

# FINAL REPORT

Human Health and Ecological Risk  
Assessment for a Former Navigational Aid  
and Weather Station located at Radio Island,  
Nunavut

PUBLIC WORKS AND  
GOVERNMENT SERVICES CANADA

**PROJECT NO. 1005418**



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**REPORT TO: Public Works and  
Government Services Canada  
Environmental Services, Western Region  
5<sup>th</sup> Floor, 10025 Jasper Avenue  
Edmonton, Alberta T5J 1S6**

**FOR: Human Health and Ecological Risk  
Assessment for a Former Navigational  
Aid and Weather Station located at  
Radio Island, Nunavut**

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**January 4, 2006**

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## EXECUTIVE SUMMARY

Jacques Whitford Limited (Jacques Whitford) was retained by Public Works and Government Services Canada (PWGSC) on behalf of Indian and Northern Affairs Canada (INAC) to conduct a human health and ecological risk assessment (HHERA) for a former navigational aid and weather station located at Radio Island, Nunavut. The primary objective of this study was to evaluate whether known concentrations of chemicals found in on-site surface soil, surface water and vegetation would present a significant risk to human or ecological receptors.

### Data Compilation

The primary source of data for this risk assessment was supplied by the Environmental Sciences Group (ESG) at the Royal Military College, who on behalf of INAC, conducted the field investigation and sampling in August 1996. Environmental data consisted of soil, surface water and vegetation samples collected from around the site. In addition to the 1996 sampling, Earth Tech Canada Inc. (Earth Tech) collected additional soil data at the same sampling locations previously chosen by the ESG. The soil and water data collected by the ESG and Earth Tech were consolidated into one data set and then screened for use in the HHERA. For the purposes of the HHERA, only soil samples that accurately reflect concentrations in the upper 10 to 15 cm below ground surface are relevant to potential exposures. This was carried out to ensure that the data used in the HHERA is representative of surface soil to which 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, MOE, DCC). 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 (ERA) they were screened specifically against ecologically-based generic guidelines. These guidelines were obtained from CCME, the Ontario Ministry of the Environment or the United States Environmental Protection Agency (US EPA). A number of metals were also screened using Defense Construction Canada (DCC) Dew Line Cleanup Criteria, as these guidelines are more site-specific (i.e., developed for arctic ecosystem). All exceedances of DCC guidelines were carried forward into the risk assessment process.

Based on screening using human health-based criteria, the chemicals carried forward into the HHRA as CoPCs were antimony, arsenic, barium, beryllium, cadmium, chromium, cobalt, copper, lead, mercury, nickel, zinc, total petroleum hydrocarbon (TPH) fractions F2 and F3, and total polychlorinated biphenyls (PCBs).

The chemicals carried forward into the ERA as CoPCs included antimony, arsenic, barium, beryllium, boron, cadmium, chromium, cobalt, copper, lead, mercury, nickel, selenium, silver, zinc, TPH fractions F1, F2, F3, F4, and total PCBs.



## Exposure Scenarios

The study area was divided into two areas, A and B. Area A located on the western end of Radio Island was comprised of the main site which included two intact buildings, a winch shed, a main house, and the foundations of two other structures. One foundation consists of eight concrete supports and the other houses a helipad. Approximately 35 m to the southwest of the main site is the area encompassing the generator building foundation. Area B represents the beach area at Radio Island. The beach area is fairly small receiving drainage from the main site. Located to the east of the beach are the remains of a burnt house. A large pile of coal, a suspected dump, and two barrel piles consisting of empty rusted barrels are also present in this area.

Because of the remote location of Radio Island and the probable use of the site by Inuit for traditional purposes, the conventional land use scenarios (residential, parkland, commercial and industrial) were expanded to incorporate a new land use scenario, Traditional 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 INAC.

The Traditional Land Use scenario was adopted as set out by Gartner Lee and Cantox (1998), with minor modifications. The original Traditional Land Use designation consisted 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 actual Inuit behavior. 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 during non-snow covered months, which resulted in the most extensive exposure scenario for human receptors. Detailed exposure values are presented in Section 4.3.

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 impacted with non-carcinogenic CoPCs by inadvertent ingestion, dermal contact, and inhalation of surface soil/dust. Additional exposure occurs through the ingestion of wild game and through ingestion and dermal contact with surface water.
- 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 CoPCs by inadvertent ingestion, dermal contact, and inhalation of surface soil/dust. Additional exposure occurs through the ingestion of wild game and through ingestion and dermal contact with surface water.

## ***Ecological Health***

The risks associated with exposure to contaminated surface soil and surface 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 surface water, as well as uptake via ingestion of terrestrial plants, terrestrial invertebrates and small mammals. The major exposure pathway considered was ingestion. Inhalation and dermal absorption were also possible exposure pathways for surface soil and surface water but these were considered to be relatively minor as compared to direct 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 valued ecosystem components (VECs) selected for evaluation in the ERA were the rock ptarmigan, snowy owl, ermine, collared lemming, arctic hare, and arctic fox. These receptors are considered to be representative of indigenous wildlife at the Radio Island 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 (ILCR) 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 the risk assessment.
- EPCs of ecological health-based CoPCs generated HQ values less than 1.0 with the exception of chromium, zinc and TPH. The VECs that were the most sensitive were ermine(TPH), collared lemming(chromium), and rock ptarmigan(zinc).
- Site specific target levels (SSTLs) were calculated for each of these CoPCs. The SSTLs were calculated by setting the HQ at 1.0, and determining the corresponding surface soil EPC for that HQ, using a backward calculation.

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# Table of Contents

<b>EXECUTIVE SUMMARY .....</b>	<b>i</b>
<b>1.0 INTRODUCTION .....</b>	<b>1</b>
1.1 Scope and Objective.....	1
1.2 Rationale for Site-Specific Risk Assessment.....	2
<b>2.0 STUDY BACKGROUND .....</b>	<b>3</b>
2.1 Site Description.....	3
2.2 Study Areas .....	3
2.3 Objective of the HHERA .....	4
2.4 Previous Environmental Investigations.....	4
2.5 Traditional Land Use.....	4
<b>3.0 DATA COMPILATION.....</b>	<b>5</b>
3.1 Sources .....	5
3.2 Selection of Data .....	5
3.2.1 Division of Data .....	5
3.3 Description and Statistical Analysis of Data .....	5
<b>4.0 HUMAN HEALTH RISK ASSESSMENT.....</b>	<b>6</b>
4.1 Risk Assessment Framework .....	6
4.2 Chemical Screening.....	8
4.2.1 Identification of Chemicals of Potential Concern .....	8
4.2.2 Summary of Chemicals of Potential Concern .....	12
4.2.3 Receptor Identification.....	12
4.2.4 Exposure Pathway Assessment .....	13
4.2.5 Potential Transport Pathways.....	13
4.2.6 Potential Exposure Mechanisms .....	13
4.2.7 Human Receptor Exposure Scenarios .....	14
4.2.8 Qualitative Risk Characterization .....	15
4.3 Receptor Characteristics .....	16
4.4 Toxicity Assessment.....	18
4.4.1 Selection of Toxicity Reference Values .....	19
4.4.2 Bioavailability.....	21
4.5 Risk Characterization.....	21
4.5.1 Non-Carcinogenic Risk Estimates .....	23
4.5.2 Carcinogenic Risk Estimates .....	24
4.5.3 Summary of Site Risk .....	25
4.6 Human Health Site-Specific Target Levels (SSTL <sub>HH</sub> ) .....	25
4.7 Uncertainty Analysis .....	25
4.7.1 Uncertainties in Toxicological Information .....	26
4.7.2 Summation of Hazards for a Single Compound .....	27
4.7.3 Summation of Hazards for Multiple Compounds .....	27
4.7.4 Modeling Assumptions .....	27

<b>5.0</b>	<b>ECOLOGICAL RISK ASSESSMENT.....</b>	<b>30</b>
5.1	Objectives.....	30
5.2	Ecological Risk Assessment Framework.....	31
5.3	Chemical Screening.....	32
5.3.1	Identifying Chemicals of Potential Concern.....	32
5.3.2	Summary of Chemicals of Potential Concern.....	37
5.4	Receptor Identification.....	37
5.5	Valued Environmental Components (VECS).....	38
5.5.1	Rare, Threatened, or Endangered Species and Species of Special Concern.....	40
5.6	Exposure Assessment.....	41
5.6.1	Potential Exposure Pathways.....	41
5.6.2	Conceptual Model.....	41
5.6.3	Selection of Assessment and Measurement Endpoints.....	43
5.7	Risk Characterization.....	44
5.7.1	Approach.....	44
5.7.2	Determination of Media to Biota Uptake Factors.....	45
5.7.3	Determination of Reference Toxicity Doses.....	46
5.7.4	Risk Characterization of Avian Receptors.....	46
5.7.4.1	Risk Estimates for Rock Ptarmigan.....	46
5.7.4.2	Risk Estimates for Snowy Owl.....	48
5.7.5	Risk Characterization of Mammalian Receptors.....	49
5.7.5.1	Risk Estimates for Collared Lemming.....	49
5.7.5.2	Risk Estimates for Ermine.....	50
5.7.5.3	Risk Estimates for Arctic Fox.....	52
5.7.5.4	Risk Estimates for Arctic Hare.....	52
5.8	Ecological Site-Specific Target Levels.....	54
5.9	Uncertainty Analysis.....	54
5.9.1	Data Limitations.....	54
5.9.2	Selection of CoPCs.....	55
5.9.3	Chemical Speciation.....	55
5.9.4	Food Chain Interactions.....	56
5.9.5	Wildlife Exposure Factors.....	56
5.9.6	Habitat Survey and VEC Selection.....	56
5.9.7	Receptor-Specific Toxicity Data.....	56
5.9.8	Measurement Endpoints from the Toxicity Data.....	57
5.10	Summary Of Uncertainty Analysis.....	57
<b>6.0</b>	<b>CONCLUSIONS.....</b>	<b>57</b>
<b>7.0</b>	<b>CLOSURE.....</b>	<b>58</b>
<b>8.0</b>	<b>REFERENCES.....</b>	<b>60</b>

---

## List of Tables

Table 4-1:	Human Health Soil Screening for Area A .....	9
Table 4-2:	Human Health Surface Water Screening .....	10
Table 4-3:	Human Health Soil Screening for Area B .....	11
Table 4-4:	Summary of CoPCs and EPCs used in HHRA.....	12
Table 4-5:	Exposure Definitions .....	14
Table 4-6:	Potential Exposure Scenarios – Traditional Land User .....	15
Table 4-7:	Summary of Receptor Characteristics.....	17
Table 4-8:	Summary of Receptor Characteristics for each age groups.....	18
Table 4-9:	Selected Carcinogenic Toxicity Values .....	19
Table 4-10:	Selected Non–Carcinogenic Toxicity Values.....	20
Table 4-11:	Selected Relative Bioavailability Factors.....	21
Table 4-12:	Hazard Quotients for Non-Carcinogens – Toddler, Area A .....	23
Table 4-13:	Hazard Quotients for Non-Carcinogens – Toddler, Area B .....	24
Table 4-14:	Incremental Lifetime Cancer Risks for Carcinogenic CoPCs in Area A .....	24
Table 4-15:	Incremental Lifetime Cancer Risks for Carcinogenic CoPCs in Area B .....	25
Table 4-16:	Modeling Assumptions .....	28
Table 5-1:	Ecological Soil Screening for Area A.....	34
Table 5-2:	Surface Water Screening .....	35
Table 5-3:	Ecological Soil Screening for Area B.....	36
Table 5-4:	Summary of CoPCs and EPCs Used in ERA.....	37
Table 5-5:	Species at Risk.....	40
Table 5-6:	Ecological Hazard Quotients for Rock Ptarmigan .....	47
Table 5-7:	Ecological Hazard Quotients for Snowy Owl.....	48
Table 5-8:	Ecological Hazard Quotients for Collared Lemming.....	50
Table 5-9:	Ecological Hazard Quotients for Ermine.....	51
Table 5-10:	Ecological Hazard Quotients for Arctic Fox.....	52
Table 5-11:	Ecological Hazard Quotients for Arctic Hare .....	53
Table 5-12:	Site Specific Target Levels in Surface Soils at Radio Island .....	54

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## List of Figures

Figure 1-1:	Relationship of Risk Assessment Components .....	1
Figure 4-1:	Human Health Conceptual Model .....	16
Figure 5-1:	ERA Conceptual Model .....	42



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## List of Drawings

Drawing 1: Site Location Plan .....	Appendix A
Drawing 2: Site Overview .....	Appendix A
Drawing 3: Impacted Zone of Area A .....	Appendix A
Drawing 4: Impacted Zone of Area B .....	Appendix A

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## List of Appendices

APPENDIX A	Site Maps
APPENDIX B	Toxicity Profiles
APPENDIX C	HHRA Model Equations, Inputs and Outputs
APPENDIX D	Species Inventory
APPENDIX E	ERA Model Inputs and Outputs

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## 1.0 INTRODUCTION

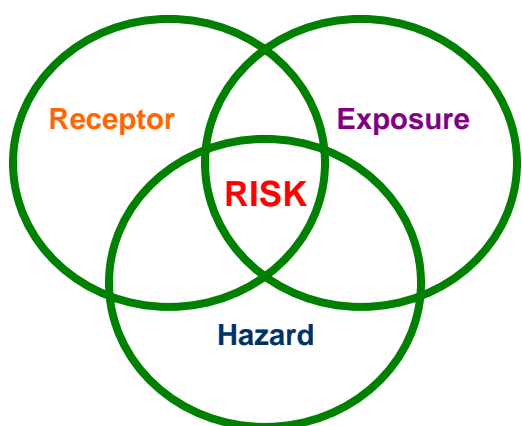
Jacques Whitford Limited was retained by Public Works and Government Services Canada (PWGSC) on behalf of Indian and Northern Affairs Canada (INAC) to conduct a quantitative human health and ecological risk assessment (HHERA) for a former navigational aid and weather station located on Radio Island, Nunavut (site).

Previous site investigations (SENES, 2003; PWGSC, 2002; ESG, 1997) identified several contaminants of potential concern (CoPCs) in soils above federal residential/parkland guidelines. The overall goal of this HHERA is to evaluate the current risk associated with chemical concentrations found on-site and, if needed, develop surface site specific target levels (SSTLs) for Radio Island below which no adverse effects would be expected.

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### 1.1 Scope and Objective

A detailed quantitative human health risk assessment (HHRA) and a quantitative ecological risk assessment (ERA) were conducted.



The basic purpose of the assessment was to determine concentrations of CoPCs in soil below which no adverse health effects would be expected. SSTLs will be established and used in preparation for reclamation work at the site. To meet this objective, a widely accepted risk assessment framework (Figure 1-1) was adopted in which potential hazards, exposure pathways, and receptors are evaluated to determine if a risk is present. The human health and ecological risk assessment framework comprises the following major components:

**Figure 1-1: Relationship of Risk Assessment Components**<sup>1</sup>

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

**Receptor Identification:** Identification of the human receptors and biota that may be exposed to the above hazard(s).

**Toxicity Assessment:** Identification of published, scientifically reviewed toxicity values against which exposures can be compared.

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

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

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<sup>1</sup> Canadian Council of Ministers of the Environment, The National Contaminated Sites Remediation Program, "A Framework for Ecological Risk Assessment General Guidance", March 1996.

**SSTL Determination:** The determination of concentrations at the site below which no adverse effects would be expected.

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

The derivation of SSTLs presented in this report follows the general methodology as outlined above.

Specific tasks included:

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

It is important to note that the assessment does not evaluate potential health issues that may have occurred in the past, rather it is designed only to evaluate current and potential future exposures to chemicals in soil, based on present day conditions and assumed future post-reclamation conditions. Previous site investigations (SENES, 2003) did not identify any CoPCs in water, therefore, any potential risks to aquatic receptors was outside the scope of this HHERA.

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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 1999a). These guidelines are conservative benchmarks for screening purposes. If soil concentrations are less than these guidelines, then the potential for human health and ecological effects is negligible. If soil concentrations exceed these guidelines it does not necessarily follow that unacceptable risks exist. The generic guidelines are intentionally conservative and do not take into account regional or site-specific information (e.g., background soil conditions) and are not appropriate for every site or region of the country.

With this in mind, in 1996 the CCME published two documents (CCME 1996a, 1996b), thereby acknowledging that these guidelines are not “set in stone” but may be modified in some instances if supported by sound reasoning and/or by the provision of site-specific data. 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 the site is a more accurate way of assessing the human health significance of contaminant concentrations in soil in the area.

Soil concentrations were initially evaluated using the Canadian Environmental Quality Guidelines published by the CCME in 1999, revised 2004 (CCME Guidelines). SSTLs were derived in accordance with the methods presented in the *Guidance Manual for Developing Site-Specific Soil Quality Objectives for Contaminated Sites in Canada* (CCME, 1996a) and *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 1996b).

The specific methods employed to develop the SSTLs are consistent with the CCME protocols as referenced above, and with standard Canadian human health and ecological risk assessment methodologies.

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## 2.0 STUDY BACKGROUND

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### 2.1 Site Description

Radio Island is located 340 km southeast of Iqaluit, Nunavut off the tip of Resolution Island (which itself is located on the southeastern tip of Baffin Island) as shown in Drawing 1 in **Appendix A**. From 1929 to 1961, the Canadian Department of Transport operated a navigational aid and weather station at the site (originally known as Resolution Island). In 1961, the station was moved to Cape Warwick, Resolution Island, Nunavut.

The site, which is composed of Canadian Shield bedrock, is approximately 1 km long and 0.5 km wide (approximately 50 hectares). The terrain consists of tilted bedrock with parallel rock ridges, knolls and gullies. The only soils identified at the site are located in the gullies and valleys formed by the bedrock and represents only 10% of the total area. Surface drainage at the site flows along the gullies to the ocean. Figure 2-2 provides an overview of the site.

Currently the site consists of two standing buildings, the remains of three other buildings, two helipads, and a beacon tower still operated by the Canadian Coast Guard at the south end of the island. Hazardous and non-hazardous debris are scattered throughout the site. The site is accessible by sea only during the summer at Acadia Cove and by air only by helicopter. Flora on the site is limited to gullies and valleys, with mosses found throughout the site where soil is present. Marine mammals are common to this region, including walrus, seals and whales. The site is also known to be a denning area for polar bears.

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### 2.2 Study Areas

Samples from previous site investigations were collected from three distinct areas: the main site (Area A), the generator building foundation (Area B), and the beach (Area C). For the purposes of this risk assessment, areas A and B were combined into one area (i.e., Area A) and area C was relabeled "Area B". In terms of the potential risk to receptors (both human and ecological), the main site and generator building areas were within short proximity of each other, and therefore should be considered as part of the same area. Drawing 2 in **Appendix A** shows the site overview including Area A and B.

#### Area A

The main site is located on the western side of Radio Island. It is comprised of two intact buildings, a winch shed, a main house, and the foundations of two other structures. One foundation consists of eight concrete supports and the other houses a helipad. All four structures are in close proximity and have common drainage pathways. Approximately 35 m to the southwest of the main site is the generator building foundation. The foundation is 12 m by 6 m and contains the remains of three generators and one boiler. The drainage from the foundation is to the west and south east of the foundation. The drainage to the west reaches the sea at approximately 30 m while the drainage to the southeast drains to the interior of the island. The operational Canadian Coast Guard radio beacon is located to the southwest of the generator building foundation. The impacted zone of Area A is assumed on Drawing 3 in **Appendix A**.

#### Area B

The beach area at Radio Island drains into Acadia Cove to the north. The beach area is fairly small receiving drainage from the main site. Located on the east side of the beach are the remains of a burnt house. A large pile of coal, a suspected dump, and two barrel piles consisting of empty rusted barrels

are also present in this area. Drawing 4 in **Appendix A** provides an assumed boundary of the impacted zone of Area B.

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## **2.3 Objective of the HHERA**

Previous site investigations (SENES, 2003; PWGSC, 2002; ESG, 1997) identified several metal and organic contaminants in soil that exceeded generic soil quality criteria provided in the CCME (2001) guidelines.

This HHERA will determine allowable risk-based levels (i.e., site-specific target levels) for the CoPCs in the soil such that the impacted soil could remain on the site without compromising the health of both human and ecological receptors that access the site. In addition, the HHERA was to determine site-specific target levels (SSTLs) for the selected contaminants.

The application of the SSTLs to the contaminant-impacted soil would allow impacted soil to remain on the site and thus reduce the amount of soil that would be removed for remediation. This is a more efficient and less expensive soil management alternative compared to the expensive off-site disposal of the contaminated soil that exceeds the CCME generic soil quality criteria suggested by PWGSC (2002), while still providing for the protection of human and ecological health.

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## **2.4 Previous Environmental Investigations**

Previous environmental investigations of Radio Island include the following:

- Public Works and Government Services Canada (PWGSC). 2002. Remedial Action Plan Former Navigational Aid and Weather Station Radio Island, Nunavut;
- Environmental Services Group (ESG) (Royal Military College). 1997. Environmental Assessment of Radio Island, NWT;
- Earth Tech Canada Inc. 2001. Environmental Site Delineation and Material Inventory; and
- SENES Consultants Limited. 2003. Human Health Screening Level Risk Assessment for Radio Island Former Military Site.

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## **2.5 Traditional Land Use**

In their 1998 Risk Assessment, Gartner Lee and Cantox argue that due to the northern location of the site and the probable use of the site by Inuit for traditional purposes, the conventional land use categories (residential, parkland, commercial, and industrial) must be expanded to incorporate an additional land use, which they term "Traditional Land Use". The parameters of this land use were developed after consultation with residents of the Eastern Arctic, the Qikiqtaaluk Corporation and INAC. "Traditional Land Use" is the receptor scenario used in the human health risk assessment.

The Traditional Land Use scenario was adopted as set out by Gartner Lee and Cantox (1998), with minor modifications. The original Traditional Land Use designation consisted 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 actual Inuit behavior. 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 during non-snow covered months, which resulted in the most extensive exposure scenario for human receptors. Detailed exposure values are presented in Section 4.3.

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## 3.0 DATA COMPILATION

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### 3.1 Sources

The primary source of data for this risk assessment was supplied by the environmental services group (ESG) at the Royal Military College, who on behalf of INAC, conducted the field investigation and sampling in August 1996. Environmental data consisted of soil, surface water and vegetation samples collected from around the site. A detailed list of samples and their locations are presented by the ESG in their report (1997). In addition to the 1996 sampling, Earth Tech Canada Inc. (Earth Tech) collected additional soil data at the same sampling locations chosen by the ESG (1997). A description of samples and sample locations can be found in their report (Earth Tech Canada Inc., 2001).

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### 3.2 Selection of Data

The soil and water data collected by the ESG (1997) and Earth Tech (2001) were consolidated into one data set and then screened for use in the HHERA. For the purposes of the HHERA, only soil samples that accurately reflect concentrations in the upper 10 to 15 cm below ground surface are relevant to potential exposures. This was done to ensure that the data used were representative of surface soil and not heavily influenced by subsurface soil characteristics.

Measured total PCBs in soil samples collected at the beach area (Drawings 2 and 4, **Appendix A**) by the ESG (1997) show one extreme outlier (i.e., ESG sample #8427, 360 ppm). Inclusion of this sample in the dataset would skew total PCBs and misrepresent the contamination at the beach area. Therefore, this sample was excluded from the dataset and not carried forward into modeling for total PCBs. It is recommended that this “hotspot” be dug up and removed from the site, thus removing the source of contamination.

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

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#### 3.2.1 Division of Data

As discussed in Section 2.2, the site was sub-divided into two areas for the risk assessment based on current and likely future land uses:

- Area A: assumed to be residential/parkland for screening purposes and used potentially for camping on site for a limited amount of time due to its remote location; and
- Area B: included beach area – assumed to be residential/parkland for screening purposes and used potentially for camping on site for a limited amount of time due to its remote location.

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### 3.3 Description and Statistical Analysis 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 conduct of the human health and ecological risk assessments, a common framework was developed (illustrated in Figure 4-1). The steps in this flowchart are described briefly below:

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

Maximum concentrations sampled on site were compared to generic CCME and MOE generic soil quality guidelines for residential/parkland land use or to Defence Construction Canada (DCC) cleanup criteria.

#### **Box 2 Maximum concentration greater than guideline?**

If the maximum soil sample CoPC concentration from the data set was less than the appropriate generic guideline, either CCME or MOE, then the CoPC was not carried forward into this quantitative risk assessment.

#### **Box 3 Determine the EPC.**

As a conservative estimate for exposure, the 95% UCL concentration values were adopted as the EPCs for the quantitative risk assessments to calculate potential risks and area-wide SSTLs.

#### **Box 4 Is the EPC greater than the background soil concentration?**

Only if the EPC is greater than the background soil concentration will the CoPC be carried forward to the risk assessment process.

#### **Box 5 Conduct quantitative risk assessment**

CoPCs that exceeded guidelines and background concentrations were carried forward into the risk assessment process. The risk assessment was conducted using EPCs. This process was conducted independently for each chemical on the site and for the HHRA and the ERA separately. In this way, the chemicals subjected to ecological risk assessment were not necessarily included in the human health risk assessment.

#### **Box 6 Do the hazard quotients in the risk assessment exceed the target hazard quotient value?**

When hazard quotients (HQs) exceed the target HQ value (0.2 (non-carcinogen) and 1.0E-05 (carcinogen) for HHRA, 1.0 for ERA), there may be an inherent risk on site.

#### **Box 7 Calculate site-specific target levels (SSTLs)**

SSTLs are calculated soil concentrations where risk to potential receptors would be considered minor; remedial action could be taken to obtain these concentrations on site.

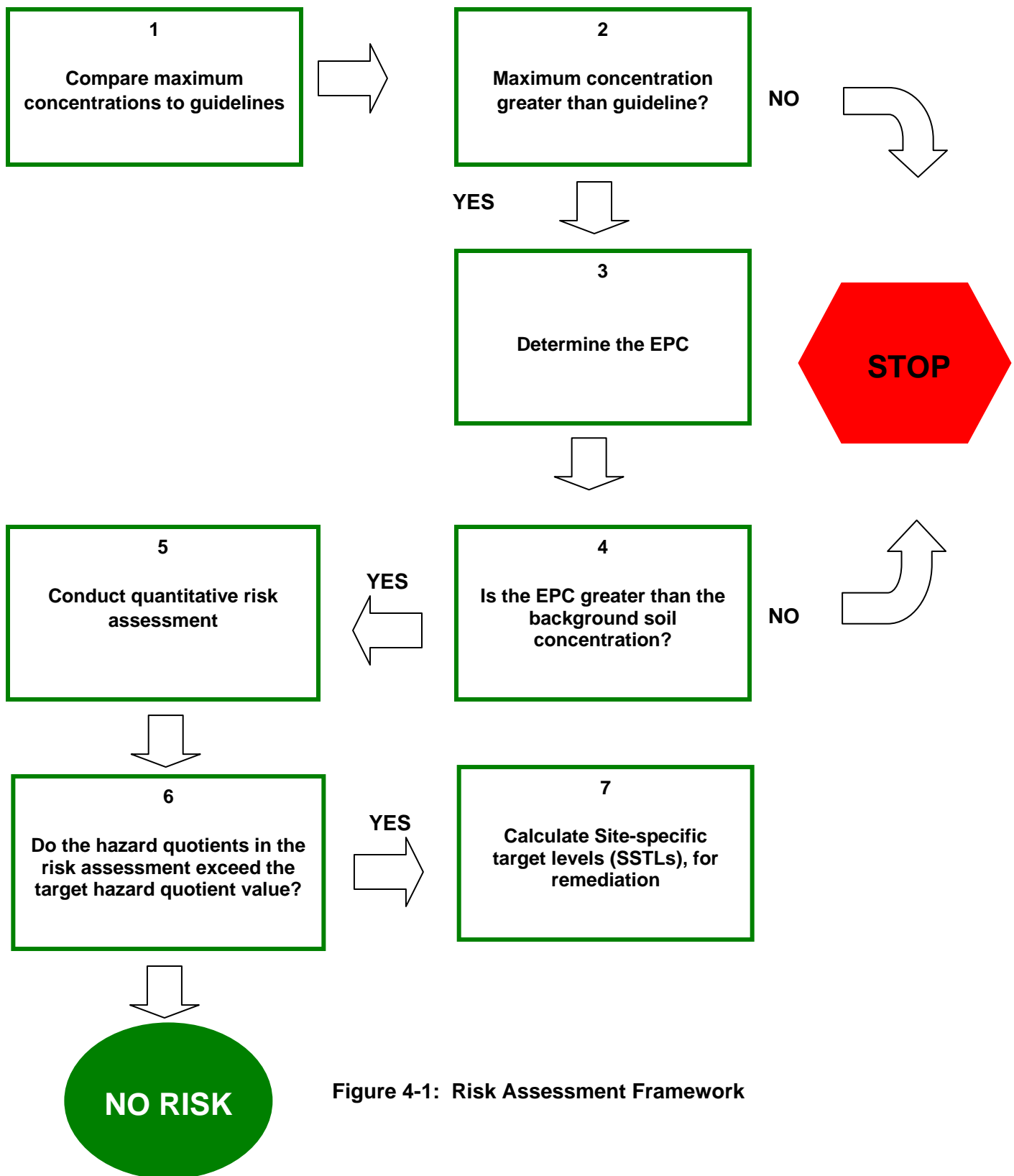


Figure 4-1: Risk Assessment Framework



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## 4.2 Chemical Screening

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### 4.2.1 Identification of Chemicals of Potential Concern

The results of human health soil and surface water screening for Areas A and B are discussed in the sections that follow.

#### Area A

##### *Soils*

As indicated in Table 4-1, and based upon the human health screening described in Box 5 of Figure 1, the maximum soil concentrations of antimony, arsenic, barium, beryllium, cadmium, lead, mercury, nickel, tin and zinc were greater than their corresponding human-health based criterion and were carried forward as CoPCs into the HHRA.

The remaining inorganic elements all had maximum concentrations that were less than their corresponding human health based criterion and were not carried forward in the HHRA, as their concentrations are below concentrations that would pose a potential risk to human health.

Although the maximum concentration of the TPH F1 and F4 fractions in soils were below the Canada Wide Standard (CWS) criterion, the maximum concentrations of the F2 and F3 fractions were greater than their CWS criteria, and were carried forward as CoPCs into the HHRA.

Maximum soil concentrations of benzene, toluene, ethylbenzene and xylene (BTEX) as well as polycyclic aromatic hydrocarbons (PAHs) were all less than their respective human-health based criteria, and were therefore not carried forward. Total polychlorinated biphenyls (PCBs) were higher than the interim CCME guideline criteria and were carried forward into the HHRA.

In summary, soil CoPCs from Area A carried forward into the HHRA included:

- Antimony
- Barium
- Cadmium
- Lead
- Nickel
- Zinc
- TPH F3 Fraction
- Arsenic
- Beryllium
- Copper
- Mercury
- Tin
- TPH F2 Fraction
- Total PCBs

##### *Surface Water*

Surface water samples were collected from two separate ponds in Area A. Additionally, one background sample was collected from a small lake adjacent to sampling location 8476. Table 4-2 presents the results of surface water screening for all parameters that were assessed in ESG (1997). None of the maximum CoPC concentrations were greater than available drinking water guidelines. For cobalt, drinking water guidelines were unavailable; however, cobalt concentrations were below background levels. Where parameters were identified as CoPCs based on soil concentrations and were also detected in surface water (i.e., copper and zinc), surface water was treated as an additional

source medium and surface water ingestion and dermal contact were included in the total dose for that CoPC. This ensures that derived total hazard quotients (i.e., risk estimates) take into consideration all potential exposure pathways.

