+00	8.35E-02
Cadmium	7440439	6.30E-02	2.06E-02	1.47E-01	5.42E-02
Copper	7440508	1.09E+01	7.51E-01	1.60E+00	3.48E+00
Lead	7439921	7.00E+00	1.18E-01	6.19E-01	8.16E-01
Nickel	7440020	1.23E+01	1.06E-01	3.29E+00	8.05E-01
Silver	7440224	7.50E-02	5.83E-03	2.46E-02	9.60E-05
Zinc	7440666	4.39E+01	5.91E+00	4.73E+01	3.70E+01

Ecological Hazard Quotients for the Rock Ptarmigan Exposed to Constituents of Interest in the CAM-D Main Area

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Invertebrate Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX										
Ethylbenzene	8.72E+01	4.68E-03	5.37E-05	2.80E-03	3.22E-05	2.94E-04	3.37E-06	--	--	8.92E-05
Toluene	9.08E+01	4.45E-03	4.90E-05	2.85E-03	3.14E-05	3.19E-04	3.52E-06	--	--	8.39E-05
Xylenes	9.01E+02	4.11E-02	4.56E-05	2.41E-02	2.67E-05	2.55E-03	2.83E-06	--	--	7.51E-05
TPH - CCME CWS										
Aliph>C06-C08 - F1	6.50E+01	9.94E-01	1.53E-02	9.47E-01	1.46E-02	4.83E-01	7.43E-03	--	--	3.73E-02
Aliph>C08-C10 - F1	6.50E+01	6.50E-01	1.00E-02	3.86E-01	5.94E-03	2.41E-01	3.71E-03	--	--	1.97E-02
Arom>C08-C10 - F1	2.17E+01	1.63E-01	7.51E-03	1.91E-01	8.84E-03	8.90E-02	4.11E-03	--	--	2.05E-02
F1 - Total										--
Aliph>C10-C12 - F2	6.50E+01	9.52E+00	1.47E-01	1.41E+00	2.16E-02	2.70E+00	4.15E-02	--	--	2.10E-01
Aliph>C12-C16 - F2	6.50E+01	1.16E+01	1.79E-01	8.66E-01	1.33E-02	1.79E+00	2.75E-02	--	--	2.20E-01
Arom>C10-C12 - F2	2.17E+01	2.38E+00	1.10E-01	2.02E+00	9.32E-02	1.23E+00	5.67E-02	--	--	2.60E-01
Arom>C12-C16 - F2	2.17E+01	2.91E+00	1.34E-01	2.11E+00	9.72E-02	1.37E+00	6.33E-02	--	--	2.95E-01
F2 - Total										--
Aliph>C16-C21 - F3	8.66E+01	2.92E+01	3.37E-01	6.70E-01	7.74E-03	5.97E+00	6.90E-02	--	--	4.13E-01
Aliph>C21-C34 - F3	8.66E+01	1.25E+01	1.44E-01	2.87E-01	3.32E-03	1.46E+00	1.68E-02	--	--	1.64E-01
Arom>C16-C21 - F3	4.33E+01	7.29E+00	1.68E-01	2.03E+00	4.68E-02	2.37E+00	5.47E-02	--	--	2.70E-01
Arom>C21-C34 - F3	4.33E+01	3.13E+00	7.22E-02	5.40E-01	1.25E-02	1.94E+00	4.47E-02	--	--	1.29E-01
F3 - Total										--
Aliph>C34-C50 - F4	1.30E+02	1.91E+01	1.47E-01	8.76E-02	6.75E-04	9.37E-01	7.21E-03	--	--	1.55E-01
Arom>C34-C50 - F4	1.73E+01	4.77E+00	2.75E-01	2.25E-01	1.30E-02	8.38E+00	4.84E-01	--	--	7.72E-01
F4 - Total										--
PAHs										
Naphthalene	6.99E+01	6.02E-02	8.62E-04	2.17E+00	3.11E-02	3.52E-02	5.04E-04	--	--	3.25E-02
PCBs										
Aroclor 1254 (Total PCBs)	1.56E+00	1.83E-03	1.17E-03	1.90E-03	1.22E-03	3.72E-03	2.38E-03	--	--	4.78E-03
Inorganics										
Arsenic	1.11E+01	4.39E-02	3.95E-03	4.85E-02	4.36E-03	8.06E-03	7.25E-04	6.38E-05	5.74E-06	9.04E-03
Barium	1.10E+02	3.42E+00	3.10E-02	1.42E+01	1.29E-01	3.27E-01	2.96E-03	5.21E-03	4.72E-05	1.63E-01
Cadmium	1.73E+01	1.20E-02	6.90E-04	1.52E-01	8.77E-03	1.08E-01	6.26E-03	1.21E-05	6.99E-07	1.57E-02
Copper	8.15E+01	2.21E-01	2.71E-03	3.56E+00	4.37E-02	1.68E-01	2.06E-03	--	--	4.85E-02
Lead	1.43E+01	2.98E-01	2.08E-02	2.10E+00	1.47E-01	1.41E-01	9.83E-03	2.76E-04	1.93E-05	1.77E-01
Lithium	4.02E+00	--	--	8.70E-01	2.17E-01	--	--	7.33E-04	1.82E-04	2.17E-01
Manganese	7.16E+03	1.46E-22	2.04E-26	9.74E+01	1.36E-02	1.57E-16	2.19E-20	7.63E-03	1.07E-06	1.36E-02
Nickel	9.27E+01	1.23E-01	1.33E-03	4.00E+00	4.31E-02	3.49E-01	3.77E-03	3.81E-04	4.12E-06	4.82E-02
Silver	6.32E+00	1.05E-03	1.67E-04	5.76E-02	9.12E-03	2.27E-03	3.59E-04	1.09E-05	1.73E-06	9.65E-03
Uranium	1.33E+02	--	--	--	--	--	--	9.35E-04	7.05E-06	7.05E-06
Zinc	1.01E+02	1.89E+00	1.86E-02	8.55E+01	8.44E-01	6.47E+00	6.39E-02	5.14E-03	5.07E-05	9.27E-01

Ecological Hazard Quotients for the Ermine Exposed to Constituents of Interest in the CAM-D Main Area

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Invertebrate Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX										
Ethylbenzene	4.43E+02	9.11E-04	2.06E-06	1.37E-05	3.08E-08	2.78E-06	6.27E-09	--	--	2.09E-06
Toluene	2.44E+02	8.65E-04	3.55E-06	1.48E-05	6.09E-08	9.71E-07	3.98E-09	--	--	3.62E-06
Xylenes	2.42E+03	7.99E-03	3.31E-06	1.18E-04	4.90E-08	2.64E-05	1.09E-08	--	--	3.37E-06
TPH - CCME CWS										
Aliph>C06-C08 - F1	1.20E+02	1.93E-01	1.61E-03	2.24E-02	1.86E-04	5.53E-03	4.59E-05	--	--	1.84E-03
Aliph>C08-C10 - F1	1.20E+02	1.27E-01	1.05E-03	1.12E-02	9.31E-05	2.20E-02	1.82E-04	--	--	1.33E-03
Arom>C08-C10 - F1	2.58E+01	3.16E-02	1.23E-03	4.14E-03	1.60E-04	4.13E-04	1.60E-05	--	--	1.40E-03
F1 - Total										--
Aliph>C10-C12 - F2	1.20E+02	1.85E+00	1.54E-02	1.25E-01	1.04E-03	1.58E+00	1.31E-02	--	--	2.95E-02
Aliph>C12-C16 - F2	1.20E+02	2.26E+00	1.88E-02	8.31E-02	6.90E-04	2.24E+01	1.86E-01	--	--	2.06E-01
Arom>C10-C12 - F2	2.58E+01	4.63E-01	1.79E-02	5.70E-02	2.21E-03	7.78E-03	3.01E-04	--	--	2.05E-02
Arom>C12-C16 - F2	2.58E+01	5.66E-01	2.19E-02	6.37E-02	2.47E-03	1.73E-02	6.71E-04	--	--	2.51E-02
F2 - Total										--
Aliph>C16-C21 - F3	2.41E+02	5.67E+00	2.36E-02	2.78E-01	1.15E-03	5.17E+01	2.15E-01	--	--	2.39E-01
Aliph>C21-C34 - F3	2.41E+02	2.43E+00	1.01E-02	6.77E-02	2.81E-04	2.21E+00	9.19E-03	--	--	1.96E-02
Arom>C16-C21 - F3	5.16E+01	1.42E+00	2.75E-02	1.10E-01	2.13E-03	8.99E-02	1.74E-03	--	--	3.14E-02
Arom>C21-C34 - F3	5.16E+01	6.08E-01	1.18E-02	9.00E-02	1.74E-03	2.68E-01	5.20E-03	--	--	1.87E-02
F3 - Total										--
Aliph>C34-C50 - F4	2.24E+03	3.71E+00	1.66E-03	4.35E-02	1.95E-05	3.27E+01	1.46E-02	--	--	1.63E-02
Arom>C34-C50 - F4	2.32E+02	9.28E-01	3.99E-03	3.89E-01	1.68E-03	4.80E+00	2.07E-02	--	--	2.63E-02
F4 - Total										--
PAHs										
Naphthalene	1.87E+02	1.17E-02	6.25E-05	1.63E-03	8.73E-06	1.95E-03	1.04E-05	--	--	8.17E-05
PCBs										
Aroclor 1254 (Total PCBs)	6.37E-01	3.56E-04	5.58E-04	1.73E-04	2.71E-04	3.12E-03	4.90E-03	--	--	5.73E-03
Inorganics										
Arsenic	1.18E+00	8.54E-03	7.23E-03	3.75E-04	3.17E-04	1.05E-03	8.88E-04	4.79E-05	4.05E-05	8.48E-03
Barium	2.77E+01	6.65E-01	2.40E-02	1.52E-02	5.48E-04	2.99E-02	1.08E-03	3.91E-03	1.41E-04	2.58E-02
Cadmium	1.09E+01	2.32E-03	2.14E-04	5.04E-03	4.64E-04	3.20E-02	2.95E-03	9.09E-06	8.37E-07	3.62E-03
Copper	1.75E+01	4.30E-02	2.46E-03	7.81E-03	4.46E-04	6.19E-01	3.54E-02	--	--	3.83E-02
Lead	8.69E+01	5.79E-02	6.66E-04	6.53E-03	7.52E-05	2.22E-01	2.56E-03	2.07E-04	2.38E-06	3.30E-03
Lithium	2.04E+01	--	--	--	--	1.31E-01	6.42E-03	5.50E-04	2.69E-05	6.45E-03
Manganese	3.08E+02	2.85E-18	9.23E-21	1.87E-14	6.07E-17	1.11E-18	3.61E-21	5.72E-03	1.86E-05	1.86E-05
Nickel	8.69E+01	2.40E-02	2.76E-04	1.62E-02	1.87E-04	1.15E-01	1.32E-03	2.86E-04	3.30E-06	1.78E-03
Silver	3.39E+00	2.05E-04	6.04E-05	1.05E-04	3.11E-05	1.56E-05	4.61E-06	8.19E-06	2.42E-06	9.85E-05
Uranium	5.74E+00	--	--	--	--	1.63E-05	2.84E-06	7.01E-04	1.22E-04	1.25E-04
Zinc	3.47E+02	3.67E-01	1.06E-03	3.01E-01	8.66E-04	6.80E+00	1.96E-02	3.85E-03	1.11E-05	2.15E-02

Ecological Hazard Quotients for the Arctic Fox Exposed to Constituents of Interest in the CAM-D Main Area

