GAHCHO KUÉ PROJECT

ENVIRONMENTAL IMPACT STATEMENT

HUMAN HEALTH RISK ASSESSMENT

October 2012 **11-1365-0012**

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1 INTRODUCTION

The proposed Gahcho Kué Project (Project) is a diamond mine located at Kennady Lake, which is north of the north-eastern arm of Great Slave Lake. The Project is situated in a remote location with no road access and no utilities. The site is about 280 kilometres (km) northeast of Yellowknife and 140 km north-northeast of the First Nation community of Łutselk'e. Figure 1-1 shows the location of the Project site within the Northwest Territories (NWT).

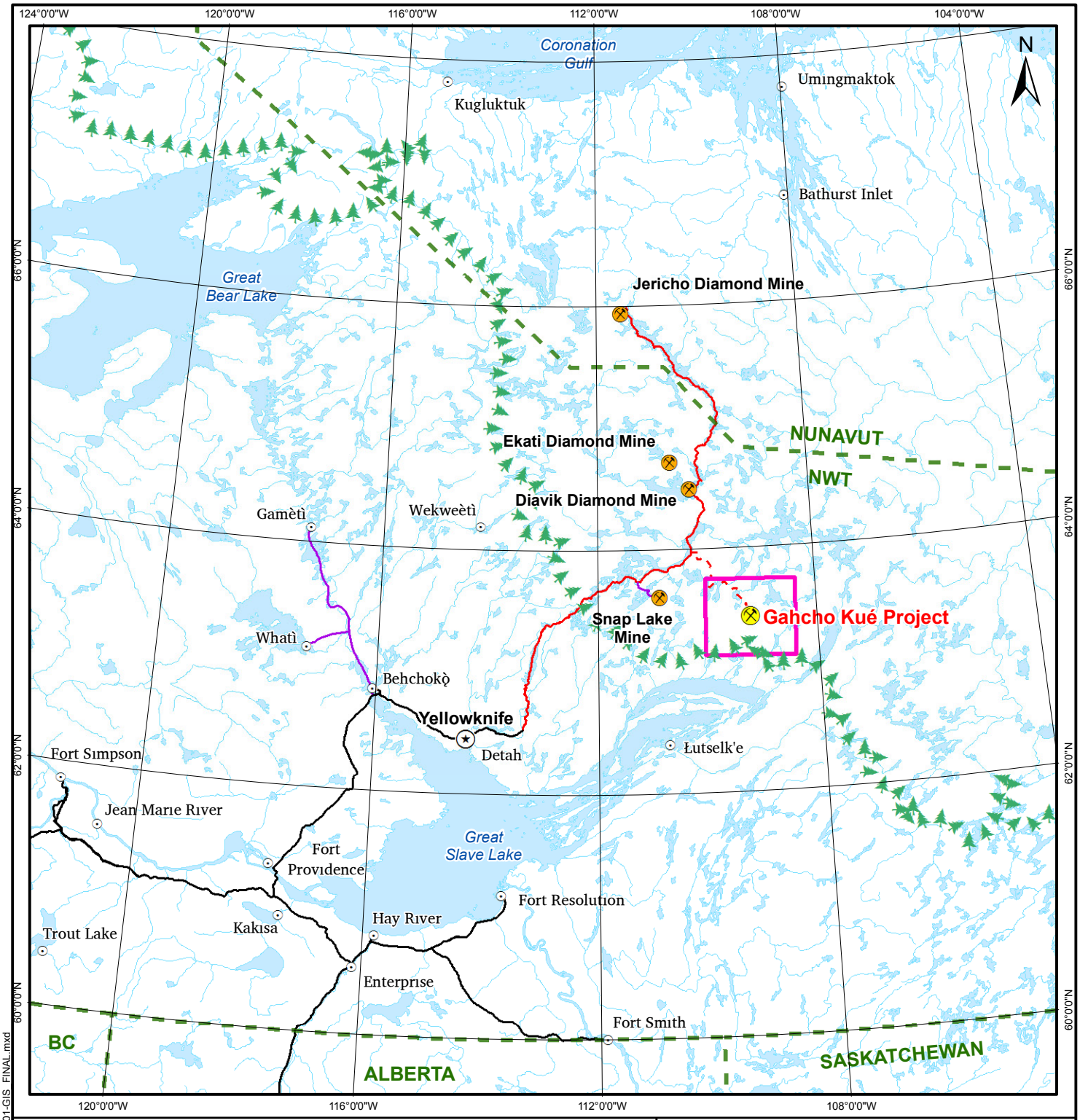
1.1 PROJECT SUMMARY

1.1.1 Site Infrastructure

No mining services are currently available at the Project site. The necessary mining infrastructure will be established on the site before the start of mining. The following major infrastructure will be required:

- processing plant;
- accommodations complex and administrative offices;
- maintenance complex and warehouse;
- electrical power and heating;
- storage for oil, fuel, and glycol;
- production and storage of explosives;
- winter access road;
- site roads;
- traffic management;
- airstrip; and
- sewage treatment.

Most of the Project infrastructure will be placed in a compact footprint as shown in Figure 1.1-1. The airstrip will be located southeast of the plant site, and the ammonium nitrate storage areas, emulsion plant, and explosives storage magazines are sited to the north and northeast of the plant site, with separation distances in accordance with the guidelines set out in the *Quantity-Distance Principles User's Manual* published by the Explosives Regulatory Division of Natural Resources Canada.



LEGEND

- Gahcho Kué Project
- Existing Mine
- Territorial Capital
- Populated Place
- Highway
- Existing Winter Road
- Tibbitt-to-Contwoyto Winter Road
- Winter Access Road
- Watercourse
- Waterbody
- Territorial/Provincial Boundary
- Treeline
- Regional Study Area

NOTES

Base data source: The Atlas of Canada

GAHCHO KUÉ PROJECT

Location of the Gahcho Kué Project

PROJECTION: Canadian Lambert Conf. Conic	DATUM: NAD83	
Scale: 1:5,000,000 (100 50 0 100 Kilometres)		
FILE No: B2011-Wild-001-GIS		DATE: December 12, 2011
JOB NO: 11-1365-0001	REVISION NO: 1	
OFFICE: GOLD-CAL	DRAWN: DC	CHECK: JV

De Beers Canada

Figure 1-1

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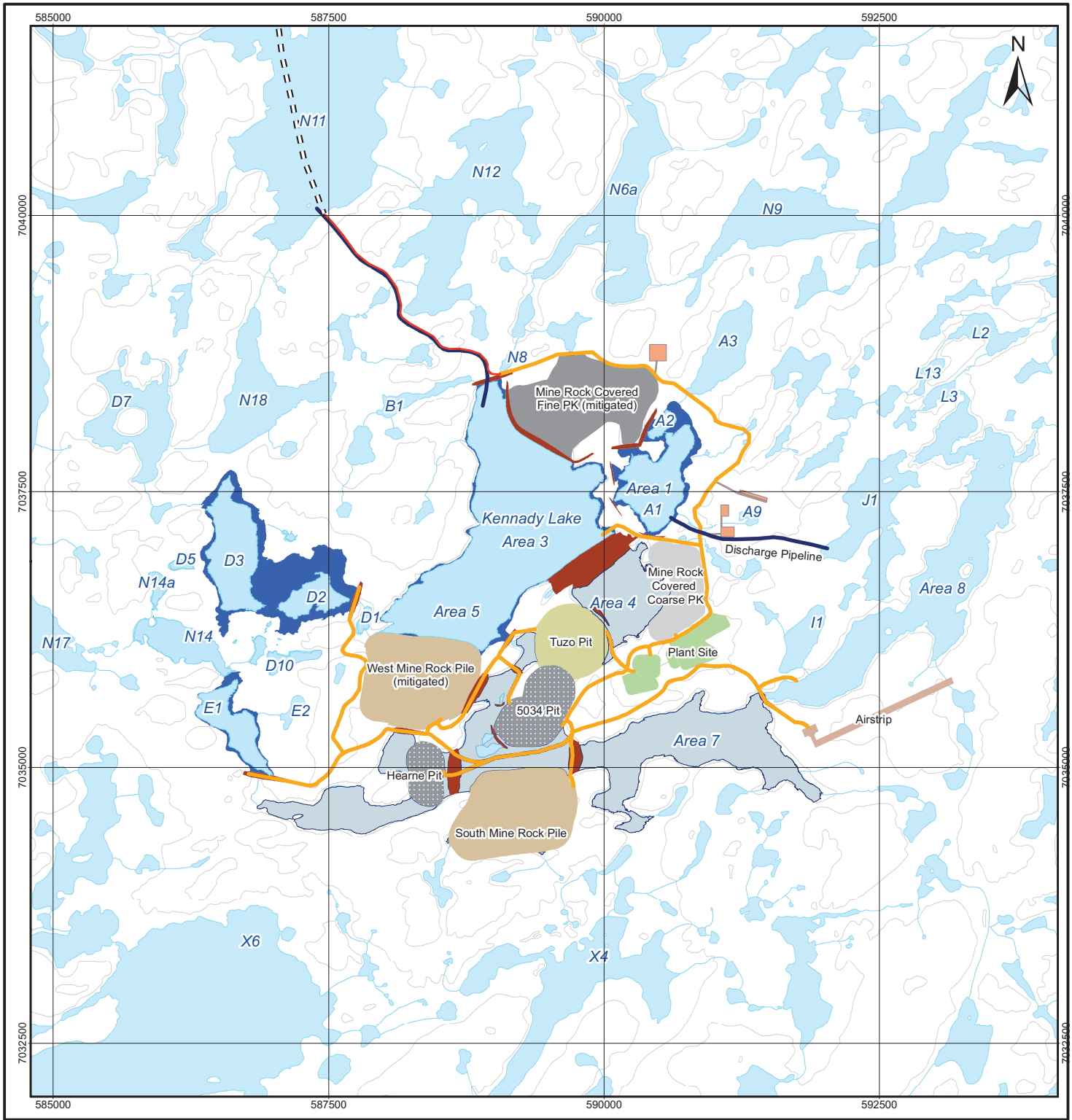

LEGEND

- Waterbody
- Watercourse
- N12 Lake Identifier
- Contour (10m interval)
- = = Proposed Winter Access Road

Project Footprint

- Service Road
- Site Road
- Water Pipeline
- Airstrip
- Back-filled Pit
- Building
- Coarse PK Pile
- De-watered Lake Bed
- Dyke or Berm
- Fine PK Containment Facility
- Flooded Area
- Mine Rock Pile
- Open Pit
- Plant Site
- Water

NOTES
Source: adapted from Figure 1.3-2 of De Beers 2010
Base data source: National Topographic Base Data (NTDB) 1:50,000
PK = Processed Kimberlite

GAHCHO KUÉ PROJECT

Revised Project Footprint

PROJECTION: UTM Zone 12	DATUM: NAD83	
Scale: 1:50,000 (500 250 0 500 Metres)		
FILE No: E2011-Perm-001-GIS		DATE: February 7. 2012
JOB NO: 11-1365-0001	REVISION NO: 1	Figure 1.1-1
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De Beers Canada

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1.1.2 Mining and Processing

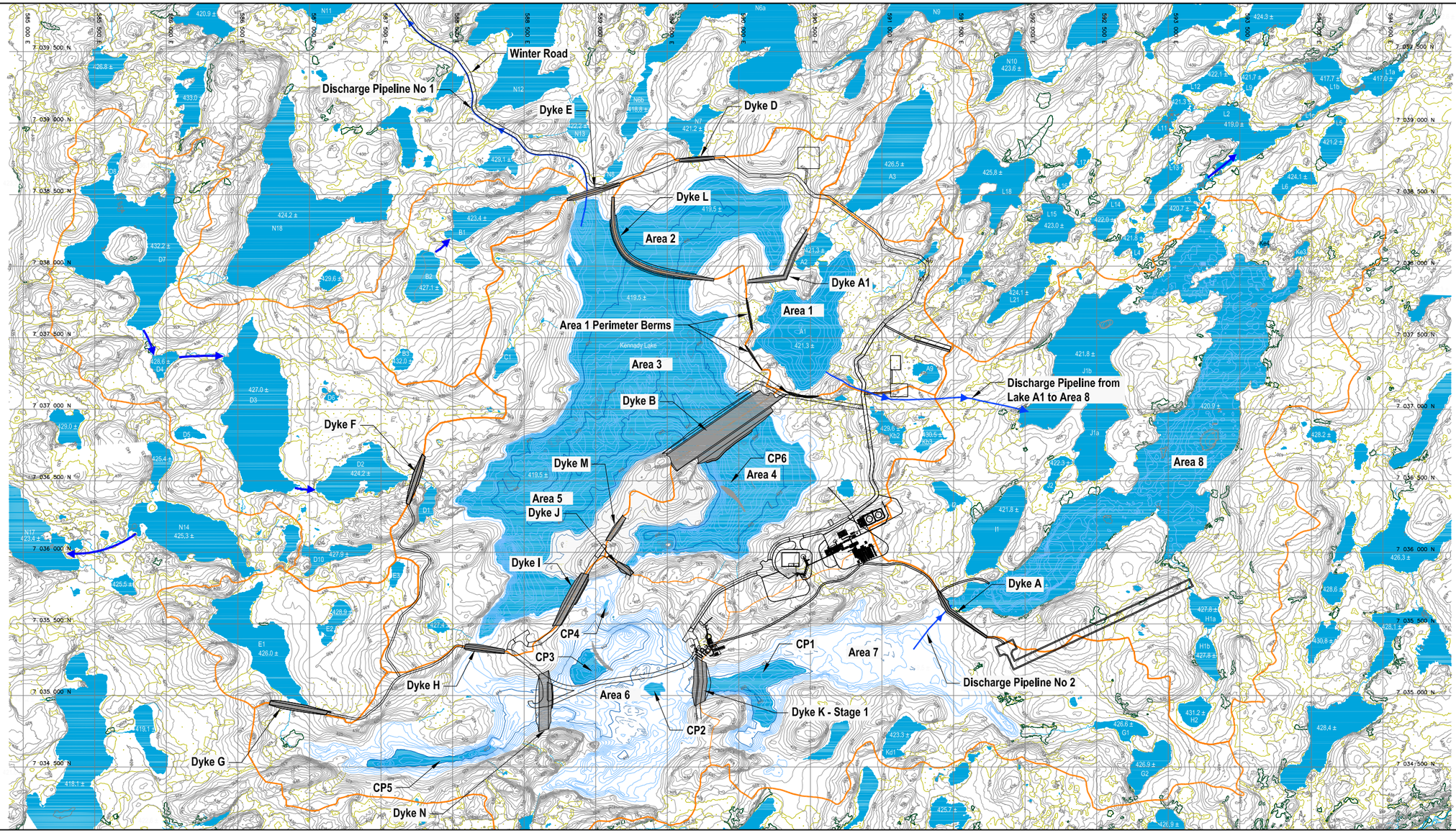
The diamond-bearing kimberlite occurs in vertical pipes located mainly beneath Kennady Lake. Ore from three ore bodies (5034, Hearne, and Tuzo) will be extracted by open pit mining (Figure 1.1-1). Pit closures, including backfilling the 5034 and Hearne pits, will occur progressively as each pit is mined out.

Kimberlite extracted from the mine will be processed on-site. The process plant will be designed to process the 3.0 million tonnes (Mt) of kimberlite per year produced by the mine. Kimberlite ore will be crushed, cleaned, and screened to a specific size range. Then the ore will be mixed with ferrosilicon and water, and diamonds will be separated using a difference in density. In the recovery plant, x-ray machines and a grease diamond recovery system will separate diamonds from the concentrate.

1.1.3 Water Management

Water management is a key component of the Project because the diamond bearing kimberlite pipes are mainly located under Kennady Lake. The Project footprint created by the Water Management Plan will consist of eight major sub-watershed areas: Area 1 is located northeast of Kennady Lake and includes Lakes A1, A2, A3, and A9, while Areas 2 to 8 are within Kennady Lake (Figure 1.1-2). Areas 2 to 7 will form the controlled area for water management purposes. Area 8 is a sub-watershed of Kennady Lake, but it is outside the controlled area boundary. The objective of the dewatering program will be to dewater Areas 2 to 7 of Kennady Lake to the maximum extent possible to safely access and mine the ore bodies. After the initial dewatering, Areas 6 and 7 will be isolated and drained completely into Areas 2 to 5.

Before dewatering can take place, Areas 2 to 7 will be isolated. Various dykes will be built to both divert the upper watersheds from Kennady Lake and close the outlet of Area 7. The isolation of Areas 2 to 7 establishes the controlled area, which will retain water affected by the Project (Section 3.9.2 of the 2012 EIS Supplemental [De Beers 2012a]). A critical activity during the initial construction will be the construction of Dyke A at the narrows separating Area 7 and Area 8. Area 8 represents the eastern section of Kennady Lake that will remain at the existing lake elevation (Figure 1.1-2). As the level of water in Areas 2 to 7 decreases, the sills separating the northwest portions of the lake (Areas 2 to 5) from the areas above the 5034 and Hearne ore bodies (Areas 6 and 7) will be exposed. Internal water retention dykes will be constructed isolating the northern portion of the lake (Area 2 to 5) from the southern portion of the lake (Areas 6 and 7), effectively splitting the partially dewatered lake into two major sections and allowing the complete drainage of the remaining water from Areas 6 and 7 into the northern part of the basin.



LEGEND

- Existing Ground Contours, 5m Index - 1m Intermediate
- Bathymetry Contours, 5m Index - 1m Intermediate
- CP — Collection Pond
- Winter Road
- Marsh Area
- Scrub
- Sub-watershed Boundary
- Drainage Flow Direction
- Discharge Pipeline
- Lake/Pond

NOTES

Base data source: EBA Figure 4.2 - Stage 1 - Initial Lake Dewatering (June-July, 2013)
Source: Adapted from Figure 3.9-1 of De Beers 2010

GAHCHO KUÉ PROJECT

Water Management Areas, Dykes, Collection Ponds, and Lakes Associated with the Project

PROJECTION: UTM Zone 12	DATUM: NAD83	De Beers Canada
SCALE: 500 0 500 METRES		
FILE No: P2011-Other-001-CAD_Figure 1.1-2		DATE: March 3, 2012
JOB No: 11-1365-0001	REVISION No: 0	Figure 1.1-2
OFFICE: GOLD - SAS	DRAWN: TAH	CHECK:

G:\2011\1365\11-1365-0001 Gahcho Kue EIS Post-Submission\Mapping\MXD\Other\P2011-Other-001-CAD_Figure 1.1-2.dwg Aug 30, 2012 - 2:56pm

During the first phase of dewatering, the lake water would be pumped via pipelines to two principal locations simultaneously:

- Area 8 of Kennady Lake, which is the natural outlet for Kennady Lake; and
- Lake N11 in the N watershed (Figure 1.1-2).

Later, as the water level in Kennady Lake is lowered, sediment from the lake bottom could become suspended due to wave action on the exposed shorelines. Areas 2, 3, and 5 will be dewatered to the maximum extent possible, before suspension of lake-bottom sediments result in TSS levels in Areas 2, 3, and 5 that are too high to discharge to Lake N11. Lake dewatering discharge will be sampled regularly to monitor for compliance with TSS discharge limits to be specified by the Mackenzie Valley Land and Water Board in the water license. Monitoring data will be used to identify the water level in the lake needed to minimize the suspension of lake-bottom suspended solids.

As the water level decreases, the sills separating the northwest portions of the lake (Areas 2 to 5) from the areas containing the 5034 and Hearne ore bodies (Areas 6 and 7) would be exposed. Construction of small dykes at these points will separate Areas 6 and 7 from the remainder of the basin and allow the complete drainage of the remaining water from Areas 6 and 7 into the northern part of the basin consisting of Areas 2 to 5. Areas 3 and 5 will serve as the water management pond (WMP) for the Project. If necessary, water in Areas 6 and 7 will be treated in-line as it is pumped to the WMP (Areas 3 and 5) for flocculation and settled in the WMP before being subsequently discharged to Lake N11. Between Year 4 and Year 6, a dyke will be constructed allowing the area near the Tuzo Pit to be dewatered so that the Tuzo Pit can be mined.

During operations, groundwater will flow into the open pits; however, to allow uninterrupted mining all water entering the active open pits will be transferred to the WMP.

At the completion of mine operations, the Hearne Pit will have been partially backfilled with fine PK; the 5034 Pit will be backfilled with fine PK and mine rock, while the Tuzo Pit will be open and empty. Area 2 will be filled with fine PK and reclaimed with a cover layer that will be comprised of mine rock, and coarse PK depending on material availability. The water elevation in Areas 3 and 5 at the end of operations is expected to be approximately 422.0 m; however, Area 4 will be drained, as this area is adjacent to the Tuzo Pit. Area 7 will have been filled to a water elevation of 420.3 m with natural runoff water at the end of mine operations. During closure, a large proportion of the water within the controlled area, especially the WMP and Area 6, will be transferred to Tuzo pit in advance

of refilling Kennady Lake. Following closure, the temporary diversion dykes will be removed to restore the upper A, B, D, and E watershed boundaries of Kennady Lake. These watersheds will be returned to their natural drainage patterns. Natural runoff into the watershed and supplemental pumping from Lake N11 will be used to refill Kennady Lake and all pits. The estimated time required to refill Kennady Lake back to the original levels is eight years.

1.1.3.1 Water for Potable Consumption

Fresh water for potable consumption will be drawn from Area 8. About 60,000 cubic metres per year (m^3/y) of fresh water will be required for potable water during construction. During operations, with a smaller workforce, the potable water required will decrease to about 27,000 m^3/y.

The freshwater intake and pumphouse will be located on the north western shore of Area 8. The intake will consist of vertical filtration wells fitted with vertical turbine pumps that supply water on demand. The intake will be connected to the pumphouse with piping buried under a rockfill embankment. The embankment will act as a secondary screen to prevent fish from becoming entrained.

Fresh water will be pumped through an overland pipeline to the freshwater storage tank in the accommodations complex, and will be chlorinated before distribution as potable water. The freshwater pipe will be insulated and heat-traced. Potable water will be monitored according to NWT health regulations for total and residual chlorine and microbiological parameters. Treated water will be piped to areas in the process plant and truck shop requiring potable water and to the accommodations and service complexes. Insulated pipes will distribute water through the utilidors between the plant, service complex, and the camp. Potable water will be trucked to washrooms in satellite areas as needed.

1.1.4 Waste Management

Five major types of waste will be produced and managed on-site:

- lake-bed sediment and overburden from pre-stripping;
- mine rock (rock, primarily granite, surrounding the kimberlite ore body) that has been excavated from the open pit mines;
- barren (non-diamondiferous) kimberlite rock;
- kimberlite that has been processed to remove diamonds; and
- general waste (domestic, industrial, hazardous materials, and sewage) waste produced as part of normal Project operations.

An estimated 226.4 Mt of mine rock and 3.3 million cubic metres (Mm^3) of overburden will be produced during the operational phase of the Project. Overburden will be used for constructing dykes, dams, and for re-grading the lake-bed. Excess overburden material will be deposited in the designated areas of the mine rock piles.

Mine rock will be used for construction of roads, dykes, dams, and reclamation of the Coarse Processed Kimberlite (PK) Pile and Fine Processed Kimberlite Containment (PKC) Facility. The mine rock will primarily be composed of granite (95%). Most of the mine rock from the excavation of open pits will be stored in one of the following locations:

- mine rock piles in and adjacent to Area 5 (West Mine Rock Pile) and Area 6 (South Mine Rock Pile); and
- the mined-out 5034 Pit.

Waste management plans are in place to reduce acid rock drainage and metal leaching. Also, geochemical testing of mine rock will occur throughout the operational period (Section 3 of the 2012 EIS Supplement [De Beers 2012a]). Only non-reactive mine rock will be placed on the upper and outer surfaces of the mine rock pile. Standard best practices for management of other types of solid waste will be followed. Food wastes and non-toxic combustible wastes will be burned in approved oil-fired incinerators. Non-combustible items will be placed in the designated landfill area or recycled if practical. Hazardous materials will be sorted in sealed steel or plastic drums in the waste transfer area before being shipped to an approved off-site hazardous waste disposal location.

A modular sewage treatment system adequate for 432 workers will be installed as part of the initial construction. The sewage treatment system will be housed in a building adjacent to the accommodations complex. Treated liquid effluent from the sewage treatment system will be discharged to Area 3 of Kennady Lake initially and then later in operations directed to the process plant for disposal with the fine PK stream. The sewage sludge will be dewatered and disposed of in the landfill on-site. If possible, the sludge may be composted or used as a soil treatment.

1.1.5 Site Access and the Winter Access Road

The site will be accessible by air for mine staff, supplies, and emergency transport. To provide seasonal overland access, a 120 km winter access road will be constructed from Kennady Lake to the north end of MacKay Lake and will intersect the Tibbitt-to-Contwoyto Winter Road at kilometre 271. The winter road

will be in operation from late January or early February through March and, under favourable conditions, into early April.

1.1.6 Project Schedule

Following necessary environmental assessment and regulatory approvals, a construction period will be required to install the infrastructure and to dewater part of Kennady Lake prior to production mining. Construction activities will take place over two years (Year -2 to -1). After the water above the ore bodies has been drained to an acceptable level, pre-stripping of the first open pit and initial production mining will begin (Year 1) will commence after commissioning is complete in the last quarter of construction (Year -1).

The construction period will be followed by an eleven-year operational period (Year 1 to 11), during which the kimberlite will be mined and processed. Most of the site infrastructure will be removed and the Project site decommissioned two years after the completion of mining (i.e., by the end of Year 13, assuming mining is completed by Year 11). Final closure of the site will take place over an extended period (Year 14 to 19). All remaining site infrastructure (e.g., airstrip and reclamation camp) will be removed after the water level in the planned reclamation areas of Kennady Lake has been restored. Monitoring of the Project site will continue after lake refilling until it is shown that the Project site and Kennady Lake meets all regulatory closure objectives.

2 ASSESSMENT APPROACH

2.1 COMPONENT DESCRIPTION

The Human Health Risk Assessment (HHRA) component of the Application presents a stand-alone assessment of the potential effects of chemical emissions on the health of people in the vicinity of the Project.

The HHRA is based on the predicted changes in air and water quality caused by the Project emissions. Indirect effects of Project emissions to soil, vegetation and animal tissues were also evaluated as part of the HHRA. The main sources of exposure for the HHRA are related to air and water emissions; no changes in sediment quality are expected as discussed in the Water Management Plan, Section 8 of the 2011 EIS Update (De Beers 2011). The HHRA is also supported by the following appendices:

- Appendix I: Air Quality Assessment (Screening Criteria and Toxicity Reference Values)
- Appendix II Summary of Chemical Screening and Identification of Chemicals of Potential Concern for Multi-media Assessment;
- Appendix III: Multi-media Toxicity Assessment;
- Appendix IV: Multi-media Exposure Assessment;
- Appendix V: Multi-media Exposure Doses and Risk Estimates; and
- Appendix VI: Particulate Matter Literature Review.

2.2 TERMS OF REFERENCE

The Terms of Reference for the Gahcho Kué Environmental Impact Statement (Gahcho Kué Panel 2007) identified seven key lines of inquiry representing the highest priority issues to be assessed. The key lines of inquiry facilitate a comprehensive analysis of the Project-related issues that engendered significant public concern. The Terms of Reference also identified eighteen subjects of note. Though not considered to have priority equal to the key lines of inquiry, the subjects of note are still important and require consideration in the HHRA.

This HHRA was completed in accordance with review of the Report of Environmental Assessment by the Mackenzie Valley Environmental Impact Review Board (MVEIRB 2006) and the Terms of Reference (Gahcho Kué Panel 2007).

Terms of Reference specific to the HHRA are listed in Table 2.2-1.

The HHRA addressed possible short-term and long-term effects to human health from potential exposure during the construction and operations periods of the Project. Effects to human health during and post Project closure will be assessed as part of the Closure Plan.

Table 2.2-1 Terms of Reference Addressed by the Environmental Health Assessment

TOR Section	Environmental Assessment or Topic	Location TOR Addressed
4.1.2 Water Quality and Fish in Kennady Lake		
	Possible fish contamination and wildlife and human health effects from contaminated fish consumption, including pathways and long and short term exposure levels and health effects of toxic exposure levels on wildlife and humans	Section 5.4 Chronic Multi-media Risk Assessment Methods Section 7 Chronic Multi-media Risk Assessment Results
5.2.2 Air Quality		
	The air quality assessment must include an assessment of risk to human health, including worker camps.	Section 5.2 Air Quality Assessment Methods Section 5.3 Particulate Matter Assessment Methods Section 5.4 Chronic Multi-media Risk Assessment Methods Section 5.5 Acute Air Quality Risk Assessment Results Section 5.6 Chronic Air Quality Risk Assessment Results Section 6 Particulate Matter Assessment Results Section 7 Chronic Multi-media Risk Assessment Results

2.3 CONSULTATION AND ASSESSMENT FOCUS

Aboriginal communities, regulators and the public have raised concerns related to human health from development in the regional study area (RSA). Based on these concerns, the following issues relevant to the human risk assessment were identified:

- the effect of both the Project and the cumulative environmental effects of development in the region on human health;
- the effect of the Project and the cumulative environmental effects of development in the region on surface water and groundwater quality;
- the effects of the Project and the cumulative environmental effects of development in the region on air quality; and
- the effects of the Project and the cumulative environmental effects in quality of country foods (i.e., plants, fish and wildlife) for human consumption.

Further discussion about the information received through the consultation process can be found in Section 12 of the 2010 EIS (De Beers 2010).

2.4 ASSESSMENT METHODOLOGY

2.4.1 General Methodology

The HHRA presents an assessment of the potential effects of chemical emissions from the Project on the health of people. Each potential linkage between environmental changes and human health was evaluated qualitatively to determine its validity based on specific activities of the Project and their likelihood to adversely affect human health. Next, an assessment was carried out for each valid linkage to assess whether activities associated with the Project might adversely affect human health.

The HHRA is comprised of three components:

- an air quality risk assessment that evaluates the acute and chronic effects associated with certain airborne or gaseous substances (i.e., only present in air);
- a qualitative particulate matter assessment; and
- a multi-media assessment to evaluate the chronic effects associated with contaminants that might be present in air, soil, water and food.

The HHRA quantifies the potential health risks to people from baseline (present-day) and Application (i.e., Project) case (predicted using modeling) environmental quality in the Project area. The framework of risk assessment provides a structured and clear approach for evaluating potential adverse effects to receptors (e.g., humans) from environmental stressors (e.g., metals in soil). Health risks were evaluated using the Baseline and predicted Application case quality of water, soil, sediment, air and food (including fish, game and wild vegetation). The Baseline scenario provides context for understanding the incremental effects predicted for the Project.

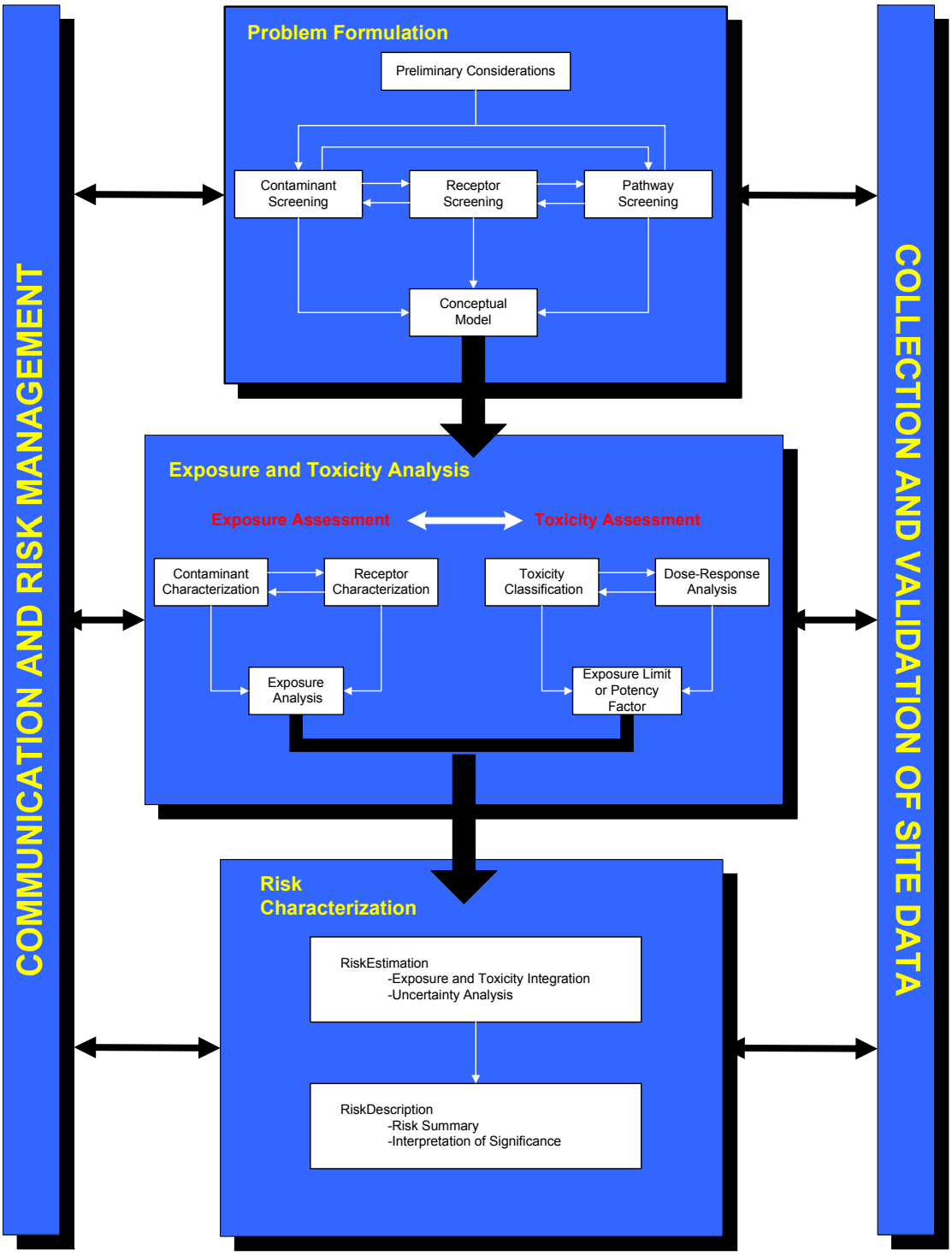
The methodology used in this HHRA is consistent with guidance provided by Health Canada (2009a,b), the Canadian Council of Ministers of the Environment (CCME 2006), the World Health Organization (WHO 1999), and the United States Environmental Protection Agency (U.S. EPA 1989, 2004, 2009).

The overall human health risk assessment framework is presented in Figure 2.4-1 and the main HHRA components are described below:

- **Problem Formulation**. This stage develops a focused understanding of how environmental quality might affect the health of people near the proposed Project site. The problem formulation step identifies the receptors (e.g., sub-populations of people), contaminants (e.g., metals), and exposure pathways (e.g., direct contact with soil) that are of greatest concern. If no unacceptable risks are predicted for the receptors, contaminants, and exposure pathways of greatest concern (i.e., the worst-case scenarios), it is highly unlikely that unacceptable risks would occur for other scenarios.
- **Exposure Assessment**. The exposure assessment step characterizes the degree to which receptors are exposed to contaminants of concern via the identified exposure pathways. For human receptors, exposure is calculated for most contaminants as a total daily dose (i.e., amount taken in per day) from all relevant pathways in a multi-media evaluation (e.g., inhalation, ingestion of drinking water and dietary items, direct contact with soil, sediment and surface water). Background dietary exposure (e.g., from supermarket foods) was also included. Exposure to certain airborne or gaseous substances (e.g., nitrogen dioxide) only occurs via the inhalation pathway, and therefore is expressed as the concentrations of these contaminants in air.
- **Toxicity Assessment**. The toxicity assessment step involves the determination of exposures that are considered to be 'safe', or associated with an acceptably low level of risk of adverse effects. Toxicity is an inherent property of a substance, which is brought about by the physical-chemical properties of the substance and its chemical reactivity within living organisms.

  Toxicity assessment in this context involves identification of the potential toxic effects of contaminants, and determination of the rate of intake of contaminants that can be tolerated over a lifetime without experiencing adverse health effects. The toxicity assessment also considered the following concepts:
  - non-carcinogens (contaminants that do not cause cancer);
  - carcinogens (contaminants that have the potential to cause cancer); and
  - bioavailability (the proportion of contaminant in a medium that is considered to be available for uptake by a human after the human contacts the medium).
- **Risk Characterization**. The potential for adverse effects to occur is assessed by comparing the estimated exposures (from the Exposure Assessment) with those exposures that are determined to be acceptable (from the Toxicity Assessment). The characterization of risk always includes an explicit consideration of uncertainty and conservatism in all elements of the risk assessment.

Figure 2.4-1 Human Health Risk Assessment Framework



Source: Health Canada (1995).

To estimate and characterize health risks for non-carcinogenic compounds, the predicted contaminant exposure (daily dose) for a specific receptor was compared to a toxicity reference value (TRV, also known as tolerable daily intake [TDI]; or reference dose, [RfD]), which signifies a safe level of long-term exposure. The ratio of the predicted exposure to the TRV is the hazard quotient (HQ):

$$HQ = \frac{D_{total}}{TRV}$$

Where:

HQ = hazard quotient (unitless)
D_{total} = estimated total dose (milligrams per kilogram per day [mg/kg-day])
TRV = toxicity reference value from literature (mg/kg-day)

Hazard quotients are calculated for individual exposure pathways when there are pathway-specific TRVs, although where both oral and dermal exposures occur, the HQ is typically calculated for the summed exposure for both pathways. A hazard index (HI) is the sum of the HQ for all exposure pathways for each contaminant or the sum of HQs for chemicals which act on the same organ systems or toxicological endpoints. The units of mg/kg-day refer to milligrams of substance per kilogram of body weight per day. These units are used for assessment of doses received by receptors on a long-term basis, and for establishment of TRVs.

To provide a framework for interpretation of the calculated risk levels and of the corresponding uncertainty associated with these risk estimates, the following categories were used to describe the risk magnitudes for non-carcinogenic compounds:

- **Negligible risk:** HI less than or equal to 1. This is accepted common practice for a multi-media risk assessment (Health Canada 2009a).
- **Low risk and likely to be negligible:** HI greater than 1 but not more than 10. This categorization is generally true but should be reviewed on contaminant-specific basis as the conservatism of the analysis is dependent on the uncertainty factor(s) used to derive the toxicity reference value that drive the HI value.
- **Potentially elevated risk:** HI greater than 10.

The above categorizations are not intended to be highly precise; for example, an HI value of 9 is not qualitatively different from an HI of 11, provided that the

conservatism of assumptions is similar. The purpose of the above categorizations is to provide a simple and transparent means for grouping risks of similar magnitude, and for eliminating pathways that clearly present negligible risk.

For carcinogenic compounds, a different calculation procedure is applied in recognition that there may not be any threshold or "safe" dose, rather only a continuum of risk with increased exposure level. Carcinogenic risk is the product of the dose of carcinogen received by the receptor and the "potency" of the carcinogen, or ability to cause cancer. The carcinogenic risk is presented as the estimated frequency of incidence in the population (e.g., one incidence per million people equals 1.0×10^{-6}) for the effect under consideration.

To calculate cancer risk, the estimated lifetime-averaged daily dose (LADD) is multiplied by the appropriate cancer slope factor to derive a conservative estimate of the potential incremental lifetime cancer risk (ILCR) associated with the modeled scenario. Based on the conservative assumptions used in this assessment, the magnitude of the cancer risk is characterized as follows:

- **Negligible risk**: no change from baseline case or ILCR less than or equal to 1.0×10^{-5} (also expressed as 1.0E-05) – equivalent to one incidence or less per 100,000 individuals (Health Canada 2009a).
- **Low risk and likely to be negligible**: ILCR greater than 1.0×10^{-5} but not greater than 1.0×10^{-4} (also expressed as ILCR greater than 1.0E-05 but not greater than 1.0E-04) – equivalent to an incidence rate between one per ten thousand [10,000] individuals and one per hundred-thousand [100,000] individuals).
- **Potentially elevated risk**: ILCR greater than 1.0×10^{-4} (also expressed as 1.0E-04) – equivalent to incidence rate greater than one per ten thousand [10,000] individuals).

The risk estimates derived for human health already incorporate exposure duration, frequency and geographic extent as these factors are input parameters in the calculations of exposure to the chemicals of concern (COC). The direction of all effects identified in the HHRA is negative. Thus, for the HHRA, the magnitude of risk and resulting consequence to human health are defined by the risk estimates.

Situations may occur where, even though an HQ exceeds 1 or an ILCR value exceeds 1.0×10^{-5}, the magnitude of risk after further evaluation will be considered to be negligible (e.g., exceedance of an acute threshold for a few hours per year at the boundary of the Project [Project Boundary]). In these

situations, the rationale for concluding a negligible effect is discussed on a chemical by chemical and location by location basis. In other words, the characterization of risk includes more than assessment of the absolute magnitude of HQ or ILCR, but also the uncertainty and conservatism in the risk assessment procedure, risks relative to background, and other contextual information.

Factors considered in risk characterization include:

- determination of the Project sources, exposure medium(s) and pathway(s) that contribute most significantly to the potential risk;
- comparison of Application Case concentrations to Baseline Case concentrations for the relevant exposure medium(s);
- evaluation of the conservatism in the modelling approaches used to predict future concentrations in the relevant exposure medium(s);
- evaluation of the conservatism in the exposure assumptions used for the relevant exposure pathway(s);
- evaluation of the conservatism in the toxicity reference value for that parameter; and
- evaluation of the potential chronic health effects that may occur at the predicted concentrations.

2.4.2 Assessment Cases

The two development scenarios addressed in the HHRA are the Baseline Case and the Application Case. The Baseline Case describes the environmental conditions that include the effects resulting from existing and approved projects or activities. The Application Case describes the Baseline Case with the effects of the Project added.

Quantitative exposure and risk predictions were not estimated for the closure and reclamation phase.

2.4.3 Temporal Considerations

The HHRA considers reasonable worst-case potential emissions through the construction and operations stages of the Project. The proposed schedule for development of the major components of the Project is summarized in Section 1.1.6. The HHRA evaluated both long-term (chronic) and short-term (acute) effects of chemical exposures on human health.

For the long-term assessment it was assumed that Seasonal Users spent six months of each year for their entire life (i.e., up to 80 years) within the RSA defined as part of the air quality assessment (Figure 2.4-2), rather than only for the length of the Project. However, the contribution of chemical emissions to existing or natural conditions (e.g., plants and soil) in the region was assumed to occur only for the life of the Project (i.e., 11 years). Exposure durations of 24 hours or less were evaluated in the short-term (acute) air quality exposure assessment and in the particulate matter assessment.

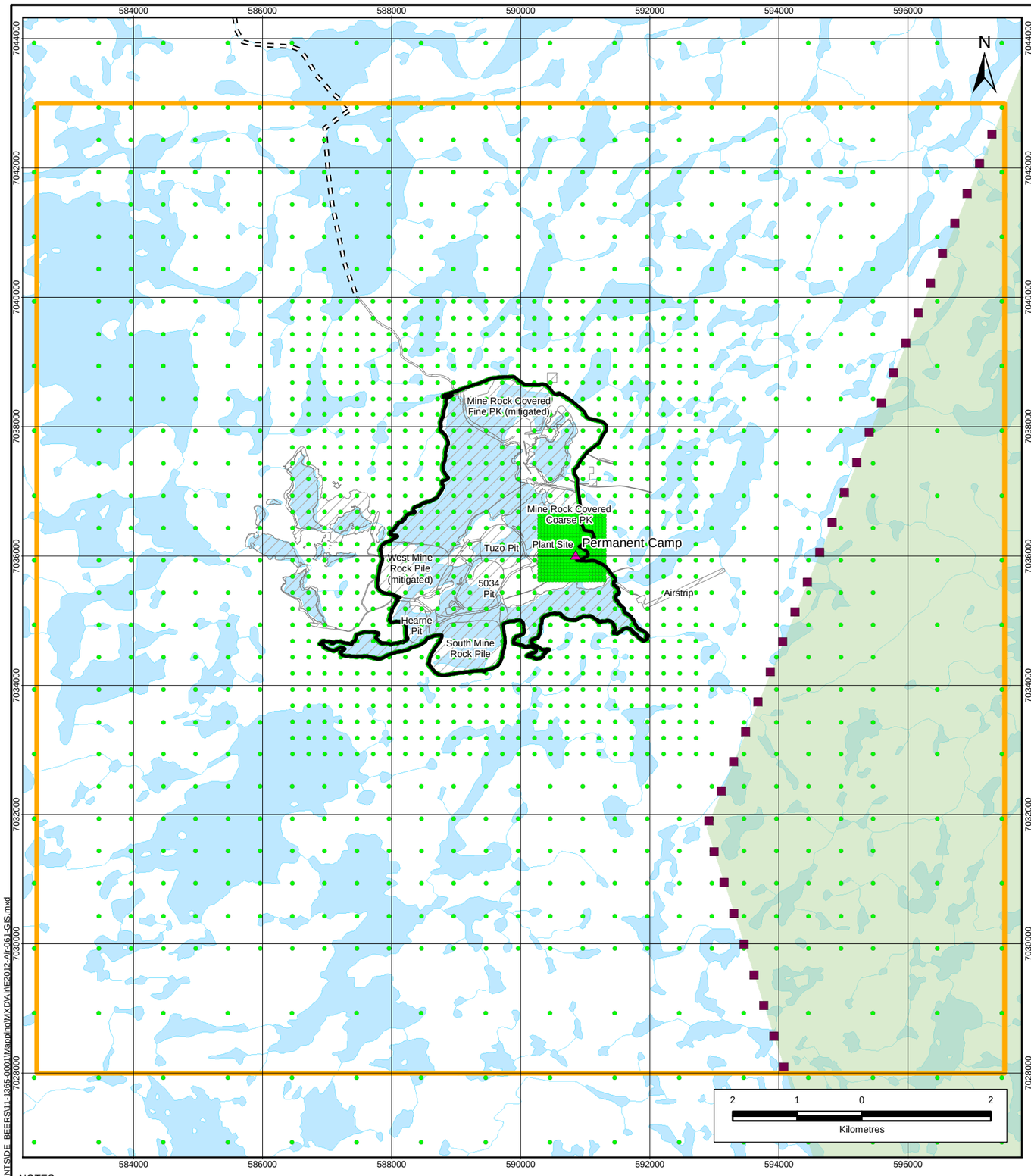
The human health RSA for the Project is defined by an 80 km by 160 km area. The RSA was selected to capture air quality cumulative effects associated with emissions from existing and approved industrial sources within the region in combination with the proposed Project. The human health RSA is the same as the RSA used in the air quality assessment in order to include the locations where humans could come into contact with air emissions from the project. The air quality RSA includes the water quality LSA within in its boundaries; the air quality RSA is larger than that the LSA for the water quality assessment, which is the other primary media through which humans could come into contact through Project-related emissions. Potential Project-related effects to water quality are not expected to extend beyond the LSA, which represents the Kennady Lake watershed to the outlet of Kirk Lake.

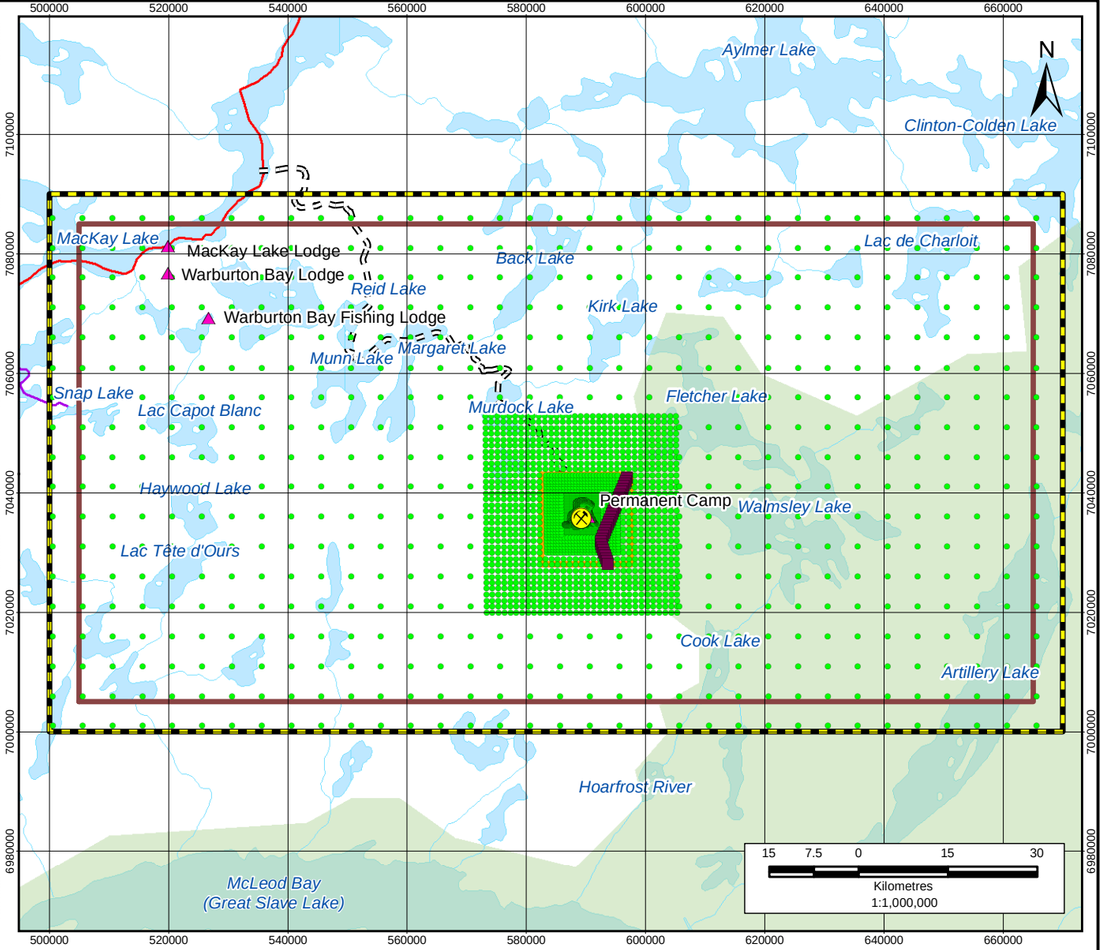
2.4.4 Spatial Considerations

Effects to human health were evaluated on a local and regional basis. Locations for assessment were selected based on areas where receptors are known to be present (i.e., communities, recreational areas, hunter/trapper cabins and worker camps). In October 2006, a Memorandum of Understanding (MOU) was signed between the Łutselk'e Dene First Nation (LKDFN) and the Canada Parks Agency that formally launched a feasibility study for a proposed national park at the East Arm of Great Slave Lake. There are no Aboriginal communities present within the RSA. The receptor locations included in this HHRA are identified below and are also presented on Figure 2.4-2:

- Gahcho Kué Worker Camp;
- Gahcho Kué Project Boundary;
- Warburton Bay Lodge;
- Warburton Bay Fishing Lodge;
- MacKay Lake Lodge; and
- Proposed National Park Boundary.

All receptor locations were assessed for the air quality and particulate matter assessments. Only the most exposed receptors, Aboriginal Seasonal Users and Gahcho Kué Workers were assessed in the multi-media risk assessment. Seasonal Users are Aboriginal people who live in communities outside the RSA but spend time (six months) within the local study area (LSA) and/or RSA throughout the year while pursuing traditional lifestyle activities (hunting, fishing, and gathering of traditional foods) and therefore may be exposed to air, soil, water and food items impacted by the Project.





LEGEND

- Gahcho Kué Project
- Existing Winter Road
- Tibbitt-to-Contwoyto Winter Road
- Proposed Winter Access Road
- Watercourse
- Waterbody
- Air Quality Receptor
- Human Health Assessment Receptor
- Proposed National Park Boundary Receptor
- Development Area Boundary
- Local Study Area
- Modelling Domain
- Project Footprint
- Regional Study Area
- The Study Area for a National Park on the East Arm of Great Slave Lake

NOTES
Base data source: National Topographic Base Data (NTDB) 1:50,000.

GAHCHO KUÉ PROJECT

Receptor Locations Assessed for the Human Health Risk Assessment

PROJECTION: UTM Zone 12	DATUM: NAD83	De Beers Canada
Scale: 1:70,000 (LSA)		
FILE No: E2012-Air-061-GIS		DATE: August 21, 2012
JOB NO: 11-1365-0012	REVISION NO: 0	**Figure 2.4-2**
OFFICE: GOLD-CAL	DRAWN: CW / CHECK: DC	

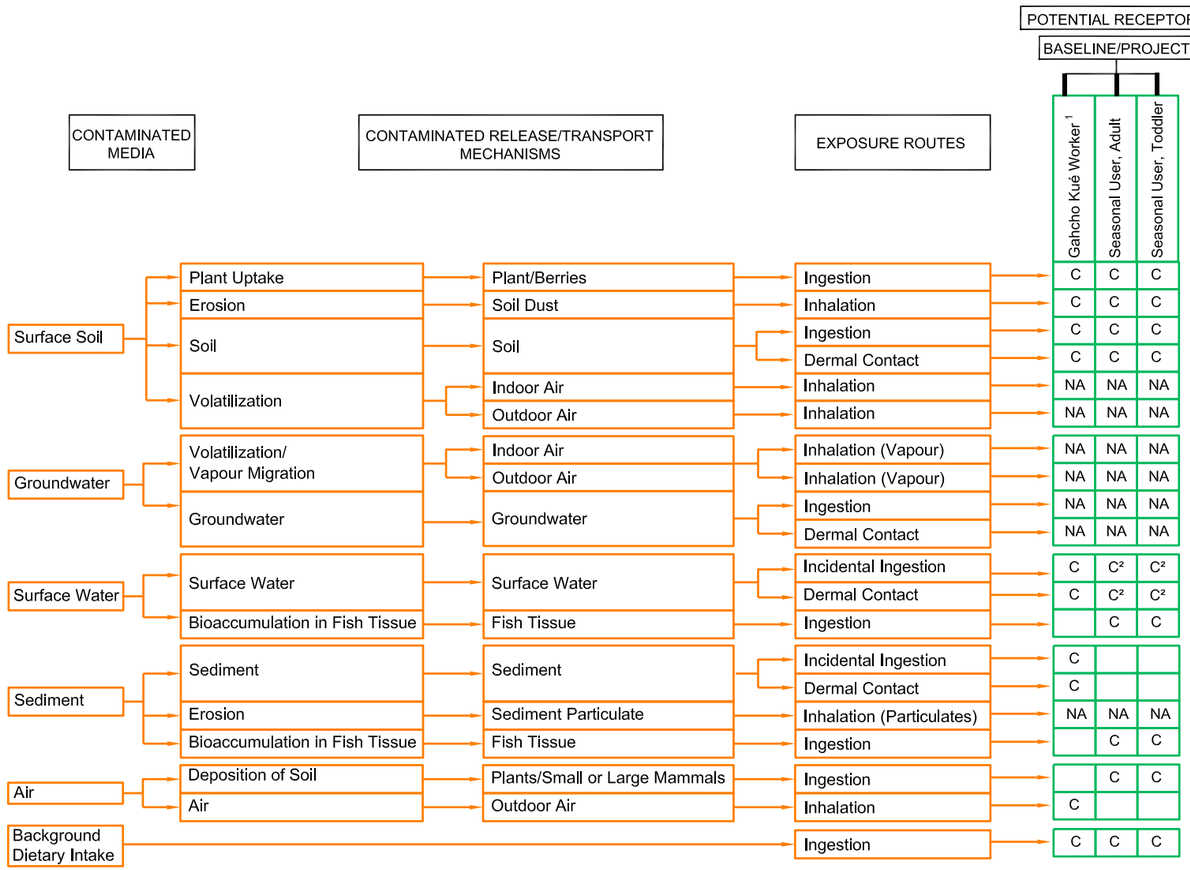
2.4.5 Key Issues

Each key line of inquiry and subject of note includes a pathway analysis that identifies and screens the linkages between individual Project components or activities. The key lines of inquiry and subject of notes were used to identify Key Issues. Primary sources of contaminants considered for the HHRA include fugitive dust, air emissions, exposed sediments, exposed soil and mine rock, and Project-related discharges and runoff to water bodies. The health of human receptors could be influenced by resulting changes to concentrations of metals in exposure media (secondary sources), including surface water, soil, plant tissue, fish tissue, and animal tissue. Further details of linkages between chemical sources and changes to exposure media concentrations are provided in the Human Health Conceptual Model (Figure 2.4-3).

The Key Issues consider the potential effects of the Project under the Application Case. The HHRA component addressed three Key Issues:

- The potential effects of emissions from existing and approved developments and the Project on short-term (acute) exposure and human health.
- The potential effects of emissions from existing and approved developments and the Project on particulate matter with a mean aerodynamic diameter of 2.5 microns (µm) or less ($PM_{2.5}$), and on particulate matter with a mean aerodynamic diameter of less than 10 µm (PM_{10}) on human health.
- The potential effects of emissions from existing and approved developments and the Project on long-term (chronic) exposure and human health.

The main sources of exposure for the HHRA are related to air and water emissions. The pathways evaluated in this HHRA are described in Section 4.



LEGEND

C	Complete Exposure Pathway
NA	Not Applicable Pathway
☐	Incomplete Exposure Pathway
1	Gahcho Kué Worker is assumed to not be consuming traditional food while on site.
2	Incidental ingestion and dermal content with drinking water.

GAHCHO KUÉ PROJECT

Human Health Conceptual Model

PROJECTION: UTM Zone 12	DATUM: NAD83	De Beers Canada
FILE No: P2011-Other-015-CAD		DATE: March 3, 2012
JOB No: 11-1365-0001	REVISION No: 1	Figure 2.4-3
OFFICE: GOLD - SAS	DRAWN: GG/TAH / CHECK:	

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3 COMPILATION OF CHEMICAL CONCENTRATIONS

Chemical concentrations in various media (e.g., soil, water and sediment) were screened against relevant regulatory guidelines and benchmarks. Screening included concentrations from two sources:

- baseline sampling surveys conducted from 1999 through 2011; and
- estimated chemical concentrations in media during the Project (i.e., construction, operations and closure phases).

Soil concentrations during the Project were estimated by summing:

- the baseline upper limit concentrations (e.g., 95% UCLM); plus
- incremental increases in concentrations estimated from dust deposition rates based on the results of the 2012 Updated Air Quality Assessment (De Beers 2012b).

Surface water concentrations of COCs for the Baseline Case and Application Case were predicted from water quality models. For potable water, predicted long term average concentrations for Kennady Lake (including Area 8) were used for the Baseline Case, and predicted maximum Project concentrations for Area 8 were used for the Application Case. For surface water, predicted long term average concentrations for Kennady Lake (including Area 8) were used for the Baseline Case, and predicted long-term averages for Kennady Lake (including Area 6, Area 8 and WMP) were used for the Application Case.

For prediction of fish and mammal tissue concentrations, maximum surface water concentrations in Lakes 410 and N11 during the Project, as summarized in Section 9 of the 2012 EIS Supplement (De Beers 2012a), were used for screening purposes, while baseline water quality was considered as long-term average concentrations.

Sediment COC concentrations from Kennady Lake, Kirk Lake, Lake 410, and Control Lake were reported from grab and core samples collected in 1999, 2004, 2005, 2010 and 2011. For more information regarding the sediment sampling methods and results, consult Annex J (Fisheries and Aquatic Resources Baseline) to the 2010 EIS (De Beers 2010). Changes in sediment quality are not expected as discussed in the Water Management Plan, Section 8 of the 2011 EIS Update, 2010 EIS Addendum JJ, and the 2011 Water Quality and Sediment Quality Supplemental Monitoring Report (De Beers 2011, 2010; Golder 2012a). Therefore, sediment concentrations were assumed to be the same in the Baseline Case and Application Case.

A summary of sources of exposure concentrations for COCs is provided in Table 3-1 below.

Table 3-1 Sources of Exposure Concentrations of Chemicals of Concern

Phase	Soil	Potable Water	Surface Water	Sediment	Vegetation	Fish Tissue	Small Mammal Tissue (Snowshoe Hare)	Large Mammal Tissue (Caribou)
Baseline Case	95% upper confidence limit of the mean (UCLM), 90th percentile, or maximum value (if statistics not possible)	Long-term average (Kennady Lake including Area 8)	Predicted long-term averages for Kennady Lake (including Area 6, Area 8 and WMP)	95% UCLM or 90th percentile, or maximum value (if statistics not possible)	95% UCLM or 90th percentile, or maximum value (if statistics not possible)	Predicted from baseline water concentrations in Lake N11 and Lake 410 using site specific BAFs	Predicted using food chain model and literature biotransfer factors	Predicted using food chain model and literature biotransfer factors
Application Case (Construction and Operations Phases of the Project)	Predicted from baseline and wet and dry deposition rates	Water concentrations for Area 8 (predicted from the Baseline water quality data) were used to assess ingestion and dermal exposure; the higher of the 95th UCLM for the Operations and Construction phases was used as the exposure concentration	Maximum annual average concentrations predicted for Kennady Lake (including Area 6, Area 8 and WMP)	95% UCLM or 90th percentile, or maximum value (if statistics not possible)	Predicted from maximum baseline vegetation concentration, wet and dry deposition rates and site-specific soil-to-plant BAFs (applied to project soil concentrations).	Predicted from maximum Project water concentrations predicted for Lake N11 and Lake 410 using site specific BAFs	Predicted using food chain model and literature biotransfer factors	Predicted using food chain model and literature biotransfer factors

95% UCLM = 95% upper confidence limit of the mean; % = percent; WMP = Water Management Pond; BAF = bioaccumulation factor.

3.1 SOILS DATA

Soil data for samples collected throughout the LSA were compiled to determine baseline soil concentrations of metals and polycyclic aromatic hydrocarbons (PAHs; Figure 3.1-1).

For the Project phase, the incremental increases in soil concentrations in the LSA were estimated using Equation 1:

$$Cs = 10{,}000 \times \frac{D_{yd} + D_{yw}}{Z_s \times BD} \times tD \times 1000$$

Where:

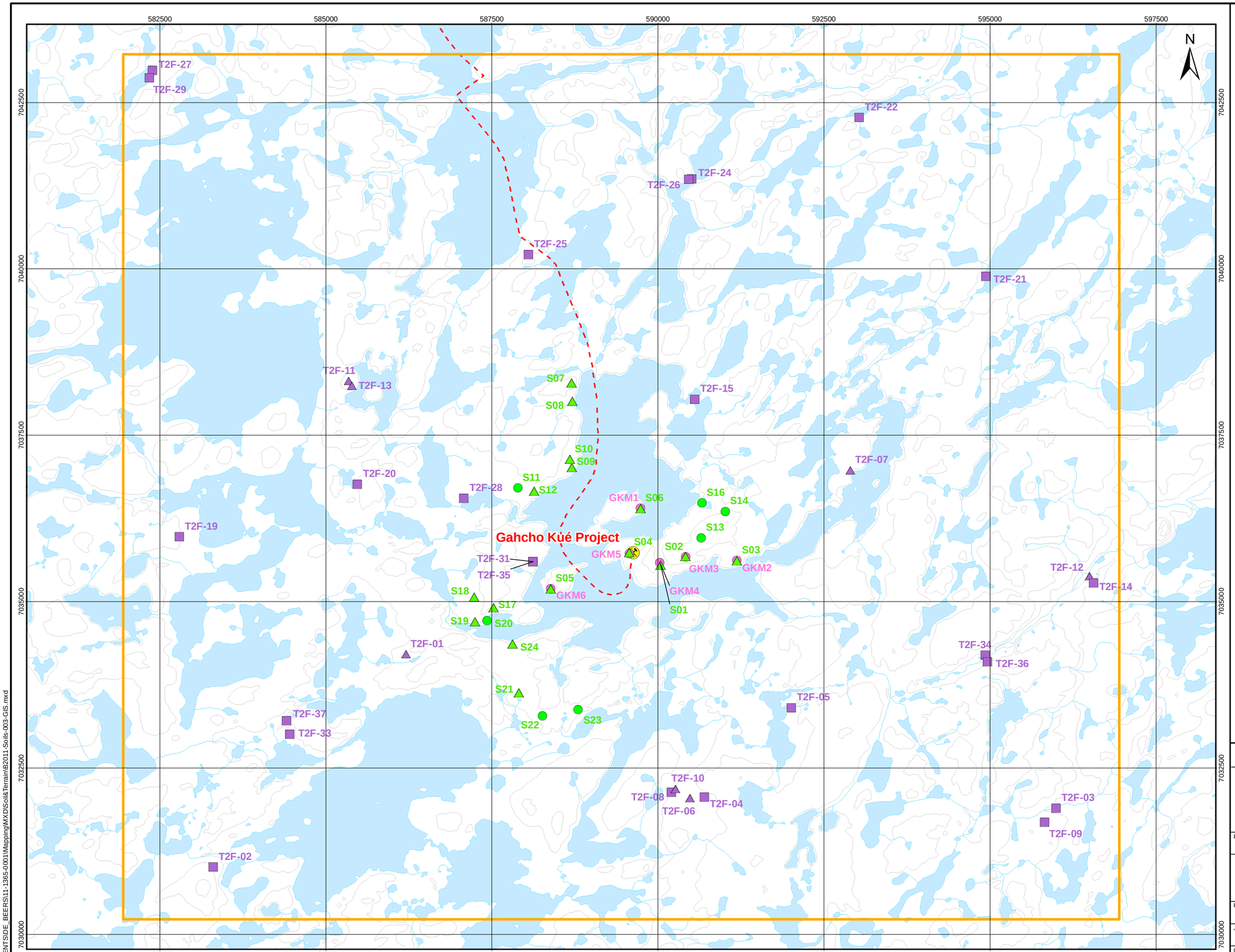
- Cs = average incremental soil concentration over exposure duration (mg COC/kg soil dry weight)
- 10000 = conversion factor (cm^2/m^2)
- D_{yd} = yearly dry deposition rate of COC (g COC/m^2-yr)
- D_{yw} = yearly wet deposition rate of COC (g COC/m^2-yr)
- tD = time period over which deposition occurs (yr)
- Z_s = soil mixing depth (cm)
- BD = soil bulk density (g soil/cm^3 soil)
- 1000 = conversion factor (mg/g)

Wet and dry deposition rates of COCs were based on the values reported in the 2012 Updated Air Quality Assessment (De Beers 2012b).