**Table 4-1: Human Health Soil Screening for Area A**

Contaminant	Maximum Soil Concentration (mg/kg)	CCME Soil Quality Guideline <sup>HH</sup> <sup>a</sup> (mg/kg)	MOE Soil Quality Guideline <sup>HH</sup> <sup>b</sup> (mg/kg)	DCC Generic Soil Quality Guideline (mg/kg) <sup>c</sup>		Maximum Exceeds Guideline
				Tier I	Tier II	
<b>Metals</b>						
Antimony	254		13			✓
Arsenic	53	12	13		30	✓
Barium	1120	500	3700			✓
Beryllium	1.5		0.37			✓
Boron	14		1600 <sup>d</sup>			
Cadmium	115	14	14		5	✓
Chromium	85	220			250	
Cobalt	188		2700		50	
Copper	2160	1100	1100		100	✓
Lead	115000	140		200	500	✓
Mercury	31	6.6	13		2	✓
Molybdenum	10.1		170			
Nickel	840		310		100	✓
Selenium	9.7	28	320			
Silver	40		98			
Thallium	0.5	1	4.1			
Tin	1700	50				✓
Vanadium	8.4		470			
Zinc	26640		16000		500	✓
<b>BTEXs</b>						
Benzene	0.02	110	35			
Toluene	0.15	22000	6100			
Ethylbenzene	0.05	10000	2300			
Xylene	0.79	150000	61000			
<b>TPH</b>						
TPH – F1 Fraction	50	15000 <sup>e</sup>				
TPH – F2 Fraction	29000	8000 <sup>e</sup>				✓
TPH – F3 Fraction	30000	18000 <sup>e</sup>				✓
TPH – F4 Fraction	15000	25000 <sup>e</sup>				
<b>PCBs</b>						
Total PCBs	490	1.3		1	5	✓
<b>PAHs<sup>d</sup></b>						
Acenaphylene	0.00001		1400			
Acenaphthene	0.00001		1000			
Anthracene	0.00001		5300			
Benzo(a)anthracene	0.00018		120			
Benzo(a)pyrene	0.00001	0.7				
Benzo(b)fluoranthene	0.00001		12			
Benzo(ghi)perylene	0.00001		12			
Benzo(k)fluoranthene	0.00001		120			
Chrysene	0.0011		12			
Dibenz(a,h)anthracene	0.00001		1.2			
Fluorene	0.00001		910			
Fluroanthene	0.00054		910			
Indeno(l,2,3-cd)pyrene	0.00001		12			
Naphthalene	0.00001		1300			
Phenanthrene	0.0014		120			
Pvrene	0.00094		680			

<sup>a</sup> CCME (1999) Last Revised 2004. Guidelines for residential/parkland soil quality;

<sup>b</sup> OME 1996. Table B Criteria Components - Non-Potable Groundwater Situation, Coarse Textured Soils, Residential Parkland, Human health based criteria.;

<sup>c</sup> INAC, 2005. Deline Clean up Criteria;

<sup>d</sup> EPA, 2005, Region III Risk Based Criteria Table;

<sup>e</sup> CCME. 2001. Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil.; Shaded boxes – criteria used to screen for risk assessment

**Table 4-2: Human Health Surface Water Screening**

CoPC	Maximum Surface Water Concentration (mg/L)	Screening Criteria		
		CCME <sub>HH</sub> <sup>a</sup> MAC (mg/L)	MOE Drinking Water <sup>b</sup>	Site Background Water Concentration <sup>c</sup>
Arsenic	0.0005 <sup>e</sup>	0.005		<0.001
Cadmium	0.0005 <sup>e</sup>	0.005		<0.001
Cobalt	0.005 <sup>e</sup>			<0.01
Chromium	0.005 <sup>e</sup>	0.05		<0.01
Copper	0.01	1 <sup>d</sup>		0.03
Nickel	0.005 <sup>e</sup>		0.1	<0.01
Lead	0.005 <sup>e</sup>	0.01		<0.01
Zinc	0.3	5 <sup>d</sup>		<0.02

<sup>a</sup> CCME (2004) Guidelines for Canadian Drinking Water Quality - MAC – Maximum Acceptable Concentration;

<sup>b</sup> MOE. 2004. Soil, Groundwater and Sediment Standards for Use under Part XV.1 of the Environmental Protection Act. Table 2 – Full Depth Generic Site Condition Standards in a Potable Groundwater Condition.

<sup>c</sup> Background concentrations from ESG (1997)

<sup>d</sup> Aesthetic Objective

<sup>e</sup> Measured value was less than method detection limit (MDL), thus was set to half MDL.

## Area B

### Soils

As indicated in Table 4-3, and based upon the human health screening described in Box 5 of Figure 1, the maximum soil concentrations of beryllium, cadmium, lead, and zinc in soils were greater than their corresponding human health based criterion and thus were carried forward as CoPCs in the HHRA.

The remaining inorganic elements all had maximum concentrations that were less than their corresponding human health based criterion and were not carried forward in the HHRA, as their concentrations are below concentrations that would pose a potential risk to health.

Maximum soil concentrations of all TPH fractions were less than their corresponding Canada Wide Standard (CWS) criterion; therefore, these contaminants were not considered further in the HHRA.

Maximum soil concentrations of BTEX and PAH and were all less than their respective human health based criteria, and were therefore not carried forward. Total polychlorinated biphenyls (PCBs) were higher than the interim CCME guideline criteria and were thus carried forward in the HHRA.

The soil CoPCs from Area B carried forward into the HHRA included:

- Beryllium
- Lead
- Cadmium
- Total PCBs

**Table 4-3: Human Health Soil Screening for Area B**

Contaminant	Maximum Soil Concentration (mg/kg)	CCME Soil Quality Guideline <sup>a</sup> HH (mg/kg)	MOE Soil Quality Guideline <sup>b</sup> HH (mg/kg)	DCC Generic Soil Quality Guideline <sup>c</sup> (mg/kg)		Maximum Exceeds Guideline
				Tier I	Tier II	
<b>Metals</b>						
Antimony	1.00		13			
Arsenic	8.90	12	13		30	
Barium	117	500	3700			
Beryllium	0.70		0.37			✓
Boron	5.00		1600 <sup>d</sup>			
Cadmium	50.0	14	14		5	✓
Chromium	40.0	220			250	
Cobalt	15.0		2700		50	
Copper	79.0	1100	1100		100	
Lead	2900	140		200	500	✓
Mercury	1.85	6.6	13		2	
Molybdenum	1.00		170			
Nickel	85.0		310		100	
Selenium	3.90	28	320			
Silver	0.50		98			
Thallium	0.50	1	4.1			
Tin	17.4	50				
Vanadium	25.4		470			
Zinc	3300		16000		500	
<b>BTEXs</b>						
Benzene	0.02	110	35			
Toluene	0.05	22000	6100			
Ethylbenzene	0.05	10000	2300			
Xylene	0.05	150000	61000			
<b>TPH</b>						
TPH – F1 Fraction	5.00	15000 <sup>e</sup>				
TPH – F2 Fraction	5.00	8000 <sup>e</sup>				
TPH – F3 Fraction	5.00	18000 <sup>e</sup>				
TPH – F4 Fraction	5.00	25000 <sup>e</sup>				
<b>PCBs</b>						
Total PCBs	2.7	1.3		1	5	✓
<b>PAHs</b>						
Acenaphylene	-		1400			
Acenaphthene	-		1000			
Anthracene	-		5300			
Benzo(a)anthracene	-		120			
Benzo(a)pyrene	-	0.7				
Benzo(b)fluoranthene	-		12			
Benzo(ghi)perylene	-		12			
Benzo(k)fluoranthene	-		120			
Chrysene	-		12			
Dibenz(a,h)anthracene	-		1.2			
Fluorene	-		910			
Fluroanthene	-		910			
Indeno(l ,2,3-cd)pyrene	-		12			
Naphthalene	-		1300			
Phenanthrene	-		120			
Pyrene	-		680			

<sup>a</sup> CCME (1999) Last Revised 2004. Guidelines for residential/parkland soil quality;

<sup>b</sup> OMOE 1996. Table B Criteria Components - Non-Potable Groundwater Situation, Coarse Textured Soils, Residential Parkland, Human health based criteria.;

<sup>c</sup> INAC, 2005. Dewline Clean up Criteria;

<sup>d</sup> EPA, 2005, Region III Risk Based Criteria Table;

<sup>e</sup> CCME. 2001. Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil.; Shaded boxes – criteria used to screen for risk assessment

## 4.2.2 Summary of Chemicals of Potential Concern

Table 4-4 provides a summary of the CoPCs identified in soil that were carried forward into the HHRA. Although the HHRA was concerned primarily with CoPCs detected in surface soil, wild game and drinking water were also included as a potential exposure pathway to provide a more complete exposure scenario.

**Table 4-4: Summary of CoPCs and EPCs used in HHRA**

Compound	C <sub>soil</sub> Site A (mg/kg)	C <sub>soil</sub> Site B (mg/kg)	C <sub>wildgame</sub> (mg/kg)	C <sub>water</sub> (mg/L)
<b>Inorganics</b>				
Antimony	4.69	--	0.000403	--
Arsenic	3.96	--	0.00775	--
Barium	203	--	0.16	--
Beryllium	0.40	0.70	0.000716	--
Cadmium	1.82	1.95	0.278	--
Copper	125	--	4.95	0.01
Lead	673	124.36	6.15	--
Mercury	2.60	--	0.00028	--
Nickel	562	--	1.63	--
Tin	31.0	--	2.74	--
Zinc	887	--	46.2	0.3
<b>TPH – CCME CWS</b>				
Aliph>C10-C12 -F2	5.28E+03	--	7.54E+01	--
Aliph>C12-C16 -F2	6.46E+03	--	1.07E+03	--
Arom>C10-C12 -F2	1.32E+03	--	3.72E-01	--
Arom>C12-C16 -F2	1.61E+03	--	8.29E-01	--
F2 - Total	1.47E+04	--		--
Aliph>C16-C21-F3	1.47E+04	--	2.24E+03	--
Aliph>C21-C34 -F3	6.29E+03	--	9.61E+01	--
Arom>C16-C21 -F3	3.67E+03	--	3.90E+00	--
Arom>C21-C34 -F3	1.57E+03	--	1.16E+01	--
F3 - Total	2.60E+04	--	2.24E+03	--
<b>Organics</b>				
PCBs – Total	2.60E-02	2.70E-3	5.26E-03	4.40E-04

-- = Parameter not evaluated for this pathway

## 4.2.3 Receptor Identification

Existing and intended land use is an important factor in evaluating the potential exposures and estimating risk. This risk assessment was directed toward the following potential end use of the land:

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

Therefore the potential human “receptors”, or people who may be most affected by the potential hazards are people hunting on the land. For the purposes of this assessment, the human receptor is characterized as an adult or child with no extreme sensitivities. Carcinogenic and non-carcinogenic chemicals were evaluated differently as illustrated below:

	RECREATIONAL EXPOSURE
NON-CARCINOGENIC CHEMICAL	Most sensitive receptor modelled as a toddler aged six months to four years old.
CARCINOGENIC CHEMICAL	Composite receptor assumed to grow up using the site from birth to 75 years lifetime. Exposures averaged over five age groups: (0 to 6 months) + (6 months to 4 yrs) + (5 to 11 yrs) + (12 to 19 yrs) + (20 to 75 yrs).

The above assumptions regarding receptors are the most protective approaches for the intended land uses. Important characteristics of the receptors (including body weight, soil ingestion rate, *etc.*) considered in the analysis are presented Section 4.3.

#### 4.2.4 Exposure Pathway Assessment

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

#### 4.2.5 Potential Transport Pathways

The principal pathways through which environmental hazards can typically contact a receptor include:

- direct contact (with soil, dust, liquid product phase hazards, or water);
- transport of liquid product phase contaminants;
- transport in groundwater;
- transport in surface water;
- air borne transport (as dust); and
- transport as a vapour.

#### 4.2.6 Potential Exposure Mechanisms

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

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

## 4.2.7 Human Receptor Exposure Scenarios

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

- inhalation/ingestion/dermal contact with soil/dust;
- inhalation/ingestion/dermal contact with sediment;
- ingestion/dermal contact with surface water;
- ingestion/dermal contact with groundwater;
- inhalation of vapours;
- ingestion of wild game; and,
- ingestion of vegetation.

Jacques Whitford has evaluated the likelihood that the identified human receptors can be exposed to the identified hazards through the various exposure scenarios using a qualitative method. The likelihood of exposure is considered and evaluated in terms of the following series of definitions, presented in Table 4-5.

**Table 4-5: Exposure Definitions**

<b>Likelihood Of Exposure</b>	<b>Definition</b>
Very Unlikely	Level of exposure that could result in adverse effects is not expected.
Unlikely	Level of exposure that could result in adverse effects would probably not occur.
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. Exceedance of this exposure level might be expected.

The relevant exposure pathways are summarized in Tables 4-6. Table 4-6 includes the qualitative evaluation of each pathway for traditional land use and a justification for the likelihood of exposure assigned. The likelihood of exposure includes consideration of the duration and frequency of exposure to each potential hazard and to the relative concentrations to which the receptor is likely to be exposed. Those hazard-exposure-receptor combinations considered to have the highest likelihood to contribute a health risk are carried forward for further quantitative analysis.

**Table 4-6: Potential Exposure Scenarios – Traditional Land User**

Exposure Pathway Description	Likelihood of Exposure	Carried Forward for Quantitative Analysis?	Justification
Ingestion of soil/dust	Likely	Yes	Soil samples collected during the soil sampling programs were impacted by inorganic elements and organics at concentrations exceeding human health soil screening guidelines for direct contact
Dermal contact with soil/dust			
Inhalation of soil/dust			
Inhalation of soil vapours	Unlikely	No	Neither the inorganic CoPCs nor the TPH F2 to F4 fractions found on site were considered volatile, therefore the inhalation of vapours pathway was considered negligible.
Ingestion of sediment	Very Unlikely	No	There is little likelihood 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	Surface water was not considered a source medium for contaminants, however, where CoPCs were identified in soil, and were detected in the surface water, surface water was considered an additional exposure pathway.
Dermal contact with surface water			
Inhalation of surface water vapours	Very Unlikely	No	No volatile CoPCs were identified in surface water, therefore vapour inhalation was not considered a valid pathway.
Ingestion of groundwater	Very Unlikely	No	Groundwater was not used as a source for either drinking or showering and therefore was not considered a valid pathway.
Dermal contact with groundwater			
Inhalation of groundwater particles/vapours			
Ingestion of vegetation	Very Unlikely	No	Due to the climate, significant harvest of vegetation was not expected.
Ingestion of wild game	Likely	Yes	Visiting Inuit receptors were expected to hunt and consume wild game on the site.

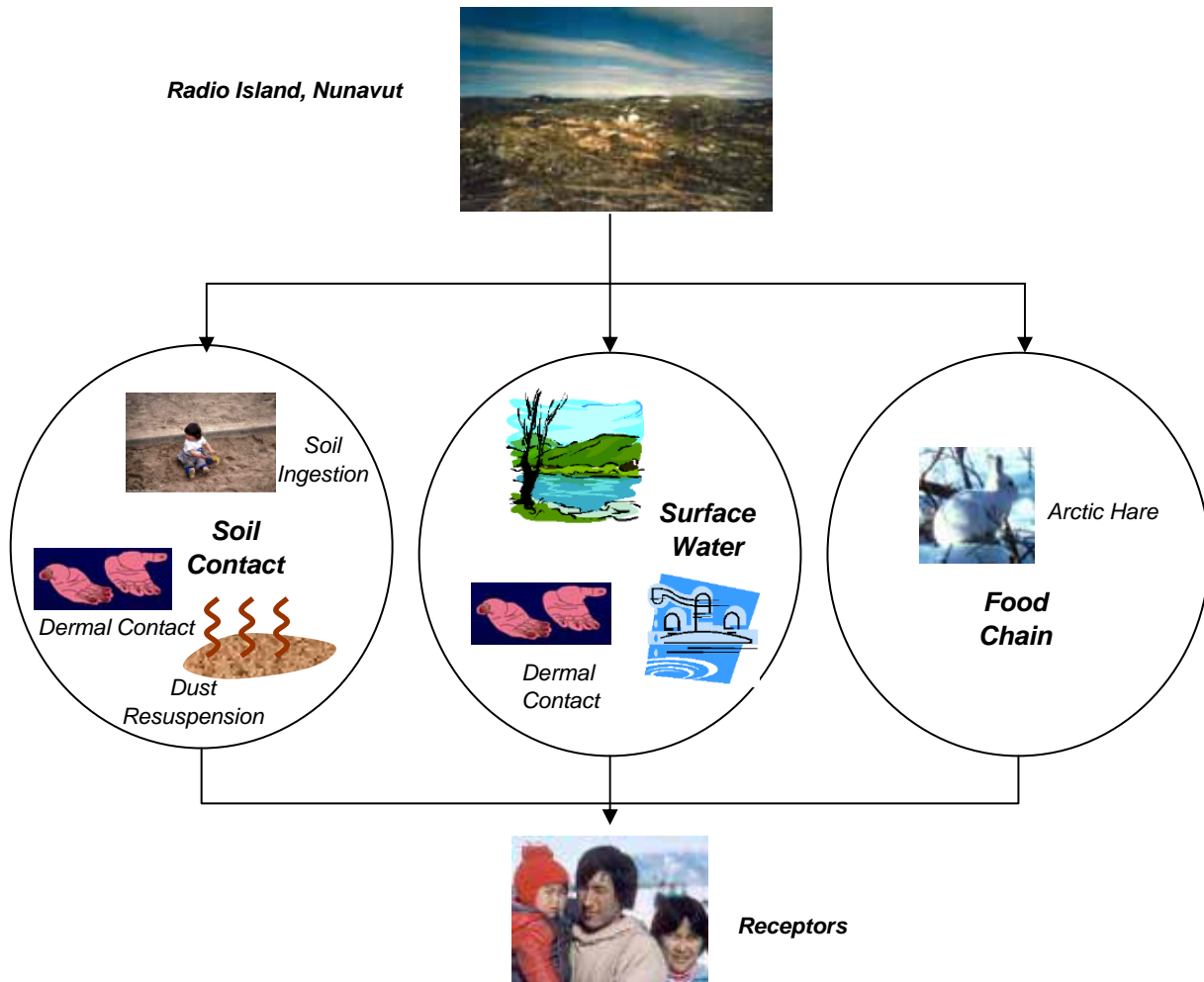
## 4.2.8 Qualitative Risk Characterization

Based on the qualitative risk screening presented above, the conceptual model (Figure 4-1) that forms the basis for the calculation of potential risk and derivation of the site specific target levels for human health ( $SST_{LHH}$ ) is as follows:

### Traditional Land Use Scenario

- A toddler aged six months to four years is exposed to surface soil impacted with non-carcinogenic contaminants by inadvertent ingestion / dermal contact / inhalation of surface soil/dust, ingestion / dermal contact with surface water and by ingestion of wild game (e.g., Arctic hare); and
- A person visits the site yearly (i.e., 3 week site-visits) from birth to 75 years of age and is exposed to carcinogenic contaminants by inadvertent ingestion / dermal contact / inhalation of surface soil/dust, ingestion / dermal contact with surface water and by ingestion of wild game (e.g., Arctic hare), throughout their lifetime.





**Figure 4-1: Human Health Conceptual Model**

### 4.3 Receptor Characteristics

It is important that the most protective assumptions are made when assessing human exposure to CoPCs at Radio Island. 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 Radio Island 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 the 95% UCL was assumed. Receptor characteristics for the toddler and composite receptor are presented below (Table 4-7).

**Table 4-7: 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	Conservative site-specific assumption
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 (carcinogens), in which any level of exposure is considered to have a potential for adverse health effects, exposures are not calculated within specific age groups (e.g., toddler) but are averaged over a lifetime. In accordance with the reasonable maximum exposure approach, it was assumed that a public visitor to the site grows up in Nunavut from birth to 75 years old. For the purposes of the risk characterization calculations, 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 4-8.

**Table 4-8: Summary of Receptor Characteristics for each age groups**

Characteristic	Receptor Values						Source
	Infant	Toddler	Child	Teen	Adult	Composite	
<b>Age</b>	<b>0 – 6 mo.</b>	<b>7 mo. – 4 yr</b>	<b>5 – 11 yr</b>	<b>12 – 19 yr</b>	<b>20 - 75 yr</b>	<b>75 yr lifetime</b>	
AT (years)	0.5	4.5	7	8	56	75	
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> )	1713	3009	5260	8283	9487	8650	Richardson, 1997
IR <sub>game</sub> (mg/d)	0	85	125	175	270	233.43	Health Canada (2004)

#### 4.4 Toxicity Assessment

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

For contaminants for which the critical effect is assumed to have no threshold (i.e., carcinogens), it is assumed that there is some probability of harm to human health at any level of exposure (CCME, 1996b). There is a linear dose-response relationship that converts estimated daily intakes averaged over a lifetime of exposure directly to an incremental risk of an individual developing cancer. For the purposes of deriving site-specific soil quality guidelines, Health Canada considers that a single increased case of cancer in an exposed population of 100,000 merits action (Health Canada, 2004a). As such, a target risk (TR) of one in one hundred thousand or  $10^{-5}$  is used in this risk assessment for carcinogenic effects.

#### 4.4.1 Selection of Toxicity Reference Values

An essential part of the risk assessment is the identification of appropriate toxicity reference values. This is typically done by a literature review of published toxicological assessments.

Toxicity values have been established by several agencies including Health Canada, the United States Environmental Protection Agency (US EPA), as well as others. Preference has been given to Health Canada toxicity reference values (TRVs) as per federal guidance (Health Canada, 2004a). In the event that a Health Canada TRV does not exist the most scientifically up to date toxicity values upon which to evaluate health risks were utilized.

Summaries of the toxicity values selected for inclusion in the risk assessment are provided in Table 4-9 and 4-10, and detailed rationales for each of the toxicity values are provided in **Appendix B**.

**Table 4-9: Selected Carcinogenic Toxicity Values**

CoPC	Route of Exposure	Exposure Limit (mg/kg-d)	Toxicological Basis	Source Agency
<b>Inorganics</b>				
Arsenic	Ingestion	2.8	Skin	Health Canada, 2004b
	Inhalation	28	Lungs	Health Canada, 2004b
Beryllium	Ingestion	na	na	na
	Inhalation	9.86	Not specified	US EPA, 1998
Cadmium	Ingestion	na	na	na
	Inhalation	40.2	Not specified	Health Canada, 2004b
Nickel	Ingestion	na	na	na
	Inhalation	2.92	Not specified	Health Canada, 2004b
PCBs (Total)	Ingestion	2	Liver	US EPA, 1997b
	Inhalation	0.4	Not specified	US EPA, 1997b

**Table 4-10: Selected Non-Carcinogenic Toxicity Values**

CoPC		Route of Exposure	Exposure Limit (mg/kg-d)	Toxicological Basis	Source Agency
Inorganics					
Antimony		Ingestion	0.0004	longevity, clinical chemistry	US EPA, 1997a
		Inhalation	na	na	na
Arsenic		Ingestion	0.0003	skin	US EPA, 2000
		Inhalation	na	na	na
Barium		Ingestion	0.016	Not specified	Health Canada, 2004b
		Inhalation	na	na	na
Beryllium		Ingestion	0.002	Small intestine	US EPA, 1998
		Inhalation	0.0000487	Beryllium sensitization and progression to CBD	US EPA,1998
Cadmium		Ingestion	0.0008	Not specified	Health Canada, 2004b
		Inhalation	na	na	na
Copper		Ingestion	0.03	Not specified	Health Canada, 2004b
		Inhalation	na	na	na
Lead		Ingestion	0.0036	Not specified	Health Canada, 2004b
		Inhalation	na	na	na
Mercury		Ingestion	0.0003	Not specified	Health Canada, 2004b
		Inhalation	na	na	na
Nickel		Ingestion	0.02	decreased body and organ weight	US EPA, 1995
		Inhalation	na	na	na
Tin		Ingestion	0.6	Not specified	HEAST, 2005
		Inhalation	na	na	na
Zinc		Ingestion	0.3	Blood	US EPA, 2005a
		Inhalation	na	na	na
Petroleum Hydrocarbons CWS Fractions					
F2	Aliph >C10-C12	Ingestion	0.1	Hepatic and hematological changes	CCME, 2001
		Inhalation	0.223		CCME, 2001
	Aliph >C12-C16	Ingestion	0.1	Hepatic and hematological changes	CCME, 2001
		Inhalation	0.223		CCME, 2001
	Arom >C10-C12	Ingestion	0.04	Decreased body weight	CCME, 2001
		Inhalation	0.004		CCME, 2001
	Arom >C12-C16	Ingestion	0.04	Decreased body weight	CCME, 2001
		Inhalation	0.004		CCME, 2001
F3	Aliph >C16-C21	Ingestion	2	Hepatic granuloma	CCME, 2001
		Inhalation	na	na	na
	Aliph >C21-C34	Ingestion	2	Hepatic granuloma	CCME, 2001
		Inhalation	na	na	na
	Arom >C16-C21	Ingestion	0.03	Nephrotoxicity	CCME, 2001
		Inhalation	na	na	na
	Arom >C21-C34	Ingestion	0.03	Nephrotoxicity	CCME, 2001
		Inhalation	na	na	na

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#### 4.4.2 Bioavailability

Bioavailability refers to “the fraction of the total amount of material in contact with a body portal-of-entry (lung, gut, skin) that enters the blood”. Relative bioavailability is the amount of a substance entering the blood via a particular route of exposure (e.g., gastrointestinal) relative to the study used to derive the TRV. These factors were then applied in the risk assessment to more realistically represent the portion of contaminants held in soil that are available. For instance, a relative bioavailability factor of 0.5 indicates that 50% of the administered (e.g., ingested) chemical is absorbed into the bloodstream compared to the absorption in the TRV study. Relative bioavailabilities via ingestion and inhalation routes of exposure are conservatively assumed to be a factor of 1.0. Table 4-11 provides the bioavailability factors used in this assessment

**Table 4-11: Selected Relative Bioavailability Factors**

CoPC	Oral	Dermal	Inhalation
Antimony	1	0.1	1
Arsenic	1	0.03	1
Barium	1	0.1	1
Beryllium	1	0.03	1
Cadmium	1	0.14	1
Lead	1	0.006	1
Mercury	1	0.05	1
Nickel	1	0.35	1
Tin	1	0.1	1
Zinc	1	0.02	1
Total PCBs	1	1	1
TPH fraction F2	1	0.2	1
TPH fraction F3	1	0.2	1

Detailed rationale supporting the selection of each of the values recommended for use in this assessment is provided in the accompanying toxicity profiles in **Appendix B**.

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#### 4.5 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 CDIs 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  
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

Five CoPCs identified at Radio Island (i.e., arsenic, beryllium, cadmium, nickel 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 Radio Island, the estimated exposure is multiplied by the appropriate slope factor or unit risk as shown below:

$$ILCR = LADD * CSF$$

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 Radio Island). 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 (2004a) 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-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 (1996b) and OME (1996b). Detailed ILCR derivations and parameter values used in the analyses are provided in **Appendix C**.

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.

#### 4.5.1 Non-Carcinogenic Risk Estimates

##### Area A

HQs for all CoPCs, which were derived using respective EPCs, are presented in Table 4-12 for the most sensitive receptor, a toddler.

Using the traditional land use scenario for Area A the total exposure risk from all CoPCs on the site were below the target HQ of 0.2, thus exposure to this portion of the Radio Island site resulted in negligible potential risk to receptors.

**Table 4-12: Hazard Quotients for Non-Carcinogens – Toddler, Area A**

CoPC	EPC (mg/kg)	Total HQ	Target HQ	Exceeds Target HQ?
Antimony	4.69	3.57E-03	2.0E-01	No
Arsenic	3.96	3.78E-03	2.0E-01	No
Barium	202.77	3.86E-03	2.0E-01	No
Beryllium	0.40	6.02E-05	2.0E-01	No
Cadmium	1.82	7.16E-04	2.0E-01	No
Copper	125.42	2.47E-03	2.0E-01	No
Lead	672.56	5.24E-02	2.0E-01	No
Mercury	2.60	2.53E-03	2.0E-01	No
Nickel	56.22	1.04E-03	2.0E-01	No
Tin	31.01	1.57E-05	2.0E-01	No
Zinc	886.83	3.83E-03	2.0E-01	No
TPH F2 Fraction	29000	1.25E-01	2.0E-01	No
TPH F3 Fraction	30000	7.00E-02	2.0E-01	No

##### Area B

HQs for all CoPCs, which were derived using respective EPCs, are presented in Table 4-13 for the most sensitive receptor, a toddler.



Using the traditional land use scenario for Area B the total exposure risks from all CoPCs on the site were below the target HQ of 0.2, thus exposure to this portion of the Radio Island site results in negligible potential risk to receptors.

**Table 4-13: Hazard Quotients for Non-Carcinogens – Toddler, Area B**

CoPC	EPC (mg/kg)	Total HQ	Target HQ	Exceeds Target HQ?
Beryllium	0.7	1.05E-04	2.0E-01	No
Cadmium	1.95	7.68E-04	2.0E-01	No
Lead	124.4	9.69E-03	2.0E-01	No

## 4.5.2 Carcinogenic Risk Estimates

### Area A

ILCRs have been derived for all carcinogenic CoPCs from Area A using the EPCs; results are presented in Table 4-14.

The ILCRs of the composite receptor engaging in traditional land use in Area A were less than the acceptable benchmark of  $1 \times 10^{-5}$  for all CoPCs. Therefore, exposure to soil in Area A poses a negligible potential risk to human receptors.

**Table 4-14: Incremental Lifetime Cancer Risks for Carcinogenic CoPCs in Area A**

CoPC	EPC (mg/kg)	Total ILCR	Target ILCR	Exceeds Target ILCR?
Arsenic	3.96	1.96E-07	1.00E-05	No
Beryllium	0.40	8.28E-11	1.00E-05	No
Cadmium	1.82	1.54E-09	1.00E-05	No
Nickel	56.22	3.44E-09	1.00E-05	No
Total PCBs	0.026	1.67E-06	1.00E-05	No

### Area B

ILCRs have been derived for all carcinogenic CoPCs from Area B using the EPCs; results are presented in Table 4-15.

The ILCRs of the composite receptor engaging in traditional land use in Area B were less than the acceptable benchmark of  $1 \times 10^{-5}$  for all CoPCs. Therefore, exposure to these contaminants in soil at Area B poses a negligible potential risk to human receptors.

**Table 4-15: Incremental Lifetime Cancer Risks for Carcinogenic CoPCs in Area B**

CoPC	EPC (mg/kg)	Total ILCR	Target ILCR	Exceeds Target ILCR?
Beryllium	0.70	2.42E-09	1.00E-05	No
Cadmium	1.95	2.75E-08	1.00E-05	No
Total PCBs	0.0027	9.58E-10	1.00E-05	No

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### 4.5.3 Summary of Site Risk

The HQs for exposure to all non-carcinogenic CoPCs under the traditional land use scenario at Areas A and B on Radio Island were less than the target HQ of 0.2, indicating that CoPCs pose a negligible risk to human health at the site.

The ILCRs for exposure to all carcinogenic CoPCs under the traditional land use scenario at Areas A and B on Radio Island were less than the target ILCR of  $1 \times 10^{-5}$ , indicating that CoPCs pose a negligible cancer risk at the site.

This assessment has incorporated a number of conservative assumptions, including exposure times for a traditional land use scenario of both Areas A and B for 21 days, and the use of the EPC concentrations as the representative on-site concentrations. However, even with such conservative assumptions, the result of the risk assessment is that the CoPCs on site do not pose a risk to human receptors.

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## 4.6 Human Health Site-Specific Target Levels (SSTL<sub>HH</sub>)

Human health site-specific target levels (SSTLs) are developed based on the exposure characteristics of the user of the site. The SSTL values represent concentrations above, which adverse health effects are possible and further action may be required. These values could be used during any future site assessment activities, only if land use has not changed from those scenarios modeled in the HHERA. However, in the case of Radio Island, none of the CoPC EPC concentrations resulted in HQs greater than 0.2 or ILCRs above  $1 \times 10^{-5}$ ; therefore there is a negligible risk to receptors on site. Because the current CoPCs do not pose a risk to human receptors, the derivation of SSTLs is unnecessary, and remediation or risk management plans are likely not required.