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX										
Ethylbenzene	3.48E+02	3.03E-05	8.71E-08	2.57E-06	7.39E-09	1.69E-07	4.85E-10	--	--	9.50E-08
Toluene	1.91E+02	2.88E-05	1.50E-07	2.62E-06	1.37E-08	5.90E-08	3.08E-10	--	--	1.64E-07
Xylenes	1.90E+03	2.66E-04	1.40E-07	2.21E-05	1.16E-08	1.61E-06	8.46E-10	--	--	1.53E-07
TPH - CCME CWS										
Aliph>C06-C08 - F1	9.46E+01	6.44E-03	6.80E-05	8.69E-04	9.18E-06	3.36E-04	3.55E-06	--	--	8.07E-05
Aliph>C08-C10 - F1	9.46E+01	4.21E-03	4.45E-05	3.54E-04	3.74E-06	1.33E-03	1.41E-05	--	--	6.24E-05
Arom>C08-C10 - F1	2.03E+01	1.05E-03	5.19E-05	1.76E-04	8.66E-06	2.51E-05	1.24E-06	--	--	6.18E-05
F1 - Total										--
Aliph>C10-C12 - F2	9.46E+01	6.17E-02	6.52E-04	1.29E-03	1.36E-05	9.57E-02	1.01E-03	--	--	1.68E-03
Aliph>C12-C16 - F2	9.46E+01	7.54E-02	7.97E-04	7.94E-04	8.39E-06	1.36E+00	1.44E-02	--	--	1.52E-02
Arom>C10-C12 - F2	2.03E+01	1.54E-02	7.60E-04	1.85E-03	9.12E-05	4.73E-04	2.33E-05	--	--	8.75E-04
Arom>C12-C16 - F2	2.03E+01	1.88E-02	9.29E-04	1.93E-03	9.52E-05	1.05E-03	5.19E-05	--	--	1.08E-03
F2 - Total										--
Aliph>C16-C21 - F3	1.89E+02	1.89E-01	9.98E-04	6.15E-04	3.25E-06	3.14E+00	1.66E-02	--	--	1.76E-02
Aliph>C21-C34 - F3	1.89E+02	8.10E-02	4.28E-04	2.63E-04	1.39E-06	1.35E-01	7.11E-04	--	--	1.14E-03
Arom>C16-C21 - F3	4.06E+01	4.72E-02	1.16E-03	1.86E-03	4.58E-05	5.47E-03	1.35E-04	--	--	1.34E-03
Arom>C21-C34 - F3	4.06E+01	2.02E-02	4.99E-04	4.96E-04	1.22E-05	1.63E-02	4.02E-04	--	--	9.13E-04
F3 - Total										--
Aliph>C34-C50 - F4	1.76E+03	1.24E-01	7.03E-05	8.04E-05	4.57E-08	1.99E+00	1.13E-03	--	--	1.20E-03
Arom>C34-C50 - F4	1.83E+02	3.09E-02	1.69E-04	2.06E-04	1.13E-06	2.92E-01	1.60E-03	--	--	1.77E-03
F4 - Total										--
PAHs										
Naphthalene	1.47E+02	3.90E-04	2.65E-06	1.99E-03	1.35E-05	1.18E-04	8.04E-07	--	--	1.70E-05
PCBs										
Aroclor 1254 (Total PCBs)	5.01E-01	1.18E-05	2.37E-05	1.74E-06	3.49E-06	1.90E-04	3.79E-04	--	--	4.06E-04
Inorganics										
Arsenic	9.28E-01	2.84E-04	3.06E-04	4.45E-05	4.79E-05	6.37E-05	6.86E-05	4.72E-06	5.09E-06	4.28E-04
Barium	2.18E+01	2.21E-02	1.02E-03	1.30E-02	5.98E-04	1.82E-03	8.37E-05	3.85E-04	1.77E-05	1.72E-03
Cadmium	8.53E+00	7.74E-05	9.07E-06	1.39E-04	1.63E-05	1.94E-03	2.28E-04	8.96E-07	1.05E-07	2.53E-04
Copper	1.38E+01	1.43E-03	1.04E-04	3.27E-03	2.38E-04	3.76E-02	2.74E-03	--	--	3.08E-03
Lead	6.82E+01	1.93E-03	2.82E-05	1.92E-03	2.82E-05	1.35E-02	1.98E-04	2.04E-05	2.99E-07	2.54E-04
Lithium	1.60E+01	--	--	7.98E-04	4.97E-05	7.96E-03	4.97E-04	5.42E-05	3.38E-06	5.50E-04
Manganese	2.42E+02	9.47E-20	3.91E-22	8.93E-02	3.69E-04	6.76E-20	2.79E-22	5.64E-04	2.33E-06	3.71E-04
Nickel	6.82E+01	7.99E-04	1.17E-05	3.66E-03	5.37E-05	6.96E-03	1.02E-04	2.82E-05	4.13E-07	1.68E-04
Silver	2.66E+00	6.82E-06	2.56E-06	5.29E-05	1.98E-05	9.50E-07	3.57E-07	8.08E-07	3.03E-07	2.31E-05
Uranium	4.51E+00	--	--	--	--	9.93E-07	2.20E-07	6.91E-05	1.53E-05	1.55E-05
Zinc	2.73E+02	1.22E-02	4.47E-05	7.84E-02	2.87E-04	4.13E-01	1.51E-03	3.80E-04	1.39E-06	1.85E-03

Ecological Hazard Quotients for the Arctic Hare Exposed to Constituents of Interest in the CAM-D Main Area

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX										
Ethylbenzene	3.51E+02	7.67E-04	2.19E-06	9.43E-04	2.69E-06	8.36E-08	2.38E-10	--	--	4.87E-06
Toluene	1.93E+02	7.29E-04	3.78E-06	9.60E-04	4.97E-06	2.92E-08	1.51E-10	--	--	8.75E-06
Xylenes	1.92E+03	6.73E-03	3.52E-06	8.10E-03	4.23E-06	7.95E-07	4.15E-10	--	--	7.75E-06
TPH - CCME CWS										
Aliph>C06-C08 - F1	9.54E+01	1.63E-01	1.71E-03	3.19E-01	3.34E-03	1.66E-04	1.74E-06	--	--	5.05E-03
Aliph>C08-C10 - F1	9.54E+01	1.07E-01	1.12E-03	1.30E-01	1.36E-03	6.61E-04	6.92E-06	--	--	2.48E-03
Arom>C08-C10 - F1	2.05E+01	2.67E-02	1.30E-03	6.44E-02	3.15E-03	1.24E-05	6.08E-07	--	--	4.45E-03
F1 - Total										--
Aliph>C10-C12 - F2	9.54E+01	1.56E+00	1.64E-02	4.73E-01	4.95E-03	4.74E-02	4.96E-04	--	--	2.18E-02
Aliph>C12-C16 - F2	9.54E+01	1.91E+00	2.00E-02	2.91E-01	3.05E-03	6.74E-01	7.06E-03	--	--	3.01E-02
Arom>C10-C12 - F2	2.05E+01	3.90E-01	1.91E-02	6.79E-01	3.32E-02	2.34E-04	1.14E-05	--	--	5.23E-02
Arom>C12-C16 - F2	2.05E+01	4.77E-01	2.33E-02	7.08E-01	3.46E-02	5.21E-04	2.55E-05	--	--	5.80E-02
F2 - Total										--
Aliph>C16-C21 - F3	1.91E+02	4.78E+00	2.50E-02	2.25E-01	1.18E-03	1.55E+00	8.14E-03	--	--	3.44E-02
Aliph>C21-C34 - F3	1.91E+02	2.05E+00	1.07E-02	9.66E-02	5.06E-04	6.66E-02	3.49E-04	--	--	1.16E-02
Arom>C16-C21 - F3	4.09E+01	1.20E+00	2.92E-02	6.81E-01	1.67E-02	2.70E-03	6.61E-05	--	--	4.60E-02
Arom>C21-C34 - F3	4.09E+01	5.12E-01	1.25E-02	1.82E-01	4.44E-03	8.07E-03	1.97E-04	--	--	1.72E-02
F3 - Total										--
Aliph>C34-C50 - F4	1.77E+03	3.13E+00	1.76E-03	2.95E-02	1.66E-05	9.85E-01	5.56E-04	--	--	2.34E-03
Arom>C34-C50 - F4	1.84E+02	7.81E-01	4.25E-03	7.56E-02	4.11E-04	1.44E-01	7.85E-04	--	--	5.44E-03
F4 - Total										--
PAHs										
Naphthalene	1.48E+02	9.87E-03	6.65E-05	7.30E-01	4.92E-03	5.86E-05	3.95E-07	--	--	4.99E-03
PCBs										
Aroclor 1254 (Total PCBs)	5.05E-01	3.00E-04	5.94E-04	6.40E-04	1.27E-03	9.40E-05	1.86E-04	--	--	2.05E-03
Inorganics										
Arsenic	9.35E-01	7.19E-03	7.69E-03	1.63E-02	1.74E-02	3.15E-05	3.37E-05	4.74E-05	5.06E-05	2.52E-02
Barium	2.19E+01	5.60E-01	2.55E-02	4.77E+00	2.18E-01	9.01E-04	4.11E-05	3.86E-03	1.76E-04	2.43E-01
Cadmium	8.60E+00	1.96E-03	2.28E-04	5.11E-02	5.94E-03	9.62E-04	1.12E-04	9.00E-06	1.05E-06	6.28E-03
Copper	1.39E+01	3.62E-02	2.61E-03	1.20E+00	8.64E-02	1.86E-02	1.34E-03	--	--	9.04E-02
Lead	6.88E+01	4.88E-02	7.09E-04	7.05E-01	1.02E-02	6.68E-03	9.70E-05	2.05E-04	2.98E-06	1.11E-02
Lithium	1.62E+01	--	--	2.93E-01	1.81E-02	3.94E-03	2.44E-04	5.44E-04	3.37E-05	1.84E-02
Manganese	2.44E+02	2.40E-18	9.81E-21	3.28E+01	1.34E-01	3.35E-20	1.37E-22	5.66E-03	2.32E-05	1.34E-01
Nickel	6.88E+01	2.02E-02	2.94E-04	1.34E+00	1.95E-02	3.44E-03	5.01E-05	2.83E-04	4.12E-06	1.99E-02
Silver	2.69E+00	1.73E-04	6.42E-05	1.94E-02	7.21E-03	4.70E-07	1.75E-07	8.11E-06	3.02E-06	7.28E-03
Uranium	4.55E+00	--	--	--	--	4.91E-07	1.08E-07	6.94E-04	1.53E-04	1.53E-04
Zinc	2.75E+02	3.09E-01	1.12E-03	2.87E+01	1.04E-01	2.04E-01	7.43E-04	3.81E-03	1.39E-05	1.06E-01



Ecological Hazard Quotients for the Snowy Owl Exposed to Constituents of Interest in the CAM-D Main Area

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX								
Ethylbenzene	1.16E+02	2.56E-05	2.20E-07	1.44E-07	1.24E-09	--	--	2.22E-07
Toluene	1.21E+02	2.43E-05	2.01E-07	5.02E-08	4.15E-10	--	--	2.01E-07
Xylenes	1.20E+03	2.25E-04	1.87E-07	1.37E-06	1.14E-09	--	--	1.88E-07
TPH - CCME CWS								
Aliph>C06-C08 - F1	8.66E+01	5.44E-03	6.28E-05	2.86E-04	3.30E-06	--	--	6.61E-05
Aliph>C08-C10 - F1	8.66E+01	3.56E-03	4.11E-05	1.14E-03	1.31E-05	--	--	5.42E-05
Arom>C08-C10 - F1	2.89E+01	8.90E-04	3.08E-05	2.14E-05	7.41E-07	--	--	3.16E-05
F1 - Total								--
Aliph>C10-C12 - F2	8.66E+01	5.21E-02	6.02E-04	8.15E-02	9.41E-04	--	--	1.54E-03
Aliph>C12-C16 - F2	8.66E+01	6.37E-02	7.36E-04	1.16E+00	1.34E-02	--	--	1.41E-02
Arom>C10-C12 - F2	2.89E+01	1.30E-02	4.51E-04	4.02E-04	1.39E-05	--	--	4.65E-04
Arom>C12-C16 - F2	2.89E+01	1.59E-02	5.52E-04	8.96E-04	3.10E-05	--	--	5.83E-04
F2 - Total								--
Aliph>C16-C21 - F3	1.15E+02	1.60E-01	1.38E-03	2.67E+00	2.32E-02	--	--	2.45E-02
Aliph>C21-C34 - F3	1.15E+02	6.84E-02	5.92E-04	1.15E-01	9.92E-04	--	--	1.58E-03
Arom>C16-C21 - F3	5.77E+01	3.99E-02	6.91E-04	4.65E-03	8.06E-05	--	--	7.72E-04
Arom>C21-C34 - F3	5.77E+01	1.71E-02	2.96E-04	1.39E-02	2.40E-04	--	--	5.37E-04
F3 - Total								--
Aliph>C34-C50 - F4	1.73E+02	1.04E-01	6.03E-04	1.69E+00	9.78E-03	--	--	1.04E-02
Arom>C34-C50 - F4	2.31E+01	2.61E-02	1.13E-03	2.48E-01	1.08E-02	--	--	1.19E-02
F4 - Total								--
PAHs								
Naphthalene	9.31E+01	3.30E-04	3.54E-06	1.01E-04	1.08E-06	--	--	4.62E-06
PCBs								
Aroclor 1254 (Total PCBs)	2.08E+00	1.00E-05	4.81E-06	1.62E-04	7.78E-05	--	--	8.26E-05
Inorganics								
Arsenic	1.48E+01	2.40E-04	1.62E-05	5.42E-05	3.66E-06	2.59E-06	1.75E-07	2.00E-05
Barium	1.47E+02	1.87E-02	1.27E-04	1.55E-03	1.05E-05	2.11E-04	1.44E-06	1.39E-04
Cadmium	2.31E+01	6.54E-05	2.83E-06	1.65E-03	7.16E-05	4.92E-07	2.13E-08	7.45E-05
Copper	1.09E+02	1.21E-03	1.11E-05	3.20E-02	2.95E-04	--	--	3.06E-04
Lead	1.91E+01	1.63E-03	8.54E-05	1.15E-02	6.02E-04	1.12E-05	5.88E-07	6.88E-04
Lithium	5.35E+00	--	--	6.78E-03	1.27E-03	2.97E-05	5.56E-06	1.27E-03
Manganese	9.54E+03	8.00E-20	8.39E-24	5.75E-20	6.03E-24	3.10E-04	3.24E-08	3.24E-08
Nickel	1.24E+02	6.75E-04	5.46E-06	5.92E-03	4.80E-05	1.55E-05	1.25E-07	5.35E-05
Silver	8.43E+00	5.76E-06	6.84E-07	8.08E-07	9.59E-08	4.43E-07	5.26E-08	8.32E-07
Uranium	1.77E+02	--	--	8.45E-07	4.78E-09	3.79E-05	2.15E-07	2.20E-07
Zinc	1.35E+02	1.03E-02	7.64E-05	3.51E-01	2.60E-03	2.09E-04	1.55E-06	2.68E-03