3.2 VEGETATION DATA

Data from several species of each vegetation type were combined into generic classes. Baseline COC concentration values were established for leaves, grasses/sedges/forbs, berries, and lichens using 95% UCLM or 90th percentile concentrations (where possible; otherwise the maximum value was used) observed in on-site samples (Figure 3.1-1 presents the sampling locations).

It was assumed that humans were directly consuming northern Labrador tea leaves (as a surrogate for leafy vegetables) and berries. Northern Labrador tea leaf, berry, lichen and grass data were used in the prediction of mammal tissue concentrations Berry species included cloudberry, mountain cranberry, crowberry, alpine bearberry, and bog bilberry. For vegetation, where a COC was not detected in any of the samples, the analytical detection limit was assumed to represent the baseline COC concentration.



LEGEND

- Gahcho Kué Project
- Contour (10m interval)
- Winter Access Road
- Watercourse
- Waterbody
- 2009 ELC/Soil Inspection and Sample Site
- 2009 ELC/Soil Inspection and Sample Site with Total Elemental Analysis
- 2009 Vegetation Baseline Metals Sample Plot Locations
- 2011 Soil Sampling Location
- 2011 Soil and Vegetation Sampling Location
- Local Study Area

NOTES

Base data source: National Topographic Base Data (NTDB) 1:50,000

GAHCHO KUÉ PROJECT

Sample Plot Locations for Soil and Vegetation Baseline Metals Sampling

PROJECTION: UTM Zone 12	DATUM: NAD83	De Beers Canada
Scale: 1:55,000 (1,000 500 0 1,000 Meters)		
FILE No: B2011-Soils-003-GIS		DATE: August 7, 2012
JOB NO: 11-1365-0001	REVISION NO: 1	Figure 3.3-1
OFFICE: GOLD-CAL	DRAWN: DC	CHECK: JF

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For the Project phase, the incremental increase in vegetation incorporated increases due to COC deposition onto plant surfaces and increased accumulation from soils. Deposition on plant surfaces was estimated using Equation 2:

$$P_d = \frac{1000 \times \left[D_{yd} + (F_w \times D_{yw}) \right] \times R_p \times \left[1 - \exp(-kp \times Tp \right]}{Yp_i \times kp}$$

Where:

- P_d = concentration of pollutant due to direct deposition on the plant group
- 1000 = conversion factor (mg/g)
- D_{yd} = yearly dry deposition rate of COC (g/m^2-yr)
- F_w = fraction of COC wet deposition that adheres to plant surface (0.2 for anions and 0.6 for cations and most organics)
- D_{yw} = yearly wet deposition rate of COC (g/m^2-yr)
- R_p = interception fraction of the edible portion of plant tissue for the plant group
- kp = plant surface loss coefficient (yr^{-1})
- Tp = length of plant exposure to deposition per harvest of the edible portion of the plant group (yr)
- Yp = yield or standing crop biomass of the edible portion of the plant productivity (kg/m^2)

Wet and dry deposition rates of COCs were based on the values reported in 2012 Updated Air Quality Assessment (De Beers 2012b). Accumulation of soil COCs in plant tissues was estimated using Equation 3:

$$Pr = C_s \times BAF$$

Where:

- Pr = concentration of COCs in plant tissue due to root uptake (mg/kg)
- Cs = average soil concentration over exposure duration (mg/kg)
- BAF = site-specific bioaccumulation factor (kg soil/kg produce)

The incremental soil COC concentration was estimated using Equation 1, and the site specific soil-to-plant bioaccumulation factor was estimated using Equation 3, with model baseline soil and plant COC concentrations.

Concentrations of the chemical resulting from direct deposition on the plant and root uptake (P_d and P_r in Equations 2 and 3, respectively) were summed to estimate the total COC concentration in plant tissues during the Project phase. The calculation was done separately for each type of plant tissue in the model: leaves, berries, lichen and grasses.

3.3 WATER AND SEDIMENT DATA

3.3.1 Potable Water

For the Baseline case, baseline water quality data (i.e., long term averages) for Kennady Lake (including Area 8) were used to assess exposure of Seasonal Users and Gahcho Kué workers ingesting and coming into contact with potable water (Appendix IV). For the Application Case, water concentrations for Area 8 during the Construction and Operations phases of the Project (predicted from the Baseline water quality data) were used to assess ingestion and dermal exposure; the higher of the 95% UCLM for the Project (Construction and Operations phases) was used as the exposure concentration (Appendix IV).

3.3.2 Surface Water

For the Baseline Case, water concentrations used to assess exposure of Gahcho Kué workers coming into contact with and incidentally ingesting surface water are predicted long-term averages for Kennady Lake (including Area 6, Area 8 and WMP) (Appendix IV). For the Application case, maximum annual average concentrations predicted for Kennady Lake (including Area 6, Area 8 and WMP) were used (Appendix IV). The Seasonal User may come into contact with surface water and sediment while conducting traditional activities such as fishing; dermal contact with water was conservatively evaluated through dermal contact with potable water (which was assumed to be used daily for showering, etc. as water quality is similar and exposure is expected to be much more frequent).

3.3.3 Water Used to Predict Tissue Concentrations

Water quality data for Lake N11 and Lake 410 were used in the prediction of fish and mammal tissue concentrations. For Lake N11 and 410 water quality, the maximum long-term average between the two lakes was used to represent baseline water quality conditions (Appendix IV). Predictions of water quality for the combined Project phases are provided in Section 9 of the 2012 EIS Supplement (De Beers 2012a). The maximum concentration of each COC during the entire operations phase in Lakes N11 and 410 (as described in Section 9 of the 2012 EIS Supplement [De Beers 2012a]) was assumed to

represent water quality conditions during the Project phase for exposure modelling.

Total metal concentrations in water were used to estimate the daily intakes in drinking water for mammals, bioaccumulation factors for fish tissues and to predict fish tissue COC concentrations.

It is acknowledged that water quality impacts will also occur in Kennady Lake and that these impacts may be of higher magnitude than those observed in Lakes N11 and 410. However, based on the following considerations, water quality changes in Kennady Lake were not deemed an appropriate representation of human exposure (i.e., through the harvesting and consumption of fish and game):

- A fish removal program will be conducted in the water management areas of Kennady Lake, removing fish in these areas as a food source for piscivorous wildlife or harvesting by people.
- Dewatering of the water management areas will result in significant disturbance and alteration of the aquatic habitats, limiting the availability of aquatic invertebrates in these areas to wildlife.
- Kennady Lake (Areas 2 to 7) will be isolated from the surrounding watersheds through the establishment of the controlled area boundary, and site access will be managed. In addition, the disturbance and mining activity within the site boundary will be a deterrent for wildlife. Seasonal Users will not have access to Kennady Lake.
- Total metal concentrations in water were used to estimate the daily intakes in drinking water for mammals, bioaccumulation factors for fish tissues and to predict fish tissue COC concentrations.
- No harvesting and subsequent of consumption of country foods will permitted by GK mine workers while on-Site

3.3.4 Sediment

For lake bottom sediments in Kennady Lake (grab samples and cores) the 95% UCLM or 90th percentile of the observed concentrations from 1999 to 2011 were assumed to provide a conservative representation of COC concentrations (Appendix IV). Based on the discussion in Section 8 of the 2011 EIS Update (De Beers 2011), sediment COC concentrations are not expected to increase during the Project phases, therefore the same COC concentrations were assumed for both the Baseline case and Application case.

3.4 FISH DATA

Lake trout, round whitefish, and slimy sculpin were collected in Kennady Lake and surrounding lakes in 1996, 1999, and 2004. Lake trout and round whitefish were assumed to provide the best representation of the species that would be consumed in the area. The median fish tissue concentrations from 1996, 1999, and 2004 (separate averages were calculated for each year) were used to estimate the water-to-fish tissue bioaccumulation factor.

4 LINKAGE ANALYSIS

The potential linkages between the Project and human health were evaluated to assess the Key Issues (Section 2.4.5). The following four linkages were analyzed:

- the linkage between changes in air quality and human health;
- the linkage between changes in water quality and human health;
- the linkage between changes in fish tissue quality and human health; and
- the linkage between changes in soil, plant and animal tissue quality and human health.

4.1 AIR QUALITY

Gahcho Kué Workers and Seasonal Users, i.e., temporary residents of hunter/trapper cabins and people spending time in recreational areas (e.g., fishing lodges or the proposed National Park) may be exposed via direct inhalation to airborne chemicals emitted from the Project. This linkage was evaluated in the Sections 5.2 and 5.3 (i.e., effects on human health due to chronic exposure to air emissions, acute exposure to air emissions and particulate matter exposure).

4.2 WATER QUALITY

4.2.1 Potable Water

Gahcho Kué workers were expected to obtain water from the mine drinking supply which will originate from Area 8. It was assumed that Aboriginal Seasonal Users will not have access to municipally treated drinking water while spending time conducting seasonal harvesting activities in the vicinity of the Project, but rather obtain drinking water from local surface waterbodies. Water quality for Area 8 was used to assess ingestion and dermal contact with drinking water by both Seasonal Users and Gahcho Kué workers.

This linkage is evaluated in the Section 5.4 (i.e., effects on human health due to chronic exposure to changes in water quality as part of the chronic multi-media assessment).

4.2.2 Surface Water

Water quality from Kennady Lake was used to assess incidental ingestion and dermal contact of Gahcho Kué workers with surface water while removing and/or stockpiling sediments from Kennady Lake.

Although the Seasonal User may come into contact with surface water and sediment while conducting traditional activities such as fishing; dermal contact with water was conservatively evaluated through dermal contact with potable water (which was assumed to be used daily for showering, etc. as water quality is similar and exposure is expected to be much more frequent).

This linkage was evaluated Section 5.4 (i.e., effects on human health due to chronic exposure to changes in water quality as part of the chronic multi-media assessment).

4.3 SEDIMENT QUALITY

Meaningful or detectable changes in concentrations of COCs (i.e., metals) are not expected to occur in sediment due to either Project related changes in water quality or to ambient air concentrations and associated deposition, resulting in an incomplete linkage to sediment quality.

Although effects from the Project on sediment quality (Water Management Plan; Section 8 of the 2011 EIS Update [De Beers 2011]) are negligible and the sediment quality linkage is incomplete, incidental ingestion and dermal contact with sediment was included for the Gahcho Kué worker to evaluate total exposure to COCs. This pathway was assessed for chronic effects associated with multi-media exposure (Section 5.4).

4.4 FISH TISSUE

Changes in concentrations of COCs (i.e., metals) in fish tissue due to Project related changes in water quality in local water bodies (i.e., Lake N11 and Lake 410) are expected. Seasonal Users are anticipated to consume fish of similar quality and therefore this linkage was evaluated in the Section 5.4 (i.e., effects on human health due to chronic exposure to changes in fish tissue quality as part of the chronic multi-media assessment).

4.5 SOIL, PLANT AND ANIMAL TISSUE QUALITY

Many of the potentially toxic chemicals released to air from the Project are volatile and will not deposit to an appreciable extent onto soil and plants. However, particulate matter containing COCs from incomplete combustion may deposit directly onto plant surfaces and soil in the area. Some COCs (e.g., metals) can accumulate in soil, plants and animals that are significant food sources for local residents. Thus, the linkage between changes in soil, plant and animal tissue quality and human health were evaluated in the assessment for chronic effects associated with multi-media exposure (Section 5.4).

5 RISK ASSESSMENT METHODS

This section presents a brief outline of the methods used in the HHRA. Specific methods employed for the air quality (acute and chronic), the particulate matter risk assessment, and the multi-media assessment are presented below in Sections 5.1 through 5.4. These three types of risk assessment align with the Key Issues for human health identified in Section 2.4.5.

5.1 CHEMICAL GROUPS AND SURROGATES

Chemicals that had sufficient toxicity information were assessed individually (e.g., benzene, toluene). However, several chemicals were evaluated as groups, for the following reasons:

- toxicity information was limited for some compounds;
- compounds with similar structures may act additively; and
- some toxicity information was available for chemical mixtures (e.g., petroleum hydrocarbon fractions).

When chemicals were assessed as a group, surrogate chemicals were used whenever possible to represent the chemical group. Use of a surrogate relies on the toxicological principle that the molecular structure of a chemical strongly influences its reactivity, biological activity and toxicity. The surrogate approach facilitates assessment of a chemical or a group of chemicals for which little or no toxicological information exists. Therefore, surrogates were selected that were structurally similar and that represented the more toxic and volatile compounds, with sufficient supporting toxicity information.

The term Total Petroleum Hydrocarbons (TPHs) refers to groups of hydrocarbon chemicals derived from a petroleum source. The TPH compounds were grouped by carbon number ranges, based on studies demonstrating that such groupings reflect commonalities in physical properties and toxic potential. Groups were then further subdivided into two fractions: aliphatic compounds and aromatic compounds. Aliphatic compounds are straight-chain or cyclical hydrocarbons (e.g., n-hexane, nonane, decane and cyclohexane). Aromatic compounds are composed of one or more benzene rings (e.g., toluene and xylene). Five groups

of TPH compounds were evaluated: C_2-C_8 aliphatics, C_9-C_{16} aliphatics, C_{16+} aliphatics, C_6-C_8 aromatics and C_9-C_{16} aromatics[1].

The following identifies the surrogate for each chemical group assessed:

- aldehydes (surrogate: acetaldehyde);
- C_2 to C_8 aliphatics (surrogate: cyclohexane);
- C_6 to C_8 aromatics (surrogate: toluene);
- C_9 to C_{16} aliphatics (surrogate: decane);
- C_9 to C_{16} aromatics (surrogate: ethylbenzene; lower weight substance applied due to the lack of toxicological information for this hydrocarbon group);
- C_{16+} aliphatics (surrogate: decane for the acute assessment [lower weight substance applied due to the lack of toxicological information for this hydrocarbon group)
- dioxins/furans (surrogate: 2,3,7,8-tetrachloro-dibenzo-*ρ*-dioxin [TCDD] for the chronic assessment);
- trimethylbenzenes (surrogate: 1,2,4-trimethylbenzene); and
- xylenes (surrogate: xylenes mixture).

For PAHs, the parent compound (i.e., the most closely structurally related PAH is considered to be the parent compound, for example naphthalene is the parent compound for 1-methylnaphthalene or 2-methylnaphthalene) was used as the surrogate where possible. Where data for a parent compound were not available, the following surrogates were used:

- Acephenanthrylene (surrogate: benzo[k]fluoranthene);
- Benzo(a)fluorene (surrogate: 2,3-benzo(b)fluorene);
- Benzo(g,h,i)fluoranthene (surrogate: benzo[g,h,i]perylene);
- Coronene (surrogate: benzo(g,h,i)perylene);
- Indeno(1,2,3-cd)fluoranthene (surrogate: fluoranthene);
- Indeno(1,2,3-W)pyrene (surrogate: indeno[1,2,3-cd]pyrene);
- 1-Methylphenanthrene (surrogate: phenanthrene);

[1] C_6-C_8 is the shortest aromatic chain possible as a minimum of 6 carbons are required to form an aromatic ring. Heavier aromatic fractions (C_{16}-C_{32}) were not assessed as they are not typically present in air due to their limited volatility and are not expected to be emitted into water.

- 2-Methylanthracene (surrogate: anthracene);
- 2-Methylfluorene (surrogate: fluorene);
- 2-Methylphenanthrene (surrogate: phenanthrene);
- 2-Methylpyrene (surrogate: pyrene);
- 3-Methyldibenzothiophene (surrogate: dibenzothiophene);
- 3-Methylphenanthrene (surrogate: phenanthrene);
- 4 + 9 Methylphenanthrene (surrogate: dibenzothiophene);
- 4-Methyldibenzothiophene (surrogate: dibenzothiophene);
- Nitropyrene (surrogate: benzo[a]pyrene); and
- Picene (surrogate: dibenzo[a,h]anthracene).

5.2 AIR QUALITY ASSESSMENT METHODS

The acute and chronic air quality assessments evaluate potential risks of chemicals/chemical groups present in air emissions from the Project, with a focus on short-term and long-term exposures, respectively, to humans. This section includes the methods used to evaluate the effects on short-term (acute) and long-term (chronic) exposure and human health of emissions from existing and approved developments and the Project (both together and separately).

Particulate matter (PM) is considered a criteria pollutant; however, since the assessment for PM follows different methods than used to evaluate other airborne substances, the assessment of PM is presented separately in Section 5.3. The available health-based chronic air screening levels are presented in Appendix I, Table I-5.

5.2.1 Acute Air Quality Assessment

5.2.1.1 Chemical Screening Process for Acute Air Quality Risk Assessment

For each chemical, air concentrations representing maximum 1-hour and 24-hour concentrations (referred to as “peak” concentrations) were predicted for receptor locations throughout the region. These exposure estimates were compared to the most conservative of the available acute health-based thresholds from the following agencies:

- Northwest Territories Guideline for Ambient Air Quality Standards (GNWT 2011);

- Agency of Toxic Substances and Disease Registry (ATSDR 2012);
- Ontario Ministry of Environment and Energy (OMOE 2012a,b);
- California Environmental Protection Agency Office of Environmental Health Hazard Assessment (CalEPA OEHHA 2012);
- World Health Organization (WHO 2000, 2005); and
- Texas Commission on Environmental Quality (TCEQ 2012).

A summary of the technical derivations for these thresholds is provided below. The available 1-hour and 24-hour health-based thresholds and the basis of these thresholds are presented in Appendix I, Tables I-1 and I-2, respectively.

Northwest Territories Guideline for Ambient Air Quality Standards

The NWT air quality standards were considered the priority source for thresholds (GNWT 2011). If a NWT threshold was available, it was used preferentially as the Project screening threshold. Thresholds based on health-based endpoints were applied preferentially, whereas other endpoints (e.g., odour) were only used in the absence of a health-based endpoint. In the absence of NWT thresholds, the lowest health-based value with supporting documentation was adopted.

Agency of Toxic Substances and Disease Registry Minimal Risk Levels

The ATSDR derives Minimal Risk Levels (MRLs) for non-carcinogenic health effects. MRLs are based on reliable and sufficient data that identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure to the substance. The ATSDR generally uses the No Observed Adverse Effect Level/Uncertainty Factor (NOAEL/UF) approach to derive MRLs. Physiologically-based pharmacokinetic (PBPK) modelling and benchmark dose (BMD) modelling have also been used in deriving MRLs. The MRLs are set below levels that may cause adverse health effects in the most sensitive subpopulations of people. The acute MRLs are derived for exposure durations of 1 to 14 days.

The ATSDR MRLs are generally based on the most sensitive substance-induced endpoint considered to be of relevance to humans. However, as MRLs are often based on animal studies (because of lack of relevant human studies), there is some degree of uncertainty associated with MRLs. Generally, precise toxicological information is lacking for people who might be most sensitive (e.g., infants, elderly and nutritionally or immunologically compromised). As a result of this uncertainty, ATSDR assumes that humans are more sensitive than animals to the effects of hazardous substances, that certain people may be particularly sensitive, and applies a protective approach (i.e., application of margins of safety) to address any uncertainties.

Ontario Ministry of the Environmental Air Quality Criteria

The OMOE has developed ambient air quality criteria (AAQC), which may be used in environmental assessments to assess air quality. An AAQC is a desirable concentration of a contaminant in air, based on protection against adverse effects on health or the environment. The term "ambient" is used to reflect general air quality independent of location or source of a contaminant. AAQCs are set with different averaging times (e.g., 24-hour, 1-hour and 10 minutes) appropriate for the effect that they are intended to protect against. The effects considered may be health, odour, vegetation, soiling, visibility, corrosion or other effects.

The OMOE has also derived air quality standards used to assess emissions from non-mobile sources of air pollution in Ontario. The Ontario air quality standards are generally derived from ambient air quality criteria, and are set at levels protective of the natural environment and sensitive populations (e.g., children, the elderly). The OMOE considers available toxicological information and supporting environmental information to establish effects-based air standards based on the limiting or critical effect(s) (health or environmental considerations) of that chemical.

California OEHHA Reference Exposure Levels

The California Office of Environmental Health Hazard Assessment (OEHHA) Reference Exposure Levels (RELs) are concentrations of a chemical at or below which adverse non-carcinogenic health effects are not anticipated to occur for a specified exposure duration. The RELs are used in risk assessments to evaluate the potential for adverse non-carcinogenic public health effects, including:

- potential effects from facility emissions or similar localized sources (Air Toxics Hot Spots Program); and
- potential effects from widespread exposures (Toxic Air Contaminants Program).

The REL is an exposure concentration at or below which adverse non-carcinogenic health effects are not expected to occur in a human population, including sensitive subgroups (e.g., infants and children). Supporting documentation was available for most of the RELs.

World Health Organization Air Quality Guidelines

The WHO has produced air quality guidelines to provide a basis for protecting public health from adverse effects of air pollution. The guidelines provide guidance to governments in making risk management decisions, particularly in setting standards, and provide additional background information for handling of

air pollution. The guidelines may be used in planning processes and various kinds of management decisions at a community or regional level. Supporting documentation was provided for all WHO guidelines.

Texas Commission on Environmental Quality (TCEQ) Effects Screening Levels

Where air quality thresholds were not available from all other jurisdictions, the criteria derived by TCEQ were used. The TCEQ has developed acute (1-hour) Effects Screening Levels (ESLs) that are used in the air permitting process to evaluate effects predicted by air dispersion modelling. The ESLs are used to evaluate the potential for effects to occur as a result of exposure to concentrations of constituents in the air. The ESLs are based on data concerning health effects, the potential for odours to be a nuisance, effects on vegetation, and corrosive effects. The ESLs are not ambient air standards. If predicted airborne levels of a constituent do not exceed the screening level, adverse health or welfare effects are not expected. If predicted ambient levels of constituents in air exceed the screening levels, it does not necessarily indicate a problem but rather triggers a review in more depth. The TCEQ has developed a guidance document titled *Guidelines to Develop Effects Screening Levels, Reference Values and Unit Risk Factors* (TCEQ 2006) that outlines the approach and methods used to derive the acute and chronic ESLs. Texas does not provide supporting documents for all compounds for which they have screening values. Accordingly, the TCEQ ESLs were not selected for use in the screening process unless adequate supporting documentation was available.

Screening Results

Predicted peak 1-hour and 24-hour concentrations in air from the Application Case were compared to NWT acute air quality thresholds, if available. If not, the predicted peak acute air quality concentrations were compared to the most conservative of the 1-hour and 24-hour acute thresholds (Appendix I, Tables I-3 and I-4), respectively. A parameter was retained for further evaluation if the predicted peak concentration (i.e., maximum from all receptor locations) was greater than the threshold, for the Application Case. A parameter that is retained for further assessment is classified as a COC and was evaluated for all receptor locations.

Based on the results of the chemical screening, the following COCs were retained for the acute air quality risk assessment based upon a 1-hour acute exposure:

- benzo(a)pyrene;
- aluminum;
- iron; and

- nickel.

The following COCs were retained for the acute air quality risk assessment based upon a 24-hour acute exposure:

- nitrogen dioxide (NO_2);
- acrolein;
- benzo(a)pyrene;
- cadmium;
- iron;
- manganese; and
- nickel.

Due to the lack of a 24-hour threshold for bismuth and several PAHs, the annual thresholds for these parameters were conservatively utilized for screening of the 24-hour data. Annual predicted concentrations of these parameters did not exceed the annual threshold and they were; therefore, not retained as COCs.

5.2.1.2 Acute Air Quality Analysis

For each COC, the following approach was used to determine the magnitude of risk (i.e., negligible, low, moderate or high) resulting from short-term air exposures at communities, recreational areas, worker camps and the local study area maximum point of impingement (LSA MPOI, i.e., the Project Boundary):

- comparison of the maximum, and selected percentile air concentrations to acute exposure limits to provide additional context to predicted risk;
- comparison of Application Case concentrations to Baseline Case concentrations;
- evaluation of the conservatism in the air modelling approach used to predict future concentrations;
- evaluation of the conservatism in the acute exposure limits for that parameter; and
- evaluation of the potential acute health effects that may occur at the predicted concentrations.

For each of the COCs, an HQ was calculated for each receptor location and all three assessment cases as follows:

$$HQ = \frac{\text{COC concentration in air } (\mu g/m^3)}{\text{Acute Threshold Concentration } (\mu g/m^3)}$$

The HQs for which toxicity reference values were based on similar target organs were added together to determine a total HQ for similar toxicological effects (Health Canada 2009a).

The toxicological basis of the 24-hour threshold for NO_2 was not provided, other than to say that it is based on odour. However, the threshold for NO_2 is likely to be based on respiratory effects based upon review of acute health effects from various health reviews such as that provided by WHO (2000). Supporting documentation on the derivation of the iron threshold was not available (OMOE 2012b). Acute inhalation of ferric salts as dusts and mists include irritation of the respiratory tract and irritation of the skin (HSDB 2010). The thresholds for acrolein and nickel were also based on respiratory effects; therefore, the HQs for NO_2, acrolein, iron and nickel were summed. The threshold for cadmium is based on kidney effects, and the manganese threshold is based on neurotoxicity and these substances were assessed individually.

Only non-carcinogenic effects are evaluated in the acute air quality assessment as cancer is associated with chronic exposure durations. Methods for the assessment of carcinogenic effects associated with exposure to Project-related changes in air quality are presented in Section 5.2.2.

5.2.2 Chronic Air Quality Risk Methods

5.2.2.1 Chronic Air Quality Risk Assessment

The chronic air quality risk assessment evaluates potential risks of chemicals/chemical groups present in air emissions from the Project. This section includes the methods used to evaluate the effects of emissions from the Project on long-term (chronic) exposure and human health.

5.2.2.1.1 Chemical Screening Process for Chronic Air Quality Risk Assessment

The peak annual predicted air concentrations for all the receptor locations were compared to air quality guidelines or objectives or screening levels (referred to herein as screening levels) derived for the protection of chronic inhalation to human health. The screening levels were obtained from:

- Northwest Territories Guideline for Ambient Air Quality Standards (GNWT 2011);
- United States Environmental Protection Agency (U.S. EPA) Regional Screening Levels (U.S. EPA 2012a);
- Ontario Ministry of Environment and Energy (OMOE 2012a,b);
- Agency for Toxic Substances and Disease Registry (ATSDR 2012);
- California Environmental Protection Agency Office of Environmental Health Hazard Assessment (CalEPA OEHHA 2012);
- World Health Organization Air Quality Guidelines (WHO 2000; 2005); and
- Texas Commission on Environmental Quality Effects Screening Levels (TCEQ 2012).

Carcinogenicity classification of the substances evaluated in the chronic air quality assessment is provided in Section 5.4.2.1.

Risk levels for which the screening levels/guidelines were derived were standardized to Canadian target risk levels (Health Canada 2009a) which have been adopted by the NWT. For non-carcinogens, this involved adjusting air quality standards using a target hazard quotient of 0.2, and for carcinogens, this involved adjusting air quality standards to a target incremental lifetime cancer risk of 1.0×10^{-5} (i.e., one in one hundred thousand), where applicable.

A summary of the technical derivations for these thresholds is provided in the following paragraphs.

Northwest Territories Guideline for Ambient Air Quality Standards

The NWT air quality standards (GNWT 2011) were considered the preferred source for selecting values. If a NWT value was available, it was used preferentially to other sources. Preference was given to screening levels that were health-based and had supporting documentation. In the absence of a NWT value, the most conservative of the available screening levels for a given chemical was used.

U.S. EPA Regional Screening Levels

The U.S. EPA has developed Regional Screening Levels (RSLs) for air which are based on the protection of human health. Regional Screening Levels are risk-based concentrations derived from standardized equations combining exposure information assumptions with U.S. EPA toxicity data. The RSLs are considered by the U.S. EPA to be protective for human exposure (including sensitive groups) over a lifetime. Chemical concentrations above the RSL would not automatically designate a health risk; however, exceeding a RSL suggests that further evaluation of the potential risks is appropriate. The RSLs shown in Appendix I, Table I-5 are the values derived for the protection of residential land use. The U.S. EPA RSLs for non-carcinogens are based on a hazard quotient of 1.0, and for carcinogens are based on a risk level of 1.0×10^{-6}. Therefore, the non-carcinogenic RSLs were multiplied by a factor of 0.2 to adjust to a hazard quotient of 0.2, and the carcinogenic RSLs were multiplied by a factor of 10 to adjust to a risk level of 1.0×10^{-5}.

Ontario Ministry of the Environment Air Quality Criteria

The OMOE has developed ambient air quality criteria (AAQC), which may be used in environmental assessments to assess air quality. An AAQC is a desirable concentration of a contaminant in air, based on protection against adverse effects on health or the environment. The term “ambient” is used to reflect general air quality independent of location or source of a contaminant. AAQCs are set with different averaging times (e.g., annual, 24-hour, 1-hour and 10 minutes) appropriate for the effect that they are intended to protect against. The effects considered may be health, odour, vegetation, soiling, visibility, corrosion or other effects.

The OMOE has also developed air quality standards used to assess emissions from all non-mobile sources of air pollution in Ontario. The Ontario air quality standards are generally derived from the ambient air quality criteria, and are set at levels protective of the natural environment and sensitive populations (e.g., children, the elderly). The OMOE considers available toxicological information and supporting environmental information to establish effects-based air standards based on the limiting or critical effect(s) (health or environmental considerations) of that chemical. In general, the OMOE air standards for carcinogens are set at an incremental lifetime cancer risk (ILCR) of one incidence in one million individuals. The air standards for non-carcinogens are generally set at a target hazard quotient of 1.0.

Agency of Toxic Substances and Disease Registry Minimal Risk Levels

The ATSDR derives Minimal Risk Levels (MRLs) for non-carcinogenic health effects based on reliable and sufficient data that identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given

route of exposure to the substance. The ATSDR generally uses the No Observed Adverse Effect Level/Uncertainty Factor (NOAEL/UF) approach to derive MRLs. Physiologically-based pharmacokinetic (PBPK) modelling and benchmark dose (BMD) modelling have also been used in deriving MRLs. The MRLs are set below levels that may cause adverse health effects in the most sensitive subpopulations of people. The chronic MRLs are derived for exposure durations greater than or equal to 365 days.

The MRLs are generally based on the most sensitive substance-induced endpoint considered to be of relevance to humans. However, as MRLs are often based on animal studies (because of lack of relevant human studies); there is some degree of uncertainty associated with MRLs. Generally, precise toxicological information is lacking for the people who might be most sensitive (e.g., infants, elderly and nutritionally or immunologically compromised). As a result of this uncertainty, ATSDR assumes that humans are more sensitive than animals to the effects of hazardous substances, that certain people may be particularly sensitive, and applies a protective approach (i.e., application of margins of safety) to address any uncertainties.

California OEHHA Reference Exposure Levels

The CalEPA OEHHA RELs are concentrations of a chemical at or below which adverse non-carcinogenic health effects are not anticipated to occur for a specified exposure duration (CalEPA OEHHA 2012). The RELs are used in risk assessments to evaluate the potential for adverse non-carcinogenic public health effects from facility emissions or similar localized sources in the Air Toxics Hot Spots Program, and from widespread exposures in the Toxic Air Chemicals program. The REL is an exposure at or below which adverse non-carcinogenic health effects are not expected to occur in a human population, including sensitive subgroups (e.g., infants and children), exposed to that concentration for a specified duration. Supporting documentation was available for most of the RELs. The chronic RELs were used in the screening process and are shown in Appendix I, Table I-5. The chronic RELs for non-carcinogens are based on a hazard quotient of 1.0. Therefore, the non-carcinogenic RELs were multiplied by a factor of 0.2 to adjust to a hazard quotient of 0.2.

The CalEPA OEHHA does not develop RELs or air quality guidelines or objectives for carcinogens. Rather, the CalEPA OEHHA has developed inhalation unit risks for use in cancer risk assessments (CalEPA OEHHA 2012). However, to consider the carcinogenic endpoints provided by CalEPA OEHHA (2012), they were contacted regarding the use of their unit risk factors for screening purposes. Mr. Chris Halm of the CalEPA Air Resources Board indicated that the unit risks can be adjusted based on an applicable cancer risk level and used as screening values (Halm 2010, pers. comm.). The CalEPA OEHHA unit risks are based on a cancer risk level of 1.0×10^{-6}; this level was

divided by the unit risk and multiplied by a factor of 10 to derive a screening value for a risk level of 1.0×10^{-5}.

World Health Organization Air Quality Guidelines

The WHO has developed air quality guidelines that provide a basis for protecting public health from adverse effects of air pollution. The guidelines are intended to provide background information and guidance to governments in making risk management decisions, particularly in setting standards, but their use is not restricted to this. The guidelines may be used in planning processes and various kinds of management decisions at a community or regional level. Supporting documentation was provided for all WHO guidelines.

Texas Commission on Environmental Quality Effects Screening Levels

The TCEQ has developed chronic ESLs that are used in the air permitting process to evaluate air dispersion modelling's predicted effects. The ESLs are used to evaluate the potential for effects to occur as a result of exposure to concentrations of constituents in the air. The ESLs are based on data concerning health effects, the potential for odours to be a nuisance, effects on vegetation and corrosive effects. They are not ambient air standards. If predicted airborne levels of a constituent do not exceed the screening level, adverse health or welfare effects are not expected. If predicted ambient levels of constituents in air exceed the screening levels, it does not necessarily indicate a problem but rather triggers a review in more depth. The TCEQ have developed a guidance document titled, *Guidelines to Develop Effects Screening Levels, Reference Values and Unit Risk Factors* (TCEQ 2006), that outlines the approach and methods used to derived the acute and chronic ESLs. Texas ESLs were only used in the absence of values from other regulatory agencies and jurisdictions. The chronic ESLs were used in the screening process and are shown in Appendix I, Table I-5. The TCEQ chronic ESLs for non-carcinogens are based on a hazard quotient of 0.3, and for carcinogens the ESLs are based on a risk level of 1.0×10^{-5}. Therefore, the non-carcinogenic ESLs were multiplied by a factor of 0.2/0.3 (i.e., 0.667) to adjust to a hazard quotient of 0.2.

Screening Results

Chemical screening was conducted by comparing the highest maximum annual predicted concentrations in air to the selected air screening levels for all receptor locations for the Application Case (Appendix I, Table I-6).

If chemicals or chemical group concentrations exceeded screening levels, they were defined as a COC and were retained for further analysis in the risk assessment.

Based on the screening results for the Baseline and Application Cases, the following COCs were retained for the chronic air inhalation risk assessment:

- nitrogen dioxide (NO_2); and
- acrolein.

Several metals were identified as having concentrations exceeding the chronic thresholds in the Application Case (i.e., aluminum, cadmium, cobalt, iron, manganese, nickel, titanium and vanadium). These parameters were evaluated as part of the multi-media risk assessment, along as exposure to these substances can occur via other exposure pathways in addition to air.

5.2.2.1.2 Exposure Pathways

The chronic air inhalation exposure pathway was evaluated in the Chronic Air Quality Risk Assessment.

5.2.2.1.3 Toxicity Assessment

Toxicity assessment involves the classification of the toxic effects of chemicals and the estimation of the amounts of chemicals that can be received by an organism without adverse health effects. For each COC, an appropriate TRV was identified based on reported mode of action (i.e., threshold versus non-threshold mode of action). For threshold chemicals (i.e., generally not a carcinogen) adverse effects are expected to only occur above a certain dose rate. However, for non-threshold chemicals (i.e., most carcinogens) theoretically all doses can exert a toxic effect. Carcinogenic classification of substances retained for chronic inhalation assessment is provided below.

Carcinogenicity Classification

The carcinogenicity classification for each of the COCs retained in the chronic air quality risk assessment is summarized in Table 5.2-1. The sources of carcinogenicity classification are as follows:

- U.S. EPA IRIS database (U.S. EPA 2012b);
- Health Canada (2009a,b); and
- International Agency for Research on Cancer (IARC 2012).

Table 5.2-1 Carcinogenicity Classification of Chemicals of Potential Concern for the Chronic Air Quality Risk Assessment

Chemical of Potential Concern	U.S. EPA IRIS[a]	Health Canada[b]	IARC[c]	Assessed as a Carcinogen?
Nitrogen dioxide (NO_2)	n/a	n/a	n/a	No
Acrolein	n/a	n/a	Group 3	No

[a] United States Environmental Protection Agency Integrated Risk Information System (U.S. EPA 2012b).

[b] Health Canada (2009b).

[c] International Agency for Research on Cancer (IARC 2012).

n/a = Not assessed.

Group 3 = not classifiable as to carcinogenicity to humans.

Toxicity Reference Values

The toxicity assessment involves identification of the potentially toxic effects of chemicals and determination of the amount of chemicals that a receptor can be exposed to without experiencing unacceptable effects. This value is called the TRV or toxicity benchmark. The TRVs are based simply on critical effects observed from studies in exposed human populations or animal species.

For the Chronic Air Quality Risk Assessment, TRVs for non-carcinogenic chemicals are called Reference Concentrations (RfC) and TRVs for carcinogenic chemicals are called Unit Risks (UR). An RfC is an estimate of continuous inhalation exposure to a chemical by the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects over a lifetime. A UR is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 $\mu g/m^3$ in air.

For the Chronic Air Quality Risk Assessment, nitrogen dioxide and acrolein were identified as COCs. As nitrogen dioxide and acrolein are not considered carcinogenic, only RfCs were considered in the assessment.

The following agencies were used to find available RfCs:

- Health Canada (Health Canada 2009b);
- United States Environmental Protection Agency Integrated Risk Information System (U.S. EPA 2012b);
- World Health Organization (WHO 2000);
- Agency of Toxic Substances and Disease Registry (ATSDR 2012); and
- National Institute of Public Health and the Environment (RIVM 2001, 2009).

The most conservative (i.e., lowest RfC) was selected for use in the risk assessment. The available RfCs, selected RfCs and toxicological basis of the

RfCs are presented in Appendix I, Table I-7. The RfCs were compiled only for the COCs identified in the problem formulation.

5.2.2.1.4 Risk Characterization

Long-term health effects were evaluated by calculating HQs for chemicals that do not cause cancer (non-carcinogens) and ILCR values for chemicals that are suspected to cause cancer (carcinogens).

In the risk characterization step, HQs were calculated for non-carcinogenic COCs as the ratio of the predicted concentration in air to the RfC, according to the following equation:

$$HQ = \frac{\text{COC Concentration in air } \left(\mu g / m^3\right)}{\text{RfC } \left(\mu g / m^3\right)}$$

An HQ less than or equal to 1.0 indicates that the estimated exposure is less than the reference concentration, signifying negligible health effects.

5.2.2.1.5 Chemical Mixtures

The HQs for COCs for which toxicity reference values were based on the similar target organs are added together to determine a total HQ for similar toxicological effects (Health Canada 2009a). The thresholds for acrolein and nitrogen dioxide do not have the same target effect; therefore, the HQs were not summed. Detailed information on the health effects associated each COC and the target endpoints/organs upon which the selected TRVs are based is presented in Appendix I, Table I-7.

5.3 PARTICULATE MATTER ASSESSMENT METHODS

Many epidemiological studies to identify the relationship between health effects and particulate matter (PM) have been conducted over the past 20 years. Many of these studies have shown that there is a relationship between increases in ambient particulate matter concentrations with mortality and hospitalizations for respiratory and cardiac health effects (Health Canada and Environment Canada 1999). This relationship has been demonstrated to be stronger for PM with a mean aerodynamic diameter of 2.5 µm ($PM_{2.5}$) than PM with a mean aerodynamic diameter of 10 µm (PM_{10}) (Health Canada and Environment Canada 1999). However, there has also been some uncertainty regarding the causal linkage between particulate matter and health effects. Many

epidemiological studies have been confounded by the presence of other air pollutants (e.g., sulphur dioxide), temperature, and smoking habits. In addition, there is uncertainty regarding whether epidemiological studies can discern between responses initiated by PM versus health effects of people who already have advanced and serious illnesses (Health Canada and Environment Canada 1999). Therefore, there is no prescribed method for assessing health risks of particulate matter, nor does the assessment of particulate matter lend itself to risk assessment methods in the same manner as other parameters.

The effects on human health as a result of exposure to PM due to emissions from existing and approved developments and the Project were evaluated qualitatively for $PM_{2.5}$ and PM_{10}.

5.4 CHRONIC MULTI-MEDIA RISK ASSESSMENT METHODS

The multi-media risk assessment evaluates potential risks of chemicals/chemical groups that are present in water, air and particulate emissions from the Project that may accumulate in other terrestrial media (e.g., soil, fish, plants and animals). Sediment was evaluated as a constant exposure (i.e., no incremental changes) for both the Baseline and Application Case in the multi-media exposure model.

The multi-media HHRA was conducted for both the Baseline and Application Cases. The risk assessments were conducted according to established human health risk assessment protocols endorsed by Health Canada (Health Canada 2009a,b). The process followed a widely recognized framework involving four phases: problem formulation, exposure assessment, toxicity assessment and risk characterization, as described briefly in Section 2.4.1 and as shown on Figure 2.4-1 (Health Canada 1995).

5.4.1 Problem Formulation

The potential for a human health risk to arise from environmental substances is predicated on the co-existence of three elements:

- chemicals must be present at hazardous levels;
- people must be present; and
- the potential for people to come into contact with the chemicals (called an exposure pathway) must exist.

The objective of the problem formulation was to develop an understanding of how chemicals emitted from the Project might affect human health. The problem

formulation helps to focus the risk assessment on the chemicals, people and exposure pathways of greatest concern, specifically:

- chemicals with the greatest toxic potential;
- people with the greatest likelihood of being exposed and the greatest susceptibilities; and
- exposure pathways that account for the majority of exposure to the chemicals emitted).

If acceptable human health risks are predicted for the most susceptible receptors, it is highly likely that acceptable human health risks would exist for other chemicals, people or exposure pathways.

5.4.1.1 People Evaluated in the Risk Assessment

Human receptors were selected based on the identification of persons with the greatest potential to be adversely affected by chemical exposures originating from the Project. For the purposes of the multi-media risk assessment, only the most exposed receptors were assessed; these were identified to be Seasonal Users and Gahcho Kué Workers. Seasonal Users are Aboriginal people who live in communities outside the RSA but spend time within the LSA/RSA while pursuing traditional activities (hunting, fishing, and gathering of traditional foods). It was assumed that Seasonal Users may spend up to six months within the LSA/RSA. These Seasonal Users may be exposed to air, soil, water and food items impacted by the Project. Recreational users and hunter/trappers that would spend less time than the Seasonal User within the LSA/RSA were not assessed as part of the multi-media risk assessment because the Seasonal User is a composite of these exposures and considered to be protective of these receptor exposures.

For chemicals that do not cause cancer (non-carcinogens), all life stages were evaluated. Toddlers are considered to be more susceptible to the effects of most non-carcinogens relative to other age categories because they typically have a greater intake rate to body weight ratio and because certain behavioural activities may expose them to larger quantities of chemicals (e.g., playing in soil). In addition, some chemicals have been shown to be more toxic to toddlers than to other age categories (e.g., adults). Consistent with risk assessment guidance (Health Canada 2009a), the toddler life phase (i.e., 7 months to 4 years) was chosen as the most sensitive child life stage.

For chemicals that cause cancer (carcinogens), a composite receptor was employed to amortize exposure over the average lifetime expectancy (80 years), consistent with Health Canada guidance (Health Canada 2009a). A composite

receptor is used to assess risk across all life stages combined over a lifetime according to the following age categories:

- infants (i.e., 0 to 6 months of age);
- toddlers (i.e., 7 months to 4 years of age);
- children (i.e., 5 to 11 years of age);
- adolescents (i.e., 12 to 19 years of age); and
- adults (i.e., greater than 20 years of age).

The assessment considered the specialized diets and lifestyles of Aboriginal Residents in the region, including the reliance on wild plants and animals as food sources and the use of local trap lines.

5.4.1.2 Chemicals Evaluated in the Risk Assessment

Comprehensive chemical screening processes were used to determine the COCs in each media (i.e., air, water, soil, sediment and food), as outlined below. Results of the chemical screening process are provided in Appendix II.

- For the Baseline Case, the screening entailed comparison between measured concentrations of contaminants in exposure media (i.e., air, water, soil, sediment and dietary items such as crops and fish) and environmental quality guidelines or regulations.
- For the Application Case, the screening entailed comparison between predicted concentrations of contaminants in exposure media (i.e., air, water, soil and dietary items such as game and fish) against both regulatory guidelines/standards and a 10 percent or greater increase above the maximum baseline concentration at any location. Detailed methods for the prediction of environmental concentrations in soil, air, and dietary items are provided in Appendix IV. No changes in sediment quality are expected as discussed in the Water Management Plan; Section 8 of the 2011 EIS Update (De Beers 2011).

Canadian environmental quality regulations and guidelines (e.g., CCME and Health Canada) were used preferentially to identify COCs. In the absence of Canadian environmental quality criteria for a particular substance, environmental quality criteria from other international regulatory jurisdictions (e.g., U.S. EPA) were used. Environmental quality regulations or guidelines used in this assessment are summarized below by media type with the exception of the air quality criteria which have already been presented in Section 5.2.2.1.1:

- Surface Water:
  - Health Canada (2008) – Guidelines for Canadian Drinking Water Quality.
  - United States Environmental Protection Agency (U.S. EPA 2012c) – *Regional Screening Levels. Regional Residential Tap Water Screening Levels for Chemical Contaminants at Superfund Sites*.
- Soil and Sediment:
  - Canadian Council of Ministers of the Environment (CCME 1999) – Canadian environmental quality guidelines for protection of soil and sediment.
  - United States Environmental Protection Agency (U.S. EPA 2012d) – Regional Screening Levels. Regional Residential Soil Screening Levels for Chemical Contaminants at Superfund Sites.
- Fish:
  - United States Environmental Protection Agency (U.S. EPA 2012e). *Region 3 Fish Tissue Screening Levels*; U.S. EPA Region III.

The COCs were identified as those substances in the Application Case that both exceeded the regulatory guideline/standard and exhibited a 10% or greater increase above the maximum Baseline concentration at any location. Comparison to regulatory values was considered to represent a conservative evaluation of the potential for the predicted concentrations to cause adverse effects. Comparison to Baseline concentrations was included in the screening procedure to evaluate whether a measureable Project-related impact on environmental quality was likely to occur. Given temporal variability, variability in sampling and laboratory methods, and the uncertainty inherent in estimates from water, air and soil quality models, any predicted increase of less than 10% above Baseline concentrations was considered unlikely to reflect a meaningful Project-related change in environmental quality.

Several metals that were COCs in the human health and/or ecological risk assessment are known to exist in two or more forms in environmental media. For example, chromium exists in two oxidation states (chromate and chromite), and arsenic exists in organic (arsenosugars) and inorganic (elemental arsenic) forms. These different forms can have very different bioavailability and very different toxicity. In the absence of information about which forms are present in environmental samples, the conservative assumption is that the entire amount is bioavailable and is present in the more-toxic form.

A summary of the COCs identified in the Application Case for further evaluation in the multi-media assessment is provided in Table 5.4-1. Table 5.4-1 also indicates the media for which each COC was identified.

Table 5.4-1 Chemicals of Concern Retained for Further Evaluation in the Multi-media Assessment Media

Parameter	Soil	Fish	Surface Water	Sediment	Plants and Game	Air
Metals						
Aluminum	–	–	–	–	–	√
Antimony	–	√	–	–	–	–
Arsenic	–	√	–	–	–	–
Cadmium	–	–	–	–	–	√
Cobalt	–	√	–	–	–	√
Iron	–	√	–	–	–	√
Manganese	–	–	–	–	–	√
Nickel	–	–	–	–	–	√
Thallium	–	√	–	–	–	–
Titanium	–	–	–	–		√
Vanadium	–	–	–	–	–	√

√ indicates that the parameter has been identified as a COC in a particular medium.

- = not identified as a COC.

5.4.1.3 Exposure Pathways Evaluated in the Risk Assessment

The objective of the exposure pathway screening process is to identify potential routes by which people could be exposed to chemicals and the relative significance of these pathways to total exposure. All potential pathways between chemicals and people were considered (see conceptual model in Figure 2.4-3). The air inhalation pathway was evaluated in the air quality risk assessment (Section 5.2). The exposure pathways evaluated in the multi-media risk assessment are listed in Table 5.4-2.

Table 5.4-2 Exposure Pathways Evaluated in the Multi-Media Risk Assessment

Exposure Pathway	Rationale
Inhalation of air	People may be exposed to airborne chemicals released to air from the Project.
Inhalation of dust	Airborne chemicals may deposit to soil and people may inhale soil dust particulates.
Ingestion of water	People may be exposed to waterborne chemicals via ingestion. It was assumed that potable water was surface water from Area 8 (potable water will be clarified by settlement and chlorinated), as municipally treated water is not expected to be available to Seasonal Users. Surface water for Area 8, which is to be used as the drinking water supply for the mine, was assumed to be the source of drinking water for both Seasonal Users and Gahcho Kué mine workers as it represents water quality of the lakes in the area. Water quality from Kennady Lake was used for incidental ingestion for workers who may come in contact with surface water when removing and/or stockpiling sediments from Kennady Lake
Dermal contact with water	People may be exposed to waterborne chemicals dermally. It was assumed that Seasonal Users and Gahcho Kué workers would come into contact with potable water (e.g., via showering). Gahcho Kué workers were also conservatively assumed to come into dermal contact with water from Kennady Lake while removing and/or stockpiling sediments. For the Seasonal User, dermal contact with surface water through traditional activities (e.g., fishing, hunting) was evaluated through dermal contact with potable water.
Ingestion of fish	Waterborne chemicals may bioaccumulate in fish and people may ingest the fish.
Ingestion of soil	Airborne chemicals may deposit to soil and people may incidentally ingest soil.
Dermal contact with soil	Airborne chemicals may deposit to soil and people may come into dermal contact with soil.
Ingestion of plants	People may consume plants that have received airborne deposition or that have taken up chemicals from the soil. Plants include traditional use plants and garden produce. Only Seasonal Users were assumed to be consuming traditional plants and garden produce.
Ingestion of game meat	People may consume animals harvested from areas near the Project. Caribou meat is a staple of the diet of Aboriginal people in the region and wild meat can be a significant component of their overall meat intake. Caribou and snowshoe hare meat was used to represent the meat ingestion pathway for Seasonal Users only.
Background dietary intake (i.e., ingestion of supermarket foods)	Background exposure from dietary sources (e.g., supermarket food) was included in the human health risk assessment for Seasonal Users and Gahcho Kué workers.

5.4.2 Toxicity Assessment

A toxicity assessment is the process of determining the amount of a chemical a person may take into his/her body through all applicable exposure pathways without risk of adverse health effects. This parameter is typically referred to as a TRV. For the multi-media risk assessment, TRVs for non-carcinogenic chemicals are called Reference Doses (RfDs) and TRVs for carcinogenic chemicals are called Slope Factors (SF) (Appendix III for further description of TRVs used in the assessment). Carcinogenicity classifications for COCs are also provided in Table 5.4-3.

Available TRVs from the following agencies were reviewed:

- Health Canada (Health Canada 2009b);
- United States Environmental Protection Agency (U.S. EPA) Integrated Risk Information System (U.S. EPA 2012b);

- World Health Organization (WHO 2000);
- Agency for Toxic Substances and Disease Registry (ATSDR 2012); and
- Netherlands National Institute of Public Health and the Environment (RIVM 2001, 2009).

Toxicity reference values were selected based on the currency of the study, study duration (i.e., chronic duration preferred) and whether the critical endpoint was based a no-observed-adverse effect level (NOAEL).

Although toxicity information for humans is selected wherever possible, the basis for many of the TRVs is laboratory toxicity studies conducted on test animals such as rats or mice. These studies provide dose-response information that is extrapolated to humans by applying safety factors. In most cases safety factors of 100 to 1,000 are applied to the highest concentration in a toxicity test where no chronic health effects were observed or measured to account for interspecies extrapolation and protection of the most susceptible in a population (e.g., toddlers and the elderly). Therefore, TRVs generally have large margins of safety so that the toxicity or risk of a chemical to people is not underestimated.

5.4.2.1 Carcinogenicity Classification

Carcinogenicity classifications for COCs are presented in Table 5.4-3.

Table 5.4-3 Carcinogenicity Classification for Chemicals of Concern in Multi-media Assessment

Compound	Health Canada	IARC	U.S. EPA	Assessed as a Carcinogen?
Metals				
Aluminum	ND	ND	ND	No
Antimony	ND	Group 2B (antimony trioxide); Group 3 (antimony trisulphide)	ND	No
Arsenic	Group 1	Group 1	Group A	Yes
Cadmium	Group II	Group I	Group B1 (inhalation only)	Yes (inhalation only)
Cobalt	ND	Group 2B	ND	Yes (inhalation only)
Iron	ND	ND	ND	No
Manganese	ND	ND	Group D	No
Nickel	Group VI (metallic)/ Group I (soluble)	Group 2B (metallic); Group I (nickel compounds)	Group A (refinery dust, nickel subsulphide); Group B2 (nickel carbonyl)	Yes
Thallium	ND	ND	ND	No
Titanium	ND	ND	ND	No
Vanadium	ND	Group 2B (vanadium pentoxide)	ND	No

Sources: Health Canada 2009b; IARC 2008; U.S. EPA 2012a.

ND = not determined.

5.4.2.2 Toxicity of Mixtures

Toxicity of chemical mixtures was addressed by summing hazard quotients and ILCR values for COCs that contribute to the same type of toxic effect as discussed in Section 5.4.3 below.

5.4.3 Exposure Assessment

An exposure assessment is the process of estimating the amount of a chemical that a person may take into his/her body (referred to as a dose) through all applicable exposure pathways. The dose of a chemical depends on the concentrations in various media (e.g., air, water, soil, food), the amount of time a person is in contact with these media and the biological characteristics of the person (e.g., ingestion rates, body weights, dietary preferences).

The multi-media assessment daily exposure is determined as a dose. This value is called the Estimated Daily Intake (EDI) and is typically expressed as milligrams per kilogram body weight (BW) per day (mg/kg BW/day).

Specific exposure assumptions, input values and equations are provided in Appendix IV. Example calculations are also provided in Appendix IV.

The following data were used in this assessment (Appendix IV):

- Predicted annual air deposition rates at receptor locations for the Baseline and Application cases.
- Predicted soil, plant and meat concentrations based on air emissions data for the Baseline and Application cases.
- Surface water concentrations predicted for Area 8 (which will be the source of the drinking water supply for the mine) and for Kennady Lake (incidental ingestion for Gahcho Kué workers only).
- Sediment concentrations measured during baseline surveys of Kennady Lake.
- Predicted fish tissue concentrations from the Project area.
- Background dietary intake values (i.e., for supermarket foods).

Two exposure scenarios were evaluated for the fish consumption pathway:

- High fish consumption: Fish consumption rates for First Nations' subsistence fishers (Health Canada 2009a) were used to calculate exposure dose for the fish ingestion pathway (conservative approach).
- Low fish consumption: A lower consumption rate was selected based on average seafood consumption for the general public, as recommended in Health Canada's Human Health Risk Assessment of Mercury (Health Canada 2007).

5.4.4 Risk Characterization

5.4.4.1 Risk Estimates

Long-term health effects were evaluated by calculating HQs for chemicals that do not cause cancer (non-carcinogens) and ILCR values for chemicals that are suspected to cause cancer (carcinogens).

In the risk characterization step, HQs were calculated for non-carcinogenic COCs as the ratio of the predicted exposure dose to the RfD, according to the following equation:

$$HQ = \frac{\text{estimated daily intake (mg/kg-day)}}{\text{RfD (mg/kg-day)}}$$

A hazard index (HI) is the sum of the HQ for all exposure pathways for each contaminant. The units of mg/kg-day refer to milligrams of substance per kilogram of body weight per day.

A hazard index (HI) that is less than or equal to 1.0 indicates that the estimated exposure is less than the reference dose, signifying negligible health effects.

Unlike non-carcinogens, cancer risk estimates are based on the ILCR, which is the additional cancer cases attributed to the incremental exposures to carcinogenic COCs released by the Project. Interpretation of these ILCRs was based on comparison of the calculated ILCR values with the "benchmark" of 1 in 100,000 (i.e., one extra cancer case in a population of 100,000 people). Health Canada considers cancer risks from chemical exposure to be essentially negligible if the ILCR is less than 1.0×10^{-5} (Health Canada 2009a). For carcinogenic COCs, ILCRs were calculated as the product of the predicted COC exposure dose and the oral slope factor, according to the following equation:

ILCR = estimated daily intake (mg/kg BW-day) x SF $(mg/kg\text{-}day)^{-1}$

When risk estimates exceed target risk thresholds (i.e., when the HI is greater than one or the ILCR is greater than 1.0×10^{-5}), the scenarios pose a potential concern and require further scrutiny. However, risk estimates greater than the target risk thresholds do not necessarily indicate that adverse health effects will occur as a large margin of safety has been included in their estimation.

The equations used to calculate risk estimates and example calculations are presented in Appendix IV.

5.4.4.1.1 Chemical Mixtures

The HIs for COCs for which TRVs were based on similar target organs were added together to determine a total HI for similar toxicological effects (Health Canada 2009a). Likewise, ILCRs are summed for carcinogenic COCs with the same target organ and form of cancer. The COCs for which HIs and ILCRs were summed by target organ endpoints are shown in Table 5.4-4. For details on the target organ(s) and effect(s) of each COC see Appendix III.

Table 5.4-4 Potential Additive Interactions of the Chemicals of Potential Concern for the Multi-Media Risk Assessment

Chemicals of Potential Concern		Target Organ	Effects
Non-carcinogens	Aluminum, manganese	nervous system	neurotoxicity
	Antimony, arsenic, cobalt, nickel, titanium and vanadium (inhalation exposure)	lungs	respiratory effects
Carcinogens	Arsenic, cadmium, cobalt and nickel	respiratory tract	tumours

5.5 ACUTE AIR QUALITY RISK ASSESSMENT RESULTS

5.5.1 Summary of Exposure Ratios

Hazard quotients (HQ) were calculated for parameters identified as COCs by comparing the concentration predicted for each location with toxicity benchmarks for the Baseline and Application cases.

The HQ values calculated for maximum 1-hour exposure to aluminum, iron and nickel for the Baseline Case and Application Case are presented in Table 5.5-1. A summary of 1-hour acute HQs is provided below:

- Hazard quotients were less than 1.0 for aluminum for the Baseline Case and Application Case at all locations except the Project Boundary (HQ=1.1) for the Application Case.
- Hazard quotients were less than 1.0 for iron for the Baseline Case and Application Case at all locations except the Employee Camp and the Project Boundary (HQ=5.2 and 10.3, respectively) for the Application Case.
- Hazard quotients were less than 1.0 for nickel for the Baseline Case and Application Case at all locations except the Employee Camp and Project Boundary (HQ=2.1 and 3.5, respectively) for the Application Case.
- Hazard quotients were less than 1.0 for benzo(a)pyrene for the Baseline Case and Application Case at all locations except the Employee Camp and Project Boundary (HQ=1.9 and 2.9, respectively) for the Application Case.

Table 5.5-1 Exposure Ratios for Maximum 1-Hour Predicted Concentrations at All Locations

Parameter	Air Threshold	Hazard Quotient (HQ)					
		Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary
Baseline Case							
Aluminum	50	0.00020	0.00019	0.00011	0.000025	0.000025	0.000028
Iron	10	0.0019	0.0018	0.0011	0.0002	0.0002	0.0003
Nickel	0.2	0.0015	0.0015	0.00095	0.00017	0.00016	0.00018
Benzo(a)pyrene	0.00015	0.0052	0.0047	0.0027	0.00086	0.00095	0.0010
Application Case							
Aluminum	50	0.00075	0.00089	0.00059	0.55	0.0677	**1.1**
Iron	10	0.0070	0.0083	0.0056	**5.2**	0.63	**10.3**
Nickel	0.2	0.0028	0.0033	0.0022	**2.1**	0.23	**3.5**
Benzo(a)pyrene	0.00015	5.21E-03	4.67E-03	2.71E-03	**1.9**	2.75E-01	**2.9**

Notes: Bold and shaded values exceed the target hazard quotient of 1.
Units are in $\mu g/m^3$.

The HQ values calculated for maximum 24-hour exposure to cadmium, iron, manganese and nickel for the Baseline Case and Application Case are presented in Table 5.5-2. A brief summary is provided below:

- Hazard quotients were less than 1.0 for cadmium for the Baseline Case and Application Case at all locations except the Project Boundary for the Application Case (HQ=2.1).
- Hazard quotients were less than 1.0 for iron for the Baseline Case and Application Case at all locations except the Employee Camp and the Project Boundary for the Application Case (HQ=1.8 and 5.8, respectively).
- Hazard quotients were less than 1.0 for manganese for the Baseline Case and Application Case at all locations except the Employee Camp and Project Boundary for the Application Case (HQ=1.1 and 3.4, respectively).
- Hazard quotients were less than 1.0 for nickel for the Baseline Case and Application Case at all locations except the Project Boundary for the Application Case (HQ=1.6).
- Hazard quotients were less than 1.0 for acrolein for the Baseline Case and Application Case at all locations except the Employee Camp and the Project Boundary for the Application Case (HQ=2.7 and 4.2, respectively).
- Hazard quotients were less than 1.0 for benzo(a)pyrene for the Baseline Case and Application Case at all locations except the Employee Camp and the Project Boundary for the Application Case (HQ=2.8 and 4.3, respectively).
- The sum of the acrolein, nitrogen dioxide, iron and nickel HQ values for respiratory effects was less than 1.0 for the Baseline Case and Application Case at all locations except the Employee Camp and the Project Boundary (HQ=6.3 and 13, respectively) for the Application Case.