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## 4.7 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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#### **4.7.1 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; and
- 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 are used 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; and
- 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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#### **4.7.2 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 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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#### **4.7.3 Summation of Hazards for 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, the toxicity values for the metals assessed 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.

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#### **4.7.4 Modeling Assumptions**

Table 4-16 summarizes the modeling assumptions used in this risk analysis, and provides an evaluation of each assumption and an opinion as to whether the assumption is acceptable.

**Table 4-16: 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>Hazard Identification</b>			
1. Screening of CoPC against human-health based generic CCME, US EPA, or DDC guidelines	Generic guidelines by nature are very conservative in order that they can be reliably applied to any situation, potentially with little site-specific information available. Substances present at concentrations less than generic guidelines are unlikely to be of concern.	Neutral	Yes
2. Exposure point concentrations (EPCs) based on the maximum concentrations from the site.	Using the 95% UCL of the geometric 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 geometric mean are likely to be much greater than the true mean concentrations of CoPCs on site.	Overestimate	Yes
<b>Receptor Characteristics</b>			
1. For analysis of non-carcinogenic exposure, a toddler (0.5 – 4 years old) was chosen as the receptor.	Young children are the most sensitive age group for assessing non-carcinogenic effects. Resulting risks are over protective for an adult population. This approach is in accordance with accepted practice from Health Canada and the US EPA.	Neutral for young children but will over-estimate risks to adults.	Yes
2. For analysis of potential carcinogenic effects, a lifetime average was used representing yearly exposure to the site from birth to 75 years old. (i.e. composite receptor).	For carcinogenic chemicals this is the most protective approach. In contrast, CCME only model adult exposure (20 to 75 years old) and US EPA only model exposure for 25 years (0 to 25 years old) averaged over a lifetime, both of which are less protective approaches.	Approach likely to over-estimate the risk.	Yes
3. For the Traditional Land Use scenario, both potential receptors (toddler and lifetime) assumed to be present on the upper site 24 hours per day, 21 days per year. While on the site they are assumed to be hunting on the site.	These are maximum values providing a reasonable maximum exposure estimate for a toddler but likely overestimating lifetime exposure.	Neutral to over-estimate.	Yes

**Table 4-16: Modeling Assumptions – cont'd.**

<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. Most current toxicity information available from Health Canada, US EPA Integrated Risk Information (IRIS) database.	This approach is in accordance with standard practice and provides the most recent scientific basis for toxicity values.	Neutral	Yes
2. Potential antagonistic/additive/synergistic effects of chemical mixtures were not quantitatively assessed.	The summation of hazards between compounds that do not have the same biological end-point or mechanism of action has little practical meaning. Summation of hazard indices across compounds 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 three potential exposure pathways: soil/dust ingestion; dermal contact, and inhalation; wild game ingestion; and ingestion and dermal contact with surface water.	CCME base the generic guidelines on only soil ingestion. Therefore, this multi-pathway approach is more protective.	Neutral	Yes
2. Assessment of the current nutritional health status of local receptors was not included in the scope of work.	Inuit peoples of the Canadian Arctic can have different nutritional status to southern populations due to dietary differences. Micronutrient deficiency (e.g., Ca, Fe, Vitamin D) has been reported and may affect the absorption of other chemicals into the body (e.g., increased absorption of lead).  Assessment 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
3. Target risk for ILCR set at 1 in 100,000 ( $10^{-5}$ ).	This is the value adopted by CCME for "acceptable" target risk. Health Canada uses target risks in the range of $10^{-5}$ to $10^{-6}$ . The CCME soil quality guidelines correspond to ILCRs of approximately $10^{-5}$ to $10^{-6}$ .	Neutral	Yes
4. Target Hazard Quotient for evaluating CoPC exposure = 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

The following sections present the results of an ecological risk assessment (ERA) which evaluated the potential for adverse effects to non-domesticated fauna based on current conditions and habitats at the former navigational aid and weather station located at Radio Island, Nu.

Ecological risk assessment is a process that evaluates the likelihood that adverse environmental effects may occur, or are occurring, because of exposure to one or more stressors (Suter, 1993). The potential hazards, or chemicals of potential concern (CoPCs), identified within the study area are chemical substances in environmental media (soil, water and terrestrial vegetation) from sources associated with past operations at the site. Therefore, the purpose of this ERA was to provide a qualitative and quantitative analysis of the likelihood and potential magnitude of adverse environmental effects to Valued Environmental Components (VECs), which are ecological receptors (mammals and birds) present, or potentially present, in the study area.

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### 5.1 Objectives

This ERA has been conducted according to established health risk assessment protocols endorsed by Health Canada (1994), CCME (1996c) and the US EPA (1999). The 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;
- Assess potential exposures of ecological receptors to CoPCs in various environmental media within terrestrial habitats under current conditions;
- Quantify the risks associated with exposures of ecological receptors to CoPCs in various environmental media under current conditions; and
- If unacceptable risk is identified, determine acceptable concentrations of contaminants (site specific target levels, or SSTLs) that would allow re-establishment of the habitat and would not pose ongoing risks.

This ERA used a general framework similar in concept to the approach used for the human health risk assessment, but is distinctive in its emphasis in three areas.

- With the exception of rare, threatened and/or endangered species, the ecological risk assessment does not consider effects on individuals of a single species rather it is concerned with potential effects at the population, community, or ecosystem levels. In order to achieve this goal, the toxicity reference values that are used to evaluate whether ecological exposures may lead to effects are based on Lowest Observed Adverse Effect Level (LOAEL) data from the ecotoxicological literature, with a focus on sublethal reproductive or developmental endpoints;



- There is no single set of ecological values or resources to be protected that can be generally applied to every site, so the selection of VECs and exposure pathways for the ERA is site-specific; and
- If appropriate, the ecological risk assessment can consider non-chemical, as well as chemical, stressors.

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## 5.2 Ecological Risk Assessment Framework

The ecological risk assessment consists of four main stages:

- **Problem Formulation** – The objective of this stage is to develop a focused understanding of how on-site chemicals/stressors might affect the health of ecological receptors that spend time on, or near, the site. There are three main components to problem formulation: i) receptor screening - identify potential ecological receptors (i.e., biological communities, populations, individuals, or habitats potentially at risk); ii) chemical screening - determine on-site contaminants of concern and other stressors of ecological receptors; iii) exposure pathway screening - identify potential exposure pathways. Each of these elements is integrated into a conceptual model that is specific to the site.
- **Exposure Assessment** – In an ERA, exposure assessment is the process of estimating the exposure(s) of ecological receptors to on-site CoPCs under a given exposure scenario. An exposure assessment is conducted for each chemical of potential concern identified in the previous stage. For wildlife, exposure is determined as a dose (i.e., estimated daily intake, EDI) and is typically expressed as milligram of a chemical per kilogram body weight per day (i.e., mg/kg-day). The EDI is derived using: i) site-specific concentrations of chemicals/stressors in soil/sediment, water and food, ii) the amount of time a receptor spends on or near the site, and iii) receptor specific parameters such as body weight, ingestion rates and dietary preferences.
- **Toxicity Assessment** – This stage involves identification of the potential toxic effects of chemicals and determination of the amount of a chemical that a receptor can be exposed to without experiencing unacceptable risks. This value is termed the toxicity reference value (TRV) or toxicity benchmark. Toxicity assessment provides the basis for evaluating what is acceptable exposure and what level of exposure may adversely affect ecological health.
- **Risk Characterization** – The nature and magnitude of potential ecological risks are evaluated by deriving quantitative estimates (hazard quotients, HQ) of the potential for adverse effects to ecological receptors. HQs were calculated by dividing exposure (i.e., EPC or total ingested dose) values for each receptor by the appropriate reference toxicity dose as follows:

$$HQ = (\text{Exposure}) / (\text{toxicity benchmark})$$

For birds and mammals, the exposure measure is the total ingested dose (mg/kg-day) summed over all exposure pathways. 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, toxicity benchmarks 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 consistent with these elements of an ecological risk assessment.

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## **5.3 Chemical Screening**

The ERA dealt exclusively with substances present in environmental media that are accessible to wildlife. To this end, Radio Island data was screened such that only surface soils (0 to 30 cm depth), surface water and vegetation were considered. Subsurface soils were not included as they were deemed inaccessible to wildlife.

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### **5.3.1 Identifying Chemicals of Potential Concern**

This step involved the selection of chemical substances that have the potential for adversely impacting ecological receptors in habitats associated with the site. CoPCs were selected based on their concentration in soils or surface water and their potential toxicity to ecological receptors. Identification of CoPCs was based on a comparison of site data to both toxicity-based screening criteria and (for inorganic substances) site-specific background concentrations.

An initial generic assessment of the potential for adverse effects associated with site-originated chemicals was conducted. This assessment compared the maximum detected concentration in soils with the generic soil quality guidelines for the protection of ecological health (CCME 1999b, 2001). For those substances for which CCME guidelines have not been developed, concentrations were screened using guidelines from the Ontario Ministry of Environment (OMOE 1997) and/or DCC cleanup criteria.

Tables 5-1, 5-2 and 5-3 illustrate the screening of CoPCs for the site. The tables list the maximum observed soil concentration and the relevant guidelines. If the substance is carried forward, its Exposure Point Concentration (EPC) is calculated. The EPC is intended to be a conservative (i.e., pessimistic, but not necessarily worst-case) estimate of the average on-site concentration to which wildlife may be exposed. Where sufficient data are available (i.e., if  $n \geq 5$ ) the EPC is estimated as the 95% upper confidence limit (UCL) of the geometric mean value. Where few data are available (i.e.,  $n < 5$ ), the EPC was assumed to be the maximum observed soil concentration from Radio Island site-specific data. Finally, a decision is rendered regarding whether each substance is present at a concentration that is below a threshold for concern, or whether the concentration could potentially cause harm to one or more ecological receptors. In the former case, the substance is not of interest to the ERA. In the latter case, the substance is deemed to be a CoPC and is carried forward into the quantitative ERA.

## **Area A**

### **Soils**

As indicated in Table 5-1, maximum soil concentrations of antimony, arsenic, barium, cadmium, chromium, cobalt, copper, lead, mercury, nickel, selenium, silver, and zinc were greater than their corresponding ecological-based soil quality criterion and were carried forward as CoPCs into the ERA.

The remaining inorganic elements all had maximum concentrations that were less than their corresponding ecological-based criterion and were not carried forward into the ERA, as their concentrations are below that which would pose a potential risk to ecological receptors.

Maximum soil concentrations of TPH fractions F2, F3 and F4 were greater than their corresponding CWS criteria and thus were carried forward as CoPCs into the ERA. The ERA process considers all fractions of TPH when assessing risk and establishing SSTLs, therefore the F1 fraction was also carried forward as a CoPC into the ERA.

Maximum soil concentrations of BTEX and PAHs were all less than their respective ecological-based criteria, and were therefore not carried forward. Total PCBs were higher than the CCME ecological guideline criteria and were carried forward as a CoPC.

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 and avian species, it was necessary to assess this environmental medium for CoPCs. Maximum concentrations of all substances measured in surface water (i.e., ponds in Area A) were screened against generic CCME guidelines developed for the protection of freshwater aquatic life (CCME, 1999b). The results of ecological health surface water screening for Radio Island are presented in Table 5-2.

No new CoPCs were identified via ecological health surface water screening. All substances assessed in surface water had previously been identified as CoPCs through screening of soil data, and were already carried forward into the quantitative ERA. However, the ingestion of surface water pathway was only carried forward for those CoPCs measured above the level of detection in this media (i.e., copper and zinc).

**Table 5-1: Ecological Soil Screening for Area A**

Contaminant	Maximum Soil Concentration (mg/kg)	CCME Soil Quality Guideline <sup>a</sup> <sub>Eco</sub> (mg/kg)	MOE Soil Quality Guideline <sup>b</sup> <sub>Eco</sub> (mg/kg)	DCC Generic Soil Quality Guideline <sup>c</sup> (mg/kg)		Maximum Exceeds Guideline
				Tier I	Tier II	
<b>Metals</b>						
Antimony	254		20			✓
Arsenic	53	17			30	✓
Barium	1120		750			✓
Beryllium	1.5		4			
Boron	14		1.5			✓
Cadmium	115	10			5	✓
Chromium	85	64			250	✓
Cobalt	188		40		50	✓
Copper	2160	63			100	✓
Lead	115000	300		200	500	✓
Mercury	31	12			2	✓
Molybdenum	10.1		40			
Nickel	840	50			100	✓
Selenium	9.7	1	10			✓
Silver	40		20			✓
Thallium	0.5	1.4				
Tin	1700	50 <sup>i</sup>				✓
Vanadium	8.4	130				
Zinc	26640	200			500	✓
<b>BTEXs</b>						
Benzene	0.02	31				
Toluene	0.15	75				
Ethylbenzene	0.05	55				
Xylene	0.79	95				
<b>TPH</b>						
TPH – F1 Fraction	50	130 <sup>d</sup>				
TPH – F2 Fraction	29000	450 <sup>d</sup>				✓
TPH – F3 Fraction	30000	400 <sup>d</sup>				✓
TPH – F4 Fraction	15000	2800 <sup>d</sup>				✓
<b>PCBs</b>						
Total PCBs	490	1.3		1	5	✓
<b>PAHs</b>						
Acenaphylene	0.00001		1000			
Acenapthene	0.00001		100			
Anthracene	0.00001		40			
Benzo(a)anthracene	0.00018		40			
Benzo(a)pyrene	0.00001	0.7				
Benzo(b)fluoranthene	0.00001		12			
Benzo(ghi)perylene	0.00001		40			
Benzo(k)fluoranthene	0.00001		12			
Chrysene	0.0011		12			
Dibenz(a,h)anthracene	0.00001		1.2			
Fluorene	0.00001		350			
Fluroanthene	0.00054		40			
Indeno(1,2,3-cd)pyrene	0.00001		12			
Naphthalene	0.00001	0.6				
Phenanthrene	0.0014		40			
Pvrene	0.00094		1.3			

<sup>a</sup> CCME (1999) Last Revised 2004. Guidelines for residential/parkland soil quality;

<sup>b</sup> OMOE 1996. Table B Criteria Components - Non-Potable Groundwater Situation, Coarse Textured Soils, Residential Parkland, Ecological health based criteria.;

<sup>c</sup> INAC, 2005. Deline Clean up Criteria;

<sup>d</sup> CCME. 2001. Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil.; Shaded boxes – criteria used to screen for risk assessment

**Table 5-2: Surface Water Screening**

CoPC	Maximum Surface Water Concentration (mg/L)	CCME Guideline <sup>a</sup> (mg/L)	Exceeds Guideline?	Site Background Water Concentration <sup>b</sup> (mg/L)	Exceeds Background Concentration
Arsenic	0.0005 <sup>c</sup>	0.005	No	<0.001	-
Cadmium	0.0005 <sup>c</sup>	1.7E-05	Yes	<0.001	-
Cobalt	0.005 <sup>c</sup>	-	No	<0.01	-
Chromium	0.005 <sup>c</sup>	0.009	No	<0.01	-
Copper	0.01	0.002-0.004	Yes	0.03	No
Nickel	0.005 <sup>c</sup>	0.025-0.150	No	<0.01	-
Lead	0.005 <sup>c</sup>	0.001-0.007	Yes	<0.01	-
Zinc	0.3	0.03	Yes	<0.02	Yes

<sup>a</sup> CCME Freshwater Aquatic Life Guidelines (2003)

<sup>b</sup> Background concentrations from ESG (1996)

<sup>c</sup> Measured value was less than method detection limit (MDL), thus was set to half MDL.

## Area B

### Soils

As indicated in Table 5-3, maximum soil concentrations of boron, cadmium, copper, lead, nickel, selenium and zinc were greater than their corresponding ecological-based soil quality criterion and were carried forward as CoPCs into the ERA.

The remaining inorganic elements all had maximum concentrations that were less than their corresponding ecological-based criterion and were not carried forward into the ERA, as their concentrations are below that which would pose a potential risk to ecological receptors.

Maximum soil concentrations of all four TPH fractions in Area B were below their corresponding CWS criteria; however, based on Area A screening TPH would already be carried forward in any case as part of the overall evaluation.

Maximum soil concentrations of BTEX and PAHs were all less than their respective ecological-based criteria, and were therefore not carried forward. Total PCBs were higher than the CCME ecological guideline criteria and were carried forward as a CoPC.

### Surface Water

Because no surface water is present within Area B, the background surface water sample was used for evaluation of potential exposures to chemicals via this media. Only copper was measured above the level of detection in the background sample, and therefore the surface water ingestion pathway was carried forward only for copper (Table 5-2).

**Table 5-3: Ecological Soil Screening for Area B**

Contaminant	Maximum Soil Concentration (mg/kg)	CCME Soil Quality Guideline <sub>Eco</sub> <sup>a</sup> (mg/kg)	MOE Soil Quality Guideline <sub>Eco</sub> <sup>b</sup> (mg/kg)	DCC Generic Soil Quality Guideline <sup>c</sup> (mg/kg)		Maximum Exceeds Guideline
				Tier I	Tier II	
<b>Metals</b>						
Antimony	1.00		20			
Arsenic	8.90	17			30	
Barium	117		750			
Beryllium	0.70		4			
Boron	5.00		1.5			✓
Cadmium	50.0	10			5	✓
Chromium	40.0	64			250	
Cobalt	15.0		40		50	
Copper	79.0	63			100	✓
Lead	2900	300		200	500	✓
Mercury	1.85	12			2	
Molybdenum	1.00		40			
Nickel	85.0	50			100	✓
Selenium	3.90	1	10			✓
Silver	0.50		20			
Thallium	0.50	1.4				
Tin	17.4	50 <sup>i</sup>				
Vanadium	25.4	130				
Zinc	3300	200			500	✓
<b>BTEXs</b>						
Benzene	0.02	31				
Toluene	0.05	75				
Ethylbenzene	0.05	55				
Xylene	0.05	95				
<b>TPH</b>						
TPH – F1 Fraction	5.00	130 <sup>d</sup>				
TPH – F2 Fraction	5.00	450 <sup>d</sup>				
TPH – F3 Fraction	5.00	400 <sup>d</sup>				
TPH – F4 Fraction	5.00	2800 <sup>d</sup>				
<b>PCBs</b>						
Total PCBs	2.7	1.3		1	5	✓
<b>PAHs</b>						
Acenaphylene	-		1000			
Acenaphthene	-		100			
Anthracene	-		40			
Benzo(a)anthracene	-		40			
Benzo(a)pyrene	-	0.7				
Benzo(b)fluoranthene	-		12			
Benzo(ghi)perylene	-		40			
Benzo(k)fluoranthene	-		12			
Chrysene	-		12			
Dibenz(a,h)anthracene	-		1.2			
Fluorene	-		350			
Fluroanthene	-		40			
Indeno(l ,2,3-cd)pyrene	-		12			
Naphthalene	-	0.6				
Phenanthrene	-		40			
Pvrene	-		1.3			

<sup>a</sup> CCME (1999) Last Revised 2004. Guidelines for residential/parkland soil quality;

<sup>b</sup> OMOE 1996. Table B Criteria Components - Non-Potable Groundwater Situation, Coarse Textured Soils, Residential Parkland, Ecological health based criteria.;

<sup>c</sup> INAC, 2005. Deline Clean up Criteria;

<sup>d</sup> CCME. 2001. Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil.; Shaded boxes – criteria used to screen for risk assessment

### 5.3.2 Summary of Chemicals of Potential Concern

Table 5-4 provides a summary of the CoPCs identified in soil that were carried forward into the ERA. Although the ERA is concerned primarily with CoPCs in surface soil, ingestion of surface water, terrestrial plants, invertebrates and small mammals were also included as potential exposure pathways.

**Table 5-4: Summary of CoPCs and EPCs Used in ERA**

CoPC	Area A Soil EPC (mg/kg)	Area B Soil EPC (mg/kg)	Background Soil Concentration (mg/kg)
Antimony	4.69		
Arsenic	3.96		1.18
Barium	202.8		
Beryllium	0.40	0.7	
Boron	6.95		
Cadmium	1.82	1.95	0.5
Chromium	26.78		41
Cobalt	22.48		13.4
Copper	125.4	44.9	25.1
Lead	672.5	124.4	
Mercury	2.60		
Nickel	56.22	40.6	42.8
Selenium	2.63	3.9	
Silver	0.82		
Zinc	887	247.3	46.8
TPH			
F1 Fraction	50		
F2 Fraction	29000		
F3 Fraction	30000		
F4 Fraction	15000		
Total PCBs	0.026	0.0027	0.004

### 5.4 Receptor Identification

Receptor selection was based on fundamental ecological considerations, and was also guided by observations made during previous site visits, 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 soils, and/or freshwater at the site;
- representative of lower and higher trophic feeding levels (i.e., herbivorous and carnivorous animals);
- present on or near the site for some or most of the year;
- of significant cultural and/or economic significance; and
- possible endangered or sensitive species.

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## 5.5 Valued Environmental Components (VECS)

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 using information gathered from previous site investigations (SENES, 2003; PWGSC, 2002; ESG, 1997) the following ecological receptors were selected for inclusion in the quantitative ERA for Radio Island:

- 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*).

Receptors were selected to be typical and representative of those likely to be found at Radio Island, 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 (1996b, c), 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 D**). In this assessment it was assumed that a collared lemming would be present at Site A for 100% of the time. It was also assumed that a collared lemming would be present at Site B for 5% of the time and in background areas for 95% of the time.



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 (US EPA, 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 D**). Based on the size of the island, the size of the site and the home range of the ermine it was assumed that the ermine spent 49.5% of its time at Site A, 0.5% of its time at Site B and 50% of its time in background areas.

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 D**). Native peoples consume the meat and use the hides of arctic hare for clothing and bandages (Gorog, 2003). Based on the size of the island, the size of the site and the home range of the arctic hare it was assumed that the hare spent 50.86% of its time at Site A, 0.45% of its time at Site B and 48.69% of its time in background areas.

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 D**). The Arctic fox is highly valued for its fur and is an important source of income for Native peoples (CWS & CWF, 2005). Based on the size of the island, the size of the site and the home range of the arctic fox it was assumed that the fox spent 9.80% of its time at Site A, 0.20% of its time at Site B and 90.00% of its time in background areas.

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 D**). They are valued for their meat and hunted by local residents (CWS & CWF, 2005). Based on the size of the island, the size of the site and the home range of the rock ptarmigan it was assumed that the ptarmigan spent 19.80% of its time at Site A, 0.20% of its time at Site B and 80.00% of its time in background areas.

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 D**). Based on the size of the island, the size of the site and the home range of the snowy owl it was assumed that the owl spent 9.80% of its time at Site A, 0.20% of its time at Site B and 90.00% of its time in background areas.

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.

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

Three species at risk are found in the Radio Island region (see Table 5-5).

**Table 5-5: Species at Risk**

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

These species were not chosen as representative receptors because the snowy owl, ermine, and arctic fox have similar exposure pathways but have smaller home ranges and subsequently greater exposure to contaminants at the site. Therefore, if contaminant levels are below toxic thresholds for these species, it can be deduced that concentrations are also safe for the peregrine falcon, wolverine, and polar bear.

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## 5.6 Exposure Assessment

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### 5.6.1 Potential Exposure Pathways

In order for chemicals to have deleterious effects, they need to gain access to the 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 the following four criteria (USEPA 1989):

- a source of contaminants of concern must be present;
- release and transport mechanisms and media must be available to move the chemicals from the source to the ecological receptors;
- an opportunity must exist for the ecological receptors to contact the affected media; and
- a means must exist by which the chemical is taken up by ecological receptors, such as ingestion, inhalation, or direct contact.

The sources of the contaminants of concern for the study area were mainly surface soil and surface water. Subsurface soils and groundwater were not considered as potential sources of contaminant exposure for wildlife. There were no direct exposure pathways for ecological receptors for either of these environmental media and transport of contaminants from these sources to surface soil and surface water was expected to be negligible.

An exposure route is the mechanism by which a receptor species might be exposed to a chemical from the source. For surface soils and terrestrial receptors, including mammals and birds, exposure to contaminants of concern may occur through the following routes:

- dermal contact with soils;
- incidental ingestion of soil (i.e., as a result of feeding or grooming);
- ingestion of plants or prey species that have accumulated chemicals from the soil; or
- inhalation of volatile contaminants migrating from the soil to ambient air.

The inhalation pathway is typically of negligible importance for wildlife receptors in open air situations, and the CoPCs for the ERA typically have low or negligible vapour pressures. Therefore, inhalation has not been considered a significant exposure pathway for the ERA at the site. Assessment of dermal contact with soils is included with incidental ingestion of soil.

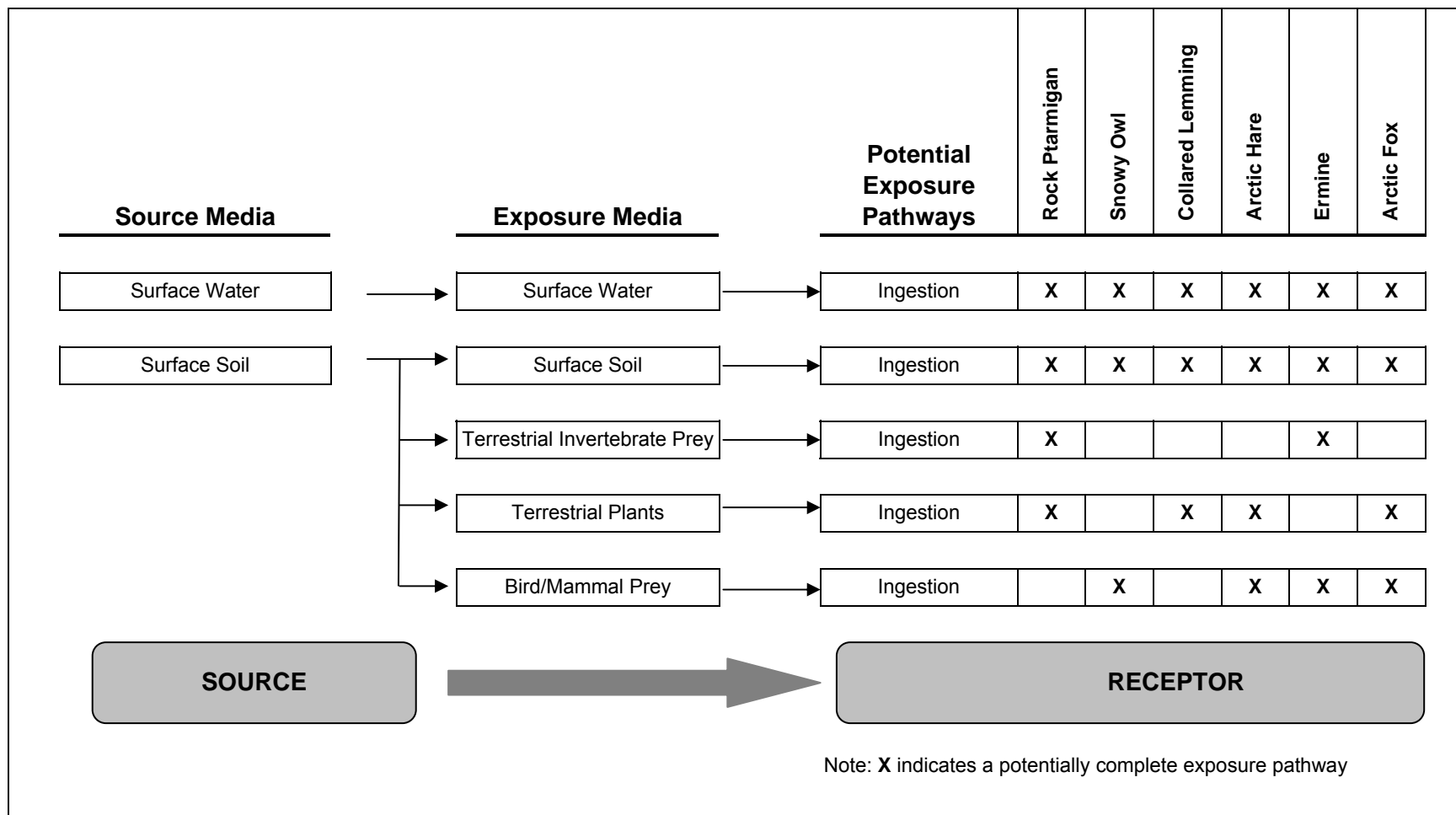
The choice of site-specific exposure pathways is dependent on the nature of the CoPCs, their source environmental media, and nature of VECs being considered in the ecological risk assessment. These are explained, for the site, in the following section.

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### 5.6.2 Conceptual Model

A conceptual site model was developed for the site and is presented in Figure 5-1. This figure schematically represents the interactions between the VECs and the CoPCs, via the exposure pathways identified. The relevant exposure pathways are designated by arrows leading from the contaminant source media to each VEC. The pathway is considered to be complete (i.e., functioning) for a VEC when the exposure pathway box is marked with an X. The conceptual model shows how the pathways representing soil and water ingestion, and ingestion of food items (including terrestrial plants, and small mammal prey items) have been conceptualized and implemented for each VEC.

Figure 5-1: ERA Conceptual Model



### 5.6.3 Selection of Assessment and Measurement Endpoints

Assessment endpoints are explicit expressions of environmental values or characteristics to be protected at a site, 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 importance of the species to the function of the ecosystem. Therefore, evaluation of 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 Radio Island, assessment endpoints focus on maintenance and protection of their populations, such that contaminants in the surface water, sediment, or soil would not significantly impact either species abundance or diversity through increased mortality or decreased reproduction.

The information needed to deal directly with the assessment endpoint is difficult to generate and rarely available; thus measurement endpoints are used to bridge the gap. Measurement endpoints are measurable responses to stressors related to assessment endpoints, and are intended to provide a basis for assessing risk potential for the assessment endpoint. They may be defined in terms of an unacceptable level 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 interaction between a VEC and a contaminant of concern is typically limited by available toxicity data. Those most commonly used to quantify the survival, growth and reproduction of receptors in bioassays include the  $LC_{50}$  and  $LD_{50}$  (concentrations or doses that will be lethal to 50% of exposed organisms, over a defined period of exposure); the  $EC_{50}$  and  $ED_{50}$  (concentrations or doses that elicit a defined 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 included in order to consider the response of species in the natural environment.

The measurement endpoints for the assessment endpoints focus on whether observed concentrations of chemicals in water or soils are likely to result in doses to birds or mammals that are greater than those observed to result in increased mortality or decreased reproduction upon chronic exposure.

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

- characterization of relationships between amount of a chemical present in surface water or sediments and a thresholds for adverse effects; and
- characterization of relationships between the dose resulting from the amount of a chemical present in surface soils and a threshold dose for adverse effects.

The dose-response relationships that have been incorporated into this ERA are based upon LOAELs, in relation to 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 body weight of the test organism (i.e., the reference toxicity dose or RTD value 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  $LD_{50}$  value. Standard conversion factors were implemented including division by 5 to convert an acute dose to a chronic dose; dividing by 6 to convert an  $LD_{50}$  value to a LOAEL value, or multiplication by 5 to convert a NOAEL to a LOAEL value. These

conversion factors are cumulative, so an acute LD<sub>50</sub> would be converted to a chronic LOAEL value by dividing by 30.

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

$$\text{Mammal Body Weight SF} = (BW_t/BW_r)^{0.06}$$

where:

SF = scaling factor

BW<sub>t</sub> = mean body weight for test species

BW<sub>r</sub> = mean body weight for receptor species

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

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

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## 5.7 Risk Characterization

Risk characterization is the final step of the ecological risk assessment. It includes a quantification of the potential nature and magnitude of adverse effects that may occur to receptor species due to presence of chemicals in identified ecological habitats at the site. In this step, characterization of exposure and characterization of ecological effects for each chemical is integrated into quantitative estimates (hazard quotients or HQ values) of the potential for adverse effects to ecological receptors.