Ecological Hazard Quotients for the Barren-ground Caribou Exposed to Constituents of Interest in the CAM-D Main Area

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX								
Ethylbenzene	2.88E+02	4.09E-05	1.42E-07	3.57E-05	1.24E-07	--	--	2.66E-07
Toluene	1.58E+02	3.89E-05	2.46E-07	3.64E-05	2.30E-07	--	--	4.75E-07
Xylenes	1.57E+03	3.59E-04	2.29E-07	3.07E-04	1.95E-07	--	--	4.24E-07
TPH - CCME CWS								
Aliph>C06-C08 - F1	7.83E+01	8.69E-03	1.11E-04	1.21E-02	1.54E-04	--	--	2.65E-04
Aliph>C08-C10 - F1	7.83E+01	5.69E-03	7.27E-05	4.92E-03	6.28E-05	--	--	1.36E-04
Arom>C08-C10 - F1	1.68E+01	1.42E-03	8.48E-05	2.44E-03	1.45E-04	--	--	2.30E-04
F1 - Total								--
Aliph>C10-C12 - F2	7.83E+01	8.33E-02	1.06E-03	1.79E-02	2.29E-04	--	--	1.29E-03
Aliph>C12-C16 - F2	7.83E+01	1.02E-01	1.30E-03	1.10E-02	1.41E-04	--	--	1.44E-03
Arom>C10-C12 - F2	1.68E+01	2.08E-02	1.24E-03	2.57E-02	1.53E-03	--	--	2.77E-03
Arom>C12-C16 - F2	1.68E+01	2.55E-02	1.52E-03	2.68E-02	1.60E-03	--	--	3.12E-03
F2 - Total								--
Aliph>C16-C21 - F3	1.57E+02	2.55E-01	1.63E-03	8.54E-03	5.46E-05	--	--	1.68E-03
Aliph>C21-C34 - F3	1.57E+02	1.09E-01	6.99E-04	3.66E-03	2.34E-05	--	--	7.22E-04
Arom>C16-C21 - F3	3.35E+01	6.38E-02	1.90E-03	2.58E-02	7.70E-04	--	--	2.67E-03
Arom>C21-C34 - F3	3.35E+01	2.73E-02	8.15E-04	6.88E-03	2.05E-04	--	--	1.02E-03
F3 - Total								--
Aliph>C34-C50 - F4	1.45E+03	1.67E-01	1.15E-04	1.12E-03	7.68E-07	--	--	1.16E-04
Arom>C34-C50 - F4	1.51E+02	4.17E-02	2.76E-04	2.86E-03	1.90E-05	--	--	2.95E-04
F4 - Total								--
PAHs								
Naphthalene	1.22E+02	5.27E-04	4.33E-06	2.77E-02	2.27E-04	--	--	2.32E-04
PCBs								
Aroclor 1254 (Total PCBs)	4.14E-01	1.60E-05	3.86E-05	2.42E-05	5.86E-05	--	--	9.72E-05
Inorganics								
Arsenic	7.67E-01	3.84E-04	5.01E-04	6.18E-04	8.05E-04	3.43E-06	4.48E-06	1.31E-03
Barium	1.80E+01	2.99E-02	1.66E-03	1.81E-01	1.00E-02	2.80E-04	1.56E-05	1.17E-02
Cadmium	7.05E+00	1.05E-04	1.48E-05	1.94E-03	2.74E-04	6.52E-07	9.25E-08	2.89E-04
Copper	1.14E+01	1.93E-03	1.70E-04	4.54E-02	3.99E-03	--	--	4.16E-03
Lead	5.64E+01	2.60E-03	4.61E-05	2.67E-02	4.73E-04	1.49E-05	2.63E-07	5.20E-04
Lithium	1.33E+01	--	--	1.11E-02	8.36E-04	3.95E-05	2.98E-06	8.39E-04
Manganese	2.00E+02	1.28E-19	6.39E-22	1.24E+00	6.19E-03	4.11E-04	2.05E-06	6.20E-03
Nickel	5.64E+01	1.08E-03	1.91E-05	5.09E-02	9.02E-04	2.05E-05	3.64E-07	9.22E-04
Silver	2.20E+00	9.21E-06	4.18E-06	7.34E-04	3.33E-04	5.88E-07	2.67E-07	3.38E-04
Uranium	3.73E+00	--	--	--	--	5.03E-05	1.35E-05	1.35E-05
Zinc	2.26E+02	1.65E-02	7.31E-05	1.09E+00	4.82E-03	2.77E-04	1.23E-06	4.90E-03

Ecological Hazard Quotients for the Collared Lemming Exposed to Constituents of Interest in the CAM-D Main Area

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX								
Ethylbenzene	4.44E+02	1.78E-03	4.02E-06	2.29E-03	5.16E-06	--	--	9.17E-06
Toluene	2.44E+02	1.69E-03	6.94E-06	2.33E-03	9.54E-06	--	--	1.65E-05
Xylenes	2.42E+03	1.57E-02	6.46E-06	1.97E-02	8.12E-06	--	--	1.46E-05
TPH - CCME CWS								
Aliph>C06-C08 - F1	1.21E+02	3.79E-01	3.14E-03	7.73E-01	6.40E-03	--	--	9.54E-03
Aliph>C08-C10 - F1	1.21E+02	2.48E-01	2.05E-03	3.15E-01	2.61E-03	--	--	4.66E-03
Arom>C08-C10 - F1	2.59E+01	6.20E-02	2.39E-03	1.56E-01	6.04E-03	--	--	8.43E-03
F1 - Total								--
Aliph>C10-C12 - F2	1.21E+02	3.63E+00	3.00E-02	1.15E+00	9.50E-03	--	--	3.95E-02
Aliph>C12-C16 - F2	1.21E+02	4.44E+00	3.67E-02	7.07E-01	5.85E-03	--	--	4.26E-02
Arom>C10-C12 - F2	2.59E+01	9.07E-01	3.51E-02	1.65E+00	6.36E-02	--	--	9.87E-02
Arom>C12-C16 - F2	2.59E+01	1.11E+00	4.28E-02	1.72E+00	6.64E-02	--	--	1.09E-01
F2 - Total								--
Aliph>C16-C21 - F3	2.42E+02	1.11E+01	4.60E-02	5.47E-01	2.27E-03	--	--	4.83E-02
Aliph>C21-C34 - F3	2.42E+02	4.76E+00	1.97E-02	2.34E-01	9.71E-04	--	--	2.07E-02
Arom>C16-C21 - F3	5.18E+01	2.78E+00	5.37E-02	1.65E+00	3.20E-02	--	--	8.56E-02
Arom>C21-C34 - F3	5.18E+01	1.19E+00	2.30E-02	4.41E-01	8.52E-03	--	--	3.15E-02
F3 - Total								--
Aliph>C34-C50 - F4	2.24E+03	7.27E+00	3.24E-03	7.15E-02	3.19E-05	--	--	3.27E-03
Arom>C34-C50 - F4	2.33E+02	1.82E+00	7.80E-03	1.83E-01	7.88E-04	--	--	8.59E-03
F4 - Total								--
PAHs								
Naphthalene	1.88E+02	2.30E-02	1.22E-04	1.77E+00	9.44E-03	--	--	9.56E-03
PCBs								
Aroclor 1254 (Total PCBs)	6.39E-01	6.97E-04	1.09E-03	1.55E-03	2.43E-03	--	--	3.52E-03
Inorganics								
Arsenic	1.18E+00	1.67E-02	1.41E-02	3.96E-02	3.34E-02	1.10E-04	9.33E-05	4.77E-02
Barium	2.78E+01	1.30E+00	4.69E-02	1.16E+01	4.17E-01	9.01E-03	3.24E-04	4.64E-01
Cadmium	1.09E+01	4.55E-03	4.18E-04	1.24E-01	1.14E-02	2.10E-05	1.93E-06	1.18E-02
Copper	1.76E+01	8.42E-02	4.80E-03	2.91E+00	1.66E-01	--	--	1.71E-01
Lead	8.71E+01	1.13E-01	1.30E-03	1.71E+00	1.97E-02	4.78E-04	5.48E-06	2.10E-02
Lithium	2.05E+01	--	--	7.10E-01	3.47E-02	1.27E-03	6.20E-05	3.48E-02
Manganese	3.09E+02	5.57E-18	1.80E-20	7.95E+01	2.57E-01	1.32E-02	4.27E-05	2.57E-01
Nickel	8.71E+01	4.70E-02	5.40E-04	3.26E+00	3.75E-02	6.60E-04	7.58E-06	3.80E-02
Silver	3.40E+00	4.01E-04	1.18E-04	4.71E-02	1.38E-02	1.89E-05	5.56E-06	1.40E-02
Uranium	5.76E+00	--	--	--	--	1.62E-03	2.81E-04	2.81E-04
Zinc	3.48E+02	7.19E-01	2.06E-03	6.98E+01	2.00E-01	8.89E-03	2.55E-05	2.02E-01

Ecological Hazard Quotients for the Rock Ptarmigan Exposed to Constituents of Interest in the CAM-D Main Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Invertebrate Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs										
Aroclor 1254 (Total PCBs)	1.56E+00	8.05E-04	5.16E-04	8.37E-04	5.37E-04	1.22E-03	7.80E-04	--	--	1.83E-03
Inorganics										
Arsenic	1.11E+01	2.33E-02	2.10E-03	7.67E-03	6.90E-04	5.16E-03	4.64E-04	6.38E-05	5.74E-06	3.26E-03
Barium	1.10E+02	1.35E+00	1.22E-02	3.42E-01	3.10E-03	1.29E-01	1.17E-03	5.21E-03	4.72E-05	1.65E-02
Cadmium	1.73E+01	8.85E-03	5.11E-04	2.04E-02	1.18E-03	8.54E-02	4.93E-03	1.21E-05	6.99E-07	6.62E-03
Copper	8.15E+01	2.21E-01	2.71E-03	2.46E-01	3.02E-03	1.68E-01	2.06E-03	--	--	7.79E-03
Lead	1.43E+01	2.98E-01	2.08E-02	6.21E-02	4.34E-03	1.41E-01	9.83E-03	2.76E-04	1.93E-05	3.50E-02
Lithium	4.02E+00	--	--	--	--	--	--	7.33E-04	1.82E-04	1.82E-04
Manganese	7.16E+03	1.46E-17	2.04E-21	7.35E-17	1.03E-20	4.03E-13	5.63E-17	7.63E-03	1.07E-06	1.07E-06
Nickel	9.27E+01	1.70E-01	1.83E-03	2.93E-02	3.16E-04	3.21E-01	3.47E-03	3.81E-04	4.12E-06	5.62E-03
Silver	6.32E+00	1.46E-03	2.31E-04	2.24E-03	3.54E-04	3.15E-03	4.98E-04	1.09E-05	1.73E-06	1.09E-03
Uranium	1.33E+02	--	--	--	--	--	--	9.35E-04	7.05E-06	7.05E-06
Zinc	1.01E+02	1.51E+00	1.49E-02	2.74E+00	2.70E-02	6.02E+00	5.94E-02	5.14E-03	5.07E-05	1.01E-01