Table 5.5-2 Hazard Quotients for 24-Hour Predicted Concentrations at All Locations

Parameter	Air Threshold	Hazard Quotient (HQ)					
		Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary
Baseline Case							
Cadmium	0.025	0.0012	0.0018	0.0011	0.00030	0.00042	0.00041
Iron	4	0.00088	0.0010	0.00075	0.00017	0.00025	0.00025
Manganese	0.1	0.00059	0.00069	0.00050	0.00011	0.00017	0.00017
Nickel	0.1	0.00057	0.00057	0.00045	0.00010	0.00014	0.00014
Nitrogen Dioxide	200	0.049	0.056	0.051	0.036	0.039	0.038
Acrolein	0.40	0.0055	0.011	0.0055	0.0029	0.0039	0.0036
Benzo(a)pyrene	0.0001	0.00079	0.0012	0.00079	0.00036	0.00051	0.00048
Sum Respiratory Effects	-	0.056	0.068	0.058	0.039	0.043	0.042
Application Case							
Cadmium	0.025	0.001	0.0018	0.0011	0.8	0.05	**2.1**
Iron	4	0.003	0.003	0.003	**1.8**	0.2	**5.8**
Manganese	0.1	0.002	0.002	0.0016	**1.1**	0.13	**3.4**
Nickel	0.1	0.001	0.0010	0.000847	0.9	0.0694	**1.6**
Nitrogen Dioxide	200	0.049	0.056	0.051	0.85	0.27	**1.1**
Acrolein	0.40	0.054	0.05	0.030	**2.7**	0.40	**4.2**
Benzo(a)pyrene	0.0001	0.0078	0.0070	0.0041	**2.8**	0.41	**4.3**
Sum Respiratory Effects	-	0.11	0.11	0.084	**6.3**	0.97	**13**

Notes: Bold and shaded values exceed the target hazard quotient of 1.0.
Units are in $\mu g/m^3$.

5.5.2 Further Analyses of Parameters with Exposure Ratios Greater than One

For parameters and locations where HQ values were greater than 1.0 for the Application Case, the frequency of exceedances of the 1-hour and 24-hour peak (i.e., maximum) concentrations of each COC over the course of a year was calculated to determine the magnitude of the risk. The frequency of exceedances for COCs with HQs greater than 1.0 is summarized in Table 5.5-3 (1-hour) and Table 5.5-4 (24-hour).

Results of the magnitude of risks assessments for the acute air quality assessment (1-hour and 24-hour) are presented in Tables 5.5-5 to 5.5-14.

Table 5.5-3 Predicted 1-Hour Summary Statistics for Baseline Case and Application Case

Parameter/ Location	Acute Exposure Limit [µg/m^3]	Baseline Case		Application Case			
		1-Hour Peak[(a)] [µg/m^3]	Frequency of Exceedance (Number of Exceedances in a Year)	Peak 1-Hour [µg/m^3]	95th Percentile [µg/m^3]	75th Percentile [µg/m^3]	Frequency of Exceedance (Number of Exceedances in a Year)
Aluminum							
Project Boundary	50	0.0014	0	55	8.80	2.92	2
Iron							
Employee Camp	10	0.0023	0	51.7	3.16	0.354	115
Project Boundary	10	0.0026	0	103	16.5	5.48	1,058
Nickel							
Employee Camp	0.2	0.000034	0	0.42	0.0359	0.0039	22
Project Boundary	0.2	0.000036	0	0.69	0.1106	0.0376	112
Benzo(a)pyrene							
Employee Camp	0.00015	0.00025	0	0.00028	0.000029	0.000004	46
Project Boundary	0.00015	0.00030	0	0.00043	0.00011	0.000023	246

(a) Peak concentration is based on the highest value predicted at a given receptor location for the maximum year.

Table 5.5-4 Predicted 24-Hour Summary Statistics for Baseline Case and Application Case

Parameter/ Location	Acute Exposure Limit [µg/m^3]	Baseline Case		Application Case			
		24-Hour Peak[(a)] [µg/m^3]	Frequency of Exceedance (Number of Exceedances in a Year)	24-Hour Peak [µg/m^3]	95th Percentile [µg/m^3]	75th Percentile [µg/m^3]	Frequency of Exceedance (Number of Exceedances in a Year)
Cadmium							
Project Boundary	0.025	0.000010	0	0.052	0.022	0.0081	13
Iron							
Employee Camp	4	0.00068	0	7.32	2.85	0.854	8
Project Boundary	4	0.00098	0	23.3	13.6	6.4	143
Manganese							
Employee Camp	0.1	0.000011	0	0.11	0.042	0.013	2
Project Boundary	0.1	0.000017	0	0.34	0.196	0.095	87
Nickel							
Project Boundary	0.1	0.00014	0	0.16	0.092	0.045	15
Nitrogen Dioxide							
Project Boundary	200	7.6	0	224.4	101.9	82.8	2
Acrolein							
Employee Camp	0.4	0.00117	0	0.483	0.136	0.039	1
Project Boundary	0.4	0.0014	0	1.09	0.301	0.111	13
Benzo(a)pyrene							
Employee Camp	0.0001	0.0000000358	0	0.000123	0.000019	0.000008	1
Project Boundary	0.0001	0.0000000477	0	0.000280	0.000079	0.000029	14

(a) Peak concentration is based on the highest value predicted at a given receptor location for the maximum year.

Table 5.5-5 Further Analysis of Acrolein and Determination of Magnitude of Risk (Acute 24-hour Assessment)

Analysis Criteria	Discussion
Comparison of 75th and 95th percentiles, and peak concentrations to acute limits	**Acute 24-hour Application Case:** The peak predicted 24-hour concentrations at the Employee Camp (0.483 µg/m^3) and the Project Boundary (1.09 µg/m^3) exceeded the acute exposure limit of 0.4 µg/m^3. The 75th and 95th percentile concentrations did not exceed the acute exposure limit at either location.
Frequency of exceedance	There was 1 predicted daily exceedance of the threshold at the Employee Camp and 13 predicted exceedances at the Project Boundary.
Conservatism and uncertainty in air predictions	This project and all existing and approved developments were assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time.
Conservatism in the acute limits for acrolein	The selected 24-hour acute limit for acrolein (0.40 µg/m^3) is health-based and is from the Ontario Ministry of Environment (OMOE). The OMOE derived a threshold based on olfactory epithelial pathology in a rat study. The NOAEL was 0.6 ppm and the estimated human equivalent concentration (HEC) NOAEL was 11 µg/m^3. An uncertainty factor of 30 was applied to the HEC NOAEL (3 for interspecies extrapolation and 10 to protect sensitive individuals).
Potential acute health effects of acrolein	Eye irritation is the most common endpoint in humans following acute exposure to acrolein (OMOE 2004). The lowest concentration at which eye irritation was reported was 137 µg/m^3 for 5 minutes (Darley et al. 1960). Nose and throat irritation and other respiratory effects typically occur at higher concentrations than eye irritation (>340 µg/m^3).
Magnitude of risk	The predicted peak 24-hour exposure concentration for acrolein in the Application Case at the Employee Camp and the Project Boundary exceeded the acute exposure limit and was higher than the Baseline Case. For Employee Camp, only 1 exceedance was predicted and the peak concentration is predicted to be only slightly above the threshold. At the Project Boundary, the 24-hour exposure concentration is predicted to be almost three times the threshold; however, receptors are not expected to be present at this location frequently. In addition, 75th and 95th percentile concentrations were less than the acute exposure limit for all receptor locations for the Baseline Case and the Application Case. The above suggests that it is unlikely that someone would experience health effects based on the predicted concentrations at these receptor locations. Based on this information the magnitude of risk for acrolein is considered to be negligible.

Table 5.5-6 Further Analysis of Aluminum and Determination of Magnitude of Risk (Acute 1-hour Assessment)

Analysis Criteria	Discussion
Comparison of 75th and 95th percentiles, and peak concentrations to 1-hour acute limits	**Acute 1-hour, Application Case:** The 75th and 95th percentiles do not exceed the 1-hour threshold at any of the locations for the baseline and application case. The application case peak concentration (55 µg/m^3) exceeded the 1-hour acute limit of 50 µg/m^3
Frequency of exceedances	Predictions indicated that the 1-hour acute threshold would only be exceeded a maximum of twice per year at the Project Boundary.
Conservatism and uncertainty in air predictions	This project and all existing and approved developments are assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staged over time.
Conservatism in the acute threshold for aluminum	The TCEQ 1-hour threshold (50 µg/m^3) used in this assessment is based on a health endpoint; however, details regarding the derivation methods were not available, therefore the level of conservatism is unknown.
Potential acute health effects of aluminum	No supporting documentation for the basis of the TCEQ threshold is available and no other jurisdictions provide information regarding the potential acute health effects of aluminum.
Magnitude of risk	The predicted peak 1-hour exposure concentration for aluminum exceeds the selected air quality criteria (i.e., TCEQ threshold) only marginally. Due to the infrequency of exceedances and the low likelihood of a receptor being present at the Project Boundary, the magnitude of risk for aluminum is considered to be negligible.

Table 5.5-7 Further Analysis of Benzo(a)pyrene and Determination of Magnitude of Risk (Acute 1-hour and 24-hour Assessment)

Analysis Criteria	Discussion
Comparison of 75th and 95th percentiles, and peak concentrations to acute limits	**Acute 1-hour Application Case:** The predicted peak 1-hour concentrations for the Employee Camp and Project Boundary (0.00028 and 0.00043 μg/m^3, respectively) exceeded the acute exposure limit of 0.00015 μg/m^3. The 75th and the 95th percentile concentrations were below the 1-hour threshold at all locations. **Acute 24-hour Application Case:** The predicted peak 24-hour concentrations for the Employee Camp and Project Boundary (0.000123 and 0.00028 μg/m^3, respectively) exceeded the acute exposure limit of 0.0001 μg/m^3. The 75th and the 95th percentile 24-hour concentrations were below the screening threshold at all locations.
Frequency of exceedance	**Acute 1-hour Application Case:** There were 46 predicted hourly exceedances of the threshold at the Employee Camp and 246 predicted hourly exceedances of the threshold at the Project Boundary. **Acute 24-hour Application Case:** There was 1 predicted exceedance of the 24-hour threshold at the Employee Camp and 14 predicted exceedances of at the Project Boundary.
Conservatism and uncertainty in air predictions	This project and all existing and approved developments were assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time.
Conservatism in the acute threshold for benzo(a)pyrene	**Acute 1-hour Application Case:** The OMOE threshold of 0.00015 μg/m^3 is a half-hour average standard based on carcinogenicity associated with exposure to PAH compounds. It was derived by applying a conversion factor of 15 to the annual ambient air quality criterion (AAQC). The AAQC is based on a WHO (2000) evaluation of coke-oven workers epidemiological studies that derived an inhalation unit risk (IUR) of 0.000085 ng/m^3 (BaP as a surrogate for total PAHs at a 1x10^{-5} risk level) equivalent to 0.1 ng/m^{3}of BaP. The carcinogenic threshold was used in the absence of an acute non-carcinogenic value. The use of a scaled-chronic threshold to protect against acute health effects is a conservative approach and of the jurisdictions reviewed for air quality criteria, the approach was found to be unique to OMOE. Carcinogenic endpoints are generally not used to assess acute exposure as they are more typically utilized for the assessment of chronic endpoints. The use of a carcinogenic endpoint is conservative for the assessment of acute exposure. Predicted annual (i.e., chronic) concentrations for benzo(a)pyrene are below the chronic threshold (see Appendix I, Table I-6). **Acute 24-hour Application Case:** The chronic OMOE threshold of 0.0001 μg/m^3 (based on a 1x10^{-5} risk level) was used to evaluate acute (24-hour) exposure, as a 24-hour AAQC was not available. The OMOE threshold is based on a carcinogenic potential endpoint based on the WHO (2000) evaluation of coke-oven workers epidemiological studies (as described above). Carcinogenic endpoints are used for annual data and for this project the annual data for benzo(a)pyrene is below the threshold therefore it is conservative to apply this threshold to the acute data. In addition, annual concentrations are below the chronic threshold.
Potential acute health effects of benzo(a)pyrene	Information on potential acute health effects of benzo(a)pyrene via inhalation was not available.
Magnitude of risk	**Acute 1-hour Application Case:** The predicted peak 1-hour exposure concentration for benzo(a)pyrene exceeded the selected air quality criteria 36 hours of the year at the Employee Camp and 246 hours at the Project Boundary. Receptors are not expected to be at the Project Boundary and, additionally, the predicted 95th and 75th percentile concentrations are less than those where health effects were observed. In addition, the acute threshold is actually based on a carcinogenic endpoint, and predicted annual concentrations are below the carcinogenic chronic threshold. Based on this information the magnitude of risk for benzo(a)pyrene is considered to be low. **Acute 24-hour Application Case:** The predicted peak 24-hour exposure concentration for benzo(a)pyrene exceeded the selected air quality criteria (i.e., the OMOE chronic threshold) but only on 1 day of the year at the Employee Camp and on 14 days at the Project Boundary. Receptors are not expected to be at the Project Boundary and, additionally, the predicted 95th and 75th percentile concentrations are less than those where health effects were observed. Based on this information the magnitude of risk for benzo(a)pyrene is considered to be negligible.

Table 5.5-8 Further Analysis of Cadmium and Determination of Magnitude of Risk (Acute 24-hour Assessment)

Analysis Criteria	Discussion
Comparison of 75th and 95th percentiles, and peak concentrations to acute limits	**Acute 24-hour Application Case:** The peak predicted 24-hour concentration of cadmium at the Project Boundary (0.052 µg/m^3) exceeded the acute exposure limit of 0.025 µg/m^3. The 75th and 95th percentile concentrations were below the threshold.
Frequency of exceedance	There were 13 predicted daily exceedances of the threshold at the Project Boundary.
Conservatism and uncertainty in air predictions	This project and all existing and approved developments were assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time.
Conservatism in the acute threshold for cadmium	The OMOE 24-hour threshold (0.025 µg/m^3) used in this assessment is based on health endpoints from occupational exposure studies extrapolated to low doses.
Potential acute health effects of cadmium	The OMOE 24-hour threshold is based on kidney effects and carcinogenicity in humans from occupational exposure studies.
Magnitude of risk	The predicted peak 24-hour exposure concentration for cadmium exceeds its selected air quality criterion only marginally (approximately 2x), and infrequently at the Project Boundary. Based on this information the magnitude of risk for cadmium is considered to be negligible.

Table 5.5-9 Further Analysis of Iron and Determination of Magnitude of Risk (Acute 1-hour and 24-hour Assessments)

Analysis Criteria	Discussion
Comparison of 75th and 95th percentiles, and peak concentrations to acute limits	**Acute 1-hour, Application Case:** The predicted 1-hour peak concentration at the Employee camp (51.7 µg/m^3) and the 95th percentile (16.5 µg/m^3) and peak (103 µg/m^3) concentrations at the Project Boundary exceeded the acute exposure limit of 10 µg/m^3. The 75th and 95th percentile concentrations at the Employee camp, and the 75th percentile concentration at the project boundary were below the exposure limit. **Acute 24-hour, Application Case**: The 75th percentile, the 95th percentile, and the 24-hour peak concentration (6.4 µg/m^3, 13.6 µg/m^3, and 23.3 µg/m^3, respectively) exceeded the acute exposure limit of 4 µg/m^3 at the Project Boundary. Only the peak 24-hour concentration (7.32 µg/m^3) exceeded the threshold at the Employee Camp.
Frequency of exceedance	**Acute 1-hour, Application Case::** There were a maximum of 115 predicted yearly exceedances of the 1-hour threshold at the Employee Camp and 1,058 predicted yearly exceedances of the 1-hour threshold at the Project Boundary. **Acute 24-hour, Application Case:** There were 8 days at the Employee Camp and 143 days at the Project Boundary for which exceedances of the 24-h threshold were predicted in the worst case year.
Conservatism and uncertainty in air predictions	This Project and all existing and approved developments are assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staged over time.
Conservatism in the acute threshold for iron	The TCEQ 1-hour threshold (10 µg/m^3) used in this assessment is based on a health endpoint; however, supporting documentation was not available. The OMOE 24-hour standard (4 µg/m^3) is based on a health-based endpoint; however, supporting documentation was not available. Due to the lack of supporting documentation, the conservatism incorporated into the derivation of the iron acute thresholds is unknown.
Potential acute health effects of iron	No supporting documentation for the basis of the 1-hour TCEQ threshold and 24-hour OMOE standard is available. Acute inhalation of ferric salts as dusts and mists include irritation of the respiratory tract and irritation of the skin (HSDB 2010).
Magnitude of risk	The predicted peak 1-hour and 24-hour exposure concentrations for iron exceed the selected air quality criteria at the Employee Camp and the peak and upper percentile concentrations exceeded the criteria at the Project Boundary. Due to the infrequency and relatively minor magnitude of exceedances (<10x the 1hr threshold, <2x the 24hr threshold) at the Employee Camp, and the low likelihood of a receptor being present at the Project Boundary, the magnitude of risk for iron is considered to be low.

Table 5.5-10 Further Analysis of Manganese and Determination of Magnitude of Risk (Acute 24-hour Assessment)

Analysis Criteria	Discussion
Comparison of 75th and 95th percentiles, and peak concentrations to acute limits	**Acute 24-hour Application case:** The 95th percentile and peak predicted 24-hour concentrations (0.196 µg/m^{3}and 0.34 µg/m^3, respectively), exceed the threshold of 0.1 µg/m^3 at the Project Boundary. At the Employee Camp, the peak concentration (0.11 µg/m^3) only slightly exceeded the threshold, and the 95th percentile concentration was below the threshold.
Frequency of exceedance	There were 2 predicted daily exceedances of the threshold at the Employee Camp and 87 predicted daily exceedances at the Project Boundary.
Conservatism and uncertainty in air predictions	This project and all existing and approved developments were assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time.
Conservatism in the acute threshold for manganese	The OMOE 24-hour threshold (0.1 µg/m^3) used in this assessment is based on a neurological endpoint from two occupational studies which estimated the level of occupational exposure to manganese associated with the appearance of subtle neurological deficits. The study measured the eye-hand coordination impairment in workers exposed to MnO_2 within the respirable fraction defined as $PM_{2.5}$ for 5.3 years. The study was subchronic and the threshold was set by applying a safety factor to set a chronic air quality criterion as the 24-hour threshold. The use of a scaled-chronic threshold to protect against acute health effects is a conservative approach and of the jurisdictions reviewed for air quality criteria, the approach was found to be unique to OMOE.
Potential acute health effects of manganese	There is no information available about the acute (1- or 24-hour exposure duration) effects of inhaled manganese in humans. Inflammatory response of the lungs has been reported in acute studies with laboratory animals. According to the OMOE, the lowest no observable adverse effect concentration of 2,800 µg/m^{3}was reported after mice were exposed to a manganese compound for 2 hours. Chronic studies have shown that exposure to elevated levels of manganese can result in accumulations within the basal ganglia of the central nervous system. Symptoms of this condition usually result from high levels of manganese (1,000 µg/m^3) and include, but are not limited to, irritability, changes in mood, aggression, loss of facial expression and staggered gait. The threshold for manganese is based on two occupational studies which found that subtle neurological deficits began to occur at 30 to 50 µg/m^3. The 24 hour threshold is based on the $PM_{2.5}$ fraction of manganese with neurotoxicity as the critical endpoint.
Magnitude of risk	The predicted peak 24-hour and the 95th percentile exposure concentration for manganese, exceeded the selected air quality criteria (i.e., OMOE threshold) at the Project boundary; however, the magnitude of exceedance was low (<5x) and infrequent, and receptors are not expected to be present at the project boundary for any length of time. At the Employee camp, the peak 24-hour concentration exceeded the screening threshold only marginally (<2x) and very infrequently (2 times in the worst case year). Based the predicted concentrations and conservative screening threshold, the magnitude of risk for manganese is considered to be low.

Table 5.5-12 Further Analysis of Nickel and Determination of Magnitude of Risk (Acute 1-hour and 24 hour Assessments)

Analysis Criteria	Discussion
Comparison of 75th and 95th percentiles, and peak concentrations to acute limits	**Acute 1-hour, Application Case:** The 75th and 95th percentiles for the Employee Camp and Project Boundary did not exceed the acute exposure limit (0.2 μg/m^3). The peak concentrations for the Employee Camp and Project Boundary (0.42 and 0.69 μg/m^3, respectively) exceeded the acute exposure limit. **Acute 24-hour, Application Case:** The peak for the Project Boundary (0.16 μg/m^3) exceeded the acute exposure limit of 0.1 μg/m^3. None of the 24-hour predicted concentrations exceeded the threshold at the Employee Camp.
Frequency of exceedance	**Acute 1-hour, Application Case:** There were 22 predicted hourly exceedances of the threshold at the Employee Camp and 112 predicted hourly exceedances at the Project Boundary in the worst case year. **Acute 24-hour, Application Case:** There were 15 days at the Project Boundary when exceedances of the threshold were predicted.
Conservatism and uncertainty in air predictions	This project and all existing and approved developments were assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time.
Conservatism in the acute threshold for nickel	The California OEHHA 1-hour threshold (0.2 μg/m^3) used in this assessment is based immune system effects on mice. There is some degree of conservatism associated with extrapolating a threshold from a mouse study to humans. An uncertainty factor of 1000 was applied to the extrapolated one hour concentration of 233 μg/m^3 to derive the 1-hour REL (SQRT10) because the BMDL was calculated for a benchmark response rate which was considered to be clearly measurable and biologically significant response, 10 for interspecies variability, and 30 for intraspecies variability). The OMOE 24-hour threshold (0.1 μg/m^3) is based on carcinogenic and non-carcinogenic effects on the respiratory system from nickel as component of PM_{10}. The 24-hour value was derived from the annual screening value (0.02 μg/m^3) with the application of a conversion factor of 5, based on empirical monitoring data, ratios of concentrations observed for different averaging times and meteorological considerations. The use of a scaled-chronic threshold to protect against acute health effects is a conservative approach and of the jurisdictions reviewed for air quality criteria, the approach was found to be unique to OMOE.
Potential acute health effects of nickel	Acute effects in humans and experimental animals exposed to nickel in the air include lung lesions, decreased lung function and immunotoxicity. The California OEHHA 1-hour threshold is based on immune system effects on mice. The OMOE 24 hour threshold is based on the annual screening value which is protective of both carcinogenic and non-carcinogenic health effects.
Magnitude of risk	The predicted peak air concentrations for nickel exceeded the 1-hour threshold at the Employee Camp and the Project Boundary, and the 24 hour threshold at the Project Boundary. However, the 75th and 95th percentile concentrations were below screening thresholds at all locations. In addition, it is unlikely that a receptor would be present at the Project Boundary through the year. Based on this information the magnitude of risk for nickel is considered to be low.

Table 5.5-13 Further Analysis of NO_2 and Determination of Magnitude of Risk (Acute 24-hour Assessment)

Analysis Criteria	Discussion
Comparison of 75th and 95th percentiles and peak concentrations to acute limits	**Acute 24-hour Application Case:** The 75th and 95th percentile concentrations predicted at the Project Boundary did not exceed the acute exposure limit of 200 µg/m^3. The peak (224.4 µg/m^3) slightly exceeded the acute exposure limit.
Frequency of exceedance	There were 2 predicted daily exceedances of the threshold at the Project Boundary in the worst case year.
Conservatism and uncertainty in air predictions	This project and all existing and approved developments were assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time.
Conservatism in the acute threshold for NO_2	The NWT 24-hour threshold (200 µg/m^3) used in this assessment was adopted from the Canadian National Ambient Air Quality Objectives. This threshold is not health based but was derived from a maximum acceptable limit at which odour is perceived. Additional information on the derivation of this threshold was not found; however, the use of an aesthetic threshold instead of a health threshold can be a conservative approach.
Potential acute health effects of NO_2	The peak concentration in the application case is predicted to be only slightly higher than the odour-based threshold (200 µg/m^3) on 2 days of the year. The 75th and 95th percentiles are predicted to be below the threshold.
Magnitude of risk	The predicted peak 24-hour exposure concentration for NO_2 slightly (<2x) exceeds the selected air quality criteria (i.e., NWT threshold) on 2 days of the year at the Project Boundary. Receptors are not expected to be at the Project Boundary and, additionally, the predicted 95th and 75th percentile concentrations are less than those where odour is detected. Based on this information the magnitude of risk for NO_2 is considered to be negligible.

Table 5.5-14 Further Analysis of Sum of Exposure Ratios (24-hour) for Respiratory Irritants (Nitrogen Dioxide, Iron, Nickel and Acrolein) and Determination of Magnitude of Risk

Analysis Criteria	Discussion
Magnitude of Risk Estimate	The HQ values were greater than 1.0 in the Application Case for the Employee Camp (HQ=6.3) and the Project Boundary (HQ=12.7).
Conservatism and uncertainty in air predictions	This Project and all Baseline developments were assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time.
Conservatism in the acute limits for nitrogen dioxide, iron, nickel and acrolein	For Nitrogen dioxide the NWT 24-hour threshold (200 µg/m^3) used in this assessment was adopted from the Canadian National Ambient Air Quality Objectives. This threshold is not health based but was derived from a maximum acceptable limit at which odour will be perceived. Additional information on the derivation of this threshold was not located; however, the use of an aesthetic threshold instead of a health threshold can be a conservative approach. For iron, the OMOE 24-hour threshold (4 µg/m^3) is based on a health endpoint. Due to the lack of supporting documentation, the conservatism incorporated into the derivation of the iron acute threshold is unknown. The OMOE 24-hour threshold (0.1 µg/m^3) for nickel is based on carcinogenic and non-carcinogenic effects on the respiratory system of nickel as a metal/parameter in PM_{10}. The 24-h value was derived from the annual screening value (0.02 µg/m^3) and a conversion factor of 5, which is based on empirical monitoring data, ratios of concentrations observed for different averaging times and meteorological considerations. There is often conservatism associated with the use of an annual value in determining a 24 hour threshold. The selected 24-hour acute limit for acrolein (0.40 µg/m^3) is health-based and is from the Ontario Ministry of Environment (OMOE). The OMOE derived a threshold based olfactory epithelial pathology in a rat study. The NOAEL was 0.6 ppm and the estimated human equivalent concentration (HEC) NOAEL was 11 µg/m^3. An uncertainty factor of 30 was applied to the HEC NOAEL (3 for interspecies extrapolation and 10 to protect sensitive individuals).
Potential acute health effects of nitrogen dioxide, iron, nickel and acrolein	**Nitrogen dioxide** The peak concentration in the application case is predicted to be only slightly higher than the odour-based threshold (200 µg/m^3) on 2 days of the year. The 75th and 95th percentiles are predicted to be below the threshold. **Iron** Supporting documentation on the derivation of the iron threshold was not available (OMOE 2012b). Acute inhalation of ferric salts as dusts and mists include irritation of the respiratory tract and irritation of the skin (HSDB 2010). **Nickel** Acute effects in humans and experimental animals exposed to nickel in the air include lung lesions, decreased lung function and immunotoxicity. The California OEHHA 24 hour threshold is based on the annual screening value which is based on carcinogenic and non-carcinogenic health effects. **Acrolein** The peak acrolein concentration at the Employee Camp is only slightly higher than the threshold and an exceedance of the threshold is only expected to occur once in the year. At the Project Boundary, the peak concentration is almost 3 times the threshold; however, receptors are not expected to be in this location. In addition, the 75th and the 95th percentiles did not exceed the threshold at any of the locations.
Magnitude of risk	The HQ values increase from the Baseline Case to the Application Case and are greater than 1.0 for the Employee Camp and the Project Boundary. The primary contributors to the HQ values for respiratory irritants are iron and acrolein. The predicted peak 24-hour exposure concentration for NO_2 exceeds the selected air quality criteria (i.e., NWT threshold) but only on 2 days of the year at the Project Boundary. Receptors are not expected to be at the Project Boundary and, additionally, the predicted 95th and 75th percentile concentrations are less than those where health effects were observed. Based on this information the magnitude of risk for respiratory irritant COCs is considered to be low.

5.5.3 Residual Effect Classification

Residual effects to human health for short-term (acute) exposure to emissions from the Project are classified in Table 5.5-15. The effect classification criteria are already incorporated into the risk estimates as described in Section 2.4.1; therefore, residual effects are defined by the magnitude of risk as determined from risk estimates.

Table 5.5-15 Residual Effect Classification for Acute Inhalation Exposure for the Application Case

Parameter	Location	Magnitude of Chronic Risks as a Result of the Project
Acute Inhalation Risk Assessment(1-hour)		
Aluminum	Employee Camp, Project Boundary	negligible
Benzo(a)pyrene	Employee Camp, Project Boundary	low
Iron	Employee Camp, Project Boundary	low
Nickel	Employee Camp, Project Boundary	low
Remainder of COCs	all locations	negligible
Acute Inhalation Risk Assessment(24-hour)		
Acrolein	Employee Camp, Project Boundary	negligible
Benzo(a)pyrene	Employee Camp, Project Boundary	negligible
Cadmium	Project Boundary	negligible
Iron	Employee Camp, Project Boundary	low
Manganese	Project Boundary	low
Nickel	Project Boundary	low
Nitrogen dioxide	Project Boundary	negligible
Respiratory Irritants (acrolein, nitrogen dioxide, iron and nickel)	Employee Camp, Project Boundary	low
Remainder of COCs	all locations	negligible

5.5.3.1 Prediction Confidence

This acute health risk assessment was based on many layers of safety, including the following:

- peak (i.e., maximum) predicted ambient air concentrations based on conservative modelling methods;
- maximum exposure durations such that people are exposed to peak predicted air concentrations 24-hours/day for 365 days per year for a lifetime; and
- evaluation of chemical mixtures and summing HQ values where applicable.

Collectively, these assumptions contribute to HQ values that overestimate the true risk that is likely to be caused by the Application Case.

5.6 CHRONIC AIR QUALITY RISK ASSESSMENT RESULTS

For the chronic inhalation of non-carcinogens, the HQ values calculated for chronic exposure to air COCs. Results are presented for the Baseline and Application Cases for comparison purposes.

Table 5.6-1 below provides the HQ values for non-carcinogenic COCs.

Table 5.6-1 Hazard Quotients for Annual Predicted Concentrations at All Locations for Non-Carcinogens

Parameter	Reference Concentration	Hazard Quotients					
		Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary
Baseline Case							
Nitrogen dioxide	60	0.1	0.1	0.1	0.1	0.1	0.1
Acrolein	0.02	0.007	0.008	0.006	0.003	0.003	0.003
Application Case							
Nitrogen dioxide	60	0.1	0.1	0.1	0.9	0.2	1.0
Acrolein	0.02	0.009	0.01	0.007	**1.6**	0.1	**4.9**

Notes: Units are in µg/m^3

Bold and shaded values exceed the target hazard quotient of 1.0.

n/a = Not applicable.

For non-carcinogens, HQ values are equal to or less than 1.0 in the Application Case, with the exception of acrolein at the Employee Camp (HQ=1.6) and at the Project Boundary (HQ=4.9). Further analyses of the potential risks posed by acrolein are provided below in Table 5.6-2.

No chronic air quality COCs with carcinogenic effects were identified for the Project.

Table 5.6-2 Further Analysis (Chronic Effects) of Acrolein and Determination of Magnitude of Risk

Analysis Criteria	Discussion
Magnitude of risk estimate	The HQ values were less than 1.0 at all locations except for the Employee Camp and Project Boundary where the values were 1.6 and 4.9, respectively, for the Application Case.
Comparison of Application Case to Baseline Case	The largest increase in HQ values was 4.9 between the Baseline Case and Application Case for the Project Boundary. The increase in HQ value for the Employee Camp was 1.6 between the Baseline Case and Application Case.
Conservatism and uncertainty in air predictions	The Project and all Baseline developments were assumed to operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time.
Conservatism in the exposure assumptions	The exposure assessment was conducted using the assumption that receptors are exposed to the maximum predicted annual average concentrations 24-hours/day for 365 days/year for a lifetime.
Conservatism in the toxicity reference value for acrolein	The HQ values calculated for acrolein were determined using the U.S. EPA IRIS RfC of 0.02 µg/m^3. The U.S. EPA IRIS (U.S. EPA (2012b) derived the RfC for acrolein based on a LOAEL of 0.9 mg/m^3 (0.4 ppm) for nasal lesions in male and female rats exposed to acrolein for 6 hours/day, 5 days/week for 13 weeks. The LOAEL was adjusted for continuous exposure. An uncertainty factor of 1,000 was applied (3 for use of a minimal LOAEL rather than a NOAEL, 3 for interspecies extrapolation using dosimetric adjustments, 10 for extrapolation from subchronic to chronic duration, and 10 to account for human variability and sensitive subpopulations). No other regulatory jurisdiction was identified that had a chronic toxicity reference value for acrolein.
Potential health effects	The target organ for chronic toxicity of acrolein is the respiratory tract. Chronic exposure to acrolein typically results in respiratory congestion and irritation of the eyes, nose and throat. Repeated exposures to acrolein can cause damage to the epithelium of the nasal cavity (ATSDR 2012; U.S. EPA 2012b). No studies on human subjects were found in the literature reviewed. A variety of animal studies have reported nasal lesions and epithelium damage caused by chronic acrolein exposure (ATSDR 2007). The TRV for acrolein (0.02 µg/m^3; U.S. EPA 2012b) is based on the incidence of nasal lesions in rats.
Magnitude of risk	The predicted annual concentrations of acrolein exceed the RfC at the Employee Camp (HQ=1.6) and Project Boundary (HQ=4.9). Due to the relatively minor magnitude of exceedances (<2x the chronic threshold) at the Employee Camp, and the low likelihood of a receptor being present at the Project Boundary, the magnitude of risk for acrolein is considered to be low.

5.6.1 Further Analysis of Parameters Exceeding Target Risk Levels

5.6.1.1 Acrolein

The HQ values for acrolein are greater than 1.0 at the Employee Camp and the Project Boundary for the Application Case. Further analysis of the potential risks posed by acrolein is provided in Table 5.6-2.

5.6.2 Residual Effect Classification

Residual effects to human health for long-term (chronic) exposure to emissions from the Project are classified in Table 5-6-3. The effect classification criteria are already incorporated into the risk estimates as described in Section 2.4.1; therefore, residual effects are defined by the magnitude of risk as determined from risk estimates.

Table 5.6-3 Residual Effect Classification for Chronic Inhalation Exposure for Application Case

Parameter	Location	Magnitude of Chronic Risks as a Result of the Project
Chronic Inhalation Risk Assessment		
Acrolein	Project Boundary and Employee Camp	low
Remainder of COCs	all locations	negligible

5.6.2.1 Prediction Confidence

This chronic health risk assessment was based on many layers of safety, including the following:

- maximum predicted ambient air concentrations based on conservative modelling methods; and
- maximum exposure durations such that people are assumed to be exposed to maximum predicted air concentrations 24-hours/day for 365 days per year for a lifetime.

Collectively, these assumptions contribute to risk estimates that overestimate the true risk that is likely to be caused by the Application Case.

6 PARTICULATE MATTER ASSESSMENT RESULTS

The PM assessment evaluated the potential health effects resulting from inhalation exposure to $PM_{2.5}$ and PM_{10} in air emissions from the Project. Many epidemiological studies to identify the relationship between health effects and particulate matter have been conducted over the past 20 years. Many of these studies have shown that there is a relationship between increases in ambient particulate matter concentrations with mortality and hospitalizations for respiratory and cardiac health effects (Health Canada and Environment Canada 1999). This relationship has been stronger for $PM_{2.5}$ than PM_{10} (Health Canada and Environment Canada 1999). However, there has also been some uncertainty regarding what specifically, the relationship is between particulate matter and health effects. Many epidemiological studies have been confounded by the presence of other air pollutants (e.g., sulphur dioxide), temperature and smoking habits. In addition, there is uncertainty regarding whether epidemiological studies have properly accounted for exposure by individuals if ambient concentrations are based on a fixed monitoring station and whether the particulate matter only advances health effects of people who already have advanced and serious illnesses (Health Canada and Environment Canada 1999). Therefore, there is no prescribed method for assessing health risks of particulate matter, nor does the assessment of particulate matter lend itself to risk assessment methods in the same manner as other parameters. For this assessment, a qualitative approach was taken to assess health effects from exposure to $PM_{2.5}$ and PM_{10}.

6.1 PARTICULATE MATTER RESULTS

Particulate matter results for the predicted Baseline and Application Cases are presented below in Table 6.1-1 (for $PM_{2.5}$) and Table 6.1-2 (for PM_{10}). A literature review on the health effects associated with exposure to particulate matter is provided in Appendix VI.

Table 6.1-1 Comparison of Predicted 75th and 95th Percentiles, and Maximum $PM_{2.5}$ Concentrations with the Canada-Wide Standard

Location	24-hour Peak $PM_{2.5}$ Concentration[µg/m^3]					
	Baseline Case		Application Case			
	Peak [µg/m^3]	Frequency of Exceedances[(b)] (Number of 24-hour Exceedances in a Year)	Peak [µg/m^3]	95th Percentile [µg/m^3]	75th Percentile [µg/m^3]	Frequency of Exceedances[(b)] (Number of 24-hour Exceedances in a Year)
Warburton Bay Lodge	2.1	0	2.1	2.0	1.9	0
Warburton Bay Fishing Lodge	2.1	0	2.1	2.0	1.9	0
MacKay Lake Lodge	2.1	0	2.1	2.0	1.9	0
Employee Camp	2.1	0	**89.2**	20.5	9.7	7
Proposed National Park Boundary	2.1	0	6.4	3.7	2.3	0
Project Boundary	2.2	0	**137.7**	**51.1**	23.6	62
Canada-Wide Standard [(a)]	**30**					

(a) (CCME 2007).

(b) Frequency of exceedances shown are for Year 8 for the Employee Camp and for Year 1 for the Project Boundary.

Note: Bold values indicate an exceedance of the Canada Wide Standard.

Table 6.1-2 Comparison of Predicted 75th and 95th percentiles, and maximum PM_{10} Concentrations with the Canada-Wide Standard

Location	24-hour Peak PM_{10} Concentration [µg/m^3]					
	Baseline Case		Application Case			
	Peak [µg/m^3]	Frequency of Exceedances[(b)] (Number of 24-hour Exceedances in a Year)	Peak [µg/m^3]	95th Percentile [µg/m^3]	75th Percentile [µg/m^3]	Frequency of Exceedances [(b)] (Number of 24-hour Exceedances in a Year)
Warburton Bay Lodge	3.1	0	3.3	3.1	3.0	0
Warburton Bay Fishing Lodge	3.1	0	3.3	3.1	3.0	0
MacKay Lake Lodge	3.1	0	3.2	3.0	3.0	0
Employee Camp	3.0	0	**171.7**	**62.6**	22.7	23
Proposed National Park Boundary	3.0	0	22.6	8.7	4.1	0
Project Boundary	3.0	0	**515.1**	**271.5**	**134.7**	81
Canada-Wide Standard[(a)]	**25**					

(a) CCME 2007.

(b) Frequency of exceedances shown are for Year 8 for the Employee Camp and for Year 1 for the Project Boundary.

Note: Bold values indicate an exceedance of the Canada Wide Standard.

For $PM_{2.5}$ (Table 6.1-1), predicted maximum concentrations for the Baseline Case (ranging from 2.1 to 2.2 µg/m^3) did not exceed the Canada-Wide Standard (30 µg/m^3) at any of the receptor locations. The predicted 95th percentile and

maximum concentration for the Application Case at the Project Boundary (51.1 µg/m^3 and 137.7 µg/m^3, respectively) and the maximum concentration at the Project Boundary (89.2 µg/m^3) exceeded the Canada-Wide Standard. The maximum $PM_{2.5}$ concentration at the Employee Camp is predicted to exceed the Canada-Wide Standard seven days of the year and 62 days of the year at the Project Boundary. Note that no concentration above the air quality standard of 30 µg/m^3 is predicted beyond approximately 1.6 km from the development area boundary (Section 3.5 of the 2012 Updated Air Quality Assessment [De Beers 2012b]).

For PM_{10} (Table 6.1-2), predicted maximum baseline concentrations (ranging from 3.0 to 3.1 µg/m^3) did not exceed the Canada-Wide Standard (25 µg/m^3) at any of the receptor locations. The predicted 95th percentile and maximum concentrations at the Employee Camp (62.6 and 171.7 µg/m^3, respectively) and at the Project Boundary (271.5 and 515.1 µg/m^3, respectively), and the 75th percentile concentration at the Project Boundary (134.7 µg/m^3) exceeded the Canada-Wide Standard. The maximum PM_{10} concentration at the Employee Camp is predicted to exceed the Canada-Wide Standard 84 days of the year and 294 days of the year at the Project Boundary.

Appendix I summarizes the potential sources of particulate matter from the Project operations. One of the sources of particulate matter is the haul road. During the summer months of May to September, road dust emissions can be mitigated by frequent watering of the haul roads. Watering of the haul roads will not be possible in the winter due to freezing conditions. However, a certain level of natural mitigation of the road dust emissions can be expected from precipitation and snow accumulation on the road surface (Golder 2012b).

6.1.1 Seasonal Variations

Table 6.1-3 shows the number of $PM_{2.5}$ exceedances in a 24-hour period per month at the Employee Camp (at Year 8) and at the Project Boundary (at Year 1). The highest number of exceedances occurs in January (3 days/month) at the Employee Camp and there were no expected exceedances from May to October. The predicted total number of exceedances is 7 days per year (1.9% of the year) at the Employee Camp. The highest number of exceedances at the Project Boundary occurs in the winter months (7 days in January and 4 days in December) and no exceedances are expected in late summer (August and September). The predicted total number of exceedances is 22 days per year (6.0% of the year) at the Project Boundary.

Table 6.1-3 Seasonality Breakdown by the Number of $PM_{2.5}$ Exceedances in a 24-hour period

Month	Number of Days per Year Exceeding the Canada-Wide Standard	
	Employee Camp (at Year 8) [(a)]	Project Boundary (at Year 1) [(a)]
January	3	7
February	1	1
March	0	1
April	0	0
May	0	1
June	0	1
July	0	2
August	0	0
September	0	0
October	0	3
November	2	2
December	1	4
Total	**7 (1.9%)**	**22 (6.0%)**

(a) Note that the time series was only modelled for specific years at certain receptors, and not all three years, so the sum total exceedances may not match Table 6.1-1, and will be equal to or less than Table 6.1-1.

Particle size in an important determinant in site and efficiency of pulmonary deposition, but particle size is also a surrogate for particle source and composition. Fine particulate $PM_{2.5}$ is generally associated with combustion particles from combustion sources such as motor vehicles and burning of coal, wood sources and fuel. Coarse particulate matter $PM_{2.5-10}$ is generally associated with crustal particles that are generated mechanically (i.e., not combustion related) from agriculture, mining, construction and road traffic. $PM_{2.5-10}$ may also contain particles of biological origin such as endotoxins. Particles between 2.5 and 10 µm in size are readily inhaled but are also deposited in the upper airways (Slaughter et al. 2005). The conditions at the proposed Mine Site include a haul road along the project boundary which is suspected to generate coarse particulate matter. For the Application Case, emissions from road dust appear to be the main contributor to particulate matter, making up approximately 77% of the predicted maximum total suspended particulate (TSP) emission rate, and $PM_{2.5}$ making up 35% of the predicted maximum $PM_{2.5}$ emission rate (Section 3.1, Table 3-1 of the 2012 Updated Air Quality Assessment [De Beers 2012b]).

6.2 CONCLUSIONS OF THE PARTICULATE MATTER LITERATURE REVIEW

Potential health effects of particulate matter increases as a result of the Project were assessed qualitatively by a review of key epidemiological studies focussed on health effects associated with particulate matter from crustal sources.

Overall, a great deal of uncertainty remains in evaluating the predicted particulate matter concentrations. $PM_{2.5}$ and PM_{10} concentrations were above Canada Wide Standards at the Employee Camp and Project Boundary. Road dust (i.e., crustal sources) appears to be the main contributor to PM concentrations predicted for the Project (Section 3.1 of the 2012 Updated Air Quality Assessment [De Beers 2012b]). Most epidemiology studies suggest that dust derived from crustal sources is less hazardous than dust derived from combustion sources. This may be attributable to contaminants adsorbed onto dust derived from combustion sources, but may also be attributable to the generally smaller size of dust from combustion sources.

One study (Laden 2000) suggested no increased daily mortality as a result of exposure to fine crustal particulate matter; which may indicate no effects associated with increased $PM_{2.5}$ concentrations at the Employee Camp and Project Boundary; however, epidemiological data on fine crustal particulate matter are conflicting. For PM_{10} derived from crustal sources, one study (Schwartz et al. 1999) indicates no increase in mortality as the result of exposure to high concentrations of PM in dust storms in Spokane, Washington. Several other studies indicate possible increases in daily mortality (Ostro 1999; Staniswalis et al. 2005) or other morbidity effects (Gordian et al. 1996) associated with increased PM_{10} concentrations from crustal sources. This indicates the potential for increased mortality or health effects at the Employee Camp and Project Boundary are uncertain as some studies would indicate the potential for adverse health effects and others would not.

The methods of assessment of health effects from exposure to respirable particulate matter are derived from epidemiology studies based on large urban centres making comparisons to small rural communities challenging. In addition, the database related to health effects from particulate matter relies heavily on studies where the particulates are derived from combustion sources. Few studies were available concerning possible health outcomes from wind-blown dust (i.e., road dust in this case) – particularly for fine particulates (i.e., $PM_{2.5}$); however, some studies have found adverse health effects (Fuentes et al. 2006). These studies would suggest that health effects at the Employee Camp and Project Boundary are possible. In light of these uncertainties, no firm conclusions regarding potential health effects from respirable particulate matter exposure at the Employee Camp and Project Boundary could be drawn. It should be noted that it is unlikely for receptors to spend time at the Project Boundary.

Discussions of the conservatism and mitigation associated with managing risk from particulate matter are provided in Sections 8.2.

7 CHRONIC MULTI-MEDIA RISK ASSESSMENT RESULTS

The risk estimates for the COCs evaluated in the multi-media risk assessment are presented in Tables 7-1 (Seasonal Users [high fish consumers] and Gahcho Kué workers) and 7-2 (Season Users [low fish consumers]). Results are presented for the Baseline Case and Application Case for comparison purposes. For comparison purposes, risk estimates were also calculated without the addition of background dietary intake, and these results are available in Appendix V.

In the Application Case, arsenic, cobalt, manganese, nickel and thallium have HI values greater than 1.0 for Seasonal Users, and arsenic, cobalt and thallium have HI values greater than 1.0 for Gahcho Kué workers. For carcinogens, ILCR values for arsenic were greater than 1.0×10^{-5} for Seasonal Users and Gahcho Kué workers.

The resulting HIs and ILCRs were summed for COCs that had toxicity reference values based on similar target organs and modes of action (Tables 7-1 and 7-2). For non-carcinogens in the Application Case, the sum HI values for neurotoxic effects (aluminum and manganese) and respiratory effects (antimony, arsenic, cobalt, nickel, titanium and vanadium) were greater than 1.0 for Seasonal Users, and were greater than 1.0 for Gahcho Kué workers for respiratory effects.

Tables 7-3 through 7-9 provide further analysis and a determination of the magnitude of risks for each COC or group of COCs that exceeded target risk levels. While results are shown for all age categories for the Seasonal User in Tables 7-1 and 7-2, the magnitude of risk tables (Tables 7-3 to 7-9) focus on the toddler, which was the most sensitive receptor for non-carcinogenic effects in most cases.

Figure 7-1 shows the HQ contribution from all the exposure pathways for the toddler Seasonal User (high fish consumer scenario) for the Application Case, and Figure 7-2 shows the same results with the absence of background dietary intake for discussion purposes. Figures 7-3 and 7-4 show the relative contribution of exposure pathways to the ILCR for the adult Seasonal Users in the Application Case, including all exposure pathways (Figure 7-3) and with the exclusion of the background dietary intake (Figure 7-4).

Table 7-1 Risk Estimates for Aboriginal Seasonal Users (High Fish Consumers) and Gahcho Kué Workers

Parameter	Baseline Case						Application Case						Baseline Case	Application Case	Baseline Case	Application Case
	Hazard Index					Total ILCR	Hazard Index					Total ILCR	Hazard Index		ILCR	
	Infant	Toddler	Child	Adolescent	Adult		Infant	Toddler	Child	Adolescent	Adult		GK Worker			
Metals																
Aluminum (NC)	1.4E-01	3.1E-01	2.4E-01	2.0E-01	1.5E-01	N/A	2.5E-01	4.2E-01	3.6E-01	3.1E-01	2.6E-01	N/A	1.5E-01	2.7E-01	N/A	N/A
Antimony (NC)	9.5E-02	1.3E-01	9.6E-02	6.0E-02	5.5E-02	N/A	9.5E-02	5.0E-01	4.3E-01	2.8E-01	2.6E-01	N/A	1.1E-02	1.2E-02	N/A	N/A
Arsenic (C)	***2.4E+00***	***4.9E+00***	***6.0E+00***	***4.1E+00***	***4.9E+00***	**2.6E-03**	***2.4E+00***	***5.1E+00***	***6.2E+00***	***4.3E+00***	***5.0E+00***	**2.7E-03**	***4.8E+00***	***4.8E+00***	**2.0E-03**	**2.0E-03**
Cadmium (C)	2.3E-01	6.0E-01	5.0E-01	3.4E-01	2.7E-01	2.2E-10	4.0E-01	8.0E-01	6.8E-01	5.1E-01	4.5E-01	2.6E-06	2.5E-01	4.2E-01	2.0E-10	2.3E-06
Cobalt (C)	***2.3E+00***	***2.6E+00***	***1.8E+00***	***1.3E+00***	***1.1E+00***	6.5E-10	***2.5E+00***	***3.2E+00***	***2.3E+00***	***1.7E+00***	***1.5E+00***	1.8E-06	8.4E-01	***1.0E+00***	5.9E-10	1.6E-06
Iron (NC)	1.4E-01	3.0E-01	1.1E-01	7.2E-02	8.2E-02	N/A	1.6E-01	3.5E-01	1.4E-01	9.8E-02	1.1E-01	N/A	6.4E-02	6.5E-02	N/A	N/A
Manganese (NC)	8.9E-01	***1.3E+00***	9.4E-01	6.7E-01	4.0E-01	N/A	***1.2E+00***	***1.6E+00***	***1.2E+00***	9.6E-01	6.8E-01	N/A	3.3E-01	6.6E-01	N/A	N/A
Nickel (C)	***1.3E+00***	1.0E+00	7.5E-01	6.1E-01	5.1E-01	3.9E-09	***1.7E+00***	***1.4E+00***	***1.2E+00***	***1.0E+00***	9.2E-01	4.6E-06	4.6E-01	8.9E-01	3.6E-09	4.1E-06
Thallium (NC)	***4.2E+00***	***7.9E+00***	***5.9E+00***	***4.2E+00***	***4.2E+00***	N/A	***4.3E+00***	***1.6E+01***	***1.3E+01***	***9.0E+00***	***8.7E+00***	N/A	***1.5E+00***	***1.6E+00***	N/A	N/A
Titanium (NC)	2.5E-04	2.5E-04	2.5E-04	2.5E-04	2.5E-04	N/A	4.9E-01	4.9E-01	4.9E-01	4.9E-01	4.9E-01	N/A	3.0E-04	5.8E-01	N/A	N/A
Vanadium (NC)	2.2E-01	1.4E-01	8.4E-02	5.8E-02	7.6E-02	N/A	2.4E-01	1.9E-01	1.2E-01	8.8E-02	1.1E-01	N/A	7.0E-02	9.0E-02	N/A	N/A
SUM HQ - chemicals with similar target organs/effects																
Neurotoxicity (Al, Mn)	***1.0E+00***	***1.6E+00***	***1.2E+00***	8.8E-01	5.5E-01	N/A	***1.5E+00***	***2.0E+00***	***1.6E+00***	***1.3E+00***	9.5E-01	N/A	4.8E-01	9.3E-01	N/A	N/A
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)	6.3E-04	6.3E-04	6.3E-04	6.3E-04	6.3E-04	N/A	***1.0E+00***	***1.0E+00***	***1.0E+00***	***1.0E+00***	***1.0E+00***	N/A	7.6E-04	***1.2E+00***	N/A	N/A
SUM ILCR - chemicals with similar target organs/effects																
Lung/respiratory tract tumours (As, Cd, Co, Ni)	N/A	N/A	N/A	N/A	N/A	5.6E-09	N/A	N/A	N/A	N/A	N/A	9.3E-06	***N/A***	***N/A***	5.1E-09	8.3E-06

Notes: Bold text shaded yellow indicates ILCR values greater than 1.0×10^{-5} (1.0E-05).
Bold italic text shaded orange indicates an HI greater than 1.0.
NC is a non-carcinogen; C is a carcinogen; N/A is not assessed because the substance was not a carcinogen or not measured in a particular media.

Table 7-2 Risk Estimates for Aboriginal Seasonal Users (Low Fish Consumers)

Parameter	Baseline Case						Application Case					
	Hazard Index					Total ILCR	Hazard Index					Total ILCR
	Infant	Toddler	Child	Adolescent	Adult		Infant	Toddler	Child	Adolescent	Adult	
Metals												
Aluminum (NC)	1.4E-01	2.9E-01	2.3E-01	2.0E-01	1.4E-01	N/A	2.5E-01	4.0E-01	3.4E-01	3.0E-01	2.5E-01	N/A
Antimony (NC)	9.5E-02	5.6E-02	3.0E-02	1.8E-02	1.6E-02	N/A	9.5E-02	9.5E-02	5.7E-02	4.2E-02	3.6E-02	N/A
Arsenic (C)	***2.4E+00***	***4.8E+00***	***5.9E+00***	***4.1E+00***	***4.8E+00***	**2.6E-03**	***2.4E+00***	***4.9E+00***	***6.0E+00***	***4.1E+00***	***4.9E+00***	**2.6E-03**
Cadmium (C)	2.3E-01	5.9E-01	4.9E-01	3.3E-01	2.6E-01	2.2E-10	4.0E-01	7.8E-01	6.7E-01	5.0E-01	4.5E-01	2.6E-06
Cobalt (NC)	***2.3E+00***	***2.4E+00***	***1.6E+00***	***1.1E+00***	9.7E-01	6.5E-10	***2.5E+00***	***2.7E+00***	***1.9E+00***	***1.4E+00***	***1.3E+00***	1.8E-06
Iron (NC)	1.4E-01	2.7E-01	7.6E-02	5.4E-02	6.4E-02	N/A	1.6E-01	3.0E-01	9.7E-02	7.0E-02	8.8E-02	N/A
Manganese (NC)	8.9E-01	***1.2E+00***	9.4E-01	6.7E-01	4.0E-01	N/A	***1.2E+00***	***1.5E+00***	***1.2E+00***	9.6E-01	6.8E-01	N/A
Nickel (C)	***1.3E+00***	9.7E-01	7.3E-01	6.0E-01	5.0E-01	3.9E-09	***1.7E+00***	***1.4E+00***	***1.1E+00***	9.7E-01	8.8E-01	4.6E-06
Thallium (NC)	***4.2E+00***	***5.0E+00***	***3.2E+00***	***2.5E+00***	***2.6E+00***	N/A	***4.3E+00***	***6.0E+00***	***3.9E+00***	***3.1E+00***	***3.1E+00***	N/A
Titanium (NC)	2.5E-04	2.5E-04	2.5E-04	2.5E-04	2.5E-04	N/A	4.9E-01	4.9E-01	4.9E-01	4.9E-01	4.9E-01	N/A
Vanadium (NC)	2.2E-01	1.4E-01	8.0E-02	5.6E-02	7.3E-02	N/A	2.4E-01	1.6E-01	9.9E-02	7.4E-02	9.3E-02	N/A
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)	***1.0E+00***	***1.5E+00***	***1.2E+00***	8.7E-01	5.4E-01	N/A	***1.5E+00***	***2.0E+00***	***1.6E+00***	***1.3E+00***	9.3E-01	N/A
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)	6.3E-04	6.3E-04	6.3E-04	6.3E-04	6.3E-04	N/A	***1.0E+00***	***1.0E+00***	***1.0E+00***	***1.0E+00***	***1.0E+00***	N/A
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)	NA	NA	NA	NA	NA	5.6E-09	NA	NA	NA	NA	NA	9.3E-06

Notes: Bold text shaded yellow indicates ILCR values greater than 1.0×10^{-5} (1.0E-05).
Bold italic text shaded orange indicates an HI greater than 1.0.
NC is a non-carcinogen; C is a carcinogen; N/A is not assessed because the substance was not a carcinogen or not measured in a particular media.

Table 7-3 Further Analysis of Arsenic and Determination of Magnitude of Risk

Analysis Criteria	Discussion
Magnitude of Risk Estimates in the Application Case	**Arsenic as a Non-Carcinogen** For the toddler Seasonal User, the HI for arsenic in the Application Case exceeded the target threshold of 1.0 for the high fish consumer (HI = 5.1), and the low fish consumer (HI=4.9). The HI was higher for the child receptor in both the low and high fish consuming scenarios (HI=6.0 and 6.2, respectively), primarily based on the higher background dietary intakes for the child compared to the toddler. For the Gahcho Kué Worker, the HI for arsenic in the Application Case was greater than 1.0 (HI = 4.8). **Arsenic as a Carcinogen** ILCRs for all receptors exceeded the target ILCR of 1.0×10^{-5}. For the Seasonal User (composite receptor), the ILCRs for arsenic in the Application Case were 2.7 E-03 (high fish consumer) and 2.6E-03 (low fish consumer). For the Worker, the ILCR for arsenic in the Application Case was 2.0E-03.
Comparison of Baseline Case to Application Case	**Arsenic as a Non-Carcinogen** The HIs for the toddler Seasonal User were only slightly higher in the Application Case compared to the Baseline Case (e.g., HI of 4.9 in baseline versus 5.1 in Application case for the high fish consumer). For the worker, the HI in the Baseline and Application Case were nearly identical (HI of 4.8). **Arsenic as Carcinogen** The ILCRs for the Seasonal User were higher in the Application Case compared to the Baseline Case, and differences in ILCRs were greater for the high fish consumer versus the low fish consumer (increase of 8 in 100,000 for the high fish consumer and increase of 1 in 100,000 for the low fish consumer, from the Baseline Case to the Application Case). For the Worker, the ILCR in the Application Case was similar but was slightly higher in the Application Case compared to the Baseline Case (increase of 0.6 in 100,000 from the Baseline Case to the Application Case).
Conservatism and uncertainty in predictions	Several sources of uncertainty in the HHRA relate to the air quality predictions and deposition rates used to predict COC concentrations in country food items. The air quality and deposition rate predictions use the maximum emission rates from the Project; however, this is a conservative assumption due to the fact that most equipment does not operate at its maximum capacity on a continuous basis. This assumption can lead to overestimation of the potential Project impacts for the longer averaging periods (24-hour and annual). Country food predictions are based on soil quality and deposition rates for the Project Boundary which is where the highest deposition rates occur.
Conservatism in the exposure assumptions	The exposure assessment used site-specific chemistry data and exposure intake values for the Northern Aboriginal populations as available. The exposure assessment assumes the consumption of country foods for six months of the year which is a conservative assumption as many country foods are only available seasonally; however, this assumption is protective of receptors that may treat and store country food items for consumption throughout the year. It is also possible that there will not be sufficient quantity of country food items present in the immediate vicinity of the Site to support this assumption and as a result, fifty percent (50%) of the daily country food consumption was assumed to be from the Site. Concentrations of substances in fish are predicted using changes in water quality in Kennady Lake and site-specific bioconcentration factors, where available. Drinking water quality for Seasonal Users and for Gahcho Kué Workers is assumed to be similar to that found in Area 8 (which will be used for the mine water supply). Arsenic concentrations in drinking water result in ILCR values for the Baseline Case which exceed target risk levels. Arsenic is naturally elevated in many areas in the Northwest Territories (Government of the Northwest Territories [GNWT] 2003). In all exposure scenarios, the ingestion of background dietary items was the primary contributor to the risk estimate (e.g., contributing 92% of the HI in the high fish consumer Application Case, followed by the 6% for the fish ingestion pathway; Figure 7-1). Excluding the dietary intake would results in an HI of 0.4 for the high fish consumer Toddler in the Application Case (Figure 7-2). The ingestion of background dietary items was also the primary pathway that contributed to the ILCR for both the Seasonal User and Gahcho Kué worker. Removal of background intake results in an ILCR of 1.2E-04 for the high fish consumer and 1.9E-05 for the worker in the Application Case. The consumption of fish was the second greatest contributor to the ILCR for the Seasonal User.

Table 7-3 Further Analysis of Arsenic and Determination of Magnitude of Effect (continued)

Analysis Criteria	Discussion
Conservatism in the toxicity reference values for arsenic	**Arsenic as a Non-Carcinogen** The most conservative value of 0.0003 mg/kg day was chosen from U.S. EPA IRIS as the RfD. The non-carcinogenic RfD for arsenic is based on the NOAEL for hyperpigmentation and keratosis in an epidemiological study for a population in Taiwan. An uncertainty factor of 3 was applied to the NOAEL to account for the lack of reproductive data and uncertainty in whether the NOAEL accounts for all sensitive individuals. An RfC was obtained from RIVM, which derived a tolerable concentration in air (TCA) of 0.001 mg/m^3. RIVM indicated that lung cancer occurs in humans at concentrations greater than 0.01 mg/m^3, but that the mechanism for tumours is not directly genotoxic, and therefore a threshold exists for this effect. RIVM therefore decided that this value was a TCA, not a cancer risk value, and applied an uncertainty factor of 10 to account for intra-human variability. **Arsenic as a Carcinogen** The most conservative arsenic slope factor and unit risk were used from Health Canada. Health Canada has derived an oral slope factor of 1.8 (mg/kg/day)-1 (Health Canada 2009b). The slope factor was derived based on an epidemiological study where humans were naturally exposed to arsenic in drinking water for up to 60 years. Overall, using a 1% increase in risk, the unit risks associated with ingestion of 1 µg/L of arsenic in drinking water were estimated to range from 3.06E-06 to 3.85E-05, with 95% upper bounds ranging from 6.49E-06 to 4.64E-05 (Health Canada 2009b). The most sensitive endpoint for both males and females was lung cancer. The overall unit risk associated with the ingestion of arsenic in drinking water was reported as a range, given that lifetime exposure to arsenic results in more than one cancer endpoint in different individuals. The above unit risk range has the liver cancer unit risk (3.06E-06) as its lower bound and the lung cancer unit risk (3.85E-05) as its upper bound (Health Canada 2009b). Health Canada has derived an inhalation unit risk of 6.4 per mg/m^3 (Health Canada 2009b) based on an epidemiological study where mortality as of 1976 was documented in a cohort of 2802 smelter workers employed for at least one year between 1940 and 1964 was followed. Based on monitoring data and conversion levels of arsenic in urine to airborne concentrations, it was found that the standardized mortality ratios for respiratory cancer increased with increased cumulative exposure to arsenic. A variety of toxicity reference values are available for arsenic ranging from 1.5 to 1.8 $(mg/kg\ BW/day)^{-1}$ for oral slope factor and 0.0043 to 0.0064 $(\mu g/m^3)^{-1}$ for air unit risk. However, the risk assessment for the Project used the more conservative Health Canada (Health Canada 2009b) oral slope factor and unit risk.
Potential health effects	**Arsenic as a Non-Carcinogen** Epidemiological studies used to derive RfDs for arsenic are based on observations of dermal effects in humans exposed to arsenic. **Arsenic as a Carcinogen** Epidemiological studies used to derive slope factors for arsenic are based on observations of cancer of the urinary bladder, lung and skin in communities exposed to high concentrations of arsenic. The Government of Canada's Priority Substances List Assessment Report on Arsenic (1993) provides estimates of total daily exposure to inorganic arsenic from environmental sources ranging from 0.1 to 2.6 µg/kg-body weight/day, which includes exposure via drinking water. In areas near point sources, exposure may be up to 35 µg/kg-bw/day. These exposures would correspond to cancer risks of 1.5 $x10^{-4}$ to 3.9 $x10^{-3}$ for the general population, and up to 5.9 $x10^{-2}$ in populations near point sources. The estimated ILCRs for the Seasonal Users and Gahcho Kué workers fall within the range of background exposure for the general Canadian population.

Table 7-3 Further Analysis of Arsenic and Determination of Magnitude of Effect (continued)

Analysis Criteria	Discussion
Magnitude of risk	**Arsenic as a Non-Carcinogen** For the toddler Seasonal User, the HIs increased only slightly between the Baseline Case and Application Case, and exceed the target HI for the high and low fish consumer, indicating that incremental COC air concentrations from the Project may influence human health. The majority of the risk associated with arsenic was attributable to background dietary intake rather than exposure as a result of the Project. Therefore, the magnitude of risk from the Project was considered to be low for Seasonal Users. For the Gahcho Kué Worker, the HI did not increase from the Baseline Case and Application Case, indicating that incremental COC air concentrations from the Project is unlikely to influence worker health. Therefore, for workers, the magnitude of risk from the Project was considered to be negligible. **Arsenic as a Carcinogen** For the Seasonal User, ILCRs exceeded the target threshold, and the ILCR increased between the Baseline Case and Application Case. However, the majority of the ILCR is attributable to background dietary intake and not site exposure. Therefore, the magnitude of risk from the Project was considered to be low for the Seasonal User. For the Gahcho Kué worker, the ILCR increase from the Baseline Case and Application Case was small (0.7 in 100,000), indicating that incremental COC air concentrations from the Project are unlikely to influence worker health. Therefore, for workers, the magnitude of risk from the Project was considered to be negligible.