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### 5.7.1 Approach

For this assessment, ecological hazard quotient (HQ) values were calculated by dividing exposure (as the exposure point concentrations or total ingested dose values) derived for each receptor by their appropriate reference toxicity dose (RTD), as follows:

$$\text{HQ} = (\text{Exposure}) / (\text{RTD})$$

For birds and mammals, the exposure measure is the total ingested dose (mg/kg-day) summed over all exposure pathways.

An HQ value of less than 1.0 indicates the exposure concentration is less than the threshold for adverse effects, and a low probability exists that adverse effects might occur. Given the overall tendency to introduce conservatism (through the use of data or assumptions that are likely to overstate, rather than understate risk) into risk assessments, it is likely no adverse effect would occur. Alternatively, a HQ value of >1.0 does not automatically indicate that there is an unacceptable level of risk. In this case, the conservative approach reduces the certainty of this conclusion, and dictates a need for more careful review of both predicted exposure levels and exposure limit derivations. As a result, HQ values greater than 1.0 should be examined carefully, and further more focused investigations may be required to reduce conservatism and provide a more realistic assessment of the

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

### 5.7.2 Determination of Media to Biota Uptake Factors

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

Substances retained in the ERA as CoPCs are listed in Table 5-4. The approaches used to estimate uptake factors for each of these groups of substances, for each of the required biological food groups, are described below. The specific uptake factors used, with references as to the source of the uptake factor, can be found in the ERA model outputs located in **Appendix E**. Some general information on the sources of data used for media to biota accumulation factors is presented below.

#### Soil to Plant Uptake Factors

Soil to plant uptake factors for organic substances are generally calculated using the equation of Travis and Arms (1988):

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

where UFSP is the uptake factor from 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 inorganic substances, data on plant tissue metal concentrations were collected from the site by the Environmental Sciences Group of the Royal Military College in 1996 (ESG 1997). EPC values were calculated by taking the average value for each inorganic substance and multiplying them by a conversion factor representing the average dry solids fraction (0.40) to obtain wet-weight tissue concentrations.

#### Soil to Animal Uptake Factors

For organic substances, soil to animal (i.e., caribou meat) uptake factors were generally calculated using the equation of Travis and Arms (1988):

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

where  $\text{Ba}_{p,s,w}$  is the transfer factor from soil to beef (day/kg), which is assumed to also be applicable to caribou meat. These 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) to estimate the concentration in meat (mg/kg). For the TPH substances, which are not as readily absorbed and which are more readily metabolized than the pesticide compounds that form the basis of the Travis and Arms data, a bioavailability factor (which can range from 0 to 1) is also applied.

For inorganic substances in meat, a similar approach is used for concentrations in meat, except that the transfer factors to meat (Bap,s,w) were obtained from the compilation of Baes *et al.* (1984).

For small animal prey items, including lemming, ptarmigan and Arctic hare, a variety of approaches and data sources were used. For organics, the approach of Travis and Arms (1988) was followed. For inorganic substances, the equations of Sample *et al.* (1998), which directly calculate the CoPC concentration in small mammal tissues from the soil concentration, are generally preferred. Where these equations are not available, the approach of Baes *et al.* (1984) was also used.

### **Soil to Soil Invertebrate Uptake Factors**

For soil to soil invertebrate uptake factors (UPSI) for the TPH compounds, conservative default uptake factors of 0.1 were assumed. For inorganic substances, the equations of Sample *et al.* (1998) were preferred.

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## **5.7.3 Determination of Reference Toxicity Doses**

RTDs for terrestrial receptors were included in the risk assessment model results for each receptor, and are presented in **Appendix E**. The RTD values are unique to each CoPC.

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## **5.7.4 Risk Characterization of Avian Receptors**

Tables showing the derivation of risk estimates for avian receptors can be found in **Appendix E**. The text below provides a synopsis of the risk estimates for each avian VEC. HQ values for the rock ptarmigan and snowy owl are presented in Table 5-6 and 5-7, respectively. Tables present HQs calculated for each CoPC from Area A, Area B and background concentrations and total HQ (i.e., summation of the Area A, Area B and background HQs).

### **5.7.4.1 Risk Estimates for Rock Ptarmigan**

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

TPH (HQ = 3.90), chromium (HQ = 1.8) and zinc (HQ = 1.21) were above the target HQ (i.e., 1.0) for the ptarmigan. The TPH risk value represents the total HQ from all four fractions. TPH fractions F2 and F3 from Area A contribute the largest risk, with HQs for each fraction equaling 1.58 and 1.42, respectively. For chromium, a majority of the total risk on-site is from the background soil concentrations (HQ = 1.42), not from samples collected in areas A and B. For zinc, the majority of total risk on-site comes from both background soil concentrations (HQ = 0.47) and from area A (HQ = 0.74). Ingestion of soil is the dominant exposure pathways leading to a high HQ value for TPH; risks due to ingestion of plants and surface water are negligible. For chromium, the dominant exposure pathway was ingestion of terrestrial plants with negligible risk from ingestion of soil and surface water. Ingestion of terrestrial plants is the dominant exposure pathway for zinc; negligible risk comes from other pathways.

All other substances that were assessed had HQ values that were below 1.0.



**Table 5-6: Ecological Hazard Quotients for Rock Ptarmigan**

CoPC	Total HQ	Area A	Area B	Background
<b>TPH</b>				
Aliph>C06-C08 - F1	1.17E-03	1.17E-03		
Aliph>C08-C10 - F1	6.16E-04	6.16E-04		
Arom>C08-C10 - F1	6.41E-04	6.41E-04		
F1 – Total	2.43E-03	2.43E-03		
Aliph>C10-C12 - F2	3.37E-01	3.37E-01		
Aliph>C12-C16 - F2	3.54E-01	3.54E-01		
Arom>C10-C12 - F2	4.18E-01	4.18E-01		
Arom>C12-C16 - F2	4.74E-01	4.74E-01		
F2 – Total	<b>1.58E+00</b>	<b>1.58E+00</b>		
Aliph>C16-C21 - F3	6.02E-01	6.02E-01		
Aliph>C21-C34 - F3	2.40E-01	2.40E-01		
Arom>C16-C21 - F3	3.93E-01	3.93E-01		
Arom>C21-C34 - F3	1.88E-01	1.88E-01		
F3 – Total	<b>1.42E+00</b>	<b>1.42E+00</b>		
Aliph>C34-C50 - F4	1.49E-01	1.49E-01		
Arom>C34-C50 - F4	7.41E-01	7.41E-01		
F4 – Total	8.90E-01	8.90E-01		
Total TPH	<b>3.90E+00</b>	<b>3.90E+00</b>		
<b>PCBs</b>				
Total PCBs	4.73E-04	1.59E-04	1.29E-07	3.14E-04
<b>Inorganics</b>				
Antimony	4.17E-02	4.17E-02		
Arsenic	5.85E-03	2.23E-03		3.62E-03
Barium	7.19E-03	7.19E-03		
Boron	1.73E-02	1.72E-02	1.25E-04	
Cadmium	3.78E-02	1.81E-02	3.28E-05	1.97E-02
Chromium (Total)	<b>1.80E+00</b>	3.77E-01		<b>1.42E+00</b>
Cobalt	3.71E-01	1.05E-01		2.66E-01
Copper	6.46E-02	3.00E-02	5.50E-08	3.46E-02
Lead	5.30E-01	4.02E-01	3.63E-04	1.28E-01
Mercury - Inorganic	4.34E-02	1.28E-02		3.06E-02
Nickel	8.80E-02	1.59E-02	1.94E-05	7.21E-02
Selenium	2.54E-02	2.50E-02	3.72E-04	
Silver	1.76E-03	1.76E-03		
Tin	6.57E-03	6.57E-03		
Zinc	<b>1.21E+00</b>	7.38E-01	3.18E-04	4.70E-01



### 5.7.4.2 Risk Estimates for Snowy Owl

For the Snowy Owl the intake pathways included ingestion of surface water, surface soil, and small mammals. The Snowy Owl feeds mainly on small mammals.

Risks (HQ values) for the Snowy Owl were much less than 1.0 for all substances at both sites and background, which suggests that the Snowy Owl is not at risk from any of these substances at Radio Island.

**Table5-7: Ecological Hazard Quotients for Snowy Owl**

CoPC	Total HQ	Area A	Area B	Background
<b>TPH</b>				
Aliph>C06-C08 - F1	1.56E-05	1.56E-05		
Aliph>C08-C10 - F1	1.28E-05	1.28E-05		
Arom>C08-C10 - F1	7.47E-06	7.47E-06		
F1 - Total	3.60E-05	3.60E-05		
Aliph>C10-C12 - F2	1.87E-02	1.87E-02		
Aliph>C12-C16 - F2	1.72E-01	1.72E-01		
Arom>C10-C12 - F2	5.65E-03	5.65E-03		
Arom>C12-C16 - F2	7.08E-03	7.08E-03		
F2 - Total	2.03E-01	2.03E-01		
Aliph>C16-C21 - F3	2.70E-01	2.70E-01		
Aliph>C21-C34 - F3	1.74E-02	1.74E-02		
Arom>C16-C21 - F3	8.50E-03	8.50E-03		
Arom>C21-C34 - F3	5.91E-03	5.91E-03		
F3 - Total	3.02E-01	3.02E-01		
Aliph>C34-C50 - F4	7.54E-02	7.54E-02		
Arom>C34-C50 - F4	8.63E-02	8.63E-02		
F4 - Total	1.62E-01	1.62E-01		
Total TPH	6.67E-01	6.67E-01		
<b>PCBs</b>				
Total PCBs	2.29E-04	2.71E-05	5.45E-08	2.02E-04
<b>Inorganics</b>				
Antimony	9.74E-04	9.74E-04		
Arsenic	1.50E-04	3.90E-05		1.11E-04
Barium	1.80E-04	1.80E-04		
Boron	6.74E-05	6.64E-05	9.74E-07	
Cadmium	9.71E-04	1.68E-04	3.55E-06	7.99E-04
Chromium (Total)	3.55E-02	2.55E-03		3.29E-02
Cobalt	8.85E-03	1.46E-03		7.39E-03
Copper	5.36E-03	7.37E-04	3.35E-08	4.62E-03
Lead	1.33E-02	8.46E-03	5.69E-05	4.74E-03
Mercury - Inorganic	2.07E-04	2.07E-04		
Nickel	2.02E-03	2.28E-04	3.85E-06	1.79E-03
Selenium	3.82E-03	3.73E-03	9.00E-05	
Silver	1.33E-05	1.33E-05		
Tin	1.39E-03	1.39E-03		
Zinc	3.90E-02	5.29E-03	8.79E-05	3.36E-02

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## 5.7.5 Risk Characterization of Mammalian Receptors

Tables showing the derivation of risk estimates for mammalian receptors can be found in **Appendix E**. The text below provides a synopsis of the risk estimates for each mammalian VEC. HQ values for the collared lemming, ermine, arctic fox and arctic hare are presented in Table 5-8 to 5-11, respectively. Tables present HQs calculated for each CoPC from Area A, Area B and background concentrations and total HQ (i.e., summation of the Area A, Area B and background HQs).

### 5.7.5.1 Risk Estimates for Collared Lemming

For the collared lemming the intake pathways include ingestion of surface water, surface soil, and terrestrial plants. The lemming feeds on vegetation including grasses and shrubs, and bark and twigs of willow and birch.

TPH (HQ = 3.78) and chromium (HQ = 2.45) were above the target HQ (i.e., 1.0) for the lemming. The TPH risk value represents the total HQ from all four fractions. TPH fractions F2 and F3 from Area A contribute the largest risk, with HQs for each fraction equaling 2.35 and 1.37, respectively. For chromium, the majority of the total risk on-site was from the background soil concentration (HQ = 1.14) and Area A (HQ = 1.31), with a negligible risk from Area B.

Ingestion of surface soils is the dominant exposure pathway leading to a high HQ value for TPH; risks due to ingestion of plants and surface water are negligible. For chromium, the dominant exposure pathway was ingestion of terrestrial plants with negligible risk from ingestion of soil and surface water

All other substances that were assessed had HQ values that were below 1.0.

**Table 5-8: Ecological Hazard Quotients for Collared Lemming**

CoPC	Total HQ	Area A	Area B	Background
<b>TPH</b>				
Aliph>C06-C08 - F1	1.51E-03	1.51E-03		
Aliph>C08-C10 - F1	7.37E-04	7.37E-04		
Arom>C08-C10 - F1	1.33E-03	1.33E-03		
F1 - Total	3.58E-03	3.58E-03		
Aliph>C10-C12 - F2	3.21E-01	3.21E-01		
Aliph>C12-C16 - F2	3.46E-01	3.46E-01		
Arom>C10-C12 - F2	8.01E-01	8.01E-01		
Arom>C12-C16 - F2	8.87E-01	8.87E-01		
F2 - Total	<b>2.35E+00</b>	<b>2.35E+00</b>		
Aliph>C16-C21 - F3	3.55E-01	3.55E-01		
Aliph>C21-C34 - F3	1.52E-01	1.52E-01		
Arom>C16-C21 - F3	6.30E-01	6.30E-01		
Arom>C21-C34 - F3	2.32E-01	2.32E-01		
F3 - Total	1.37E+00	1.37E+00		
Aliph>C34-C50 - F4	1.59E-02	1.59E-02		
Arom>C34-C50 - F4	4.17E-02	4.17E-02		
F4 - Total	5.75E-02	5.75E-02		
<b>Total TPH</b>	<b>3.78E+00</b>	<b>3.78E+00</b>		
<b>PCBs</b>				
Total PCBs	1.45E-03	7.79E-04	3.80E-06	6.63E-04
<b>Inorganics</b>				
Antimony	2.73E-02	2.73E-02		
Arsenic	8.26E-02	5.84E-02		2.42E-02
Barium	6.29E-02	6.29E-02		
Boron	6.10E-02	5.89E-02	2.12E-03	
Cadmium	1.28E-01	1.03E-01	1.95E-04	2.49E-02
Chromium (Total)	<b>2.45E+00</b>	<b>1.31E+00</b>		<b>1.14E+00</b>
Cobalt	1.16E-01	7.23E-02		4.37E-02
Copper	6.53E-01	5.14E-01	1.11E-05	1.39E-01
Lead	2.21E-01	2.01E-01	4.78E-04	1.96E-02
Mercury - Inorganic	6.28E-03	6.28E-03		
Nickel	1.33E-01	6.36E-02	1.65E-04	6.93E-02
Selenium	2.00E-01	1.86E-01	1.42E-02	
Silver	5.75E-03	5.75E-03		
Tin	8.47E-03	8.47E-03		
Zinc	9.61E-01	8.39E-01	7.19E-04	1.21E-01

### 5.7.5.2 Risk Estimates for Ermine

For the ermine the intake pathways included ingestion of surface water, surface soil, small mammals, soil invertebrates, and terrestrial plants. The ermine feeds mainly on small mammals, but also consumes invertebrates and plant material as minor components of its diet.

TPH had a HQ (4.49) above the target HQ (i.e., 1.0) for the ermine. The TPH risk value represents the total HQ from all four fractions. TPH fractions F2 and F3 from Area A contribute the largest risk, with HQs for each fraction equaling 2.13 and 2.16.

Ingestion of surface soil and small mammals are the dominant exposure pathways leading to the high HQ value for TPH; risks due to ingestion of plants and surface water are negligible.

All other substances that were assessed had HQ values that were below 1.0.

**Table 5-9: Ecological Hazard Quotients for Ermine**

CoPC	Total HQ	Area A	Area B	Background
<b>TPH</b>				
Aliph>C06-C08 - F1	1.66E-04	1.66E-04		
Aliph>C08-C10 - F1	1.30E-04	1.30E-04		
Arom>C08-C10 - F1	1.26E-04	1.26E-04		
F1 – Total	4.22E-04	4.22E-04		
Aliph>C10-C12 - F2	1.87E-01	1.87E-01		
Aliph>C12-C16 - F2	1.73E+00	1.73E+00		
Arom>C10-C12 - F2	9.43E-02	9.43E-02		
Arom>C12-C16 - F2	1.16E-01	1.16E-01		
F2 – Total	<b>2.13E+00</b>	<b>2.13E+00</b>		
Aliph>C16-C21 - F3	1.82E+00	1.82E+00		
Aliph>C21-C34 - F3	1.13E-01	1.13E-01		
Arom>C16-C21 - F3	1.31E-01	1.31E-01		
Arom>C21-C34 - F3	9.86E-02	9.86E-02		
F3 – Total	<b>2.16E+00</b>	<b>2.16E+00</b>		
Aliph>C34-C50 - F4	8.15E-02	8.15E-02		
Arom>C34-C50 - F4	1.28E-01	1.28E-01		
F4 – Total	2.09E-01	2.09E-01		
Total TPH	<b>4.49E+00</b>	<b>4.49E+00</b>		
<b>PCBs</b>				
Total PCBs	2.29E-03	1.27E-03	1.24E-06	1.02E-03
<b>Inorganics</b>				
Antimony	6.40E-03	6.40E-03		
Arsenic	8.45E-03	6.39E-03		2.06E-03
Barium	1.19E-02	1.19E-02		
Boron	1.11E-03	1.10E-03	7.97E-06	
Cadmium	8.96E-03	5.93E-03	6.23E-05	2.97E-03
Chromium (Total)	1.02E-01	4.24E-02		5.92E-02
Cobalt	7.94E-03	5.11E-03		2.83E-03
Copper	1.08E-01	6.33E-02	1.40E-06	4.46E-02
Lead	2.71E-02	2.54E-02	8.66E-05	1.62E-03
Mercury - Inorganic	7.19E-04	7.19E-04		
Nickel	8.49E-03	4.52E-03	3.82E-05	3.93E-03
Selenium	1.70E-01	1.68E-01	2.00E-03	
Silver	7.84E-04	7.84E-04		
Tin	1.70E-02	1.70E-02		
Zinc	5.11E-02	3.00E-02	2.48E-04	2.09E-02

### 5.7.5.3 Risk Estimates for Arctic Fox

For the Arctic fox the intake pathways included ingestion of surface water, surface soil, small mammals, and terrestrial plants. The Arctic fox feeds mainly on small mammals, but also consumes plant material as a minor component of its diet.

Risks (HQ values) for the arctic fox were less than 1.0 for all substances at both sites and background, which suggests that the arctic fox is not at risk from any of these substances at Radio Island.

**Table 5-10: Ecological Hazard Quotients for Arctic Fox**

CoPC	Total HQ	Area A	Area B	Background
<b>TPH</b>				
Aliph>C06-C08 - F1	1.91E-05	1.91E-05		
Aliph>C08-C10 - F1	1.48E-05	1.48E-05		
Arom>C08-C10 - F1	1.46E-05	1.46E-05		
F1 – Total	4.85E-05	4.85E-05		
Aliph>C10-C12 - F2	2.04E-02	2.04E-02		
Aliph>C12-C16 - F2	1.85E-01	1.85E-01		
Arom>C10-C12 - F2	1.06E-02	1.06E-02		
Arom>C12-C16 - F2	1.31E-02	1.31E-02		
F2 – Total	2.29E-01	2.29E-01		
Aliph>C16-C21 - F3	1.94E-01	1.94E-01		
Aliph>C21-C34 - F3	1.26E-02	1.26E-02		
Arom>C16-C21 - F3	1.48E-02	1.48E-02		
Arom>C21-C34 - F3	1.01E-02	1.01E-02		
F3 – Total	2.31E-01	2.31E-01		
Aliph>C34-C50 - F4	8.73E-03	8.73E-03		
Arom>C34-C50 - F4	1.28E-02	1.28E-02		
F4 – Total	2.16E-02	2.16E-02		
Total TPH	4.81E-01	4.81E-01		
<b>PCBs</b>				
Total PCBs	4.18E-04	1.34E-04	2.68E-07	2.84E-04
<b>Inorganics</b>				
Antimony	7.40E-04	7.40E-04		
Arsenic	2.98E-03	8.24E-04		2.16E-03
Barium	1.48E-03	1.48E-03		
Boron	2.42E-04	2.38E-04	3.50E-06	
Cadmium	2.00E-03	7.51E-04	1.14E-05	1.24E-03
Chromium (Total)	5.61E-02	7.25E-03		4.88E-02
Cobalt	3.42E-03	7.11E-04		2.71E-03
Copper	2.39E-02	7.87E-03	4.41E-07	1.60E-02
Lead	4.02E-03	3.12E-03	1.88E-05	8.84E-04
Mercury – Inorganic	9.02E-05	9.02E-05		
Nickel	3.62E-03	6.15E-04	8.23E-06	3.00E-03
Selenium	1.87E-02	1.83E-02	4.44E-04	
Silver	5.91E-05	5.91E-05		
Tin	1.82E-03	1.82E-03		
Zinc	1.28E-02	4.85E-03	5.16E-05	7.87E-03

### 5.7.5.4 Risk Estimates for Arctic Hare

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

TPH (HQ = 1.69) and chromium (HQ = 1.01) were above the target HQ (i.e., 1.0) for the arctic hare. The TPH risk value represents the total HQ from all four fractions. TPH fraction F2 from Area A contributes the largest risk, with a HQ equaling 1.03. For chromium, the majority of the total risk on-site was from the background soil concentration (HQ = 0.47) and Area A (HQ = 0.54), with a negligible risk from Area B.

Ingestion of surface soil and terrestrial plants are the dominant exposure pathways leading to a high HQ value for TPH; risks due to surface water ingestion are negligible. For chromium, the dominant exposure pathway was ingestion of terrestrial plants with negligible risk from ingestion of soil and surface water.

All other substances that were assessed had HQ values that were below 1.0.

**Table 5-11: Ecological Hazard Quotients for Arctic Hare**

CoPC	Total HQ	Area A	Area B	Background
<b>TPH</b>				
Aliph>C06-C08 - F1	6.26E-04	6.26E-04		
Aliph>C08-C10 - F1	3.08E-04	3.08E-04		
Arom>C08-C10 - F1	5.52E-04	5.52E-04		
F1 – Total	1.49E-03	1.49E-03		
Aliph>C10-C12 - F2	1.39E-01	1.39E-01		
Aliph>C12-C16 - F2	1.92E-01	1.92E-01		
Arom>C10-C12 - F2	3.33E-01	3.33E-01		
Arom>C12-C16 - F2	3.69E-01	3.69E-01		
F2 – Total	<b>1.03E+00</b>	<b>1.03E+00</b>		
Aliph>C16-C21 - F3	1.98E-01	1.98E-01		
Aliph>C21-C34 - F3	6.69E-02	6.69E-02		
Arom>C16-C21 - F3	2.65E-01	2.65E-01		
Arom>C21-C34 - F3	9.90E-02	9.90E-02		
F3 – Total	6.29E-01	6.29E-01		
Aliph>C34-C50 - F4	8.89E-03	8.89E-03		
Arom>C34-C50 - F4	2.07E-02	2.07E-02		
F4 – Total	2.96E-02	2.96E-02		
Total TPH	<b>1.69E+00</b>	<b>1.69E+00</b>		
<b>PCBs</b>				
Total PCBs	6.83E-04	3.55E-04	3.07E-07	3.28E-04
<b>Inorganics</b>				
Antimony	1.16E-02	1.16E-02		
Arsenic	3.43E-02	2.42E-02		1.01E-02
Barium	2.65E-02	2.65E-02		
Boron	2.43E-02	2.41E-02	1.53E-04	
Cadmium	5.25E-02	4.21E-02	1.54E-05	1.04E-02
Chromium (Total)	<b>1.01E+00</b>	5.37E-01		4.73E-01
Cobalt	4.80E-02	2.98E-02		1.82E-02
Copper	2.72E-01	2.12E-01	8.41E-07	5.96E-02
Lead	9.15E-02	8.33E-02	3.74E-05	8.14E-03
Mercury - Inorganic	2.61E-03	2.61E-03		
Nickel	5.50E-02	2.62E-02	1.31E-05	2.88E-02
Selenium	8.21E-02	8.10E-02	1.08E-03	
Silver	2.38E-03	2.38E-03		
Tin	3.98E-03	3.98E-03		
Zinc	3.95E-01	3.44E-01	5.80E-05	5.11E-02

---

## 5.8 Ecological Site-Specific Target Levels

Based upon the results of the ecological risk assessment, TPH, chromium and zinc all had HQ values greater than 1.0. The following VECs were identified as having the highest HQ for each of these CoPCs:

- Ermine (TPH);
- Collared Lemming (chromium); and
- Rock Ptarmigan (zinc)

Consequently, site-specific target levels (SSTLs) were calculated for each of these CoPCs. SSTLs are generally calculated by setting the HQ to 1.0, and determining the corresponding surface soil EPC for that HQ using a backward calculation. The elevated HQ values for chromium and zinc at Site A were due to high concentrations observed in on-site plant materials. As our model does not permit back calculations from plant concentrations to corresponding soil concentrations, we recommend that SSTL values for chromium and zinc be set to background concentrations. The SSTLs for each CoPCs are presented in Table 5-12.

**Table 5-12: Site Specific Target Levels in Surface Soils at Radio Island**

VEC	CoPC	Maximum Soil Concentration (mg/kg)	Surface Soil SSTL (mg/kg)
Ermine	Total Petroleum Hydrocarbons	74,050	10,883
Collared Lemming	Chromium	85	41
Rock Ptarmigan	Zinc	26,640	46.8

SSTLs for TPH, chromium and zinc are all well below the maximum concentrations measured in surface soils; thus clean-up to SSTL levels will help protect ecological receptors from adverse effects of exposure to contaminated areas of Radio Island.

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## 5.9 Uncertainty Analysis

Uncertainties are inherent in every aspect of the ERA process. The most effective way to decrease uncertainty is to collect site-specific data. Application of site-specific information assists in reduction of uncertainty by allowing removal of generic data that may be broadly and inaccurately applicable to a wide range of sites and cases. For Radio Island, a variety of site-specific data has been collected, representing soils, surface water, and vegetation.

Despite incorporation of a considerable amount of site-specific data, the ERA involves many assumptions, and incorporates simplifications and uncertainty with respect to the characteristics of the receptors, exposure pathways, and CoPC concentrations in the environment. This section qualitatively discusses some significant aspects of uncertainty inherent in this risk assessment.

---

### 5.9.1 Data Limitations

The quality of a risk assessment calculation often hinges on the size, extent and condition of the supporting data. In addition to making use of existing site data, a large number of samples were collected for this risk assessment, and a significant amount of data was collected for this study,

including both chemical and biological data. The time available for collection of data precluded consideration of fluctuations in measured concentrations due to daily or seasonal influences. Because some of these data sets were summarized statistically, including calculation of a conservative representative value, such as the 95% UCL as the EPC, the values presented are conservative estimators of the true concentration to which native species would be exposed.

Key limitations in the ERA included insufficient background data for inorganic substances in soils, inadequate sampling of plants and TPH onsite. It is possible that the concentrations of some of the substances that were carried forward as CoPCs are not elevated as a result of human activities, but reflect natural background levels. In particular, the concentrations of inorganic substances measured in plant tissue samples from the Area A of Radio Island by ESG (1997) are high, and suggest very high soil-to-plant concentration ratios. General practice typically collects seven to ten background samples; in contrast, data for the current site is limited to four soil and three vegetation samples. Additional sampling of both soil and vegetation would decrease the uncertainty in the model.

Conversely, there was insufficient sampling done for vegetation and TPH onsite. In total, nine vegetation samples and six TPH sample, with only one sample taken at Area B. Of the nine vegetation samples, only five would be available to receptors, two were plant roots which are generally inaccessible, and two samples could not be identified as root or shoot or whole plant and were therefore excluded from calculations. Increase sampling would give a better profile onsite vegetation and TPH levels, as well as reduce any uncertainty in the model.

---

### **5.9.2 Selection of CoPCs**

CoPCs were selected independently in each of the media evaluated in the ecological risk assessment, and the analysis was completed to include all media (surface water, soils, and biota exposed to these media) if the substance exceeded screening criteria for any one of these. For each of the media, there are gaps in understanding of the toxicology of CoPCs, and the physical and chemical properties of these chemicals. The approach for selecting CoPCs included comparison of each detected chemical value to values that are believed to be protective of most North American species, in most ecosystems. Because empirical data do not exist for all possible 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.

---

### **5.9.3 Chemical Speciation**

The fate, food chain interactions, and toxicity of a number of inorganic and organic contaminants (including TPH and the metals evaluated here) depend to a large extent upon their chemical form, and the context in which they are ingested. As such, conservative assumptions about chemical form, bioavailability, and absorption over the gut were generally carried forward in the risk assessment, and the potential for toxicity is likely to be overstated. For example, it has been generally assumed that 100% of each ingested CoPC is absorbed from ingested soil, water, or food, and is available to the organism as a potentially toxic substance. This may be reasonable for some CoPCs, but will be highly conservative for others.



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#### **5.9.4 Food Chain Interactions**

Very limited "real world" data exist that allow quantification of the true relationship between a chemical in an environmental medium and chemical transfer through the food chain. Only a few classes of chemicals appear to be magnified through the food chain. These substances include methyl mercury, some PCBs, some chlorinated pesticides (such as DDT), and some PCDD/PCDF compounds. These substances all have a tendency to partition into fatty tissue rather than water. They are also resistant to natural degradation processes by metabolic enzymes. The TPH substances and PAHs are also hydrophobic classes of chemicals present in the environment. While they are hydrophobic, they may only partially absorbed following ingestion, and may also be metabolized and/or excreted by some invertebrates and most vertebrates. For this reason, food chain magnification does not tend to occur with TPH or PAHs. The extent of food chain magnification is another uncertainty that is generally treated in a conservative manner. Additional collection and chemical analysis of tissue samples from mammalian and avian species could have further reduced uncertainties associated with these values but were beyond the scope of the ecological field program.

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#### **5.9.5 Wildlife Exposure Factors**

Virtually every factor incorporated into dose calculations for wildlife species possesses a site-specific component. Validity of each exposure factor is dependent on consideration of the site-specific nature of these factors. In the absence of site-specific validation, exposure factors are incorporated based on validations performed elsewhere for other cases and sometimes for other species. Considerations such as food ingestion rates, 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 locations. It has been assumed that each receptor organism spends its entire life cycle at the Radio Island site. On the basis of this assumption, the VECs are modeled as being exposed to the 95% UCL concentration for each CoPC. Therefore, it is likely that the level of wildlife exposure has been substantially overestimated, particularly for migratory VECs.

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#### **5.9.6 Habitat Survey and VEC Selection**

This risk assessment invested significant effort into consideration of existing habitats and the species that exist within them. Both aquatic and terrestrial habitats were evaluated to identify relevant species, and to support the selection of appropriate VECs. Therefore, the VECs that were selected are known to be present, or can reasonably be expected to be present on the site. These VECs are also known to be reasonably or conservatively representative of other species that may be present on the site and exposed to CoPCs. Use of site-specific receptors decreases uncertainty since local species are considered rather than highly sensitive non-native species.

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#### **5.9.7 Receptor-Specific Toxicity Data**

For most of the CoPCs and VECs, toxicity data were available in some form. However, it is important to note that toxicity data are generally not available for the particular VEC species under consideration. Toxicity values are not necessarily specific to the VEC species, or to a reproductive or population-level endpoint. As a result, there is uncertainty associated with the extrapolations that are used to translate toxicity data from a test species in the laboratory, to a receptor species in the wild. The conversion

factors that are used are scientifically based, and are applied in a manner that is believed to be conservative.

In some cases, there is 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 for a bird by incorporating an additional safety factor of 5.