Ecological Hazard Quotients for the Ermine Exposed to Constituents of Interest in the CAM-D Main Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Invertebrate Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs										
Aroclor 1254 (Total PCBs)	6.37E-01	1.25E-05	1.96E-05	9.91E-06	1.56E-05	2.41E-04	3.79E-04	--	--	4.14E-04
Inorganics										
Arsenic	1.18E+00	3.61E-04	3.06E-04	4.21E-05	3.56E-05	1.10E-04	9.28E-05	8.40E-06	7.12E-06	4.42E-04
Barium	2.77E+01	2.09E-02	7.54E-04	1.05E-03	3.79E-05	2.74E-03	9.89E-05	6.86E-04	2.48E-05	9.15E-04
Cadmium	1.09E+01	1.37E-04	1.26E-05	6.96E-04	6.41E-05	4.85E-03	4.47E-04	1.60E-06	1.47E-07	5.24E-04
Copper	1.75E+01	3.42E-03	1.96E-04	1.37E-03	7.83E-05	1.09E-01	6.21E-03	--	--	6.48E-03
Lead	8.69E+01	4.61E-03	5.31E-05	1.15E-03	1.32E-05	3.90E-02	4.49E-04	3.63E-05	4.19E-07	5.15E-04
Lithium	2.04E+01	--	--	--	--	1.12E-04	5.51E-06	9.65E-05	4.73E-06	1.02E-05
Manganese	3.08E+02	2.27E-19	7.36E-22	3.29E-15	1.07E-17	1.95E-19	6.34E-22	1.00E-03	3.26E-06	3.26E-06
Nickel	8.69E+01	2.63E-03	3.03E-05	2.62E-03	3.02E-05	2.33E-02	2.69E-04	5.02E-05	5.78E-07	3.30E-04
Silver	3.39E+00	2.27E-05	6.69E-06	2.57E-05	7.58E-06	3.81E-06	1.12E-06	1.44E-06	4.24E-07	1.58E-05
Uranium	5.74E+00	--	--	--	--	2.87E-06	4.99E-07	1.23E-04	2.14E-05	2.19E-05
Zinc	3.47E+02	2.34E-02	6.74E-05	4.91E-02	1.41E-04	1.17E+00	3.38E-03	6.77E-04	1.95E-06	3.59E-03

Ecological Hazard Quotients for the Arctic Fox Exposed to Constituents of Interest in the CAM-D Main Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs										
Aroclor 1254 (Total PCBs)	5.01E-01	9.15E-07	1.83E-06	1.35E-07	2.69E-07	1.47E-05	2.93E-05	--	--	3.14E-05
Inorganics										
Arsenic	9.28E-01	2.65E-05	2.86E-05	1.24E-06	1.33E-06	6.66E-06	7.18E-06	8.28E-07	8.93E-07	3.80E-05
Barium	2.18E+01	1.53E-03	7.03E-05	5.51E-05	2.53E-06	1.66E-04	7.65E-06	6.76E-05	3.11E-06	8.36E-05
Cadmium	8.53E+00	1.01E-05	1.18E-06	3.29E-06	3.85E-07	2.95E-04	3.46E-05	1.57E-07	1.84E-08	3.61E-05
Copper	1.38E+01	2.51E-04	1.83E-05	3.96E-05	2.88E-06	6.60E-03	4.80E-04	--	--	5.01E-04
Lead	6.82E+01	3.38E-04	4.96E-06	1.00E-05	1.47E-07	2.37E-03	3.47E-05	3.58E-06	5.25E-08	3.99E-05
Lithium	1.60E+01	--	--	--	--	6.83E-06	4.26E-07	9.52E-06	5.93E-07	1.02E-06
Manganese	2.42E+02	1.66E-20	6.87E-23	1.18E-20	4.88E-23	1.19E-20	4.90E-23	9.91E-05	4.09E-07	4.09E-07
Nickel	6.82E+01	1.93E-04	2.83E-06	4.71E-06	6.90E-08	1.42E-03	2.08E-05	4.95E-06	7.26E-08	2.37E-05
Silver	2.66E+00	1.66E-06	6.24E-07	3.61E-07	1.35E-07	2.32E-07	8.69E-08	1.42E-07	5.32E-08	9.00E-07
Uranium	4.51E+00	--	--	--	--	1.74E-07	3.86E-08	1.21E-05	2.69E-06	2.73E-06
Zinc	2.73E+02	1.72E-03	6.29E-06	4.41E-04	1.61E-06	7.13E-02	2.61E-04	6.67E-05	2.44E-07	2.69E-04

Ecological Hazard Quotients for the Arctic Hare Exposed to Constituents of Interest in the CAM-D Main Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs										
Aroclor 1254 (Total PCBs)	5.05E-01	2.31E-05	4.58E-05	4.94E-05	9.79E-05	7.25E-06	1.44E-05	--	--	1.58E-04
Inorganics										
Arsenic	9.35E-01	6.70E-04	7.17E-04	4.53E-04	4.84E-04	3.29E-06	3.52E-06	8.31E-06	8.89E-06	1.21E-03
Barium	2.19E+01	3.87E-02	1.76E-03	2.02E-02	9.20E-04	8.23E-05	3.75E-06	6.78E-04	3.09E-05	2.72E-03
Cadmium	8.60E+00	2.55E-04	2.96E-05	1.20E-03	1.40E-04	1.46E-04	1.70E-05	1.58E-06	1.83E-07	1.87E-04
Copper	1.39E+01	6.35E-03	4.58E-04	1.45E-02	1.05E-03	3.27E-03	2.36E-04	--	--	1.74E-03
Lead	6.88E+01	8.56E-03	1.24E-04	3.66E-03	5.32E-05	1.17E-03	1.70E-05	3.60E-05	5.22E-07	1.95E-04
Lithium	1.62E+01	--	--	--	--	3.38E-06	2.09E-07	9.55E-05	5.90E-06	6.11E-06
Manganese	2.44E+02	4.21E-19	1.72E-21	4.34E-18	1.78E-20	5.87E-21	2.40E-23	9.94E-04	4.07E-06	4.07E-06
Nickel	6.88E+01	4.88E-03	7.09E-05	1.73E-03	2.51E-05	7.01E-04	1.02E-05	4.97E-05	7.22E-07	1.07E-04
Silver	2.69E+00	4.21E-05	1.57E-05	1.32E-04	4.92E-05	1.15E-07	4.26E-08	1.42E-06	5.29E-07	6.54E-05
Uranium	4.55E+00	--	--	--	--	8.62E-08	1.89E-08	1.22E-04	2.68E-05	2.68E-05
Zinc	2.75E+02	4.34E-02	1.58E-04	1.62E-01	5.87E-04	3.53E-02	1.28E-04	6.69E-04	2.43E-06	8.75E-04

**Ecological Hazard Quotients for the Snowy Owl Exposed to Constituents of Interest in the CAM-D Main Dump**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs								
Aroclor 1254 (Total PCBs)	2.08E+00	7.73E-07	3.72E-07	1.25E-05	6.01E-06	--	--	6.38E-06
Inorganics								
Arsenic	1.48E+01	2.24E-05	1.51E-06	5.67E-06	3.82E-07	4.55E-07	3.07E-08	1.92E-06
Barium	1.47E+02	1.29E-03	8.80E-06	1.42E-04	9.64E-07	3.71E-05	2.53E-07	1.00E-05
Cadmium	2.31E+01	8.50E-06	3.68E-07	2.51E-04	1.09E-05	8.63E-08	3.74E-09	1.12E-05
Copper	1.09E+02	2.12E-04	1.95E-06	5.62E-03	5.17E-05	--	--	5.37E-05
Lead	1.91E+01	2.86E-04	1.50E-05	2.02E-03	1.06E-04	1.97E-06	1.03E-07	1.21E-04
Lithium	5.35E+00	--	--	5.82E-06	1.09E-06	5.22E-06	9.75E-07	2.06E-06
Manganese	9.54E+03	1.41E-20	1.47E-24	1.01E-20	1.06E-24	5.44E-05	5.70E-09	5.70E-09
Nickel	1.24E+02	1.63E-04	1.32E-06	1.21E-03	9.77E-06	2.72E-06	2.20E-08	1.11E-05
Silver	8.43E+00	1.41E-06	1.67E-07	1.97E-07	2.34E-08	7.78E-08	9.23E-09	1.99E-07
Uranium	1.77E+02	--	--	1.48E-07	8.40E-10	6.66E-06	3.77E-08	3.86E-08
Zinc	1.35E+02	1.45E-03	1.07E-05	6.07E-02	4.50E-04	3.66E-05	2.71E-07	4.61E-04



**Ecological Hazard Quotients for the Barren-ground Caribou Exposed to Constituents of Interest in the CAM-D Main Dump**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs								
Aroclor 1254 (Total PCBs)	4.14E-01	1.24E-06	2.99E-06	1.87E-06	4.52E-06	--	--	7.51E-06
Inorganics								
Arsenic	7.67E-01	3.58E-05	4.67E-05	1.72E-05	2.24E-05	6.03E-07	7.86E-07	6.98E-05
Barium	1.80E+01	2.07E-03	1.15E-04	7.65E-04	4.25E-05	4.92E-05	2.73E-06	1.60E-04
Cadmium	7.05E+00	1.36E-05	1.93E-06	4.57E-05	6.47E-06	1.15E-07	1.62E-08	8.42E-06
Copper	1.14E+01	3.39E-04	2.98E-05	5.50E-04	4.84E-05	--	--	7.82E-05
Lead	5.64E+01	4.57E-04	8.10E-06	1.39E-04	2.46E-06	2.61E-06	4.62E-08	1.06E-05
Lithium	1.33E+01	--	--	--	--	6.93E-06	5.22E-07	5.22E-07
Manganese	2.00E+02	2.25E-20	1.12E-22	1.64E-19	8.21E-22	7.21E-05	3.60E-07	3.60E-07
Nickel	5.64E+01	2.61E-04	4.62E-06	6.54E-05	1.16E-06	3.60E-06	6.39E-08	5.84E-06
Silver	2.20E+00	2.25E-06	1.02E-06	5.01E-06	2.27E-06	1.03E-07	4.68E-08	3.34E-06
Uranium	3.73E+00	--	--	--	--	8.84E-06	2.37E-06	2.37E-06
Zinc	2.26E+02	2.32E-03	1.03E-05	6.12E-03	2.71E-05	4.86E-05	2.15E-07	3.76E-05

**Ecological Hazard Quotients for the Collared Lemming Exposed to Constituents of Interest in the CAM-D Main Dump**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs								
Aroclor 1254 (Total PCBs)	6.39E-01	3.07E-04	4.80E-04	6.83E-04	1.07E-03	--	--	1.55E-03
Inorganics								
Arsenic	1.18E+00	8.88E-03	7.51E-03	6.26E-03	5.29E-03	1.10E-04	9.33E-05	1.29E-02
Barium	2.78E+01	5.13E-01	1.85E-02	2.79E-01	1.01E-02	9.01E-03	3.24E-04	2.89E-02
Cadmium	1.09E+01	3.37E-03	3.10E-04	1.67E-02	1.53E-03	2.10E-05	1.93E-06	1.84E-03
Copper	1.76E+01	8.42E-02	4.79E-03	2.01E-01	1.14E-02	--	--	1.62E-02
Lead	8.71E+01	1.13E-01	1.30E-03	5.07E-02	5.82E-04	4.78E-04	5.48E-06	1.89E-03
Lithium	2.05E+01	--	--	--	--	1.27E-03	6.20E-05	6.20E-05
Manganese	3.09E+02	5.57E-18	1.80E-20	6.00E-17	1.94E-19	1.32E-02	4.27E-05	4.27E-05
Nickel	8.71E+01	6.47E-02	7.42E-04	2.39E-02	2.74E-04	6.60E-04	7.58E-06	1.02E-03
Silver	3.40E+00	5.57E-04	1.64E-04	1.83E-03	5.38E-04	1.89E-05	5.56E-06	7.07E-04
Uranium	5.76E+00	--	--	--	--	1.62E-03	2.81E-04	2.81E-04
Zinc	3.48E+02	5.75E-01	1.65E-03	2.24E+00	6.42E-03	8.89E-03	2.55E-05	8.09E-03