Table 7-4 Further Analysis of Cobalt and Determination of Magnitude of Risk (Non-carcinogenic Effects)

Analysis Criteria	Discussion
Magnitude of risk estimate in the Application Case	For the toddler Seasonal User the HI for cobalt in the Application Case exceeded the target threshold of 1.0 for the high fish consumer (HI = 3.2) and the low fish consumer (HI = 2.7). The HI for the Gahcho Kué Worker also marginally exceeded 1.0 for cobalt (HI=1.04).
Comparison of Baseline Case to Application Case	The HIs for the toddler Seasonal User and the Worker were higher in the Application Case compared to the Baseline Case. For the toddler, differences in HIs were greater for the high fish consumer versus the low fish consumer (difference in HI of 0.6 for the high fish consumer and difference of 0.3 for the low fish consumer).
Conservatism and uncertainty in predictions	Several sources of uncertainty in the HHRA relate to the air quality predictions and deposition rates used to predict COC concentrations in country food items. The air quality and deposition rate predictions use the maximum emission rates from the Project; however, this is a conservative assumption because most equipment does not operate at its maximum capacity on a continuous basis. This assumption can lead to overestimation of the potential Project impacts for the longer averaging periods (24-hour and annual). Country food predictions are based on soil quality and deposition rates for the Project Boundary which is where the highest deposition rates occur.

Table 7-4 Further Analysis of Cobalt and Determination of Magnitude of Effect (Non-carcinogenic Effects) (continued)

Analysis Criteria	Discussion
Conservatism in the exposure assumptions	The exposure assessment used site-specific chemistry data and exposure intake values for the Northern Aboriginal populations as available. The exposure assessment assumes the consumption of country foods for six months of the year which is a conservative assumption as many country foods are only available seasonally; however, this assumption is protective of receptors that may treat and store country food items for consumption throughout the year. It is also possible that there will not be sufficient quantity of country food items present in the immediate vicinity of the Site to support this assumption and as a result, fifty percent (50%) of the daily country food consumption was assumed to be from the Site. In the Application Case for the high fish consuming toddler Seasonal User, the ingestion of background dietary items was the primary exposure pathway contributing 59% of the risk HI, followed by the ingestion of fish (17% of the HI), the ingestion of caribou (7% of HI), the incidental sediment ingestion pathway (5% of the HI) and the inhalation of air pathway (5% of the HI). For the low fish consuming toddler Seasonal User, the ingestion of background dietary items was also the driver of risk (contributing 70% of the HI), followed by ingestion of caribou (9% of HI), inhalation of air (6%) and incidental sediment ingestion (6%). Ingestion of fish contributed 2% to the HQ for the low fish consumer. Excluding the background dietary intake results in a HI of 1.3 (high fish consumer, Figure 7-2) and 0.48 (low fish consumer) for the Toddler Seasonal User. Fish consumption is likely to be lower than the 'high fish' scenario, given that Seasonal User are not only relying on fish as a protein source (e.g., caribou and hare are also being consumed). In the Application Case for the Worker, the ingestion of background dietary items was the primary exposure pathway contributing to the risk estimate (contributing 74% of the HI), followed by the inhalation of air (19% of HI), incidental ingestion of sediment (5% of HI) and incidental ingestion of soil (1% of HI). Exclusion of the background dietary intakes results in a HI of 0.27 for the Worker.
Conservatism in the toxicity reference values for cobalt	The provisional value of 0.0003 mg/kg day was chosen from U.S. EPA NCEA as the RfD. The non-carcinogenic RfD for cobalt is based on the LOAEL of 1 mg/kg-day for decreased iodine uptake by the thyroid in humans. An uncertainty factor of 3,000 was applied to the LOAEL to derive the RfD; the uncertainty factor considered four separate factors (a factor of 10 for extrapolation from a subchronic to chronic study, a factor of 10 for extrapolation from a LOAEL to a NOAEL, a factor of 10 to account for lack of data on human variability and sensitive populations; and a factor of 3 to account for the lack of multigenerational studies). The provisional value of 0.006 $\mu g/m^3$ was chosen from U.S. EPA NCEA as the RfC. The non-carcinogenic RfC for cobalt is based on decreased pulmonary function in workers (Nemery et al. 1992). The NOAEL of 5.3 $\mu g/m^3$ was adjusted for continuous exposure (to 1.9 $\mu g/m^3$) and an uncertainty factor of 300 was applied to account for three separate factors (a factor of 3 for extrapolation from an assumed subchronic to chronic study, a factor of 10 for database insufficiencies including lack of developmental inhalation studies and multigenerational studies, and a factor of 10 to account for lack of data on human variability and sensitive populations).
Potential health effects	Cobalt is an essential element for humans and is required for the production of vitamin B12. Vitamin B12 is a coenzyme in many biological reactions including the production of red blood cells, and cobalt has been used to treat anemia. Oral exposure to high levels of cobalt has occurred in humans who consumed beer containing cobalt salts, resulting in death in extreme circumstances, and more commonly in nausea, vomiting and diarrhea.
Magnitude of risk	For the toddler Seasonal User, the HIs increased between the Baseline Case and Application Case (difference in HI of 0.6 for the high fish consumer and difference of 0.3 for the low fish consumer), and exceed the target HI for the high and low fish consumers (HI = 3.2 and 2.7, respectively). The majority of the risk associated with cobalt was attributable to background dietary intake and fish ingestion, which are based on conservative assumptions. Therefore, the magnitude of risk from the Project was considered to be low for Seasonal Users. For the Gahcho Kué Worker, the HI marginally exceeded 1.0 in the Application Case (HI=1.04). Based on the marginal exceedance, the magnitude of risk from the Project was considered to be negligible for the Gahcho Kué Worker.

Table 7-5 Further Analysis of Manganese and Determination of Magnitude of Risk

Analysis Criteria	Discussion
Magnitude of risk estimate in the Application Case	For the toddler Seasonal User, the HI for manganese in the Application Case exceeded the target threshold of 1.0 for both the high and low fish consumers (HI = 1.6 and 1.5, respectively). For the Worker, the HI for manganese in the Application Case did not exceed the target threshold of 1.0.
Comparison of Baseline Case to Application Case	The HIs for the toddler Seasonal User were higher in the Application Case compared to the Baseline Case, and differences in HIs between the Baseline Case and Application Case were similar for both the high fish consumer and low fish consumer (difference in HI = 0.3).
Conservatism and uncertainty in predictions	Several sources of uncertainty in the HHRA relate to the air quality predictions and deposition rates used to predict COC concentrations in country food items. The air quality and deposition rate predictions use the maximum emission rates from the Project; however, this is a conservative assumption because most equipment does not operate at its maximum capacity on a continuous basis. This assumption can lead to overestimation of the potential Project impacts for the longer averaging periods (24-hour and annual).
Conservatism in the exposure assumptions	The exposure assessment used site-specific chemistry data and exposure intake values for the Northern Aboriginal populations as available. In the Application Case for the high and low fish consuming toddler Seasonal Users, the ingestion of background dietary items was the driver of risk (contributing 63% of the HI), followed by the inhalation of air (18% of HI), ingestion of berries (7% of HI) and ingestion of Labrador tea leaves (6% of HI). In all exposure scenarios, the ingestion of background dietary items was the primary contributor to the risk estimate (Figure 7-1). Excluding the dietary intake would result in an HI of 0.6 for the high fish consumer Toddler in the Application Case (Figure 7-2).
Conservatism in the toxicity reference values for manganese	The oral RfDs for manganese (0.1 mg/kg-day for the toddler life stage and 0.2 mg/kg-day for the adult life stage) were based upon weight-of-evidence from several epidemiological and experimental studies (Health Canada 2009b). Manganese was administered via food and water and no uncertainty factors were applied. The inhalation RfC for manganese of 0.05 µg/m^3 was based upon impairment of neurological function in an occupational study using manganese dioxide (U.S. EPA 2012b). The LOAEL of 0.15 mg/m^3 was adjusted for continuous exposure to a $LOAEL_{adj}$ of 0.05 mg/m^3. An uncertainty factor of 1000 was applied (10 for the use of a LOAEL, 10 for the protection of sensitive individuals, and 10 for database limitations including the lack of developmental data, the less than chronic exposure periods, and potential differences in toxicity of different forms of manganese).
Potential health effects	Manganese is an essential element for humans and is found widely throughout the body; adverse health effects can be linked to both manganese deficiency as well as excessive manganese levels. Bone mineralization, protein and energy metabolism, metabolic regulation, cellular protection from free radicals are all functions that require manganese. Manganese is also a component of metalloenzymes and can act as an enzyme activator (ATSDR 2008).
Magnitude of risk	For the toddler Seasonal User, the HIs increase between the Baseline Case and Application Case (difference in HI=0.3), and exceed the target HI for the high and low fish consumers (HI=1.6 and 1.5, respectively). The majority of the risk associated with manganese was attributable to background dietary intake rather than exposure as a result of the Project. Therefore, the magnitude of risk from the Project was considered to be low for Seasonal Users. For the Gahcho Kué Worker, the HI increased between the Baseline Case and Application Case, but did not exceed the target HI, indicating that incremental COC air concentrations from the Project are not likely to influence human health. Therefore, for Gahcho Kué Workers, the magnitude of risk from the Project was considered to be negligible.

Table 7-6 Further Analysis of Nickel and Determination of Magnitude of Risk

Analysis Criteria	Discussion
Magnitude of risk estimate in the Application Case	For the toddler Seasonal User, the HI for nickel in the Application Case exceeded the target threshold of 1.0 for the high and low fish consumer (HI = 1.4). The infant receptor had an HI higher than the toddler for this COC (HI=1.7 for the high and low fish consumer). The ILCR for Seasonal Users did not exceed the target threshold of 1.0×10^{-5}. For the Gahcho Kué Worker, the HI for nickel did not exceed the target threshold of 1.0 and the ILCR did not exceed the target threshold of 1.0×10^{-5}.
Comparison of Baseline Case to Application Case	The HIs for the toddler Seasonal User were only slightly higher in the Application Case compared to the Baseline Case with differences in HIs of approximately 0.4 in each case.
Conservatism and uncertainty in predictions	Several sources of uncertainty in the HHRA relate to the air quality predictions and deposition rates used to predict COC concentrations in country food items. The air quality and deposition rate predictions use the maximum emission rates from the Project; however, this is a conservative assumption because most equipment does not operate at its maximum capacity on a continuous basis. This assumption can lead to overestimation of the potential Project impacts for the longer averaging periods (24-hour and annual).
Conservatism in the exposure assumptions	The exposure assessment used site-specific chemistry data and exposure intake values for the Northern Aboriginal populations as available. In the Application Case for the toddler Seasonal User, the ingestion of background dietary items was the primary exposure pathway contributing to the risk estimates (Figure 7-1). Exclusion of the background dietary intake would result in a HI of 0.54 for the high fish consumer (Figure 7-2) The secondary exposure pathway contributing to the risk estimates was the inhalation of air followed by the ingestion of fish and snowshoe hare.
Conservatism in the toxicity reference values for nickel	Health Canada (2009b) has derived an oral tolerable daily intake (TDI) of 0.011 mg/kg-day for soluble nickel. The TDI is based on a 2-generation reproductive study where rats were exposed to 0, 0.22, 0.55, 1.1 and 2.2 mg/kg-day nickel in drinking water. The F0 generation was exposed prior to and during mating and throughout gestation lactation. The F1 generation was exposed from weaning through reproduction until weaning of F2 pups. A NOAEL of 1.1 mg/kg/day was derived, the critical effect being post-implantation perinatal lethality. An uncertainty factor of 100 was applied to the NOAEL (a factor of 10 each for intra- and inter-species variation). Health Canada (2009b) has derived an inhalation tolerable concentration (TC) of 1.8E-05 mg/m^3 for sulphidic nickel (nickel subsulphide). The TC is based on a subchronic study where rats and mice were exposed to 0, 0.11, 0.2, 0.4, 0.9 and 1.8 mg/m^3 nickel subsulphide for 6 hours/day, 5 days/week, for 13 weeks. A NOAEL of 0.1 mg/m^3 was determined for mice, and a LOAEL of 0.1 mg/m^3 was determined for rats, based on respiratory track effects (alveolar macrophages, hyperplasia). The dose was adjusted for continuous exposure by applying conversion factors of 6 hours/24 hours and 5 days/7 days. An uncertainty factor of 1000 was applied to the adjusted dose (a factor of 10 each for intraspecies variation, interspecies variation, and for a less than chronic study). Health Canada (2009b) has derived an inhalation unit risk (UR) of 0.71 (mg/m^3) for soluble nickel (primarily nickel chloride and nickel sulphate). The UR is based on epidemiological studies where workers (cohort of 3250 to 54509) at two nickel refineries were occupationally exposed to nickel by inhalation for at least 12 months. The estimate of the concentration in air associated with a 5% increase in tumour incidence or mortality due to tumours (i.e., TC_{05}) for lung cancer mortality for soluble nickel was 0.07 mg/m^3. In all exposure scenarios, the ingestion of background dietary items was the primary contributor to the risk estimate (Figure 7-1). Excluding the dietary intake would results in an HI of 0.5 for the high fish consumer Toddler in the Application Case.
Potential health effects	Data available for chronic nickel inhalation exposure for humans are limited to occupational data. Respiratory effects found in nickel workers included chronic bronchitis, emphysema, and reduced vital capacity. These workers were also exposed to other metals, so it cannot be concluded that nickel is the sole causative agent of the effects observed. Asthma from primary irritation and as the result of dermal sensitization has also been documented amongst nickel workers. Nickel refinery workers with elevated urinary nickel concentrations also showed a significant increase in urinary β2-microglobulin levels, which is indicative of tubular dysfunction in the kidneys (ATSDR 2005).
Magnitude of risk	For the toddler Seasonal User, the HIs increase between the Baseline Case and Application Case (difference in HI=0.4), and exceed the target HI for the high and low fish consumers (HI=1.4). The majority of the risk associated with nickel was attributable to background dietary intake rather than exposure as a result of the Project. Therefore, the magnitude of risk from the Project was considered to be low for Seasonal Users. For the Gahcho Kué Worker, the HI increased between the Baseline Case and Application Case, but did not exceed the target HI, indicating that incremental COC air concentrations from the Project are not likely going to influence human health. Therefore, for Gahcho Kué Workers, the magnitude of risk from the Project was considered to be negligible.

Table 7-7 Further Analysis of Thallium and Determination of Magnitude of Risk

Analysis Criteria	Discussion
Magnitude of risk estimate in the Application Case	For the toddler Seasonal User, the HI for thallium in the Application Case exceeded the target threshold of 1.0 for the high and low fish consumers (HI = 16 and HI = 6, respectively). For the Gahcho Kué worker, the HI also exceeded the target threshold (HI=1.6).
Comparison of Baseline Case to Application Case	The HIs for the toddler Seasonal User were higher in the Application Case compared to the Baseline Case with a differences in HI of 8.1 and 1.0 for the high and low fish consumers, respectively. The difference in HIs for the Gahcho Kué Worker was 0.1 between the Baseline Case and Application Case.
Conservatism and uncertainty in predictions	Several sources of uncertainty in the HHRA relate to the air quality predictions and deposition rates used to predict COC concentrations in country food items. The air quality and deposition rate predictions use the maximum emission rates from the Project; however, this is a conservative assumption because most equipment does not operate at its maximum capacity on a continuous basis. This assumption can lead to overestimation of the potential Project impacts for the longer averaging periods (24-hour and annual).
Conservatism in the exposure assumptions	The exposure assessment used site-specific chemistry data and exposure intake values for the Northern Aboriginal populations as available. In the Application Case, for the high fish consuming toddler Seasonal User (Figure 7-1), the fish ingestion pathway was the primary exposure pathway that contributed to the HI value (70% of the HI), followed by background dietary intake (20%) and ingestion of caribou (6%). For the low fish consuming toddler, the fish ingestion pathway contributed 20% to the HI value, while background dietary intake was the primary exposure pathway, contributing 55%. Exclusion of the background dietary intake would result in an HI of 12.8 (high fish consumer, Figure 7-2) and 2.7 (low fish consumer) for the Toddler Seasonal User. The fish tissue concentrations were derived from predicted water concentrations and a BAF derived based on non-detect concentrations of thallium in fish tissues (<0.04 mg/kg wet weight). A BAF derived based on non-detect concentrations is likely to over-estimate fish tissue concentrations and therefore over-estimate risks to human health from fish ingestion.
Conservatism in the toxicity reference values for thallium	The provisional value of 0.00001 mg/kg day was chosen from U.S. EPA Regional Screening Levels as the RfD. The non-carcinogenic RfD for thallium is based on a study which looked at the critical effects of thallium (I) sulphate administered to male and female rats. The rats were administered 0, 0.01,0.05 or 0.25 mg/kg/-day of an aqueous solution by gavage for 90 days. The endpoint chosen for RfD development was hair follicle atrophy in female rats that also had alopecia (hair loss). The endpoint was chosen because atrophy of hair follicles is consistent with the changes observed in humans poisoned by thallium. The mid-dose in the study (0.04 mg/kg/-day was assumed to approximate the NOAEL and therefore this value was used as a point of departure. An uncertainty factor of 3000 was applied to the NOAEL (10 for interspecies extrapolation, 10 for lack of studies and 3 for extrapolating from subchronic to chronic exposure. An RfC was not available for thallium.
Potential health effects	Effects in humans following oral exposure to thallium include: death due to nerve damage; respiratory system, cardiovascular system, liver, kidney and muscle damage; and possible hair loss (ATSDR 1992). Lung and nervous system damage was caused following exposure to 54 to 110 mg/kg thallium nitrate. It has also been found that thallium can cross the human placenta, although developmental effects are not well characterized (ATSDR 1992).
Magnitude of risk	For the toddler Seasonal User and Gahcho Kué workers, the HI increased between the Baseline Case and Application Case (increases of 8.1 and 1.0 for the high and low fish consumers, respectively) and exceeds the target HI (HI=16 and 6 for the high and low fish consumers, respectively), indicating that incremental concentrations from the Project may influence human health. The fish tissue concentrations which are the primary contributor to the risk estimates were derived from predicted water concentrations and a BAF derived based on non-detect concentrations of thallium in fish tissues (<0.04 mg/kg wet weight). A BAF derived based on non-detect concentrations is likely to over-estimate fish tissue concentrations and therefore over-estimate risks to human health from fish ingestion. Therefore, the magnitude of risk from the Project was considered to be low. For Gahcho Kué workers, the HI exceeded the target HI (HI=1.6); however, the increase from the Baseline Case to the Application Case was small (difference in HI values of 0.1). Therefore, for the Gahcho Kué worker, the magnitude of risk was considered negligible.

Table 7-8 Further Analysis of Neurotoxic Effects and Determination of Magnitude of Risk (Non-carcinogenic Effects)

Analysis Criteria	Discussion
Magnitude of risk estimate in the Application Case	For the toddler Seasonal User, the HI for neurotoxic effects in the Application Case exceeded the target threshold of 1.0 for the high and low fish consumers (HI = 2.0). The HI for the Gahcho Kué worker was below 1.0.
Comparison of Baseline Case to Application Case	The HIs for the toddler Seasonal User were higher in the Application Case compared to the Baseline Case (difference in HI of 0.4 for the high fish consumer and difference of 0.5 for the low fish consumer).
Conservatism and uncertainty in predictions	Several sources of uncertainty in the HHRA relate to the air quality predictions and deposition rates used to predict COC concentrations in country food items. The air quality and deposition rate predictions use the maximum emission rates from the Project; however, this is a conservative assumption because most equipment does not operate at its maximum capacity on a continuous basis. This assumption can lead to overestimation of the potential Project impacts for the longer averaging periods (24-hour and annual). Country food predictions are based on soil quality and deposition rates for the Project Boundary which is where the highest deposition rates occur.
Conservatism in the exposure assumptions	The exposure assessment used site-specific chemistry data and exposure intake values for the Northern Aboriginal populations as available. The exposure assessment assumes the consumption of country foods for six months of the year which is a conservative assumption as many country foods are only available seasonally; however, this assumption is protective of receptors that may treat and store country food items for consumption throughout the year. It is also possible that there will not be sufficient quantity of country food items present in the immediate vicinity of the Site to support this assumption and as a result, fifty percent (50%) of the daily country food consumption was assumed to be from the Site. In the Application Case, the primary exposure pathway that contributed to the HI values for the toddler Seasonal User was background dietary intake, with manganese contributing the majority of risk to the HI. In all exposure scenarios, the ingestion of background dietary items was the primary contributor to the risk estimate, followed by inhalation of manganese in air. Excluding the dietary intake would result in an HI of 0.8 for the high fish consumer Toddlers and 0.6 for the low fish consumer Toddlers in the Application Case.
Conservatism in the toxicity reference values	Below is a summary of the TRVs for the pathways that had the greatest contribution to the HI values. **Aluminum** A U.S. EPA provisional RfD of 1 mg/kg/day has been derived for aluminum (U.S. EPA 2012a). The RfD is based on the LOAEL of 100 mg Al/kg-day for minimal neurotoxicity in the offspring of mice (U.S. EPA 2006). An uncertainty factor of 100 was applied to the LOAEL (3 for use of a minimal LOAEL, 10 for interspecies extrapolation and 3 for intrahuman variability where the critical effects have been observed in a sensitive sub-group. **Manganese** Health Canada (2009b) has developed an oral TDI of 0.1 mg/kg/day for toddlers and children and of 0.2 mg/kg/day for adults based on a weight of evidence approach from human epidemiological and experimental studies. The toxicological endpoint upon which the oral TDI is based is Parkinsonian-like neurotoxicity. No uncertainty factors have been utilized in the derivation of the oral TDI. The inhalation RfC for manganese of 0.05 µg/m^3 was based upon impairment of neurological function in an occupational study using manganese dioxide (U.S. EPA 2012b). The LOAEL of 0.15 mg/m^3 was adjusted for continuous exposure to a $LOAEL_{adj}$ of 0.05 mg/m^3. An uncertainty factor of 1000 was applied (10 for the use of a LOAEL, 10 for the protection of sensitive individuals, and 10 for database limitations including the lack of developmental data, the less than chronic exposure periods, and potential differences in toxicity of different forms of manganese).
Potential health effects	The toxicity reference values for aluminum are based on neurotoxic effects (i.e., psychomotor and cognitive impairment). Manganese is an essential element for humans and is found widely throughout the body; adverse health effects can be linked to both manganese deficiency as well as excessive manganese levels. Bone mineralization, protein and energy metabolism, metabolic regulation, cellular protection from free radicals are all functions that require manganese. Manganese is also a component of metalloenzymes and can act as an enzyme activator (ATSDR 2008).

Table 7-8 Further Analysis of Neurotoxic Effects and Determination of Magnitude of Effect (Non-carcinogenic Effects) (continued)

Analysis Criteria	Discussion
Magnitude of risk	For the toddler Seasonal User, the HIs for additive effects of aluminum and manganese increased between the Baseline Case and Application Case (increase in HI of 0.4 and 0.5 for the high and low fish consumers, respectively) and exceed the target HI for both the high and low fish consumers (HI = 2.0). The majority of the risk associated with aluminum and manganese was attributable to background dietary intake rather than exposure as a result of the Project. Therefore, the magnitude of risk from the Project was considered to be low for Seasonal Users.

Table 7-9 Further Analysis of Respiratory Tract Effects (Additive Inhalation Effects of Antimony, Arsenic, Cobalt, Nickel, Titanium and Vanadium) and Determination of Magnitude of Risk (Non-carcinogenic Effects)

Analysis Criteria	Discussion
Magnitude of risk estimate in the Application Case	For the toddler Seasonal User, the HI for respiratory tract effects in the Application Case was equal to the target threshold of 1.0 (HI = 1.0). The HI for the Gahcho Kué Worker also exceeded the target threshold of 1.0 (HI = 1.2).
Comparison of Baseline Case to Application Case	The HIs for the Baseline Case were less than the target threshold of 1.0 for both the Seasonal User and the Gahcho Kué Worker. The HI for the toddler Seasonal User was higher in the Application Case compared to the Baseline Case (difference in HI of 1.0). For the Gahcho Kué Worker, the HI in the Application Case was higher than that in the Baseline Case (difference in HI of 1.2).
Conservatism and uncertainty in predictions	Several sources of uncertainty in the HHRA relate to the air quality predictions and deposition rates used to predict COC concentrations. The air quality and deposition rate predictions use the maximum emission rates from the Project; however, this is a conservative assumption because most equipment does not operate at its maximum capacity on a continuous basis. This assumption can lead to overestimation of the potential Project impacts for the longer averaging periods (24-hour and annual).
Conservatism in the exposure assumptions	The exposure assessment used site-specific chemistry data and exposure intake values for the Northern Aboriginal populations as available. In the Application Case, the primary inhalation exposure pathway that contributed to the HI value for the toddler Seasonal User and for the Worker was the inhalation of air (rather than dust) with cobalt, nickel and titanium being the main contributors.
Conservatism in the toxicity reference values	Below is a summary of the inhalation TRVs for cobalt, nickel and titanium, which had the greatest contribution to the HI values. For cobalt, the provisional value of 0.006 µg/m^3 was chosen from U.S. EPA NCEA as the RfC. The non-carcinogenic RfC for cobalt is based on decreased pulmonary function in workers (Nemery et al. 1992). The NOAEL of 5.3 µg/m^3 was adjusted for continuous exposure (to 1.9 µg/m^3) and an uncertainty factor of 300 was applied to account for three separate factors (a factor of 3 for extrapolation from an assumed subchronic to chronic study, a factor of 10 for database insufficiencies including lack of developmental inhalation studies and multigenerational studies, and a factor of 10 to account for lack of data on human variability and sensitive populations). Health Canada (2009b) has derived an inhalation TC of 1.8E-05 mg/m3 for sulphidic nickel (nickel subsulphide). The TC is based on a subchronic study where rats and mice were exposed to 0, 0.11, 0.2, 0.4, 0.9 and 1.8 mg/m^3 nickel subsulphide for 6 hours/day, 5 days/week, for 13 weeks. A NOAEL of 0.1 mg/m^3 was determined for mice, and a LOAEL of 0.1 mg/m3 was determined for rats, based on respiratory track effects (alveolar macrophages, hyperplasia). The dose was adjusted by applying factors of 6 hours/24 hours and 5 days/7 days for conversion to continuous exposure. An uncertainty factor of 1000 was applied to the adjusted dose (a factor of 10 each for intraspecies variation, interspecies variation, and for a less than chronic study). The inhalation RfC for titanium tetrachloride of 0.0001 mg/m^3 is based upon increased irregular breathing and rhinitis (ATSDR 1997). Vapours of titanium tetrachloride where generated by passing nitrogen over liquid titanium tetrachloride. An uncertainty factor of 100 was applied to the LOAEL of 0.1 mg/m^3 which was adjusted to a human equivalent concentration ($LOAEL_{HEC}$ 0.012 mg/m^3). The uncertainty factor was comprised of a factor of 3 for the use of a LOAEL rather than a NOAEL, a factor of 3 for extrapolation from animals to humans and 10 for the protection of sensitive human populations.

Table 7-9 Further Analysis of Respiratory Tract Effects (Additive Inhalation Effects of Antimony, Arsenic, Cobalt, Nickel, Titanium and Vanadium) and Determination of Magnitude of Effect (Non-carcinogenic Effects) (continued)

Analysis Criteria	Discussion
Potential health effects	Below is a summary of the potential health effects of cobalt, nickel and titanium, which had the greatest contribution to the HI values. Inhalation of cobalt can affect the respiratory system and if sufficient quantities are inhaled (0.003 mg/m^3), irritation, wheezing, asthma and pneumonia can result. Occupational exposure to cobalt concentrations of 0.038 mg/m^3 for six hours resulted in breathing difficulties, although these levels are approximately 10,000 to 100,000 times the typical outdoor air concentration. Individuals can also develop sensitivity to cobalt through occupational exposure to concentrations ≥0.007 mg/m^3, and subsequent exposures can result in skin rashes or asthma attacks. Data available for chronic nickel inhalation exposure for humans are limited to occupational data. Respiratory effects found in nickel workers included chronic bronchitis, emphysema, and reduced vital capacity. These workers were also exposed to other metals, so it cannot be concluded that nickel is the sole causative agent of the effects observed. Asthma from primary irritation and as the result of dermal sensitization has also been documented amongst nickel workers. Nickel refinery workers with elevated urinary nickel concentrations also showed a significant increase in urinary β2-microglobulin levels, which is indicative of tubular dysfunction in the kidneys (ATSDR 2005). Inhalation of titanium tetrachloride can result in irritation to the skin, eyes, mucous membranes, and the lungs. Long term effects can result from short-term inhalation exposure to titanium tetrachloride. Short term effects such as coughing, tightness in the chest, chemical bronchitis or pneumonia and congestion of mucous membranes, can result in long-term effects such as narrowing of the vocal cords, windpipe and upper airways (ATSDR 1997). Laboratory animals exposed to titanium tetrachloride chronically (for two years) by inhalation have developed lung tumours (ATSDR 1997).
Magnitude of risk	For the toddler Seasonal User, the HI for additive effects of antimony, arsenic, cobalt, nickel, titanium and vanadium equalled the target threshold (HI = 1.0), and increased between the Baseline Case (where the HI was less than 1.0) and the Application Case),. The magnitude of risk from the Project was considered to be negligible for Seasonal Users. For Gahcho Kué Workers, the HI for respiratory tract effects exceeded the target threshold of 1.0 (HI = 1.2), and increased between the Baseline Case (where the HI was less than 1.0) and the Application Case, indicating that incremental COC air concentrations from the Project may influence human health and result in adverse effects on the respiratory tract. Therefore, the magnitude of risk from the Project was considered to be low for Workers.

Figure 7-1 Hazard Index Breakdown by Pathway for the Toddler Seasonal User (Application Case)

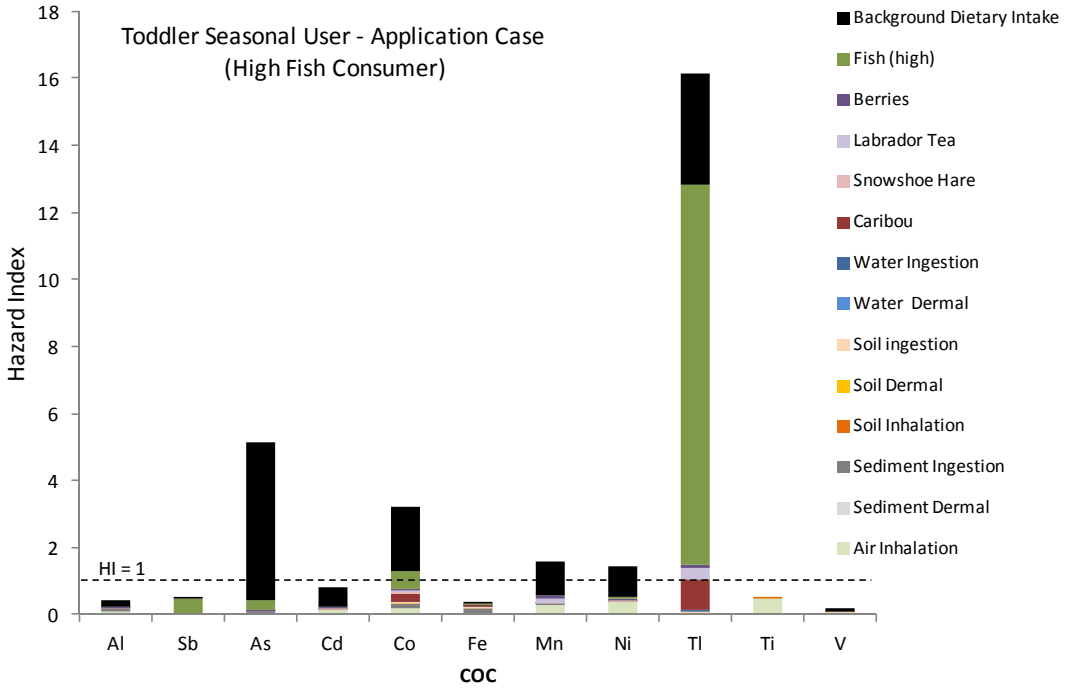


Figure 7-2 Hazard Index Breakdown by Pathway for the Toddler Seasonal User (Application Case) Without Background Dietary Intake

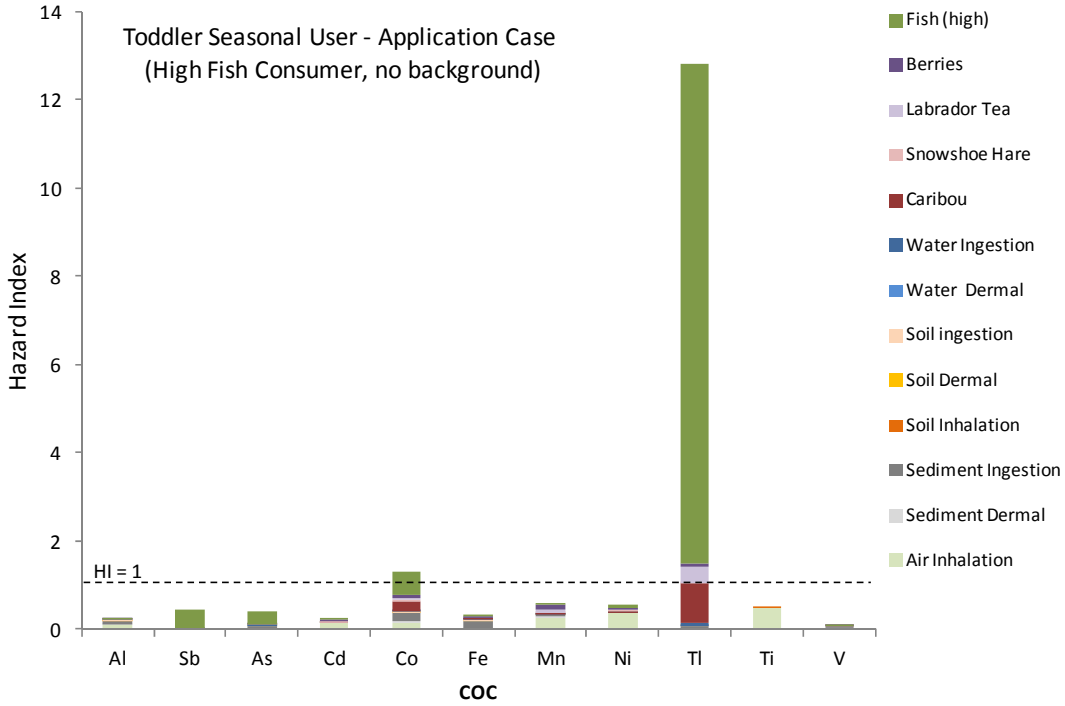


Figure 7-3 ILCR Breakdown by Pathway for the Adult Seasonal User (Application Case)

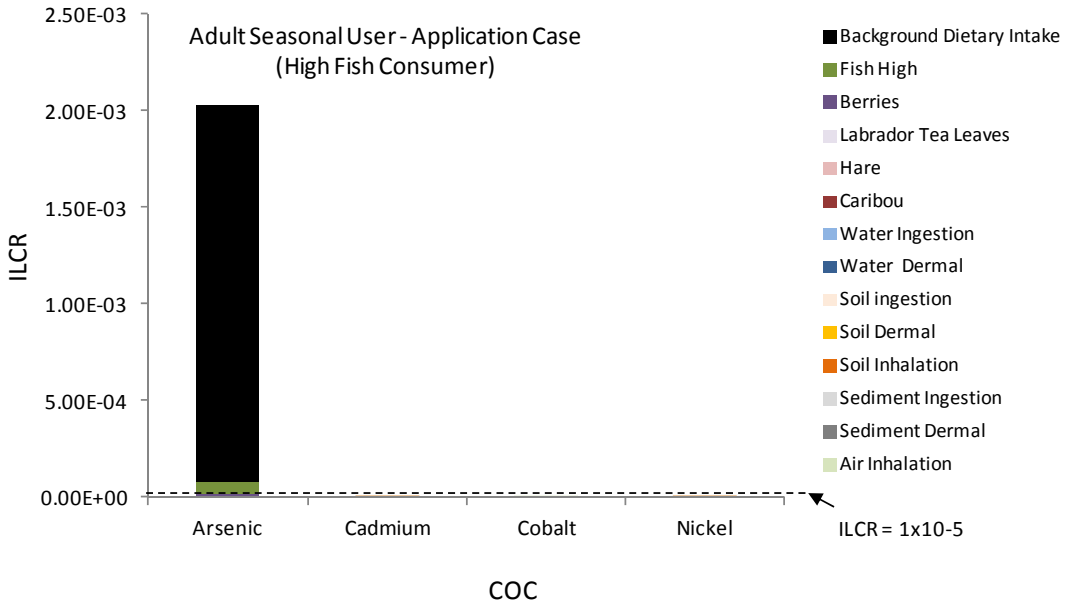
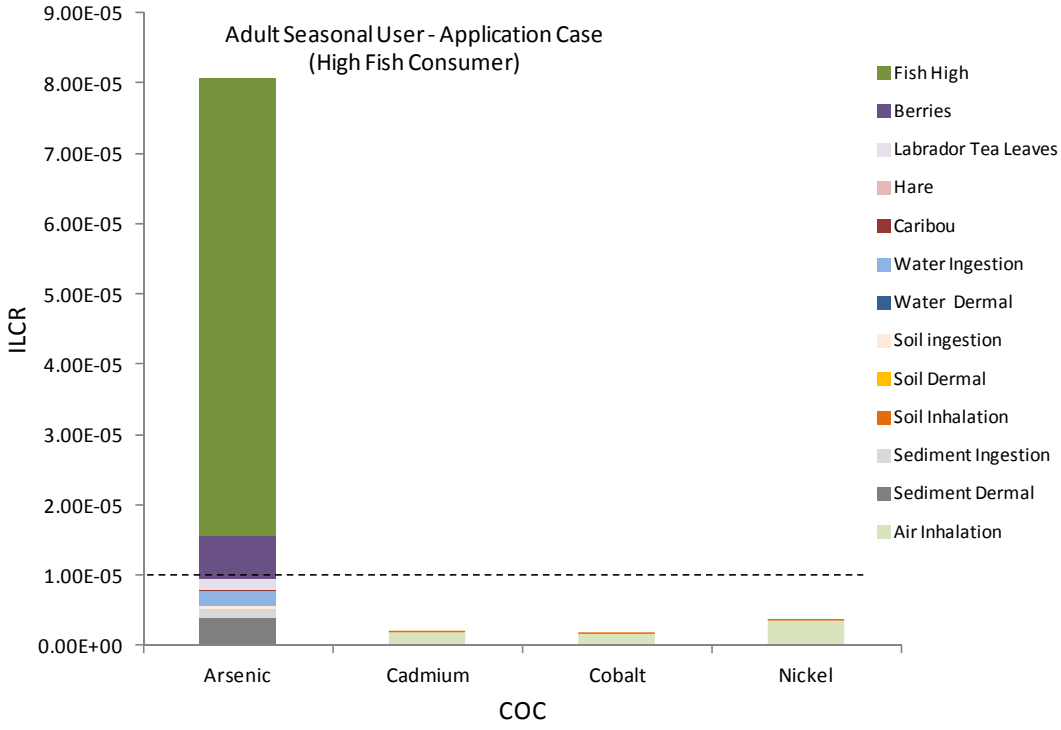


Figure 7-4 ILCR Breakdown by Pathway for the Adult Seasonal User (Application Case) Without Background Dietary Intake



7.1 FURTHER ANALYSES OF PARAMETERS EXCEEDING TARGET RISK LEVELS

7.1.1 Arsenic

The HI values for arsenic are greater than the target threshold of 1.0 for the toddler Seasonal User (high and low fish consumers). For the Worker, the HI values also exceeded the target threshold of 1.0. The primary exposure pathway that contributed to the HI value for all receptors was the ingestion of background dietary items.

Based on the analysis of arsenic as a carcinogen, ILCR values were greater than the target threshold of 1.0×10^{-5} for both the Seasonal User and the Worker. For all receptors, the ingestion of background dietary items was the primary pathway that contributed to the ILCR.

Further analysis of the potential risks posed by arsenic is provided in Table 7-3.

7.1.2 Cobalt

The HI values for cobalt are greater than the target threshold of 1.0 for the toddler Seasonal User (high fish consumer and low fish consumer) and for the Gahcho Kué Worker. The primary exposure pathway that contributed to the HI values was the ingestion of background dietary items. For the Seasonal Users, fish was the secondary exposure pathway contributing to the risk estimate whereas the inhalation of air was the secondary exposure pathway for the Worker.

Further analysis of the potential risks posed by cobalt is provided in Table 7-4.

7.1.3 Manganese

The HI values for manganese are greater than the target threshold of 1.0 for the toddler Seasonal User (high fish consumer and low fish consumer) but were below 1.0 for the Gahcho Kué Worker. The primary exposure pathway that contributed to the HI values for the toddler Seasonal User was the ingestion of background dietary food items followed by the inhalation of air, ingestion of berries, fish, Labrador tea, and sediment.

Further analysis of the potential risks posed by manganese is provided in Table 7-5.

7.1.4 Nickel

The HI values for nickel are greater than the target threshold of 1.0 for the toddler Seasonal Users (high and low fish consumer) but not for the Gahcho Kué Workers. The ILCR value did not exceed the target risk level of 1.0E-5 for Seasonal Users (composite receptor) or the Gahcho Kué Workers. The primary exposure pathway that contributed to risk estimates exceeding target levels for the Seasonal User was the ingestion of background dietary items followed by inhalation of air, ingestion of fish and ingestion of snowshoe hare.

Further analysis of the potential risks posed by nickel is provided in Table 7-6.

7.1.5 Thallium

The HI values for thallium are greater than the target threshold of 1.0 for the toddler Seasonal User (high and low fish consumers) as well as the Gahcho Kué worker. The primary exposure pathway that contributed to the HI value for these receptors was fish ingestion, followed by background dietary intake and ingestion of caribou.

Further analysis of the potential risks posed by thallium is provided in Table 7-7.

7.1.6 Neurotoxic Effects (Additive Effects of Aluminum and Manganese)

The HI values for the additive effect of neurotoxicity from aluminum and manganese are greater than the target threshold of 1.0 for the toddler Seasonal User (high fish consumer and low fish consumer), but below 1.0 for the Gahcho Kué worker. The primary exposure pathway that contributed to the HI values for the toddler Seasonal User was background dietary intake.

Further analysis of the potential risk of neurotoxic effects is provided in Table 7-8.

7.1.7 Non-Carcinogenic Respiratory Effects (Additive Inhalation Effects of Antimony, Arsenic, Cobalt, Nickel, Titanium and Vanadium)

The HI values for the additive effects on the respiratory tract from inhalation exposure to antimony, arsenic, cobalt, nickel, titanium and vanadium are greater than the target threshold of 1.0 for the toddler Seasonal User and the Gahcho Kué Worker. The primary inhalation exposure pathway that contributed to the HI

values was the inhalation of air (rather than dust), with cobalt, nickel and titanium being the main contributors to risk.

Further analysis of the potential risk of respiratory tract effects is provided in Table 7-9.

7.2 RESIDUAL EFFECTS CLASSIFICATION

Residual effects to human health for chronic multi-media exposure to emissions from the Project are classified in Table 7.2-1. The effect classification criteria are already incorporated into the risk estimates as described in Section 2.4.1; therefore, residual effects are defined by the magnitude of risk as determined from risk estimates.

Table 7.2-1 Residual Effect Classification for Chronic Multi-Media Assessment for the Application Case

Parameter	Location	Magnitude of Chronic Risks as a Result of the Project
Chronic Multi-Media Risk Assessment		
Arsenic (carcinogenic and non-carcinogenic effects)	Seasonal Users	low
Arsenic (carcinogenic and non-carcinogenic effects)	Gahcho Kué workers	negligible
Cobalt	Seasonal Users	low
Cobalt	Gahcho Kué worker	negligible
Manganese	Seasonal Users	low
Manganese	Gahcho Kué workers	negligible
Nickel	Seasonal Users	low
Nickel	Gahcho Kué workers	negligible
Thallium	Seasonal Users – high fish consumers	low
Thallium	Seasonal Users – low fish consumers	low
Thallium	Gahcho Kué workers	low
Sum of neurotoxicants	Seasonal Users	low
Sum of neurotoxicants	Gahcho Kué workers	negligible
Sum of respiratory toxicants (via inhalation)	Seasonal Users	negligible
Sum of respiratory toxicants (via inhalation)	Gahcho Kué workers	low
Remainder of COCs	all locations	negligible

7.3 PREDICTION CONFIDENCE

7.3.1 Prediction Confidence

Uncertainty is associated with risk assessment predictions, depending on the quality, quantity and variability associated with available information. When information is uncertain, it is standard practice in a risk assessment to make assumptions that are biased towards safety (i.e., conservative assumptions). Conservative assumptions are used so that risks are not underestimated for the maximally exposed individual. If chemical emissions are safe for this maximally exposed individual, then chemical exposures will be safe for all people. By using these conservative assumptions, there is considerable confidence that potential risks are not underestimated. Rather, these conservative assumptions weigh heavily toward an overestimation of the true risk.

The magnitude of conservatism differs for each parameter, medium and exposure pathway. For all chemicals which were identified as a COC and have a risk estimate greater than the target risk level, the layers of safety, including conservatism in the assumptions and level of uncertainty, are discussed when evaluating the magnitude of the risk.

8 RISK ASSESSMENT CONCLUSIONS

The HHRA section of the Application presents an assessment of the potential effects of chemical emissions from the Project on the health of people. Both the potential short-term (acute) and long-term (chronic) human health effects that may occur as a result of chemical emissions from the Project were assessed, following a risk assessment approach. The conclusions of the HHRA are summarized below.

8.1 SHORT-TERM EFFECTS

Short-term effects to human health as result of the Project were predicted to be negligible for exposure to all chemicals in air at all locations for the Application Case, except for iron. Risks for acute exposure to benzo(a)pyrene, iron, manganese and nickel effects were predicted to be low (non-negligible) for the Gahcho Kué worker camp and/or the Project Boundary.

It is noted that the predicted acute air concentrations are very conservative and likely overestimate risk. The conservatism in the particulate matter predictions is discussed further in Section 5.3.

8.2 PARTICULATE MATTER

Potential health effects of particulate matter ($PM_{2.5}$ and PM_{10}) increases as a result of the Project were assessed qualitatively by a review of key epidemiological studies focussed on health effects associated with particulate matter from crustal sources.

Overall, a great deal of uncertainty remains in evaluating the predicted particulate matter concentrations. $PM_{2.5}$ and PM_{10} concentrations were above Canada Wide Standards at the Employee Camp and Project Boundary. Road dust (i.e., crustal sources) appears to be the main contributor to PM concentrations predicted for the Project (Section 3.1 of the 2012 Updated Air Quality Assessment [De Beers 2012b]). Most epidemiology studies suggest that dust derived from crustal sources is less hazardous than dust derived from combustion sources. This may be attributable to contaminants adsorbed onto dust derived from combustion sources, but may also be attributable to the generally smaller size of dust from combustion sources.

Based on the literature review, the potential for increased mortality or health effects at the Employee Camp and Project Boundary are uncertain as some

studies would indicate the potential for adverse health effects and others would not.

The methods of assessment of health effects from exposure to respirable particulate matter are derived from epidemiology studies based on large urban centres making comparisons to small rural communities challenging. In addition, the database related to health effects from particulate matter relies heavily on studies where the particulates are derived from combustion sources. Few studies were available concerning possible health outcomes from wind-blown dust (i.e., road dust in this case) – particularly for fine particulates (i.e., $PM_{2.5}$); however, some studies have found adverse health effects (Fuentes et al. 2006). These studies would suggest that health effects at the Employee Camp and Project Boundary are possible. In light of these uncertainties, no firm conclusions regarding potential health effects from respirable particulate matter exposure at the Employee Camp and Project Boundary could be drawn. The assessment conducted is similar to that conducted for other mines. Occupational health and safety policies and procedures are designed to reduce air quality risks to workers, and such policies and procedures will be consistently applied to this Project.

Based on the conclusions above, the following is recommended:

- Further monitoring of particulate matter concentrations during the construction and operations phases of the Project may be useful to capture real (i.e., measured) concentrations, as current data are based on modeling, or to determine the success of dust control measures.
- Further investigation of risk management options for minimizing the generation of dust. The following conceptual measures are provided for consideration:
  - Assessment of heating and ventilation air conditioning systems in the buildings at the Employee camp to understand the potential for reduction of exposure to particulate matter.
  - Confirm that DeBeers Health and Safety Plan covers the mitigation of exposure of workers to dust and particulate matter.
  - If the current DeBeers Health and Safety Plan does not cover the mitigation of exposure of workers to dust and particulate matter, an industrial hygienist should develop one.
- There is an indication from the available information that road dust significantly contributes to dust exposure. Management of road dust is a cost-effective method of dust exposure control that may contribute significantly to improved air quality.

It is noted that the predicted particulate matter concentrations are very conservative and likely overestimate risk. It is also unlikely that people will spend extended periods of time adjacent to the Project Boundary. The conservatism in the particulate matter predictions is discussed further in Section 5.3.

8.3 LONG-TERM EFFECTS

Long-term inhalation effects to human health as a result of the Project were predicted to be negligible for exposure to all COCs except for acrolein (at the Project Boundary and Gahcho Kué worker camp). Uncertainties associated with the air modelling predictions are the main contributor to these elevated risk estimates. A preliminary analysis conducted following completion of the Air Quality Assessment indicates that the air concentrations may be overestimated by an order of magnitude.

The following provides a summary of the magnitude of long-term risks for combined exposures to COCs in air, water, soil, plants, fish and wild game:

- Magnitude of long-term risks to human health as a result of the Project were predicted to be negligible for the combined exposures for arsenic, cobalt, manganese, nickel and neurotoxicants for Gahcho Kué Workers;
- Magnitude of long-term risks to human health as a result of the Project were predicted to be low for the combined exposures for arsenic, cobalt, manganese, nickel, neurotoxicants, respiratory toxicants and thallium for Seasonal Users;
- Magnitude of long-term risks to human health as a result of the Project were predicted to be low for the combined exposures for thallium and respiratory toxicants for Gahcho Kué Workers.

Although the overall magnitude of risk was considered low for several COCs, many of the assumptions applied in the multi-media assessment overestimate risk. The primary assumptions that would contribute to uncertainty in this risk assessment include the following:

- Revision of the conservatism in the method used to predict air concentrations and deposition rates such as refinement of emission factors and deposition velocity rates selected.
- Parameterization of emissions from diffuse area sources is difficult to simulate in dispersion models. The Project area emission sources include mine pits, roads, and mine rock piles. The Project fugitive particulate matter emissions are difficult to quantify accurately. The estimated fugitive particulate matter emissions assessed in the EIS are

decidedly conservative. Based on a review of the PM monitoring data at the Snap Lake Mine and the Ekati Mine, the high particulate matter impacts identified in this assessment are due in part to the conservative emission estimates.

- Predictive air modeling using maximum emission rates from the Project will provide conservative results because most equipment does not operate at its maximum capacity on a continuous basis. This assumption can lead to overestimation of the potential Project impacts for the longer averaging periods (24-hour and annual).
- Receptors were assumed to obtain a large portion (50%) of their food sources (i.e., plants, meat and fish) from the location where the maximum deposition occurs or obtain. Furthermore, receptors are assumed to eat these food items for six months of the year for their lifetime.
- Background dietary intake was included in the determination of risk estimates; for several parameters (e.g., arsenic), background dietary intake was the main driver of the elevated risk estimates, and this pathway alone would result in an HQ greater than the target threshold of 1.0.

9 MONITORING AND FOLLOW-UP

The human health risk assessment identified negligible to low potential for adverse health effects associated with dust impacts at the Gahcho Kué worker camp and at the Project Boundary. Note that identification of these potential effects only indicates the possibility that they could occur, not a probability that they could or will occur. Note further that such identification of possible effects is due to the conservative emission estimates used in this assessment, as discussed previously. In other words, the potential risks identified will not occur at the levels indicated because of the conservative nature of the assumptions.

De Beers is committed to the following management practices for dust control which will mitigate or reduce potential effects to human health:

- water spray application to control dust emissions on haul roads during summer;
- managing vehicle speed to limit wind-blown dust from vehicle wheel entrainment; and,
- regularlygrading the haul roads to maintain a surface of coarse material on the road surface.

Finally, De Beers plans to incorporate the results of its ambient air quality monitoring program for all substances of potential concern into its emission management plans as part of its response to the principle of continuous improvement and as part of its plans to link monitoring to adaptive management.

10 REFERENCES

Andersen, Z.J., P. Wahlin, Raaschou-Nielsen O, Scheike T. and S. Loft. 2007. *Ambient particle source apportionment and daily hospital admissions among children and elderly in Copenhagen*. Journal of Exposure Science and Environmental Epidemiology 17:625-636.

ATSDR (Agency for Toxicological Substances and Disease Registry). 1992. *Toxicological profile for thallium. July 1992*. ATSDR, US Public Health Service.

ATSDR. 1997. *Toxicological Profile for Titanium Tetrachloride*. US Department of Health and Human Services. Public Health Service, Agency for Toxic Substances and Disease Registry. September 1997. Atlanta, GA.

ATSDR. 2005. *Toxicological Profile for Nickel*. US Department of Health and Human Services. Public Health Service, Agency for Toxic Substances and Disease Registry. August 2005. Atlanta, Georgia.

ATSDR. 2007. *Toxicological Profile for Acrolein*. U.S. Department of Health and Human Services, Public Health Service. August 2007. CRC Press Inc. Atlanta, GA.

ATSDR. 2008. *Toxicological Profile for Manganese*. U.S. Department of Health and Human Services, Public Health Service. August 2007. CRC Press Inc. Atlanta, GA.

ATSDR. 2012. *ATSDR ToxProfiles*. Available at: http://www.atsdr.cdc.gov/toxprofiles/index.asp. Accessed July 2012.

Bourcier, T., F. Thomas, T. Bury, L. Cadiot, V. Borderie and L. Laroche. 2003. *Effects of air pollution and climatic conditions on the frequency of opthalmological emergency examinations*. British Journal of Ophthalmology 87: 809-811.

Buist, A.S., L.R. Johnson, W.M. Vollmer, G.J. Sexton and P.H. Kanarek. 1983. *Acute effects of volcanic ash from Mount St. Helens on lung function in children*. American Review of Respiratory Disease 127:714-719.

CalEPA OEHHA (California Environmental Protection Agency, Office of Environmental Health Hazard Assessment). 2012. *Table 1*. *Consolidated Table of OEHHA/ARB Approved Risk Assessment Health Values*. Updated May 3, 2012. California Air Resources Board, California Environmental Protection Agency.

CCME (Canadian Council of Ministers of the Environment). 1999. *Canadian Environmental Quality Guidelines*. Available on-line at: http://ceqg-rcqe.ccme.ca/. Accessed July 2012.

CCME. 2006. *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines*. PN1332.

CCME. 2007. *Guidance Document on Achievement Determination*. Canada-Wide Standards for Particulate Matter and Ozone. Revised. PN 1391. Canadian Council of Ministers of the Environment.

Chang, C., I. Lee, S. Tsai and C. Yang. 2006. *Correlation of Asian Storm Events with Daily Clinic Visits for Allergic Rhinitis in Taipei, Taiwan*. Journal of Toxicology and Environmental Health, Part A, 69: 229-235.

Chen, Y. and C. Yang. 2005. *Effects of Asian Dust Storm Events on the Daily Hospital Admission for Cardiovascular Disease in Taipei, Taiwan*. Journal of Toxicology and Environmental Health, Part A, 68: 1457-1464.

Chen, Y.S., P.C. Sheen, E.R. Chen, Y.K. Liu, T.N. Wu and C.Y. Yang. 2004. *Effects of Asian dust storm events on daily mortality in Taipei, Taiwan*. Environmental Research 95(2): 151–155.

De Beers (De Beers Canada Inc.). 2010. *Environmental Impact Statement for the Gahcho Kué Project. Volumes 1, 2, 3a, 3b, 4, 5, 6a, 6b, 7 and Annexes A through N*. Submitted to Mackenzie Valley Environmental Impact Review Board. December 2010.

De Beers. 2011. *Environmental Impact Statement for the Gahcho Kué Project. Volumes 3a Revision 2, 3b Revision 2, 4 Revision 2, and 5 Revision 2*. Submitted to the Mackenzie Valley Environmental Impact Review Board in Response to the Environmental Impact Statement Conformity Review. July 2011.

De Beers. 2012a. *Environmental Impact Statement Supplemental Information Submission for the Gahcho Kué Project*. Submitted to the Mackenzie Valley Environmental Impact Review Board, Yellowknife, NWT, Canada. May 2012.

De Beers. 2012b. *Environmental Impact Statement for the Gahcho Kué Project - 2012 Updated Air Quality Assessment*. Submitted to the Mackenzie Valley Environmental Impact Review Board, Yellowknife, NWT, Canada. August 2012.

Fuentes, M., H.R. Song, S.K. Ghosh, D.M. Holland and J.M. Davis. 2006. *Spatial association between speciated fine particles and mortality*. Biometrics 62(3):855-63.

Gahcho Kué Panel. 2007. *Terms of Reference for the Gahcho Kué Environmental Impact Statement*. Mackenzie Valley Environmental Impact Review Board. Yellowknife, NWT. October 5, 2007.

GNWT (Government of the Northwest Territories). 2003. *Environmental Guideline for Contaminated Sites*. Appendix 4. NWT. November 2003. Available on-line at: http://www.enr.gov.nt.ca/library/pdf/eps/siteremediation.pdf. Accessed November 2010.

GNWT. 2011. *Ambient Air Quality Standards*. Available at: http://www.enr.gov.nt.ca/_live/documents/content/air_quality_standards_guideline.pdf. Accessed July 2012.

Golder (Golder Associates Ltd.). 2012a. 2011 Water Quality and Sediment Quality Supplemental Monitoring Report. Report No. 11-1365-0001/DCN-050. Submitted to De Beers Canada Inc. March 2012.

Golder (Golder Associates Ltd.). 2012b. Determination of Natural Winter Mitigation of Road Dust Emissions from Mining Operations in Northern Canada. Submitted to De Beers Canada Inc.

Gordian, M.E., H. Ozkaynak, J. Xue, S. Morris and J.D. Spengler. 1996. *Particulate Air Pollution and Respiratory Disease in Anchorage, Alaska*. Environmental Health Perspectives 104 (3): 290-297.

Government of Canada. 1993. *Canadian Environmental Protection Act*. Priority Substances List Assessment Report – Arsenic and its Compounds. Government of Canada, Health and Welfare Canada, Environment Canada. Minister of Supply and Services Canada, 1993.

Gupta, S.K., S.C. Gupta, R. Agarwal, S. Sushma, S.S. Agrawal and S. Rohit. 2007. *A multicentric case-control study on the impact of air pollution on eyes in a metropolitan city of India*. Indian Journal of Occupational and Environmental Medicine 11(1): 37-40.

Halm, C. 2010. Phone conversation with Chris Halm (Air Pollution Specialist) RE: Consolidated Table of OEHHA / ARB Approved Risk Assessment Health Values. California Air Resources Board, California Environmental Protection Agency. June 7, 2010.

Health Canada. 1995. *Human Health Risk Assessment of Chemicals from Contaminated Sites: Volumes I and II*. Health Canada. Ottawa, ON.

Health Canada. 2007. *Human Health Risk Assessment of Mercury in Fish and Health Benefits of Fish Consumption*. Bureau of Chemical safety Food Directorate Health Products and Food Branch.

Health Canada. 2008. *Guidelines for Canadian Drinking Water Quality Summary Table*. Prepared by the Federal-Provincial-Territorial Committee on Drinking Water of the Federal-Provincial-Territorial Committee on Health and the Environment, May 2008. Available at: http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/sum_guide-res_recom/index-eng.php. Accessed July 2012.

Health Canada. 2009a. *Federal Contaminated Site Risk Assessment in Canada Part 1: Guidance on Human Health Preliminary Quantitative Risk Assessment Version 2.0, May 2009*. Health Canada. Ottawa, ON.

Health Canada. 2009b. *Federal Contaminated Site Risk Assessment in Canada Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors*. Health Canada. Ottawa, ON.

Health Canada and Environment Canada. 1999. *National Ambient Air Quality Objectives For Particulate Matter Addendum To The Science Assessment Document A Report By The Federal - Provincial Working Group on Air Quality Objectives and Guidelines December 1997, Revised April 1999*. Available on-line at: http://www.city.grandforks.bc.ca/air/airshed/simmsresources/particule_matter_9_e.pdf

Hefflin, B.J., B. Jalaludin and E. McClure. 1991. *Surveillance for dust storms and respiratory diseases in Washington State*. Archives of Environmental Health 49: 170-174.

HSDB (Hazardous Substances Database). 2010. *Iron*. United States National Library of Medicine.TOXNET Toxicology Data Network. US National Library of Medicine, Bethesda, MD. National Institutes of Health. Health and Human Services. Last Revised and reviewed in 2010. Available at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed August 2012.

IARC (International Agency for Research on Cancer). 2012. *Agents classified by the IARC monographs*. Volumes 1 to 105. Available at: http://monographs.iarc.fr/ENG/Classification/ClassificationsAlphaOrder.pdf. Accessed August 2012.

Jin, A. 2010. E-mail correspondence between Mike Demarchi of LGL Limited Environmental Research Associates and Andrew Jin, Health Canada. Dated June 15, 2010.

Kwon, H-J., S-H. Cho, Y. Chun, F. Lagarde and G. Pershagen. 2002. *Effects of the Asian dust events on daily mortality in Seoul, Korea*. Environmental Research Section A 90: 1-5.

Laden, F., L.M. Neas, D.W. Dockery and J. Schwartz. 2000. *Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities*. Environmental Health Perspectives 108: 941-947.

MVEIRB (Mackenzie Valley Environmental Impact Review Board). 2006. *Reasons for Decision and Report of Environmental Assessment for the DeBeers Gahcho Kué Diamond Mine, Kennady Lake, NT*. June 28, 2006.

Nemery, B., P. Casier, D. Roosels, D. Lahaye and M. Demedts. 1992. *Survey of cobalt exposure and respiratory health in diamond polishers*. American Review of Respiratory Disease 145(3): 610-616.

OMOE (Ontario Ministry of the Environment). 2012a. *Ontario's Ambient Air Quality Criteria*. Standards Development Branch, Ontario Ministry of the Environment. April 2012. Available at: http://www.ene.gov.on.ca/environment/en/resources/STDPROD_096586.html. Accessed July 2012.

OMOE. 2012b. *Summary of Standards and Guidelines to support Ontario Regulation 419: Air Pollution — Local Air Quality*. Standards Development Branch, Ontario Ministry of the Environment. April 2012. Available at: http://www.ene.gov.on.ca/environment/en/resources/STDPROD_096528.html. Accessed July 2012.

Ostro, B.D., R. Broadwin and M.J. Lipsett. 2000. *Coarse and fine particles and daily mortality in the Coachella Valley California: a follow-up study*. Journal of Exposure Analysis and Environmental Epidemiology 10: 412-419.

Ostro, B.D., S. Hurley and M. Lipsett. 1999. *Air Pollution and Daily Mortality in the Coachella Valley, California: A Study of PM10 Dominated by Coarse Particles*. Environmental Research, Section A, 81:231-238.

Penttinen, P., P. Tiittanen and J. Pekkanen. 2004. *Mortality and air pollution in metropolitan Helsinki, 1988--1996*. Scandinavian Journal of Work, Environmental and Health. 30(Suppl 2):19-27.

Pope, C.A., R.W. Hill and G.M. Villegas. 1999. *Particulate Air Pollution and Daily Mortality on Utah's Wasatch Front*. Environmental Health Perspectives 107: 567-573.

RIVM (National Institute of Public Health and the Environment). 2001. *Re-evaluation of Human Toxicological Maximum Permissible Risk Levels*. RIVM Report No. 711701 025.