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### **5.9.8 Measurement Endpoints from the Toxicity Data**

The paucity of toxicity data for many chemicals limited the measurement endpoints that were available. Where LOAEL values were not available, it was necessary to extrapolate from NOAEL values. Correction factors used for this extrapolation are relatively conservative and tend to under-estimate the LOAEL value. This approach is conservative, and if observed chemical concentrations are lower than the RTD values, there is little potential for observable adverse effects at the population level. This approach is more conservative than the suggestion of Suter (1993), that a 20% effect level (such as a 20% reduction in survivorship or growth of exposed biota) be treated as a conservative approximation of the threshold for regulatory concern. Therefore, use of these reference toxicity doses would overestimate the potential for significant adverse effects on species of concern, and overestimate the potential for significant ecological risks.

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### **5.10 Summary Of Uncertainty Analysis**

As a result of the scientific investigations, literature reviews, and risk assessment guidance that have been undertaken or followed in the preparation of this ERA, it is believed that the risk assessment results present a reasonable yet conservative evaluation of the risk to ecological receptors present at the site. Where uncertainty or lack of knowledge were encountered in the development of the risk estimates, reasonable yet conservative assumptions were made, or data were selected, in order to ensure that risks were not underestimated.

---

## **6.0 CONCLUSIONS**

Jacques Whitford has completed a human health and ecological risk assessment for the former navigational aid and weather station located at Radio Island, Nunavut. The purpose of the risk assessment was to evaluate potential risks associated with contaminants in soil samples within two areas on the Radio Island site. Concentrations were compared with applicable generic CCME soil quality criteria and carried forward into the risk assessment only if they exceeded the criteria. The following points summarize the major findings of this report:

- For the purposes of assessing potential exposures, the Radio Island site was separated into two areas, A and B;
- Data from previous reports were compiled into one dataset and soil sample concentrations were compared to applicable generic soil guidelines. In regards to the human health risk assessment component of this HHERA, maximum concentrations of antimony, arsenic, barium, beryllium, cadmium, lead, mercury, nickel, tin, zinc, TPH fractions F2 and F3, and total PCBs were found to

exceed generic human health guidelines in Areas A and B; therefore these contaminants were chosen as CoPCs;

- Antimony, arsenic, barium, beryllium, boron, cadmium, chromium, cobalt, copper, lead, mercury, nickel, selenium, silver, zinc, all TPH fractions and total PCBs exceeded generic ecological soil guidelines and were therefore chosen as CoPCs in the ERA;
- Once CoPCs were determined, EPCs were calculated for each CoPC as the 95% UCL of the geometric mean of the data sets and inputted into both the human health and ecological risk models; and
- Potential environmental and human health risks were then calculated, based on the soil EPCs, for each potential receptor for all applicable elements (varied by exposure area and receptor type), with the following outcomes:
  - Surface soil EPCs of the identified CoPCs are not anticipated to produce any adverse effects to human receptors using the Radio Island site for traditional land use purposes;
  - Surface soil EPCs of the identified CoPCs are not anticipated to produce any adverse effects to ecological receptors included in this risk assessment, with the exception of chromium, zinc and total petroleum hydrocarbons; and
  - EPC concentrations for chromium, zinc and total petroleum hydrocarbons pose a potential risk to ecological receptors. Consequently, SSTLs were developed for these specific CoPCs based on the most sensitive ecological receptors in the model. Remediation to these site-specific levels will help protect all ecological receptors from adverse effects from exposure to affected areas of Radio Island.

---

## 7.0 CLOSURE

This report has been prepared for the sole benefit of INAC. The report may not be relied upon by any other person or entity without the expressed written consent of Jacques Whitford and INAC.

Any use that a third party makes of this report, or any reliance on decisions made based on it, are the responsibility of such third parties.

Jacques Whitford accepts no responsibility for damages, if any, suffered by any third party as a result of decisions made or actions taken based on this report.

The information and conclusions contained in this report are based upon work undertaken by trained professional and technical staff in accordance with generally accepted engineering and scientific practices current at the time the work was performed. Any site-specific information provided by other parties and used or referenced by Jacques Whitford has been assumed by Jacques Whitford to be accurate. Conclusions presented in this report should not be construed as legal advice.

This risk assessment was undertaken exclusively for the purpose outlined herein and was limited to those contaminants, exposure pathways, receptors, and related uncertainties specifically referenced in this report. This work was specific to the site conditions and land use considerations described herein. The report cannot be used or applied under any circumstances to another location or situation or for any other purpose without further evaluation of the data and related limitations.

This document describes only the applicable risks associated with the identified environmental hazards, and is not intended to imply a risk-free site. Should any conditions at the site be observed or discovered that differ from those at the sample locations, or should the land use surrounding the identified hazards change significantly, we request that we be notified immediately to reassess the conclusions provided herein.

Yours truly,

**JACQUES WHITFORD LIMITED**

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## 8.0 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).
- 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.
- 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). 2001. Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil.
- CCME (Canadian Council of Ministers of the Environment). 1999a, 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). 1999b. Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment, Winnipeg. Originally published in 1999, updated in 2004.
- 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). 1996c. A Framework for Ecological Risk Assessment: General Guidance. CCME Subcommittee on Environmental Quality Criteria for Contaminated Sites. March, 1996.
- CWS & CWF (Canadian Wildlife Service & Canadian Wildlife Federation). 2005. Hinterland Who's Who. Accessed October, 2005 at <http://www.hww.ca>.
- Earth Tech Canada Inc. 2001. Environmental Site Delineation and Material Inventory.
- Environmental Services Group (ESG) (Royal Military College). 1997. Environmental Assessment of Radio Island, NWT.
- 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).
- Health Canada. 2004a Federal Contaminated Site Risk Assessment in Canada. Part 1: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA). September, 2004.
- Health Canada. 2004b. Federal Contaminated Site Risk Assessment in Canada Part II: Health Canada Toxicological Reference Values (TRVs), September 2004.
- Health Canada. 1994. Canadian Environmental Protection Act. Human Health Risk Assessment for Priority Substances. Ottawa. Cat. No. En40-215/41E. pp 36.
- Health Effects Assessment Summary Tables (HEAST) FY 2005. Update. Risk Assessment Information System (RAIS). Office of Research and Development; Office of Emergency and Remedial Response, U.S. Environmental Protection Agency, Washington, DC.
- Indian and Northern Affairs Canada. 2005. Abandoned Military Site Remediation Protocol. DEW Line Cleanup Criteria (DCC), Appendix B.
- 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). 1996. Rationale for the Development and Application of Generic Soil, Groundwater and Sediment Criteria for use at Contaminated Sites in Ontario.
- OMOE. 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>.
- Public Works and Government Services Canada (PWGSC). 2002. Remedial Action Plan Former Navigational Aid and Weather Station Radio Island, Nunavut.
- Richardson GM. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment. O'Connor Associates Environmental Inc., Ottawa, ON.
- Sample BE, Arenal CA. 1999. Allometric models for interspecies extrapolation of wildlife toxicity data. Bull Environ Contam Toxicol. 1999 Jun; 62(6):653-63.

- 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.
- 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).
- SENES Consultants Limited. 2003. Human Health Screening Level Risk Assessment for Radio Island Former Military Site.
- 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). 2005a. Integrated Risk Information System (IRIS) Database – Zinc. Confirmed current as of December 2005. Available on-line at: <http://www.epa.gov/iris/>
- United States Environmental Protection Agency (US EPA). 2005b. Region 3: Mid-Atlantic Region Hazardous Site Cleanup Division Risk Based Concentration Tables. <http://www.epa.gov/reg3hwmd/risk/rbc1002.pdf> October 25, 2005.
- United States Environmental Protection Agency (US EPA). 2000. Integrated Risk Information System (IRIS) Database – Arsenic. Confirmed current as of December 2005. Available on-line at: <http://www.epa.gov/iris/>
- United States Environmental Protection Agency (US EPA). 1999. Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Peer review draft (FRL-6535-1).
- United States Environmental Protection Agency (US EPA). 1998. Integrated Risk Information System (IRIS) Database – Beryllium. Confirmed current as of December 2005. Available on-line at: <http://www.epa.gov/iris/>
- United States Environmental Protection Agency (US EPA). 1997. Integrated Risk Information System (IRIS) Database – Antimony. Confirmed current as of December 2005. Available on-line at: <http://www.epa.gov/iris/>
- United States Environmental Protection Agency (US EPA). 1997. Integrated Risk Information System (IRIS) Database – Polychlorinated Biphenyls. Confirmed current as of December 2005. Available on-line at: <http://www.epa.gov/iris/>
- United States Environmental Protection Agency (US EPA). 1995. Integrated Risk Information System (IRIS) Database – Nickel. Confirmed current as of December 2005. Available on-line at: <http://www.epa.gov/iris/>

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.





# APPENDIX A

## Site Maps



SCALE: 1 : 3 000 000

DATE: 27/10/05

DRAWN BY: LDP

APPROVED BY:

CLIENT :

TITLE :

**PUBLIC WORKS AND GOVERNMENT SERVICES CANADA**

**HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT**

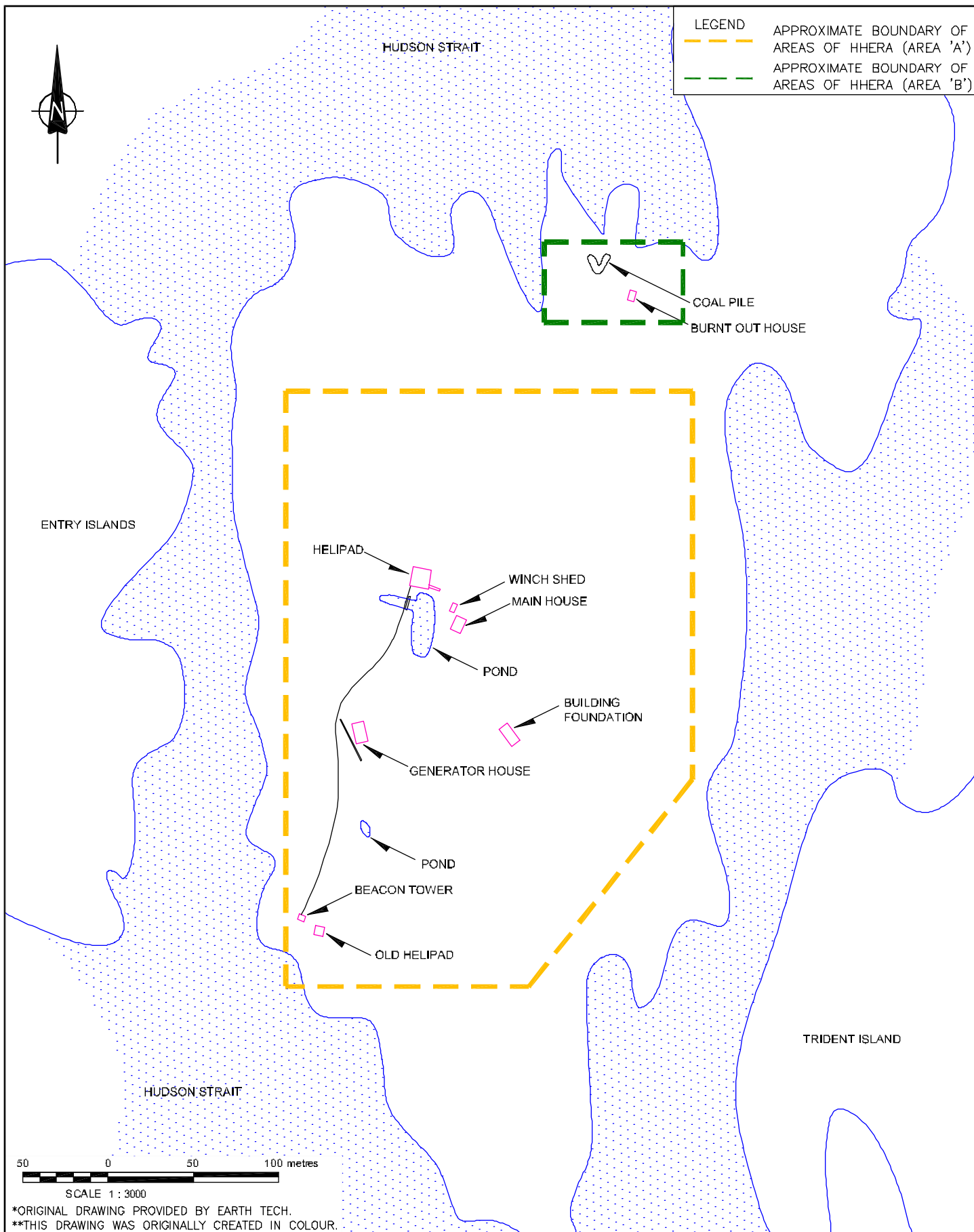
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**FORMER NAVIGATIONAL AID & WEATHER STATION**

**RADIO ISLAND, NUNAVUT**

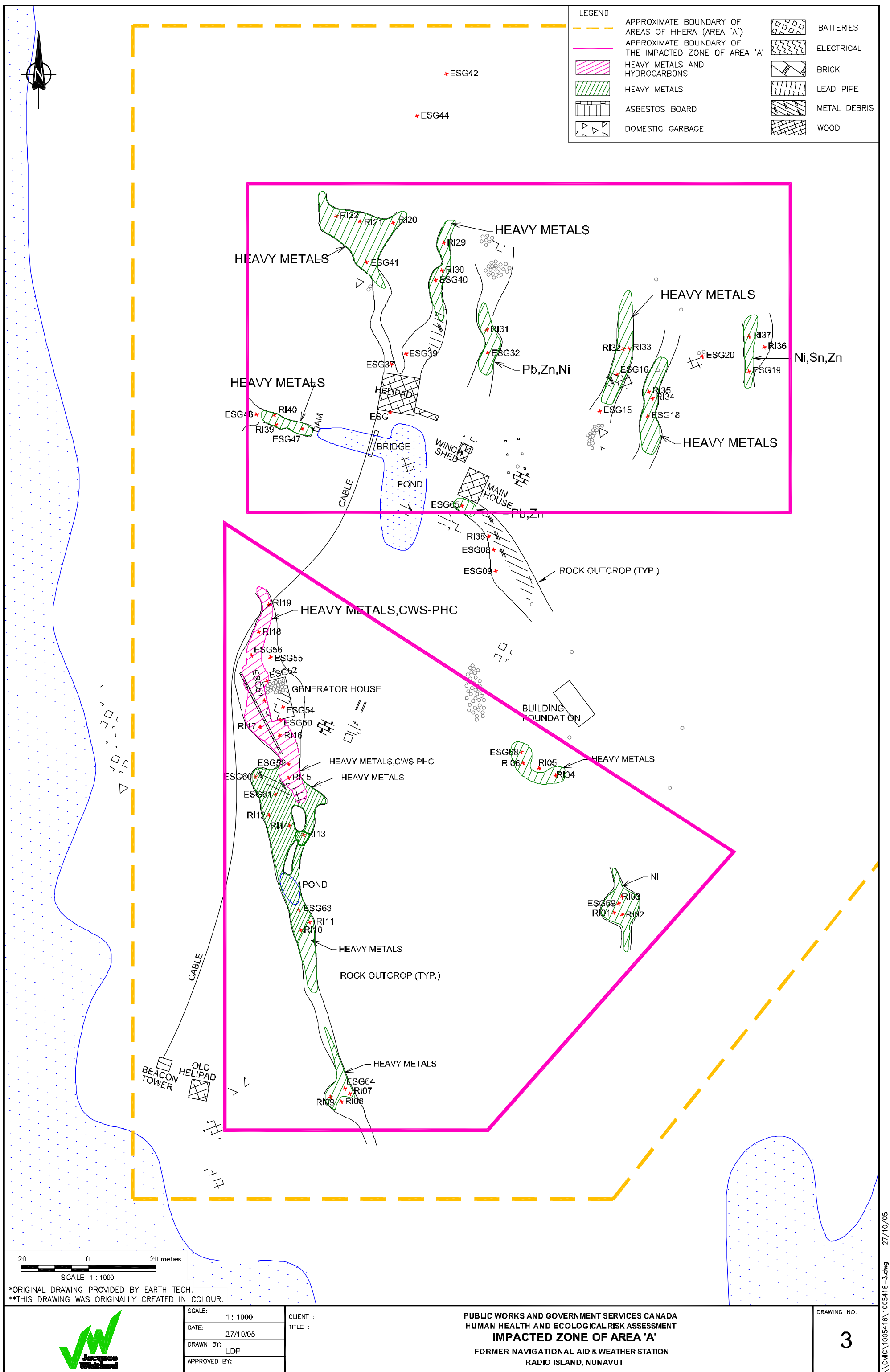
DRAWING NO.

**1**



	<p>SCALE: 1 : 3000</p> <p>DATE: 27/10/05</p> <p>DRAWN BY: LDP</p> <p>APPROVED BY:</p>	<p>CLIENT : PUBLIC WORKS AND GOVERNMENT SERVICES CANADA</p> <p>TITLE : HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT</p> <p><b>SITE OVERVIEW</b></p> <p>FORMER NAVIGATIONAL AID &amp; WEATHER STATION</p> <p>RADIO ISLAND, NUNAVUT</p>	<p>DRAWING NO.</p> <p><b>2</b></p>
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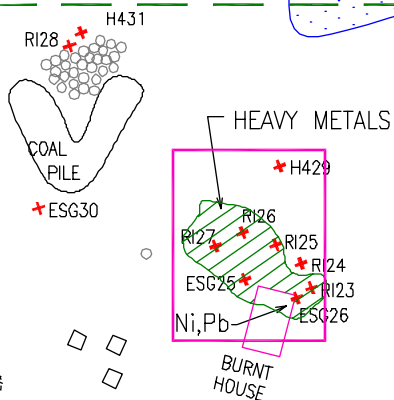
\\CMIC\1005418\1005418-2.dwg 27/10/05





#### LEGEND

	APPROXIMATE BOUNDARY OF AREAS OF HHERA (AREA 'B')		BATTERIES
	APPROXIMATE BOUNDARY OF THE IMPACTED ZONE OF AREA 'B'		ELECTRICAL
	HEAVY METALS AND HYDROCARBONS		BRICK
	HEAVY METALS		LEAD PIPE
	ASBESTOS BOARD		METAL
	DOMESTIC GARBAGE		WOOD



10 5 0 10 20 metres

SCALE 1:750

\*ORIGINAL DRAWING PROVIDED BY EARTH TECH.

\*\*THIS DRAWING WAS ORIGINALLY CREATED IN COLOUR.



SCALE:

1:750

DATE:

27/10/05

DRAWN BY:

LDP

APPROVED BY:

CLIENT :

TITLE :

PUBLIC WORKS AND GOVERNMENT SERVICES CANADA  
HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT  
**IMPACTED ZONE OF AREA 'B'**  
FORMER NAVIGATIONAL AID & WEATHER STATION  
RADIO ISLAND, NUNAVUT

DRAWING NO.

4

# APPENDIX B

## Toxicity Profiles

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# Table of Contents

<b>GLOSSARY AND ACRONYMS .....</b>	<b>vii</b>
<b>1.0 INTRODUCTION.....</b>	<b>1</b>
1.1 Non-Carcinogenic TRVs.....	1
1.2 Carcinogenic TRVs.....	2
1.3 Bioavailability.....	2
<b>2.0 ANTIMONY .....</b>	<b>3</b>
2.1 Assessment of Carcinogenicity .....	3
2.2 Susceptible Populations .....	3
2.3 Selection of Toxicity Values.....	3
2.3.1 Non-Cancer Oral Toxicity Reference Values .....	3
2.3.2 Cancer Oral Toxicity Reference Values.....	3
2.3.3 Non-Cancer Inhalation Toxicity Reference Values .....	4
2.3.4 Cancer Inhalation Toxicity Reference Values .....	4
2.4 Bioavailability.....	4
2.5 Oral Bioavailability.....	4
2.5.1 Inhalation Bioavailability.....	4
2.5.2 Dermal Bioavailability.....	4
2.6 Conclusion.....	4
2.7 References .....	5
<b>3.0 ARSENIC.....</b>	<b>6</b>
3.1 Assessment of Carcinogenicity .....	6
3.2 Susceptible Populations .....	6
3.3 Selection of Toxicity Values.....	6
3.3.1 Oral Non-Cancer Toxicity Reference Values .....	6
3.3.2 Oral Cancer Toxicity Reference Values.....	7
3.3.3 Non-Cancer Inhalation Toxicity Reference Values .....	7
3.3.4 Cancer Inhalation Toxicity Reference Values .....	7
3.4 Bioavailability.....	8
3.4.1 Oral Bioavailability .....	8
3.4.2 Inhalation Bioavailability.....	8
3.4.3 Dermal Bioavailability.....	8
3.5 CONCLUSION.....	9
3.6 References .....	9
<b>4.0 BARIUM.....</b>	<b>11</b>
4.1 Assessment of Carcinogenicity .....	11
4.2 Susceptible Populations .....	11
4.3 Selection of Toxicity Values.....	11
4.3.1 Non-Cancer Oral Toxicity Reference Values .....	11
4.3.2 Cancer Oral Toxicity Reference Values.....	11
4.3.3 Non-Cancer Inhalation Toxicity Reference Values .....	12
4.3.4 Cancer Inhalation Toxicity Reference Values .....	12

4.4	Bioavailability.....	12
4.4.1	Oral Bioavailability .....	12
4.4.2	Inhalation Bioavailability.....	12
4.4.3	Dermal Bioavailability.....	12
4.5	Conclusion.....	12
4.6	References .....	13
<b>5.0</b>	<b>BERYLLIUM .....</b>	<b>14</b>
5.1	Assessment of Carcinogenicity .....	14
5.2	Susceptible Populations .....	14
5.3	Selection of Toxicity Values.....	14
5.3.1	Non-Cancer Oral Toxicity Reference Values .....	14
5.3.2	Cancer Oral Toxicity Reference Values.....	14
5.3.3	Non-Cancer Inhalation Toxicity Reference Values .....	15
5.3.4	Cancer Inhalation Toxicity Reference Values .....	15
5.4	Bioavailability.....	15
5.5	Oral Bioavailability .....	15
5.5.1	Inhalation Bioavailability.....	16
5.5.2	Dermal Bioavailability.....	16
5.6	Conclusion.....	16
5.7	References .....	16
<b>6.0</b>	<b>CADMIUM.....</b>	<b>18</b>
6.1	Assessment of Carcinogenicity .....	18
6.2	Susceptible Populations .....	18
6.3	Selection of Toxicity Values.....	18
6.3.1	Non-Cancer Oral Toxicity Reference Values .....	18
6.3.2	Cancer Oral Toxicity Reference Values.....	19
6.3.3	Non-Cancer Inhalation Reference Toxicity Values .....	19
6.3.4	Cancer Inhalation Reference Toxicity Values .....	19
6.4	Bioavailability.....	19
6.4.1	Oral Bioavailability .....	20
6.4.2	Inhalation Bioavailability.....	20
6.4.3	Dermal Bioavailability.....	20
6.5	Conclusion.....	20
6.6	References .....	21
<b>7.0</b>	<b>COPPER.....</b>	<b>22</b>
7.1	Assessment of Carcinogenicity .....	22
7.2	Susceptible Populations .....	22
7.3	Toxicity Reference Values.....	22
7.3.1	Non-Cancer Oral Toxicity Reference Values .....	22
7.3.2	Cancer Oral Toxicity Reference Values.....	24
7.3.3	Non-Cancer Inhalation Toxicity Reference Values .....	24
7.3.4	Cancer Inhalation Toxicity Reference Values .....	24
7.4	Bioavailability.....	24
7.4.1	Oral Bioavailability .....	24
7.4.2	Inhalation .....	24



7.4.3	Bioavailability .....	25
7.4.4	Dermal Bioavailability.....	25
7.5	Conclusion.....	25
7.6	References .....	25
<b>8.0</b>	<b>LEAD .....</b>	<b>27</b>
8.1	Assessment of Carcinogenicity .....	27
8.2	Susceptible Populations .....	27
8.3	Selection of Toxicity Values.....	27
8.3.1	Non-Cancer Oral Toxicity Reference Values .....	27
8.3.2	Cancer Oral Toxicity Reference Values.....	28
8.3.3	Non-Cancer Inhalation Toxicity Reference Values .....	28
8.3.4	Cancer Inhalation Toxicity Reference Values .....	28
8.4	Bioavailability.....	28
8.4.1	Oral Bioavailability .....	28
8.4.2	Inhalation Bioavailability.....	29
8.4.3	Dermal Bioavailability.....	29
8.5	Conclusion.....	29
8.6	References .....	29
<b>9.0</b>	<b>MERCURY.....</b>	<b>31</b>
9.1	Assessment of Carcinogenicity .....	31
9.2	Susceptible Populations .....	31
9.3	Selection of Toxicity Values.....	31
9.4	Bioavailability.....	32
9.5	References .....	32
<b>10.0</b>	<b>NICKEL.....</b>	<b>33</b>
10.1	Assessment of Carcinogenicity .....	33
10.2	Susceptible Populations .....	33
10.3	Selection of Toxicity Values .....	33
10.3.1	Non-Cancer Oral Toxicity Reference Value.....	33
10.3.2	Cancer Inhalation Toxicity Reference Value.....	33
10.4	Bioavailability.....	34
10.4.1	Oral Bioavailability .....	34
10.4.2	Inhalation Bioavailability.....	34
10.4.3	Dermal Bioavailability.....	34
10.5	References .....	34
<b>11.0</b>	<b>TIN .....</b>	<b>36</b>
11.1	Assessment of Carcinogenicity .....	36
11.2	Susceptible Populations .....	36
11.3	Selection of Toxicity Values .....	36
11.3.1	Non-Cancer Oral Toxicity Reference Values .....	36
11.3.2	Cancer Oral Toxicity Reference Values.....	36
11.3.3	Non-Cancer Inhalation Toxicity Reference Values .....	36
11.3.4	Cancer Inhalation Toxicity Reference Values .....	36
11.4	Bioavailability.....	37

11.4.1	Oral Bioavailability .....	37
11.4.2	Inhalation Bioavailability.....	37
11.4.3	Dermal Bioavailability.....	37
11.5	Conclusion.....	37
11.6	References .....	37
<b>12.0</b>	<b>ZINC.....</b>	<b>38</b>
12.1	Assessment of Carcinogenicity .....	38
12.2	Susceptible Populations .....	38
12.3	Selection of Toxicity Values .....	38
12.3.1	Non-Cancer Oral Toxicity Reference Value.....	38
12.4	Bioavailability.....	39
12.4.1	Oral Bioavailability .....	39
12.4.2	Inhalation Bioavailability.....	39
12.4.3	Dermal Bioavailability.....	39
12.4.4	References.....	39
<b>13.0</b>	<b>PETROLEUM HYDROCARBON CWS FRACTIONS F2 AND F3.....</b>	<b>40</b>
13.1	Assessment of Carcinogenicity .....	41
13.2	Susceptible Populations .....	41
13.3	Selection of Toxicity Reference Values.....	41
13.3.1	Non-Cancer Oral Toxicity Reference Value.....	41
13.3.1.1	Aromatic Fractions >C10-C12 and >C12-C16 .....	41
13.3.1.2	Aromatic Fractions >C16-C21 and >C21-C34 .....	41
13.3.1.3	Aliphatic Fractions >C10-C12 and >C12-C16.....	42
13.3.1.4	Aliphatic Fractions >C16-C21 and >C21-C34.....	42
13.3.2	Cancer Oral Toxicity Reference Value.....	42
13.3.3	Non-Cancer Inhalation Toxicity Reference Value .....	42
13.3.3.1	Aromatic Fractions >C10-C12 and >C12-C16 .....	42
13.3.3.2	Aromatic Fractions >C16-C21 and >C21-C34 .....	42
13.3.3.3	Aliphatic Fractions >C10-C12 and >C12-C16.....	42
13.3.3.4	Aliphatic Fractions >C16-C21 and >C21-C34.....	43
13.3.4	Cancer Inhalation Toxicity Reference Value.....	43
13.4	Bioavailability.....	43
13.4.1	Oral Bioavailability .....	43
13.4.2	Inhalation Bioavailability.....	43
13.4.3	Dermal Bioavailability.....	43
13.5	Conclusion.....	43
13.6	References .....	44
<b>14.0</b>	<b>POLYCHLORINATED BIPHENYLS (PCBS) .....</b>	<b>45</b>
14.1	Assessment of Carcinogenicity .....	45
14.2	Susceptible Populations .....	45
14.3	Selection of Toxicity Reference Values.....	46
14.3.1	Non-Cancer Oral Toxicity Reference Values.....	46
14.3.2	Cancer Oral Toxicity Reference Value.....	47
14.3.3	Non-Cancer Inhalation Toxicity Reference Value .....	47
14.3.4	Cancer Inhalation Toxicity Reference Values .....	47

14.4	Bioavailability.....	47
14.4.1	Oral Bioavailability .....	48
14.4.2	Inhalation Bioavailability.....	48
14.4.3	Dermal Bioavailability.....	48
14.5	Conclusion.....	48
14.6	References .....	49

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## List of Tables

Table 1:	Selected Toxicity Reference Values for Antimony .....	4
Table 2:	Selected Bioavailabilities for Antimony.....	5
Table 3:	Selected Toxicity Reference Values for Arsenic .....	9
Table 4:	Selected Relative Bioavailabilities for Arsenic.....	9
Table 5:	Selected Toxicity Reference Values for Barium .....	12
Table 6:	Selected Bioavailabilities for Barium .....	13
Table 7:	Selected Toxicity Reference Values for Cadmium .....	20
Table 8:	Selected Relative Bioavailabilities for Cadmium .....	21
Table 9:	Selected Toxicity Reference Values for Copper.....	25
Table 10:	Selected Relative Bioavailabilities for Copper.....	25
Table 11:	Selected Toxicity Reference Values for Lead .....	29
Table 12:	Selected Bioavailabilities for Lead.....	29
Table 13:	Selected Toxicity Values for Elemental Mercury .....	32
Table 14:	Selected Toxicity Values for Nickel .....	34
Table 15:	Selected Toxicity Values for Zinc .....	38
Table 16:	Composition of Petroleum Hydrocarbon CWS Fractions F2 and F3.....	41
Table 17:	Selected TRVs for Petroleum Hydrocarbon CWS Fractions F2 and F3.....	43
Table 18:	Selected Bioavailabilities for Petroleum Hydrocarbon CWS Fractions F2 and F3 .....	44
Table 19:	Oral Reference Dose for PCB Mixtures .....	46
Table 20:	Minimal Risk Levels for Oral Exposure to PCBs .....	47
Table 21:	Selected Toxicity Reference Values for PCBs .....	49
Table 22:	Selected Bioavailabilities for PCBs .....	49

## 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.

---

## 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.



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## **2.0 ANTIMONY**

Antimony is a silvery-white metal that is found in the earth's crust. Exposure to antimony at high levels can result in a variety of adverse health effects. Chronic inhalation of high levels of antimony can irritate the eyes and lungs as well as cause heart and lung problems as well as digestive problems. Ingesting large doses of antimony can cause vomiting. Chronic animal studies have reported that ingesting antimony can cause liver damage and blood changes. (ATSDR, 1992)

---

### **2.1 Assessment of Carcinogenicity**

The US EPA's IRIS program has not evaluated the carcinogenicity of antimony. The Agency for Toxic Substances and Disease Registry (ATSDR, 1992) state that no information is available on the carcinogenic potential of antimony. The International Agency for Research on Cancer (IARC, 1989) found that there is insufficient supporting evidence to list antimony trioxide or antimony trisulphide as carcinogenic agents at this time.

---

### **2.2 Susceptible Populations**

Individuals with existing chronic respiratory or cardiovascular disease or problems would probably be at special risk, since antimony probably exacerbates one or both types of health problems. Because antimony is excreted in the urine, individuals with kidney dysfunction may be unusually susceptible (ATSDR, 1992).