**Ecological Hazard Quotients for the Barren-ground Caribou Exposed to Constituents of Interest in the CAM-D Background Area**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
Inorganics						
Arsenic	7.67E-01	2.99E-03	3.90E-03	1.39E-03	1.82E-03	5.71E-03
Barium	1.80E+01	1.41E-01	7.83E-03	5.21E-02	2.90E-03	1.07E-02
Cadmium	7.05E+00	1.11E-04	1.57E-05	1.04E-03	1.47E-04	1.63E-04
Copper	1.14E+01	1.92E-02	1.68E-03	3.79E-02	3.33E-03	5.01E-03
Lead	5.64E+01	1.23E-02	2.18E-04	5.98E-03	1.06E-04	3.24E-04
Nickel	5.64E+01	2.16E-02	3.83E-04	5.35E-03	9.48E-05	4.78E-04
Silver	2.20E+00	1.32E-04	5.98E-05	2.94E-04	1.33E-04	1.93E-04
Zinc	2.26E+02	7.72E-02	3.42E-04	2.98E-01	1.32E-03	1.66E-03

**Ecological Hazard Quotients for the Arctic Fox Exposed to Constituents of Interest in the CAM-D Background Area**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Mammal Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
Inorganics								
Arsenic	9.28E-01	2.21E-03	2.39E-03	1.00E-04	1.08E-04	5.49E-04	5.92E-04	3.09E-03
Barium	2.18E+01	1.04E-01	4.79E-03	3.75E-03	1.73E-04	1.18E-02	5.44E-04	5.51E-03
Cadmium	8.53E+00	8.20E-05	9.61E-06	7.48E-05	8.77E-06	7.68E-03	9.00E-04	9.18E-04
Copper	1.38E+01	1.42E-02	1.03E-03	2.73E-03	1.98E-04	4.93E-01	3.59E-02	3.71E-02
Lead	6.82E+01	9.11E-03	1.34E-04	4.30E-04	6.30E-06	1.16E-01	1.69E-03	1.83E-03
Nickel	6.82E+01	1.60E-02	2.35E-04	3.85E-04	5.64E-06	1.14E-01	1.67E-03	1.91E-03
Silver	2.66E+00	9.76E-05	3.66E-05	2.12E-05	7.94E-06	1.36E-05	5.10E-06	4.97E-05
Zinc	2.73E+02	5.71E-02	2.09E-04	2.15E-02	7.86E-05	5.24E+00	1.92E-02	1.95E-02

**Ecological Hazard Quotients for the Arctic Hare Exposed to Constituents of Interest in the CAM-D Background Area**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Mammal Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
Inorganics								
Arsenic	9.35E-01	6.28E-05	6.72E-05	4.13E-05	4.41E-05	3.05E-07	3.26E-07	1.12E-04
Barium	2.19E+01	2.96E-03	1.35E-04	1.54E-03	7.04E-05	6.57E-06	2.99E-07	2.06E-04
Cadmium	8.60E+00	2.33E-06	2.71E-07	3.08E-05	3.58E-06	4.26E-06	4.96E-07	4.34E-06
Copper	1.39E+01	4.03E-04	2.91E-05	1.12E-03	8.09E-05	2.74E-04	1.97E-05	1.30E-04
Lead	6.88E+01	2.59E-04	3.76E-06	1.77E-04	2.57E-06	6.42E-05	9.33E-07	7.27E-06
Nickel	6.88E+01	4.55E-04	6.61E-06	1.58E-04	2.30E-06	6.33E-05	9.20E-07	9.83E-06
Silver	2.69E+00	2.77E-06	1.03E-06	8.71E-06	3.24E-06	7.55E-09	2.81E-09	4.28E-06
Zinc	2.75E+02	1.62E-03	5.90E-06	8.83E-03	3.21E-05	2.91E-03	1.06E-05	4.86E-05

**Ecological Hazard Quotients for the Ermine Exposed to Constituents of Interest in the CAM-D Background Area**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Invertebrate Ingestion		Terrestrial Mammal Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
Inorganics								
Arsenic	1.18E+00	1.48E-03	1.26E-03	1.69E-04	1.44E-04	4.45E-04	3.77E-04	1.78E-03
Barium	2.77E+01	6.99E-02	2.53E-03	3.52E-03	1.27E-04	9.57E-03	3.46E-04	3.00E-03
Cadmium	1.09E+01	5.50E-05	5.06E-06	4.44E-04	4.09E-05	6.21E-03	5.72E-04	6.18E-04
Copper	1.75E+01	9.51E-03	5.43E-04	4.84E-03	2.76E-04	3.99E-01	2.28E-02	2.36E-02
Lead	8.69E+01	6.11E-03	7.03E-05	1.87E-03	2.15E-05	9.35E-02	1.08E-03	1.17E-03
Nickel	8.69E+01	1.07E-02	1.24E-04	9.93E-03	1.14E-04	9.23E-02	1.06E-03	1.30E-03
Silver	3.39E+00	6.55E-05	1.93E-05	7.42E-05	2.19E-05	1.10E-05	3.24E-06	4.44E-05
Zinc	3.47E+02	3.83E-02	1.10E-04	1.43E-01	4.11E-04	4.24E+00	1.22E-02	1.27E-02

**Ecological Hazard Quotients for the Snowy Owl Exposed to Constituents of Interest in the CAM-D Background Area**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Mammal Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
Inorganics						
Arsenic	1.48E+01	1.87E-03	1.26E-04	4.68E-04	3.15E-05	1.58E-04
Barium	1.47E+02	8.81E-02	6.00E-04	1.01E-02	6.85E-05	6.68E-04
Cadmium	2.31E+01	6.93E-05	3.00E-06	6.53E-03	2.83E-04	2.86E-04
Copper	1.09E+02	1.20E-02	1.10E-04	4.20E-01	3.86E-03	3.97E-03
Lead	1.91E+01	7.70E-03	4.04E-04	9.84E-02	5.16E-03	5.57E-03
Nickel	1.24E+02	1.35E-02	1.10E-04	9.71E-02	7.86E-04	8.95E-04
Silver	8.43E+00	8.25E-05	9.79E-06	1.16E-05	1.37E-06	1.12E-05
Zinc	1.35E+02	4.83E-02	3.58E-04	4.46E+00	3.30E-02	3.34E-02

Ecological Hazard Quotients for the Arctic Fox Exposed to Constituents of Interest in the CAM-D Barrel Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX										
Ethylbenzene	3.48E+02	3.33E-07	9.56E-10	2.82E-08	8.11E-11	1.85E-09	5.32E-12	--	--	1.04E-09
Toluene	1.91E+02	4.29E-07	2.24E-09	3.90E-08	2.04E-10	8.79E-10	4.59E-12	--	--	2.45E-09
Xylenes	1.90E+03	3.33E-07	1.75E-10	2.76E-08	1.46E-11	2.01E-09	1.06E-12	--	--	1.91E-10
TPH - CCME CWS										
Aliph>C06-C08 - F1	9.46E+01	3.48E-05	3.68E-07	4.70E-06	4.96E-08	1.82E-06	1.92E-08	--	--	4.37E-07
Aliph>C08-C10 - F1	9.46E+01	2.28E-05	2.41E-07	1.91E-06	2.02E-08	7.22E-06	7.62E-08	--	--	3.37E-07
Arom>C08-C10 - F1	2.03E+01	5.69E-06	2.81E-07	9.49E-07	4.68E-08	1.36E-07	6.70E-09	--	--	3.34E-07
F1 - Total										--
Aliph>C10-C12 - F2	9.46E+01	9.17E-04	9.69E-06	1.92E-05	2.03E-07	1.42E-03	1.50E-05	--	--	2.49E-05
Aliph>C12-C16 - F2	9.46E+01	1.12E-03	1.18E-05	1.18E-05	1.25E-07	2.03E-02	2.14E-04	--	--	2.26E-04
Arom>C10-C12 - F2	2.03E+01	2.29E-04	1.13E-05	2.75E-05	1.36E-06	7.03E-06	3.47E-07	--	--	1.30E-05
Arom>C12-C16 - F2	2.03E+01	2.80E-04	1.38E-05	2.87E-05	1.42E-06	1.56E-05	7.72E-07	--	--	1.60E-05
F2 - Total										--
Aliph>C16-C21 - F3	1.89E+02	1.56E-02	8.22E-05	5.06E-05	2.67E-07	2.59E-01	1.37E-03	--	--	1.45E-03
Aliph>C21-C34 - F3	1.89E+02	6.67E-03	3.52E-05	2.17E-05	1.15E-07	1.11E-02	5.86E-05	--	--	9.39E-05
Arom>C16-C21 - F3	4.06E+01	3.89E-03	9.59E-05	1.53E-04	3.77E-06	4.50E-04	1.11E-05	--	--	1.11E-04
Arom>C21-C34 - F3	4.06E+01	1.67E-03	4.11E-05	4.08E-05	1.01E-06	1.34E-03	3.31E-05	--	--	7.52E-05
F3 - Total										--
Aliph>C34-C50 - F4	1.76E+03	1.25E-02	7.11E-06	8.13E-06	4.63E-09	2.01E-01	1.15E-04	--	--	1.22E-04
Arom>C34-C50 - F4	1.83E+02	3.12E-03	1.71E-05	2.09E-05	1.14E-07	2.95E-02	1.62E-04	--	--	1.79E-04
F4 - Total										--
PAHs										
Naphthalene	1.47E+02	8.32E-07	5.65E-09	4.25E-06	2.88E-08	2.52E-07	1.71E-09	--	--	3.62E-08
PCBs										
Aroclor 1254 (Total PCBs)	5.01E-01	6.32E-07	1.26E-06	9.31E-08	1.86E-07	1.01E-05	2.02E-05	--	--	2.17E-05
Inorganics										
Arsenic	9.28E-01	3.39E-05	3.65E-05	1.92E-06	2.07E-06	9.23E-06	9.95E-06	1.66E-06	1.79E-06	5.03E-05
Barium	2.18E+01	3.13E-03	1.44E-04	1.13E-04	5.17E-06	3.38E-04	1.55E-05	1.35E-04	6.21E-06	1.71E-04
Cadmium	8.53E+00	1.96E-05	2.29E-06	6.47E-06	7.59E-07	5.82E-04	6.82E-05	3.15E-07	3.69E-08	7.13E-05
Copper	1.38E+01	2.50E-04	1.81E-05	6.01E-05	4.37E-06	1.19E-02	8.68E-04	--	--	8.91E-04
Lead	6.82E+01	2.59E-04	3.80E-06	1.17E-05	1.71E-07	3.10E-03	4.54E-05	7.17E-06	1.05E-07	4.95E-05
Lithium	1.60E+01	--	--	--	--	1.37E-05	8.52E-07	1.90E-05	1.19E-06	2.04E-06
Manganese	2.42E+02	3.33E-20	1.37E-22	2.37E-20	9.77E-23	2.37E-20	9.80E-23	1.98E-04	8.18E-07	8.18E-07
Nickel	6.82E+01	1.77E-04	2.59E-06	5.26E-06	7.70E-08	1.97E-03	2.89E-05	9.91E-06	1.45E-07	3.17E-05
Silver	2.66E+00	1.66E-06	6.24E-07	3.61E-07	1.35E-07	2.32E-07	8.69E-08	2.84E-07	1.06E-07	9.53E-07
Uranium	4.51E+00	--	--	--	--	3.49E-07	7.73E-08	2.43E-05	5.38E-06	5.46E-06
Zinc	2.73E+02	1.60E-03	5.88E-06	5.78E-04	2.12E-06	1.35E-01	4.94E-04	1.33E-04	4.89E-07	5.03E-04