RIVM. 2009. *Re-evaluation of some Human Toxicological Maximum Permissible Risk Levels Earlier Evaluated in the Period 1991-2001*. Bilthoven, the Netherlands.

Schwartz, J., G. Norris, T. Larson, L. Sheppard, C. Claiborne and J. Koenig. 1999. *Episodes of High Coarse Particle Concentrations Are Not Associated with Increased Mortality*. Environmental Health Perspectives 107 (5): 339-342.

Schwarze, P.E., J. Ovrevik, M. Lag, M. Refsnes, P. Nafstad, R.B. Hetland and E. Dybing. 2006. *Particulate matter properties and health effects: consistency of epidemiological and toxicological studies*. Human and Experimental Toxicology 25: 559-579.

Slaughter, J.C., E. Kim, L. Sheppard, J.H. Sullivan, T. Larson and C. Claiborn. 2005. *Association between Particulate Matter and Emergency Room Visits, Hospital Admissions, and Mortality in Spokane, Washington*. Journal of Exposure Analysis and Environmental Epidemiology 15: 153-159.

Staniswalis, J.G., N.J. Parks, J.O. Bader and Y.M. Maldonado. 2005. *Temporal analysis of airborne particulate matter reveals a dose-rate effect on mortality in El Paso: indications of differential toxicity for different particle mixtures*. Journal of the Air and Waste Management Association 55(7): 893-902.

TCEQ (Texas Commission on Environmental Quality). 2006. *Guidelines to Develop Effects Screening Levels, Reference Values, and Unit Risk Factors*. Prepared by Toxicology Section, Chief Engineer's Office, November, 2006. Available at: http://www.tceq.state.tx.us/comm_exec/forms_pubs/pubs/rg/rg-442.html. Accessed November 2010.

TCEQ. 2012. *March 2012 Effects Screening Levels (ESLs)*. Accessed July 2012. http://www.tceq.state.tx.us/implementation/tox/esl/list_main.html#esl_1

U.S. EPA (United States Environmental Protection Agency) 1989. *Risk assessment guidance for Superfund Volume 1. Human health evaluation manual*. (Part A). EPA/540/1-89/002. Available at: http://www.epa.gov/oswer/riskassessment/ragsa. Accessed July 2012.

U.S. EPA 2004. *Risk assessment guidance for Superfund Volume 1. Human health evaluation manual. (Part E Supplemental Guidance for Dermal Risk Assessment, Final)*. EPA/540/R/99/005. Available at: http://www.epa.gov/oswer/riskassessment/ragsa/. Accessed October 2010.

U.S. EPA. 2006. *Provisional Peer Reviewed Toxicity Values for Aluminum*. Superfund Health Risk Technical Support Center, National Centre for Environmental Assessment, Office of Research and Development, U.S. EPA, Cincinnati, OH. 10-23-2006.

U.S. EPA. 2009. *Risk assessment guidance for Superfund Volume 1. Human health evaluation manual. Part F, Supplemental Guidance for Inhalation Risk Assessment.* EPA-540-R-070-002. Available at: http://www.epa.gov/oswer/riskassessment/ragsa/. Accessed October 2010.

U.S. EPA. 2012a. *U.S. EPA Regional Screening Level Air Supporting Table*. May 2012. Available at: http://www.epa.gov/region09/superfund/prg/. Accessed August 2012.

U.S. EPA. 2012b. *Integrated Risk Information System (IRIS)*. Cincinnati, OH. Online Database. http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showSubstanceList. Accessed July 2012.

U.S. EPA. 2012c. *Regional Screening Levels. Regional Residential Tap Water Screening Levels for Chemical Contaminants at Superfund Sites*. http://www.epa.gov/region9/superfund/prg/. Accessed July 2012.

U.S. EPA. 2012d. *Regional Screening Levels. Regional Residential Soil Screening Levels for Chemical Contaminants at Superfund Sites*. http://www.epa.gov/region9/superfund/prg/. Accessed July 2012.

U.S. EPA. 2012e. *Region III Fish Tissue Screening Levels, updated April 2012*. U.S. EPA Region III. Available at: http://www.epa.gov/reg3hwmd/risk/human/index.htm. Accessed July 2012.

WHO (World Health Organization). 1999. *International Program on Chemical Safety. Environmental Health Criteria 210*. Principles for the Assessment of Risks to Human Health from Exposure to Chemicals. Available at: http://www.inchem.org/documents/ehc/ehc/ehc210.htm. Accessed July 2012.

WHO. 2000. *Air Quality Guidelines for Europe, Second Edition*. WHO Regional Publications, European Series. No. 91. Copenhagen, DK.

WHO. 2005. *WHO Air Quality Guidelines Global Update 2005 Report on a Working Group meeting*, Bonn, Germany 18-20 October 2005.

Yang, C. 2006. *Effects of Asian Dust Storm Events on Daily Clinical Visits for Conjunctivitis in Taipei, Taiwan*. Journal of Toxicology and Environmental Health, Part A, 69: 1673-1680.

Yang, C., S. Tsai, C. Chang and S. Ho. 2005a. *Effects of Asian Dust Storm Events on Daily Admissions for Asthma in Taipei, Taiwan*. Inhalation Toxicology 17: 817-821.

Yang, C., Y. Chen, H. Chiu and W. Goggins. 2005b. *Effects of Asian Dust Storm Events on Daily Stroke Admissions in Taipei, Taiwan*. Environmental Research 99: 79-84.

Yang, C., M. Cheng and C. Chen. 2009. *Effects of the Asian Dust Storm Events on Hospital Admissions for Congestive Heart Failure in Taipei, Taiwan*. Journal of Toxicology and Environmental Health. Part A, 72: 324-328.

11 ACRONYMS AND GLOSSARY

11.1 ACRONYMS AND ABBREVIATIONS

Al	Aluminum
As	Arsenic
ATSDR	Agency for Toxic Substances and Disease Registry
BAF	Bioaccumulation factor
BMD	Benchmark dose
C	Carcinogen
CalEPA	California Environmental Protection Agency
CalEPA OEHHA	California Environmental Protection Agency Office of Environmental Health Hazard Assessment
CCME	Canadian Council of Ministers of the Environment
Cd	Cadmium
Co	Cobalt
COC	Chemical of Concern
D_{total}	estimated total dose
e.g.	For example
EDI	Estimated Daily Intake
EIS	Environmental Impact Statement
ESL	Effects screening level
et al.	Group of authors
HEC	Human equivalent concentration
HHRA	Human Health Risk Assessment
HI	Hazard index
HQ	Hazard quotient
HSDB	Hazardous Substances Data Bank
i.e.	That is
IARC	International Agency for Research on Cancer
ILCR	Incremental Lifetime Cancer Risk
LADD	Lifetime-averaged daily dose
LKDFN	Łutselk'e Dene First Nation
LOAEL	Lowest Observed Adverse Effect Level
LSA	Local Study Area
Mn	Manganese

MOU	Memorandum of understanding
MPOI	Maximum Point of Impingement
MRL	Minimal risk level
MVEIRB	Mackenzie Valley Environmental Impact Review Board
N/A	Not applicable
NA	Not applicable
NC	Non-carcinogen
Ni	Nickel
NO_2	Nitrogen dioxide (gas)
NOAEL	No Observed Adverse Effect Level
NWT	Northwest Territories
OEHHA	Office of Environmental Health Hazard Assessment
OMOE	Ontario Ministry of the Environment
PAH	Polycyclic aromatic hydrocarbon
PBPK	Physiologically-based pharmacokinetic
PKC	Processed kimberlite containment
PM	Particulate matter
PM_{10}	Particulate matter with a mean aerodynamic diameter of 10 microns (µm) or smaller
$PM_{2.5}$	Particulate matter with a mean aerodynamic diameter of 2.5 microns (µm) or smaller
REL	Reference exposure level
RfC	Reference Concentration
RfD	Reference Dose
RIVM	Netherlands National Institute of Public Health and the Environment
RSA	Regional Study Area
Sb	Antimony
SF	Slope Factor
TC	Tolerable concentration
TC_{05}	estimate of the concentration in air associated with a 5% increase in tumour incidence or mortality due to tumours
TCA	Tolerable concentration in air
TCDD	tetrachloro-dibenzo-*ρ*-dioxin
TCEQ	Texas Commission on Environmental Quality
TDI	Tolerable Daily Intake
Ti	Titanium
TPH	Total Petroleum Hydrocarbon

TRV	Toxicity Reference Value
TSP	Total suspended particulate
U.S. EPA	United States Environmental Protection Agency
U.S. EPA IRIS	United States Environmental Protection Agency Integrated Risk Information System
UCLM	Upper confidence level of the mean
UF	Uncertainty factor
UR	Unit Risk
V	Vanadium
WHO	World Health Organization

11.2 UNITS OF MEASURE

%	percent
<	less than
>	greater than
µg/m^3	micrograms per cubic metre
µm	micrometres/microns
kg	kilogram
km	kilometre
m	metres
m^3	cubic metres
m^3/y	cubic metres per year
mg	milligram
mg/kg	milligrams per kilogram
mg/kg BW/day	milligrams per kilograms body weight per day
mg/kg-day	milligrams per kilograms per day
mg/m^3	milligrams per cubic metre
Mt	million tonnes
ppm	parts per million

11.3 GLOSSARY

Acute	A stimulus severe enough to rapidly induce an effect; in aquatic toxicity tests, an effect observed in 96 hours or less is typically considered acute. When referring to aquatic toxicology or human health, an acute effect is not always measured in terms of lethality.
Application Case	The Environmental Impact Assessment (EIA) case including the project that is the subject of the application, existing environmental conditions, and existing and approved projects or activities.
Baseline	A surveyed or predicted condition that serves as a reference point to which later surveys are coordinated or correlated.
Baseline Case	The EIA assessment case that includes existing environmental conditions as well as existing and approved projects or activities.
Bioaccumulation	When an organism stores within its body a higher concentration of a substance than is found in the environment. This is not necessarily harmful. For example, freshwater fish must bioaccumulate salt to survive in intertidal waters.
Carcinogen	An agent that is reactive or toxic enough to act directly to cause cancer.
Chemical of Potential Concern	A chemical that is emitted or released into the environment and poses a potential risk of exposure to humans.
Chronic	The development of adverse effects after extended exposure to a given substance. In chronic toxicity tests, the measurement of a chronic effect can be reduced growth, reduced reproduction or other non-lethal effects, in addition to lethality. Chronic should be considered a relative term depending on the life span of the organism.
Concentration	Quantifiable amount of a chemical in environmental media.
Country Foods	Country foods are dietary items from the local region which are used for sustenance. Country food items include: fruit, vegetables, herbs, medicinal plants, fish and game.
Dermal Contact	A person can be exposed to chemicals in soil when soil particles adhere to skin. That is, chemicals in soil may be absorbed through the skin and enter the bloodstream. This is typically a minor exposure pathway that is included in a multi-media risk assessment.
Exposure	The contact reaction between a chemical and a biological system, or organism. Estimated dose of chemical that is received by a particular receptor through a specific exposure pathway (e.g., ingestion, inhalation); expressed as the amount of chemical received, per body weight, per unit time (i.e., mg/kg day).

Exposure Pathway	The route by which a receptor comes into contact with a chemical or physical agent. Examples of exposure pathways include: the ingestion of water, food and soil; the inhalation of air and dust; and dermal absorption.
Groundwater	That part of the subsurface water that occurs beneath the water table, in soils and geologic formations that are fully saturated.
Hydrogen Sulphide	Hydrogen sulphide is a colourless gas with strong odour of rotten eggs. It comes from industrial fugitive emissions by way of petroleum refineries, tank farms for unrefined petroleum products, natural gas plants, petrochemical plants, oil sands plants, sewage treatment facilities, pulp and paper plants using the Kraft pulping process and animal feedlots. Natural sources include sulphur hot springs, sloughs, swamps and lakes.
Incremental Lifetime Cancer Risk (ILCR)	The risk associated with daily exposure to a carcinogenic chemical that is separate from the risk associated with assumed background exposures.
Local Study Area - Maximum Point of Impingement (LSA MPOI)	The LSA MPOI (maximum point of impingement) is the highest ground-level concentration as predicted by the air quality model within this area.
Lowest Observed Adverse Effect Level (LOAEL)	In toxicity testing, it is the lowest concentration at which adverse effects on the measurement end point are observed.
Multi-Media Risk Assessment	Multiple exposure pathways, including air inhalation, water ingestion, food ingestion, incidental soil ingestion, dermal contact and dust inhalation, are evaluated in a multi-media risk assessment. Exposures to the chemicals of concern for each pathway are summed to determine total exposure for each chemical.
Nitrogen Dioxide	One of the component gases of oxides of nitrogen which also includes nitric oxide. In burning natural gas, coal, oil and gasoline, atmospheric nitrogen may combine with molecular oxygen to form nitric oxide, an ingredient in the brown haze observed near large cities. Nitric oxide is converted to nitrogen dioxide in the atmosphere. Cars, trucks, trains and planes are the major source of oxides of nitrogen in Alberta. Other major sources include oil and gas industries and power plants.
Non-Carcinogen	A chemical that does not cause cancer and has a threshold concentration, below which adverse effects are unlikely.
Particulate Matter	A mixture if small particles and liquid droplets, often including a number of chemicals, dust and soil particles.

Polycyclic Aromatic Hydrocarbon (PAH)	A chemical by-product. Aromatics are considered to be highly toxic components of petroleum products. PAHs, many of which are potential carcinogens, are composed of at least two fused benzene rings. Toxicity increases along with molecular size and degree of alkylation of the aromatic nucleus.
Receptor	The person or organism subjected to exposure to chemicals or physical agents.
Reference Concentration (RfC)	For a specific chemical that is conceptually equivalent to an air quality objective, and is expressed in $\mu S/m^3$. It is an exposure limit that is established for chemicals which are locally acting (e.g., irritant chemicals), whose toxicity is dependent solely on the air concentration and not on the total internal dose received by multiple exposure pathways.
Reference Dose	Refers to the safe level or dose of a chemical for which exposure occurs through multiple pathways (i.e., inhalation, ingestion and dermal). It is most commonly expressed in terms of the total intake of the chemical per unit of body weight (e.g., mg/kg BW/day). This term applies only to threshold chemicals.
Regional Study Area (RSA)	Defines the spatial extent related to the cumulative effects resulting from the project and other regional developments.
Risk	The likelihood or probability that the toxic effects associated with a chemical or physical agent will be produced in populations of individuals under their actual conditions of exposure. Risk is usually expressed as the probability of occurrence of an adverse effect, i.e., the expected ratio between the number of individuals that would experience an adverse effect at a given time and the total number of individuals exposed to the factor. Risk is expressed as a fraction without units and takes values from 0 (absolute certainty that there is no risk, which can never be shown) to 1.0, where there is absolute certainty that a risk will occur.
Risk Assessment	Process that evaluates the probability of adverse effects that may occur, or are occurring on target organism(s) as a result of exposure to one or more stressors.
Risk Characterization	The process of evaluating the potential risk to a receptor based on comparison of the estimated exposure to the toxicity reference value.
Runoff	The portion of water from rain and snow that flows over land to streams, ponds or other surface water bodies. It is the portion of water from precipitation that does not infiltrate into the ground, or evaporate.
Slope Factor	An upper-bound estimate of risk per increment of dose calculated using linear extrapolation for carcinogens.

Sulphur Dioxide	Sulphur dioxide is a colourless gas with a pungent odour. In Alberta, natural gas processing plants are responsible for close to half of the emissions of this gas. Oil sands facilities and power plants are also major sources. Others include gas plant flares, oil refineries, pulp and paper mills and fertilizer plants.
Total Petroleum Hydrocarbons	Groups of hydrocarbon chemicals derived from a petroleum source.
Toxicant	A toxicant is a chemical compound that has an effect on organisms.
Toxicity	The inherent potential or capacity of a material to cause adverse effects in a living organism.
Toxicity Assessment	The process of determining the amount (concentration or dose) of a chemical to which a receptor may be exposed without the development of adverse effects.
Toxicity Reference Value (TRV)	For a non-carcinogenic chemical, the maximum acceptable dose (per unit body weight and unit of time) of a chemical to which a specified receptor can be exposed, without the development of adverse effects. For a carcinogenic chemical, the maximum acceptable dose of a chemical to which a receptor can be exposed, assuming a specified risk (e.g., 1 in 100,000). May be expressed as a Reference Dose (RfD) for non-carcinogenic (threshold-response) chemicals or as a Risk Specific Dose (RsD) for carcinogenic (non-threshold response) chemicals. Also referred to as exposure limit.
Traditional Use Plants	Plants used by First Nations people of a region as part of their traditional lifestyle for food, ceremonial, medicinal and other purposes.
Wildlife	Under the *Species at Risk Act*, wildlife is defined as a species, subspecies, variety or geographically or genetically distinct population of animal, plant or other organism, other than a bacterium or virus that is wild by nature and is native to Canada or has extended its range into Canada without human intervention and has been present in Canada for at least 50 years.

APPENDIX I

AIR QUALITY ASSESSMENT (SCREENING CRITERIA AND TOXICITY REFERENCE VALUES)

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I INTRODUCTION

This appendix provides the chemical screening for acute and chronic air exposure, as well as the toxicity reference values used for the air assessment.

I.1 CHEMICAL SCREENING FOR THE ACUTE AIR QUALITY RISK ASSESSMENT

For each chemical, peak air concentrations representing maximum 1-hour and 24-hour concentrations were predicted for receptor locations throughout the region and compared to acute health-based thresholds from the following agencies:

- Northwest Territories Guideline for Ambient Air Quality Standards (GNWT 2011, internet site);
- Agency of Toxic Substances and Disease Registry (ATSDR 2012, internet site);
- Ontario Ministry of Environment and Energy (OMoE 2012a, internet site; OMoE 2012b, internet site);
- California Environmental Protection Agency Office of Environmental Health Hazard Assessment (CalEPA OEHHA 2012, internet site);
- Texas Commission on Environmental Quality (TCEQ 2012, internet site); and
- World Health Organization (WHO 2000, 2006).

The available 1-hour and 24-hour acute health-based thresholds and the basis of these thresholds are presented in Table I-1 and Table I-2, respectively.

The NWT air quality standards (GNWT 2011, internet site) were considered the priority source for selecting values for screening purposes. In the absence of a NWT value, the most conservative of the available health-based screening levels for a given chemical was used. Priority was given to screening levels that were health-based and had supporting documentation.

Predicted 1-hour and 24-hour peak concentrations in air from the Application Case were compared to the most conservative of the 1-hour and 24-hour acute thresholds, respectively (Table I-3 and Table I-4). A parameter was retained for further evaluation if the predicted peak concentration (i.e., maximum from all receptor locations) was greater than the threshold. A parameter that was retained for further assessment was classified as a chemical of potential concern (COPC) and was evaluated for all receptor locations.

Table I-1 Acute Inhalation 1-Hour Health-Based Thresholds

Parameter	Air Screening Levels and Guidelines [µg/m^3]									Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2]			OMoE[3]	ATSDR[4]	California OEHHA[5]	WHO[6,7]	TCEQ[8]	
	Standard	Desirable	Acceptable	Tolerable						
Acid Gases										
Sulfur dioxide (SO_2)	450	450	900	-	690	26 (0.01 ppm) [a]	660	500 [a]	-	NWT: Adopted from CCME NAAQO; OMoE: Threshold based on health and vegetation endpoints (supporting documentation not available); Cal OEHHA: Threshold based on impairment of airway function (bronchoconstriction) especially in asthmatics; ATSDR: Respiratory effects on exercising asthmatics, uncertainty factors of 3 for use of LOAEL and 3 for human variability. The MRL of 0.01 ppm was converted to µg/m^3 using MW = 64.07 g/mol. MRL for a 10 minute averaging time; WHO: Threshold based on changes in pulmonary function and respiratory symptoms in exercising asthmatics. Threshold for a 10 minute averaging time.
Nitrogen dioxide (NO_2)	400	-	400	1,000	400	-	470	200	-	NWT: Adopted from CCME NAAQO; CCMENAAQO: Supporting documentation not available; OMoE: Threshold based on health endpoint (supporting document not available); Cal OEHHA: Threshold based on increased airway rectivity in asthmatics; WHO: Threshold based on studies of bronchial responsiveness among asthmatics.
Carbon monoxide (CO)	15,000	15,000	35,000	-	36,200	-	23,000	30,000	-	NWT: Adopted from CCME NAAQO; CCME NAAQO: Supporting documentation not available; Cal OEHHA: Threshold based on effects of angina in people with known cardiovascular diseases that are exercising heavily; WHO: Threshold of 30 mg/m^3 based on a maximum concentration of 2.5% carboxyhaemoglobin (COHb - carbon monoxide bound to blood haemoglobin) to protect nonsmoking, middle-aged and elderly population groups with coronary artery disease, and to protect fetuses of nonsmoking pregnant women (1 hour).
Particulate Matter										
$PM_{2.5}$	-	-	-	-	-	-	-	-	-	-
PM_{10}	-	-	-	-	-	-	-	-	-	-
Total Suspended Particulate (TSP)	-	-	-	-	-	-	-	-	-	-
Volatile Organic Compounds										
1,1,1-Trichloroethane	-	-	-	-	-	-	-	-	2,800	TCEQ: Screening level based on impaired psychomotor performance in adult males for 1 hour. The LOAEL was 954 mg/m^3 (175 ppm) and an uncertainty factor of 100 (10 for intraspecies differences and 10 for LOAEL to NOAEL extrapolation) was applied for a value of 9.54 mg/m^3 (HQ=1).
1,3-Butadiene	-	-	-	-	-	-	-	-	100 to 510	TCEQ: Odour endpoint for 1,3-butadiene (510 µg/m^3), health endpoint for 1,3-butadiene homopolymers (100 µg/m^3) (supporting documentation is not available).
Acetaldehyde	-	-	-	-	500 [b]	-	470	-	-	OMoE: Threshold based on health (irritation and tissue damage in the upper respiratory tract from prolonged exposure) endpoint (supporting documentation not available), half-hour averaging time; Cal OEHHA: Threshold based on sensory irritation, bronchi, eyes, nose and throat. Sixty-one adult asthmatic human volunteers were exposed to acetylaldehyde via inhalation by nebulizer for 2-4 minutes experienced bronchoconstriction, PC_{20} > 20% drop in forced expiratory volume in one secnd (FEV_1). LOAEL = 142 mg/m^3. Uncertainties applied include 10 for LOAEL, 1 for interspecies, 1 for intraspecies, and 30 for asthma exacerbation in children, hyper-responsiveness to methacholine for a total of 300.

Table I-1 Acute Inhalation 1-Hour Health-Based Thresholds (continued)

Parameter	Air Screening Levels and Guidelines [µg/m^3]									Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2]			OMoE[3]	ATSDR[4]	California OEHHA[5]	WHO[6,7]	TCEQ[8]	
	Standard	Desirable	Acceptable	Tolerable						
Acetone	-	-	-	-	-	61760 (26 ppm) [c]	-	-	-	ATSDR: Threshold based on neurobehaviour effects in humans. The threshold is based on a LOAEL value of 237 ppm for 4 hours. An uncertainty factor of 9 was used.
Acrolein	-	-	-	-	4.5	6.88 (0.003 ppm)	2.5	-	-	OMoE: Threshold based on irritation following an acute exposure (1 hour) to acrolein. A LOAEL of 137 µg/m^3 and an uncertainty factor of 30 (3 for LOAEL to NOAEL extrapolation and 10 for interspecies variability) was used; ATSDR: Acute inhalation MRL was derived based on a LOAEL of 0.3 ppm for nasal and throat irritation and decreased respiratory rate in volunteers exposed for 1 h. An uncertainty factor of 100 was applied (10 for using a LOAEL and 10 for human variability). The MRL of 0.003 ppm was converted to µg/m^3 using MW = 56.06 g/mol; Cal OEHHA: Threshold based on eye irritation in humans in two studies. A LOAEL of 138 mg/m^3 (0.06 ppm) and 160 mg/m^3 was estimated and an uncertainty factor of 60 (6 for using LOAEL for mild effects in the absense of a NOAEL and 10 for greater sensitivity in children with asthma) was applied to both LOAELs, the average of the two were taken.
Aldehydes (surrogate: acetaldehyde)	-	-	-	-	500 [b]	-	470	-	-	See acetaldehyde
Benzene	-	-	-	-	-	-	1,300	-	-	Cal OEHHA: Threshold based on reproductive and developmental toxicity.
C2 to C8 aliphatics (surrogate: cyclohexane)	-	-	-	-	-	-	-	-	3,400	TCEQ: Screening level based on health effects (interim, supporting documentation not available).
C9 to C16 aliphatics (surrogate: decane)	-	-	-	-	60,000	-	-	-	-	OMoE: Threshold based on health and odour effects for 1 hour (supporting documentation not available).
C16+ aliphatics (surrogate: decane)	-	-	-	-	60,000	-	-	-	-	OMoE: Threshold based on health and odour effects for 1 hour (supporting documentation not available).
C6 to C8 aromatics (surrogate: toluene)	-	-	-	-	-	-	37,000	1000	-	See toluene
C9 to C16 aromatics (surrogate ethylbenzene)	-	-	-	-	1,900 [a]	-	-	-	-	OMoE: Threshold based on odour and for a 10 minute averaging time (supporting documentation not available).
Ethylbenzene	-	-	-	-	1,900 [a]	-	-	-	-	OMoE: Threshold based on odour and for a 10 minute averaging time (supporting documentation not available).
Formaldehyde	-	-	-	-	-	49.1 (0.04 ppm)	55	100 [b]	-	Cal OEHHA: Based on mild and moderate eye irritation. Nineteen (19) non-asthmatic, non-smoking humans exposed to 0.5 to 3.0 ppm (single exposure per concentration) for 3 hrs experienced mild and moderate eye irritation. LOAEL = 1 ppm and NOAEL = 0.5 ppm. Uncertainty factors of 1 for interspecies, 1 for intraspecies, and 10 for asthma exacerbation in children were applied; ATSDR: Threshold based on LOAEL of 0.4 ppm (nasal and eye irritation in humans following 2-hour exposure); uncertainty factors: 3 for use of LOAEL, 3 for human variability. The MRL of 0.04 ppm was converted using MW = 30.03 g/mol; WHO: Threshold based on throat and nose irritation, and represents an exposure at which there is a negligible risk of upper repiratory tract cancer in humans.
Methyl ethyl ketone	-	-	-	-	-	-	13,000	-	-	Cal OEHHA: Threshold based on respiratory system and eye effects in humans
Toluene	-	-	-	-	-	-	37,000	1000	-	Cal OEHHA: Threshold based on nervous system, eyes, respiratory system, and reproductive/developmental effects. Sixteen (16) young, health males exposed to 98 ppm for 6 hrs experienced impaired reaction time and symptoms of headache, dizziness, a feeling of intoxication and slight eye and nose irritation. LOAEL = 100 ppm and NOAEL = 40 ppm. Uncertainty factors were applied for LOAEL (1), interspecies (1), and intraspecies (10) for a total of 10; WHO: odour detection threshold; 30 minute averaging time.

Table I-1 Acute Inhalation 1-Hour Health-Based Thresholds (continued)

Parameter	Air Screening Levels and Guidelines [µg/m^3]									Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2]			OMoE[3]	ATSDR[4]	California OEHHA[5]	WHO[6,7]	TCEQ[8]	
	Standard	Desirable	Acceptable	Tolerable						
Trimethylbenzenes	-	-	-	-	-	-	-	-	1250	TCEQ: Screening level based on health effects (interim, supporting documentation not available), as the solvent Aromatic 100.
Xylene (total)	-	-	-	-	3,000 [a]	8680 (2 ppm)	22,000	-	-	OMoE: Threshold based on odour and a 10 minute averaging time (supporting documentation not available); ATSDR:Threshold based on a minimal LOAEL of 50 ppm (217 µg/m^3) for mild objective and subjective respiratory effects and subjective neurological effects in subjects exposed to *m*-xylene vapour for 2 hours (uncertainty factor of 30). The MRL of 2 ppm was converted to µg/m^3 using MW = 106.16 g/mol; Cal OEHHA: Threshold based on nervous system, respiratory system, and eye effects in humans.
Polycyclic Aromatic Hydrocarbons										
1-methylnaphthalene	-	-	-	-	-	-	-	-	30	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
1-methylphenanthrene (surrogate: phenanthrene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-methylanthracene (surrogate: anthracene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-methylfluorene (surrogate: fluorene)	-	-	-	-	-	-	-	-	10	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-methylnaphthalene	-	-	-	-	-	-	-	-	30	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-methylphenanthrene (surrogate: phenanthrene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-methylpyrene (surrogate: pyrene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
3-Methyldibenzothiophene (surrogate: dibenzothiophene)	-	-	-	-	-	-	-	-	25	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
3-Methylphenanthrene (surrogate: phenanthrene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
4 + 9 Methylphenanthrene (surrogate: dibenzothiophene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health (supporting documentation not available); as 2,5-dimethylphenanthrene and generic PAHs.
4-Methyldibenzothiophene (surrogate: dibenzothiophene)	-	-	-	-	-	-	-	-	25	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Acenaphthene	-	-	-	-	-	-	-	-	1	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Acenaphthylene	-	-	-	-	-	-	-	-	1	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Acephenanthrylene (surrogate: benzo(k)fluoranthene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Anthracene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benz(a)anthracene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health (interim, supporting documentation not available); as anthracene.
Benzo(a)fluorene (surrogate: 2,3-benzo(b)fluorene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).

Table I-1 Acute Inhalation 1-Hour Health-Based Thresholds (continued)

Parameter	Air Screening Levels and Guidelines [µg/m^3]									Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2]			OMoE[3]	ATSDR[4]	California OEHHA[5]	WHO[6,7]	TCEQ[8]	
	Standard	Desirable	Acceptable	Tolerable						
Benzo(a)pyrene	-	-	-	-	0.00015	-	-	-	-	A half-hour average standard of 0.00015 µg/m3 for B[a]P, (as a surrogate of total PAHs), based on carcinogenicity associated with exposure to PAH compounds.
Benzo(b)fluoranthene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benzo(e)pyrene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benzo(g,h,i)fluoranthene (surrogate: benzo(g,h,i)perylene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benzo(g,h,i)perylene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benzo(k)fluoranthene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Chrysene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Coronene (surrogate: benzo(g,h,i)perylene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Cyclopenta(c,d)pyrene (surrogate: benzo(a)pyrene)	-	-	-	-	-	-	-	-	0.03	TCEQ: Threshold based on health (under review, supporting documentation not available).
Dibenz(a,h)anthracene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available); as anthracene.
Dibenzothiophene	-	-	-	-	-	-	-	-	25	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Fluoranthene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Fluorene	-	-	-	-	-	-	-	-	10	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Indeno(1,2,3-cd)fluoranthene (surrogate: fluoranthene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Indeno(1,2,3-cd)pyrene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Indeno(1,2,3-W)pyrene (surrogate: indeno(1,2,3-cd)pyrene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Naphthalene	-	-	-	-	50 [a]	-	-	-	440	OMoE: Threshold based on odour and a 10 minute averaging time (supporting documentation not available)[5]; TCEQ: Odour based endpoint, guideline under review
Nitropyrene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health (supporting documentation not available), half-hour averaging time; as 1-nitropyrene.
Perylene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Phenanthrene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).

Table I-1 Acute Inhalation 1-Hour Health-Based Thresholds (continued)

Parameter	Air Screening Levels and Guidelines [µg/m^3]									Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2]			OMoE[3]	ATSDR[4]	California OEHHA[5]	WHO[6,7]	TCEQ[8]	
	Standard	Desirable	Acceptable	Tolerable						
Picene (surrogate: dibenzo(a,h)anthracene)	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health (supporting documentation not available); as anthracene.
Pyrene	-	-	-	-	-	-	-	-	0.5	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Metals and Inorganics										
Aluminum	-	-	-	-	-	-	-	-	50	TCEQ: Threshold based on health (interim, supporting documentation not available); for aluminum as a metal and insoluble.
Antimony	-	-	-	-	-	-	-	-	5	TCEQ: Threshold based on health (interim, supporting documentation not available)
Arsenic	-	-	-	-	-	-	0.2	-	-	Cal OEHHA: Threshold based on decreased fetal weight in mice following maternal inhalation of As_2O_3 during gestation period. A LOAEL of 0.26 mg/m^3 and an uncertainty factor of 1000 (10 for no NOAEL value, 10 for interspecies differences between mice and humans, 10 for human interindividual differences) were used; TCEQ: Threshold based on health (interim, supporting documentation not available), as inorganic arsenic.
Barium	-	-	-	-	-	-	-	-	5	TCEQ: Threshold based on health (interim, supporting documentation not available), for barium and compounds.
Beryllium	-	-	-	-	-	-	-	-	0.02	TCEQ: Threshold based on health (interim, supporting documentation not available); for berylium as a particulate.
Bismuth	-	-	-	-	-	-	-	-	50	TCEQ: Threshold based on health (interim, supporting documentation not available); for bismuth and compounds
Boron	-	-	-	-	-	-	-	-	50	TCEQ: Threshold based on health (interim, supporting documentation not available)
Cadmium	-	-	-	-	-	-	-	-	0.1	TCEQ: Threshold based on health (interim, supporting documentation not available); for cadmium and compounds
Calcium	-	-	-	-	-	-	-	-	20	TCEQ: Threshold based on health (interim, supporting documentation not available); for calcium oxide.
Chromium (as Cr[III])	-	-	-	-	-	-	-	-	3.6	TCEQ: Threshold (for Cr(III) based on increased acid phosphotase activity in lavage fluid and increased acid phosphotase and β-glucuronidase activity in lung tissue (precursors to adverse effects) in hamsters. The NOAEL was 77 mg/m^3 and adjusted to 10.82 mg/m^3 for a human equivalent concentration. An uncertainty factor of 300 (3 for interspecies differences, 10 for intraspcies differences and 10 for incomplete database) was used for a threshold of 36 µg/m^3 (HQ=1).
Cobalt	-	-	-	-	-	-	-	-	0.2	TCEQ: Threshold based on health (interim, supporting documentation not available); for cobalt and inorganic compounds.
Copper	-	-	-	-	-	-	100	-	-	Cal OEHHA: Threshold based on respiratory system effects in humans.
Iron	-	-	-	-	-	-	-	-	10	TCEQ: Threshold based on health (interim, supporting documentation not available
Lead	-	-	-	-	-	-	-	-	-	-
Lithium	-	-	-	-	-	-	-	-	10	TCEQ: Threshold based on health (interim, supporting documentation not available), as LiOH, LiO and lithium silicate as Li.
Magnesium	-	-	-	-	-	-	-	-	50	TCEQ: Threshold based on health (interim, supporting documentation not available), as Mg, except magnesium chromate.

Table I-1 Acute Inhalation 1-Hour Health-Based Thresholds (continued)

Parameter	Air Screening Levels and Guidelines [µg/m^3]									Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2]			OMoE[3]	ATSDR[4]	California OEHHA[5]	WHO[6,7]	TCEQ[8]	
	Standard	Desirable	Acceptable	Tolerable						
Manganese	-	-	-	-	-	-	-	-	2	TCEQ: Threshold based on health (interim, supporting documentation not available), as dust and inorganic compounds.
Mercury	-	-	-	-	-	-	0.6	-	-	Cal OEHHA: Threshold based on CNS disturbances in rat offspring. Maternal rats were exposed to metallic Hg vapour (1.8 mg/m^3) for 3 hours/day during gestation. The offspring displayed significant dose-dependent deficits in behaviour 3-7 months after birth compared to controls. An uncertainty factor of 3000 (10 for using LOAEL for moderate to severe effects in the absence of a NOAEL, 3 for interspecies toxicokinetic differences, 10 for interspecies toxicodynamic differences, 3 for individual variability, and 3 for intraspecies differences in age differences) were used.
Molybdenum	-	-	-	-	-	-	-	-	30	TCEQ: Threshold based on health (interim, supporting documentation not available)
Nickel	-	-	-	-	-	-	0.2	-	-	Cal OEHHA: Threshold based on immune system effects in mice, as nickel and compounds.
Phosphorus	-	-	-	-	-	-	-	-	-	-
Potassium	-	-	-	-	-	-	-	-	20	TCEQ: Threshold based on health (interim, supporting documentation not available)
Selenium	-	-	-	-	-	-	-	-	2	TCEQ: Threshold based on health (interim, supporting documentation not available), as selenium and compounds.
Silver	-	-	-	-	-	-	-	-	0.1	TCEQ: Threshold based on health (interim, supporting documentation not available), as silver and compounds.
Sodium	-	-	-	-	-	-	8	-	-	Cal OEHHA: Threshold based on respiratory system, eyes and skin effects in humans, as NaOH.
Strontium	-	-	-	-	-	-	-	-	20	TCEQ: Threshold based on health (interim, supporting documentation not available), as strontium and compounds.
Thallium	-	-	-	-	-	-	-	-	1	TCEQ: Threshold based on health (interim, supporting documentation not available), as thallium and compounds.
Tin	-	-	-	-	-	-	-	-	20	TCEQ: Threshold based on health (interim, supporting documentation not available), as tin oxide and inorganic compounds.
Titanium	-	-	-	-	-	-	-	-	50	TCEQ: Threshold based on health (interim, supporting documentation not available).
Tungsten	-	-	-	-	-	-	-	-	50	TCEQ: Threshold based on health (interim, supporting documentation not available); for insoluble tungsten.
Uranium	-	-	-	-	-	-	-	-	2	TCEQ: Threshold based on health (interim, supporting documentation not available); for insoluble uranium.
Vanadium	-	-	-	-	-	-	30	-	-	OMoE: Threshold based on health endpoint (supporting documentation not available); Cal OEHHA: Threshold based on effects on the respiratory system and eyes in humans, as vanadium pentoxide.
Zinc	-	-	-	-	-	-	-	-	20	TCEQ: Threshold based on health (interim, supporting documentation not available), for zinc and compounds.
Zirconium	-	-	-	-	-	-	-	-	50	TCEQ: Threshold based on health (interim, supporting documentation not available), for zirconium and compounds.

Table I-1 Acute Inhalation 1-Hour Health-Based Thresholds (continued)

Parameter	Air Screening Levels and Guidelines [µg/m^3]									Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2]			OMoE[3]	ATSDR[4]	California OEHHA[5]	WHO[6,7]	TCEQ[8]	
	Standard	Desirable	Acceptable	Tolerable						
Dioxins/Furans										
Total Dioxins/Furans	-	-	-	-	3E-07 TEQ [9]	-	-	-	-	A conversion factor of 3 was applied to the effects-based 24-hour average AAQC to set a half-hour average air standard of 0.0000003 µg WHO 2006-TEQ/m^3 for total dioxins, furans, and dioxinlike PCBs, based on the developmental effects associated with exposure to these compounds.

Notes: All values are in µg/m^3, unless otherwise noted.
- = Value not available.
Shaded acute thresholds were used in the risk assessment.

*Value converted from parts-per-million (ppm) to micrograms per meter cubed (µg/m^3) using the molecular weight (ML) of the specified compound, in the following formula:Y mg/m^3=(X ppm)(molecular weight)/24.45, (assumptions: 25 ºC and 1 atm).
a. 10-minute averaging time.

b. Half-hour averaging time.

c. 4-hour averaging time.

1. Northwest Territories Department of Environment and Natural Resources – Ambient Air Quality Standards (NWT 2011)
2. Canadian Council of Ministers of the Environment (CCME 1999)
3. Ontario Ministry of the Environment (OMoE 2012a)
4. Agency of Toxic Substances and Disease Registry (ATSDR), Minimum Risk Levels (MRLs) (ATSDR 2012)
5. California Office of Environmental Health Hazard Assessment (CalEPA OEHHA 2012)
6. World Health Organization (WHO), WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide (WHO 2006)
7. WHO Air Quality Guidelines for Europe, Second Edition. (WHO 2000).
8. Texas Commission on Environmental Quality (TCEQ), Effects Screening Levels (TCEQ 2012). These values were only provided when values for no other jurisdictions were available. Thresholds based on an HQ = 0.3 unless otherwise stated.
9. Ontario Air Standards for Dioxins, Furans and Dioxin-like PCBs (OMoE 2011).
HQ - Hazard Quotient, LOAEL - Lowest Observed Adverse Effects Level, NOAEL - No Observed Adverse Effects Level, NAAQO - Canadian National Ambient Air Quality Objectives, MRL - Minimal Risk Level, MW - Molecular Weight (grams per moles), REL - Reference Exposure Level, PAH - Polycyclic Aromatic Hydrocarbons, PM - Particulate Matter, ppm - parts per million .

Table I-2 Acute Inhalation 24-Hour Health-Based Thresholds

Parameter	Air Screening Levels and Guidelines [µg/m^3]							Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2,3]			OMoE[4,5]	ATSDR[6]	WHO[7,8]	
	Standard	Desirable	Acceptable	Tolerable				
Acid Gases								
Sulfur dioxide (SO_2)	150	150	300	800	275	-	20	NWT: Adopted from CCMENAAQO; CCME NAAQO: Supporting documentation not available; OMoE: Threshold based on health and vegetation endpoints (supporting document not available); WHO: Threshold based childhood respiratory disease and all-age mortality.
Nitrogen dioxide (NO_2)	200	-	200	300	200	-	-	NWT: Adopted from CCME NAAQO; CCMENAAQO: Supporting documentation not available.
Carbon monoxide (CO)	6,000	6,000	15,000	20,000	15,700	-	-	NWT: Adopted from CCME NAAQO. Standard is for 8-hour exposure; CCME NAAQO: Guideline for 8-hour exposure, supporting documentation not available; OMoE: Guideline for 8-hour exposure, supporting documentation not available.
Particulate Matter								
$PM_{2.5}$	30	30	-	-	30	-	25	NWT: Adopted from Canada-wide Standard (CWS); CCME and OMoE thresholds based on Canada-wide Standards, intended for the protection of respiratory effects; WHO: Based on relationship between 24-hr and annual PM levels.
PM_{10}	-	25			50	-	50	CCME: reference level, above which there are demonstrated effects on human health and/or the environment; WHO: air quality guideline reflects the relationship between the distributions of 24-hour means (and its 99th percentile) and annual average concentrations. OMoE: interim ambient air quality criterion.
Total Suspended Particulate (TSP)	120	-	120	400	120	-	-	OMoE: Threshold based on visibility, for particulate less than 44 µm diameter; NWT: Adopted from CCME NAAQO.
Volatile Organic Compounds								
1,1,1,-trichloroethane	-	-	-	-	115000 (2 ppm)	10,900 (2 ppm)	-	OMoE: Threshold based on health endpoint (supporting document not available); ATSDR: Threshold based on a LOAEL of 175 ppm for reduced performance of psychomotor tests in human volunteers (uncertainty factor of 100). The MRL of 2 ppm was converted to µg/m^3 using MW = 133.40 g/mol.
1,3-Butadiene	-	-	-	-	10	220 (0.1 ppm)	-	OMoE: Threshold based on carcinogenicity associated with exposure to this compound. Threshold based on a unit risk estimate from the State of Texas that was based on cancer studies to occupationally-exposed workers. The inhalation unit risk estimates from Texas is 5x10^{-7}µg/m^3, giving an annual threshold of 2 µg/m^3. A conversion factor of 5 (based on empirical monitoring data, ratios of concentrations observed for different averaging times and meterological considerations) was used for the 24-h threshold; ATSDR: An acute-duration inhalation MRL of 0.1 ppm was derived using the LOAEL of 40 ppm for reduced male fetal body weight gain from exposed pregnant mice. The LOAEL of 40 ppm was adjusted for intermittent exposure (6 hours/day) resulting in a duration-adjusted LOAEL of 10 ppm. A $LOAEL_{HEC}$ (human equivalent concentration) of 10 ppm was derived, which was divided by an uncertainty factor of 90 (3 for use of a minimally adverse effect, 3 for extrapolation from animals to humans, and 10 for human variability). The MRL was converted from 0.1 ppm to 220 µg/m^3 using MW = 64.07 g/mol.
Acetaldehyde	-	-	-	-	500	-	-	OMoE: Threshold based on health (to prevent irritation & possible tissue damage in upper respiratory tract resulting from prolonged exposure) endpoint (supporting document available, but details not provided).
Acetone	-	-	-	-	11,880	-	-	OMoE: Threshold based on health (irritation & neurological) endpoint (supporting document available). A LOAEL of 594 mg/m^3 (based on a number of human studies) was used as the LOAEL, an uncertainty factor of 50 (10 for sensitive individuals in the population, 5 for extrapolating from LOAEL to a NOAEL and to account for the lack of toxicological data characterizing the dose-response & chronic effects of acetone) was applied.
Acrolein	-	-	-	-	0.40	-	-	OMoE: Threshold based on olfactory epithelial pathology in a rat study. The NOAEL was 0.6 ppm and the estimated HEC NOAEL was 11 µg/m^3. An uncertainty factor of 30 (3 for interspecies extrapolation and 10 to protect sensitive individuals in humans) was used.
Aldehydes (surrogate: acetaldehyde)	-	-	-	-	500	-	-	OMoE: Threshold based on health (avoid irritation & possible tissue damage in upper respiratory tract resulting from prolonged exposure) endpoint (supporting document available, but details not provided).
Benzene	-	-	-	-	2.3	28.75 (0.009 ppm)	-	OMoE: Threshold based on methods used by U.S. EPA and the European Union (EU) that extrapolated occupational exposure concentrations to annual ambient air exposures based on mortality due to acute myeloid leukemia. The 24-h threshold was calculated from the annual threshold (0.45 µg/m^3) based on a conversion factor of 5 (based on empirical monitoring data, ratios of concentrations observed for different averaging times, and meterological considerations); ATSDR: Threshold from LOAEL of 10.2 ppm (reduced lymphocyte proliferation following mitogen stimulation in mice, adjusted for continuous exposure (10.2 ppm x 6hr/24/hr = $LOAEL_{ADJ}$ of 2.55 ppm; uncertainty factor of 300 applied: 10 for use of a LOAEL, 3 for extrapolation from animals to humans, 10 for human variability; 2.55/300 = 0.009 ppm x molecular weight of 78.11/24.45 = 0.02875 mg/m^3).
C2 to C8 aliphatics (surrogate: cyclohexane)	-	-	-	-	6,100	-	-	OMoE: Threshold based on health endpoint (supporting documentation not available)
C9 to C16 aliphatics (surrogate: decane)	-	-	-	-	6,100	-	-	No 24-hr thresholds for the C9-C16 aliphatic surrogate, decane, were available. The 24-hr threshold for C2-C8 aliphatics, cyclohexane, was conservatively used in the risk assessment.
C16+ aliphatics (surrogate: decane)	-	-	-	-	6,100	-	-	No 24-hr thresholds were available for the C16+ aliphatics. The 24-hr threshold for C2-C8 aliphatics, which was based on cyclohexane, was conservatively used in the risk assessment.

Table I-2 Acute Inhalation 24-Hour Health-Based Thresholds (continued)

Parameter	Air Screening Levels and Guidelines [µg/m^3]							Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2,3]			OMoE[4,5]	ATSDR[6]	WHO[7,8]	
	Standard	Desirable	Acceptable	Tolerable				
C6 to C8 aromatics (surrogate: toluene)	-	-	-	-	2,000	3,770 (1 ppm)	-	OMoE: Odour-based threshold (supporting documentation not available); ATSDR: Threshold from NOAEL of 40 ppm (neurological effects on 16 healthy young men exposed for 6 hours/day on 4 consecutive days; exposure concentration adjusted from intermittent to continuous exposure (40 ppm x 5d/7d x 8hr/24hr); uncertainty factors: 10 for human variability). The MRL of 1 ppm was converted to µg/m^3 using MW = 94.14 g/mol.
C9 to C16 aromatics (surrogate ethylbenzene)	-	-	-	-	1,000	21,710 (5 ppm)	-	OMoE: Threshold based on health endpoint (supporting documentation not available); ATSDR: An acute-duration inhalation MRL was identified using benchmark dose (BMD) analysis of auditory threshold data. The lowest human equivalent concentration (HEC) of 154.26 ppm was selected as the point of departure. This HEC was divided by an uncertainty factor of 30 (3 for extrapolation from animals to humans with dosimetric adjustments and 10 for human variability) resulting in an acute-duration inhalation MRL of 5 ppm, which was converted to µg/m^3 using MW = 106.17 g/mol.
Ethylbenzene	-	-	-	-	1,000	2,1710 (5 ppm)	-	OMoE: Threshold based on health endpoint (supporting documentation not available); ATSDR: An acute-duration inhalation MRL was identified using benchmark dose (BMD) analysis of auditory threshold data. The lowest human equivalent concentration (HEC) of 154.26 ppm was selected as the point of departure. This HEC was divided by an uncertainty factor of 30 (3 for extrapolation from animals to humans with dosimetric adjustments and 10 for human variability) resulting in an acute-duration inhalation MRL of 5 ppm, which was converted to µg/m^3 using MW = 106.17 g/mol.
Formaldehyde	-	-	-	-	65	-	-	OMoE: Threshold based on health endpoint and odour (supporting document not available).
Methyl ethyl ketone	-	-	-	-	1,000	-	-	OMoE: Threshold based on health endpoint (supporting document not available).
Toluene	-	-	-	-	2000	3,770 (1 ppm)	-	OMoE: Odour-based threshold (supporting documentation not available); ATSDR: Threshold from NOAEL of 40 ppm (Neurological effects on 16 healthy young men exposed for 6 hours/day on 4 consecutive days; exposure concentration adjusted from intermittent to continuous exposure (40 ppm x 5d/7d x 8hr/24hr); Uncertainty factors: 10 for human variability). The MRL of 1 ppm was converted to µg/m^3 using MW = 94.14 g/mol.
Trimethylbenzenes	-	-	-	-	220	-	-	OMoE: Threshold based on CNS effects. A 4-week inhalation rat exposure study concluded that exposure to any of the trimethylbenzene isomers to concentrations as low as 492 mg/m^3 produced long-term CNS effects. The LOAEL is adjusted with an uncertainty factor of 300 (3 for extrapolating from LOAEL to NOAEL, 3 for interspecies extrapolation, 10 for intraspecies variability, and 3 for subchronic to chronic extrapolation). For trimethylbenzene as an individual isomer or as a mixture.
Xylenes (total)	-	-	-	-	730		-	OMoE: Threshold based on health endpoint (supporting document not available).
Polycyclic Aromatic Hydrocarbons								
1-methylnaphthalene	-	-	-	-	12	-	-	OMoE: Jurisdictional Screening Level; 30 minute averaging time.
2-methylanthracene (surrogate: anthracene)	-	-	-	-	0.2	-	-	OMoE: Jurisdictional Screening Level.
2-methylnaphthalene	-	-	-	-	10	-	-	OMoE: Jurisdictional Screening Level.
2-methylpyrene (surrogate: pyrene)	-	-	-	-	0.2	-	-	OMoE: Jurisdictional Screening Level.
Acenaphthylene	-	-	-	-	3.5	-	-	OMoE: Jurisdictional Screening Level.
Anthracene	-	-	-	-	0.2	-	-	OMoE: Jurisdictional Screening Level.
Benzo(a)pyrene	-	-	-	-	0.0001	-	-	A chronic threshold was conservatively used in the absence of an appropriate 24-hour threshold. OMoE: Threshold based on carcinogenic potential endpoint that is based on WHO's evaluation of coke-oven worker epidemiological studies that derived an inhalation unit risk (IUR) value of 0.000087 ng/m3 (BaP as a surrogate for total PAHs) equivalent to 0.01 ng/m3 of BaP.
Benzo(g,h,i)fluoranthene (surrogate: benzo(g,h,i)perylene)	-	-	-	-	1.2	-	-	OMoE: Jurisdictional Screening Level.
Benzo(g,h,i)perylene	-	-	-	-	1.2	-	-	OMoE: Jurisdictional Screening Level.
Coronene (surrogate: benzo(g,h,i)perylene)	-	-	-	-	1.2	-	-	OMoE: Jurisdictional Screening Level.
Fluoranthene	-	-	-	-	140	-	-	OMoE: Jurisdictional Screening Level.
Indeno(1,2,3-cd)fluoranthene (surrogate: fluoranthene)	-	-	-	-	140	-	-	OMoE: Jurisdictional Screening Level.
Naphthalene	-	-	-	-	22.5	-	-	OMoE: Threshold based on health endpoint (supporting document not available).
Pyrene	-	-	-	-	0.2	-	-	OMoE: Jurisdictional Screening Level.
Metals and Inorganics								
aluminum	-	-	-	-	120	-	-	OMoE: Threshold based on particulate (supporting document not available); for the metal parameter/oxide.
antimony	-	-	-	-	25	-	-	OMoE: Threshold based on health endpoint (supporting document not available).
arsenic	-	-	-	-	0.3	-	-	OMoE: Threshold based on health endpoint (supporting document not available).

Table I-2 Acute Inhalation 24-Hour Health-Based Thresholds (continued)

Parameter	Air Screening Levels and Guidelines [µg/m^3]							Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2,3]			OMoE[4,5]	ATSDR[6]	WHO[7,8]	
	Standard	Desirable	Acceptable	Tolerable				
barium	-	-	-	-	10	-	-	OMoE: Threshold based on health endpoint (supporting document not available), for total water soluble barium compounds.
beryllium	-	-	-	-	0.01	-	-	OMoE: Threshold based on health endpoint (supporting document not available), for beryllium and compounds.
boron	-	-	-	-	120	300	-	OMoE: Threshold based on particulate endpoint (supporting documentation not available); ATSDR: Threshold based on significant increased nasal secretions in volunteers. A NOAEL of 0.6 mg/m^3 and an uncertainty factor of 3 (for human variability in the pharmacodynamic response to boron) were used.
cadmium	-	-	-	-	0.025	0.3	-	OMoE: Threshold based on health endpoint (supporting document not available); ATSDR: Threshold baed on a LOAEL of 0.088 mg/m^3 for respiratory effects in rats exposed to cadmium oxide for 6.2 hours/day, 5 days/week for 2 weeks and an uncertainty factor of 300 (3 for extrapolating from animals to humans and 10 for human variability).
calcium	-	-	-	-	10	-	-	OMoE: Threshold based on corrosion endpoint (supporting document not available), for the metal parameter/oxide.
chromium (as chromium [III])	-	-	-	-	0.5	-	-	OMoE: Threshold (for Cr(0), Cr(II) and Cr(III)) based on respiratory effects associated with exposure to Cr(III). It is based on a $BMCL_{10}$ value of 3.45 mg/m^3 subchronic inhalation exposure study (rats) and an uncertainty factor (300).
cobalt	-	-	-	-	0.1	-	-	OMoE: Threshold based on health endpoint (supporting document not available).
copper	-	-	-	-	50	-	-	OMoE: Threshold based on health endpoint (supporting document not available).
iron	-	-	-	-	4	-	-	OMoE: Threshold based on health endpoint (supporting document not available), as metallic iron.
lead	-	-	-	-	0.5	-	-	OMoE: Threshold based on health endpoint (supporting document not available), for lead and compounds.
lithium	-	-	-	-	20	-	-	OMoE: Threshold based on health endpoint (supporting document not available), excluding hydrides.
magnesium	-	-	-	-	120	-	-	OMoE: Threshold based on particulate endpoint (supporting document not available), as MgO.
manganese	-	-	-	-	0.1	-	-	OMoE: Threshold for Mn as metal/parameter in PM2.5. Based on neurological endpoint (supporting document available). Threshold based on two occupational studies in which the estimated level of occupational exposure to Mn associated with the appearance of subtle neurological deficiets ranged from 30 to 50 µg/m^3. Health Canada derived a Reference Concentration (RfC) by a range of derived $BMCL_{05}$ values (19.2 - 35.3 µg/m^3) for subtle but significant neurological effets. 10 µg/m^3 has been identified as the level of exposure at which Mn begin to accumulate in the brain.
mercury	-	-	-	-	2	-	-	OMoE: Threshold based on health endpoint (supporting document not available).
molybdenum	-	-	-	-	120	-	-	OMoE: Threshold based on particulate endpoint (supporting document not available), as a particulate.
nickel	-	-	-	-	0.1	-	-	OMoE: Threshold for nickel as a metal/parameter in PM10. Based on carcinogenic and non-carcinogenic effects (supporting documentation available). The 24-hr screening value was derived from the annual screening value (0.02 µg/m^3) and a conversion factor of 5, which is based on empirical monitoring data, ratios of concentrations observed for different averaging times, and meteriological considerations.
phosphorus	-	-	-	-	0.35	0.00002	-	OMoE: Jurisdictional Screening Level for white phosphorus; ATSDR: Respiratory effect, uncertainty factor of 30 (documentation not available) for white phosphorus.
potassium	-	-	-	-	8	-	-	OMoE: Jurisdictional Screening Level.
selenium	-	-	-	-	10	-	-	OMoE: Threshold based on health endpoint (supporting document not available).
silver	-	-	-	-	1	-	-	OMoE: Threshold based on health endpoint (supporting document not available).
sodium	-	-	-	-	10	-	-	OMoE: Threshold based on corrosion endpoint (supporting document not available), as NaOH.
strontium	-	-	-	-	120	-	-	OMoE: Threshold based on particulate endpoint (supporting document not available).
thallium	-	-	-	-	0.24	-	-	OMoE: Jurisdictional Screening Level.
tin	-	-	-	-	10	-	-	OMoE: Threshold based on health endpoint (supporting document not available).
titanium	-	-	-	-	34	-	-	OMoE: Threshold for titanium dioxide based on particulate endpoint (supporting documents not available).
tungsten	-	-	-	-	20	-	-	OMoE: Jurisdictional Screening Level as insoluble tungsten compounds.

Table I-2 Acute Inhalation 24-Hour Health-Based Thresholds (continued)

Parameter	Air Screening Levels and Guidelines [µg/m^3]							Toxicological Endpoints and Derivations
	NWT[1]	CCME NAAQO[2,3]			OMoE[4,5]	ATSDR[6]	WHO[7,8]	
	Standard	Desirable	Acceptable	Tolerable				
uranium	-	-	-	-	0.15	-	-	OMoE: Thresholdfor uranium as a metal/parameter in PM10. Based on kidney toxicity (based on U accumulation in the kidney over a 50 year exposure period that is considered to be protective for long-term continuous inhalation exposure) for uranium in PM_{10} and TSP, respectively. A factor of 5 was applied to the annual average of 0.03 µg/m^3 for U for the 24 hour average.
vanadium	-	-	-	-	2	0.8	1	OMoE: Threshold based on health endpoint (supporting document not available yet); ATSDR: Threshold baesd on a LOAEL of 0.56 mg/m^3 for lung inflammation in rats exposed to vanadium pentoxide for 6 hours/day, 5 days/week for 13 days. The human equivalent concentration of the LOAEL (0.073 mg/m^3) was divided by an uncertainty factor of 90 (3 for using minimal LOAEL, 3 for animal to human extrapolation and 10 for human variability); WHO: Threshold based on occupational studies suggesting that the LOAEL of V can be assumed to be 20 µg/m^3, based on chronic upper respiratory tract symptoms. The adverse nature of the observed effects were minimal at 20 µg/m^3 and a susceptible subpopulation was not identified, a protection factor of 20 was selected.
zinc	-	-	-	-	120	-	-	OMoE: Threshold based on particulate endpoint (supporting document not available).
zirconium	-	-	-	-	20	-	-	OMoE: Jurisdictional Screening Level, for zirconium and compounds.
Dioxins and Furans								
Total dioxins / furans	-	-	-	-	1.00E-7 TEQ	-	-	OMoE: The TEQ guideline is based on developmental effects in offspring of experimental animals exposed to TCDD *in utero* at low maternal doses during gestation. This endpoint appears to be the most sensitive for TCDD toxicity (supporting documentation available). Assuming TCDD half-life of 2774 days and 80% bioavailability from food, an animal steady-state maternal body burden of 33 mg/kg bw gives an equivalent human daily intake (EHDI) of 10.3 pg/kg bw/day. An uncertainty factor of 10 was applied and a 3% apportionment of the TRV to exposure from air and subsequent route-to-route extrapolation gives a value of 0.1 pg/m^3. Toxicity equivalency factors (TEFs) are applied to 17 dioxin and furan isomers of concern to convert them into 2,3,7,8-TCDD (tetrachlorodibenzo-p-dioxin) toxicity equivalents. The conversion involves multiplying the concentration of the isomer by the appropriate TEF to yield the TEQ for this isomer. Summing the individual TEQ values for each of the isomers of concern provides the total toxicity equivalent level for the sample mixture.

1. Northwest Territories Department of Environment and Natural Resources – Ambient Air Quality Standards (NWT 2011)
2. Canadian Council of Ministers of the Environment (CCME 1999)
3. Guidelines from the Canadian Council of Ministers of the Environment (CCME), Guidance Document on Achievement Determination: Canada-wide Standards for Particulate Matter and Ozone (CCME 2007).
4. Ontario Ministry of the Environment (OMoE 2012)
5. OMoE Jurisdictional Screening Levels (JSL) (OMoE 2008).
6. Agency of Toxic Substances and Disease Registry (ATSDR), Minimum Risk Levels (MRLs) (ATSDR 2012)
7. World Health Organization (WHO), WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide (WHO 2006)
8. WHO Air Quality Guidelines for Europe, Second Edition. (WHO 2000).

Notes: All values are in µg/m^3, unless otherwise noted.
- = Value not available.
Shaded acute thresholds were used in the risk assessment.
*Value converted from parts-per-million (ppm) to micrograms per meter cubed (µg/m^3) using the molecular weight (ML) of the specified compound, in the following formula:Y mg/m^3=(X ppm)(molecular weight)/24.45, (assumptions: 25 ºC and 1 atm).
BMCL10 - Benchmark Dose 95% lower confidence limit, determined using the EPA Benchmark Dose Software, CNS - Central Nervous System, HQ - Hazard Quotient, IUR - Inhalation Unit Risk (µg/m3)-1, LOAEL - Lowest Observed Adverse Effects Level, NOAEL - No Observed Adverse Effects Level, NAAQO - Canadian National Ambient Air Quality Objectives, MRL- Minimal Risk Level, MW - Molecular Weight (grams per moles), REL - Reference Exposure Level, PAH - Polycyclic Aromatic Hydrocarbons, PEF - Potency Equivalent Factor, PM - Particulate Matter, ppm - Parts per million, RfC – Reference Concentration (µg/m3), TEQ - Toxicity Equivalent Quotient, WHO - World Health Organization, ATSDR – Agency for Toxic Substances and Disease Registry: acute 1-14 days, intermediate >14 - 364 days, chronic ≥365 days.