---

### **2.3 Selection of Toxicity Values**

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

An oral reference dose (RfD) of 0.0004 mg/kg/day was provided for antimony by the U.S. EPA (1987) based on a chronic study examining ingestion by rats. The main endpoints of concern were a decrease in longevity, a decrease in blood glucose levels and an alteration in cholesterol levels. The U.S. EPA (1987) reported a lowest observable adverse effect limit (LOAEL) of 0.35 mg/kg-day, and applied an uncertainty factor of 1000 (10 for interspecies conversion, 10 to protect sensitive individuals, and 10 because the effect level was a LOAEL and there wasn't a no observable adverse effects level [NOAEL] established) to the LOAEL to derive the RfD.

---

#### **2.3.2 Cancer Oral Toxicity Reference Values**

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

---

### 2.3.3 Non-Cancer Inhalation Toxicity Reference Values

Inhalation toxicity values for antimony 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.

---

### 2.3.4 Cancer Inhalation Toxicity Reference Values

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

---

## 2.4 Bioavailability

The following sections describe the bioavailability of antimony.

---

### 2.5 Oral Bioavailability

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

---

#### 2.5.1 Inhalation Bioavailability

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

---

#### 2.5.2 Dermal Bioavailability

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

---

## 2.6 Conclusion

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

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

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	$4.0 \times 10^{-4}$ mg/kg-day	RfD	US EPA, 1987
Inhalation	NA	NA	NA
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	NA	NA	NA
NA – Not Applicable			

**Table 2: Selected Bioavailabilities for Antimony**

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

---

## 2.7 References

ATSDR (Agency for Toxic Substances and Disease Registry), 1992. Toxicological Profile for Antimony. September 1992.

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

International Agency for Research on Cancer (IARC). 1989. "Antimony Trioxide And Antimony Trisulfide". *Monographs*. Vol. 47, p. 291. World Health Organization.

US EPA (Environmental Protection Agency). 1987. Integrated Risk Information System (IRIS) Database – Antimony. Confirmed current as of December 2004. Available on-line at: <http://www.epa.gov/iris/>

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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  $6.4 \times 10^{-3}$  per  $\mu\text{g}/\text{m}^3$ , of  $4.9 \times 10^{-3}$  per  $\mu\text{g}/\text{m}^3$ , and of  $0.99 \times 10^{-3}$  per  $\mu\text{g}/\text{m}^3$  for the Anaconda, Tacoma and Ronnskar cohorts, respectively. Health Canada reviewed only one follow-up study for the Anaconda cohort. The Health Canada  $\text{TD}_{05}$  is based on only the Anaconda smelter data as being the most conservative. Recently, Health Canada (2003) has recommended an inhalation SF of  $28.0 (\text{mg}/\text{kg}\cdot\text{day})^{-1}$  based on a  $\text{TC}_{05}$  of  $7.8 \mu\text{g}/\text{m}^3$  for arsenic and its inorganic compounds (Health Canada, 1996).

The Health Canada (2003) inhalation SF of  $28.0 (\text{mg}/\text{kg}\cdot\text{day})^{-1}$  is used in this assessment.

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## 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).

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### 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).

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### 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).

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### 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  $33.1 \mu\text{g}/\text{cm}^2\cdot\text{hour}$  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).

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### 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

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### 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 BERYLLIUM

According to the ATSDR (2002), beryllium is a hard, grayish metal naturally found in mineral rocks, coal, soil, and volcanic dust. Beryllium compounds are commercially mined, and the beryllium is purified for use in nuclear weapons and reactors, aircraft and space vehicle structures, instruments, x-ray machines, and mirrors. Beryllium ores are used to make speciality ceramics for electrical and high-technology applications. Beryllium alloys are used in automobiles, computers, sports equipment (golf clubs and bicycle frames), and dental bridges.

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### 5.1 Assessment of Carcinogenicity

The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have determined that beryllium is a human carcinogen. The U.S. EPA has determined that beryllium is a probable human carcinogen.

---

### 5.2 Susceptible Populations

There are no studies on the health effects of children exposed to beryllium. It is likely that the health effects seen in children exposed to beryllium will be similar to the effects seen in adults. We do not know whether children differ from adults in their susceptibility to beryllium (ATSDR, 2002).

It is not known if exposure to beryllium will result in birth defects or other developmental effects in people: the studies on developmental effects in animals are not conclusive.

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### 5.3 Selection of Toxicity Values

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

The oral reference dose (RfD) for beryllium published by the US EPA (1998) is 0.002 mg/kg-d. The US EPA oral RfD is based on a long-term study of dogs fed diets containing beryllium by Morgareidge, *et al* (1976). The oral RfD is based on the development of intestinal lesions. A BMD<sub>10</sub> (the lower 95% confidence limit on the dose from the maximum likelihood estimate [MLE] of a 10% relative change) of 0.46 mg/kg-day (MLE = 1.4 mg/kg-day) was derived for the lesions and used for further quantitation in this assessment in the US EPA's assessment. (U.S. EPA, 1995). An uncertainty factor of 300 was applied: 10 for extrapolation for interspecies differences, 10 for consideration of intraspecies variation, and 3 for database deficiencies. The USEPA has low to medium confidence in this RfD.

An oral RfD of 0.002 mg/kg-day has been adopted in this assessment based on the US EPA's recommended oral RfD.

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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 beryllium.

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### 5.3.3 Non-Cancer Inhalation Toxicity Reference Values

The inhalation reference concentration (RfC) for beryllium published by the US EPA (1998) is  $2\text{E-}2 \mu\text{g}/\text{m}^3$ . The RfC is based on beryllium sensitization and progression to chronic beryllium disease (CBD) identified in the co-principal studies by Kreiss, *et al.* (1996) and Eisenbud, *et al.* (1949). The Kreiss, *et al.* (1996) occupational exposure study identified a lowest observed adverse effects level (LOAEL) for beryllium sensitization in workers exposed to  $0.55 \mu\text{g}/\text{m}^3$  (median of average concentrations). The Eisenbud, *et al.* (1949) study, using relatively insensitive screening methods, suggests a no observed adverse effects level (NOAEL) of  $0.01\text{-}0.1 \mu\text{g}/\text{m}^3$  in community residents living near a beryllium plant. The LOAEL from the Kreiss, *et al.* study was used for the operational derivation of the RfC because the screening method used in the Eisenbud, *et al.* (1949) study was less sensitive than the method used in the Kreiss, *et al.* (1996) study.

Because individuals developing beryllium sensitization and CBD are the most sensitive subpopulation, an uncertainty factor of 1 was used to account for human variability. An uncertainty factor of 1 was also used to adjust for the less-than-chronic exposure duration of the Kreiss, *et al.* (1996) study; use of this uncertainty factor is supported by the evidence that the occurrence of CBD does not appear to be related to exposure duration. A database uncertainty factor of 3 was used to account for the poor quality of exposure monitoring in the co-principal studies and other epidemiology studies that assessed the incidence of beryllium sensitization and CBD among exposed workers and community residents. The US EPA has medium confidence in this RfD.

An RfD of  $4.47\text{E-}6 \text{ mg}/\text{kg-d}$  was then calculated based on the US EPA RfC, by dividing by an adult body weight of 70.7 kg and multiplying by an adult inhalation rate of  $15.8 \text{ m}^3/\text{d}$ , which was then used in this assessment.

---

### 5.3.4 Cancer Inhalation Toxicity Reference Values

The US EPA (1998) has published an inhalation unit risk for beryllium of  $2.4\text{E-}3 (\mu\text{g}/\text{m}^3)^{-1}$ . The unit risk value is based on an occupational exposure epidemiological study by Wagoner, *et al.* (1980) which was used to estimate the lifetime cancer risk from exposure to beryllium oxide based on the estimated lower and upper bounds of exposure estimated by the National Institute of Occupational Safety and Health (NIOSH); namely, 100 and  $1,000 \mu\text{g}/\text{m}^3$ .

An inhalation slope factor of  $10.9 (\text{mg}/\text{kg-d})^{-1}$  was then calculated by multiplying the US EPA inhalation unit risk, by an adult body weight of 70.7 kg and dividing by an adult inhalation rate of  $15.8 \text{ m}^3/\text{d}$ , which was then used in this assessment.

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## 5.4 Bioavailability

The following sections describe the bioavailability of beryllium.

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### 5.5 Oral Bioavailability

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

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## 5.5.1 Inhalation Bioavailability

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

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## 5.5.2 Dermal Bioavailability

Health Canada (2003) recommends a relative dermal absorption factor of 0.03 for beryllium. Therefore, a relative dermal bioavailability of 0.03 was adopted for this assessment.

## 5.6 Conclusion

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

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

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	2.00E-03 mg/kg-day	RfD	US EPA, 1998
Inhalation	4.47E-06 mg/kg-day	RfC	US EPA, 1998
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	10.7 (mg/kg-day) <sup>-1</sup>	Slope Factor	US EPA, 1998
NA – Not Applicable			

**Table 6: Selected Bioavailabilities for Beryllium**

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

---

## 5.7 References

ATSDR (Agency for Toxic Substances and Disease Registry), 2002. Toxicological Profile for Beryllium. September 2002. Available on-line at: <http://www.atsdr.cdc.gov/toxpro2.html>.

Eisenbud, M; Wanta, RC; Dustan, C; *et al.*, 1949. Non-occupational berylliosis. J Ind Hyg Toxicol 31:282-294. Cited In: US EPA, 1998.

Kreiss, K; Mroz, MM; Newman, LS; *et al.*, 1996. Machining risk of beryllium disease and sensitization with median exposures below 2 MU-G/M(3). Am J Ind Med 30(1):16-25. Cited In: US EPA, 1998.

Morgareidge, K; Cox, GE; Gallo, MA. 1976. Chronic feeding studies with beryllium in dogs. Food and Drug Research Laboratories, Inc. Submitted to the Aluminum Company of America, Alcan

Research & Development, Ltd., Kawecki-Berylco Industries, Inc., and Brush-Wellman, Inc. Cited In: US EPA, 1998.

US EPA (United States Environmental Protection Agency), 1998. Integrated Risk Information System (IRIS) Database. Beryllium and compounds (inorganic). Confirmed current as of December 2004. Available on-line at: <http://www.epa.gov/iris/>

Wagoner, JK; Infante, PF; Bayliss, DL. (1980) Beryllium: an etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease, and heart disease among industrially exposed workers. Environ Res 21:15-34. Cited In: US EPA, 1998.

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## **6.0 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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### **6.1 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.

---

### **6.2 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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### **6.3 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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#### **6.3.1 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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### 6.3.2 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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### 6.3.3 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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### 6.3.4 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 µg 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.

---

## 6.4 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).

---

#### 6.4.1 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.

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#### 6.4.2 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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#### 6.4.3 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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### 6.5 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	$4.29E+01$ (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

---

## 6.6 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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## **7.0 COPPER**

Copper is widely distributed in nature and is an essential element. Copper deficiency is characterized by hypochromic, microcytic anemia which is a result of defect in hemoglobin synthesis. Many oxidative enzymes (e.g., catalase, peroxidase, cytochrome oxides) require copper. The importance of copper in human nutrition has been reviewed in detail (IPCS, 1998; IOM, 2001).

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### **7.1 Assessment of Carcinogenicity**

The toxicity of copper has been the subject of several comprehensive reviews (US EPA, 1991; IPCS, 1998; ATSDR, 2002). Copper is classified as Group D – not classifiable as a human carcinogen due to a lack of human data, inadequate animal data from assays of copper compounds, and equivocal mutagenicity data (US EPA, 1991). Health Canada also does not consider copper to be carcinogenic to humans (CCME, 1999).

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### **7.2 Susceptible Populations**

Infants and children under 1 year old are unusually susceptible to copper toxicity because they have not developed the homeostatic mechanism to remove copper from the body (ATSDR, 2002). Wilson's Disease is a genetic disorder associated with impaired transport of copper from the liver to the bile, thereby resulting in increased copper concentrations in the liver as they are not able to maintain homeostasis (ATSDR, 2002). Another genetic condition which increases the susceptibility to copper toxicity is a deficiency in the enzyme glucose-6-phosphate dehydrogenase (ATSDR, 2002). Individuals with liver disease are also susceptible to copper toxicity because of the critical role the liver plays in eliminating copper from the body (ATSDR, 2002).

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### **7.3 Toxicity Reference Values**

As copper is considered an essential element for humans, there are two types of toxicity values that are considered: (a) the minimal daily intake so that a person will not suffer from copper deficiency and (b) the maximal permissible daily intake so that a person will not suffer from copper toxicity. Major non-carcinogenic effects observed in humans after exposure to excessive amounts of copper include diarrhea, vomiting, hypotension, skin irritation, lung disease, kidney damage and liver damage.

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

The Recommended Dietary Allowance (RDA) for US adults is 0.900 mg/day or about 0.13 mg/kg-day (IOM, 2001). This RDA is a combination of indicators, including plasma copper and ceruloplasmin concentrations, erythrocyte superoxide dismutase (ESOD) activity and platelet copper concentration in controlled human depletion/repletion studies. The US Reference Daily Intake (RDI) (a term which replaces RDA) for copper is 2.0 mg/day or about 0.030 mg/kg-day for adults (US FDA, 1999).

The tolerable upper intake level for US adults is 10 mg/day or about 0.140 mg/kg-day, and is based on protection from liver damage (IOM, 2001). A reference dose (RfD) for elemental copper has not been developed by the US EPA (1991).

Health Canada has developed lower and upper bound tolerable daily intake (TDI) values for copper of 0.050 mg/kg-day and 0.500 mg/kg-day respectively. The lower limit is comparable to that set by the WHO for a young child. The upper limit set by Health Canada is appreciably higher (more than 3-fold) than the tolerable limit set in the US. Health Canada and Environment Canada have also developed TDIs of 0.100 mg/kg-day and 0.030 mg/kg-day for toddlers and adults, respectively as part of the CCME soil quality guideline development process (CCME, 1999).

ATSDR (2002) has developed a minimal risk level (MRL) for acute duration oral exposure to copper. The MRL of 20 µg/kg-day is based on a no observable adverse effects level (NOAEL) of 27.2 µg/kg-day copper administered as copper sulphate in drinking water (Pizzaro *et al.* 1999). The total copper exposure was estimated at 53.8 µg/kg-day by adding estimated daily dietary intake of copper. An uncertainty factor of 3 was used for human variability. A statistically significant difference was seen between the reported NOAEL and a lowest observable adverse effects level (LOAEL) of 73.1 µg/kg-day (total intake of about 100 µg/kg-day) for gastrointestinal effects in women. The Pizzaro *et al.* study was also reviewed by the Food and Nutrition Board (IOM, 2001).

The Food and Nutrition Board (IOM, 2001) of the US National Academy of Sciences Institute of Medicine, in a joint activity with Health Canada, has published a series of tolerable upper limits of copper for various life stages. The tolerable upper limit is the highest level of daily intake of a nutrient (over a lifetime) likely not to result in an adverse health effect to almost all individuals. The UL for copper were based on a NOAEL of 10 mg/day of copper. A double blind study, conducted over 12 weeks, involved administering seven adults 10 mg of copper gluconate capsules daily. The resulting liver function tests were normal in all cases.

The World Health Organization (WHO, 1996) proposed 12 mg copper/day as a safe upper level of intake for a 65 kg adult.

The Food and Nutrition Board (IOM, 2001) also recommended a “safe range” for oral intake of copper between 10 and 130 µg/kg-day for chronic ingestion by a 76 kg adult. Age specific upper limits (UL) are recommended based on extrapolation by body weight:

- Child 1-3 years, UL of 1,000 µg/day
- Child 4-8 years, UL of 3,000 µg/day
- Child 9-13 years, UL of 5,000 µg/day
- Adolescents 14-18 years, UL of 8,000 µg/day
- Adult 19 years and older, UL of 10,000 µg/day

Tolerable upper intake levels are considered to be the highest level of daily intake of a nutrient likely to not pose an adverse health effect to almost all individuals. These were developed through a risk approach, using a NOAEL for copper of 10 mg/day.

The Health Canada and Environment Canada TDIs for toddlers and adults of  $1.0 \times 10^{-1}$  and  $3.0 \times 10^{-2}$ , respectively have been selected for this assessment.

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### **7.3.2 Cancer Oral Toxicity Reference Values**

The carcinogenic potential of copper has not been adequately assessed, therefore an oral slope factor or unit risk is not available.

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### **7.3.3 Non-Cancer Inhalation Toxicity Reference Values**

The California Air Resources Board approved a risk assessment health value of 0.0024 mg/m<sup>3</sup> (CARB, 1998, last reviewed 01/1992) for chronic inhalation of copper based on respiratory effects. This value was derived from the ACGIH (1992) threshold limit value (TLV) of 1 mg/m<sup>3</sup> by dividing by a factor 420. This factor is made up of a conversion from a 40 hour work week (4.2), a factor to protect sensitive individuals (10) and a factor to account for the deficiency of using a TLV rather than a NOAEL (10) (CAPCOA, 1993).

Due to insufficient data, a non-cancer TRV for inhalation of copper has not been selected for this assessment.

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### **7.3.4 Cancer Inhalation Toxicity Reference Values**

The carcinogenic potential of copper has not been adequately assessed, therefore an inhalation slope factor or unit risk is not available.

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## **7.4 Bioavailability**

The following sections describe the oral, inhalation and dermal bioavailabilities of copper.

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### **7.4.1 Oral Bioavailability**

Oral bioavailability of copper in humans depends on dietary intake of copper, as this is regulated by homeostasis mechanisms in the body. A range of 25 to 40% bioavailability from the diet was estimated (CCME, 1999). Venugopal and Luckey (1978) report an oral absorption of 30% for dietary intake.

In the gastrointestinal tract, all copper is present as the cupric ion or bound to amino acids and is absorbed as such (ATSDR, 2002). The absorption of ingested dietary copper (bioavailability) in humans is subject to strict homeostatic control and varies widely according to the daily oral intake (IPCS, 1998). It has been reported (IOM, 2001) that the bioavailability of copper is over 50% at daily intakes less than 1 mg/day and less than 20% at daily intakes above 5 mg/day. About 35% of a 2 mg/day intake is absorbed (near the upper range of daily intakes of copper in North America).

The selected toxicity reference value is based on copper gluconate capsules. Insufficient information is available to determine whether the oral bioavailability of copper in this form would be greater than copper from dietary intakes, so no adjustment for this will be made in this assessment.

For this study, the relative oral bioavailability of copper in soil was assumed to be 1.0.

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### **7.4.2 Inhalation**

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### 7.4.3 Bioavailability

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

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### 7.4.4 Dermal Bioavailability

No information was identified regarding dermal absorption of copper. Health Canada (2003) recommends a relative dermal absorption factor of 0.1 for copper. The Health Canada relative dermal bioavailability has been adopted for this assessment.

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## 7.5 Conclusion

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

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

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion – toddler	$1.0 \times 10^{-1}$ mg/kg-day	RfD	CCME, 1999
Ingestion – adult	$3.0 \times 10^{-2}$ mg/kg-day	RfD	CCME, 1999
Inhalation	NA	NA	NA
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	NA	NA	NA
NA – Not Applicable			

**Table 10: Selected Relative Bioavailabilities for Copper**

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

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## 7.6 References

ACGIH (American Conference of Governmental and Industrial Hygienists), 1992. Threshold Limit Values. Cincinnati, OH.

ATSDR (Agency for Toxic Substances and Disease Registry), 2002. Toxicological Profile for Copper. September 2002. Available on-line at: <http://www.atsdr.cdc.gov/toxpro2.html>.

CAPCOA (California Air Pollution Control Officers Association), 1993. *Revised 1992 Risk Assessment Guidelines*. Air Toxics “Hot spots” Program, October.



- CARB (California Air Resources Board), 1998. "List of Toxic Air Contaminants – Compound Summary Table." <http://www.arb.ca.gov/toxics/tac/tac.htm>.
- CCME (Canadian Council of Ministers of the Environment), 1999. Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health: Copper. Winnipeg, MN, Canada.
- Health Canada, 2003. Federal Contaminated Site Risk Assessment in Canada, Part I: Guidance on Human Health Screening Level Risk Assessment (SLRA). October 3, 2003.
- IOM (Institute of Medicine), 2001. 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.
- IPCS (International Programme on Chemical Safety), 1998. Copper. Geneva: International Programme on Chemical Safety. World Health Organization Environmental Health Criteria 200. Available on-line at: <http://www.inchem.org/documents/ehc/ehc/ehc200.htm>.
- Pizzaro F, Olivares M and Uauy R, 1999. Acute gastrointestinal effects of graded levels of copper in drinking water. *Environmental Health Perspectives* 107(2): 117-121. Cited in: ATSDR, 2002.
- US EPA (United States Environmental Protection Agency) Region III, 1995. Preliminary Remediation Goals Database.
- US EPA (United States Environmental Protection Agency), 1991. Integrated Risk Information System (IRIS) Database: Copper. Confirmed current as of December 2004. Available on-line at: <http://www.epa.gov/iris/>
- US FDA (United States Food and Drug Administration), 1999. Center for Food Safety and Applied Nutrition. A Food Labeling Guide. September, 1994 (Editorial revisions June, 1999).
- WHO (World Health Organization), 1996. "Copper:" In: *Trace Elements in Human Nutrition and Health*. Geneva, pp. 123-143.
- Venugopal B and Luckey TD, 1978. Metal Toxicity in Mammals – 2. Chemical Toxicity of Metals and Metalloids. Plenum Press. pp. 104-112.



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## 8.0 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).

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### 8.1 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.

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### 8.2 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.

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### 8.3 Selection of Toxicity Values

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#### 8.3.1 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.

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### 8.3.2 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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### 8.3.3 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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### 8.3.4 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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## 8.4 Bioavailability

The following section describes the bioavailabilities of lead.

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### 8.4.1 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%.

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## 8.4.2 Inhalation Bioavailability

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

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## 8.4.3 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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## 8.5 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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## 8.6 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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## 9.0 MERCURY

The US EPA (2003) provides toxicity information for three forms of mercury: elemental mercury ( $\text{Hg}^0$ ), inorganic mercury compounds (primarily mercuric chloride), and organic mercury compounds (primarily methyl mercury,  $\text{MeHg}$ ). All forms of mercury are quite toxic, and each form exhibits different health effects.

Elemental mercury is one of the most frequently emitted forms to air by anthropogenic emissions (OECD, 1993). Approximately 80% of mercury released from human activities is elemental mercury released to the air (ASTDR, 1999). Elemental mercury will vaporize at room temperatures (ASTDR, 1999).

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### 9.1 Assessment of Carcinogenicity

Several studies have been carried out regarding elemental mercury and cancer in humans. These studies are inconclusive due to lack of exposure data and confounding factors (ASTDR, 1999). The US Environmental Protection Agency (EPA) has classified elemental mercury as Group D – not classifiable as to human carcinogenicity, based on inadequate human and animal data (US EPA, 1995).

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### 9.2 Susceptible Populations

The occurrence of acrodynia (or pink disease) is the most widely recognized form of hypersensitivity to mercury; however, the physiological basis for this sensitivity is unknown (ASTDR, 1999). Other known susceptible populations to the toxic effects of mercury include unborn children, newborns, and individuals with diseases of the liver, kidneys, lungs and nerves. Individuals with a dietary insufficiency of zinc, glutathione, antioxidants, or selenium, or those who are malnourished may be more susceptible to mercury poisoning because of the diminished capacity of these substances to protect against mercury toxicity (ASTDR, 1999).

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### 9.3 Selection of Toxicity Values

The central nervous system (CNS) is the major target organ for elemental toxicity in humans, with effects including increased excitability, irritability, excessive shyness, insomnia, severe salivation, gingivitis, and tremors (ASTDR, 1999). Chronic exposure to elemental mercury also affects the kidneys in humans (ASTDR, 1999). The US EPA (1995) has not established a RfD for elemental mercury. However, because of the low bioavailability similar to that of mercuric sulfide, the oral chronic RfD for mercuric sulfide (0.003 mg/kg/day) was used to estimate human health risks for oral exposure from elemental mercury (ORNL, 2003).

The RfC for elemental mercury is 0.0003 mg/m<sup>3</sup> based on CNS effects in humans (US EPA, 1995). The inhalation reference dose of 6.6E-05 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. For the purposes of this assessment, the RfC was used to estimate human health risks for inhalation exposures from elemental mercury.

**Table 13: Selected Toxicity Values for Elemental Mercury**

Route of Exposure	TRV	Toxicological Basis	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	3.0E-03 mg/kg/day	Unknown	ORNL
Inhalation	6.6E-05 mg/kg/day	CNS	US EPA
<b>Cancer Effects</b>			
Ingestion	N/A	N/A	N/A
Inhalation	N/A	N/A	N/A
N/A – not applicable			

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## 9.4 Bioavailability

Few studies in humans were located regarding the absorption of ingested elemental mercury, although reported of ingestion of substantial amounts of elemental mercury indicate that absorption is negligible (ASTDR, 1999). The bioavailability associated with oral ingestion was estimated to be 0.01% (ORNL, 2003). An oral bioavailability factor of 1.0 was used in this assessment.

There are limited quantitative data on the absorption of elemental mercury vapour by humans after inhalation exposure; however, it is reported to be substantial, with at least one study indicating up to 80% of inhaled elemental mercury vapour being retained in human tissues (ASTDR, 1999). An inhalation bioavailability factor of 1.0 was used in this assessment.

Dermal absorption of elemental mercury vapour is considered to be low compared to inhalation exposure, with one estimate at 2.6% of the amount absorbed by the lung (ASTDR, 1999). A dermal bioavailability factor of 0.021 (2.6% of 0.80) was used in this assessment.

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## 9.5 References

ASTDR (Agency for Toxic Substances and Disease Registry). 1999. Toxicological Profile for Mercury. Public Health Service, US Department of Health and Human Services, Atlanta, GA.

OECD (Organization for Economic Cooperation and Development). 1993. Co-operative risk production activities for certain dangerous chemicals: Mercury. Draft Status Report. As cited in: Health Canada (1996).

ORNL. 2003. Oak Ridge National Laboratory. Risk Assessment Program. Risk Assessment Information System. Toxicity and Chemical-specific Factors Data Base [http://risk/lcd.ornl.gov/cgi-bin/tox/TOX\\_9801](http://risk/lcd.ornl.gov/cgi-bin/tox/TOX_9801)

Health Canada. 1996. Canadian Soil Quality Guidelines for Contaminated Sites – Human Health Effects: Inorganic Mercury. Final Report, March 1996. Prepared by Health Canada for The National Contaminated Sites Remediation Program.

US EPA (Environmental Protection Agency). 1995. Integrated Risk Information System (IRIS) Database – Mercury, elemental. Confirmed current as of May 2003. Available on-line at: <http://www.epa.gov/iris/>



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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  $2.4\text{E-}1 \text{ (mg/m}^3\text{)}^{-1}$ . The inhalation slope factor of  $1.1 \text{ (mg/kg/day)}^{-1}$  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  $15.33 \text{ m}^3\text{/day}$ .

**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

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## 10.4 Bioavailability

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### 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.

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### 10.4.2 Inhalation Bioavailability

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

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### 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).

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## 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 TIN**

Tin is a silvery-white metal that is found in the earth's crust. Exposure to high levels of tin can result in stomach aches, anemia as well as liver and kidney problems. It can also result in breathing problems, eye irritations as well as affect the nervous system. (ATSDR, 1992)

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### **11.1 Assessment of Carcinogenicity**

The US EPA's IRIS program has not evaluated the carcinogenicity of tin. The Agency for Toxic Substances and Disease Registry (ATSDR, 1992) state that there is no conclusive information available on the carcinogenic potential of tin.

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### **11.2 Susceptible Populations**

There are no specific populations that have been identified that are unusually susceptible to either inorganic tin or organotin compounds with respect to health effects (ATSDR, 1992).

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### **11.3 Selection of Toxicity Values**

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

An oral reference dose (RfD) of 0.6 mg/kg/day was provided for tin in the EPA's Health Effects Summary Tables (ORNL, 2004). The RfD was derived by the U.S. EPA based on a chronic study examining ingestion by rats. The main endpoint of concern was lesions appearing on the liver. The U.S. EPA reported a no observable adverse effects level (NOAEL) of 600 mg/kg-day, and applied an uncertainty factor of 100 to derive the RfD.

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#### **11.3.2 Cancer Oral Toxicity Reference Values**

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

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#### **11.3.3 Non-Cancer Inhalation Toxicity Reference Values**

Inhalation toxicity values for tin 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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#### **11.3.4 Cancer Inhalation Toxicity Reference Values**

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

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## 11.4 Bioavailability

The following sections describe the bioavailability of tin.

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### 11.4.1 Oral Bioavailability

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

---

### 11.4.2 Inhalation Bioavailability

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

---

### 11.4.3 Dermal Bioavailability

The relative dermal absorption factor for tin has been conservatively assumed to be 0.1.

---

## 11.5 Conclusion

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

**Table 20: Selected Toxicity Reference Values for Tin**

Route of Exposure	TRV	TRV Type	Source Agency
<b>Non-Cancer Effects</b>			
Ingestion	$6.0 \times 10^{-1}$ mg/kg-day	RfD	ORNL, 2004
Inhalation	NA	NA	NA
<b>Cancer Effects</b>			
Ingestion	NA	NA	NA
Inhalation	NA	NA	NA

NA – Not Applicable

**Table 21: Selected Bioavailabilities for Tin**

Route of Exposure	Relative Bioavailability	Reference
Ingestion	1.0	Assumed
Inhalation	1.0	Assumed
Dermal	0.1	Assumed

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## 11.6 References

ATSDR (Agency for Toxic Substances and Disease Registry), 1992. Toxicological Profile for Tin. September 1992.

ORNL (Oak Ridge National Laboratory). 2004. Risk Assessment Program. Risk Assessment Information System. Toxicity and Chemical-specific Factors Data Base Search.

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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).

---

### 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 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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### 13.1 Assessment of Carcinogenicity

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

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### 13.2 Susceptible Populations

There is no information readily available on susceptible populations.

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### 13.3 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 16: 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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#### 13.3.1 Non-Cancer Oral Toxicity Reference Value

##### 13.3.1.1 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.

##### 13.3.1.2 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.

#### **13.3.1.3 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.

#### **13.3.1.4 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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### **13.3.2 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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### **13.3.3 Non-Cancer Inhalation Toxicity Reference Value**

#### **13.3.3.1 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).

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

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

#### **13.3.3.3 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).



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

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

### 13.3.4 Cancer Inhalation Toxicity Reference Value

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

## 13.4 Bioavailability

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

### 13.4.1 Oral Bioavailability

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

### 13.4.2 Inhalation Bioavailability

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

### 13.4.3 Dermal Bioavailability

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

## 13.5 Conclusion

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

**Table 17: 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 17: 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 18: 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

## 13.6 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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## 14.0 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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### 14.1 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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### 14.2 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).

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## 14.3 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).

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### 14.3.1 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 19: 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 20: 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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### 14.3.2 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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### 14.3.3 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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### 14.3.4 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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## 14.4 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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#### 14.4.1 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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#### 14.4.2 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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#### 14.4.3 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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### 14.5 Conclusion

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

**Table 21: 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 22: Selected Bioavailabilities for PCBs**

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

## 14.6 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 putorius 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.



# APPENDIX C

## HHRA Model Assumptions and Equations

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

Receptor characteristics were selected based on site-specific assumptions for a worker scenario at the site as well as information from other sources. These include CCME (1996), Richardson (1997) and Health Canada (2004). The relevant receptor was an on-site adult.