Ecological Hazard Quotients for the Arctic Hare Exposed to Constituents of Interest in the CAM-D Barrel Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX										
Ethylbenzene	3.51E+02	8.43E-06	2.40E-08	1.04E-05	2.95E-08	9.18E-10	2.62E-12	--	--	5.36E-08
Toluene	1.93E+02	1.09E-05	5.64E-08	1.43E-05	7.42E-08	4.36E-10	2.26E-12	--	--	1.31E-07
Xylenes	1.92E+03	8.43E-06	4.40E-09	1.01E-05	5.30E-09	9.96E-10	5.20E-13	--	--	9.70E-09
TPH - CCME CWS										
Aliph>C06-C08 - F1	9.54E+01	8.82E-04	9.24E-06	1.73E-03	1.81E-05	9.01E-07	9.44E-09	--	--	2.73E-05
Aliph>C08-C10 - F1	9.54E+01	5.77E-04	6.05E-06	7.03E-04	7.36E-06	3.58E-06	3.75E-08	--	--	1.34E-05
Arom>C08-C10 - F1	2.05E+01	1.44E-04	7.06E-06	3.49E-04	1.70E-05	6.73E-08	3.29E-09	--	--	2.41E-05
F1 - Total										--
Aliph>C10-C12 - F2	9.54E+01	2.32E-02	2.44E-04	7.04E-03	7.38E-05	7.06E-04	7.39E-06	--	--	3.25E-04
Aliph>C12-C16 - F2	9.54E+01	2.84E-02	2.98E-04	4.34E-03	4.54E-05	1.00E-02	1.05E-04	--	--	4.48E-04
Arom>C10-C12 - F2	2.05E+01	5.81E-03	2.84E-04	1.01E-02	4.94E-04	3.49E-06	1.70E-07	--	--	7.78E-04
Arom>C12-C16 - F2	2.05E+01	7.10E-03	3.47E-04	1.05E-02	5.16E-04	7.76E-06	3.79E-07	--	--	8.63E-04
F2 - Total										--
Aliph>C16-C21 - F3	1.91E+02	3.94E-01	2.07E-03	1.86E-02	9.74E-05	1.28E-01	6.72E-04	--	--	2.83E-03
Aliph>C21-C34 - F3	1.91E+02	1.69E-01	8.85E-04	7.97E-03	4.17E-05	5.49E-03	2.88E-05	--	--	9.56E-04
Arom>C16-C21 - F3	4.09E+01	9.86E-02	2.41E-03	5.62E-02	1.37E-03	2.23E-04	5.45E-06	--	--	3.79E-03
Arom>C21-C34 - F3	4.09E+01	4.22E-02	1.03E-03	1.50E-02	3.66E-04	6.65E-04	1.63E-05	--	--	1.42E-03
F3 - Total										--
Aliph>C34-C50 - F4	1.77E+03	3.17E-01	1.79E-04	2.99E-03	1.69E-06	9.98E-02	5.63E-05	--	--	2.37E-04
Arom>C34-C50 - F4	1.84E+02	7.92E-02	4.30E-04	7.66E-03	4.16E-05	1.46E-02	7.95E-05	--	--	5.51E-04
F4 - Total										--
PAHs										
Naphthalene	1.48E+02	2.11E-05	1.42E-07	1.56E-03	1.05E-05	1.25E-07	8.42E-10	--	--	1.06E-05
PCBs										
Aroclor 1254 (Total PCBs)	5.05E-01	1.60E-05	3.17E-05	3.42E-05	6.78E-05	5.02E-06	9.95E-06	--	--	1.09E-04
Inorganics										
Arsenic	9.35E-01	8.58E-04	9.18E-04	7.05E-04	7.53E-04	4.57E-06	4.89E-06	1.67E-05	1.78E-05	1.69E-03
Barium	2.19E+01	7.93E-02	3.61E-03	4.13E-02	1.88E-03	1.68E-04	7.64E-06	1.36E-03	6.20E-05	5.57E-03
Cadmium	8.60E+00	4.96E-04	5.76E-05	2.38E-03	2.76E-04	2.88E-04	3.35E-05	3.16E-06	3.68E-07	3.68E-04
Copper	1.39E+01	6.33E-03	4.56E-04	2.21E-02	1.59E-03	5.92E-03	4.27E-04	--	--	2.48E-03
Lead	6.88E+01	6.58E-03	9.56E-05	4.29E-03	6.23E-05	1.54E-03	2.23E-05	7.21E-05	1.05E-06	1.81E-04
Lithium	1.62E+01	--	--	--	--	6.77E-06	4.19E-07	1.91E-04	1.18E-05	1.23E-05
Manganese	2.44E+02	8.43E-19	3.45E-21	8.69E-18	3.56E-20	1.18E-20	4.82E-23	1.99E-03	8.15E-06	8.15E-06
Nickel	6.88E+01	4.48E-03	6.52E-05	1.93E-03	2.81E-05	9.78E-04	1.42E-05	9.96E-05	1.45E-06	1.09E-04
Silver	2.69E+00	4.22E-05	1.57E-05	1.32E-04	4.93E-05	1.15E-07	4.27E-08	2.85E-06	1.06E-06	6.61E-05
Uranium	4.55E+00	--	--	--	--	1.73E-07	3.80E-08	2.44E-04	5.36E-05	5.37E-05
Zinc	2.75E+02	4.07E-02	1.48E-04	2.12E-01	7.71E-04	6.68E-02	2.43E-04	1.34E-03	4.87E-06	1.17E-03

Ecological Hazard Quotients for the Barren-ground Caribou Exposed to Constituents of Interest in the CAM-D Barrel Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion			Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	SSL for Ter. Plant Ingestion (mg/kg)	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX									
Ethylbenzene	2.88E+02	4.49E-07	1.56E-09	3.92E-07	1.36E-09	4.46E+04	--	--	2.92E-09
Toluene	1.58E+02	5.80E-07	3.66E-09	5.42E-07	3.42E-09	2.45E+04	--	--	7.08E-09
Xylenes	1.57E+03	4.49E-07	2.86E-10	3.84E-07	2.44E-10	2.44E+05	--	--	5.31E-10
TPH - CCME CWS									
Aliph>C06-C08 - F1	7.83E+01	4.70E-05	6.01E-07	6.53E-05	8.34E-07	1.21E+04	--	--	1.43E-06
Aliph>C08-C10 - F1	7.83E+01	3.08E-05	3.93E-07	2.66E-05	3.40E-07	1.21E+04	--	--	7.33E-07
Arom>C08-C10 - F1	1.68E+01	7.69E-06	4.59E-07	1.32E-05	7.86E-07	2.60E+03	--	--	1.24E-06
F1 - Total									--
Aliph>C10-C12 - F2	7.83E+01	1.24E-03	1.58E-05	2.66E-04	3.40E-06	1.21E+04	--	--	1.92E-05
Aliph>C12-C16 - F2	7.83E+01	1.51E-03	1.93E-05	1.64E-04	2.10E-06	1.21E+04	--	--	2.14E-05
Arom>C10-C12 - F2	1.68E+01	3.10E-04	1.85E-05	3.82E-04	2.28E-05	2.60E+03	--	--	4.13E-05
Arom>C12-C16 - F2	1.68E+01	3.79E-04	2.26E-05	3.99E-04	2.38E-05	2.60E+03	--	--	4.63E-05
F2 - Total									--
Aliph>C16-C21 - F3	1.57E+02	2.10E-02	1.34E-04	7.03E-04	4.49E-06	2.43E+04	--	--	1.39E-04
Aliph>C21-C34 - F3	1.57E+02	9.00E-03	5.75E-05	3.01E-04	1.93E-06	2.43E+04	--	--	5.95E-05
Arom>C16-C21 - F3	3.35E+01	5.25E-03	1.57E-04	2.13E-03	6.34E-05	5.20E+03	--	--	2.20E-04
Arom>C21-C34 - F3	3.35E+01	2.25E-03	6.71E-05	5.67E-04	1.69E-05	5.20E+03	--	--	8.40E-05
F3 - Total									--
Aliph>C34-C50 - F4	1.45E+03	1.69E-02	1.16E-05	1.13E-04	7.77E-08	2.25E+05	--	--	1.17E-05
Arom>C34-C50 - F4	1.51E+02	4.22E-03	2.80E-05	2.90E-04	1.92E-06	2.34E+04	--	--	2.99E-05
F4 - Total									--
PAHs									
Naphthalene	1.22E+02	1.12E-06	9.23E-09	5.90E-05	4.85E-07	1.89E+04	--	--	4.94E-07
PCBs									
Aroclor 1254 (Total PCBs)	4.14E-01	8.54E-07	2.06E-06	1.29E-06	3.13E-06	6.42E+01	--	--	5.19E-06
Inorganics									
Arsenic	7.67E-01	4.57E-05	5.96E-05	2.67E-05	3.47E-05	1.19E+02	1.21E-06	1.57E-06	9.60E-05
Barium	1.80E+01	4.22E-03	2.35E-04	1.56E-03	8.69E-05	2.79E+03	9.84E-05	5.47E-06	3.27E-04
Cadmium	7.05E+00	2.64E-05	3.75E-06	8.99E-05	1.27E-05	1.09E+03	2.29E-07	3.25E-08	1.65E-05
Copper	1.14E+01	3.37E-04	2.96E-05	8.35E-04	7.34E-05	1.76E+03	--	--	1.03E-04
Lead	5.64E+01	3.50E-04	6.21E-06	1.62E-04	2.87E-06	8.75E+03	5.22E-06	9.24E-08	9.18E-06
Lithium	1.33E+01	--	--	--	--	--	1.39E-05	1.04E-06	1.04E-06
Manganese	2.00E+02	4.49E-20	2.24E-22	3.29E-19	1.64E-21	3.11E+04	1.44E-04	7.20E-07	7.20E-07
Nickel	5.64E+01	2.39E-04	4.23E-06	7.30E-05	1.29E-06	8.75E+03	7.21E-06	1.28E-07	5.66E-06
Silver	2.20E+00	2.25E-06	1.02E-06	5.01E-06	2.27E-06	3.42E+02	2.06E-07	9.37E-08	3.39E-06
Uranium	3.73E+00	--	--	--	--	--	1.77E-05	4.74E-06	4.74E-06
Zinc	2.26E+02	2.17E-03	9.60E-06	8.03E-03	3.56E-05	3.50E+04	9.71E-05	4.30E-07	4.56E-05

Ecological Hazard Quotients for the Ermine Exposed to Constituents of Interest in the CAM-D Barrel Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Invertebrate Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX										
Ethylbenzene	4.43E+02	4.54E-06	1.03E-08	1.50E-07	3.39E-10	3.05E-08	6.90E-11	--	--	1.07E-08
Toluene	2.44E+02	5.86E-06	2.41E-08	2.21E-07	9.09E-10	1.45E-08	5.95E-11	--	--	2.50E-08
Xylenes	2.42E+03	4.54E-06	1.88E-09	1.48E-07	6.13E-11	3.31E-08	1.37E-11	--	--	1.96E-09
TPH - CCME CWS										
Aliph>C06-C08 - F1	1.20E+02	4.75E-04	3.95E-06	1.21E-04	1.01E-06	3.00E-05	2.49E-07	--	--	5.20E-06
Aliph>C08-C10 - F1	1.20E+02	3.11E-04	2.58E-06	6.07E-05	5.04E-07	1.19E-04	9.87E-07	--	--	4.07E-06
Arom>C08-C10 - F1	2.58E+01	7.78E-05	3.01E-06	2.24E-05	8.68E-07	2.24E-06	8.67E-08	--	--	3.97E-06
F1 - Total										--
Aliph>C10-C12 - F2	1.20E+02	1.25E-02	1.04E-04	1.87E-03	1.55E-05	2.35E-02	1.95E-04	--	--	3.14E-04
Aliph>C12-C16 - F2	1.20E+02	1.53E-02	1.27E-04	1.24E-03	1.03E-05	3.34E-01	2.77E-03	--	--	2.91E-03
Arom>C10-C12 - F2	2.58E+01	3.13E-03	1.21E-04	8.50E-04	3.29E-05	1.16E-04	4.49E-06	--	--	1.59E-04
Arom>C12-C16 - F2	2.58E+01	3.83E-03	1.48E-04	9.49E-04	3.68E-05	2.58E-04	9.99E-06	--	--	1.95E-04
F2 - Total										--
Aliph>C16-C21 - F3	2.41E+02	2.13E-01	8.82E-04	2.29E-02	9.50E-05	4.26E+00	1.77E-02	--	--	1.87E-02
Aliph>C21-C34 - F3	2.41E+02	9.11E-02	3.78E-04	5.58E-03	2.32E-05	1.83E-01	7.58E-04	--	--	1.16E-03
Arom>C16-C21 - F3	5.16E+01	5.31E-02	1.03E-03	9.07E-03	1.76E-04	7.42E-03	1.44E-04	--	--	1.35E-03
Arom>C21-C34 - F3	5.16E+01	2.28E-02	4.41E-04	7.42E-03	1.44E-04	2.21E-02	4.29E-04	--	--	1.01E-03
F3 - Total										--
Aliph>C34-C50 - F4	2.24E+03	1.71E-01	7.63E-05	4.41E-03	1.97E-06	3.32E+00	1.48E-03	--	--	1.56E-03
Arom>C34-C50 - F4	2.32E+02	4.27E-02	1.84E-04	3.95E-02	1.70E-04	4.87E-01	2.09E-03	--	--	2.45E-03
F4 - Total										--
PAHs										
Naphthalene	1.87E+02	1.14E-05	6.06E-08	3.49E-06	1.86E-08	4.16E-06	2.22E-08	--	--	1.01E-07
PCBs										
Aroclor 1254 (Total PCBs)	6.37E-01	8.63E-06	1.36E-05	4.68E-06	7.34E-06	1.67E-04	2.62E-04	--	--	2.83E-04
Inorganics										
Arsenic	1.18E+00	4.63E-04	3.92E-04	6.14E-05	5.20E-05	1.52E-04	1.29E-04	1.68E-05	1.43E-05	5.87E-04
Barium	2.77E+01	4.27E-02	1.54E-03	2.15E-03	7.76E-05	5.57E-03	2.01E-04	1.37E-03	4.96E-05	1.87E-03
Cadmium	1.09E+01	2.67E-04	2.46E-05	1.36E-03	1.26E-04	9.59E-03	8.83E-04	3.20E-06	2.95E-07	1.03E-03
Copper	1.75E+01	3.41E-03	1.95E-04	2.28E-03	1.30E-04	1.97E-01	1.12E-02	--	--	1.16E-02
Lead	8.69E+01	3.54E-03	4.08E-05	1.06E-03	1.22E-05	5.11E-02	5.88E-04	7.28E-05	8.39E-07	6.42E-04
Lithium	2.04E+01	--	--	--	--	2.25E-04	1.10E-05	1.93E-04	9.48E-06	2.05E-05
Manganese	3.08E+02	4.54E-19	1.47E-21	6.59E-15	2.14E-17	3.91E-19	1.27E-21	2.01E-03	6.53E-06	6.53E-06
Nickel	8.69E+01	2.42E-03	2.78E-05	6.43E-03	7.40E-05	3.25E-02	3.74E-04	1.01E-04	1.16E-06	4.77E-04
Silver	3.39E+00	2.27E-05	6.70E-06	2.57E-05	7.59E-06	3.82E-06	1.13E-06	2.88E-06	8.50E-07	1.63E-05
Uranium	5.74E+00	--	--	--	--	5.75E-06	1.00E-06	2.47E-04	4.30E-05	4.40E-05
Zinc	3.47E+02	2.19E-02	6.31E-05	7.66E-02	2.21E-04	2.22E+00	6.40E-03	1.36E-03	3.90E-06	6.69E-03