Table I-3 Chemicals of Potential Concern Screening based on Predicted Peak 1-Hour Concentrations at Each Receptor Location

Parameter	Air Threshold	Predicted Air Concentrations at the Following Locations:						Predicted Maximum Concentration (All Locations)	Measured Maximum Baseline Concentration	Measured Maximum Baseline Concentration + 10%	Above Air Threshold?	Above Measured Maximum Baseline Concentration + 10%?	Retained as a COC?
		Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary						
Acid Gases													
Sulfur dioxide (SO_2)	450	3.7	4.1	3.5	35.3	7.2	43.4	43.4	4.1	4.5	No	Yes	No
Nitrogen dioxide (NO_2)	400	43.1	38.1	23.2	234	101	315	314.8	43.1	47.4	No	Yes	No
Carbon monoxide (CO)	15,000	130	129	124	1715	344	2144	2144	13.8	15.2	No	Yes	No
Particulate Matter													
$PM_{2.5}$	-	3.5	3.2	3.7	253	39	311	311	3.7	4.1	n/a	Yes	Yes
PM_{10}	-	4.6	4.8	4.2	1118	143	1922	1922	3.7	4.1	n/a	Yes	Yes
TSP	-	7.8	8.0	7.8	1668	140	5741	5741	7.8	8.6	n/a	Yes	Yes
Volatile Organic Compounds													
1,1,1-Trichloroethane	2,800	0.0000048	0.0000076	0.0000038	0.000169	0.0000071	0.00013	0.000169	0.0000076	0.00000836	No	Yes	No
1,3-Butadiene	100 to 510	0.0020	0.0018	0.0011	0.0998	0.0147	0.1530	0.153	0.00198	0.00218	No	Yes	No
Acetaldehyde	470	0.27	0.25	0.15	13.5	1.98	20.6	20.6	0.267	0.294	No	Yes	No
Acetone	6,1760	0.141	0.129	0.0763	7.08	1.04	10.9	10.9	0.141	0.155	No	Yes	No
Acrolein	2.5	0.0217	0.02	0.0118	1.09	0.161	1.68	1.68	0.0217	0.0239	No	Yes	No
Aldehydes (surrogate: acetaldehyde)	470	0.371	0.341	0.201	18.7	2.75	28.6	28.6	0.371	0.408	No	Yes	No
Benzene	1,300	0.0175	0.0161	0.00953	0.882	0.13	1.35	1.35	0.0175	0.0193	No	Yes	No
C2 to C8 aliphatics (surrogate: cyclohexane)	3,400	0.218	0.2	0.118	11	1.62	16.8	16.8	0.218	0.24	No	Yes	No
C9 to C16 aliphatics (surrogate: decane)	60,000	0.0285	0.0262	0.0155	1.43	0.212	2.2	2.2	0.0285	0.0313	No	Yes	No
C16+ aliphatics (surrogate: decane)	60,000	0.0224	0.0206	0.0121	1.13	0.166	1.73	1.73	0.0224	0.0246	No	Yes	No
C6 to C8 aromatics (surrogate: toluene)	37,000	0.0326	0.0299	0.0177	1.64	0.242	2.52	2.52	0.0326	0.0359	No	Yes	No
C9 to C16 aromatics (surrogate ethylbenzene)	1,900	0.0322	0.0296	0.0175	1.62	0.239	2.48	2.48	0.0322	0.0354	No	Yes	No
Ethylbenzene	1,900	0.00301	0.00277	0.00164	0.151	0.0223	0.232	0.232	0.00301	0.00331	No	Yes	No
Formaldehyde	49.1	0.143	0.131	0.0775	7.18	1.06	11	11	0.143	0.157	No	Yes	No
Methyl ethyl ketone	13,000	0.0479	0.044	0.026	2.41	0.356	3.7	3.7	0.0479	0.0527	No	Yes	No
Toluene	37,000	0.0256	0.0235	0.0139	1.28	0.189	1.96	1.96	0.0256	0.0281	No	Yes	No
trimethylbenzenes	1,250	0.00729	0.00669	0.00395	0.367	0.0541	0.562	0.562	0.00729	0.00801	No	Yes	No
Xylene (total)	8,680	0.0202	0.0186	0.011	1.02	0.15	1.56	1.56	0.0202	0.0222	No	Yes	No
Dioxins/Furans (µg TEQ/m^3)	3.00E-07	1.48E-11	2.07E-11	1.05E-11	8.58E-10	8.76E-11	2.02E-09	2.02E-09	2.07E-11	2.28E-11	No	Yes	No
Polyaromatic Hydrocarbons													
1-Methylnaphthalene	30	0.000179	0.00016	0.0000931	0.0638	0.00944	0.099	0.099	0.000179	0.000197	No	Yes	No
1-Methylphenanthrene (surrogate: phenanthrene)	0.5	0.00000805	0.00000722	0.00000419	0.00287	0.000425	0.00445	0.00445	0.00000805	0.00000886	No	Yes	No
2-Methylanthracene (surrogate: anthracene)	0.5	0.00000493	0.00000441	0.00000256	0.00176	0.00026	0.00273	0.00273	0.00000493	0.00000542	No	Yes	No
2-Methylfluorene (surrogate: fluorene)	10	0.000000166	0.000000149	8.64E-08	0.0000592	0.00000876	0.0000919	0.0000919	0.000000166	0.000000183	No	Yes	No
2-Methylnaphthalene	30	0.000289	0.000259	0.000151	0.103	0.0153	0.16	0.16	0.000289	0.000318	No	Yes	No

Table I-3 Chemicals of Potential Concern Screening Based on Predicted Peak 1-Hour Concentrations at Each Receptor Location (continued)

Parameter	Air Threshold	Predicted Air Concentrations at the Following Locations:						Predicted Maximum Concentration (All Locations)	Measured Maximum Baseline Concentration	Measured Maximum Baseline Concentration + 10%	Above Air Threshold?	Above Measured Maximum Baseline Concentration + 10%?	Retained as a COC?
		Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary						
2-Methylphenanthrene (surrogate: phenanthrene)	0.5	0.0000199	0.0000178	0.0000103	0.00709	0.00105	0.011	0.011	0.0000199	0.0000219	No	Yes	No
2-Methylpyrene (surrogate: pyrene)	0.5	0.00000147	0.00000132	0.000000767	0.000525	0.0000777	0.000815	0.000815	0.00000147	0.00000162	No	Yes	No
3-Methyldibenzothiophene (surrogate: dibenzothiophene)	25	0.000000304	0.000000273	0.000000158	0.000108	0.0000161	0.000168	0.000168	0.000000304	0.000000335	No	Yes	No
3-Methylphenanthrene (surrogate: phenanthrene)	0.5	0.0000143	0.0000129	0.00000747	0.00511	0.000757	0.00794	0.00794	0.0000143	0.0000158	No	Yes	No
4 + 9 Methylphenanthrene (surrogate: dibenzothiophene)	0.5	0.0000108	0.00000972	0.00000564	0.00386	0.000572	0.006	0.006	0.0000108	0.0000119	No	Yes	No
4-Methyldibenzothiophene (surrogate: dibenzothiophene)	25	0.000000194	0.000000174	0.000000101	0.0000691	0.0000102	0.000107	0.000107	0.000000194	0.000000213	No	Yes	No
Acenaphthene	1	0.0000093	0.0000083	0.00000488	0.00326	0.000482	0.00506	0.00506	0.0000093	0.0000102	No	Yes	No
Acenaphthylene	1	0.0000332	0.0000298	0.0000173	0.0118	0.00175	0.0184	0.0184	0.0000332	0.0000365	No	Yes	No
Acephenanthrylene (surrogate: benzo(k)fluoranthene)	0.5	0.00000568	0.00000509	0.00000296	0.00203	0.0003	0.00314	0.00314	0.00000568	0.00000625	No	Yes	No
Anthracene	0.5	0.00000593	0.00000531	0.00000309	0.00211	0.000312	0.00328	0.00328	0.00000593	0.00000652	No	Yes	No
Benz(a)anthracene	0.5	0.00000144	0.00000129	0.000000758	0.000503	0.0000744	0.000781	0.000781	0.00000144	0.00000158	No	Yes	No
Benzo(a)fluorene (surrogate: 2,3-benzo(b)fluorene)	0.5	0.00000179	0.0000016	0.00000093	0.000637	0.0000943	0.000989	0.000989	0.00000179	0.00000197	No	Yes	No
Benzo(a)pyrene	0.00015	0.000000782	0.000000701	0.000000407	**0.000279**	0.0000412	**0.000433**	**0.000433**	0.000000782	0.00000086	Yes	Yes	Yes
Benzo(b)fluoranthene	0.5	0.00000657	0.00000589	0.00000342	0.00234	0.000346	0.00363	0.00363	0.00000657	0.00000723	No	Yes	No
Benzo(e)pyrene	0.5	0.000000111	9.92E-08	5.76E-08	0.0000394	0.00000584	0.0000612	0.0000612	0.000000111	0.000000122	No	Yes	No
Benzo(g,h,i)fluoranthene (surrogate: benzo(g,h,i)perylene)	0.5	0.00000276	0.00000247	0.00000143	0.000982	0.000145	0.00152	0.00152	0.00000276	0.00000303	No	Yes	No
Benzo(g,h,i)perylene	0.5	0.00000181	0.00000162	0.000000945	0.000638	0.0000944	0.000991	0.000991	0.00000181	0.00000199	No	Yes	No
Benzo(k)fluoranthene	0.5	0.000000746	0.000000669	0.000000388	0.000266	0.0000393	0.000413	0.000413	0.000000746	0.000000821	No	Yes	No
Chrysene	0.5	0.0000016	0.00000143	0.00000084	0.000565	0.0000837	0.000878	0.000878	0.0000016	0.00000176	No	Yes	No
Coronene (surrogate: benzo(g,h,i)perylene)	0.5	1.38E-08	1.24E-08	7.2E-09	0.00000493	0.00000073	0.00000766	0.00000766	1.38E-08	1.52E-08	No	Yes	No
Cyclopenta(c,d)pyrene (surrogate: benzo(a)pyrene)	0.03	0.000000976	0.000000874	0.000000508	0.000348	0.0000515	0.00054	0.00054	0.000000976	0.00000107	No	Yes	No
Dibenz(a,h)anthracene	0.5	0.00000206	0.00000185	0.00000108	0.000731	0.000108	0.00113	0.00113	0.00000206	0.00000227	No	Yes	No
Dibenzothiophene	25	0.000000127	0.000000114	6.95E-08	0.000042	0.00000622	0.0000652	0.0000652	0.000000127	0.000000139	No	Yes	No
Fluoranthene	0.5	0.0000251	0.0000225	0.0000131	0.00894	0.00132	0.0139	0.0139	0.0000251	0.0000277	No	Yes	No
Fluorene	10	0.0000473	0.0000424	0.0000246	0.0168	0.00249	0.0262	0.0262	0.0000473	0.000052	No	Yes	No
Indeno(1,2,3-cd)fluoranthene (surrogate: fluoranthene)	0.5	6.92E-08	0.000000062	0.000000036	0.0000247	0.00000365	0.0000383	0.0000383	6.92E-08	7.61E-08	No	Yes	No
Indeno(1,2,3-cd)pyrene	0.5	4.35E-08	6.89E-08	3.44E-08	0.00000153	6.48E-08	0.00000118	0.00000153	6.89E-08	7.58E-08	No	Yes	No
Indeno(1,2,3-W)pyrene (surrogate: indeno(1,2,3-cd)pyrene)	0.5	0.00000124	0.00000111	0.000000647	0.000443	0.0000656	0.000688	0.000688	0.00000124	0.00000137	No	Yes	No
Naphthalene	440	0.000717	0.000641	0.000376	0.253	0.0374	0.392	0.392	0.000717	0.000789	No	Yes	No
Nitro-pyrene	0.5	0.00000111	0.000000991	0.000000576	0.000394	0.0000584	0.000612	0.000612	0.00000111	0.00000122	No	Yes	No
Perylene	0.5	1.38E-08	1.24E-08	7.2E-09	0.00000493	0.00000073	0.00000766	0.00000766	1.38E-08	1.52E-08	No	Yes	No

Table I-3 Chemicals of Potential Concern Screening Based on Predicted Peak 1-Hour Concentrations at Each Receptor Location (continued)

Parameter	Air Threshold	Predicted Air Concentrations at the Following Locations:						Predicted Maximum Concentration (All Locations)	Measured Maximum Baseline Concentration	Measured Maximum Baseline Concentration + 10%	Above Air Threshold?	Above Measured Maximum Baseline Concentration + 10%?	Retained as a COC?
		Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary						
Phenanthrene	0.5	0.0000442	0.0000396	0.000023	0.0157	0.00233	0.0244	0.0244	0.0000442	0.0000486	No	Yes	No
Picene (surrogate: dibenzo(a,h)anthracene)	0.5	1.38E-08	1.24E-08	7.2E-09	0.00000493	0.00000073	0.00000766	0.00000766	1.38E-08	1.52E-08	No	Yes	No
Pyrene	0.5	0.0000341	0.0000306	0.0000178	0.0121	0.0018	0.0188	0.0188	0.0000341	0.0000375	No	Yes	No
Metals & Inorganics													
Aluminum	50	0.0373	0.0443	0.0295	27.5	3.37	**55**	**55**	0.00984	0.0108	Yes	Yes	Yes
Antimony	5	0.00000129	0.00000154	0.00000102	0.00095	0.000121	0.00204	0.00204	0.000000251	0.000000276	No	Yes	No
Arsenic	0.2	0.0000167	0.0000158	0.0000116	0.00272	0.000338	0.00555	0.00555	0.0000167	0.0000184	No	Yes	No
Barium	5	0.000791	0.000938	0.000628	0.582	0.0699	1.12	1.12	0.000237	0.00026	No	Yes	No
Beryllium	0.02	0.00000768	0.00000741	0.0000054	0.0003	0.000011	0.000231	0.0003	0.00000768	0.00000845	No	Yes	No
Bismuth	50	0.000000403	0.000000478	0.00000032	0.000285	0.0000358	0.000594	0.000594	8.48E-08	9.33E-08	No	Yes	No
Boron	50	0.0000322	0.0000384	0.0000254	0.0301	0.00309	0.0438	0.0438	0.0000245	0.0000269	No	Yes	No
Cadmium	0.1	0.000261	0.000233	0.000138	0.0445	0.00504	0.0985	0.0985	0.000261	0.000288	No	Yes	No
Chromium (as chromium [III])	3.6[(a)]	0.00034	0.000403	0.000271	0.249	0.0295	0.467	0.467	0.000151	0.000166	No	Yes	No
Cobalt	0.2	0.0000684	0.0000812	0.0000543	0.0499	0.00596	0.0931	0.0931	0.0000535	0.0000589	No	Yes	No
Copper	100	0.0000579	0.0000687	0.000046	0.0415	0.00502	0.0787	0.0787	0.0000575	0.0000632	No	Yes	No
Iron	10	0.0701	0.0833	0.0556	**51.7**	6.33	**103**	**103**	0.0187	0.0206	Yes	Yes	Yes
Lead	0.5[(c)]	0.000055	0.0000475	0.0000302	0.0226	0.00287	0.0465	0.0465	0.000055	0.0000605	No	Yes	No
Lithium	10	0.00000609	0.00000708	0.00000495	0.00769	0.000819	0.018	0.018	0	0	No	Yes	No
Manganese	2	0.00102	0.00121	0.000804	0.752	0.0917	1.49	1.49	0.000304	0.000334	No	Yes	No
Mercury	0.6	0.0000551	0.0000513	0.0000381	0.00405	0.000394	0.00957	0.00957	0.0000551	0.0000606	No	Yes	No
Molybdenum	30	0.000012	0.0000143	0.00000949	0.00882	0.00113	0.0191	0.0191	0.0000023	0.00000253	No	Yes	No
Nickel	0.2	0.000551	0.000652	0.00044	**0.417**	0.046	**0.692**	**0.692**	0.0003	0.00033	Yes	Yes	Yes
Selenium	2	0.0000388	0.0000373	0.0000272	0.00151	0.000179	0.00297	0.00297	0.0000388	0.0000427	No	Yes	No
Silver	0.1	0.0000417	0.0000371	0.0000214	0.00747	0.000866	0.0165	0.0165	0.0000417	0.0000459	No	Yes	No
Sodium	8	0.00133	0.00158	0.00106	1	0.117	1.85	1.85	0.000478	0.000525	No	Yes	No
Strontium	20	0.00021	0.000249	0.000168	0.161	0.0178	0.267	0.267	0.000102	0.000112	No	Yes	No
Thallium	1	0.000000601	0.000000715	0.000000474	0.00044	0.0000558	0.000931	0.000931	0.000000123	0.000000135	No	Yes	No
Tin	20	6.54E-08	7.61E-08	5.31E-08	0.0000826	0.00000879	0.000194	0.000194	0	0	No	Yes	No
Titanium	50	0.00346	0.00412	0.00273	2.53	0.32	5.33	5.33	0.000715	0.000787	No	Yes	No
Tungsten	50[(b)]	0.000000388	0.000000462	0.000000307	0.000285	0.0000358	0.000594	0.000594	8.48E-08	9.33E-08	No	Yes	No
Uranium	2[(b)]	0.00000343	0.00000408	0.00000271	0.00253	0.000316	0.00521	0.00521	0.000000803	0.000000883	No	Yes	No
Vanadium	30	0.000116	0.000138	0.0000921	0.0853	0.0106	0.174	0.174	0.000028	0.0000308	No	Yes	No
Zinc	20	0.000305	0.000271	0.000157	0.0794	0.01	0.157	0.157	0.000305	0.000336	No	Yes	No
Zirconium	50	0.00000275	0.00000319	0.00000223	0.00347	0.000369	0.00814	0.00814	0	0	No	Yes	No

(a) Screening value for chromium (III).

(b) Screening value for the insoluble metal form.

(c) 24-hour screening value used in the absence of a 1-hour screening value.

Notes: Units are in $\mu g/m^3$.
Shaded and bold values exceed the air threshold.
NG = No Guideline.
'-' = no threshold available

Table I-4 Chemicals of Potential Concern Screening based on Predicted Peak 24-Hour Concentrations at Each Receptor Location

Parameter	Air Threshold	Predicted Air Concentrations at the Following Locations:						Predicted Maximum Concentration (All Locations)	Measured Maximum Baseline Concentration	Measured Maximum Baseline Concentration + 10%	Above Air Threshold?	Above Measured Maximum Baseline Concentration + 10%?	Retained as a COC?
		Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary						
Acid Gases													
Sulfur dioxide (SO_2)	150	2.9	2.9	2.9	16.9	3.5	33.3	33.3	2.9	3.2	No	Yes	No
Nitrogen dioxide (NO_2)	200	9.7	11.2	10.2	170	54.3	**224**	**224**	11.2	12.3	Yes	Yes	**Yes**
Carbon monoxide (CO) [(a)]	6,000	120	122	119	1249	216	1856	1856	5.1	5.6	No	Yes	No
Particulate Matter													
$PM_{2.5}$	30	2.1	2.1	2.1	**89**	6.4	**138**	**138**	2.2	2.4	Yes	Yes	**Yes**
PM_{10}	25	3.3	3.3	3.2	**172**	22.6	**515**	**515**	3.1	3.4	Yes	Yes	**Yes**
Total Suspended Particulate (TSP)	120	7.3	7.4	7.3	**281**	31.8	**1308**	**1308**	7.2	8.0	Yes	Yes	**Yes**
Volatile Organic Compounds													
1,1,1-Trichloroethane	10,900	0.00000138	0.00000136	0.00000121	0.0000741	0.00000114	0.0000468	0.0000741	0.00000138	0.00000152	No	Yes	No
1,3-Butadiene	10	0.000199	0.000402	0.0002	0.044	0.00307	0.0994	0.0994	0.000401	0.000441	No	Yes	No
Acetaldehyde	500	0.0269	0.0542	0.027	5.94	0.414	13.4	13.4	0.0541	0.0595	No	Yes	No
Acetone	11,880	0.0142	0.0285	0.0142	3.13	0.218	7.05	7.05	0.0285	0.0313	No	Yes	No
Acrolein	0.4	0.00219	0.00441	0.0022	**0.483**	0.0337	**1.09**	**1.09**	0.0044	0.00484	Yes	Yes	**Yes**
Aldehydes (surrogate: acetaldehyde)	500	0.0373	0.0752	0.0375	8.24	0.575	18.6	18.6	0.075	0.0825	No	Yes	No
Benzene	2.3	0.00177	0.00355	0.00178	0.389	0.0272	0.878	0.878	0.00354	0.0039	No	Yes	No
C2 to C8 aliphatics (surrogate: cyclohexane)	6,100	0.022	0.0442	0.022	4.85	0.338	10.9	10.9	0.0441	0.0485	No	Yes	No
C9 to C16 aliphatics (surrogate: decane)	6,100	0.00287	0.00577	0.00288	0.633	0.0442	1.43	1.43	0.00576	0.00634	No	Yes	No
C16+ aliphatics (surrogate: decane)	6,100	0.00225	0.00454	0.00226	0.497	0.0347	1.12	1.12	0.00453	0.00498	No	Yes	No
C6 to C8 aromatics (surrogate: toluene)	3,770	0.00328	0.00661	0.00329	0.724	0.0506	1.63	1.63	0.0066	0.00726	No	Yes	No
C9 to C16 aromatics (surrogate ethylbenzene)	1,000	0.00324	0.00652	0.00325	0.715	0.0499	1.61	1.61	0.00651	0.00716	No	Yes	No
Ethylbenzene	1,000	0.000304	0.000609	0.000306	0.0668	0.00467	0.151	0.151	0.000608	0.000669	No	Yes	No
Formaldehyde	65	0.0144	0.0289	0.0145	3.17	0.221	7.15	7.15	0.0289	0.0318	No	Yes	No
Methyl ethyl ketone	1,000	0.00483	0.00972	0.00484	1.07	0.0744	2.4	2.4	0.0097	0.0107	No	Yes	No
Toluene	3,770	0.0026	0.00516	0.00261	0.565	0.0396	1.28	1.28	0.00515	0.00567	No	Yes	No
Trimethylbenzenes	220	0.000734	0.00148	0.000736	0.162	0.0113	0.365	0.365	0.00147	0.00162	No	Yes	No
Xylene (total)	730	0.00204	0.00409	0.00205	0.449	0.0314	1.01	1.01	0.00409	0.0045	No	Yes	No
Dioxins/Furans [(b)] (µg TEQ/m^3)	1.00E-07	2.06E-12	1.78E-12	1.80E-12	1.55E-10	8.64E-12	7.78E-10	7.78E-10	2.06E-12	2.27E-12	No	Yes	**No**
Polyaromatic Hydrocarbons													
1-Methylnaphthalene	12	0.0000261	0.0000282	0.0000283	0.0282	0.00185	0.0642	0.0642	0.0000279	0.0000307	No	Yes	No
1-Methylphenanthrene (surrogate: phenanthrene)	-	0.00000118	0.00000127	0.00000127	0.00127	0.0000831	0.00289	0.00289	0.00000126	0.00000138	NG	Yes	No [(c)]
2-Methylanthracene (surrogate: anthracene)	0.2	0.000000719	0.000000777	0.000000778	0.000777	0.0000508	0.00177	0.00177	0.000000768	0.000000845	No	Yes	No
2-Methylfluorene (surrogate: fluorene)	-	2.42E-08	2.62E-08	2.62E-08	0.0000262	0.00000171	0.0000596	0.0000596	2.59E-08	2.85E-08	NG	Yes	No [(c)]
2-Methylnaphthalene	10	0.0000422	0.0000456	0.0000457	0.0456	0.00299	0.104	0.104	0.0000451	0.0000496	No	Yes	No
2-Methylphenanthrene (surrogate: phenanthrene)	-	0.0000029	0.00000314	0.00000314	0.00314	0.000205	0.00713	0.00713	0.0000031	0.00000341	NG	Yes	No [(c)]

Table I-4 Chemicals of Potential Concern Screening Based on Predicted Peak 24-Hour Concentrations at Each Receptor Location (continued)

Parameter	Air Threshold	Predicted Air Concentrations at the Following Locations:						Predicted Maximum Concentration (All Locations)	Measured Maximum Baseline Concentration	Measured Maximum Baseline Concentration + 10%	Above Air Threshold?	Above Measured Maximum Baseline Concentration + 10%?	Retained as a COC?
		Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary						
2-Methylpyrene (surrogate: pyrene)	0.2	0.000000215	0.000000232	0.000000233	0.000232	0.0000152	0.000528	0.000528	0.00000023	0.000000253	No	Yes	No
3-Methyldibenzothiophene (surrogate: dibenzothiophene)	-	4.44E-08	0.000000048	4.81E-08	0.000048	0.00000314	0.000109	0.000109	4.75E-08	5.22E-08	NG	Yes	No [(c)]
3-Methylphenanthrene (surrogate: phenanthrene)	-	0.00000209	0.00000226	0.00000227	0.00226	0.000148	0.00514	0.00514	0.00000224	0.00000246	NG	Yes	No [(c)]
4 + 9 Methylphenanthrene (surrogate: dibenzothiophene)	-	0.00000158	0.00000171	0.00000171	0.00171	0.000112	0.00389	0.00389	0.00000169	0.00000186	NG	Yes	No [(c)]
4-Methyldibenzothiophene (surrogate: dibenzothiophene)	-	2.83E-08	3.06E-08	3.06E-08	0.0000306	0.000002	0.0000695	0.0000695	3.02E-08	3.33E-08	NG	Yes	No [(c)]
Acenaphthene	-	0.00000135	0.0000015	0.00000147	0.00144	0.0000943	0.00328	0.00328	0.0000015	0.00000165	NG	Yes	No [(c)]
Acenaphthylene	3.5	0.00000485	0.00000524	0.00000524	0.00524	0.000343	0.0119	0.0119	0.00000518	0.0000057	No	Yes	No
Acephenanthrylene (surrogate: benzo(k)fluoranthene)	-	0.000000829	0.000000896	0.000000898	0.000896	0.0000586	0.00204	0.00204	0.000000886	0.000000975	NG	Yes	No [(i)]
Anthracene	0.2	0.000000865	0.000000935	0.000000937	0.000934	0.0000611	0.00212	0.00212	0.000000927	0.00000102	No	Yes	No
Benz(a)anthracene	-	0.000000208	0.000000234	0.000000228	0.000223	0.0000146	0.000506	0.000506	0.000000234	0.000000257	NG	Yes	No [(c)]
Benzo(a)fluorene (surrogate: 2,3-benzo(b)fluorene)	-	0.000000261	0.000000282	0.000000282	0.000282	0.0000184	0.000641	0.000641	0.000000279	0.000000307	NG	Yes	No [(c)]
Benzo(a)pyrene	0.0001	0.000000114	0.000000123	0.000000124	**0.000123**	0.00000807	**0.00028**	**0.00028**	0.000000122	0.000000134	Yes	Yes	**Yes**
Benzo(b)fluoranthene	-	0.000000958	0.00000104	0.00000104	0.00103	0.0000677	0.00235	0.00235	0.00000103	0.00000113	NG	Yes	No [(c)]
Benzo(e)pyrene	-	1.61E-08	1.74E-08	1.75E-08	0.0000174	0.00000114	0.0000397	0.0000397	1.73E-08	0.000000019	NG	Yes	No [(c)]
Benzo(g,h,i)fluoranthene (surrogate: benzo(g,h,i)perylene)	1.2	0.000000402	0.000000435	0.000000435	0.000435	0.0000284	0.000988	0.000988	0.00000043	0.000000473	No	Yes	No
Benzo(g,h,i)perylene	1.2	0.000000263	0.000000287	0.000000286	0.000282	0.0000185	0.000642	0.000642	0.000000287	0.000000316	No	Yes	No
Benzo(k)fluoranthene	-	0.000000109	0.000000118	0.000000118	0.000118	0.0000077	0.000267	0.000267	0.000000116	0.000000128	NG	Yes	No [(c)]
Chrysene	-	0.000000233	0.000000255	0.000000254	0.00025	0.0000164	0.000569	0.000569	0.000000255	0.000000281	NG	Yes	No [(c)]
Coronene (surrogate: benzo(g,h,i)perylene)	1.2	2.02E-09	2.18E-09	2.19E-09	0.00000218	0.000000143	0.00000496	0.00000496	2.16E-09	2.37E-09	No	Yes	No
Cyclopenta(c,d)pyrene	-	0.000000142	0.000000154	0.000000154	0.000154	0.0000101	0.00035	0.00035	0.000000152	0.000000167	NG	Yes	No [(c)]
Dibenz(a,h)anthracene	-	0.0000003	0.000000325	0.000000326	0.000323	0.0000212	0.000735	0.000735	0.000000325	0.000000358	NG	Yes	No [(c)]
Dibenzothiophene	-	1.99E-08	2.19E-08	2.22E-08	0.0000186	0.00000123	0.0000423	0.0000423	2.19E-08	2.41E-08	NG	Yes	No [(c)]
Fluoranthene	140	0.00000367	0.00000396	0.00000398	0.00396	0.000259	0.009	0.009	0.00000393	0.00000433	No	Yes	No
Fluorene	-	0.0000069	0.00000746	0.00000747	0.00745	0.000488	0.0169	0.0169	0.00000739	0.00000812	NG	Yes	No [(c)]
Indeno(1,2,3-cd)fluoranthene (surrogate: fluoranthene)	140	1.01E-08	1.09E-08	1.09E-08	0.0000109	0.000000714	0.0000248	0.0000248	1.08E-08	1.19E-08	No	Yes	No
Indeno(1,2,3-cd)pyrene	-	1.25E-08	1.23E-08	1.09E-08	0.000000672	1.04E-08	0.000000424	0.000000672	1.25E-08	1.37E-08	NG	Yes	No [(c)]
Indeno(1,2,3-W)pyrene (surrogate: indeno(1,2,3-cd)pyrene)	-	0.000000181	0.000000196	0.000000196	0.000196	0.0000128	0.000446	0.000446	0.000000194	0.000000213	NG	Yes	No [(c)]
Naphthalene	22.5	0.000104	0.000114	0.000113	0.112	0.00731	0.254	0.254	0.000114	0.000126	No	Yes	No
Nitro-pyrene	-	0.000000161	0.000000174	0.000000175	0.000174	0.0000114	0.000397	0.000397	0.000000173	0.00000019	NG	Yes	No [(c)]
Perylene	-	2.02E-09	2.18E-09	2.19E-09	0.00000218	0.000000143	0.00000496	0.00000496	2.16E-09	2.37E-09	NG	Yes	No [(c)]
Phenanthrene	-	0.00000646	0.00000696	0.000007	0.00695	0.000455	0.0158	0.0158	0.00000693	0.00000763	NG	Yes	No [(c)]
Picene	-	2.02E-09	2.18E-09	2.19E-09	0.00000218	0.000000143	0.00000496	0.00000496	2.16E-09	2.37E-09	NG	Yes	No [(c)]
Pyrene	0.2	0.00000498	0.00000537	0.00000539	0.00537	0.000351	0.0122	0.0122	0.00000533	0.00000586	No	Yes	No
Metals and Inorganics													
Aluminum	120 [(d)]	0.0064	0.00707	0.00572	3.87	0.481	12.4	12.4	0.00217	0.00238	No	Yes	No
Antimony	25	0.000000223	0.000000246	0.000000199	0.000114	0.0000169	0.000462	0.000462	5.88E-08	6.47E-08	No	Yes	No

Table I-4 Chemicals of Potential Concern Screening Based on Predicted Peak 24-Hour Concentrations at Each Receptor Location (continued)

Parameter	Air Threshold	Predicted Air Concentrations at the Following Locations:						Predicted Maximum Concentration (All Locations)	Measured Maximum Baseline Concentration	Measured Maximum Baseline Concentration + 10%	Above Air Threshold?	Above Measured Maximum Baseline Concentration + 10%?	Retained as a COC?
		Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary						
Arsenic	0.3	0.00000314	0.00000297	0.00000281	0.000382	0.0000484	0.00126	0.00126	0.00000314	0.00000346	No	Yes	No
Barium	10	0.000136	0.00015	0.000122	0.0897	0.0101	0.254	0.254	0.0000494	0.0000544	No	Yes	No
Beryllium	0.01	0.00000144	0.00000137	0.00000126	0.000132	0.00000192	0.0000832	0.000132	0.00000144	0.00000159	No	Yes	No
Bismuth	-	6.91E-08	7.64E-08	6.19E-08	0.0000351	0.00000512	0.000135	0.000135	1.94E-08	2.13E-08	NG	Yes	No [c]
Boron	120	0.00000555	0.00000612	0.00000495	0.00831	0.000449	0.00982	0.00982	0.00000438	0.00000482	No	Yes	No
Cadmium	0.025	0.0000297	0.0000438	0.0000279	0.0191	0.00129	**0.0517**	**0.0517**	0.0000438	0.0000481	Yes	Yes	**Yes**
Chromium (as chromium III)	0.5 [e]	0.0000583	0.0000645	0.0000523	0.0433	0.00431	0.105	0.105	0.0000346	0.0000381	No	Yes	No
Cobalt	0.1	0.0000117	0.000013	0.0000105	0.0104	0.000865	0.0222	0.0222	0.0000105	0.0000115	No	Yes	No
Copper	50	0.00000994	0.0000123	0.00000891	0.00879	0.000727	0.0195	0.0195	0.0000123	0.0000136	No	Yes	No
Iron	4	0.012	0.0133	0.0108	7.32	0.904	**23.3**	**23.3**	0.00406	0.00446	Yes	Yes	**Yes**
Lead	0.5	0.00000889	0.0000117	0.00000808	0.00517	0.000407	0.0134	0.0134	0.0000117	0.0000129	No	Yes	No
Lithium	20	0.00000103	0.00000115	0.000000929	0.000927	0.000102	0.00334	0.00334	0	0	No	Yes	No
Manganese	0.1 [f]	0.000174	0.000193	0.000156	**0.111**	0.0131	**0.336**	**0.336**	0.0000687	0.0000755	Yes	Yes	**Yes**
Mercury	2	0.0000103	0.00000923	0.00000975	0.00147	0.0000803	0.00738	0.00738	0.0000103	0.0000113	No	Yes	No
Molybdenum	120	0.00000207	0.00000229	0.00000185	0.00106	0.000157	0.00432	0.00432	0.000000534	0.000000587	No	Yes	No
Nickel	0.1 [g]	0.0000943	0.000104	0.0000847	0.0916	0.00694	**0.156**	**0.156**	0.0000569	0.0000626	Yes	Yes	**Yes**
Selenium	10	0.0000073	0.00000697	0.00000638	0.00066	0.0000271	0.000673	0.000673	0.0000073	0.00000803	No	Yes	No
Silver	1	0.00000432	0.00000652	0.00000412	0.00322	0.000216	0.00868	0.00868	0.00000652	0.00000717	No	Yes	No
Sodium	10	0.000228	0.000252	0.000204	0.17	0.0171	0.417	0.417	0.0000937	0.000103	No	Yes	No
Strontium	120	0.000036	0.0000398	0.0000323	0.0351	0.00267	0.0601	0.0601	0.0000183	0.0000201	No	Yes	No
Thallium	0.24	0.000000103	0.000000114	9.23E-08	0.0000527	0.00000782	0.000211	0.000211	2.87E-08	3.16E-08	No	Yes	No
Tin	10	0.000000011	1.23E-08	9.98E-09	0.00000996	0.0000011	0.0000358	0.0000358	0	0	No	Yes	No
Titanium	34 [d]	0.000595	0.000657	0.000532	0.302	0.0449	1.21	1.21	0.000166	0.000183	No	Yes	No
Tungsten	20 [h]	6.68E-08	7.37E-08	5.97E-08	0.0000351	0.00000504	0.000135	0.000135	1.94E-08	2.13E-08	No	Yes	No
Uranium	0.15 [g]	0.00000059	0.000000652	0.000000528	0.000326	0.0000446	0.00118	0.00118	0.00000018	0.000000198	No	Yes	No
Vanadium	0.8	0.00002	0.0000221	0.0000179	0.0113	0.0015	0.0395	0.0395	0.0000062	0.00000683	No	Yes	No
Zinc	120	0.0000339	0.0000504	0.0000318	0.0279	0.00172	0.0696	0.0696	0.0000504	0.0000555	No	Yes	No
Zirconium	20	0.000000462	0.000000518	0.000000419	0.000418	0.0000462	0.0015	0.0015	0	0	No	Yes	No

[a] Predicted carbon monoxide concentrations are averaged over an 8-hour period.

[b] Screening values as toxicity equivalent factors (TEF).

[c] Air thresholds are available for 1-hour and annual periods, and the parameter does not screen in as a COC for both time periods. When there are no guidelines for the 24-hour period, the parameter does not screen in.

[d] Screening value as the metal oxide.

[e] Screening value for chromium compounds (metallic, divalent and trivalent forms).

[f] Screening value as the metal in $PM_{2.5}$.

[g] Screening value as the metal in PM_{10}.

[h] Screening value as the insoluble metal compound.

Notes: Units are in $\mu g/m^3$.
Shaded and bold values exceed the air threshold.
NG = No Guideline.

Based on the acute air screening, the following COCs were identified as exceeding air thresholds:

1-hour

- benzo(a)pyrene;
- aluminum;
- iron; and
- nickel

Benzo(a)pyrene, aluminum, iron and nickel have been evaluated under the Acute Air Quality Risk Assessment.

24-hour

- nitrogen dioxide;
- acrolein;
- benzo(a)pyrene;
- cadmium;
- iron;
- manganese;
- nickel; and
- particulate Matter ($PM_{2.5}$, PM_{10} and TSP). PM_{10} was used as a surrogate to assess TSP due to the lack of health based criteria for TSP.

Nitrogen dioxide, acrolein, benzo(a)pyrene, cadmium, iron, manganese and nickel have been evaluated under the Acute Air Quality Risk Assessment. Particulate matter has been evaluated under the Particulate Matter Assessment.

I.1.1 Chemical Screening for the Chronic Air Quality Risk Assessment

The highest maximum annual predicted air concentrations for all the receptor locations were compared to air quality guidelines or objectives or screening levels (referred to herein as screening levels) derived for the protection of chronic inhalation to human health. The screening levels were obtained from:

- Northwest Territories Guideline for Ambient Air Quality Standards (GNWT 2011, internet site);
- United States Environmental Protection Agency (U.S. EPA) Regional Screening Levels (U.S. EPA 2012a, internet site);
- Ontario Ministry of Environment and Energy (OMoE 2012a, internet site; OMoE 2012b, internet site);
- Agency for Toxic Substances and Disease Registry (ATSDR 2012, internet site);
- Texas Commission on Environmental Quality Effects Screening Levels (TCEQ 2012, internet site);
- California Environmental Protection Agency Office of Environmental Health Hazard Assessment (CalEPA OEHHA 2012, internet site); and
- World Health Organization Air Quality Guidelines (WHO 2000, 2006).

The NWT air quality standards (GNWT 2011, internet site) were considered the priority source for selecting values. If a NWT value was available, it was used regardless of the availability of values from other sources. In the absence of a NWT value, the most conservative of the available health-based screening levels for a given chemical was used. Priority was given to screening levels that were health-based and had supporting documentation.

Risk levels for which the screening levels/guidelines were derived were standardized to Canadian federal acceptable risk levels. For non-carcinogens this involved adjusting to a hazard quotient of 0.2 and for carcinogens this involved adjusting to a risk level of $1x10^{-5}$ (i.e., one in one hundred thousand). The risk levels for which the screening levels/guidelines were developed are noted in the column headers for each regulatory agency. Further information on the approach used to develop the screening levels/guidelines/objectives for each of the agencies is provided below.

The available health-based screening criteria are presented in Table I-5.

Chemical screening was conducted by comparing the highest peak annual predicted concentrations in air to the selected air screening levels for all receptor locations for the Application Case (Table I-6).

Table I-5 Chronic Air Screening Levels and Guidelines Used in the Chemical Screening Process

Parameter	Carcinogen / Non-Carcinogen	Air Screening Levels and Guidelines [µg/m^3]										Toxicological Endpoints and Derivations
		NWT[1]	CCME NAAQO[2]			U.S. EPA RSL[3]	OMoE[4]	ATSDR[5]	California OEHHA[6,7]	WHO[8,9]	TCEQ[10]	
		Standard	Desirable	Acceptable	Tolerable							
Acid Gases												
Sulfur dioxide (SO_2)	Non-carcinogenic	30	30	60	-	-	11 (55)	-	-	10 (50)	-	NWT: annual arithmetic mean, adopted from NAAQO; CCME: Supporting documentation not available; OMoE: Threshold based on health and vegetation endpoints (supporting document not available); WHO: Threshold based on health endpoint (Epidemiological studies show that prevalence of respiratory symptoms, respiratory illness frequencies, differences in lung function and mortalities are associated with elevated levels of SO_2)
Nitrogen dioxide (NO_2)	Non-carcinogenic	60	60	100	-	-	-	-	-	8 (40)	-	NWT: annual arithmetic mean, adopted from NAAQO; CCME: Supporting documentation not available; WHO: Threshold based on health endpoint (Epidemiological studies show that reduced lung function growth in children is linked to elevated NO_2 concentrations within communities already at current North American and European urban ambient air levels).
Particulate Matter												
$PM_{2.5}$	Non-carcinogenic	-	-	-	-	-	-	-	-	10	-	WHO: Threshold represents the lowest level at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95% confidence in response to long term exposure.
PM_{10}	Non-carcinogenic	-	-	-	-	-	-	-	-	20	-	WHO: Threshold represents the lowest level at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95% confidence in response to long term exposure.
Total Suspended Particulate (TSP)	Non-carcinogenic	60	60	70	-	-	60	-	-	-	-	OMoE: Threshold (geometric mean) based on visibility; NWT: annual arithmetic mean, adopted from NAAQO.
Volatile Organic Compounds												
1,1,1-trichloroethane	Non-carcinogenic	-	-	-	-	1040 (5200)	-	-	-	-	-	U.S. EPA: PPRTV value, supporting documentation not available.
1,3-Butadiene	Non-carcinogenic	-	-	-	-	0.42 (2.1)		-	4 (20)	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Threshold based on an increased incidence of ovarian atrophy in mice in a 103 week study. The benchmark concentration (BMC_{05}) was 3.1 mg/m^3 (1.40 ppm) and the human equivalent concentration was 0.55 mg/m^3 (0.25 ppm). An uncertainty factor of 30 (3 for interspecies differences and 10 for intraspecies differences) was applied.
	Carcinogenic	-	-	-	-	0.81 (0.081)	20 (2)	-	0.059	-	-	U.S. EPA: Supporting documentation not available; OMoE: Threshold based on carcinogenic effects. Threshold based on a unit risk estimate from the State of Texas that was based on a cancer studies to occupationally-exposed workers. The inhalation unit risk estimates from Texas is 5x10-7 µg/m^3, based on a a risk level of 1 in 100,000 excess cancer risk, giving an annual threshold of 2 µg/m^3 for a risk level of one in one million, or 20 µg/m^3 for a risk level of one in one hundred thousand; Cal OEHHA: Calculated from lung alveolar and bronchiolar neoplasms in female mice using a linearized multistage procedure.
Acetaldehyde	Non-carcinogenic	-	-	-	-	1.88 (9.4)	-	-	28 (140)	-	-	Cal OEHHA: Threshold based on incidence of degeneration of nasal olfactory epithelium effects on rats (a sub-chronic study for 4 weeks, exposed for 6 hours/day, 5 days/week). The Ltime-adjusted exposure from a LOAEL of 720 mg/m^3 was 43.2 mg/m^3 and was used to calculate a chronic threshold. An uncertainty factor of 300 (3 for subchronic to chronic, 3 for interspecies differences, 3 for intra-individual differences, and 10 to account for for sensitive individuals).
	Carcinogenic	-	-	-	-	11 (1.1)	-	-	3.7	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Calculated from rat nasal tumor incidence data using a linearized, time-dependent multistage procedure.
Acetone	Non-carcinogenic	-	-	-	-	6400 (32000)	-	6,176 (13 ppm /30,880 µg/m^3)	-	-	-	U.S. EPA: Supporting documentation not available; ATSDR: Chronic MRL was derived based on a LOAEL of 1250 ppm for neurological effects in humans in a 6-week study. A uncertainty factor of 100 was applied (10 for use of a LOAEL and 10 for human variability). The MRL of 13 ppm was converted to µg/m^3 using MW = 58.08 g/mol.
Acrolein	Non-carcinogenic	-	-	-	-	0.0042 (0.021)	-	-	0.07 (0.35)	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Threshold based on rat studies where the NOAEL was 459 mg/m^3 (0.2 ppm) for respiratory lesions. The equivalent human exposure was 69 mg/m^3 (30 ppb). An uncertainty factor of 200 (3 for subchronic to chronic extrapolation, 2 for analogue chemical, 3 for interspecies differences, 10 for potential asthma exacerbation in children).
Aldehydes (surrogate: acetaldehyde)	Non-carcinogenic	-	-	-	-	1.88 (9.4)	-	-	28 (140)	-	-	Cal OEHHA: Threshold based on degeneration of olfactory epithelium in rats in a 4 week study. Benchmark concentration (BMC_{05}) was 178 mg/m^3 and the time-adjusted human equivalent concentration was 43.2 mg/m^3. An uncertainty factor of 300 (3 for extrapolating from subchronic to chronic, 3 for interspecies toxicodynamic differences, 3 for intraspecies toxicokinetic differences and 10 for intraspecies toxicodynamic differences) was applied.
	Carcinogenic	-	-	-	-	11 (1.1)	-	-	3.7	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Calculated from rat nasal tumor incidence data using a linearized, time-dependent multistage procedure.
Benzene	Non-carcinogenic	-	-	-	-	6.2 (31)	-	1.9 (0.003 ppm / 9.58 µg/m^3)	12 (60)	-	-	U.S. EPA: Threshold based on BMCL of 8.2 mg/m^3(Decreased lymphocyte count in 44 occupationally exposed workers, Conversion factors: MW = 78.11, BMCL = 7.2 ppm, 8-hour TWA, BMCL (mg/m^3) = 7.2 ppm x MW/24.45 = 23.0 mg/m^3. $BMCL_{ADJ}$ = 23.0 mg/m^3 x 10 m^3/20 m^3 x 5 days/7days = 8.2 mg/m^3; Uncertainty factor of 300 applied: 3 for extrapolation from a BMD, 10 for human variability, 3 for subchronic to chronic extrapolation, 3 for databased deficiencies); ATSDR: Threshold from BMD of 0.1 ppm (Significantly decreased counts of B-lymphocytes in 250 occupationally exposed workers, Exposure adjustment: 0.1 x 8hr/24hr x 6d/7d, divided by uncertainty factor of 10 for human variability); Cal OEHHA: Non-carcinogenic threshold based on hematological effects in occupationally exposed workers for an average of 7.4 years (32% had been exposed to >10 years). This study involved 303 male workers exposed to benzene for 1 to 21 years. Data from 1394 air samples indicated that 84% of all benzene samples were < 3.2 mg/m^3 (< 1 ppm) and the median (and NOAEL) was 1.7 mg/m^3 (0.52 ppm). An uncertainty factor of 10 (for intraspecies differences) was applied; WHO: Carcinogenic threshold based on the geometric mean of a range of estimates associated with an excess lifetime risk (RL=6 x 10^{-6})of leukaemia at an air concentration of 1 µg/m^3 (adjusted to RL = 10^{-5}).

Table I-5 Chronic Air Screening Levels and Guidelines Used in the Chemical Screening Process

Parameter	Carcinogen / Non-Carcinogen	Air Screening Levels and Guidelines [µg/m^3]										Toxicological Endpoints and Derivations
		NWT[1]	CCME NAAQO[2]			U.S. EPA RSL[3]	OMoE[4]	ATSDR[5]	California OEHHA[6,7]	WHO[8,9]	TCEQ[10]	
		Standard	Desirable	Acceptable	Tolerable							
Benzene (continued)	Carcinogenic	-	-	-	-	3.1 (0.31)	0.45 (added, changed to car)	-	0.34	1.7 (0.17)	-	OMoE: Threshold based on methods used by U.S. EPA and the EU that extrapolated occupational exposure concentrations to ambient air exposures based on mortality due to acute myeloid leukemia (AML); WHO: Threshold based an increase of mortality from leukaemia from workers occupationally exposed, tumours of the Zymbal gland, mammary gland and nasal cavity have been observed in mice and rats after inhalation exposure at 320 - 960 mg/m^3. Threshold based on the geometric mean of the range of estimates of the excess lifetime risk of leukaemia from the Pliofilm cohort study. Cal OEHHA: Based on an inhalation unit risk of 2.9x10-5 [µg/m3]$^{-1}$, which corresponds to the upper 95% confidence bound derived by U.S. EPA for human leukemia incidence data from 2 occupational studies.
C2 to C8 aliphatics (surrogate: cyclohexane)	Non-carcinogenic	-	-	-	-	1260 (6300)	-	-	-	-	-	U.S. EPA: Supporting documentation not available
C9 to C16 aliphatics (surrogate: decane)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	667 (1000)	TCEQ: Health endpoint, interim guideline. The TCEQ value for decane (C10 alkane) was selected in the absence of other values.
C16+ aliphatics (surrogate: nonacosane)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	6.67 (10)	TCEQ: Health endpoint, interim guideline. The TCEQ value for nonocosane (C17 and above alkane) was selected in the absence of other values.
C6 to C8 aromatics (surrogate: toluene)	Non-carcinogenic	-	-	-	-	1040 (5200)	-	60 (0.08 ppm, 300 µg/m^3)	60 (300)	-	-	U.S. EPA: Threshold from NOAEL of 34 ppm (Neurological effects in exposed rats, adjusted to continuous exposure, Uncertainty factor of 10 applied to account for potentially susceptible human subpopulations); ATSDR: Threshold from LOAEL of 35 ppm (Medical examination, interviews, color testing and blood testing of occupationally exposed workers; exposure adjustment: 35 ppm x 5d/7d x 8hr/24hr; Uncertainty factors: 10 for use of LOAEL, 10 for human variability). The MRL of 0.08 ppm was converted to µg/m3 using MW = 94.14 g/mol; Cal OEHHA: Threshold based on decreased brain (subcortical limbic area) weight and altered dopamine receptor (caudate-putamen) binding in rats in a 4 week study. The NOAEL was 154 mg/m3 (40 ppm) and the human equivalent concentration was 27 mg/m^3 (7 ppm). An uncertainty factor of 100 (10 for subchronic to chronic extrapolation and 10 for intraspecies differences) was used.
C9 to C16 aromatics (surrogate: ethylbenzene)	Non-carcinogenic	-	-	-	-	200 (1000)	-	52 (0.06 ppm, 260 µg/m^3)	400 (2,000)	-	-	ATSDR: Human equivalent concentrations (HECs) were calculated based on renal toxicity in rats. The lowest HEC value (17.45 ppm) was selected as the point of departure for the MRL. The HECMCA of 17.45 ppm was divided by an uncertainty factor of 300 (10 for the use of a LOAEL, 3 for extrapolation from animals to humans with dosimetric adjustments, and 10 for human variability) resulting in a chronic-duration inhalation MRL of 0.06 ppm, which was converted to µg/m^3 using MW = 106.17 g/mol. Cal OEHHA: Threshold based on nephrotoxicity and body weight reduction in rats and hyperplasia of the pituitary gland, liver cellular alterations and necrosis in mice in a 103 week study. The experimental NOAEL and human equivalent concentration was 56 mg/m^3 (13 ppm). An uncertainty factor of 30 (3 for interspecies differences and 10 for intraspecies differences) was applied.
	Carcinogenic	-	-	-	-	9.7 (0.97)	-	-	4	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Calculated from male rat renal tumor data, using the linearized multistage methodology with lifetime weighted average doses.
Ethylbenzene	Non-carcinogenic	-	-	-	-	200 (1000)	-	52 (0.06 ppm, 260 µg/m^3)	400 (2,000)	-	-	U.S. EPA: Threshold baesd on developmental toxicity in rat and rabbit studies. The NOAEL was 434 mg/m3 and the HEC NOAEL was also 434 mg/m3. An uncertainty factor of 300 (3 for interspecies conversion and 10 for the absense of multigenerational reproductive and chronic studies) was used; ATSDR: Human equivalent concentrations (HECs) were calculated based on renal toxicity in rats. The lowest HEC value (17.45 ppm) was selected as the point of departure for the MRL. The HECMCA of 17.45 ppm was divided by an uncertainty factor of 300 (10 for the use of a LOAEL, 3 for extrapolation from animals to humans with dosimetric adjustments, and 10 for human variability) resulting in a chronic-duration inhalation MRL of 0.06 ppm, which was converted to µg/m^3 using MW = 106.17 g/mol; Cal OEHHA: Threshold based on nephrotoxicity and body weight reduction in rats and hyperplasia of the pituitary gland, liver cellular alterations and necrosis in mice in a 103 week study. The experimental NOAEL and human equivalent concentration was 56 mg/m^3 (13 ppm). An uncertainty factor of 30 (3 for interspecies differences and 10 for intraspecies differences) was applied.
	Carcinogenic	-	-	-	-	9.7 (0.97)	-	-	4	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Calculated from male rat renal tumor data, using the linearized multistage methodology with lifetime weighted average doses.
Formaldehyde	Non-carcinogenic	-	-	-	-	2.0 (10)	-	1.97(0.008 ppm, 9.83 µg/m3)	1.8 (9)	-	-	U.S. EPA: Guideline derived from ATSDR MRL; ATSDR: Threshold MRL from LOAEL of 0.24 ppm (Histological changes (mild eye and upper respiratory tract irritation, mild damage to epithelium) in 70 workers in formaldehyde-producing chemical plant, 100 furniture factor workers, and 36 nonexposed office workers for 10.4 years; Uncertainty factors: 3 for use of LOAEL, 10 for human variability). The MRL was converted to µg/m3 using MW = 30.03 g/mol; Cal OEHHA: Non-carcinogenic threshold based on respiratory effects (nasal obstruction/discomfort, lower airway discomfort, eye irritation) in an occupational study of 66 workers. A time-adjusted exposure (also the NOAEL) of 0.09 mg/m^3 and an uncertainty factor of 10 (for potential asthma exacerbation in children) was applied. For carcinogenic: threshold based on rat nasal squamous carcinoma incidence data, linearized multistage procedure, with pharmacokinetic interpolation of molecular dosimetry data to the tumor incidence data.
	Carcinogenic	-	-	-	-	1.9 (0.19)	-	-	1.7	-	-	
Methyl ethyl ketone	Non-carcinogenic	-	-	-	-	1,040 (5,200)	-	-	-	-	-	U.S. EPA: Supporting documentation not available.
Toluene	Non-carcinogenic	-	-	-	-	1,040 (5,200)	-	60 (0.08 ppm, 300 µg/m^3)	60 (300)	-	-	U.S. EPA: Threshold from NOAEL of 34 ppm (Neurological effects in exposed rats, adjusted to continuous exposure, Uncertainty factor of 10 applied to account for potentially susceptible human subpopulations); ATSDR: Threshold from LOAEL of 35 ppm (Medical examination, interviews, color testing and blood testing of occupationally exposed workers; exposure adjustment: 35 ppm x 5d/7d x 8hr/24hr; Uncertainty factors: 10 for use of LOAEL, 10 for human variability). The MRL of 0.08 ppm was converted to µg/m^3 using MW = 94.14 g/mol; Cal OEHHA: Threshold based on decreased brain (subcortical limbic area) weight and altered dopamine receptor (caudate-putamen) binding in rats in a 4 week study. The NOAEL was 154 mg/m^3 (40 ppm) and the human equivalent concentration was 27 mg/m^3 (7 ppm). An uncertainty factor of 100 (10 for subchronic to chronic extrapolation and 10 for intraspecies differences) was used.

Table I-5 Chronic Air Screening Levels and Guidelines Used in the Chemical Screening Process

Parameter	Carcinogen / Non-Carcinogen	Air Screening Levels and Guidelines [µg/m^3]										Toxicological Endpoints and Derivations
		NWT[1]	CCME NAAQO[2]			U.S. EPA RSL[3]	OMoE[4]	ATSDR[5]	California OEHHA[6,7]	WHO[8,9]	TCEQ[10]	
		Standard	Desirable	Acceptable	Tolerable							
Trimethylbenzenes	Non-carcinogenic	-	-	-	-	1.04 (5.2), 1.46 (7.3)	-	-	-	-	125	U.S. EPA: PPRTV (supporting documentation not available) for 1,2,3-trimethylbenzene and 1,2,4-trimethylbenzene, respectively; TCEQ: Screening level based on health effects for Aromatic 100 as trimethylbenzenes.
Xylenes (total)	Non-carcinogenic	-	-	-	-	20 (100)	-	44 (0.05 ppm, 220 µg/m^3)	140 (700)	-	-	U.S. EPA: Threshold based on impaired motor coordination in rats. An experimental NOAEL of 217 mg/m^3 (50 ppm) was calculated and a HEC NOAEL was 30 mg/m3. An uncertainty factor of 300 (3 for interspecies differences, 10 for intraspecies differences, 3 for extrapolation from subchronic to chronic and 3 for uncertainties in the database) was applied; ATSDR: Neurological, uncertainty factor of 300. The MRL of 0.05 ppm was converted to µg/m^3 using MW=106.16. Cal OEHHA: Threshold based on eye irritation, sore throat, floating sensation and poor appetite in a occupational study for an average of 7 years. The human equivalent concentration (and LOAEL) was (22.1 mg/m^3) 5.1 ppm and an uncertainty factor of 30 (3 for LOAEL uncertainty and 10 for intraspecies differences) was applied.
Polycyclic Aromatic Hydrocarbons												
1-Methylnaphthalene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.8 (3)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
1-Methylphenanthrene (surrogate: phenanthrene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-Methylanthracene (surrogate: anthracene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-Methylfluorene (surrogate: fluorene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.67 (1.0)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-Methylnaphthalene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.8 (3)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-Methylphenanthrene (surrogate: phenanthrene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
2-Methylpyrene (surrogate: pyrene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
3-Methyldibenzothiophene (surrogate: dibenzothiophene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.5 (2.5)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
3-Methylphenanthrene (surrogate: phenanthrene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
4 + 9 Methylphenanthrene (surrogate: phenanthrene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold baesd on health effects (interim, supporting documentation not available).
4-Methyldibenzothiophene (surrogate: dibenzothiophene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.5 (2.5)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Acenaphthene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.07 (0.1)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Acenaphthylene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.07 (0.1)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Acephenanthrylene (surrogate: benzo(k)fluoranthene)		-	-	-	-	0.087 (0.0087)	-	-	0.091	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Anthracene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benz(a)anthracene	Carcinogenic	-	-	-	-	0.087 (0.0087)	-	-	0.091	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Benzo(a)fluorene (surrogate: 2,3-benzo(b)fluorene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benzo(a)pyrene	Carcinogenic	-	-	-	-	0.0087 (0.00087)	0.0001	-	0.0091	0.00012 TEQ (0.000012)	-	OMoE: Threshold based on carcinogenic potential endpoint that is based on WHO's evaluation of coke-oven worker epidemiological studies that derived an inhalation unit risk (IUR) value of 0.000087 ng/m^3 (BaP as a surrogate for total PAHs) equivalent to 0.01 ng/m^3 of BaP; U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available; WHO: Threshold is the unit risk for BaP (8.7 x 10^{-5}) caused by an air concentration of 1 µg/m^3 (adjusted to RL=10^{-5}). Threshold based on epidemiological data from studies in coke-oven workers.
Benzo(b)fluoranthene	Carcinogenic	-	-	-	-	0.087 (0.0087)	-	-	0.091	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Benzo(e)pyrene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benzo(g,h,i)fluoranthene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benzo(g,h,i)perylene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Benzo(k)fluoranthene	Carcinogenic	-	-	-	-	0.087 (0.0087)	-	-	0.091	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Chrysene	Carcinogenic	-	-	-	-	0.87 (0.087)	-	-	0.91	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Coronene (surrogate: benzo(g,h,i)perylene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Cyclopenta(c,d)pyrene	Carcinogenic	-	-	-	-	0.087 (0.0087)	-	-	0.091	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Dibenz(a,h)anthracene	Carcinogenic	-	-	-	-	0.008 (0.0008)	-	-	0.0083	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Dibenzothiophene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.5 (2.5)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).

Table I-5 Chronic Air Screening Levels and Guidelines Used in the Chemical Screening Process

Parameter	Carcinogen / Non-Carcinogen	Air Screening Levels and Guidelines [µg/m^3]										Toxicological Endpoints and Derivations
		NWT[1]	CCME NAAQO[2]			U.S. EPA RSL[3]	OMoE[4]	ATSDR[5]	California OEHHA[6,7]	WHO[8,9]	TCEQ[10]	
		Standard	Desirable	Acceptable	Tolerable							
Fluoranthene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Fluorene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.67 (1.0)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Indeno(1,2,3-cd)fluoranthene (surrogate: fluoranthene)	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Indeno(1,2,3-cd)pyrene	Carcinogenic	-	-	-	-	0.087 (0.0087)	-	-	0.091	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Indeno(1,2,3-W)pyrene (surrogate: indeno(1,2,3-cd)pyrene)	Carcinogenic	-	-	-	-	0.087 (0.0087)	-	-	0.091	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Naphthalene	Non-carcinogenic	-	-	-	-	0.62 (3.1)	-	0.73 (0.0007 ppm, 3.7 µg/m^3)	1.8 (9.0)	-	-	U.S. EPA: Threshold from $LOAEL_{HEC}$ of 9.3 mg/m^3 (Nasal effects (hyperplasia and metaplasia in respiratory and olfactory epithelium) in mice; Conversion Factors: adjusted to a continuous exposure (6/24 hr x 5/7 days), blood:gas (air) default ratio of 1 used, $LOAEL_{HEC}$ calculated for an extrarespiratory effect for a category 3 gas, a default b:a lambda for humans was 1.0, $LOAEL_{HEC}$ x [b:a lambda(animal)/b:a lambda(human)] = 9.3 mg/m3; Uncertainty factor of 3000: 10 for sensitive individuals, 10 for interspecies extrapolation, 10 for use of LOAEL, 3 for database deficiencies); Cal OEHHA: Non-carcinogenic threshold based on respiratory effects (nasal inflammation, olfactory epithelial metaplasia, respiratory epithelial hyperplasia) in mice. No supporting documentation for carcinogenic threshold; ATSDR: Threshold based on respiratory effects. The MRL of 0.0007 ppm was converted to µg/m^3 using MW = 128.17 g/mol.
	Carcinogenic	-	-	-	-	0.72 (0.072)	-	-	0.29	-	-	
Nitro-pyrene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
	Carcinogenic		-	-	-	0.22 (0.022)	-	-	0.091	-	-	U.S. EPA: as 4-nitropyrene, supporting documentation not available. Cal OEHHA: as 1- and 4-nitropyrene, supporting documentation not available.
Perylene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Phenanthrene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Picene (surrogate: dibenzo(a,h)anthracene)	Carcinogenic	-	-	-	-	0.008 (0.0008)	-	-	0.0083	-	-	U.S. EPA: Supporting documentation not available; Cal OEHHA: Supporting documentation not available.
Pyrene	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.03 (0.05)	TCEQ: Threshold based on health effects (interim, supporting documentation not available).
Metals & Inorganics												
Aluminum	Non-carcinogenic	-	-	-	-	1.04 (5.2)	-	-	-	-	-	U.S. EPA: PPRTV (supporting documentation not available).
Antimony	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.3 (0.5)	TCEQ: Health endpoint, interim guideline.
Arsenic	Non-carcinogenic	-	-	-	-	0.0032 (0.016)		-	0.003 (0.015)	-	-	U.S. EPA: Threshold based on inorganic arsenic (supporting documentation not available); Cal OEHHA: Threshold based on decrease in intellectual function and adverse effects on neurobehaviourl development in humans. An inhalation dose was estimated from an oral dose (drinking water) to give a value of 0.46 ug/m^3. An uncertainty factor of 30 (3 for estimating a LOAEL based on quantitative dose-response analysis and 10 for inter-individual variation) was used.
	Carcinogenic	-	-	-	-	0.0057 (0.00057)	-		0.003	0.0066 (0.00066)	-	U.S. EPA: Threshold based on lung cancer in occupational exposure studies for inorganic arsenic. An IUR of 4.3E-3 $(µg/m^3)^{-1}$ was used. WHO: Threshold based on lung tumours from studies in exposed human populations in Sweden and US.
Barium	Non-carcinogenic	-	-	-	-	0.104 (0.52)	-	-	-	-	-	U.S. EPA: Threshold based on HEAST (supporting documentation not available.
Beryllium	Non-carcinogenic	-	-	-	-	0.0042 (0.021)	-	-	0.0014 (0.007)	-	-	U.S. EPA: Threshold based on beryllium sensitization and progressing to chronic beryllium disease (CBD) in an occupational study for beryllium and compounds. The LOAEL (HEC) was 0.2 µg/m^3 and an uncertainty factor of 10 (3 for sensitive individuals in the population and 3 for database uncertainty) was applied; Cal OEHHA: Threshold based on beryllium-sensitized (chronic beryllium disease) workers in a beryllia ceramics plant. The LOAEL (median exposure of sensitized workers) was 0.55 µg/m^3 and the average experimental exposure was 0.2 µg/m^3. An uncertainty factor of 30 (10 for LOAEL uncertainty where there is a low incidence of disease but with serious, irreversible chronic effects and 3 for intraspecies differences).
	Carcinogenic	-	-	-	-	0.01 (0.001)	-		0.0042	-	-	U.S. EPA: Threshold based on lung cancer effects on an occupational study. An IUR of 2.4E-4 $(µg/m^3)^{-1}$ was used. Cal OEHHA: Based on a unit risk of 2.4E-03 $(µg/m^3)^{-1}$, based on lung cancer in beryllium processing workers.
Bismuth	Non-carcinogenic	-	-	-	-	-	-	-	-	-	3 (5)	TCEQ: Health endpoint, interim guideline.
Boron	Non-carcinogenic	-	-	-	-	4.2 (21)	-	-	-	-	-	U.S. EPA: Threshold based on HEAST (1997) (supporting documentation not available) for borates only.
Cadmium	Non-carcinogenic	-	-	-	-	0.0042 (0.021)°	0.005	0.002 (0.01)	0.004 (0.02) n	0.005	-	U.S. EPA: Threshold derived from Cal OEHHA; OMoE: Threshold based on health effects (supporting document not available); ATSDR: Threshold based on a urinary Cd level associated with 10% extra risk of low molecular weight proteinuria. The estimated air concentration of 0.1 µg/m^3 was divided by an uncertainty factor of 9 (3 for human variability and 3 as a modifying factor); Cal OEHHA: Threshold based on kidney and respiratory system effects in humans (occupational study with an average exposure duration of 28 years). A LOAEL of 0.5 µg/m^3 was derived and an uncertainty factor of 30 (3 for use of a subchronic study and 10 for intraspecies uncertainty) were used; WHO: Threshold based on data collected in industrial workers with lung cancer and renal alterations effects. The threshold is to prevent a further increase of cadmium in agricultural soils for future generations, which is likely to increase the dietary intake.
	Carcinogenic	-	-	-	-	0.014 (0.0014)	-		0.0024	-	-	U.S. EPA: Threshold based on lung, trachea and bronchus cancer deaths in occupation exposure studies. An IUR of 1.8E-3 $(µg/m^3)^{-1}$ was used.
Calcium	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.2 (2)	TCEQ: Health endpoint, interim guideline, as calcium oxide.