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

**Table C-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	Selected following consultation with INAC
ET	Exposure Time (hr/d)	24	24	Conservative site-specific assumption
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 C-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> )	1713	3009	5260	8283	9487	8650	Richardson, 1997
IR <sub>game</sub> (mg/d)	0	85	125	175	270	233.43	Health Canada (2004)

**Table C-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								
Antimony	0.0004	--	--	--	0.2	1	1	0.1
Arsenic	0.0003	--	2.8	28	0.2	1	1	0.03
Barium	0.016	--	--	--	0.2	1	1	0.1
Beryllium	0.002	0.0000487	--	9.8	0.2	1	1	0.03
Cadmium	0.0008	--	--	40.2	0.2	1	1	0.14
Copper	0.03	--	--	--	0.2	1	1	0.1
Lead	0.0036	--	--	--	0.2	1	1	0.006
Mercury	0.0003	--	--	--	0.2	1	1	0.05
Nickel	0.02	--	--	2.92	0.2	1	1	0.35
Tin	0.6	--	--	--	0.2	1	1	0.1
Zinc	0.3	--	--	--	0.2	1	1	0.02
TPH - CCME CWS								
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								
Organics								
PCBs – Total	0.001	--	2.0	0.4	0.2	1	1	0.1

-- not available:

**Table C-4 Dermal Permeability Coefficient (Kp) Values**

Compound	Kp (cm/hr)	Reference
<b>Inorganics</b>		
Antimony	1.0E-03	US EPA (2004)
Arsenic	1.0E-03	US EPA (2004)
Barium	1.0E-03	US EPA (2004)
Beryllium	1.0E-03	US EPA (2004)
Cadmium	1.0E-03	US EPA (2004)
Copper	1.0E-03	US EPA (2004)
Lead	3.42E-04	US EPA (2004)
Mercury	1.0E-03	US EPA (2004)
Nickel	2.0E-04	US EPA (2004)
Tin	1.0E-03	US EPA (2004)
Zinc	6.0E-04	US EPA (2004)

**Table C-5 CoPC Concentrations Used in HHRA**

Compound	C <sub>soil</sub> Site A (mg/kg)	C <sub>soil</sub> Site B (mg/kg)	C <sub>wildgame</sub> (mg/kg)	C <sub>water</sub> (mg/L)
<b>Inorganics</b>				
Antimony	4.69E+00	--	4.03E-04	--
Arsenic	3.96E+00	--	7.75E-03	5.00E-4
Barium	2.03E+02	--	1.60E-01	--
Beryllium	4.00E-01	0.7	7.16E-04	--
Cadmium	1.82E+00	1.95	2.78E-01	5.00E-4
Copper	1.25E+02	--	4.95E+00	1.13E-2
Lead	6.73E+02	124.36	6.15E+00	5.00E-3
Mercury	2.60E+00	--	2.80E-04	--
Nickel	5.62E+01	--	1.63E+00	5.00E-3
Tin	3.10E+01	--	2.74E+00	--
Zinc	8.87E+02	--	4.62E+01	8.50E-2
<b>TPH – CCME CWS</b>				
Aliph>C10-C12 -F2	5.28E+03	--	7.54E+01	--
Aliph>C12-C16 -F2	6.46E+03	--	1.07E+03	--
Arom>C10-C12 -F2	1.32E+03	--	3.72E-01	--
Arom>C12-C16 -F2	1.61E+03	--	8.29E-01	--
F2 - Total	1.47E+04	--		--
Aliph>C16-C21-F3	1.47E+04	--	2.24E+03	--
Aliph>C21-C34 -F3	6.29E+03	--	9.61E+01	--
Arom>C16-C21 -F3	3.67E+03	--	3.90E+00	--
Arom>C21-C34 -F3	1.57E+03	--	1.16E+01	--
F3 - Total	2.60E+04	--	2.24E+03	--
<b>Organics</b>				
PCBs – Total	2.60E-02			4.40E-04
-- = Parameter not evaluated for this pathway				

## C-2.0 EQUATIONS FOR THE EXPOSURE ASSESSMENT

Exposure of human receptors to arsenic at the site may occur via inadvertent ingestion, dermal contact and inhalation of dust in air. Receptor characteristics and model parameters for these pathways are provided in Section E-1 of this Appendix. The potential for adverse health effects of arsenic increases with increasing exposure. Exposure from inadvertent ingestion, dermal contact and inhalation of dust in air will be considered in the QRA.

## C-3.0 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.

### C-3.1 Inhalation

#### C-3.1.1 Inhalation of Resuspended Soil/Dust – Summer - Outdoor

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

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>
$\text{FR}_{\text{soilo}}$ = 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

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

Where:

**Units**

$\text{CDI}_{\text{INHS/DSO}}$  = 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

$C_{\text{soil}}$  = concentration of chemical in soil mg/kg

## C-3.2 Oral/Dermal Exposure

### C-3.2.1 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:

**Units**

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

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

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

$\text{ET}$  = exposure time hours/day

$\text{EF}$  = exposure frequency days/year

$\text{ED}$  = exposure duration years

$\text{CF}$  = conversion factor kg/mg

$\text{BW}$  = 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

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

Where:

**Units**

$\text{CDI}_{\text{SDINGSO}}$  = chronic daily intake from ingestion of soil/dust – summer outdoor mg/kg-day

$\text{LADD}_{\text{SDINGSO}}$  = lifetime average daily dose from ingestion of soil/dust – summer outdoor mg/kg-day

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

$C_{\text{soil}}$  = concentration of chemical in soil mg/kg

### C-3.2.2 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:

**Units**

$\text{Intake}_{\text{SDERMSO}}$	= 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:

**Units**

$\text{CDI}_{\text{SDERMSO}}$	= 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

### C-3.2.3 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:

**Units**

$\text{Intake}_{\text{dermwater}}$	= 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:

**Units**

$\text{CDI}_{\text{DERMWATER}}$	= 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:

**Units**

$\text{DA}_{\text{event}}$	= 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



## C-3.3 Food Ingestion

### C-3.3.1 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

### C-3.3.2 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:

**Units**

Intake <sub>DWATER</sub>	= daily intake from the ingestion of drinking water	mg/kg-day
C <sub>dwater</sub>	= concentration in drinking water	mg/L
IR <sub>dwater</sub>	= ingestion rate of drinking water	L/day
F <sub>dwater</sub>	= fraction of drinking water consumed from site	unitless
AF <sub>oral</sub>	= oral absorption factor	unitless
EF	= exposure frequency	days/year
ED	= exposure duration	years
BW	= body weight of receptor	kg
AT <sub>c</sub>	= averaging time carcinogen = (365 days/year) x (75 years)	days
AT <sub>nc</sub>	= averaging time non-carcinogen = (365 days/year) x (exposure duration)	days

$$\text{CDI}_{\text{AHARE}} = \text{Intake}_{\text{AHARE}} \times C_{\text{AHARE}}$$

Where:

**Units**

CDI <sub>AHARE</sub>	= chronic daily intake from ingestion of arctic hare	mg/kg-day
Intake <sub>AHARE</sub>	= daily intake from ingestion of arctic hare	kg/kg-day
C <sub>ahare</sub>	= concentration of chemical in arctic hare	mg/kg

### C-3.4 Risk Characterization

After the various intakes are derived, the final step is the 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 IELCRs and HQs for specific receptors.

#### C-3.4.1 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 that chemical (US EPA, 1998a). Since carcinogenic risk estimates are over a lifetime of exposure, a composite receptor comprising five separate lifestages (infant, toddler, child, teen, adult) was used to evaluate carcinogenic intakes. A lifetime average daily dose was derived for each exposure 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

### C-3.4.2 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 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:

		<b><u>Units</u></b>
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:

		<b><u>Units</u></b>
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:

		<b><u>Units</u></b>
Total HI	= total hazard index from multiple exposure pathways	unitless
HI	= hazard index for a specific exposure pathway	unitless

## **C-4.0 REFERENCES**

- CCME. 1996. A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines. Canadian Council of Ministers of the Environment.
- 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 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 D

## Species Inventory

# Intake Parameters - Rock Ptarmigan (*Lagopus mutus*)

Body Weight <sup>a</sup>	4.88E+02	grams
	4.88E-01	kilograms
Food Intake Rate <sup>b</sup>	7.67E+01	g dry-wt/day
	7.67E-02	kg dry-wt/day
	1.87E-01	kg wet-wt/day
Water Intake Rate <sup>c</sup>		
	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*)

Body Weight <sup>a</sup>	2.05E+03	grams
	2.05E+00	kilograms
Food Intake Rate <sup>b</sup>	9.28E+01	g dry-wt/day
	9.28E-02	kg dry-wt/day
	2.89E-01	kg wet-wt/day
Water Intake Rate <sup>c</sup>	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)



# 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 - 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 - Collared Lemming (*Dicrostonyx torquatus*)**

<b>Body Weight<sup>a</sup></b>	8.50E+01	grams
	8.50E-02	kilograms
<b>Food Intake Rate<sup>b</sup></b>	7.61E+00	g dry-wt/day
	7.61E-03	kg dry-wt/day
	2.02E-02	kg wet-wt/day
<b>Water Intake Rate<sup>c</sup></b>	1.08E-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	1.00E+00	6.24E-01	3.76E-01	3.76E-01	2.02E-02			
invertebrates								
mammals/birds/herps								
Aquatic (sediments):								
plants								
invertebrates								
fish								
<b>Total</b>	<b>1.00E+00</b>			<b>3.76E-01</b>	<b>2.02E-02</b>	<b>6.30E-02</b>	<b>6.30E-02</b>	<b>0.00E+00</b>

References:

<sup>a</sup>CWS & CWF 2005

<sup>b</sup>USEPA 1993: equation 3-8 (rodents)

<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, fruit)

<sup>f</sup>Table 3.1; USEPA 1993: Table 4-5 (jackrabbit)

# 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)

# APPENDIX E

ERA Model 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>	Radio Island	
<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	0.098	
Intake factor (IFing-sl)	0.00011974	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	0.098	
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	0.098	
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	0.098	
Intake factor (IFing-tm)	0.013124829	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	0.098	
Intake factor (IFing-sw)	0.004541463	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	0.098	
Intake factor (IFing-sed)	4.43481E-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	0.098	
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	0.098	
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	0.098	
Intake factor (IFing-fsh)	0.00069078	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>	Radio Island	
<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	0.198	
Intake factor (IFing-sl)	0.002896243	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	0.198	
Intake factor (IFing-tp)	0.057072204	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	0.198	
Intake factor (IFing-ti)	0.019024068	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	0.198	
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	0.198	
Intake factor (IFing-sw)	0.014807631	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	0.198	
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	0.198	
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	0.198	
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	0.198	
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>	Radio Island	
<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>	Radio Island	
<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	0.495	
Intake factor (IFing-sl)	0.001408581	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	0.495	
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	0.495	
Intake factor (IFing-ti)	0.004866573	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	0.495	
Intake factor (IFing-tm)	0.184929775	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	0.495	
Intake factor (IFing-sw)	0.061179775	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	0.495	
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	0.495	
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	0.495	
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	0.495	
Intake factor (IFing-fsh)	0.004866573	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>	Radio Island	
<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	0.098	
Intake factor (IFing-sl)	0.000141735	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	0.098	
Intake factor (IFing-tp)	0.000395465	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	0.098	
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	0.098	
Intake factor (IFing-tm)	0.015423121	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	0.098	
Intake factor (IFing-sw)	0.008275556	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	0.098	
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	0.098	
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	0.098	
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	0.098	
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>	Radio Island	
<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	0.5086	
Intake factor (IFing-sl)	0.001880088	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	0.5086	
Intake factor (IFing-tp)	0.076006789	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	0.5086	
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	0.5086	
Intake factor (IFing-tm)	0.004000357	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	0.5086	
Intake factor (IFing-sw)	0.043543712	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	0.5086	
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	0.5086	
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	0.5086	
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	0.5086	
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 Site A

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>TPH - CCME CWS</b>	% Composition					
Aliph>C06-C08 - F1	0.55	1.07E+01	5.20E-01	7.95E-01	5.16E-03	--
Aliph>C08-C10 - F1	0.36	7.03E+00	2.12E-01	3.97E-01	2.05E-02	--
Arom>C08-C10 - F1	0.09	1.76E+00	1.05E-01	1.47E-01	3.86E-04	--
F1 - Total	1.00					
Aliph>C10-C12 - F2	0.36	5.28E+03	3.96E+01	2.28E+02	7.54E+01	--
Aliph>C12-C16 - F2	0.44	6.46E+03	2.44E+01	1.51E+02	1.07E+03	--
Arom>C10-C12 - F2	0.09	1.32E+03	5.68E+01	1.04E+02	3.72E-01	--
Arom>C12-C16 - F2	0.11	1.61E+03	5.93E+01	1.16E+02	8.29E-01	--
F2 - Total	1.00					
Aliph>C16-C21 - F3	0.56	1.47E+04	1.71E+01	4.58E+02	2.24E+03	--
Aliph>C21-C34 - F3	0.24	6.29E+03	7.33E+00	1.12E+02	9.61E+01	--
Arom>C16-C21 - F3	0.14	3.67E+03	5.17E+01	1.81E+02	3.90E+00	--
Arom>C21-C34 - F3	0.06	1.57E+03	1.38E+01	1.48E+02	1.16E+01	--
F3 - Total	1.00					
Aliph>C34-C50 - F4	0.80	6.32E+03	1.48E+00	4.73E+01	9.36E+02	--
Arom>C34-C50 - F4	0.20	1.58E+03	3.78E+00	4.23E+02	1.37E+02	--
F4 - Total	1.00					
<b>PCBs</b>						
Aroclor 1254 (Total PCBs)	11097691	2.60E-02	1.50E-03	4.55E-03	4.06E-03	--
<b>Inorganics</b>						
Antimony	7429905	4.69E+00	2.52E-02	1.68E-01	4.03E-04	--
Arsenic	7440382	3.96E+00	2.00E-01	1.02E-01	7.75E-03	--
Barium	7440393	2.03E+02	2.62E+00	2.95E+00	1.60E-01	--
Beryllium	7440393	4.00E-01	4.48E-02	2.88E-03	7.16E-04	--
Boron	7440417	6.95E+00	2.53E+01	1.11E+00	5.20E-01	--
Cadmium	7440439	1.82E+00	4.70E+00	2.13E+00	2.78E-01	--
Chromium (Total)	7440439	2.68E+01	2.55E+01	1.53E+00	8.30E-01	--
Cobalt	7440484	2.25E+01	3.90E+00	4.39E-01	2.15E-01	--
Copper	7440508	1.25E+02	3.54E+01	3.06E+00	4.95E+00	1.00E-02
Lead	7439921	6.73E+02	5.85E+01	2.46E+01	6.15E+00	--
Mercury - Inorganic	7487947	2.60E+00	9.32E-02	9.04E-02	2.80E-04	--
Nickel	7440020	5.62E+01	2.22E+01	2.22E+00	1.63E+00	--
Selenium	7782492	2.63E+00	2.21E-01	7.31E-02	3.04E-01	--
Silver	7440224	8.20E-01	6.37E-02	2.69E-01	1.05E-03	--
Tin	7440280	3.10E+01	4.48E-01	1.32E+00	2.74E+00	--
Vanadium	7440622	2.64E+01	2.83E-02	1.77E-01	1.04E-01	--
Zinc	7440666	8.87E+02	1.22E+03	1.27E+02	4.62E+01	3.00E-01

## Final Exposure Point Concentrations for Site B

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	2.70E-03	1.43E-04	2.09E-04	3.98E-04	--
<b>Inorganics</b>						
Arsenic	7440382	1.00E-20	1.07E-13	2.93E-16	1.06E-19	5.00E-04
Boron	7440417	5.00E+00	1.82E+01	8.00E-01	3.74E-01	--
Cadmium	7440439	1.95E+00	1.34E-01	2.25E+00	2.88E-01	--
Chromium (Total)	7440439	1.00E-20	1.82E-23	4.18E+01	1.57E-16	5.00E-03
Cobalt	7440484	--	--	--	--	5.00E-03
Copper	7440508	1.00E-20	3.86E-09	4.48E-06	3.19E-03	1.13E-02
Lead	7439921	1.24E+02	5.95E-01	6.31E+00	2.91E+00	5.00E-03
Nickel	7440020	4.06E+01	2.59E-01	2.41E+00	1.40E+00	--
Selenium	7782492	3.90E+00	3.42E-01	5.47E-02	3.52E-01	--
Zinc	7440666	2.47E+02	1.54E+01	8.34E+01	4.20E+01	8.50E-02

## Final Exposure Point Concentrations for the Background 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	4.00E-03	1.80E-03	3.57E-04	3.45E-03	--
<b>Inorganics</b>						
Arsenic	7440382	1.18E+00	1.00E-01	4.34E-02	2.88E-03	--
Cadmium	7440439	5.00E-01	1.20E+00	7.64E-01	1.48E-01	--
Chromium (Total)	7440439	4.10E+01	2.30E+01	1.49E+00	1.13E+00	--
Cobalt	7440484	1.34E+01	2.50E+00	2.62E-01	1.09E-01	--
Copper	7440508	2.51E+01	1.03E+01	2.00E+00	3.93E+00	3.00E-02
Lead	7439921	5.00E+00	7.50E+00	4.72E-01	7.04E-01	--
Nickel	7440020	4.28E+01	2.60E+01	2.38E+00	1.44E+00	--
Zinc	7440666	4.68E+01	1.88E+02	4.83E+01	3.72E+01	--



	Area (ha)	Hare 7	Fox 50	Lemming 4	Ermine 10	Owl 50	Ptarmigan 24
Site A	3.56	50.86%	9.80%	100.00%	49.50%	9.80%	19.80%
Site B	0.0314	0.45%	0.20%	5.00%	0.50%	0.20%	0.20%
SUM	3.59	51.31%	10.00%	105.00%	50.00%	10.00%	20.00%
Background	1.41	48.69%	90.00%	95.00%	50.00%	90.00%	80.00%

Ecological Hazard Quotients for the Rock Ptarmigan Exposed to Constituents of Interest in the CAM-D

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	9.27E-04	1.06E-05	5.55E-04	6.37E-06	5.82E-05	6.67E-07	--	--	1.77E-05
Toluene	9.08E+01	8.80E-04	9.69E-06	5.65E-04	6.22E-06	6.32E-05	6.96E-07	--	--	1.66E-05
Xylenes	9.01E+02	8.13E-03	9.02E-06	4.77E-03	5.29E-06	5.04E-04	5.60E-07	--	--	1.49E-05
TPH - CCME CWS										
Aliph>C06-C08 - F1	6.50E+01	3.11E-02	4.79E-04	2.97E-02	4.57E-04	1.51E-02	2.33E-04	--	--	1.17E-03
Aliph>C08-C10 - F1	6.50E+01	2.04E-02	3.14E-04	1.21E-02	1.86E-04	7.56E-03	1.16E-04	--	--	6.16E-04
Arom>C08-C10 - F1	2.17E+01	5.09E-03	2.35E-04	6.00E-03	2.77E-04	2.79E-03	1.29E-04	--	--	6.41E-04
F1 - Total										--
Aliph>C10-C12 - F2	6.50E+01	1.53E+01	2.36E-01	2.26E+00	3.48E-02	4.34E+00	6.68E-02	--	--	3.37E-01
Aliph>C12-C16 - F2	6.50E+01	1.87E+01	2.88E-01	1.39E+00	2.14E-02	2.87E+00	4.42E-02	--	--	3.54E-01
Arom>C10-C12 - F2	2.17E+01	3.83E+00	1.77E-01	3.24E+00	1.50E-01	1.97E+00	9.11E-02	--	--	4.18E-01
Arom>C12-C16 - F2	2.17E+01	4.68E+00	2.16E-01	3.38E+00	1.56E-01	2.20E+00	1.02E-01	--	--	4.74E-01
F2 - Total										--
Aliph>C16-C21 - F3	8.66E+01	4.25E+01	4.91E-01	9.77E-01	1.13E-02	8.70E+00	1.00E-01	--	--	6.02E-01
Aliph>C21-C34 - F3	8.66E+01	1.82E+01	2.10E-01	4.19E-01	4.83E-03	2.12E+00	2.45E-02	--	--	2.40E-01
Arom>C16-C21 - F3	4.33E+01	1.06E+01	2.45E-01	2.95E+00	6.82E-02	3.45E+00	7.96E-02	--	--	3.93E-01
Arom>C21-C34 - F3	4.33E+01	4.55E+00	1.05E-01	7.87E-01	1.82E-02	2.82E+00	6.52E-02	--	--	1.88E-01
F3 - Total										--
Aliph>C34-C50 - F4	1.30E+02	1.83E+01	1.41E-01	8.42E-02	6.48E-04	9.00E-01	6.93E-03	--	--	1.49E-01
Arom>C34-C50 - F4	1.73E+01	4.58E+00	2.64E-01	2.16E-01	1.25E-02	8.05E+00	4.65E-01	--	--	7.41E-01
F4 - Total										--
PAHs										
Naphthalene	6.99E+01	1.19E-02	1.71E-04	4.30E-01	6.16E-03	6.97E-03	9.97E-05	--	--	6.43E-03
PCBs										
Aroclor 1254 (Total PCBs)	1.56E+00	7.52E-05	4.82E-05	8.56E-05	5.49E-05	8.66E-05	5.55E-05	--	--	1.59E-04
Inorganics										
Antimony	4.37E-01	1.36E-02	3.11E-02	1.44E-03	3.30E-03	3.20E-03	7.32E-03	--	--	4.17E-02
Arsenic	1.11E+01	1.15E-02	1.03E-03	1.14E-02	1.03E-03	1.94E-03	1.75E-04	--	--	2.23E-03
Barium	1.10E+02	5.87E-01	5.33E-03	1.49E-01	1.35E-03	5.62E-02	5.10E-04	--	--	7.19E-03
Boron	8.66E+01	2.01E-02	2.32E-04	1.45E+00	1.67E-02	2.12E-02	2.44E-04	--	--	1.72E-02
Cadmium	1.73E+01	5.27E-03	3.04E-04	2.68E-01	1.55E-02	4.06E-02	2.34E-03	--	--	1.81E-02
Chromium (Total)	4.14E+00	7.76E-02	1.87E-02	1.46E+00	3.51E-01	2.92E-02	7.05E-03	--	--	3.77E-01
Cobalt	2.83E+00	6.51E-02	2.30E-02	2.23E-01	7.86E-02	8.35E-03	2.95E-03	--	--	1.05E-01
Copper	8.15E+01	3.63E-01	4.46E-03	2.02E+00	2.48E-02	5.82E-02	7.14E-04	1.48E-04	1.82E-06	3.00E-02
Lead	1.43E+01	1.95E+00	1.36E-01	3.34E+00	2.33E-01	4.69E-01	3.28E-02	--	--	4.02E-01
Mercury - Inorganic	1.14E+00	7.53E-03	6.61E-03	5.32E-03	4.67E-03	1.72E-03	1.51E-03	--	--	1.28E-02
Nickel	9.27E+01	1.63E-01	1.76E-03	1.27E+00	1.37E-02	4.22E-02	4.55E-04	--	--	1.59E-02
Selenium	8.66E-01	7.62E-03	8.79E-03	1.26E-02	1.46E-02	1.39E-03	1.60E-03	--	--	2.50E-02
Silver	6.32E+00	2.37E-03	3.76E-04	3.64E-03	5.75E-04	5.12E-03	8.09E-04	--	--	1.76E-03
Tin	2.14E+01	8.98E-02	4.20E-03	2.56E-02	1.20E-03	2.51E-02	1.17E-03	--	--	6.57E-03
Zinc	1.01E+02	2.57E+00	2.54E-02	6.97E+01	6.88E-01	2.41E+00	2.38E-02	4.44E-03	4.39E-05	7.38E-01

Ecological Hazard Quotients for the Ermine Exposed to Constituents of Interest in the CAM-D Continued

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	
TPH - CCME CWS										
Aliph>C06-C08 - F1	1.20E+02	1.51E-02	1.26E-04	3.87E-03	3.21E-05	9.54E-04	7.92E-06	--	--	1.66E-04
Aliph>C08-C10 - F1	1.20E+02	9.91E-03	8.23E-05	1.93E-03	1.61E-05	3.79E-03	3.14E-05	--	--	1.30E-04
Arom>C08-C10 - F1	2.58E+01	2.48E-03	9.60E-05	7.13E-04	2.76E-05	7.13E-05	2.76E-06	--	--	1.26E-04
F1 - Total										--
Aliph>C10-C12 - F2	1.20E+02	7.44E+00	6.18E-02	1.11E+00	9.21E-03	1.39E+01	1.16E-01	--	--	1.87E-01
Aliph>C12-C16 - F2	1.20E+02	9.10E+00	7.55E-02	7.35E-01	6.10E-03	1.98E+02	1.65E+00	--	--	1.73E+00
Arom>C10-C12 - F2	2.58E+01	1.86E+00	7.21E-02	5.05E-01	1.96E-02	6.89E-02	2.67E-03	--	--	9.43E-02
Arom>C12-C16 - F2	2.58E+01	2.27E+00	8.81E-02	5.64E-01	2.18E-02	1.53E-01	5.94E-03	--	--	1.16E-01
F2 - Total										--
Aliph>C16-C21 - F3	2.41E+02	2.07E+01	8.58E-02	2.23E+00	9.24E-03	4.15E+02	1.72E+00	--	--	1.82E+00
Aliph>C21-C34 - F3	2.41E+02	8.86E+00	3.68E-02	5.43E-01	2.25E-03	1.78E+01	7.38E-02	--	--	1.13E-01
Arom>C16-C21 - F3	5.16E+01	5.17E+00	1.00E-01	8.82E-01	1.71E-02	7.21E-01	1.40E-02	--	--	1.31E-01
Arom>C21-C34 - F3	5.16E+01	2.21E+00	4.29E-02	7.22E-01	1.40E-02	2.15E+00	4.17E-02	--	--	9.86E-02
F3 - Total										--
Aliph>C34-C50 - F4	2.24E+03	8.91E+00	3.98E-03	2.30E-01	1.03E-04	1.73E+02	7.74E-02	--	--	8.15E-02
Arom>C34-C50 - F4	2.32E+02	2.23E+00	9.59E-03	2.06E+00	8.86E-03	2.54E+01	1.09E-01	--	--	1.28E-01
F4 - Total										--
PCBs										
Aroclor 1254 (Total PCBs)	6.37E-01	3.66E-05	5.74E-05	2.21E-05	3.48E-05	7.50E-04	1.18E-03	--	--	1.27E-03
Inorganics										
Antimony	1.17E+00	6.61E-03	5.64E-03	8.18E-04	6.99E-04	7.46E-05	6.37E-05	--	--	6.40E-03
Arsenic	1.18E+00	5.58E-03	4.73E-03	4.97E-04	4.21E-04	1.43E-03	1.21E-03	--	--	6.36E-03
Barium	2.77E+01	2.86E-01	1.03E-02	1.44E-02	5.19E-04	2.96E-02	1.07E-03	--	--	1.19E-02
Boron	1.02E+02	9.79E-03	9.63E-05	5.41E-03	5.33E-05	9.62E-02	9.47E-04	--	--	1.10E-03
Cadmium	1.09E+01	2.56E-03	2.36E-04	1.04E-02	9.56E-04	5.15E-02	4.74E-03	--	--	5.93E-03
Chromium (Total)	4.68E+00	3.77E-02	8.05E-03	7.47E-03	1.59E-03	1.53E-01	3.28E-02	--	--	4.24E-02
Cobalt	1.44E+01	3.17E-02	2.20E-03	2.14E-03	1.48E-04	3.97E-02	2.76E-03	--	--	5.11E-03
Copper	1.75E+01	1.77E-01	1.01E-02	1.49E-02	8.50E-04	9.16E-01	5.23E-02	6.12E-04	3.49E-05	6.33E-02
Lead	8.69E+01	9.47E-01	1.09E-02	1.20E-01	1.38E-03	1.14E+00	1.31E-02	--	--	2.54E-02
Mercury - Inorganic	5.78E+00	3.66E-03	6.34E-04	4.40E-04	7.61E-05	5.18E-05	8.95E-06	--	--	7.19E-04
Nickel	8.69E+01	7.92E-02	9.12E-04	1.08E-02	1.24E-04	3.02E-01	3.48E-03	--	--	4.52E-03
Selenium	3.58E-01	3.70E-03	1.03E-02	3.56E-04	9.93E-04	5.62E-02	1.57E-01	--	--	1.68E-01
Silver	3.39E+00	1.16E-03	3.41E-04	1.31E-03	3.86E-04	1.94E-04	5.72E-05	--	--	7.84E-04
Tin	3.28E+01	4.37E-02	1.33E-03	6.42E-03	1.96E-04	5.07E-01	1.55E-02	--	--	1.70E-02
Zinc	3.47E+02	1.25E+00	3.60E-03	6.17E-01	1.78E-03	8.54E+00	2.46E-02	1.84E-02	5.28E-05	3.00E-02

Ecological Hazard Quotients for the Arctic Fox Exposed to Constituents of Interest in the CAM-D (Continued)

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	
TPH - CCME CWS										
Aliph>C06-C08 - F1	9.46E+01	1.52E-03	1.61E-05	2.06E-04	2.17E-06	7.96E-05	8.41E-07	--	--	1.91E-05
Aliph>C08-C10 - F1	9.46E+01	9.97E-04	1.05E-05	8.37E-05	8.85E-07	3.16E-04	3.34E-06	--	--	1.48E-05
Arom>C08-C10 - F1	2.03E+01	2.49E-04	1.23E-05	4.15E-05	2.05E-06	5.95E-06	2.93E-07	--	--	1.46E-05
F1 - Total										--
Aliph>C10-C12 - F2	9.46E+01	7.49E-01	7.91E-03	1.57E-02	1.65E-04	1.16E+00	1.23E-02	--	--	2.04E-02
Aliph>C12-C16 - F2	9.46E+01	9.15E-01	9.67E-03	9.64E-03	1.02E-04	1.65E+01	1.75E-01	--	--	1.85E-01
Arom>C10-C12 - F2	2.03E+01	1.87E-01	9.23E-03	2.25E-02	1.11E-03	5.74E-03	2.83E-04	--	--	1.06E-02
Arom>C12-C16 - F2	2.03E+01	2.29E-01	1.13E-02	2.34E-02	1.16E-03	1.28E-02	6.30E-04	--	--	1.31E-02
F2 - Total										--
Aliph>C16-C21 - F3	1.89E+02	2.08E+00	1.10E-02	6.77E-03	3.57E-05	3.46E+01	1.83E-01	--	--	1.94E-01
Aliph>C21-C34 - F3	1.89E+02	8.91E-01	4.71E-03	2.90E-03	1.53E-05	1.48E+00	7.83E-03	--	--	1.26E-02
Arom>C16-C21 - F3	4.06E+01	5.20E-01	1.28E-02	2.05E-02	5.04E-04	6.02E-02	1.48E-03	--	--	1.48E-02
Arom>C21-C34 - F3	4.06E+01	2.23E-01	5.49E-03	5.46E-03	1.34E-04	1.79E-01	4.42E-03	--	--	1.01E-02
F3 - Total										--
Aliph>C34-C50 - F4	1.76E+03	8.96E-01	5.10E-04	5.83E-04	3.32E-07	1.44E+01	8.22E-03	--	--	8.73E-03
Arom>C34-C50 - F4	1.83E+02	2.24E-01	1.23E-03	1.50E-03	8.20E-06	2.12E+00	1.16E-02	--	--	1.28E-02
F4 - Total										--
PCBs										
Aroclor 1254 (Total PCBs)	5.01E-01	3.68E-06	7.35E-06	5.93E-07	1.19E-06	6.26E-05	1.25E-04	--	--	1.34E-04
Inorganics										
Antimony	9.20E-01	6.65E-04	7.22E-04	9.97E-06	1.08E-05	6.22E-06	6.76E-06	--	--	7.40E-04
Arsenic	9.28E-01	5.61E-04	6.05E-04	7.91E-05	8.53E-05	1.20E-04	1.29E-04	--	--	8.19E-04
Barium	2.18E+01	2.87E-02	1.32E-03	1.03E-03	4.76E-05	2.47E-03	1.13E-04	--	--	1.48E-03
Boron	7.98E+01	9.85E-04	1.23E-05	1.00E-02	1.25E-04	8.02E-03	1.01E-04	--	--	2.38E-04
Cadmium	8.53E+00	2.58E-04	3.02E-05	1.86E-03	2.18E-04	4.29E-03	5.03E-04	--	--	7.51E-04
Chromium (Total)	3.68E+00	3.80E-03	1.03E-03	1.01E-02	2.74E-03	1.28E-02	3.48E-03	--	--	7.25E-03
Cobalt	1.13E+01	3.19E-03	2.82E-04	1.54E-03	1.36E-04	3.31E-03	2.93E-04	--	--	7.11E-04
Copper	1.38E+01	1.78E-02	1.29E-03	1.40E-02	1.02E-03	7.64E-02	5.56E-03	8.28E-05	6.02E-06	7.87E-03
Lead	6.82E+01	9.53E-02	1.40E-03	2.31E-02	3.39E-04	9.48E-02	1.39E-03	--	--	3.12E-03
Mercury - Inorganic	4.54E+00	3.69E-04	8.11E-05	3.68E-05	8.11E-06	4.32E-06	9.50E-07	--	--	9.02E-05
Nickel	6.82E+01	7.97E-03	1.17E-04	8.78E-03	1.29E-04	2.52E-02	3.69E-04	--	--	6.15E-04
Selenium	2.82E-01	3.73E-04	1.32E-03	8.76E-05	3.11E-04	4.69E-03	1.66E-02	--	--	1.83E-02
Silver	2.66E+00	1.16E-04	4.36E-05	2.52E-05	9.46E-06	1.62E-05	6.07E-06	--	--	5.91E-05
Tin	2.58E+01	4.40E-03	1.71E-04	1.77E-04	6.88E-06	4.23E-02	1.64E-03	--	--	1.82E-03
Zinc	2.73E+02	1.26E-01	4.60E-04	4.83E-01	1.77E-03	7.12E-01	2.61E-03	2.48E-03	9.09E-06	4.85E-03