Ecological Hazard Quotients for the Snowy Owl Exposed to Constituents of Interest in the CAM-D Barrel Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Mammal Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX								
Ethylbenzene	1.16E+02	2.81E-07	2.42E-09	1.58E-09	1.36E-11	--	--	2.43E-09
Toluene	1.21E+02	3.63E-07	2.99E-09	7.48E-10	6.18E-12	--	--	3.00E-09
Xylenes	1.20E+03	2.81E-07	2.34E-10	1.71E-09	1.42E-12	--	--	2.35E-10
TPH - CCME CWS								
Aliph>C06-C08 - F1	8.66E+01	2.94E-05	3.40E-07	1.55E-06	1.79E-08	--	--	3.57E-07
Aliph>C08-C10 - F1	8.66E+01	1.92E-05	2.22E-07	6.14E-06	7.09E-08	--	--	2.93E-07
Arom>C08-C10 - F1	2.89E+01	4.81E-06	1.67E-07	1.16E-07	4.01E-09	--	--	1.71E-07
F1 - Total								--
Aliph>C10-C12 - F2	8.66E+01	7.75E-04	8.95E-06	1.21E-03	1.40E-05	--	--	2.29E-05
Aliph>C12-C16 - F2	8.66E+01	9.47E-04	1.09E-05	1.72E-02	1.99E-04	--	--	2.10E-04
Arom>C10-C12 - F2	2.89E+01	1.94E-04	6.71E-06	5.98E-06	2.07E-07	--	--	6.92E-06
Arom>C12-C16 - F2	2.89E+01	2.37E-04	8.20E-06	1.33E-05	4.61E-07	--	--	8.66E-06
F2 - Total								--
Aliph>C16-C21 - F3	1.15E+02	1.31E-02	1.14E-04	2.20E-01	1.91E-03	--	--	2.02E-03
Aliph>C21-C34 - F3	1.15E+02	5.63E-03	4.88E-05	9.43E-03	8.17E-05	--	--	1.30E-04
Arom>C16-C21 - F3	5.77E+01	3.29E-03	5.69E-05	3.83E-04	6.64E-06	--	--	6.36E-05
Arom>C21-C34 - F3	5.77E+01	1.41E-03	2.44E-05	1.14E-03	1.98E-05	--	--	4.42E-05
F3 - Total								--
Aliph>C34-C50 - F4	1.73E+02	1.06E-02	6.10E-05	1.71E-01	9.89E-04	--	--	1.05E-03
Arom>C34-C50 - F4	2.31E+01	2.64E-03	1.14E-04	2.51E-02	1.09E-03	--	--	1.20E-03
F4 - Total								--
PAHs								
Naphthalene	9.31E+01	7.03E-07	7.55E-09	2.15E-07	2.31E-09	--	--	9.85E-09
PCBs								
Aroclor 1254 (Total PCBs)	2.08E+00	5.34E-07	2.57E-07	8.62E-06	4.15E-06	--	--	4.41E-06
Inorganics								
Arsenic	1.48E+01	2.86E-05	1.93E-06	7.85E-06	5.30E-07	9.09E-07	6.13E-08	2.52E-06
Barium	1.47E+02	2.64E-03	1.80E-05	2.88E-04	1.96E-06	7.42E-05	5.05E-07	2.04E-05
Cadmium	2.31E+01	1.65E-05	7.16E-07	4.95E-04	2.14E-05	1.73E-07	7.48E-09	2.22E-05
Copper	1.09E+02	2.11E-04	1.94E-06	1.02E-02	9.35E-05	--	--	9.55E-05
Lead	1.91E+01	2.19E-04	1.15E-05	2.64E-03	1.38E-04	3.93E-06	2.06E-07	1.50E-04
Lithium	5.35E+00	--	--	1.16E-05	2.17E-06	1.04E-05	1.95E-06	4.12E-06
Manganese	9.54E+03	2.81E-20	2.94E-24	2.02E-20	2.12E-24	1.09E-04	1.14E-08	1.14E-08
Nickel	1.24E+02	1.49E-04	1.21E-06	1.68E-03	1.36E-05	5.44E-06	4.40E-08	1.48E-05
Silver	8.43E+00	1.41E-06	1.67E-07	1.97E-07	2.34E-08	1.56E-07	1.85E-08	2.09E-07
Uranium	1.77E+02	--	--	2.97E-07	1.68E-09	1.33E-05	7.54E-08	7.71E-08
Zinc	1.35E+02	1.36E-03	1.00E-05	1.15E-01	8.50E-04	7.32E-05	5.43E-07	8.61E-04

Ecological Hazard Quotients for the Collared Lemming Exposed to Constituents of Interest in the CAM-D Barrel Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX								
Ethylbenzene	4.44E+02	5.57E-05	1.25E-07	7.16E-05	1.61E-07	--	--	2.87E-07
Toluene	2.44E+02	7.19E-05	2.94E-07	9.89E-05	4.05E-07	--	--	6.99E-07
Xylenes	2.42E+03	5.57E-05	2.30E-08	7.01E-05	2.89E-08	--	--	5.19E-08
TPH - CCME CWS								
Aliph>C06-C08 - F1	1.21E+02	5.83E-03	4.83E-05	1.19E-02	9.86E-05	--	--	1.47E-04
Aliph>C08-C10 - F1	1.21E+02	3.82E-03	3.16E-05	4.85E-03	4.01E-05	--	--	7.17E-05
Arom>C08-C10 - F1	2.59E+01	9.54E-04	3.69E-05	2.41E-03	9.30E-05	--	--	1.30E-04
F1 - Total								--
Aliph>C10-C12 - F2	1.21E+02	1.54E-01	1.27E-03	4.86E-02	4.02E-04	--	--	1.67E-03
Aliph>C12-C16 - F2	1.21E+02	1.88E-01	1.55E-03	2.99E-02	2.48E-04	--	--	1.80E-03
Arom>C10-C12 - F2	2.59E+01	3.84E-02	1.48E-03	6.97E-02	2.69E-03	--	--	4.18E-03
Arom>C12-C16 - F2	2.59E+01	4.70E-02	1.81E-03	7.28E-02	2.81E-03	--	--	4.63E-03
F2 - Total								--
Aliph>C16-C21 - F3	2.42E+02	2.61E+00	1.08E-02	1.28E-01	5.31E-04	--	--	1.13E-02
Aliph>C21-C34 - F3	2.42E+02	1.12E+00	4.62E-03	5.50E-02	2.28E-04	--	--	4.85E-03
Arom>C16-C21 - F3	5.18E+01	6.52E-01	1.26E-02	3.88E-01	7.49E-03	--	--	2.01E-02
Arom>C21-C34 - F3	5.18E+01	2.79E-01	5.39E-03	1.03E-01	2.00E-03	--	--	7.39E-03
F3 - Total								--
Aliph>C34-C50 - F4	2.24E+03	2.09E+00	9.33E-04	2.06E-02	9.19E-06	--	--	9.43E-04
Arom>C34-C50 - F4	2.33E+02	5.23E-01	2.25E-03	5.29E-02	2.27E-04	--	--	2.47E-03
F4 - Total								--
PAHs								
Naphthalene	1.88E+02	1.39E-04	7.42E-07	1.08E-02	5.73E-05	--	--	5.80E-05
PCBs								
Aroclor 1254 (Total PCBs)	6.39E-01	1.06E-04	1.66E-04	2.36E-04	3.70E-04	--	--	5.35E-04
Inorganics								
Arsenic	1.18E+00	5.67E-03	4.79E-03	4.86E-03	4.11E-03	1.10E-04	9.33E-05	9.00E-03
Barium	2.78E+01	5.24E-01	1.89E-02	2.85E-01	1.03E-02	9.01E-03	3.24E-04	2.95E-02
Cadmium	1.09E+01	3.28E-03	3.01E-04	1.64E-02	1.51E-03	2.10E-05	1.93E-06	1.81E-03
Copper	1.76E+01	4.18E-02	2.38E-03	1.52E-01	8.68E-03	--	--	1.11E-02
Lead	8.71E+01	4.35E-02	4.99E-04	2.96E-02	3.40E-04	4.78E-04	5.48E-06	8.44E-04
Lithium	2.05E+01	--	--	--	--	1.27E-03	6.20E-05	6.20E-05
Manganese	3.09E+02	5.57E-18	1.80E-20	6.00E-17	1.94E-19	1.32E-02	4.27E-05	4.27E-05
Nickel	8.71E+01	2.96E-02	3.40E-04	1.33E-02	1.53E-04	6.60E-04	7.58E-06	5.01E-04
Silver	3.40E+00	2.79E-04	8.19E-05	9.14E-04	2.69E-04	1.89E-05	5.56E-06	3.56E-04
Uranium	5.76E+00	--	--	--	--	1.62E-03	2.81E-04	2.81E-04
Zinc	3.48E+02	2.69E-01	7.72E-04	1.47E+00	4.21E-03	8.89E-03	2.55E-05	5.00E-03

Ecological Hazard Quotients for the Rock Ptarmigan Exposed to Constituents of Interest in the CAM-D Barrel Dump