Table I-5 Chronic Air Screening Levels and Guidelines Used in the Chemical Screening Process

Parameter	Carcinogen / Non-Carcinogen	Air Screening Levels and Guidelines [µg/m^3]										Toxicological Endpoints and Derivations
		NWT[1]	CCME NAAQO[2]			U.S. EPA RSL[3]	OMoE[4]	ATSDR[5]	California OEHHA[6,7]	WHO[8,9]	TCEQ[10]	
		Standard	Desirable	Acceptable	Tolerable							
Chromium (as chromium [III])	Non-carcinogenic	-	-	-	-	-	-	-	-	-	(0.028) 0.14	TCEQ: For elemental, divalent and trivalent chromium compounds. A chronic ESL of 0.14 ug/m3 as Cr3+ based on an HQ = 1 was derived based on inhalation studies in rats exposed to chromium sulphate particulate. Critical effects were increased relative lung and trachea weight in male and female rats. An uncertainty factor of 1000 (3 for extrapolation from animals to humans, 10 to account for variability within the human population, 3 for an incomplete database, and a subchronic to chronic factor of 10) was applied to the PODHEC of 0.8086 mg/m3 based on the BMCL10 for increases in total lung and trachea weight relative to body weight in male rats.
Cobalt	Non-carcinogenic	-	-	-	-	0.00126 (0.0063)	-	0.02 (0.1)	-	-	-	U.S. EPA: PPRTV (supporting documentation not available); ATSDR: Respiratory endpoing, uncertainty factor of 10.
	Carcinogenic		-	-	-	0.0027 (0.00027)	-	-	-	-	-	U.S. EPA: PPRTV (supporting documentation not available).
Copper	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.6 (1)	TCEQ: Health endpoint, interim guideline.
Iron	Non-carcinogenic	-	-	-	-	-	-	-	-	-	3 (5)	TCEQ: Health endpoint, interim guideline; for iron as the metal parameter/oxide.
Lithium	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.6 (1)	TCEQ: Health endpoint, interim guideline, for Li as LiOH, LiO and lithium silicate.
Lead	Non-carcinogenic	-	-	-	-	0.03 (0.15)	0.2	-	-	0.1 (0.5)	-	U.S. EPA: documentation not available; OMoE: Threshold based on health effects (supporting document not available) over a 30-day period (arithmetic mean); WHO: Threshold based on having blood lead levels not exceed 100 µg/L to protect 98% of the population including children. Various international expert groups have determined that the earliest signs of adverse effects of lead in young children begin at 100 - 150 µg/L in blood. It also appears that 1 µg/m^3 of lead in air directly contributes approximately 19 µg/L of lead in blood in children and 16 µg/L in adults.
	Carcinogenic	-	-	-	-	-	-	-	0.833	-	-	Cal OEHHA: Threshold based on lead and compounds (supporting documentation not available); for lead as a metal paramter/oxide.
Magnesium	Non-carcinogenic	-	-	-	-	-	-	-	-	-	3 (5)	TCEQ: Health endpoint, interim guideline, as Mg except magnesium chromate.
Manganese	Non-carcinogenic	-	-	-	-	0.0104 (0.052)	-	0.008 (0.04)	0.018 (0.09)	0.03 (0.15)	-	U.S. EPA: Threshold based on impairment of neurobehaviourl function (occupational study). THe LOAEL was 0.15 mg/m^3 and the HEC LOAEL was 0.05 mg/m^3. An uncertainty factor of 1000 (10 to protect sensitive individuals, 10 for using a LOAEL and 10 for database limitations) was applied; ATSDR: Threshold based on abnormal performances in hand steadiness, eye-hand coordination and reaction time in an occupational study. The $BMCL_{10}$ of 74 µg/m^3 was adjusted by an uncertainty factor of 500 (10 for human variability, 10 for database deficiencies and 5 for increased susceptibility in children); Cal OEHHA: Threshold based on impairment of neurobehaviour function in humans (occupational study) for Mn and compounds. A $BMCL_{05}$ of 72 µg/m^3 was obtained and a time-adjusted exposure of 26 µg/m^3 was calculated. An uncertainty factor of 300 (3 for subchronic to chronic conversion, 100 for intraspecies differences (10 for adults to children and 10 for the more sensitive developing brains of newborns and infant children) was used; WHO: Threshold based on neurotoxic effects observed in occupationally exposed workers and an estimated NOAEL of 30 µg/m^3 was obtained. The threshold was derived by dividing by a factor of 4.2 for continuous exposure and an uncertainty factor of 50 (10 for interindividual variation and 5 for developmental effects in younger children).
Mercury	Non-carcinogenic	-	-	-	-	0.062 (0.31)	-	0.04 (0.2)	0.006 (0.03)	1	-	U.S. EPA: Threshold based on hand tremors, increases in memory disturbance, slight subjective and objective evidence of autonomic dysfunction in occupational studies. A LOAEL of 0.025 mg/m^3 was calculated and adjusted to a LOAEL of 0.009 mg/m^3. An uncertainty factor of 30 (10 to protect sensitive individuals and 3 for a lack of a database) was used; ATSDR: Threshold based on neurological effects (occupational study, exposed for an average of 15.3 years), uncertainty factor of 30; Cal OEHHA: Threshold based on nervous system effects in humans as Hg and inorganic compounds; WHO: Threshold based on the LOAELs for Hg vapour (15 - 30 µg/m^3, tremors, renal tubular effects, and changes in plasma enzymes) and applying an uncertainty factor of 20 (10 for uncertainty in variable sensitivities in higher risk populations and 2 for extrapolating from LOAEL to NOAEL). Since cationic inorganic Hg is retained only half as much as the vapour, the guideline also protects against mile renal effects caused by cationic inorganic Hg; Cal OEHHA: Threshold based on neurotoxicity as measured by intentional tremor, memory and sleep disturbances, decreased performance on neurobehaviour tests and decreased EEG activity in occupational studies. Humans were exposed to Hg for 8 hours/day, 5 days/week for a long-term exposure. A LOAEL of 0.025 mg/m^3 (time adjusted value is 0.0009 mg/m^3) was calculated and an uncertainty factor of 300 (10 for neurotoxicity being a moderate to severe effect, 3 to reflect interindividual variability and 10 for the higher susceptibility of the developing nervous system) were used.
Molybdenum	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.8 (3)	TCEQ: Health endpoint, interim guideline.
Nickel	Non-carcinogenic	-	-	-	-	0.0104 (0.052), 0.0188 (0.094)	0.02	0.018 (0.09)	0.0028 (0.014)	-	-	U.S. EPA: Threshold of 0.0104 is for nickel refinery dust and is based on Cal OEHHA; Threshold of 0.0188 is for nickel soluble salts and is based on ATSDR; OMoE: Threshold for nickel in PM10, based on carcinogenic and non-carcinogenic effects (supporting document available). Based on CSTEE/EU (Scientific Committee for Toxicity, Ecotoxicity and Environment) derivation of non-carcinogenic effects of NiSO4, OMoE picked the lower end of the 10-50 ng/m^3 range; ATSDR: Threshold based on chronic active lung inflammation and bronchialization in rats exposed to nickel sulfate (6 hours/day, 5 days/week for 2 years). The NOAEL was 0.06 mg/m^3 (human equivalent concentration of 0.0027 mg/m^3) and an uncertainty factor of 30 (3 for animal to human extrapolaiton and 10 for human variability) was applied. Respiratory endpoint, uncertainty factor of 30; Cal OEHHA: Threshold based on respiratory system and hematopoietic system effects in rats.
	Carcinogenic	-	-	-	-	0.094 (0.0094)			0.0385	0.025 (0.0025)	-	U.S. EPA: Threshold for nickel soluble salts based on Cal OEHHA; Cal OEHHA: For nickel and nickel compounds, based on a unit risk of 2.6E-04 (ug/m3)-1, based on lung cancer incidence data in humans. WHO: Threshold based on studies in occupationally exposed human populations (increased risk of lung and nasal cancers). An incremental risk of 3.8x10^{-4} is given for 1 µg/m^3 of nickel in the air.

Table I-5 Chronic Air Screening Levels and Guidelines Used in the Chemical Screening Process

Parameter	Carcinogen / Non-Carcinogen	Air Screening Levels and Guidelines [µg/m^3]										Toxicological Endpoints and Derivations
		NWT[1]	CCME NAAQO[2]			U.S. EPA RSL[3]	OMoE[4]	ATSDR[5]	California OEHHA[6,7]	WHO[8,9]	TCEQ[10]	
		Standard	Desirable	Acceptable	Tolerable							
Phosphorus	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.06 (0.1)	TCEQ: Health endpoint, interim guideline, for yellow phosphorus.
Potassium	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.2 (2)	TCEQ: Health endpoint, interim guideline.
Selenium	Non-carcinogenic	-	-	-	-	4.2 (21)	-	-	4 (20)	-	-	U.S. EPA: Threshold based on Cal OEHHA; Cal OEHHA: Threshold based on alimentary system, cardivascular system and nervous system effects in humans for selenium and compounds except for hydrogen selenide.
Silver	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.006 (0.01)	TCEQ: Health endpoint, interim guideline.
Sodium	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.2 (2)	TCEQ: Health endpoint, interim guideline, as NaOH.
Strontium	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.2 (2)	TCEQ: Health endpoint, interim guideline, as strontium and compounds.
Thallium	Non-carcinogenic	-	-	-	-	-	-	-	-	-	0.06 (0.1)	TCEQ: Health endpoint, interim guideline, as thallium and compounds.
Tin	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.2 (2)	TCEQ: Health endpoint, interim guideline, as tin, its compounds and inorganic form.
Titanium	Non-carcinogenic	-	-	-	-	0.02 (0.1)	-	0.02 (0.1)	-	-	-	U.S. EPA: Threshold based on ATSDR (as titanium tetraoxide); ATSDR: Threshold based on its ability to cause irregular breathing and lung noises in rats, along with rhinitis, tracheitis and alveolar hyperplasia. An uncertainty factor of 90 was applied as titanium tetraoxide.
Tungsten	Non-carcinogenic	-	-	-	-	-	-	-	-	-	3 (5)	TCEQ: Health endpoint, interim guideline, for insoluble tungsten
Uranium	Non-carcinogenic	-	-	-	-	-	0.03	0.16 (0.8)	-	-	-	OMoE: Threshold for uranium in PM10, based on kidney toxicity (based on U accumulation in the kidney over a 50 year exposure period that is considered to be protective for long-term continuous inhalation exposure); ATSDR: Threshold for insoluble U compounds based on respiratory endpoint, uncertainty factor of 1000.
Vanadium	Non-carcinogenic	-	-	-	-	0.00146 (0.0073)	-	-	-	-	-	U.S. EPA: PPRTV (supporting documentation not available) as vanadium pentoxide.
	Carcinogenic		-	-	-	0.0029 (0.00029)	-	-	-	-	-	U.S. EPA: PPRTV (supporting documentation not available) as vanadium pentoxide.
Zinc	Non-carcinogenic	-	-	-	-	-	-	-	-	-	1.2 (2)	TCEQ: Health endpoint, interim guideline, as zinc and compounds.
Zirconium	Non-carcinogenic	-	-	-	-	-	-	-	-	-	3 (5)	TCEQ: Health endpoint, interim guideline, as Zr and compounds.
Dioxins/Furans												
Total dioxins / furans	Non-carcinogenic	-	-	-	-	-	-	-	8E-6 (4E-5)	-	-	Cal OEHHA: Threshold for dioxins and furans (treated as 2,3,7,8-tetrachlorodibenzo-*p*-dioxin) based on mortality, alimentary, reproductive, endocrine, respiratory and development endpoints in a 2 year rat study. The observed NOAEL was 0.001 µg/kg/day in diet, an uncertainty factor of 100 (10 for interspecies differences and 10 for intraspecies differences) and an oral route to inhalation route extrapolation (3500 µg/m^3 per mg/kg/day) was applied.

1. Northwest Territories Department of Environment and Natural Resources – Ambient Air Quality Standards (NWT 2011).
2. Guidelines from the Canadian Council of Ministers of the Environment (CCME), Guidance Document on Achievement Determination: Canada-wide Standards for Particulate Matter and Ozone (CCME 2007).
3. Guidelines from US Environmental Protection Agency (U.S. EPA) Regional Screening Levels (RSL) for Region 9 - Pacifi Southwest (April 2012).
4. Ontario Ministry of the Environment (OMoE 2012a) Ambient Air Quality Criteria and OMoE Summary of Standards and Guidelines to support Ontario Regulation 419: Air Pollution (OMoE 2012b).
5. Agency of Toxic Substances and Disease Registry (ATSDR), Minimum Risk Levels (MRLs) (ATSDR 2012).
6. California Office of Environmental Health Hazard Assessment (CalEPA OEHHA 2012). Chronic Reference Exposure Levels. An ILCR of 10^{-5} was assumed in calculating thresholds from Inhalation Unit Risk factors.
7. CalEPA OEHHA 2012 - Toxicity Criteria Database. Chemical-specific inhalation unit risks used to derive carcinogenic thresholds.
8. World Health Organization (WHO), WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide (WHO 2006)
9. WHO Air Quality Guidelines for Europe, Second Edition. (WHO 2000).
10. Texas Commission on Environmental Quality (TCEQ 2012). Effects Screening Levels.

Notes: All values are in µg/m^3, unless otherwise noted.
Shaded thresholds were used in the risk assessment.
- = Value not available.
* Value converted from parts-per-million (ppm) to micrograms per meter cubed (µg/m^3) using the molecular weight (ML) of the specified compound, in the following formula:Y mg/m^3=(X ppm)(molecular weight)/24.45, (assumptions: 25 ºC and 1 atm).
BMCL10 - Benchmark Dose 95% lower confidence limit, determined using the EPA Benchmark Dose Software ,CNS - Central Nervous System, HEAST - Health Effects Assessment Summary Tables (U.S. EPA, 1997 Update), HQ - Hazard Quotient, IUR - Inhalation Unit Risk (µg/m3)-1, LOAEL - Lowest Observed Adverse Effects Level , NOAEL - No Observed Adverse Effects Level, NAAQO - Canadian National Ambient Air Quality Objectives, MRL - Minimal Risk Level, MW - Molecular Weight (grams per moles), REL - Reference Exposure Level, PAH - Polycyclic Aromatic Hydrocarbons, PEF - Potency Equivalent Factor, PPRTV - Provisional Peer-Reviewed Toxicity Value, PM - Particulate Matter, ppm - Parts per million, RfC - Reference Concentration (µg/m3), TEQ - Toxicity Equivalent Quotient, WHO - World Health Organization.
The screening levels derived by OMoE, U.S. EPA, ATSDR, California OEHHA, and WHO are based on an HQ=1.0 for non-carcinogens, and an RL=10^{-6} for carcinogens. These guidelines have been adjusted to an HQ=0.2 and RL=10^{-5}, for comparison to Canadian federal guidelines. Original values are listed in brackets beside the adjusted value.
The screening levels derived by TCEQ are based on an HQ=0.3 for non-carcinogens (unless otherwise noted), and an RL=10^{-5} for carcinogens. Guidelines for non-carcinogens have been adjusted to a HQ=0.2, for comparison to Canadian guidelines. Values for carcinogens remain unchanged. Original values are listed in brackets beside the adjusted value.

Table I-6 Chemicals of Potential Concern Screening Based on Predicted Peak Annual Concentrations at Each Receptor Location

Parameter	Air Threshold (Non-carcinogenic)	Air Threshold (Carcinogenic)	Predicted Air Concentrations at the Following Locations:						Predicted Maximum Concentration (All Locations)	Measured Maximum Baseline Concentration	Measured Maximum Baseline Concentration + 10%	Above Air Threshold?	Above Measured Maximum Baseline Concentration + 10%?	Retained as a COC?
			Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary						
Acid Gases														
Sulfur dioxide (SO_2)	30	-	2.6	2.6	2.6	3.1	2.7	4.8	4.8	2.6	2.9	No	Yes	No
Nitrogen dioxide (NO_2)	60	-	5.9	6.0	5.9	55.2	9.6	**62.1**	**62.1**	5.9	6.5	Yes	Yes	**Yes** [1]
Particulate Matter														
$PM_{2.5}$	10	-	1.9	1.9	1.9	6.4	2.3	**17.8**	**17.8**	1.9	2.1	Yes	Yes	**Yes**
PM_{10}	20		3.0	3.0	3.0	13.9	4.1	**92.4**	**92.4**	3.0	3.3	Yes	Yes	**Yes**
Total Suspended Particulate (TSP)	60	-	7.1	7.1	7.1	29.0	8.4	**278**	**278**	7.1	7.8	Yes	Yes	**Yes**
Volatile Organic Compounds														
1,1,1-Trichloroethane	1040	-	0.000000050	0.000000057	0.000000036	0.000005680	0.000000079	0.000005150	0.000005680	0.000000055	0.000000061	No	Yes	No
1,3-Butadiene	0.42	0.059	0.0000162	0.000018	0.0000131	0.00294	0.000223	0.00889	0.00889	0.0000146	0.000016	No	Yes	No
Acetaldehyde	1.88	3.7	0.00218	0.00242	0.00176	0.397	0.0301	1.2	1.2	0.00197	0.00216	No	Yes	No
Acetone	6176	-	0.00115	0.00128	0.000929	0.209	0.0158	0.631	0.631	0.00103	0.00114	No	Yes	No
Acrolein	0.0042	-	0.000178	0.000197	0.000144	**0.0323**	0.00245	**0.0975**	**0.0975**	0.00016	0.000176	Yes	Yes	**Yes** [1]
Aldehydes (surrogate: acetaldehyde)	1.88	3.7	0.00303	0.00336	0.00245	0.551	0.0418	1.66	1.66	0.00273	0.003	No	Yes	No
Benzene	1.9	0.34	0.000144	0.00016	0.000117	0.0267	0.002	0.0787	0.0787	0.00013	0.000143	No	Yes	No
C2 to C8 aliphatics (surrogate: cyclohexane)	1260	-	0.00178	0.00198	0.00144	0.324	0.0246	0.978	0.978	0.0016	0.00177	No	Yes	No
C9 to C16 aliphatics (surrogate: decane)	667	-	0.000233	0.000259	0.000188	0.0423	0.00321	0.128	0.128	0.00021	0.000231	No	Yes	No
C16+ aliphatics (surrogate: decane)	6.67	-	0.000183	0.000203	0.000148	0.0333	0.00252	0.1	0.1	0.000165	0.000181	No	Yes	No
C6 to C8 aromatics (surrogate: toluene)	60	-	0.000267	0.000296	0.000215	0.0484	0.00367	0.146	0.146	0.00024	0.000264	No	Yes	No
C9 to C16 aromatics (surrogate ethylbenzene)	52	4	0.000263	0.000292	0.000213	0.0478	0.00363	0.144	0.144	0.000237	0.00026	No	Yes	No
Ethylbenzene	52	4	0.0000248	0.0000275	0.00002	0.00459	0.000342	0.0135	0.0135	0.0000222	0.0000245	No	Yes	No
Formaldehyde	1.8	1.7	0.00117	0.0013	0.000946	0.213	0.0161	0.64	0.64	0.00106	0.00116	No	Yes	No
Methyl ethyl ketone	1040	-	0.000392	0.000435	0.000317	0.0712	0.0054	0.215	0.215	0.000353	0.000388	No	Yes	No
Toluene	60	-	0.000212	0.000236	0.000171	0.0396	0.00292	0.114	0.114	0.00019	0.000209	No	Yes	No
Trimethylbenzenes	1.04	-	0.0000596	0.0000661	0.0000481	0.0108	0.000821	0.0327	0.0327	0.0000536	0.000059	No	Yes	No
Xylene (total)	20	-	0.000166	0.000184	0.000134	0.0305	0.00229	0.0907	0.0907	0.000149	0.000164	No	Yes	No
Dioxins/Furans (µg TEQ/m^3)	8.00E-06	-	6.44E-14	7.52E-14	4.95E-14	1.18E-11	4.59E-13	3.91E-11	3.91E-11	6.36E-14	7.00E-14	No	Yes	No
Polyaromatic Hydrocarbons														
1-Methylnaphthalene	-	1.8	0.0000023	0.00000275	0.00000191	0.00127	0.000122	0.00574	0.00574	0.0000012	0.00000132	No	Yes	No
1-Methylphenanthrene (surrogate: phenanthrene)	-	0.03	0.000000103	0.000000124	8.58E-08	0.0000569	0.00000548	0.000258	0.000258	5.38E-08	5.92E-08	No	Yes	No
2-Methylanthracene (surrogate: anthracene)	-	0.03	6.33E-08	7.57E-08	5.25E-08	0.0000348	0.00000335	0.000158	0.000158	3.29E-08	3.62E-08	No	Yes	No
2-Methylfluorene (surrogate: fluorene)	-	0.67	2.13E-09	2.55E-09	1.77E-09	0.00000117	0.000000113	0.00000533	0.00000533	1.11E-09	1.22E-09	No	Yes	No
2-Methylnaphthalene	-	1.8	0.00000372	0.00000445	0.00000308	0.00205	0.000197	0.00928	0.00928	0.00000193	0.00000213	No	Yes	No
2-Methylphenanthrene (surrogate: phenanthrene)	-	0.03	0.000000255	0.000000306	0.000000212	0.000141	0.0000135	0.000638	0.000638	0.000000133	0.000000146	No	Yes	No
2-Methylpyrene (surrogate: pyrene)	-	0.03	1.89E-08	2.27E-08	1.57E-08	0.0000104	0.000001	0.0000473	0.0000473	9.85E-09	1.08E-08	No	Yes	No

Table I-6 Chemicals of Potential Concern Screening Based on Predicted Peak Annual Concentrations at Each Receptor Location (continued)

Parameter	Air Threshold (Non-carcinogenic)	Air Threshold (Carcinogenic)	Predicted Air Concentrations at the Following Locations:						Predicted Maximum Concentration (All Locations)	Measured Maximum Baseline Concentration	Measured Maximum Baseline Concentration + 10%	Above Air Threshold?	Above Measured Maximum Baseline Concentration + 10%?	Retained as a COC?
			Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary						
3-Methyldibenzothiophene (surrogate: dibenzothiophene)	-	1.5	3.91E-09	4.68E-09	3.24E-09	0.00000215	0.000000207	0.00000977	0.00000977	2.03E-09	2.24E-09	No	Yes	No
3-Methylphenanthrene (surrogate: phenanthrene)	-	0.03	0.000000184	0.000000221	0.000000153	0.000101	0.00000977	0.00046	0.00046	9.59E-08	0.000000105	No	Yes	No
4 + 9 Methylphenanthrene (surrogate: dibenzothiophene)	-	0.03	0.000000139	0.000000167	0.000000116	0.0000767	0.00000738	0.000348	0.000348	7.25E-08	7.97E-08	No	Yes	No
4-Methyldibenzothiophene (surrogate: dibenzothiophene)	-	1.5	2.49E-09	2.98E-09	2.07E-09	0.00000137	0.000000132	0.00000622	0.00000622	1.3E-09	1.42E-09	No	Yes	No
Acenaphthene	-	0.07	0.000000122	0.000000146	0.000000101	0.0000651	0.00000623	0.000293	0.000293	0.000000066	7.26E-08	No	Yes	No
Acenaphthylene	-	0.07	0.000000426	0.00000051	0.000000354	0.000235	0.0000226	0.00106	0.00106	0.000000222	0.000000244	No	Yes	No
Acephenanthrylene (surrogate: benzo(k)fluoranthene)	-	0.087	0.000000073	8.73E-08	6.06E-08	0.0000402	0.00000387	0.000182	0.000182	0.000000038	4.18E-08	No	Yes	No
Anthracene	-	0.03	7.63E-08	9.13E-08	6.33E-08	0.0000419	0.00000403	0.00019	0.00019	3.98E-08	4.38E-08	No	Yes	No
Benz(a)anthracene	-	0.087	0.000000019	2.27E-08	1.56E-08	0.0000101	0.000000962	0.0000453	0.0000453	1.04E-08	1.14E-08	No	Yes	No
Benzo(a)fluorene (surrogate: 2,3-benzo(b)fluorene)	-	0.03	0.000000023	2.75E-08	0.000000019	0.0000126	0.00000122	0.0000573	0.0000573	1.19E-08	1.31E-08	No	Yes	No
Benzo(a)pyrene	-	0.0001	0.00000001	0.000000012	8.33E-09	0.00000553	0.000000532	0.0000251	0.0000251	5.22E-09	5.75E-09	No	Yes	No
Benzo(b)fluoranthene	-	0.087	8.45E-08	0.000000101	7.01E-08	0.0000464	0.00000446	0.00021	0.00021	4.42E-08	4.86E-08	No	Yes	No
Benzo(e)pyrene	-	0.03	1.42E-09	1.7E-09	1.18E-09	0.000000782	7.53E-08	0.00000355	0.00000355	7.39E-10	8.13E-10	No	Yes	No
Benzo(g,h,i)fluoranthene (surrogate: benzo(g,h,i)perylene)	-	0.03	3.54E-08	4.24E-08	2.94E-08	0.0000195	0.00000188	0.0000884	0.0000884	1.84E-08	2.03E-08	No	Yes	No
Benzo(g,h,i)perylene	-	0.03	2.35E-08	2.81E-08	1.94E-08	0.0000127	0.00000122	0.0000574	0.0000574	1.25E-08	1.37E-08	No	Yes	No
Benzo(k)fluoranthene	-	0.087	9.58E-09	1.15E-08	7.95E-09	0.00000527	0.000000508	0.0000239	0.0000239	4.98E-09	5.48E-09	No	Yes	No
Chrysene	-	0.87	2.09E-08	0.000000025	1.73E-08	0.0000113	0.00000108	0.0000509	0.0000509	1.12E-08	1.23E-08	No	Yes	No
Coronene (surrogate: benzo(g,h,i)perylene)	-	0.03	1.78E-10	2.13E-10	1.47E-10	9.78E-08	9.42E-09	0.000000444	0.000000444	9.24E-11	1.02E-10	No	Yes	No
Cyclopenta(c,d)pyrene	-	0.087	1.25E-08	0.000000015	1.04E-08	0.0000069	0.000000664	0.0000313	0.0000313	6.52E-09	7.17E-09	No	Yes	No
Dibenz(a,h)anthracene	-	0.008	2.67E-08	3.19E-08	2.21E-08	0.0000145	0.0000014	0.0000658	0.0000658	1.41E-08	1.55E-08	No	Yes	No
Dibenzothiophene	-	1.5	1.84E-09	2.22E-09	1.52E-09	0.00000103	8.65E-08	0.00000381	0.00000381	9.97E-10	1.1E-09	No	Yes	No
Fluoranthene	-	0.03	0.000000324	0.000000387	0.000000268	0.000178	0.0000171	0.000805	0.000805	0.000000169	0.000000186	No	Yes	No
Fluorene	-	0.67	0.000000608	0.000000727	0.000000504	0.000334	0.0000322	0.00152	0.00152	0.000000317	0.000000349	No	Yes	No
Indeno(1,2,3-cd)fluoranthene (surrogate: fluoranthene)	-	0.03	8.89E-10	1.06E-09	7.37E-10	0.000000489	4.71E-08	0.00000222	0.00000222	4.62E-10	5.08E-10	No	Yes	No
Indeno(1,2,3-cd)pyrene	-	0.087	4.49E-10	5.15E-10	3.24E-10	5.15E-08	7.13E-10	4.67E-08	5.15E-08	5.02E-10	5.52E-10	No	Yes	No
Indeno(1,2,3-W)pyrene (surrogate: indeno(1,2,3-cd)pyrene)	-	0.087	0.000000016	1.91E-08	1.32E-08	0.00000879	0.000000846	0.0000399	0.0000399	8.31E-09	9.14E-09	No	Yes	No
Naphthalene	0.62	0.29	0.00000934	0.0000112	0.00000772	0.00504	0.000483	0.0227	0.0227	0.000005	0.0000055	No	Yes	No
Nitro-pyrene	0.03	0.091	1.42E-08	0.000000017	1.18E-08	0.00000782	0.000000753	0.0000355	0.0000355	7.39E-09	8.13E-09	No	Yes	No
Perylene	-	0.03	1.78E-10	2.13E-10	1.47E-10	9.78E-08	9.42E-09	0.000000444	0.000000444	9.24E-11	1.02E-10	No	Yes	No
Phenanthrene	-	0.03	0.00000057	0.000000682	0.000000473	0.000313	0.00003	0.00141	0.00141	0.000000298	0.000000328	No	Yes	No
Picene (surrogate: dibenzo(a,h)anthracene)	-	0.008	1.78E-10	2.13E-10	1.47E-10	9.78E-08	9.42E-09	0.000000444	0.000000444	9.24E-11	1.02E-10	No	Yes	No
Pyrene	-	0.03	0.000000439	0.000000525	0.000000364	0.000241	0.0000232	0.00109	0.00109	0.000000229	0.000000252	No	Yes	No
Metals and Inorganics														
Aluminum	1.04	-	0.000215	0.000266	0.000178	0.343	0.0255	**2.42**	**2.42**	0.0000798	0.0000878	Yes	Yes	**Yes** [3]
Antimony	0.03	-	6.56E-09	8.28E-09	5.55E-09	0.00000984	0.000000871	0.0000889	0.0000889	1.92E-09	2.11E-09	No	Yes	No

Table I-6 Chemicals of Potential Concern Screening Based on Predicted Peak Annual Concentrations at Each Receptor Location (continued)

Parameter	Air Threshold (Non-carcinogenic)	Air Threshold (Carcinogenic)	Predicted Air Concentrations at the Following Locations:						Predicted Maximum Concentration (All Locations)	Measured Maximum Baseline Concentration	Measured Maximum Baseline Concentration + 10%	Above Air Threshold?	Above Measured Maximum Baseline Concentration + 10%?	Retained as a COC?
			Warburton Bay Lodge	Warburton Bay Fishing Lodge	MacKay Lake Lodge	Employee Camp	Proposed National Park Boundary	Project Boundary						
Arsenic	0.003	0.003	0.00000014	0.000000156	0.000000106	0.0000591	0.00000308	0.000246	0.000246	0.000000131	0.000000145	No	Yes	No
Barium	0.104	-	0.00000477	0.00000586	0.00000392	0.00786	0.000541	0.0495	0.0495	0.00000191	0.0000021	No	Yes	No
Beryllium	0.0014	0.0042	5.75E-08	6.23E-08	4.23E-08	0.0000101	0.000000113	0.00000912	0.0000101	6.15E-08	6.76E-08	No	Yes	No
Bismuth	3	-	2.1E-09	2.64E-09	1.77E-09	0.0000032	0.000000266	0.000026	0.000026	6.71E-10	7.38E-10	No	Yes	No
Boron	4.2	-	0.000000309	0.000000362	0.000000242	0.000605	0.0000267	0.002	0.002	0.000000182	0.0000002	No	Yes	No
Cadmium	0.001	0.0024	0.00000182	0.00000202	0.00000153	**0.0018**	0.0000939	***0.00684***	***0.00684***	0.00000131	0.00000145	Yes	Yes	**Yes** [3]
Chromium (as chromium [III])	0.028	-	0.0000026	0.00000312	0.00000212	0.00379	0.000242	0.0212	0.0212	0.00000133	0.00000146	No	Yes	No
Cobalt	0.00126	0.0027	0.000000638	0.000000748	0.000000529	0.000858	0.0000541	***0.00473***	***0.00473***	0.000000343	0.000000377	Yes	Yes	**Yes** [3]
Copper	0.6	-	0.000000675	0.000000779	0.000000551	0.000738	0.0000466	0.00408	0.00408	0.000000427	0.00000047	No	Yes	No
Iron	3	-	0.000404	0.0005	0.000335	0.649	0.0479	**4.53**	**4.53**	0.000151	0.000166	Yes	Yes	**Yes** [3]
Lead	0.03	0.833	0.000000557	0.000000629	0.000000452	0.000441	0.0000289	0.00262	0.00262	0.000000406	0.000000446	No	Yes	No
Lithium	0.6	-	1.58E-08	0.000000022	1.35E-08	0.0000477	0.00000296	0.000347	0.000347	0	0	No	Yes	No
Manganese	0.008	-	0.00000625	0.00000767	0.00000516	**0.00978**	0.000703	**0.066**	**0.066**	0.00000254	0.00000279	Yes	Yes	**Yes** [3]
Mercury	0.006	-	0.000000416	0.00000046	0.000000313	0.000122	0.00000408	0.000373	0.000373	0.000000416	0.000000458	No	Yes	No
Molybdenum	1.8	-	6.02E-08	7.62E-08	5.11E-08	0.0000897	0.00000809	0.000831	0.000831	1.71E-08	1.89E-08	No	Yes	No
Nickel	0.0028	0.025	0.00000445	0.0000053	0.00000353	**0.00736**	0.000394	***0.031***	***0.031***	0.00000243	0.00000267	Yes	Yes	**Yes** [3]
Selenium	4	-	0.000000298	0.000000324	0.00000022	0.0000663	0.00000182	0.000133	0.000133	0.000000311	0.000000342	No	Yes	No
Silver	0.006	-	0.00000027	0.000000299	0.00000023	0.000292	0.0000155	0.00116	0.00116	0.000000184	0.000000202	No	Yes	No
Sodium	1.2	-	0.00000861	0.0000105	0.00000701	0.0147	0.000928	0.0818	0.0818	0.00000376	0.00000414	No	Yes	No
Strontium	1.2	-	0.00000155	0.00000186	0.00000124	0.00278	0.00015	0.012	0.012	0.000000774	0.000000851	No	Yes	No
Thallium	0.06	-	3.12E-09	3.93E-09	2.63E-09	0.00000476	0.000000405	0.0000407	0.0000407	9.69E-10	1.07E-09	No	Yes	No
Tin	1.2	-	1.69E-10	2.37E-10	1.45E-10	0.000000513	3.18E-08	0.00000373	0.00000373	0	0	No	Yes	No
Titanium	0.02	-	0.000018	0.0000227	0.0000152	**0.0275**	0.00233	**0.233**	**0.233**	0.00000564	0.0000062	Yes	Yes	**Yes** [3]
Tungsten	3	-	2.06E-09	2.59E-09	1.73E-09	0.00000319	0.000000262	0.000026	0.000026	6.71E-10	7.38E-10	No	Yes	No
Uranium	0.006	-	1.87E-08	2.34E-08	1.57E-08	0.0000294	0.00000233	0.000228	0.000228	6.38E-09	7.02E-09	No	Yes	No
Vanadium	0.00146	0.0029	0.000000642	0.000000799	0.000000535	0.00101	0.0000788	***0.00764***	***0.00764***	0.000000223	0.000000245	Yes	Yes	**Yes** [3]
Zinc	1.2	-	0.00000224	0.00000251	0.00000189	0.00237	0.000132	0.0109	0.0109	0.00000149	0.00000164	No	Yes	No
Zirconium	1.2	-	7.11E-09	9.93E-09	6.11E-09	0.0000215	0.00000133	0.000157	0.000157	0	0	No	Yes	No

1 – Parameter evaluated under the chronic air quality risk assessment.

2 – Parameter evaluated under the particulate matter assessment.

3 – Parameter evaluated under the multimedia assessment.

Notes: **Value exceeds non-carcinogenic threshold**.
Value exceeds carcinogenic threshold.
Units are in ug/m^3.

Based on the chronic air screening, the following COCs were identified as exceeding air thresholds:

- nitrogen dioxide (NO_2);
- acrolein;
- particulate matter;
- aluminum;
- cadmium;
- cobalt;
- iron;
- manganese;
- nickel;
- titanium; and
- vanadium.

Nitrogen dioxide and acrolein have been evaluated under the Chronic Air Quality Risk Assessment. Particulate matter has been evaluated under the Particulate Matter Assessment. The metal parameters identified above have been evaluated under the Multimedia Risk Assessment, along with media other than air.

I.1.1.1 Toxicity Reference Values

For the inhalation pathway, TRVs for non-carcinogenic chemicals are called Reference Concentrations (RfC) and TRVs for carcinogenic chemicals are called Unit Risks (UR). An RfC is an estimate of continuous inhalation exposure to a chemical by the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects over a lifetime. A UR is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 $\mu g/m^3$ in air.

For the Chronic Air Quality Risk Assessment, nitrogen dioxide and acrolein were identified as COCs. As nitrogen dioxide and acrolein are not considered carcinogenic, only RfCs were considered in the assessment.

The following agencies were used to find available RfCs:

- Health Canada (Health Canada 2009);
- United States Environmental Protection Agency Integrated Risk Information System (U.S. EPA 2012b, internet site);

- World Health Organization (WHO 2000);
- Agency of Toxic Substances and Disease Registry (ATSDR 2012, internet site); and
- National Institute of Public Health and the Environment (RIVM 2001, 2009).

The most conservative (i.e., lowest RfC) was selected for use in the risk assessment. The available RfCs, selected RfCs and toxicological basis of the RfCs are presented in Table I-7.

Table I-7 Reference Concentrations for Chemicals of Potential Concern Evaluated in the Chronic Air Quality Risk Assessment – Non-Carcinogens

Parameter	Reference Concentration [µg/m^3]						Toxicological Endpoints and Derivations
	Health Canada[(a)]	U.S. EPA IRIS[(b)]	ATSDR[(c)]	RIVM[(d)]	WHO[(e)]	Other[(f)]	
Acid Gases							
Nitrogen dioxide	n/a	n/a	n/a	n/a	40	60 (NWT)	The WHO guideline value of 40 µg/m^3 (annual mean) was set to protect the public from the health effects of gaseous NO_2. The NWT chronic threshold is based on the National Ambient Air Quality Objective derived from a maximum acceptable limit in which odour will be perceived (GNWT 2011).
Volatile Organic Compounds (VOCs)							
Acrolein	n/a	0.02	n/a	n/a	n/a	n/a	IRIS derived an RfC for acrolein based on a LOAEL of 0.9 mg/m^3 (0.4 ppm) for nasal lesions in male and female rats exposed to acrolein 6 hours/day, 5 days/week for 13 weeks. An uncertainty factor of 1,000 was applied (3 for use of a minimal LOAEL, 3 for interspecies extrapolation using dosimetric adjustments, 10 for extrapolation from subchronic to chronic duration, and 10 to account for human variability and sensitive subpopulations).

(a) Health Canada (2009).
(b) United States Environmental Protection Agency Integrated Risk Information System (U.S. EPA 2012b, internet site).
(c) Agency for Toxic Substances and Disease Registry (ATSDR 2012, internet site).
(d) National Institute of Public Health and the Environment (RIVM 2001, 2009).
(e) World Health Organization (2000, 2006).
(f) Source of RfC is explained in toxicological endpoint section, as RfCs were available from other jurisdictions.
n/a = Not available, NWT – Northwest Territories
Note: Bolded RfCs were used in the risk assessment. Unless otherwise stated, the most conservative of the available RfCs was chosen (i.e., the lowest).

I.2 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 2012. ATSDR ToxProfiles. Available at: http://www.atsdr.cdc.gov/toxprofiles/index.asp. Accessed July 2012.

CalEPA OEHHA. 2012. Table 1. Consolidated Table of OEHHA/ARB Approved Risk Assessment Health Values. Updated May 3, 2012. California Air Resources Board, California Environmental Protection Agency. http://www.arb.ca.gov/toxics/healthval/contable.pdf. Accessed July 2012.

CCME (Canadian Council of Ministers of the Environment). 1999. National Ambient Air Quality Objectives: Process and Status. Canadian Environmental Quality Guidelines. CCME, Winnipeg, Manitoba.

CCME (Canadian Council of Ministers of the Environment). 2007. Canada Wide Standards for Particulate Matter (PM) and Ozone. Endorsed by CCME Council of Ministers, June 5-6, 2000, Quebec City. CCME, Winnipeg, Manitoba.

Health Canada. 2009. Federal Contaminated Site Risk Assessment in Canada Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors. Health Canada. Ottawa, ON.

GNWT (Government of the Northwest Territories). 2011. Ambient Air Quality Standards. Available at: http://www.enr.gov.nt.ca/_live/documents/content/air_quality_standards_guideline.pdf. Accessed July 2012.

OMoE. 2008. Jurisdictional Screening Level (JSL) List. A Screening Tool for Ontario Regulation 419: Air Pollution - Local Air Quality. Standards Development Branch, Ontario Ministry of the Environment. PIBS #: 6547e, Version 1, February 2008. Accessed July 2012. Available at: http://www.ene.gov.on.ca/environment/en/resources/STD01_076485.html.

OMoE (Ontario Ministry of the Environment). 2011. Ontario Air Standards for Dioxins, Furans and Dioxin-Like PCBs. June 2011. Standards Development Branch, Ontario Ministry of the Environment.

OMoE (Ontario Ministry of the Environment). 2012a. Ontario's Ambient Air Quality Criteria. Standards Development Branch, Ontario Ministry of the

Environment. April 2012. Accessed July 2012. Available at: http://www.ene.gov.on.ca/environment/en/resources/STDPROD_096586.html

OMoE. 2012b. Summary of Standards and Guidelines to support Ontario Regulation 419: Air Pollution — Local Air Quality. Standards Development Branch, Ontario Ministry of the Environment. April 2012. Accessed July 2012. Available at: http://www.ene.gov.on.ca/environment/en/resources/STDPROD_096528.html.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of Human Toxicological Maximum Permissible Risk Levels. RIVM Report No. 711701 025.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of Some Human Toxicological Maximum Permissible Risk Levels Eariler Evaluated in the Period 1991-2001. RIVM Report No. 711701092.

TCEQ (Texas Commission on Environmental Quality). 2012. March 2012 Effects Screening Levels (ESLs). Accessed July 2012. http://www.tceq.state.tx.us/implementation/tox/esl/list_main.html#esl_1

U.S. EPA (United States Environmental Protection Agency). 1997. Health Effects Assessment Summary Tables - FY - 1997 Update. U.S. EPA Solid Waste and Emergency Response, Washington, DC. EPA-540-R-97 036, PB97-921199, July 1997.

U.S. EPA. 2012b. Integrated Risk Information System (IRIS). Cincinnati, OH. Online Database. Accessed July 2012. http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showSubstanceList

U.S. EPA. 2012a. U.S. EPA Regional Screening Level Air Supporting Table. April 2012. Accessed July 2012. Available at: http://www.epa.gov/region09/superfund/prg/.

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

WHO. 2006. WHO Air Quality Guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global Update 2005, Summary of Risk Assessment. Geneva, Switzerland.

APPENDIX II

SUMMARY OF CHEMICAL SCREENING AND IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN FOR MULTI-MEDIA ASSESSMENT

LIST OF TABLES

The screening tables for the Baseline case are presented in Tables II-1 to II-5. The screening tables for the Application case (Construction and Operations Phases) are presented in Tables II-6 to II-9.

Table II-1 Baseline Human Health Soil Metal Screening Results

Parameter [1]	Maximum Measured Baseline Concentration	CCME Guidelines [2]		U.S. EPA RSL [3]	Above Guideline or Screening Level? [4]
		Residential	Notes	Residential	
Total Metals					
Aluminum (Al)	12,900	NG	-	15,400	No
Antimony (Sb)	<0.1	20	G	6.2	No
Arsenic (As)	2.1	12	SI	3.9	No
Barium (Ba)	402	500	I	3,000	No
Beryllium (Be)	0.6	4	G	32	No
Bismuth (Bi)	<0.5	NG	-	NG	NG
Boron (B)	38	NG	-	3,200	No
Cadmium (Cd)	0.64	14	SI	14	No
Calcium (Ca)	17,400	NG	-	NG	NG
Chromium (Cr)	129	220	SI	2.9 [5]	No
Cobalt (Co)	29.7	50	G	4.6	No
Copper (Cu)	28.4	1,100	SI	620	No
Iron (Fe)	23,400	NG	-	11,000	Yes
Lead (Pb)	4.2	140	SI	80	No
Lithium (Li)	14.6	NG	-	32	No
Magnesium (Mg)	58,700	NG	-	NG	NG
Manganese (Mn)	348	NG	-	360	No
Mercury (Hg)	0.172	6.6	SI	2	No
Molybdenum (Mo)	1.55	10	G	78	No
Nickel (Ni)	429	NG	-	300 [6]	Yes
Phosphorus (P)	1170	NG	-	NG	NG
Potassium (K)	5300	NG	-	NG	NG
Selenium (Se)	0.37	80	SI	78	No
Silver (Ag)	0.13	20	G	78	No
Sodium (Na)	110	NG	-	NG	NG
Strontium (Sr)	180	NG	-	9,400	No
Thallium (Tl)	0.103	1	P	0.78 [7]	No
Tin (Sn)	<2	50	G	9,400	No
Titanium (Ti)	678	NG	-	28,000	No
Uranium (U)	1.66	23	DC	46 [8]	No
Vanadium (V)	30.4	NG	-	78	No
Zinc (Zn)	38.5	NG	-	4,600	No

[1] Units for all metals are milligrams per kilogram (mg/kg) as dry weight.

[2] Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health (CCME 1999), residential land use, coarse soil texture. Human health guidelines are provided where available.

[3] Guidelines from the US Environmental Protection Agency (U.S. EPA 2012a) Regional Screening Levels (RSL) for Residential Soil Region 9 (updated in July 2012). Values for carcinogens converted to a risk level (RL) of 10^{-5} (multiplied by 10) and non-carcinogens hazard index (HI) of 0.2 (multiplied by 0.2), for comparison with Canadian guidelines.

[4] For screening purposes, CCME guidelines were used as the primary source; however, if a CCME guideline was not available then U.S. EPA RSLs were applied.

[5] U.S. EPA RSL provided for chromium is for chromium (VI) compounds.

[6] U.S. EPA RSL provided for nickel soluble salts.

[7] U.S. EPA RSL provided for thallium soluble salts.

[8] U.S. EPA RSL provided for uranium soluble salts.

Notes: CCME = Canadian Council of Ministers of the Environment; U.S. EPA = United States Environmental Protection Agency; RSL = Regional Screening Level; G = Generic Guideline; I = Interim Guideline; NG = no guideline; P = Provisional Soil Quality Guideline for Human Health; DC = Direct Contact; SI = Soil Ingestion.

Table II-2 Baseline Soil PAH Screening Results

Parameter [1]	Maximum Measured Baseline Concentration	CCME Guideline [2]	U.S. EPA RSL [3]	Above Guideline or Screening Level? [4]
		Residential	Residential	
Low Molecular Weight PAHs				
1-Methylnaphthalene	<0.01 [5]	NG	160 (16)	No
1-Methylphenanthrene	<0.01 [5]	NG	340 (1700) [7]	No
2-Methylanthracene	<0.01 [5]	NG	3,400 (17,000)[8]	No
2-Methylfluorene	<0.01 [5]	NG	460 (2300) [9]	No
2-Methylnaphthalene	<0.02	NG	46 (62)	No
2-Methylphenanthrene	<0.01 [5]	NG	340 (1700) [7]	No
3-Methyldibenzothiophene	<0.01 [5]	NG	NG	NG
3-Methylphenanthrene	<0.01 [5]	NG	340 (1700) [7]	No
4-+9-Methylphenanthrene	<0.01 [5]	NG	340 (1700) [7]	No
4-Methyldibenzothiophene	<0.01 [5]	NG	NG	NG
Acenaphthene	<0.09	NG	680 (3400)	No
Acenaphthylene	<0.02	NG	NG	NG
Anthracene	<0.02	NG	3400 (17000)	No
Dibenzothiophene	<0.01 [5]	NG	NG	NG
Fluorene	0.16	NG	460 (2300)	No
Naphthalene	<0.02	NG	28 (140)	No
Phenanthrene	<0.02	NG	340 (1700) [6]	No
High Molecular Weight PAHs				
2-Methylpyrene	<0.01 [5]	NG	340 (1700) [10]	No
Acephenanthrylene	<0.01 [5]	NG	NG	NG
Benz(a)anthracene	<0.03	NG	1.5 (0.15)	No [11]
Benzo(a)fluorene	<0.01 [5]	NG	NG	NG
Benzo(a)pyrene	<0.8	NG	0.15 (0.015)	No [11,12]
Benzo(b)fluoranthene	<0.09	NG	1.5 (0.15)	No
Benzo(e)pyrene	<0.01 [5]	NG	NG	NG
Benzo(g,h,i)fluoranthene	<0.01 [5]	NG	NG	NG
Benzo(g,h,i)perylene	<0.07	NG	NG	NG [11]
Benzo(k)fluoranthene	<0.02	NG	15 (1.5)	No
Chrysene	<0.03	NG	150 (15)	No [11]
Coronene	<0.01 [5]	NG	NG	NG
Cyclopenta(c,d)pyrene	<0.01 [5]	NG	NG	NG
Dibenz(a,h)anthracene	<0.03	NG	0.15 (0.015)	No [11]
Fluoranthene	0.011	NG	460 (2300)	No
Indeno(1,2,3-cd)fluoranthene	<0.01 [5]	NG	NG	NG
Indeno(1,2,3-c,d)pyrene	<0.04	NG	1.5 (0.15)	No [11]
Indeno(1,2,3-W)pyrene	<0.01 [5]	NG	NG	NG

Table II-2 Baseline Soil PAH Screening Results (continued)

Parameter [1]	Maximum Measured Baseline Concentration	CCME Guideline [2]	U.S. EPA RSL [3]	Above Guideline or Screening Level? [4]
		Residential	Residential	
Nitro-pyrene	<0.01 [5]	NG	3.8 (0.38)	No
Perylene	<0.01 [5]	NG	NG	NG
Picene	<0.01 [5]	NG	NG	NG
Pyrene	<0.09	NG	340 (1700)	No
B[a]P Total Potency Equivalent [13]	<0.93	5.3	NG	No

1 Units for all PAHs are milligrams per kilogram (mg/kg) as dry weight.

2 Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health (CCME 1999), residential land use, coarse soil texture. Human health guidelines are provided where available.

3 Guidelines from the US Environmental Protection Agency (U.S. EPA 2012a) Regional Screening Levels (RSL) for Residential Soil Region 9 (updated in July 2012). Values for carcinogens converted to a risk level (RL) of 10^{-5} (multiplied by 10) and non-carcinogens hazard index (HI) of 0.2 (multiplied by 0.2), for comparison with Canadian guidelines.

4 For screening purposes, CCME guidelines were used as the primary source; however, if a CCME guideline was not available then U.S. EPA RSLs were applied.

5 Baseline concentrations for these PAHs were not measured, but a standard detection limit of <0.01 mg/kg was assumed for the baseline concentration.

6 The value provided for phenanthrene was adopted from pyrene.

7 The value provided for the methylphenanthrenes was adopted from pyrene.

8 The value provided for the methylanthracene was adopted from that of anthracene.

9 The value selected for methylfluorene was adopted from fluorene.

10 The value selected for methylpyrene was adopted from pyrene.

11 Assessed using the B[a]P Total Potency Equivalent.

12 The B[a]P Total Potency Equivalent guideline from CCME is not exceeded. Since CCME guidelines are used as the primary source, B[a]P does not screen in.

13 CCME does not provide screening guidelines for non-carcinogenic PAHs; CCME recommends using guidelines from another jurisdiction (CCME 2010). For carcinogenic PAHs, the Benzo[a]pyrene Total Potency Equivalent (B[a]P TPE) is for the direct contact pathway. The B[a]P TPE is calculated for several potentially carcinogenic PAHs and compared to an acceptable B[a]P TPE soil guideline of 5.3 µg/g for residential land use which corresponds to a 10^{-5} cancer risk. The soil concentration of each PAH is multiplied by its potency factor, which yields the B[a]P TPE. This calculation is shown below:

Benz[a]anthracene	0.1	Benzo[g,h,i]perylene	0.01	Indeno[1,2,3-c,d]pyrene	0.1
Benzo[a]pyrene	1	Chrysene	0.01		
Benzo[b+j+k]fluoranthene	0.1	Dibenz[a,h]anthracene	1		

$$B[a]P\ TPE = \sum_{i=1}^{n} (C_i \times PEF_i)$$

Notes: NG = no guideline; CCME = Canadian Council of Ministers of the Environment; U.S. EPA = United States Environmental Protection Agency; RSL = Regional Screening Level; PAH = Polycyclic Aromatic Hydrocarbons.

Table II-3 Baseline Sediment Metal Screening Results

Parameter [1]	Maximum Measured Baseline Concentration	CCME Guidelines [2]	U.S. EPA RSL [3]	Above Guideline or Screening Level? [4]
		Residential	Residential	
Total Metals				
Aluminum (Al)	61,700	NG	15,400	Yes
Antimony (Sb)	3	20 [I]	6.2	No
Arsenic (As)	27	12	3.9	Yes
Barium (Ba)	799	500	3,000	Yes
Beryllium (Be)	1.4	4 [I]	32	No
Bismuth (Bi)	7.7	NG	NG	NG
Boron (B)	29	NG	3,200	No
Cadmium (Cd)	1.18	14	14	No
Calcium	7800	NG	NG	No [7]
Chromium (Cr)	170	220	2.9 [5]	No
Cobalt (Co)	80.2	50 [I]	4.6	Yes
Copper (Cu)	153	1,100	620	No
Iron (Fe)	146,000	NG	11,000	Yes
Lead (Pb)	31.2	140	80	No
Lithium	23	NG	32	No
Magnesium	9900	NG	360	No [7]
Manganese (Mn)	21,000	NG	360 [6]	Yes
Mercury (Hg)	0.9	6.6	2	No
Molybdenum	7.9	10 [I]	78	No
Nickel (Ni)	93	NG	300	No
Phosphorus	2450	NG	NG	No [7]
Potassium	2000	NG	NG	No [7]
Selenium (Se)	3	80	78	No
Silver (Ag)	2.7	20 [I]	78	No
Sodium	150	NG	NG	No [7]
Strontium (Sr)	289	NG	9,400	No
Thallium (Tl)	0.4	1	0.156	No
Tin (Sn)	5.9	50 [I]	9,400	No
Titanium	370	NG	28,000	No
Uranium (U)	4	23	46	No
Vanadium (V)	85	NG	78	Yes
Zinc (Zn)	272	NG	4,600	No

[1] Units for all metals are milligrams per kilogram (mg/kg) as dry weight.

[2] Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health (CCME 1999), residential land use, coarse soil texture. Human health component values are provided where available.

[3] U.S. EPA RSLs Region 9 Superfund (U.S. EPA 2012a) (Updated in July 2012). Hazard Index (HI) for non-carcinogens were adjusted by a factor of 0.2 (multiplied by 0.2). Values for carcinogens were converted to a risk level (RL) of 10^{-5} (multiplied by 10).

[4] For screening purposes, CCME guidelines were used as the primary source; however, if a CCME guideline was not available then U.S. EPA RSLs were applied.

[5] U.S. EPA RSL provided for chromium is for chromium (IV) compounds.

[6] U.S. EPA RSL provided for manganese is for manganese (water).

[7] Calcium, sodium, potassium, magnesium, and phosphorus are all essential minerals that serve a variety of biochemical, intracellular, and ion balance purposes in human tissues. These parameters are naturally occurring substances are included in routine analytical chemical analyses. Government agencies often do not develop regulatory criteria for these and other innocuous substances. As these substances are not known or expected to be associated with on-site activities, they have been excluded from the risk assessment (Health Canada 2010a).

Notes: NG = no guideline; CCME = Canadian Council of Ministers of the Environment; U.S. EPA = United States Environmental Protection Agency; RSL = Regional Screening Level; I = CCME Interim remediation criterion.

Table II-4 Baseline Water Screening Results for the Gahcho Kué Project

Parameter	Units	Kennady Lake Baseline Water Quality[a]	Health Canada Drinking Water Quality Guidelines [1]	U.S. EPA Region 3 Regional Screening Levels for Tap Water [2]	Above Guidelines or Screening Level? [3]
Conventionals [4]					
Total Dissolved Solids (TDS)	mg/L	13	500 [5]	NG	No
Major Ions					
Sulphate (SO_4)	mg/L	0.83	500 [5]	NG	No
Nutrients					
Nitrogen - Ammonia (NH_4)	mg/L as N	0.032	NG	NG	NG
Total Metals					
Aluminum (Al)	mg/L	0.0098	0.1 [5]	3.2 (16)	No
Antimony (Sb)	mg/L	0.0001	0.006	0.0012 (0.006)	No
Arsenic (As)	mg/L	0.00014	0.01	0.00045 (0.0045)	No
Barium (Ba)	mg/L	0.0027	1	0.58 (2.9)	No
Beryllium (Be)	mg/L	0.000041	NG	0.0032 (0.016)	No
Boron (B)	mg/L	0.0031	5	0.62 (3.1)	No
Cadmium (Cd)	mg/L	0.00002	0.005	0.00138 (0.0069)	No
Chromium (Cr)	mg/L	0.0002	0.05	3.2 (16) [7] 0.00031 (0.000031) [8]	No
Cobalt (Co)	mg/L	0.000135	NG	0.00094 (0.0047)	No
Copper (Cu)	mg/L	0.0012	1 [5]	0.124 (0.62)	No
Iron (Fe)	mg/L	0.065	0.3 [5]	2.2 (11)	No [3]
Lead (Pb)	mg/L	0.000049	0.01	0.0024 (0.00024) [8]	No
Manganese (Mn)	mg/L	0.0122	0.05 [5]	0.064 (0.32)	No
Mercury (Hg)	mg/L	0.0000102	0.001	0.000126 (0.00063)	No
Molybdenum (Mo)	mg/L	0.000074	NG	0.0156 (0.078)	No
Nickel (Ni)	mg/L	0.00032	NG	0.06 (0.3) [10]	No
Selenium (Se)	mg/L	0.00019	0.01	0.0156 (0.078)	No
Silver (Ag)	mg/L	0.00008	NG	0.0142 (0.071)	No
Strontium (Sr)	mg/L	0.0082	NG	1.86 (9.3)	No
Thallium (Tl)	mg/L	0.000021	NG	0.000032 (0.00016)	No
Uranium (U)	mg/L	0.000026	0.02	0.0094 (0.047)	No
Vanadium (V)	mg/L	0.00024	NG	0.0156 (0.078)	No
Zinc (Zn)	mg/L	0.0028	5 [5]	0.94 (4.7)	No

[a] Baseline water quality data for Kennady Lake (including Area 8)

[1] Health Canada Drinking Water Guidelines (Health Canada 2010b).

[2] U.S. EPA Region 3 Regional Screening Levels for Tap Water (U.S. EPA2012b) (updated in July 2012). Non-cancer-based RSLs were adjusted for the target non-cancer risk level of 0.2 from 1.0, and cancer-based RSLs were adjusted for the target cancer risk level of 10^{-5} from 10^{-6}. U.S. EPA values were only used in the absence of a Health Canada guideline.

[3] For screening purposes, Health Canada Drinking Water Guidelines were used as the primary source; however, if a Health Canada guideline was not available or if the guideline was based on an aesthetic or operational objective, then the U.S. EPA Region 3 Regional Screening Levels for Tap Water were applied.

[4] Assumed pH value based on observed results in the baseline geochemistry test results.

[5] Guideline is an aesthetic objective or operational guideline.

[6] Guideline is equivalent to 45 mg/L as nitrate. When nitrate and nitrite are determined separately, levels of nitrite should not exceed 3.2 mg/L.

[7] Guideline is for trivalent chromium.

[8] Guideline is for hexavalent chromium.

[9] Guideline for lead acetate.

[10] Guideline is for nickel soluble salts.

Notes: NG = No Guideline; U.S. EPA = United States Environmental Protection Agency.

Table II-5 Baseline Fish Screening Results

Parameter	Baseline Water Concentration [1] [mg/L]	BAF [L water/kg fish]	Predicted Maximum Baseline Fish Tissue Concentration [2] [mg/kg wet weight]	U.S. EPA Region 3 Regional Screening Levels for Fish Ingestion [3] [mg/kg]	Above Screening Level?
Total Metals					
Aluminum	0.0185	278	5.14	280 (1400)	No
Antimony	0.0000617	2729	0.168	0.11 (0.54) [4]	Yes
Arsenic	0.000122	417	0.0509	0.021 (0.0021)	Yes
Barium	0.00274	16	0.0438	54 (270)	No
Beryllium	0.0000640	68	0.00435	0.54 (2.7)	No
Boron	0.001743	72	0.125	54 (270)	No
Cadmium	0.0000190	237	0.00450	0.28 (1.4)	No
Chromium	0.000160	78	0.0125	0.063 (0.0063) [5]	No
Cobalt	0.000190	157	0.0298	0.082 (0.41)	No
Copper	0.00128	839	1.07	10.8 (54)	No
Iron	0.0590	150	8.85	190 (950)	No
Lead	0.000061	80	0.00488	0.11 (0.011) [6]	No
Manganese	0.00570	29	0.165	38 (190)	No
Mercury	0.0000051	9450	0.0482	0.082 (0.41) [7]	No
Molybdenum	0.0000300	449	0.0135	1.36 (6.8)	No
Nickel	0.000465	232	0.108	5.4 (27) [8]	No
Selenium	0.0000320	3000	0.0960	1.36 (6.8)	No
Silver	0.00000810	2000	0.0162	1.36 (6.8)	No
Strontium	0.00690	69	0.476	162 (810)	No
Thallium	0.0000142	800	0.0114	0.0028 (0.014) [9]	Yes
Uranium	0.0000158	270	0.00427	0.82 (4.1) [10]	No
Vanadium	0.0000940	95	0.00893	1.36 (6.8) [11]	No
Zinc	0.00240	379	0.910	82 (410)	No

[1] Maximum predicted baseline water concentrations in Lake N11 and N410.

[2] Fish tissue concentrations in Lake N11 and Lake N410 were estimated by multiplying predicted maximum baseline concentrations in water by parameter-specific bioaccumulation factors (Aquatic Health Section - Appendix 8.VI of the 2012 EIS Update).

[3] U.S. EPA Region 3 Regional Screening Level (RSL) Fish Ingestion (2012c) (Updated in July 2012). U.S. EPA RSLs were adjusted by a factor of 0.2 for non-carcinogens and by a factor of 10 for carcinogens.

[4] Screening level is for antimony (metallic).

[5] Screening level is for chromium (VI).

[6] Screening level is for lead acetate.

[7] Screening level for mercuric chloride and other mercury salts.

[8] Screening level is for nickel (soluble salts).

[9] Screening level is for thallium (soluble salts).

[10] Screening level is for uranium (soluble salts).

[11] Screening level is for vanadium and compounds.

Notes: U.S. EPA = United States Environmental Protection Agency; RSL = Regional Screening Level.