Ecological Hazard Quotients for the Arctic Hare Exposed to Constituents of Interest in the CAM-D

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	
TPH - CCME CWS										
Aliph>C06-C08 - F1	9.54E+01	2.02E-02	2.12E-04	3.95E-02	4.14E-04	2.06E-05	2.16E-07	--	--	6.26E-04
Aliph>C08-C10 - F1	9.54E+01	1.32E-02	1.39E-04	1.61E-02	1.69E-04	8.19E-05	8.58E-07	--	--	3.08E-04
Arom>C08-C10 - F1	2.05E+01	3.31E-03	1.62E-04	7.99E-03	3.90E-04	1.54E-06	7.54E-08	--	--	5.52E-04
F1 - Total										--
Aliph>C10-C12 - F2	9.54E+01	9.93E+00	1.04E-01	3.01E+00	3.15E-02	3.02E-01	3.16E-03	--	--	1.39E-01
Aliph>C12-C16 - F2	9.54E+01	1.21E+01	1.27E-01	1.85E+00	1.94E-02	4.29E+00	4.50E-02	--	--	1.92E-01
Arom>C10-C12 - F2	2.05E+01	2.48E+00	1.21E-01	4.32E+00	2.11E-01	1.49E-03	7.28E-05	--	--	3.33E-01
Arom>C12-C16 - F2	2.05E+01	3.04E+00	1.48E-01	4.51E+00	2.20E-01	3.31E-03	1.62E-04	--	--	3.69E-01
F2 - Total										--
Aliph>C16-C21 - F3	1.91E+02	2.76E+01	1.45E-01	1.30E+00	6.81E-03	8.97E+00	4.70E-02	--	--	1.98E-01
Aliph>C21-C34 - F3	1.91E+02	1.18E+01	6.19E-02	5.57E-01	2.92E-03	3.84E-01	2.01E-03	--	--	6.69E-02
Arom>C16-C21 - F3	4.09E+01	6.90E+00	1.69E-01	3.93E+00	9.61E-02	1.56E-02	3.82E-04	--	--	2.65E-01
Arom>C21-C34 - F3	4.09E+01	2.96E+00	7.23E-02	1.05E+00	2.56E-02	4.65E-02	1.14E-03	--	--	9.90E-02
F3 - Total										--
Aliph>C34-C50 - F4	1.77E+03	1.19E+01	6.71E-03	1.12E-01	6.33E-05	3.75E+00	2.11E-03	--	--	8.89E-03
Arom>C34-C50 - F4	1.84E+02	2.97E+00	1.62E-02	2.88E-01	1.56E-03	5.49E-01	2.98E-03	--	--	2.07E-02
F4 - Total										--
PCBs										
Aroclor 1254 (Total PCBs)	5.05E-01	4.88E-05	9.67E-05	1.14E-04	2.26E-04	1.62E-05	3.22E-05	--	--	3.55E-04
Inorganics										
Antimony	9.28E-01	8.82E-03	9.50E-03	1.92E-03	2.07E-03	1.61E-06	1.74E-06	--	--	1.16E-02
Arsenic	9.35E-01	7.45E-03	7.96E-03	1.52E-02	1.63E-02	3.10E-05	3.32E-05	--	--	2.42E-02
Barium	2.19E+01	3.81E-01	1.74E-02	1.99E-01	9.06E-03	6.40E-04	2.92E-05	--	--	2.65E-02
Boron	8.05E+01	1.31E-02	1.62E-04	1.93E+00	2.39E-02	2.08E-03	2.59E-05	--	--	2.41E-02
Cadmium	8.60E+00	3.42E-03	3.98E-04	3.57E-01	4.15E-02	1.11E-03	1.29E-04	--	--	4.21E-02
Chromium (Total)	3.71E+00	5.03E-02	1.36E-02	1.94E+00	5.22E-01	3.32E-03	8.94E-04	--	--	5.37E-01
Cobalt	1.14E+01	4.23E-02	3.71E-03	2.96E-01	2.60E-02	8.59E-04	7.54E-05	--	--	2.98E-02
Copper	1.39E+01	2.36E-01	1.70E-02	2.69E+00	1.94E-01	1.98E-02	1.43E-03	4.35E-04	3.14E-05	2.12E-01
Lead	6.88E+01	1.26E+00	1.84E-02	4.45E+00	6.46E-02	2.46E-02	3.57E-04	--	--	8.33E-02
Mercury - Inorganic	4.58E+00	4.89E-03	1.07E-03	7.08E-03	1.55E-03	1.12E-06	2.44E-07	--	--	2.61E-03
Nickel	6.88E+01	1.06E-01	1.54E-03	1.69E+00	2.45E-02	6.54E-03	9.50E-05	--	--	2.62E-02
Selenium	2.84E-01	4.94E-03	1.74E-02	1.68E-02	5.93E-02	1.22E-03	4.28E-03	--	--	8.10E-02
Silver	2.69E+00	1.54E-03	5.74E-04	4.84E-03	1.80E-03	4.20E-06	1.56E-06	--	--	2.38E-03
Tin	2.60E+01	5.83E-02	2.24E-03	3.41E-02	1.31E-03	1.10E-02	4.22E-04	--	--	3.98E-03
Zinc	2.75E+02	1.67E+00	6.06E-03	9.28E+01	3.37E-01	1.85E-01	6.71E-04	1.31E-02	4.75E-05	3.44E-01

Ecological Hazard Quotients for the Snowy Owl Exposed to Constituents of Interest in the CAM-D (Continued)

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	
TPH - CCME CWS								
Aliph>C06-C08 - F1	8.66E+01	1.29E-03	1.49E-05	6.77E-05	7.82E-07	--	--	1.56E-05
Aliph>C08-C10 - F1	8.66E+01	8.42E-04	9.73E-06	2.69E-04	3.10E-06	--	--	1.28E-05
Arom>C08-C10 - F1	2.89E+01	2.11E-04	7.30E-06	5.06E-06	1.75E-07	--	--	7.47E-06
F1 - Total								--
Aliph>C10-C12 - F2	8.66E+01	6.33E-01	7.31E-03	9.89E-01	1.14E-02	--	--	1.87E-02
Aliph>C12-C16 - F2	8.66E+01	7.73E-01	8.93E-03	1.41E+01	1.63E-01	--	--	1.72E-01
Arom>C10-C12 - F2	2.89E+01	1.58E-01	5.48E-03	4.89E-03	1.69E-04	--	--	5.65E-03
Arom>C12-C16 - F2	2.89E+01	1.93E-01	6.70E-03	1.09E-02	3.77E-04	--	--	7.08E-03
F2 - Total								--
Aliph>C16-C21 - F3	1.15E+02	1.76E+00	1.52E-02	2.94E+01	2.55E-01	--	--	2.70E-01
Aliph>C21-C34 - F3	1.15E+02	7.53E-01	6.52E-03	1.26E+00	1.09E-02	--	--	1.74E-02
Arom>C16-C21 - F3	5.77E+01	4.39E-01	7.61E-03	5.12E-02	8.87E-04	--	--	8.50E-03
Arom>C21-C34 - F3	5.77E+01	1.88E-01	3.26E-03	1.53E-01	2.65E-03	--	--	5.91E-03
F3 - Total								--
Aliph>C34-C50 - F4	1.73E+02	7.57E-01	4.37E-03	1.23E+01	7.10E-02	--	--	7.54E-02
Arom>C34-C50 - F4	2.31E+01	1.89E-01	8.20E-03	1.80E+00	7.81E-02	--	--	8.63E-02
F4 - Total								--
PCBs								
Aroclor 1254 (Total PCBs)	2.08E+00	3.11E-06	1.50E-06	5.32E-05	2.56E-05	--	--	2.71E-05
Inorganics								
Antimony	5.82E-01	5.62E-04	9.65E-04	5.29E-06	9.10E-06	--	--	9.74E-04
Arsenic	1.48E+01	4.74E-04	3.20E-05	1.02E-04	6.87E-06	--	--	3.89E-05
Barium	1.47E+02	2.43E-02	1.65E-04	2.10E-03	1.43E-05	--	--	1.80E-04
Boron	1.15E+02	8.32E-04	7.21E-06	6.83E-03	5.92E-05	--	--	6.64E-05
Cadmium	2.31E+01	2.18E-04	9.44E-06	3.65E-03	1.58E-04	--	--	1.68E-04
Chromium (Total)	5.52E+00	3.21E-03	5.81E-04	1.09E-02	1.97E-03	--	--	2.55E-03
Cobalt	3.77E+00	2.69E-03	7.13E-04	2.82E-03	7.47E-04	--	--	1.46E-03
Copper	1.09E+02	1.50E-02	1.38E-04	6.50E-02	5.98E-04	4.54E-05	4.18E-07	7.37E-04
Lead	1.91E+01	8.05E-02	4.22E-03	8.07E-02	4.23E-03	--	--	8.46E-03
Mercury - Inorganic	1.52E+00	3.11E-04	2.05E-04	3.67E-06	2.42E-06	--	--	2.07E-04
Nickel	1.24E+02	6.73E-03	5.45E-05	2.14E-02	1.74E-04	--	--	2.28E-04
Selenium	1.15E+00	3.15E-04	2.73E-04	3.99E-03	3.45E-03	--	--	3.73E-03
Silver	8.43E+00	9.82E-05	1.17E-05	1.38E-05	1.63E-06	--	--	1.33E-05
Tin	2.85E+01	3.71E-03	1.30E-04	3.60E-02	1.26E-03	--	--	1.39E-03
Zinc	1.35E+02	1.06E-01	7.87E-04	6.06E-01	4.49E-03	1.36E-03	1.01E-05	5.29E-03

Ecological Hazard Quotients for the Collared Lemming Exposed to Constituents of Interest in the CAM-D (Continued)

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	
TPH - CCME CWS								
Aliph>C06-C08 - F1	1.21E+02	5.99E-02	4.96E-04	1.22E-01	1.01E-03	--	--	1.51E-03
Aliph>C08-C10 - F1	1.21E+02	3.92E-02	3.25E-04	4.98E-02	4.12E-04	--	--	7.37E-04
Arom>C08-C10 - F1	2.59E+01	9.80E-03	3.79E-04	2.47E-02	9.55E-04	--	--	1.33E-03
F1 - Total								--
Aliph>C10-C12 - F2	1.21E+02	2.95E+01	2.44E-01	9.31E+00	7.71E-02	--	--	3.21E-01
Aliph>C12-C16 - F2	1.21E+02	3.60E+01	2.98E-01	5.74E+00	4.75E-02	--	--	3.46E-01
Arom>C10-C12 - F2	2.59E+01	7.36E+00	2.84E-01	1.34E+01	5.16E-01	--	--	8.01E-01
Arom>C12-C16 - F2	2.59E+01	9.00E+00	3.48E-01	1.39E+01	5.39E-01	--	--	8.87E-01
F2 - Total								--
Aliph>C16-C21 - F3	2.42E+02	8.18E+01	3.39E-01	4.03E+00	1.67E-02	--	--	3.55E-01
Aliph>C21-C34 - F3	2.42E+02	3.50E+01	1.45E-01	1.73E+00	7.14E-03	--	--	1.52E-01
Arom>C16-C21 - F3	5.18E+01	2.04E+01	3.95E-01	1.22E+01	2.35E-01	--	--	6.30E-01
Arom>C21-C34 - F3	5.18E+01	8.76E+00	1.69E-01	3.25E+00	6.27E-02	--	--	2.32E-01
F3 - Total								--
Aliph>C34-C50 - F4	2.24E+03	3.53E+01	1.57E-02	3.47E-01	1.55E-04	--	--	1.59E-02
Arom>C34-C50 - F4	2.33E+02	8.81E+00	3.78E-02	8.90E-01	3.82E-03	--	--	4.17E-02
F4 - Total								--
PCBs								
Aroclor 1254 (Total PCBs)	6.39E-01	1.45E-04	2.26E-04	3.53E-04	5.52E-04	--	--	7.79E-04
Inorganics								
Antimony	1.17E+00	2.61E-02	2.23E-02	5.93E-03	5.05E-03	--	--	2.73E-02
Arsenic	1.18E+00	2.21E-02	1.86E-02	4.71E-02	3.98E-02	--	--	5.84E-02
Barium	2.78E+01	1.13E+00	4.07E-02	6.15E-01	2.22E-02	--	--	6.29E-02
Boron	1.02E+02	3.87E-02	3.80E-04	5.96E+00	5.85E-02	--	--	5.89E-02
Cadmium	1.09E+01	1.01E-02	9.32E-04	1.11E+00	1.02E-01	--	--	1.03E-01
Chromium (Total)	4.70E+00	1.49E-01	3.18E-02	6.00E+00	1.28E+00	--	--	1.31E+00
Cobalt	1.44E+01	1.25E-01	8.69E-03	9.18E-01	6.36E-02	--	--	7.23E-02
Copper	1.76E+01	6.99E-01	3.98E-02	8.33E+00	4.75E-01	1.29E-03	7.37E-05	5.14E-01
Lead	8.71E+01	3.75E+00	4.30E-02	1.38E+01	1.58E-01	--	--	2.01E-01
Mercury - Inorganic	5.80E+00	1.45E-02	2.50E-03	2.19E-02	3.78E-03	--	--	6.28E-03
Nickel	8.71E+01	3.13E-01	3.60E-03	5.22E+00	6.00E-02	--	--	6.36E-02
Selenium	3.59E-01	1.47E-02	4.08E-02	5.21E-02	1.45E-01	--	--	1.86E-01
Silver	3.40E+00	4.57E-03	1.34E-03	1.50E-02	4.41E-03	--	--	5.75E-03
Tin	3.29E+01	1.73E-01	5.26E-03	1.06E-01	3.21E-03	--	--	8.47E-03
Zinc	3.48E+02	4.94E+00	1.42E-02	2.87E+02	8.25E-01	3.88E-02	1.11E-04	8.39E-01

Ecological Hazard Quotients for the Rock Ptarmigan Exposed to Constituents of Interest in the CAM-D (Continued)

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	7.90E-08	5.07E-08	8.22E-08	5.27E-08	4.02E-08	2.58E-08	--	--	1.29E-07
Inorganics										
Boron	8.66E+01	1.46E-04	1.69E-06	1.05E-02	1.21E-04	1.54E-04	1.77E-06	--	--	1.25E-04
Cadmium	1.73E+01	5.70E-05	3.29E-06	7.74E-05	4.47E-06	4.33E-04	2.50E-05	--	--	3.28E-05
Copper	8.15E+01	2.93E-25	3.59E-27	2.23E-12	2.73E-14	8.61E-10	1.06E-11	4.49E-06	5.50E-08	5.50E-08
Lead	1.43E+01	3.64E-03	2.54E-04	3.43E-04	2.40E-05	1.21E-03	8.47E-05	--	--	3.63E-04
Nickel	9.27E+01	1.19E-03	1.28E-05	1.49E-04	1.61E-06	4.64E-04	5.01E-06	--	--	1.94E-05
Selenium	8.66E-01	1.14E-04	1.32E-04	1.97E-04	2.28E-04	1.05E-05	1.21E-05	--	--	3.72E-04
Zinc	1.01E+02	7.23E-03	7.14E-05	8.89E-03	8.78E-05	1.60E-02	1.58E-04	--	--	3.18E-04



Ecological Hazard Quotients for the Ermine Exposed to Constituents of Interest in the CAM-D (Continued)

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	3.84E-08	6.03E-08	1.03E-08	1.61E-08	7.43E-07	1.17E-06	--	--	1.24E-06
Inorganics										
Boron	1.02E+02	7.11E-05	7.00E-07	3.93E-05	3.87E-07	6.99E-04	6.88E-06	--	--	7.97E-06
Cadmium	1.09E+01	2.77E-05	2.56E-06	1.11E-04	1.02E-05	5.38E-04	4.95E-05	--	--	6.23E-05
Copper	1.75E+01	1.42E-25	8.13E-27	2.20E-10	1.26E-11	5.96E-06	3.41E-07	1.85E-05	1.06E-06	1.40E-06
Lead	8.69E+01	1.77E-03	2.04E-05	3.10E-04	3.57E-06	5.44E-03	6.27E-05	--	--	8.66E-05
Nickel	8.69E+01	5.78E-04	6.65E-06	1.19E-04	1.37E-06	2.62E-03	3.02E-05	--	--	3.82E-05
Selenium	3.58E-01	5.55E-05	1.55E-04	2.69E-06	7.51E-06	6.58E-04	1.84E-03	--	--	2.00E-03
Zinc	3.47E+02	3.52E-03	1.01E-05	4.10E-03	1.18E-05	7.85E-02	2.26E-04	--	--	2.48E-04

Ecological Hazard Quotients for the Arctic Fox Exposed to Constituents of Interest in the CAM-D (Continued)

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	7.81E-09	1.56E-08	1.15E-09	2.30E-09	1.25E-07	2.50E-07	--	--	2.68E-07
Inorganics										
Boron	7.98E+01	1.45E-05	1.81E-07	1.47E-04	1.84E-06	1.18E-04	1.48E-06	--	--	3.50E-06
Cadmium	8.53E+00	5.64E-06	6.61E-07	1.08E-06	1.27E-07	9.06E-05	1.06E-05	--	--	1.14E-05
Copper	1.38E+01	2.89E-26	2.10E-27	3.12E-14	2.27E-15	1.00E-06	7.30E-08	5.07E-06	3.68E-07	4.41E-07
Lead	6.82E+01	3.60E-04	5.27E-06	4.80E-06	7.04E-08	9.17E-04	1.34E-05	--	--	1.88E-05
Nickel	6.82E+01	1.17E-04	1.72E-06	2.09E-06	3.06E-08	4.42E-04	6.48E-06	--	--	8.23E-06
Selenium	2.82E-01	1.13E-05	4.01E-05	2.76E-06	9.81E-06	1.11E-04	3.94E-04	--	--	4.44E-04
Zinc	2.73E+02	7.15E-04	2.62E-06	1.25E-04	4.56E-07	1.32E-02	4.85E-05	--	--	5.15E-05

Ecological Hazard Quotients for the Arctic Hare Exposed to Constituents of Interest in the CAM-D (Continued)

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	4.49E-08	8.90E-08	9.59E-08	1.90E-07	1.41E-08	2.79E-08	--	--	3.07E-07
Inorganics										
Boron	8.05E+01	8.32E-05	1.03E-06	1.23E-02	1.52E-04	1.32E-05	1.65E-07	--	--	1.53E-04
Cadmium	8.60E+00	3.24E-05	3.77E-06	9.02E-05	1.05E-05	1.02E-05	1.18E-06	--	--	1.54E-05
Copper	1.39E+01	1.66E-25	1.20E-26	2.60E-12	1.87E-13	1.13E-07	8.14E-09	1.16E-05	8.33E-07	8.41E-07
Lead	6.88E+01	2.07E-03	3.01E-05	4.00E-04	5.81E-06	1.03E-04	1.50E-06	--	--	3.74E-05
Nickel	6.88E+01	6.76E-04	9.82E-06	1.74E-04	2.53E-06	4.97E-05	7.22E-07	--	--	1.31E-05
Selenium	2.84E-01	6.49E-05	2.29E-04	2.30E-04	8.11E-04	1.25E-05	4.39E-05	--	--	1.08E-03
Zinc	2.75E+02	4.11E-03	1.49E-05	1.04E-02	3.77E-05	1.49E-03	5.41E-06	--	--	5.80E-05

Ecological Hazard Quotients for the Snowy Owl Exposed to Constituents of Interest in the CAM-D (Continued)

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	6.60E-09	3.18E-09	1.07E-07	5.13E-08	--	--	5.45E-08
Inorganics								
Boron	1.15E+02	1.22E-05	1.06E-07	1.00E-04	8.69E-07	--	--	9.74E-07
Cadmium	2.31E+01	4.77E-06	2.06E-07	7.71E-05	3.34E-06	--	--	3.55E-06
Copper	1.09E+02	2.44E-26	2.25E-28	8.55E-07	7.87E-09	2.78E-06	2.56E-08	3.35E-08
Lead	1.91E+01	3.04E-04	1.59E-05	7.80E-04	4.09E-05	--	--	5.69E-05
Nickel	1.24E+02	9.92E-05	8.03E-07	3.76E-04	3.05E-06	--	--	3.85E-06
Selenium	1.15E+00	9.53E-06	8.26E-06	9.44E-05	8.18E-05	--	--	9.00E-05
Zinc	1.35E+02	6.04E-04	4.48E-06	1.13E-02	8.34E-05	--	--	8.79E-05

Ecological Hazard Quotients for the Collared Lemming Exposed to Constituents of Interest in the CAM-D (Continued)

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	7.52E-07	1.18E-06	1.68E-06	2.63E-06	--	--	3.80E-06
Inorganics								
Boron	1.02E+02	1.39E-03	1.37E-05	2.14E-01	2.10E-03	--	--	2.12E-03
Cadmium	1.09E+01	5.43E-04	4.99E-05	1.58E-03	1.45E-04	--	--	1.95E-04
Copper	1.76E+01	2.79E-24	1.59E-25	4.54E-11	2.59E-12	1.94E-04	1.11E-05	1.11E-05
Lead	8.71E+01	3.47E-02	3.98E-04	7.00E-03	8.04E-05	--	--	4.78E-04
Nickel	8.71E+01	1.13E-02	1.30E-04	3.05E-03	3.50E-05	--	--	1.65E-04
Selenium	3.59E-01	1.09E-03	3.03E-03	4.02E-03	1.12E-02	--	--	1.42E-02
Zinc	3.48E+02	6.89E-02	1.98E-04	1.82E-01	5.21E-04	--	--	7.19E-04

Ecological Hazard Quotients for the Rock Ptarmigan Exposed to Constituents of Interest in the CAM-D (Continued)

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	4.68E-05	3.00E-05	4.15E-04	2.66E-04	2.75E-05	1.76E-05	--	--	3.14E-04
Inorganics										
Arsenic	1.11E+01	1.38E-02	1.24E-03	2.31E-02	2.07E-03	3.34E-03	3.00E-04	--	--	3.62E-03
Cadmium	1.73E+01	5.85E-03	3.38E-04	2.77E-01	1.60E-02	5.87E-02	3.39E-03	--	--	1.97E-02
Chromium (Total)	4.14E+00	4.80E-01	1.16E-01	5.30E+00	1.28E+00	1.15E-01	2.77E-02	--	--	1.42E+00
Cobalt	2.83E+00	1.57E-01	5.54E-02	5.76E-01	2.04E-01	2.01E-02	7.10E-03	--	--	2.66E-01
Copper	8.15E+01	2.94E-01	3.60E-03	2.38E+00	2.91E-02	1.54E-01	1.89E-03	1.79E-03	2.20E-05	3.46E-02
Lead	1.43E+01	5.85E-02	4.09E-03	1.73E+00	1.21E-01	3.62E-02	2.53E-03	--	--	1.28E-01
Nickel	9.27E+01	5.01E-01	5.40E-03	6.00E+00	6.47E-02	1.83E-01	1.98E-03	--	--	7.21E-02
Zinc	1.01E+02	5.48E-01	5.41E-03	4.34E+01	4.28E-01	3.71E+00	3.67E-02	--	--	4.70E-01

Ecological Hazard Quotients for the Arctic Fox Exposed to Constituents of Interest in the CAM-D (Continued)

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	5.21E-06	1.04E-05	6.54E-06	1.31E-05	1.30E-04	2.61E-04	--	--	2.84E-04
Inorganics										
Arsenic	9.28E-01	1.54E-03	1.66E-03	3.63E-04	3.92E-04	1.09E-04	1.17E-04	--	--	2.16E-03
Cadmium	8.53E+00	6.51E-04	7.63E-05	4.36E-03	5.11E-04	5.61E-03	6.57E-04	--	--	1.24E-03
Chromium (Total)	3.68E+00	5.34E-02	1.45E-02	8.35E-02	2.27E-02	4.28E-02	1.16E-02	--	--	4.88E-02
Cobalt	1.13E+01	1.74E-02	1.54E-03	9.08E-03	8.03E-04	4.13E-03	3.65E-04	--	--	2.71E-03
Copper	1.38E+01	3.27E-02	2.38E-03	3.74E-02	2.72E-03	1.48E-01	1.08E-02	2.28E-03	1.66E-04	1.60E-02
Lead	6.82E+01	6.51E-03	9.54E-05	2.72E-02	3.99E-04	2.66E-02	3.89E-04	--	--	8.84E-04
Nickel	6.82E+01	5.57E-02	8.16E-04	9.44E-02	1.38E-03	5.44E-02	7.97E-04	--	--	3.00E-03
Zinc	2.73E+02	6.09E-02	2.23E-04	6.83E-01	2.50E-03	1.40E+00	5.14E-03	--	--	7.87E-03

Ecological Hazard Quotients for the Arctic Hare Exposed to Constituents of Interest in the CAM-D (Continued)

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	7.20E-06	1.43E-05	1.31E-04	2.59E-04	2.72E-05	5.38E-05	--	--	3.28E-04
Inorganics										
Arsenic	9.35E-01	2.12E-03	2.27E-03	7.28E-03	7.78E-03	2.26E-05	2.42E-05	--	--	1.01E-02
Cadmium	8.60E+00	9.00E-04	1.05E-04	8.73E-02	1.02E-02	1.17E-03	1.36E-04	--	--	1.04E-02
Chromium (Total)	3.71E+00	7.38E-02	1.99E-02	1.67E+00	4.51E-01	8.92E-03	2.40E-03	--	--	4.73E-01
Cobalt	1.14E+01	2.41E-02	2.12E-03	1.82E-01	1.60E-02	8.59E-04	7.54E-05	--	--	1.82E-02
Copper	1.39E+01	4.52E-02	3.26E-03	7.49E-01	5.40E-02	3.09E-02	2.23E-03	1.25E-03	9.02E-05	5.96E-02
Lead	6.88E+01	9.00E-03	1.31E-04	5.46E-01	7.93E-03	5.53E-03	8.04E-05	--	--	8.14E-03
Nickel	6.88E+01	7.70E-02	1.12E-03	1.89E+00	2.75E-02	1.13E-02	1.65E-04	--	--	2.88E-02
Zinc	2.75E+02	8.42E-02	3.06E-04	1.37E+01	4.97E-02	2.92E-01	1.06E-03	--	--	5.11E-02



Ecological Hazard Quotients for the Ermine Exposed to Constituents of Interest in the CAM-D (Continued)

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	5.69E-06	8.93E-06	1.76E-06	2.76E-06	6.45E-04	1.01E-03	--	--	1.02E-03
Inorganics										
Arsenic	1.18E+00	1.68E-03	1.42E-03	2.13E-04	1.81E-04	5.37E-04	4.55E-04	--	--	2.06E-03
Cadmium	1.09E+01	7.11E-04	6.55E-05	3.75E-03	3.46E-04	2.77E-02	2.55E-03	--	--	2.97E-03
Chromium (Total)	4.68E+00	5.83E-02	1.25E-02	7.33E-03	1.56E-03	2.12E-01	4.52E-02	--	--	5.92E-02
Cobalt	1.44E+01	1.91E-02	1.33E-03	1.29E-03	8.94E-05	2.04E-02	1.42E-03	--	--	2.83E-03
Copper	1.75E+01	3.57E-02	2.04E-03	9.83E-03	5.62E-04	7.34E-01	4.19E-02	1.85E-03	1.06E-04	4.46E-02
Lead	8.69E+01	7.11E-03	8.19E-05	2.32E-03	2.67E-05	1.31E-01	1.51E-03	--	--	1.62E-03
Nickel	8.69E+01	6.09E-02	7.01E-04	1.17E-02	1.35E-04	2.69E-01	3.10E-03	--	--	3.93E-03
Zinc	3.47E+02	6.66E-02	1.92E-04	2.38E-01	6.84E-04	6.94E+00	2.00E-02	--	--	2.09E-02

Ecological Hazard Quotients for the Snowy Owl Exposed to Constituents of Interest in the CAM-D (Continued)

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	4.40E-06	2.12E-06	4.16E-04	2.00E-04	--	--	2.02E-04
Inorganics								
Arsenic	1.48E+01	1.30E-03	8.75E-05	3.47E-04	2.34E-05	--	--	1.11E-04
Cadmium	2.31E+01	5.50E-04	2.38E-05	1.79E-02	7.75E-04	--	--	7.99E-04
Chromium (Total)	5.52E+00	4.51E-02	8.17E-03	1.37E-01	2.48E-02	--	--	3.29E-02
Cobalt	3.77E+00	1.47E-02	3.90E-03	1.32E-02	3.49E-03	--	--	7.39E-03
Copper	1.09E+02	2.76E-02	2.54E-04	4.73E-01	4.36E-03	1.25E-03	1.15E-05	4.62E-03
Lead	1.91E+01	5.50E-03	2.88E-04	8.48E-02	4.45E-03	--	--	4.74E-03
Nickel	1.24E+02	4.71E-02	3.81E-04	1.73E-01	1.40E-03	--	--	1.79E-03
Zinc	1.35E+02	5.15E-02	3.81E-04	4.48E+00	3.32E-02	--	--	3.36E-02

Ecological Hazard Quotients for the Collared Lemming Exposed to Constituents of Interest in the CAM-D (Continued)

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	2.12E-05	3.32E-05	4.02E-04	6.30E-04	--	--	6.63E-04
Inorganics								
Arsenic	1.18E+00	6.25E-03	5.28E-03	2.24E-02	1.89E-02	--	--	2.42E-02
Cadmium	1.09E+01	2.65E-03	2.43E-04	2.68E-01	2.46E-02	--	--	2.49E-02
Chromium (Total)	4.70E+00	2.17E-01	4.62E-02	5.14E+00	1.09E+00	--	--	1.14E+00
Cobalt	1.44E+01	7.10E-02	4.92E-03	5.59E-01	3.87E-02	--	--	4.37E-02
Copper	1.76E+01	1.33E-01	7.57E-03	2.30E+00	1.31E-01	3.69E-03	2.10E-04	1.39E-01
Lead	8.71E+01	2.65E-02	3.04E-04	1.68E+00	1.92E-02	--	--	1.96E-02
Nickel	8.71E+01	2.27E-01	2.60E-03	5.81E+00	6.67E-02	--	--	6.93E-02
Zinc	3.48E+02	2.48E-01	7.11E-04	4.20E+01	1.21E-01	--	--	1.21E-01