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Invertebrate Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
BTEX										
Ethylbenzene	8.72E+01	1.46E-04	1.68E-06	8.77E-05	1.01E-06	9.18E-06	1.05E-07	--	--	2.79E-06
Toluene	9.08E+01	1.89E-04	2.08E-06	1.21E-04	1.33E-06	1.36E-05	1.49E-07	--	--	3.56E-06
Xylenes	9.01E+02	1.46E-04	1.62E-07	8.58E-05	9.52E-08	9.07E-06	1.01E-08	--	--	2.68E-07
TPH - CCME CWS										
Aliph>C06-C08 - F1	6.50E+01	1.53E-02	2.36E-04	1.46E-02	2.25E-04	7.43E-03	1.14E-04	--	--	5.75E-04
Aliph>C08-C10 - F1	6.50E+01	1.00E-02	1.54E-04	5.94E-03	9.14E-05	3.72E-03	5.72E-05	--	--	3.03E-04
Arom>C08-C10 - F1	2.17E+01	2.50E-03	1.16E-04	2.95E-03	1.36E-04	1.37E-03	6.33E-05	--	--	3.15E-04
F1 - Total										--
Aliph>C10-C12 - F2	6.50E+01	4.03E-01	6.21E-03	5.95E-02	9.16E-04	1.14E-01	1.76E-03	--	--	8.88E-03
Aliph>C12-C16 - F2	6.50E+01	4.93E-01	7.59E-03	3.67E-02	5.64E-04	7.57E-02	1.17E-03	--	--	9.32E-03
Arom>C10-C12 - F2	2.17E+01	1.01E-01	4.66E-03	8.54E-02	3.95E-03	5.20E-02	2.40E-03	--	--	1.10E-02
Arom>C12-C16 - F2	2.17E+01	1.23E-01	5.69E-03	8.92E-02	4.12E-03	5.81E-02	2.68E-03	--	--	1.25E-02
F2 - Total										--
Aliph>C16-C21 - F3	8.66E+01	6.84E+00	7.90E-02	1.57E-01	1.81E-03	1.40E+00	1.62E-02	--	--	9.70E-02
Aliph>C21-C34 - F3	8.66E+01	2.93E+00	3.38E-02	6.74E-02	7.78E-04	3.42E-01	3.94E-03	--	--	3.86E-02
Arom>C16-C21 - F3	4.33E+01	1.71E+00	3.95E-02	4.75E-01	1.10E-02	5.55E-01	1.28E-02	--	--	6.33E-02
Arom>C21-C34 - F3	4.33E+01	7.33E-01	1.69E-02	1.27E-01	2.93E-03	4.54E-01	1.05E-02	--	--	3.03E-02
F3 - Total										--
Aliph>C34-C50 - F4	1.30E+02	5.49E+00	4.23E-02	2.53E-02	1.94E-04	2.70E-01	2.08E-03	--	--	4.46E-02
Arom>C34-C50 - F4	1.73E+01	1.37E+00	7.93E-02	6.48E-02	3.74E-03	2.41E+00	1.39E-01	--	--	2.22E-01
F4 - Total										--
PAHs										
Naphthalene	6.99E+01	3.66E-04	5.23E-06	1.32E-02	1.89E-04	2.14E-04	3.06E-06	--	--	1.97E-04
PCBs										
Aroclor 1254 (Total PCBs)	1.56E+00	2.78E-04	1.78E-04	2.89E-04	1.86E-04	2.86E-04	1.83E-04	--	--	5.47E-04
Inorganics										
Arsenic	1.11E+01	1.49E-02	1.34E-03	5.96E-03	5.36E-04	3.76E-03	3.38E-04	6.38E-05	5.74E-06	2.22E-03
Barium	1.10E+02	1.37E+00	1.25E-02	3.50E-01	3.17E-03	1.32E-01	1.19E-03	5.21E-03	4.72E-05	1.69E-02
Cadmium	1.73E+01	8.60E-03	4.97E-04	2.01E-02	1.16E-03	8.35E-02	4.82E-03	1.21E-05	6.99E-07	6.48E-03
Copper	8.15E+01	1.10E-01	1.35E-03	1.87E-01	2.29E-03	1.40E-01	1.71E-03	--	--	5.35E-03
Lead	1.43E+01	1.14E-01	7.97E-03	3.63E-02	2.54E-03	6.49E-02	4.53E-03	2.76E-04	1.93E-05	1.51E-02
Lithium	4.02E+00	--	--	--	--	--	--	7.33E-04	1.82E-04	1.82E-04
Manganese	7.16E+03	1.46E-17	2.04E-21	7.35E-17	1.03E-20	4.03E-13	5.63E-17	7.63E-03	1.07E-06	1.07E-06
Nickel	9.27E+01	7.78E-02	8.39E-04	1.63E-02	1.76E-04	3.94E-01	4.25E-03	3.81E-04	4.12E-06	5.27E-03
Silver	6.32E+00	7.31E-04	1.16E-04	1.12E-03	1.77E-04	1.58E-03	2.49E-04	1.09E-05	1.73E-06	5.44E-04
Uranium	1.33E+02	--	--	--	--	--	--	9.35E-04	7.05E-06	7.05E-06
Zinc	1.01E+02	7.06E-01	6.97E-03	1.80E+00	1.77E-02	4.69E+00	4.63E-02	5.14E-03	5.07E-05	7.10E-02

**Ecological Hazard Quotients for the Collared Lemming Exposed to Constituents of Interest in the CAM-D Simpson Lake Shoreline Area**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs								
Aroclor 1254 (Total PCBs)	6.39E-01	5.57E-04	8.73E-04	1.24E-03	1.95E-03	--	--	2.82E-03
Inorganics								
Arsenic	1.18E+00	5.57E-03	4.71E-03	4.81E-03	4.07E-03	1.10E-04	9.33E-05	8.87E-03
Barium	2.78E+01	1.56E-01	5.62E-03	8.50E-02	3.06E-03	9.01E-03	3.24E-04	9.01E-03
Cadmium	1.09E+01	2.79E-03	2.56E-04	1.50E-02	1.38E-03	2.10E-05	1.93E-06	1.64E-03
Copper	1.76E+01	9.75E-02	5.56E-03	2.13E-01	1.21E-02	--	--	1.77E-02
Lead	8.71E+01	9.98E-01	1.15E-02	1.72E-01	1.97E-03	4.78E-04	5.48E-06	1.34E-02
Lithium	2.05E+01	--	--	--	--	1.27E-03	6.20E-05	6.20E-05
Manganese	3.09E+02	5.57E-18	1.80E-20	6.00E-17	1.94E-19	1.32E-02	4.27E-05	4.27E-05
Nickel	8.71E+01	3.01E-02	3.46E-04	1.35E-02	1.55E-04	6.60E-04	7.58E-06	5.08E-04
Silver	3.40E+00	5.57E-04	1.64E-04	1.83E-03	5.38E-04	1.89E-05	5.56E-06	7.07E-04
Uranium	5.76E+00	--	--	--	--	1.62E-03	2.81E-04	2.81E-04
Zinc	3.48E+02	3.01E+00	8.64E-03	5.60E+00	1.61E-02	8.89E-03	2.55E-05	2.47E-02

Ecological Hazard Quotients for the Collared Lemming Exposed to Constituents of Interest in the CAM-D Simpson Lake Shoreline Area

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Invertebrate Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs										
Aroclor 1254 (Total PCBs)	1.56E+00	1.46E-03	9.38E-04	1.52E-03	9.76E-04	2.74E-03	1.76E-03	--	--	3.67E-03
Inorganics										
Arsenic	1.11E+01	1.46E-02	1.32E-03	5.90E-03	5.30E-04	3.71E-03	3.34E-04	6.38E-05	5.74E-06	2.19E-03
Barium	1.10E+02	4.10E-01	3.72E-03	1.04E-01	9.45E-04	3.92E-02	3.55E-04	5.21E-03	4.72E-05	5.06E-03
Cadmium	1.73E+01	7.31E-03	4.22E-04	1.84E-02	1.06E-03	7.34E-02	4.24E-03	1.21E-05	6.99E-07	5.72E-03
Copper	8.15E+01	2.56E-01	3.14E-03	2.61E-01	3.20E-03	1.75E-01	2.14E-03	--	--	8.48E-03
Lead	1.43E+01	2.62E+00	1.83E-01	2.10E-01	1.47E-02	8.13E-01	5.68E-02	2.76E-04	1.93E-05	2.55E-01
Lithium	4.02E+00	--	--	--	--	--	--	7.33E-04	1.82E-04	1.82E-04
Manganese	7.16E+03	1.46E-17	2.04E-21	7.35E-17	1.03E-20	4.03E-13	5.63E-17	7.63E-03	1.07E-06	1.07E-06
Nickel	9.27E+01	7.90E-02	8.52E-04	1.65E-02	1.78E-04	3.92E-01	4.23E-03	3.81E-04	4.12E-06	5.26E-03
Silver	6.32E+00	1.46E-03	2.31E-04	2.24E-03	3.54E-04	3.15E-03	4.98E-04	1.09E-05	1.73E-06	1.09E-03
Uranium	1.33E+02	--	--	--	--	--	--	9.35E-04	7.05E-06	7.05E-06
Zinc	1.01E+02	7.90E+00	7.80E-02	6.86E+00	6.77E-02	1.04E+01	1.02E-01	5.14E-03	5.07E-05	2.48E-01



**Ecological Hazard Quotients for the Collared Lemming Exposed to Constituents of Interest in the Outfall Area**

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs								
Aroclor 1254 (Total PCBs)	6.39E-01	3.57E-03	5.58E-03	7.95E-03	1.24E-02	--	--	1.80E-02
Inorganics								
Arsenic	1.18E+00	7.21E-02	6.09E-02	2.04E-02	1.72E-02	1.10E-04	9.33E-05	7.83E-02
Barium	2.78E+01	4.51E-01	1.63E-02	2.46E-01	8.86E-03	9.01E-03	3.24E-04	2.54E-02
Cadmium	1.09E+01	4.63E-03	4.25E-04	1.98E-02	1.82E-03	2.10E-05	1.93E-06	2.25E-03
Copper	1.76E+01	3.95E-01	2.25E-02	3.69E-01	2.10E-02	--	--	4.35E-02
Lead	8.71E+01	6.79E-02	7.80E-04	3.80E-02	4.37E-04	4.78E-04	5.48E-06	1.22E-03
Lithium	2.05E+01	--	--	--	--	1.27E-03	6.20E-05	6.20E-05
Manganese	3.09E+02	5.57E-18	1.80E-20	6.00E-17	1.94E-19	1.32E-02	4.27E-05	4.27E-05
Nickel	8.71E+01	1.50E-01	1.72E-03	4.49E-02	5.15E-04	6.60E-04	7.58E-06	2.25E-03
Silver	3.40E+00	5.57E-04	1.64E-04	1.83E-03	5.38E-04	1.89E-05	5.56E-06	7.07E-04
Uranium	5.76E+00	--	--	--	--	1.62E-03	2.81E-04	2.81E-04
Zinc	3.48E+02	2.26E+00	6.50E-03	4.78E+00	1.37E-02	8.89E-03	2.55E-05	2.02E-02

Ecological Hazard Quotients for the Rock Ptarmigan Exposed to Constituents of Interest in the Outfall Area

Constituent	Reference Toxicity Dose (mg/kg-day)	Surface Soil Ingestion		Terrestrial Plant Ingestion		Terrestrial Invertebrate Ingestion		Surface Water Ingestion		Total Ecological Hazard Quotients and Index
		Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	Average Daily Dose (mg/kg-day)	Hazard Quotient	
PCBs										
Aroclor 1254 (Total PCBs)	1.56E+00	9.36E-03	6.00E-03	9.74E-03	6.25E-03	3.43E-02	2.20E-02	--	--	3.43E-02
Inorganics										
Aluminum	6.90E+02	--	--	--	--	--	--	--	--	--
Arsenic	1.11E+01	1.89E-01	1.70E-02	2.50E-02	2.25E-03	2.26E-02	2.03E-03	6.38E-05	5.74E-06	2.13E-02
Barium	1.10E+02	1.18E+00	1.08E-02	3.01E-01	2.73E-03	1.13E-01	1.03E-03	5.21E-03	4.72E-05	1.46E-02
Cadmium	1.73E+01	1.21E-02	7.01E-04	2.43E-02	1.40E-03	1.10E-01	6.34E-03	1.21E-05	6.99E-07	8.44E-03
Copper	8.15E+01	1.04E+00	1.27E-02	4.52E-01	5.55E-03	2.53E-01	3.10E-03	--	--	2.14E-02
Iron	3.96E+01	--	--	--	--	--	--	--	--	--
Lead	1.43E+01	1.78E-01	1.25E-02	4.66E-02	3.26E-03	9.30E-02	6.50E-03	2.76E-04	1.93E-05	2.22E-02
Lithium	4.02E+00	--	--	--	--	--	--	7.33E-04	1.82E-04	1.82E-04
Manganese	7.16E+03	1.46E-17	2.04E-21	7.35E-17	1.03E-20	4.03E-13	5.63E-17	7.63E-03	1.07E-06	1.07E-06
Nickel	9.27E+01	3.94E-01	4.25E-03	5.49E-02	5.93E-04	2.58E-01	2.78E-03	3.81E-04	4.12E-06	7.63E-03
Silver	6.32E+00	1.46E-03	2.31E-04	2.24E-03	3.54E-04	3.15E-03	4.98E-04	1.09E-05	1.73E-06	1.09E-03
Uranium	1.33E+02	--	--	--	--	--	--	9.35E-04	7.05E-06	7.05E-06
Zinc	1.01E+02	5.94E+00	5.87E-02	5.86E+00	5.78E-02	9.43E+00	9.31E-02	5.14E-03	5.07E-05	2.10E-01