Table II-6 Construction and Operations Soil Metal Screening Results

Parameter[1]	Maximum Predicted Concentration for Construction and Operations Phases)	Maximum Measured Baseline Concentration + 10%	CCME Guidelines[2]	U.S. EPA RSL[3]	Above Maximum Measured Baseline Concentration + 10%?	Above Guideline or Screening Level? [4]	Chemical of Concern? [5]
			Residential	Residential			
Metals							
Aluminum	12,923	14,190	NG	15,400	No	No	No
Antimony	0.101	0.11	20	6.2	No	No	No
Arsenic	2.10	2.31	12	3.9	No	No	No
Barium	402	442	500 [I]	3,000	No	No	No
Beryllium	0.60	0.66	4 [I]	32	No	No	No
Bismuth	0.50	0.55	NG	NG	No	NG	No
Boron	38	41.8	NG	3,200	No	No	No
Cadmium	0.699	0.704	14	14	No	No	No
Chromium	129	142	220	2.9 [6]	No	No	No
Cobalt	29.7	32.7	50 [I]	4.6	No	No	No
Copper	28.4	31.2	1,100	620	No	No	No
Iron	23,442	25,740	NG	11,000	No	Yes	No
Lead	4.22	4.62	140	80	No	No	No
Lithium	14.60	14.6	NG	32	No	No	No
Manganese	348	383	NG	360 [7]	No	No	No
Mercury	0.176	0.189	6.6	2	No	No	No
Molybdenum	1.56	1.71	10 [I]	78	No	No	No
Nickel	429	472	NG	300	No	Yes	No
Selenium	0.371	0.41	80	78	No	No	No
Silver	0.1408	0.143	20 [I]	78	No	No	No
Strontium	180	198	NG	9,400	No	No	No
Thallium	0.279	0.113	1	0.78 [8]	Yes	No	No
Tin	2.0	2.2	50 [I]	9,400	No	No	No
Titanium	680	746	NG	28,000	No	No	No
Uranium	1.66	1.83	23	46	No	No	No
Vanadium	30.5	33.4	NG	78	No	No	No
Zinc	38.6	42.5	NG	4,600	No	No	No

[1] Units for all metals are milligrams per kilogram (mg/kg) as dry weight. Parameters that are of low toxicological concern such as calcium, magnesium, phosphorus, potassium, and sodium are not assessed as a chemical of concern, therefore they are not included in this table.

[2] Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health (CCME 1999), residential land use, coarse soil texture.

[3] U.S. EPA RSLs (Region 9) Residential Soils (2012a) (Updated in July 2012). Hazard Index (HI) for non-carcinogens were adjusted by a factor of 0.2 (multiplied by 0.2). Values for carcinogens were converted to a risk level (RL) of 10^{-5} (multiplied by 10).

[4] For screening purposes, CCME guidelines were used as the primary source; however, if a CCME guideline was not available then U.S. EPA RSLs were applied.

[5] If a parameter is above the appropriate guideline and the concentrations is greater than 10% above baseline, then it is considered a chemical of concern.

[6] U.S. EPA RSL provided for chromium is for chromium (VI) compounds.

[7] U.S. EPA RSL provided for manganese is for manganese (water).

[8] U.S. EPA RSL provided for thallium is for thallium soluble salts.

[9] U.S. EPA RSL provided for uranium is for uranium soluble salts.

Notes: NG = no guideline; CCME = Canadian Council of Ministers of the Environment; U.S. EPA = United States Environmental Protection Agency; RSL = Regional Screening Level; I = CCME Interim remediation criterion.

Table II-7 Construction and Operations Soil PAH Screening Results

Parameter [1]	Predicted Maximum Concentration for Construction and Operations Phases	Maximum Measured Baseline Concentration +10 %	CCME Guidelines[2]	U.S. EPA RSL[3]	Above Maximum Measured Baseline Concentration + 10%?	Above Guideline or Screening Level? [4]	Chemical of Concern? [5]
			Residential	Residential			
Low Molecular Weight PAHs							
1-Methylnaphthalene	0.0552	0.011 [6]	NG	160 (16)	Yes	No	No
1-Methylphenanthrene	0.012	0.011 [6]	NG	340 (1700)[8]	Yes	No	No
2-Methylanthracene	0.011	0.011 [6]	NG	3,400 (17,000)[9]	No	No	No
2-Methylfluorene	0.010	0.011 [6]	NG	460 (2300)[10]	No	No	No
2-Methylnaphthalene	0.093	0.022	NG	46 (62)	Yes	No	No
2-Methylphenanthrene	0.015	0.011 [6]	NG	340 (1700)[8]	Yes	No	No
3-Methyldibenzothiophene	0.010	0.011 [6]	NG	NG	No	NG	No
3-Methylphenanthrene	0.014	0.011 [6]	NG	340 (1700)[8]	Yes	No	No
4-+9-Methylphenanthrene	0.013	0.011 [6]	NG	340 (1700)[8]	Yes	No	No
4-Methyldibenzothiophene	0.010	0.011 [6]	NG	NG	No	NG	No
Acenaphthene	0.0923	0.099	NG	680 (3400)	No	No	No
Acenaphthylene	0.0284	0.022	NG	NG	Yes	NG	No [14]
Anthracene	0.0215	0.022	NG	3,400 (17,000)	No	No	No
Dibenzothiophene	0.010	0.011 [6]	NG	NG	No	NG	No
Fluorene	0.0172	0.176	NG	460 (2300)	No	No	No
Naphthalene	0.199	0.022	NG	28 (140)	Yes	No	No
Phenanthrene	0.0311	0.022	NG	340 (1700)[6]	Yes	No	No
High Molecular Weight PAHs							
2-Methylpyrene	0.010	0.011 [6]	NG	340 (1700)[11]	No	No	No
Acephenanthrylene	0.011	0.011 [6]	NG	NG	No	NG	No
Benz(a)anthracene [12]	0.030	0.033	NG	1.5 (0.15)	No	No	No
Benzo(a)fluorene	0.010	0.011 [6]	NG	NG	No	NG	No
Benzo(a)pyrene [12]	0.800	0.88	NG	0.15 (0.015)	No	Yes	No
Benzo(b)fluoranthene	0.092	0.099	NG	1.5 (0.15)	No	No	No
Benzo(e)pyrene	0.010	0.011 [6]	NG	NG	No	NG	No
Benzo(g,h,i)fluoranthene	0.01070	0.011 [6]	NG	NG	No	NG	No
Benzo(g,h,i)perylene [12]	0.07045	0.077	NG	NG	No	NG	No
Benzo(k)fluoranthene	0.02019	0.022	NG	15 (1.5)	No	No	No
Chrysene [12]	0.03040	0.033	NG	150 (15)	No	No	No
Coronene	0.0100	0.011 [6]	NG	NG	No	NG	No
Cyclopenta(c,d)pyrene	0.01025	0.011 [6]	NG	NG	No	NG	No
Dibenzo(a,h)anthracene [12]	0.03052	0.033	NG	0.15 (0.015)	No	No	No
Fluoranthene	0.01633	0.012	NG	460 (2300)	Yes	No	No
Indeno(1,2,3-cd)fluoranthene	0.01002	0.011 [6]	NG	NG	No	NG	No

Table II-7 Construction and Operations Soil PAH Screening Results (continued)

Parameter [1]	Predicted Maximum Concentration for Construction and Operations Phases	Maximum Measured Baseline Concentration +10 %	CCME Guidelines[2]	U.S. EPA RSL[3]	Above Maximum Measured Baseline Concentration + 10%?	Above Guideline or Screening Level? [4]	Chemical of Concern? [5]
			Residential	Residential			
Indeno(1,2,3-cd)pyrene [12]	0.04000	0.044	NG	1.5 (0.15)	No	No	No
Indeno(1,2,3-W)pyrene	0.01031	0.011 [6]	NG	NG	No	NG	No
Nitro-pyrene	0.01028	0.011 [6]	NG	3.8 (0.38)	No	No	No
Perylene	0.01000	0.011 [6]	NG	NG	No	NG	No
Picene	0.01000	0.011 [6]	NG	NG	No	NG	No
Pyrene	0.09859	0.099	NG	340 (1700)	No	No	No
B[a]P TPE [13]	0.8499	1.023	5.3	NG	No	No	No

1 Units for all PAHs are milligrams per kilogram (mg/kg) as dry weight.

2 Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health (CCME 1999), residential land use, coarse soil texture.

3 U.S. EPA RSLs (Region 9) Residential Soil (U.S. EPA 2012a) (Updated in July 2012).Hazard Index (HI) for non-carcinogens were adjusted by a factor of 0.2 (multiplied by 0.2). Values for carcinogens were converted to a risk level (RL) of 10^{-5} (multiplied by 10).

4 For screening purposes, CCME guidelines were used as the primary source; however, if a CCME guideline was not available then U.S. EPA RSLs were applied.

5 If a parameter is above the appropriate guideline and the concentrations is greater than 10% above baseline, then it is considered a chemical of concern.

6 These parameters were not measured but a standard detection limit of 0.01 mg/kg was used as the baseline concentration.

7 The value provided for the phenanthrenes was adopted from pyrene.

8 The value provided for the methylphenanthrenes was adopted from pyrene.

9 The value provided for the methylanthracene was adopted from that of anthracene.

10 The value selected for methylfluorene was adopted from fluorene.

11 The value selected for methylpyrene was adopted from pyrene.

12 Assessed using the B[a]P Total Potency Equivalent.

13 CCME does not provide screening guidelines for non-carcinogenic PAHs; CCME recommends using guidelines from another jurisdiction (CCME 2010). For carcinogenic PAHs, the Benzo[a]pyrene Total Potency Equivalent (B[a]P TPE) is for the direct contact pathway. The B[a]P TPE is calculated for several potentially carcinogenic PAHs and compared to an acceptable B[a]P TPE soil guideline of 5.3 µg/g for residential land use which corresponds to a 10^{-5} cancer risk. The soil concentration of each PAH is multiplied by its potency factor, which yields the B[a]P TPE. This calculation is shown below:

Benz[a]anthracene	0.1	Benzo[g,h,i]perylene	0.01	Indeno[1,2,3-c,d]pyrene	0.1
Benzo[a]pyrene	1	Chrysene	0.01		
Benzo[b+j+k]fluoranthene	0.1	Dibenz[a,h]anthracene	1		

$$B[a]P\ TPE = \sum_{i=1}^{n} (C_i \times PEF_i)$$

14 Not considered a COC, because the predicted concentrations are orders of magnitude lower than the available PAH guidelines for other parameters.

Notes: NG = no guideline; CCME = Canadian Council of Ministers of the Environment; U.S. EPA = United States Environmental Protection Agency; RSL = Regional Screening Level; PAH = Polycyclic Aromatic Hydrocarbons.

Table II-8 Construction and Operations Water Screening Results

Parameter	Units	Predicted Maximum Concentrations for Area 8 during Construction and Operation Phases	Baseline Water Concentrations for Kennady Lake[a] +10%	Health Canada Drinking Water Quality Guidelines[1]	US EPA Region 3 Regional Screening Levels for Tap Water[2]	Maximum Concentration - Above Baseline +10%?	Above Guideline or Screening Level?	Chemical of Concern?[3]
Conventionals [4]								
Total Dissolved Solids (TDS)	mg/L	14.9	14.3	500 [5]	NG	Yes	No	No
Major Ions								
Sulphate (SO_4)	mg/L	0.94	0.91	500 [5]	NG	Yes	No	No
Nutrienta								
Nitrogen - Ammonia (NH_4)	mg/L as N	0.0285	0.035	NG	NG	No	NG	No
Total Metals								
Aluminum (Al)	mg/L	0.011	0.011	0.1 [5]	3.2 (16)	No	No	No
Antimony (Sb)	mg/L	0.00011	0.00011	0.006	0.0012 (0.006)	No	No	No
Arsenic (As)	mg/L	0.00016	0.00015	0.01	0.00045 (0.0045)	Yes	No	No
Barium (Ba)	mg/L	0.003	0.003	1	0.58 (2.9)	No	No	No
Beryllium (Be)	mg/L	0.000046	0.000045	NG	0.0032 (0.016)	Yes	No	No
Boron (B)	mg/L	0.0035	0.0034	5	0.62 (3.1)	Yes	No	No
Cadmium (Cd)	mg/L	0.000026	0.000022	0.005	0.00138 (0.0069)	Yes	No	No
Chromium (Cr)	mg/L	0.00022	0.00022	0.05	3.2 (16) [7] 0.00031 (0.000031) [8]	No	No	No
Cobalt (Co)	mg/L	0.000153	0.000149	NG	0.00094 (0.0047)	Yes	No	No
Copper (Cu)	mg/L	0.0014	0.0013	1 [5]	0.124 (0.62)	Yes	No	No
Iron (Fe)	mg/L	0.0741	0.0715	0.3 [5]	2.2 (11)	Yes	No	No
Lead (Pb)	mg/L	0.0000553	0.0000539	0.01	0.0024 (0.00024) [9]	Yes	No	No
Manganese (Mn)	mg/L	0.0137	0.0134	0.05 [5]	0.064 (0.32)	Yes	No	No
Mercury (Hg)	mg/L	0.000012	0.000011	0.001	0.000126 (0.00063)	Yes	No	No
Molybdenum (Mo)	mg/L	0.000084	0.000081	NG	0.0156 (0.078)	Yes	No	No
Nickel (Ni)	mg/L	0.000366	0.000352	NG	0.06 (0.3) [10]	Yes	No	No
Selenium (Se)	mg/L	0.00021	0.00021	0.01	0.0156 (0.078)	No	No	No
Silver (Ag)	mg/L	0.000091	0.000088	NG	0.0142 (0.071)	Yes	No	No
Strontium (Sr)	mg/L	0.00928	0.00902	NG	1.86 (9.3)	Yes	No	No
Thallium (Tl)	mg/L	0.000023	0.000023	NG	0.000032 (0.00016)	No	No	No
Uranium (U)	mg/L	0.000030	0.000029	0.02	0.0094 (0.047)	Yes	No	No
Vanadium (V)	mg/L	0.00027	0.00026	NG	0.0156 (0.078)	Yes	No	No
Zinc (Zn)	mg/L	0.0032	0.0031	5 [5]	0.94 (4.7)	Yes	No	No

[a] Baseline water quality data for Kennady Lake (including Area 8).
[1] Health Canada Drinking Water Guidelines (Health Canada 2010b).
[2] U.S. EPA Region 3 Regional Screening Levels for Tap Water (U.S. EPA 2012b).
Non-cancer-based RSLs were adjusted for the target non-cancer risk level of 0.2 from 1.0, and cancer-based RSLs were adjusted for the target cancer risk level of 10^{-5} from 10^{-6}. U.S. EPA values were only used in the absence of a Health Canada guideline.
[3] Chemical of concern only if the maximum concentration from all project scenarios is greater than the baseline concentration + 10% and the applicable guideline.
[4] Assumed pH value based on observed results in the baseline geochemistry test results.
[5] Guideline is an aesthetic objective or operational guideline.
[6] Guideline is equivalent to 45 mg/L as nitrate. Where nitrate and nitrite are determined separately, levels of nitrite should not exceed 3.2 mg/L.
[7] Guideline is for chromium (III).
[8] Guideline is for chromium (VI).
[9] Guideline is for lead acetate.
[10] Guideline is for nickel soluble salts.
Notes: NG = No Guideline; US EPA = United States Environmental Protection Agency.

Table II-9 Construction and Operations Fish Screening Results

Parameter	Project Water Concentration [1] [mg/L]	BAF [L water/kg fish]	Maximum Predicted Project Fish Tissue Concentration [2] [mg/kg wet weight]	Maximum Predicted Baseline Fish Tissue Concentration + 10% [mg/kg wet weight]	U.S. EPA Region 3 Regional Screening Levels for Fish Ingestion [3] [mg/kg]	Above Maximum Predicted Baseline Fish Tissue Concentration + 10%?	Above Screening Level?	Chemical of Concern? [4]
Metals								
Aluminum	0.0294	278	8.18	5.66	280 (1400)	Yes	No	No
Antimony	0.000346	2729	0.943	0.185	0.11 (0.54) [5]	Yes	Yes	Yes
Arsenic	0.000742	417	0.310	0.056	0.021 (0.0021)	Yes	Yes	Yes
Barium	0.0103	16	0.166	0.0482	54 (270)	Yes	No	No
Beryllium	0.0000730	68	0.00497	0.00479	0.54 (2.7)	Yes	No	No
Boron	0.0256	72	1.85	0.138	54 (270)	Yes	No	No
Cadmium	0.0000236	237	0.00558	0.00495	0.28 (1.4)	Yes	No	No
Chromium	0.000378	78	0.0295	0.0137	0.063 (0.0063)[6]	Yes	No	No
Cobalt	0.000361	157	0.0567	0.0328	0.082 (0.41)	Yes	No	No
Copper	0.00147	839	1.24	1.18	10.8 (54)	Yes	No	No
Iron	0.088467	150	13.3	9.74	190 (950)	Yes	No	No
Lead	0.000111	80	0.00888	0.00537	0.11 (0.011) [7]	Yes	No	No
Manganese	0.0136	29	0.395	0.182	38 (190)	Yes	No	No
Mercury	0.00000646	9450	0.0610	0.053	0.082 (0.41)[8]	Yes	No	No
Molybdenum	0.00157	449	0.703	0.0148	1.36 (6.8)	Yes	No	No
Nickel	0.00122	232	0.283	0.119	5.4 (27)[9]	Yes	No	No
Selenium	0.0000563	3000	0.169	0.106	1.36 (6.8)	Yes	No	No
Silver	0.0000197	2000	0.0394	0.0178	1.36 (6.8)	Yes	No	No
Strontium	0.0172	69	1.19	0.524	162 (810)	Yes	No	No
Thallium	0.0000492	800	0.0393	0.0125	0.0028 (0.014)[10]	Yes	Yes	Yes
Uranium	0.000372	270	0.100	0.00469	0.82 (4.1)[11]	Yes	No	No
Vanadium	0.000513	95	0.0487	0.00982	1.36 (6.8)[12]	Yes	No	No
Zinc	0.00346	379	1.31	1	82 (410)	Yes	No	No

[1] Maximum predicted water concentrations in Lake N11 and N410, during construction, operations and closure.

[2] Fish tissue concentrations in Lake N11 and Lake N410 were estimated by multiplying predicted maximum concentrations in water during construction, operations and closure by parameter-specific bioaccumulation factors (Aquatic Health Section - Appendix 8.VI of the 2012 EIS Update).

[3] U.S. EPA Regional Screening Level (RSL) Fish Ingestion Region 3 (April 2012c) (Updated July 2012). U.S. EPA RSLs were adjusted by a factor of 0.2 for non-carcinogens and by a factor of 10 for carcinogens.

[4] If a parameter is above the screening level and the concentration is greater than 10% above baseline, then it is considered a chemical of concern.

[5] Screening level is for antimony (metallic).

[6] Screening level is for chromium VI.

[7] Screening level is for lead acetate.

[8] Screening level for mercuric chloride and other mercury salts.

[9] Screening level is for nickel (soluble salts).

[10] Screening level is for screening level is for thallium (soluble salts).

[11] Screening level is for uranium (soluble salts).

[13] Screening level is for vanadium and compounds.

Notes: NG = No Guideline; U.S. EPA = United States Environmental Protection Agency; RSL = Regional Screening Level.

REFERENCES

CCME (Canadian Council of Ministers of the Environment). 1999. Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health. Canadian Environmental Quality Guidelines. CCME. Winnipeg, Manitoba.

CCME (Canadian Council of Ministers of the Environment). 2010. Canadian Soil Quality Guidelines for the Protection nof Environmental and Human Health: Polycyclic Aromatic Hydrocarbons. CCME. Winnipeg, Manitoba.

Health Canada. 2010a. Federal Contaminated Site Risk Assessment in Canada, Part V: Guidance on Human Health Detailed Quantitative Risk Assessment for Chemicals (DQRAChem). Available at: www.healthcanada.gc.ca

Health Canada. 2010b. Guidelines for Canadian Drinking Water Quality – Summary Table. December 2010. Health Canada. Ottawa, Ontario.

U.S. EPA (United States Environmental Protection Agency). 2012a. U.S. EPA Regional Screening Level for Region 9. April 2012. Accessed July 2012. Available at: http://www.epa.gov/region09/superfund/prg/.

U.S. EPA (United States Environmental Protection Agency). 2012b. U.S. EPA Regional Screening Levels for Region 3. April 2012. Accessed July 2012. Available at: http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/Generic_Tables/index.htm

U.S. EPA (United States Environmental Protection Agenecy). 2012c. U.S. EPA Regional Screening Levels for Region 3 (Fish Ingestion). April 2012. Accessed July 2012. Available at: http://www.epa.gov/reg3hwmd/risk/human/index.htm

APPENDIX III

MULTI-MEDIA TOXICITY ASSESSMENT

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III CONTAMINANT CLASSIFICATION

Several organizations have developed classification systems based on the carcinogenic properties of chemicals. The classification systems for the International Agency for Research on Cancer (IARC 2012), U.S. EPA (2012a) and Health Canada (1996) are presented in Table III-1.

Table III-1 Carcinogen Classification Systems used by IARC, U.S. EPA and Health Canada

IARC[1]	U.S. EPA[2]	Health Canada[3]	Description
Group 1	Group A	Group I	Human carcinogen
Group 2A	Group B	Group II	Probable human carcinogen
	B1		Limited human evidence available
	B2		Inadequate human evidence, sufficient animal evidence
Group 2B	Group C	Group III	Possible human carcinogen
Group 3	Group D	Group IV	Unclassifiable as to human carcinogenicity/ Unlikely to be a carcinogen (Health Canada only)
Group 4	Group E	Group V	Probably not carcinogenic to humans
		Group VI	Unclassifiable as to human carcinogenicity

1. International Agency for Research on Cancer (IARC 2012).
2. U.S. EPA – Integrated Risk Information System (IRIS) accessed on-line (U.S. EPA 2012a).
3. Health Canada (1996).

The carcinogenicity classifications for the chemicals of concern (COCs) assessed in this risk assessment are provided in Table III-2.

Table III-2 Carcinogenicity Classification for the Chemicals of Concern at the Site

Compound	Health Canada	IARC	U.S. EPA	Assessed as a Carcinogen?
Metals				
Aluminum	ND	ND	ND	No
Antimony	ND	Group 2B (antimony trioxide); Group 3 (antimony trisulfide)	ND	No
Arsenic	Group 1	Group 1	Group A	Yes
Cadmium	Group II	Group I	Group B1 (inhalation only)	Yes (inhalation only)
Cobalt	ND	Group 2B	ND	Yes (inhalation only)
Iron	ND	ND	ND	No
Manganese	ND	ND	Group D	No
Nickel	Group VI (metallic)/ Group I (soluble)	Group 2B (metallic); Group I (nickel compounds)	Group A (refinery dust, nickel subsulfide); Group B2 (nickel carbonyl)	Yes
Thallium	ND	ND	ND	No
Titanium	ND	ND	ND	No
Vanadium	ND	Group 2B (vanadium pentoxide)	ND	No

ND = Not determined.

Source: Health Canada 2009; IARC 2012; U.S. EPA 2012a.

III.1 TOXICITY REFERENCE VALUES

The toxicity assessment was conducted for all COCs and involved the identification of the potential toxic effects of these chemicals and the selection of toxicity reference values (TRVs) for each chemical. TRVs include reference doses (RfD), and tolerable daily intakes (mg/kg/day; TDI), and reference concentrations (RfC) (mg/m^3) for non-carcinogens and cancer potency or slope factors for carcinogens. A reference dose or concentration represents an estimated daily intake, which can be received by human receptors each day over a lifetime without experiencing any significant or adverse health impact. Slope factors are used to estimate carcinogenic risk and are defined as a plausible upper bound probability of an individual developing cancer as a result of a lifetime exposure to a potential carcinogen.

Both hazard quotients and incremental lifetime cancer risks (ILCR) values were calculated for carcinogens where possible.

The TRVs used in this risk assessment were obtained from several governmental agencies using the general hierarchy given below:

- Health Canada. 2009. Federal Contaminated Sites Risk Assessment in Canada. Part II. Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors, Version 2.0, May 2009. Health Canada. Ottawa, ON.
- U.S. EPA. 2012a. Integrated Risk Information System (IRIS). http://www.epa.gov/iris/.
- Other sources of peer-reviewed TRVs (in no order of preference):
  - Agency for Toxic Substances and Disease Registry (ATSDR). 2012. http://www.atsdr.cdc.gov/.
  - California Environmental Protection Agency (Cal EPA). http://www.oehha.ca.gov/air/hot_spots/index.html.
  - U.S. EPA. 2010a. National Center for Environmental Assessment (NCEA). http://www.epa.gov/ncea/index.htm.
  - National Institute of Public Health and the Environment (RIVM). 2001. Re-evaluation of human-toxicological maximum permissible risk levels. March, 2001.
- Provisional TRVs, including the following source:
  - U.S. EPA 2010b. NCEA Provisional Peer-Reviewed Toxicity Values (PPRTV). Available upon request.

Occasionally exceptions were made to the hierarchy of selection of chemical-specific TRVs based on the currency of the study, study duration (i.e., chronic duration preferred) and whether the critical endpoint was based a no-observed-adverse effect level (NOAEL).

A summary of the toxicity reference values selected for use in the risk assessment is provided in Table III-3. Toxicity profiles for all contaminants of potential concern, outlining pharmacokinetics, toxicity, carcinogenicity classification and toxicity-based reference values selected for use in the human health risk assessment, are compiled in Section III.3.

III.2 ROUTE-TO-ROUTE EXTRAPOLATION OF TOXICITY VALUES

The toxicity of a substance can differ between exposure routes (e.g., oral versus inhalation) and as a result route-to-route extrapolation of TRVs was not conducted in the absence of inhalation specific reference values (U.S. EPA 2009). Oral RfD values and slope factors were conservatively used to assess dermal exposure.

Table III-3 Selected for the Human Health Risk Assessment

COC	Classification		Toxicity Reference Values										
			Oral				Dermal			Inhalation			
	Health Canada [a]	U.S. EPA [a]	TDI/RfD$_o$ [mg/kg-d]	SF [mg/kg-d]$^{-1}$	Target Organ(s)/Effect	Source	RfD[c] [mg/kg-d]	SF [mg/kg-d]$^{-1}$	RAF [e] (unitless)	RfC [mg/m^3]	Unit Risk [mg/m^3]$^{-1}$	Target Organ/Effect	Source
Total Metals													
Aluminum	ND	ND	1.0	N/A	neurotoxicity	U.S. EPA RSL (US EPA 2012b)	1.0	N/A	0.01 [g]	0.005	N/A	neurological effects (psychomotor and cognitive impairment)	U.S. EPA RSL (US EPA 2012b)
Antimony	ND	ND	0.006	N/A	decreased body weight/ reduced food and water intake	RIVM 2009	0.006	N/A	0.1	0.0002 (antimony trioxide)	N/A	pulmonary toxicity, chronic interstitial inflammation	U.S. EPA IRIS (US EPA 2012a)
Arsenic	I	A	0.0003	1.8	skin (hyperpigmentation, keratosis) and possible vascular complications (RfD); lung, bladder and liver cancer (SF)	U.S. EPA IRIS (US EPA 2012a) (RfD); Health Canada 2009 (SF)	0.0003	1.8	0.03	0.001	6.4	lung (RfC), lung cancer (UR)	RIVM 2001 (RfC); Health Canada 2009 (UR)
Cadmium	II	B1	0.001 (food) 0.0005 (water)	N/A	kidneys (proteinuria)	U.S. EPA IRIS (US EPA 2012a)	0.001 (food) 0.0005 (water)	N/A	0.01	0.00001	1.8	kidney (RfC); lung, trachea and bronchus cancer (UR)	ATSDR 2012 (RfC); U.S. EPA IRIS (US EPA 2012a) (UR)
Cobalt	ND	ND	0.0003	N/A	thyroid (decreased iodine uptake)	U.S. EPA RSL (US EPA 2012b)	0.0003	N/A	0.01	0.000006	9	decreased pulmonary function and respiratory tract irritation	U.S. EPA RSL (US EPA 2012b)
Iron	NC	NC	0.7	N/A	gastrointestinal effects	U.S. EPA RSL (US EPA 2012b)	0.7	N/A	0.01[i]	N/A	N/A	N/A	N/A
Manganese	NC	D	(toddler); (adult)	N/A	Parkinsonian-like neurotoxicity	Health Canada 2009	0.1 (toddler); 0.2 (adult)	N/A	0.01[i]	0.00005	N/A	nervous system (impairment of neurobehavioural function)	U.S. EPA IRIS (US EPA 2012a)
Nickel [d]	VI/I [b]	A/B2 [f]	0.011	N/A	post-implantation perinatal lethality	Health Canada 2009	0.011	N/A	0.01	0.000018	0.71	respiratory track effects i.e., alveolar macrophages, hyperplasia (RfC); lung and nasal cancer (UR)	Health Canada 2009
Thallium	ND	ND	0.00001	N/A	hair follicle atrophy	U.S. EPA RSL (US EPA 2012b)	0.00001	N/A	0.01 [g]	N/A	N/A	N/A	N/A
Titanium	ND	ND	N/A	N/A	N/A	N/A	N/A	N/A	0.01 [g]	0.0001 (titanium tetrachloride)	N/A	respiratory tract (rhinitis and tracheitis)	ATSDR 2012
Vanadium	ND	ND	0.005	N/A	decreased hair cystine	U.S. EPA IRIS (US EPA 2012a)	0.005	N/A	0.1	0.0001	N/A	respiratory tract (degeneration of the respiratory epithelium of the epiglottis)	ATSDR 2012

a - Health Canada classifications are from Health Canada 2009; U.S. EPA classifications are from U.S. EPA IRIS (2012a).

b - Classification for metallic nickel is Group VI, classification for nickel sulphate, nickel subsulphide and soluble nickel is Group I.

c - Oral TRV adopted as dermal TRV not available.

d- Soluble nickel used to assess oral non-carcinogenic effects, nickel subsulphide used to assess inhalation non-carcinogenic effects, soluble nickel used to assess carcinogenic effects for inhalation pathway (i.e., unit risk).

e- Relative dermal absorption factors (RAFs) from Health Canada (2009), unless otherwise noted.

f - U.S. EPA IRIS has classified nickel subsulphide and nickel refinery dust as Group A, and nickel carbonyl as Group B2. Soluble salts of nickel as a compound have not been classified.

g – Dermal RAF from OMOE (2011).

Notes: COC – Contaminant of Concern, TDI – Tolerable Daily Intake, RfD - Reference Dose, SF - Slope Factor, RfC - Reference Concentration, RAF - Relative Absorption Factor, UR – Unit Risk, inh – inhalation, ND – not determined, N/A – Not Available, NC – non-carcinogen; HC - Health Canada, CCME - Canadian Council of Ministers of the Environment, U.S. EPA - United States Environmental Protection Agency, IRIS - Integrated Risk Information System, RSL – Regional Screening Levels.

III.3 TOXICITY PROFILES

III.3.1 Aluminum

III.3.1.1 Pharmacokinetics

In general, absorption of aluminum in humans and animals is poor via inhalation or oral exposure pathways and dermal absorption is even less significant (ATSDR 2008).

Oral Exposure

Absorption of aluminum through normal dietary uptake is estimated as 0.1%, while more bioavailable forms (e.g., complexes with some carboxylic acids) can be absorbed at a rate closer to 1.0%. In addition, the aqueous and pH conditions of the gut will also affect absorption (ATSDR 2008). Distribution of aluminum following oral exposure in animals has been shown to occur in the brain (hippocampus), while simultaneous intake of vitamin D has been found to enhance accumulation and retention of aluminum in the bones, kidneys, muscle and heart (ATSDR 2008). Elimination of aluminum following oral uptake in humans and animals occurs in the kidneys (via urine) with unabsorbed aluminum being excreted primarily in the feces. A study on rats found a single oral dose of 11 mg aluminum resulted in a 14-fold increase in aluminum levels in the urine within 24-hours of exposure and that normal baseline levels returned after 5 days (ATSDR 2008).

Inhalation Exposure

Inhalation exposure studies on humans have found occupational exposure to aluminum fumes, dusts and flakes result in increased serum levels, and that direct absorption in the brain may occur through the olfactory tract via axonal transport. Autopsy results from a stonemason exposed to aluminum found elevated concentrations (compared to normal baseline levels) in the lungs, hilar lymph nodes, liver and spleen (ATSDR 2008). Rats and guinea pigs with subchronic or chronic exposure to aluminum chlorhydrate showed accumulation primarily in the lungs, with some additional accumulation in the adrenal glands and peribronchial lymph nodes. Excretion in humans occurs via urine, and a correlation exists between exposure duration and urinary concentrations; welders exposed to 0.2 to 5.3 mg/m^3 aluminum for 10 years had urinary aluminum half-lives of over 6 months compared to 9 days in individuals with less than 1-year exposure (ATSDR 2008).

Dermal Exposure

A study was conducted applying aluminum chlorohydrate to the underarms of two subjects, the conclusions of the study estimated 0.012% of the applied aluminum was absorbed through the skin (ATSDR 2008). A mouse study has found elevated concentrations in the liver, brain, lung and kidneys following exposure to 0.04 mg/day for 20 days during gestation (ATSDR 2008). No studies were found on the excretion of aluminum following dermal exposure in humans or animals (ATSDR 2008).

III.3.1.2 Toxicity

Non-Carcinogenic Effects

Studies on the toxicity of aluminum through inhalation, oral or dermal exposure are limited and often contradictory or provide limited information (i.e., do not specify the dose, form or bioavailability of aluminum or the identity and concentration of other compounds with concomitant exposure), making evaluations of aluminum-specific toxicity difficult.

Oral Exposure

Aluminum is commonly found in the diet of humans and animals; it is used in food additives, packaging, and present in drinking water and medication. Normal dietary intake in humans is estimated as being 0.10 to 0.12 mg/kg/day in adults (ATSDR 2008). Toxicity to humans following oral exposure to aluminum phosphide has been reported (including cardiovascular and gastrointestinal effects following acute accidental or suicide-attempt exposure), but is considered to be the result of the formation of highly toxic phosphine gas, rather than the aluminum itself. Numerous oral toxicity studies have been performed on animals, but unfortunately the base rate of dietary intake is often not reported, which underestimates total intake concentrations. Some effects of aluminum following oral intake in rodents include ataxia, splaying and dragging of hind limbs, and paralysis in maternal mice exposed to approximately 184 mg/kg/day or 250 mg/kg/day as aluminum lactate during gestation and lactation (ATSDR 2008).

Oral uptake studies of aluminum found NOAELs ranging from 0.6 mg/kg/day in female mice and male and female rats, following 5 or 7 weeks (mice) and 2.5 years (rats) exposure to aluminum chloride and aluminum potassium sulfate (administered in food and water) to 979 mg/kg/day in mice (administered in food as aluminum potassium sulfate over 20 months) (ATSDR 2008). Oral exposure LOAELs ranged from 130 mg/kg/day (administered as aluminum lactate in food over a 6-week period) in female mice (causing decreased total, vertical and horizontal neurological activity; decreased diurnal period and shortened activity periods) to 770 mg/kg/day

(administered once via gavage as aluminum chloride) in male mice (corresponding to an LD_{50}) (ATSDR 2008).

Inhalation Exposure

Toxicity following inhalation exposure has been found in occupational studies and consists of wheezing, dyspnea and impaired lung function (following exposure to unspecified aluminum fumes) and pulmonary fibrosis following exposure to aluminum-containing dusts, all of these exposures co-occur with exposure to numerous other toxic chemicals (ATSDR 2008). One individual chronically exposed to aluminum dust and metallic aluminum showed reversible effects on the lungs (sarcoid-like epitheloid granulomas). Neurological effects for chronically-exposed workers are limited to sub-clinical effects including memory impairment, electroencephalogram changes, eye-hand coordination, and motor skills, although these studies did not adequately characterize aluminum exposure, and their validity is questioned (ATSDR 2008). Hamster studies have found absolute lung weight increases following 3-day exposure to ≥ 7 mg/m^3, and correspond to similar findings in rabbits following 43 mg/m^3 exposure for 5 days. Reduction in body weight was observed in a 24-month study in rats exposed to 6.1 mg/m^3 as aluminum chlorhydrate.

Inhalation uptake studies with experimental animals found NOAELs for aluminum ranging from 0.061 mg/m^3 following long-term exposure to aluminum chlorohydrate in rats and guinea pigs to 100 mg/m^3 following 5 days exposure to aluminum powder, for 4 hours/day in male rats (ATSDR 2008). Inhalation LOAELs ranged from 0.61 mg/m^3 in rats and guinea pigs from exposure to aluminum chlorohydrate over 6 months, 5 days/week, for 6 hours/day (causing increases in alveolar macrophages and lesions in the lungs in both species) to 200 mg/m^3 in male rats following 5 days exposure to aluminum powder (causing multifocal microgranulomas in the lungs) (ATSDR 2008).

Dermal Exposure

Dermal toxicity studies are limited; effects of aluminum exposure include skin damage in female mice, rabbits and large white pigs following application of 10% aluminum chloride (0.005 to 0.1 g Al) or aluminum nitrate (0.006 to 0.013 g Al), but not other forms following a 5-day exposure study (ATSDR 2008). Studies on increased incidences of Ahlzheimer's disease following application of aluminum-containing deodorants in humans have found a trend ($p=0.03$) toward a higher risk with increasing use of these deodorants (ATSDR 2008).

Reference Dose for Chronic Oral Exposure (RfD)

Health Canada (2009) has not developed a tolerable daily intake for aluminum (Health Canada 2009).

A U.S. EPA provisional RfD of 1 mg/kg/day has been derived for aluminum (U.S. EPA 2012b). An RfD for aluminum is not available on U.S. EPA IRIS (2012a).

Reference Concentration for Chronic Inhalation Exposure (RfC)

Health Canada (2009) has not developed a tolerable concentration of aluminum.

An RfC is not available for aluminum on U.S. EPA IRIS, however a provisional RfC is provided by U.S. EPA (2012b). The inhalation chronic reference concentration is 5.00E-03 mg/m^3. The RfC is based on a study by Hosovski et. al. (1990) which studied the critical effects of aluminum on the psychomotor and cognitive impairment of humans. The RfC has a modifying factor of 1 and has an uncertainty factor of 300. The overall confidence in the RfC is low to medium.

Carcinogenic Effects

Health Canada (2009) and U.S. EPA (2012a) have not assessed the carcinogenicity of aluminum.

Carcinogenic Risk from Oral Exposure

An oral slope factor for aluminum is not available (Health Canada 2009 and U.S. EPA 2012a).

Carcinogenic Risk from Inhalation Exposure

An inhalation slope factor for aluminum is not available (Health Canada 2009; U.S. EPA 2012a).

III.3.1.3 Summary of TRVs Used

Oral Chronic RfD	1 mg/kg/day	U.S. EPA 2012b
Inhalation RfC	5.0E-3 mg/m^3	U.S. EPA 2012b
Oral Slope Factor	not available	HC 2009; U.S. EPA 2012a
Inhalation Slope Factor	not available	HC 2009; U.S. EPA 2012a

III.3.2 Antimony

III.3.2.1 Pharmacokinetics

Oral Exposure

No studies have been found on the absorption, distribution or excretion of antimony via oral exposure, although animal studies have found at least some forms will be absorbed across the gastrointestinal tract, with estimates for antimony tartrate and trichlorine ranging from 2% to 7% (ATSDR 1992). The rate of gastrointestinal absorption in humans is 10% for antimony tartrate and 1% for all other forms (ATSDR 1992). Distribution in animals following oral exposure to antimony occurs in the gastrointestinal tract, the liver, kidneys, bones, lungs, spleen and thyroid. Dose-response rates of uptake have not been observed and antimony uptake demonstrates a plateau of absorption. Animal studies have found antimony is partially absorbed from the gastrointestinal tract, with either urine or feces as the main route of excretion, depending on the ligand form.

Inhalation Exposure

Absorption via inhalation exposure in humans has not been characterized, although the presence of antimony in the blood and urine following occupational exposure to dust suggest absorption does occur across the lungs (ATSDR 1992). Particle size (i.e., ligand form) determines the rate of uptake, in addition, mucocilary clearance (swallowing) accounts for gastrointestinal absorption following inhalation exposure. Antimony is mainly transported in the bloodstream and is distributed to various tissues. Excretion occurs via urine in humans, while animals are known to eliminate antimony via feces as well. Animal studies have also shown elimination of antimony (in the form of antimony tartrate) occurs in two phases: the first phase (accounting for 90% of the initial dose) occurs in 24 hours, with the half-life of the second phase taking 16 days (ATSDR 1992).

Dermal Exposure

Dermal exposure studies were not found for humans, and only limited information was available for animals (ATSDR 1992). From these studies it is known that at least some antimony is absorbed through the skin; accumulation likely occurs in the liver, kidney, skeleton, spleen and fur; and parenteral exposure studies infer excretion occurs via the urine and feces (ATSDR 1992).

III.3.2.2 Toxicity

Non-Carcinogenic Effects

Oral Exposure

One effect to humans following oral exposure to antimony includes vomiting (after an individual ingested approximately 0.53 mg/kg potassium antimony tartrate) (ATSDR 1992). Reported animal effects include: vomiting, severe diarrhea, mild hematological alterations, and severe weight loss (ATSDR 1992).

Oral uptake studies of antimony found NOAELs ranging from 0.0748 mg/kg/day for antimony trichloride in rats (administered for 30 days via water, focusing on cardiological effects) up to 16,714 mg/kg/day for antimony trioxide administered for one day via food (ATSDR 1992). Oral exposure LOAELs ranged from 0.0748 mg/kg/day in rats for antimony trichloride (administered in water over 21 to 81 days), causing decreased hypotensive responses in newborns and decreased maternal weight gain to 16,714 mg/kg/day in rats for antimony trioxide (administered for one day via food) causing diarrhoea (ATSDR 1992).

Inhalation Exposure

Occupational studies on the toxicity of antimony have found exposure to antimony trioxide and/or pentoxide dust (at concentrations ≥ 8.87 mg/m^3) caused pneumoconiosis (lung inflammation due to dust inhalation). A second occupational study found unspecified concentrations of antimony cause pulmonary alterations (including airway obstruction, bronchospasm and hyperinflation) and chronic bronchitis, chronic emphysema, inactive tuberculosis, pleural adhesions and irritation (ATSDR 1992). Other workplace-related effects included increased blood pressure, degenerative changes in myocardium and electrocardiogram abnormalities, and gastrointestinal, ocular and reproductive effects. Toxicity in animals includes respiratory effects such as pneumoconiosis and increased alveolar macrophages leading to fibrosis, parenchmatous and fatty degeneration of the liver, tubular dilation of the kidneys, ocular conjunctivitis and dermatosis of the eyes, a decreased number of offspring and 67% failure rate for conception (ATSDR 1992). Lung cancer has also been observed in rats exposed to 4.2 and 36 mg/m^3 antimony trioxide for one year.

Inhalation uptake studies of the effects of antimony on experimental animals found NOAELs ranging from 3.81 mg/m^3 for antimony trisulfide following a 7 weeks of exposure, over 7 hours/day, for 5 days/week in dogs (focusing on cardiological effects) to 799 mg/m^3 for antimony as stibine in studies on rats and guinea pigs over a 30 minute duration (ATSDR 1992). Inhalation LOAELs for antimony ranged from 0.07 mg/m^3 for antimony trioxide from a 1-year, 5 days/week, 6 hours/day exposure

duration study in rats (chronic inflammation and proliferation of macrophages and hyperplasia in peribronciolar lymphnodes) to 1395 mg/m^3 from 30 minutes exposure duration to antimony as stibine in rats and guinea pigs (causing increased mortality and pulmonary edema) (ATSDR 1992).

Dermal Exposure

No studies were found on the toxicity of antimony to humans through dermal exposure, although animal studies have found lung hyperemia in rabbits exposed to 6 to 8 applications of antimony trioxide paste, localized edema in rabbits given 6685 mg/kg antimony trioxide and eye irritation in rabbits following direct application of 79 to 100 mg antimony trioxide and thioantimonate to the eyes (ATSDR 1992).

Dermal uptake studies with antimony are limited. Dermal NOAEL values included 209 mg and 20,900 mg in rabbits, for antimony pentasulfides and trioxides, respectively (following 13-week and one-day exposure durations) (ATSDR 1992). Dermal LOAELs (both for antimony trioxide) ranged from 79.2 mg, causing mild eye irritation in rabbits, to 6685 mg/kg causing edema in rabbits. Both studies were over a 1-day exposure period (ATSDR 1992).

Reference Dose for Chronic Oral Exposure (RfD)

A tolerable daily intake for antimony is not available from Health Canada (2009).

The U.S. EPA RfD for antimony is 4E-04 mg/kg/day based on a chronic rat oral bioassay with longevity, blood glucose and cholesterol levels as the critical effects (U.S. EPA 2012). A NOAEL value was not determined, but the LOAEL was 0.35 mg/kg body weight/day. The uncertainty factor is 1000 based on interspecies conversion (10), protection of sensitive subpopulations (10), and a factor of 10 because a NOAEL was not established. Confidence in the study, database and RfD value is reported as low based on the use of only one test species, one dose level and lack of a NOAEL and the database is deficient due to the lack of adequate oral exposure studies (U.S. EPA 2012).

The oral tolerable daily intake (TDI) from the Netherlands National Institute for Public Health and the Environment (RIVM 2009) of 0.006 mg/kg /day was used in this assessment. RIVM derived a TDI from a no observed adverse effect level (NOAEL) of 6 mg/kg/day for decreased body weight, food intake, and water intake observed in rats exposed to antimony potassium tartrate in drinking water for 90 days. A total uncertainty factor of 1000 was applied to the NOAEL (factors of 10 each for intra- and inter-species variation and use of a sub-chronic study), resulting in a TDI of 0.006 mg/kg /day. RIVM (2009) noted that the TDI applies to soluble antimony compounds. The RIVM (2009) value was chosen over the U.S. EPA value because

it is based on a more recent study (1998 study versus a 1975 study used by the U.S. EPA); the 1998 study was not available when the U.S. EPA last updated their evaluation in 1991.

Reference Concentration for Chronic Inhalation Exposure (RfC)

A tolerable concentration for antimony is not available from Health Canada (2009).

The RfC for antimony trioxide is 2E-4 mg/m^3 based on a 1-year chronic inhalation exposure study in rats (with a critical effect of pulmonary toxicity and chronic interstitial inflammation) (U.S. EPA 2012). A 10% relative increase in effects was used to derive the benchmark concentration (BMC_{10}) of 0.87 mg/m^3, which was then converted to a human equivalent concentration (BMC(HEC)) of 0.074 mg/m^3. The uncertainty factor for this RfC is 300 (based on the protection of sensitive subpopulations (10), interspecies extrapolation following dosimetric scaling (3), database deficiencies (3), and for a less-than-lifetime exposure duration (3)). The confidence in the study, database and RfC value is medium based on the lack of lifetime data and reproductive/ developmental studies in humans (U.S. EPA 2012).

Carcinogenic Effects

Health Canada and the U.S. EPA have not classified antimony for carcinogenicity (Health Canada 1996; U.S. EPA 2012).

Carcinogenic Risk from Oral Exposure

An oral slope factor is not available for antimony or antimony trioxide (Health Canada 2009 and U.S. EPA 2012).

Carcinogenic Risk from Inhalation Exposure

An inhalation slope factor is not available for antimony or antimony trioxide (Health Canada 2009 and U.S. EPA 2012).

III.3.2.3 Summary of TRVs Used

Oral Chronic RfD (Sb) 6E-3 mg/kg/day decreased body weight, food and water intake RIVM 2009

Inhalation RfC (SbO_3) 2E-4 mg/m^3 respiratory effects U.S. EPA 2012

Oral Slope Factor not available Health Canada 2009; U.S. EPA 2012

Inhalation Slope Factor not available Health Canada 2009; U.S. EPA 2012

III.3.3 Arsenic

III.3.3.1 Pharmacokinetics

Oral Exposure

Human studies show that both arsenates and arsenites are well absorbed across the gastrointestinal tract; evidence is provided by the small percentage of arsenic eliminated directly in feces (the absorption of insoluble arsenic salts following oral exposure is much lower than that of arsenates and arsenites). Absorption of arsenates and arsenites through the gastrointestinal tract is estimated to be on the order of 95%, and urinary excretion ranges from 55% to 80% of the daily intake rate. Animal studies on arsenic bioavailability indicate the absorption of arsenic ingested in dust or soil is considerably less than the absorption of arsenic from ingested salts (ATSDR 2000). The bioavailability of arsenic from soil is reduced by low solubility and accessibility due to the presence of other soil matrix components (Davis 1992 as cited in ATSDR 2000). A study where arsenic-contaminated soil from mining and smelting sites was incubated in simulated stomach acid found that only a portion of the arsenic (ranging from 3% to 50%) became soluble. The estimates of soluble or bioavailable arsenic agreed well with the bioavailability estimates for the same soil samples.

Inhalation Exposure

Inhaled airborne arsenic is rapidly deposited in the respiratory tract and absorbed into the bloodstream (Hrudey 1996). The size and solubility of the aerosols containing arsenic influence the extent of deposition, retention and clearance from the lungs. A study of arsenic absorption by inhalation in a group of lung cancer patients indicated that about 40% of the arsenic in the cigarette smoke was deposited in the lungs, and absorption was estimated to be 75% to 85%, resulting in a total absorption of about 30% to 40% of the arsenic in the cigarette smoke. The American Conference of Governmental Industrial Hygienists (ACGIH 2001) indicates approximately 77% of airborne particles with a diameter of 10 μm are generally deposited in the respiratory tract. The World Health Organization (WHO 1981; as cited in ATSDR 2000) indicates that the bioavailability of deposited, water soluble As(III) may be as high as 85% to 90%. Smelter workers exposed to arsenic trioxide absorbed about 40% to 60% of the estimated inhaled dose (Vahter et al. 1986; as cited in ATSDR 2000). Vahter et al. (1986; as cited in ATSDR 2000) also demonstrated that arsenic urinary elimination is relatively rapid, as urinary arsenic excretion increased within a few hours of the smelter workers beginning work for the week, and decreased over the weekend.

Dermal Exposure

Several studies have been conducted on the absorption of inorganic arsenic through skin in humans and animals. Wester et al. (1993; as cited in Hrudey et al. 1996) studied the absorption of arsenic from water and soil in both humans and monkeys. Absorption of arsenic and arsenic mixed with soil by the skin was tested using radiolabeled arsenic (H_3AsO_4) on cadavers. Only 0.8% of the arsenic in water solution was found to penetrate human skin in comparison to the 2% to 6% that penetrated the monkey abdominal skin. Three (3) to 4.5% of the arsenic mixed with soil was absorbed by the monkey skin, while 1.9% was absorbed by human skin.

III.3.3.2 Toxicity

Non-Carcinogenic Effects

Oral Exposure

Respiratory effects such as pulmonary edema, respiratory distress and hemorrhagic bronchitis have been reported following cases of acute oral arsenic poisoning resulting from the consumption of doses of 8 mg/kg. These effects may be secondary effects resulting from damage to the cardiovascular system. High oral doses of arsenic have resulted in cardiac arrhythmias.

Blackfoot disease (the loss of circulation in the fingers and toes) is the primary effect of chronic arsenic exposure on the cardiovascular system. Blackfoot disease has been demonstrated in an area of Taiwan with elevated arsenic levels in drinking water. However, similar but less severe cases of peripheral vascular disease such as Raynaud's disease and gangrene of the fingers and toes have been observed in populations chronically exposed to elevated arsenic concentrations in drinking water in Bangladesh, Mexico and Chile (ATSDR 2000). Peripheral neuropathy, characterized by numbness in hands and feet, is the most common neurological side effect of chronic oral arsenic exposure. Symptoms improve after the exposure stops, but recovery tends to be slow and incomplete (ATSDR 2000).

Gastrointestinal distress results from oral consumption of inorganic arsenic and symptoms typically include nausea, vomiting and diarrhoea (ATSDR 2000). These symptoms are the result of irritation of the gastrointestinal mucosa and subside several days after the exposure. Depression of the red blood cells (anemia) or white blood cells (leukopenia) is frequently observed in humans exposed orally to arsenic (ATSDR 2000). Liver cell damage has been noted only in a few cases of acute arsenic poisoning and is not usually associated with chronic, low level oral arsenic exposure. Skin lesions such as hyperkeratinization of the skin on the palms of the hands and soles of the feet, formation of corns and warts and hyper- or

hypopigmentation of the skin are the first clinical signs of chronic oral arsenic exposure (ATSDR 2000).

There is suggestive evidence that arsenic may cause developmental effects. One case of a mother who ingested arsenic at week 30 of pregnancy resulted in the birth of a premature infant who died several days later. High arsenic was found in the baby's liver, kidney and brain. The infant demonstrated severe pulmonary hemorrhaging which was attributed to the arsenic (Lugo et al. 1969 in ATSDR 2000). More recent studies on the relationship between normal levels of arsenic in drinking water and congenital heart defects and spontaneous abortions were inconclusive because of small population sample sizes and the presence of multiple contaminants (ATSDR 2000). Exposure to arsenic dusts in the workplace has resulted in cases of contact dermatitis. Repeated contact to arsenic dust may lead to arsenic sensitization in occupational settings.

Studies have not identified human subpopulations with a particular susceptibility to arsenic. However, it is possible people with a decreased ability to methylate arsenic in the liver, which is the mechanism by which arsenic toxicity is reduced, may be more susceptible (ATSDR 2000). Reduced capacity of the liver to methylate arsenic could be due to lack of choline or methionine in the diet. Decreased methlylation capacity of the liver does not seem to result from liver disease, at least at low levels of arsenic exposure (ATSDR 2000).

Inhalation Exposure

Most information on human inhalation exposure to arsenic derives from occupational settings such as smelters and chemical plants, where the predominant form of airborne arsenic is arsenic trioxide dust. Workers exposed to arsenic dusts in air have experienced irritation to the mucous membranes of the nose and throat that may lead to laryngitis, bronchitis, or rhinitis. There is some evidence that inhaled inorganic arsenic may also result in cardiovascular effects. Cohort mortality studies of arsenic-exposed workers at various smelters have all reported increased risk of mortality from cardiovascular disease (i.e., ischemic heart disease and cerebrovascular disease) and peripheral neurological effects. However, several factors (copper and other metal exposure) may have confounded the conclusions. Several case studies have reported nausea, vomiting, and diarrhea in workers with acute arsenic poisoning following occupational inhalation exposure. Dermatitis (hyperpigmentation, folliculitis, and superficial ulcerations) was observed in 11 employees in one department of a Malaysian tin smelter (total of 500 employees in the plant) exposed to mean arsenic oxide concentrations of 0.005 to 0.014 mg As_2O_3/m^3. There are also several studies suggesting that inhalation exposure to arsenic may have caused increased incidences of spontaneous abortion, significant

increases in incidences of congenital malformations and significantly decreased average birth weight in female smelter employees in Sweden (ATSDR 2000).

No studies were located regarding respiratory effects in humans exposed to organic arsenics. Short-term exposure of rats and mice to high concentrations of arsenic caused respiratory distress, and necropsy of animals revealed bright red lungs with dark spots. Respiratory distress was also observed in rats and mice exposed to high levels of arsenic.

Dermal Exposure

Several studies of humans exposed to arsenic dusts in the workplace have reported inorganic arsenic (usually arsenic trioxide) can cause contact dermatitis (erythema and swelling, with papules and vesicles). Application of organic arsenic to the skin of rabbits was reported to result in mild dermal irritation.

Tolerable Daily Intake (TDI) and Reference Dose (RfD) for Chronic Oral Exposure

A tolerable daily intake value is not available for arsenic and its inorganic compounds from Health Canada (Health Canada 2009).

An increased incidence of skin effects (hyperpigmentation and keratosis) and vascular disease such as Blackfoot disease was observed in people who consumed water containing elevated arsenic levels for long periods of time. U.S. EPA (2012) identified an RfD based on human populations with Blackfoot disease; a NOAEL was determined to be 0.009 mg/L (or 8E-4 mg/kg/day) while a LOAEL was found to be 0.17 mg/L arsenic (~0.014 mg/kg/day). The U.S. EPA has developed an oral reference dose of 3E-4 mg/kg/day by dividing the NOAEL by an uncertainty factor of 3 in order to account for data limitations with respect to the potential effects of arsenic on reproductive toxicity (U.S. EPA 2012).

Tolerable Concentration (TC) and Reference Concentration (RfC) for Chronic Inhalation Exposure

A tolerable concentration value is not available for arsenic and its inorganic compounds from Health Canada (Health Canada 2009).

A reference concentration for chronic inhalation exposure is not available for arsenic from U.S. EPA IRIS (U.S. EPA 2012).

RIVM (2001) derived a tolerable concentration in air (TCA) of 0.001 mg/m^3. RIVM (2001) indicated that lung cancer occurs in humans at concentrations greater than 0.01 mg/m^3, but that the mechanism for tumours is not directly genotoxic, and therefore a threshold exists for this effect. RIVM (2001) therefore determined that

this threshold value was a TCA, not a cancer risk value, and applied an uncertainty factor of 10 to account for intra-human variability.

Carcinogenic Effects

Health Canada has classified arsenic and its inorganic compounds as Group I – carcinogenic to humans, based on documented human carcinogenicity following inhalation and oral exposure (Health Canada 1993). Human cancers associated with occupational exposure to arsenic compounds include lung, stomach, colon, liver, and urinary system cancers, with inhalation exposure being the most significant pathway (Health Canada 1993). Oral exposure has also been characterized, and a Taiwanese study found skin cancers associated with arsenic ingested in drinking water (Health Canada 1993).

The U.S. EPA has classified inorganic arsenic as Group A – known human carcinogen by inhalation and oral exposure based on increased lung cancer mortality in multiple human populations (exposed primarily through inhalation), increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder), and increased incidence of skin cancer in populations consuming drinking water with high levels of inorganic arsenic. (U.S. EPA 2012).

Carcinogenic Risk from Oral Exposure

Health Canada has derived an oral slope factor of 1.8 $(mg/kg/day)^{-1}$ (Health Canada 2009) based on the critical endpoint of skin cancer. The slope factor was derived based on an epidemiological study where humans were naturally exposed to arsenic in drinking water for up to 60 years. Overall, using a 1% increase in risk, the unit risks associated with ingestion of 1 µg/L of arsenic in drinking water were estimated to range from 3.06E-06 to 3.85E-05, with 95% upper bounds ranging from 6.49E-06 to 4.64E-05 (Health Canada 2009). The most sensitive endpoint for both males and females was lung cancer. The overall unit risk associated with the ingestion of arsenic in drinking water was reported as a range, given that lifetime exposure to arsenic results in more than one cancer endpoint in different individuals. The above unit risk range has the liver cancer unit risk (3.06E-06) as its lower bound and the lung cancer unit risk (3.85E-05) as its upper bound (Health Canada 2009).

The U.S. EPA has developed a cancer slope factor of 1.5 $(mg/kg/day)^{-1}$ for oral exposure, which is based on studies that indicate an increased incidence of skin cancer in Taiwanese populations that were orally exposed to arsenic in drinking water (Tseng et al. 1968; Tseng 1977 in U.S. EPA 2012).

Carcinogenic Risk from Inhalation Exposure

For arsenic, Health Canada has derived an inhalation unit risk of 6.4 per mg/m^3 (Health Canada 2009) based on an epidemiological study where mortality as of 1976 was documented in a cohort of 2802 smelter workers employed for at least one year between 1940 and 1964 was followed. Based on monitoring data and conversion levels of arsenic in urine to airborne concentrations, it was found that the standardized mortality ratios for respiratory cancer increased with increased cumulative exposure to arsenic.

III.3.3.3 Summary of TRVs Used

Tolerable Daily Intake	not available	Health Canada 2009	
Oral Chronic RfD	3E-04 mg/kg/day	skin and vascular effects	U.S. EPA 2012
Tolerable Concentration	not available	Health Canada 2009	
Inhalation RfC	not available	U.S. EPA 2012	
Tolerable Concentration	0.001 mg/m^3	lungs	RIVM 2001
Oral Slope Factor	1.8 (mg/kg/day)$^{-1}$	skin cancer	Health Canada 2009
Inhalation Unit Risk	6.4 (mg/m^3)$^{-1}$	lung cancer	Health Canada 2009

III.3.4 Cadmium

III.3.4.1 Pharmacokinetics

Oral and Inhalation Exposure

The major site of cadmium absorption in humans following inhalation is the alveoli of the lung, while the majority of ingested cadmium tends to pass through the gastrointestinal tract without being absorbed and is excreted in the feces (ATSDR 1999). Absorbed cadmium from the lungs and gastrointestinal tract tends to be excreted very slowly and is found equal proportions in the urine and feces (ATSDR 1999). The two main target organs for cadmium are the kidney and liver. The half-life of cadmium in the human body is very long; an estimated half-life for cadmium in the kidney ranges from 6 to 38 years and the liver from 4 and 19 years (ATSDR 1999). The placenta may act as a partial barrier to fetal cadmium exposure (ATSDR 1999). Cadmium is not metabolized, but is bound to proteins and other

molecules; in particular, cadmium binds to albumin (a protein) in the bloodstream and is transported to the liver (ATSDR 1999). Once cadmium enters the liver it becomes bound to another protein called metallothionein and is re-released to the bloodstream. The metallothionein-bound cadmium is then filtered by the kidney glomerulus and is reabsorbed by the proximal tubule cells. Lysozymes (strong enzymes) degrade the cadmium-metallothionein complex and allow free cadmium to be released in the kidney. The free cadmium initiates the synthesis of metallothionein in the proximal tubule cells and can also cause excessive damage to the kidneys.

Dermal Exposure

There is currently not enough information to determine the potential absorption of cadmium via the dermal route of exposure (ATSDR 1999). Based on the limited information it appears that very little cadmium is absorbed through the skin

III.3.4.2 Toxicity

Non-Carcinogenic Effects

Oral Exposure

High acute oral doses of cadmium (0.07 mg/kg) may cause gastrointestinal effects such as nausea, vomiting, and abdominal pain (Nordberg et al. 1973 in ATSDR 1999). Anemia can result from oral or inhaled cadmium, and if transported to the gut, cadmium will cause reduced iron absorption. However, in populations with adequate dietary intake of iron, it is likely that cadmium-induced anemia will not be a problem, as the increased intake will compensate for decreased absorption (ATSDR 1999). Chronic cadmium exposure coupled with poor nutrition can lead to changes in the way which the kidney metabolizes vitamin D, causing painful bone diseases such as osteomalacia and osteoporosis (ATSDR 1999). Cadmium causes kidney damage, particularly to the renal tubules in the early stages and as the disease progresses or dose increases, glomerular damage is also observed (ATSDR 1999). There are limited data to suggest that cadmium exposures in pregnant women may result in decreased birth weight in their babies (ATSDR 1999). Populations that may be unusually susceptible to cadmium exposure are those with a genetic predisposition to lower inducibility of metallothionein, the enzyme that sequesters cadmium. Increased absorption of cadmium from the gastrointestinal tract may result in individuals with depleted levels of calcium or iron resulting from dietary deficiencies (ATSDR 1999). Infants and children may have increased uptake of cadmium via the gastrointestinal tract and higher concentrations of cadmium in the bone.

Inhalation Exposure

Inhalation of cadmium at 5 mg/m^3 has resulted in pulmonary edema, tracheobronchitis and pneumonitis in humans (ATSDR 1999).

Dermal Exposure

Cadmium appears to have a relatively low dermal toxicity based on occupational studies where workers who were exposed to high levels of cadmium dust did not report any dermal effects. In addition, cadmium does not appear to cause sensitization by repeated dermal contact (ATSDR 1999).

Reference Dose for Chronic Oral Exposure (RfD)

The U.S. EPA (2012) has developed oral reference doses for cadmium for food and water. The oral reference dose for food is 1E-3 mg/kg/day and for water is 5E-4 mg/kg/day (U.S. EPA 2012). The highest cadmium level in the human kidney that does not produce proteinuria has been determined to be 200 µg/g of wet kidney cortex. A toxicokinetic model was used to determine the level of chronic oral exposure that would result in a cadmium kidney concentration of 200 µg /g of wet kidney cortex. The toxicokinetic model assumes that 0.01% of the body cadmium kidney burden is eliminated daily and that absorption of cadmium from food and water are 2.5% and 5%, respectively. A NOAEL for chronic cadmium exposure via water and food was determined to be 0.005 and 0.01 mg/kg/day, respectively. An uncertainty factor of 10 to account for human variability was applied to the NOAELs to develop the reference doses for water and food.

Reference Concentration for Chronic Inhalation Exposure (RfC)

ATSDR (2012) provides an inhalation minimal risk level based on kidney effects in humans (1E-05 mg/m^3).

Carcinogenic Effects

Epidemiological studies demonstrate increased incidence of lung cancer in workers exposed to cadmium via the inhalation route, however, the studies did not control for factors such as smoking and simultaneous exposures to other metals so the causal relationship is somewhat controversial. Oral exposure to cadmium has not been associated with cancer in humans, however available studies are inadequate to assess carcinogenicity. The U.S. EPA has classified cadmium as a Group B1 – probable human carcinogen based on limited human and sufficient animal data (U.S. EPA 2012).

Carcinogenic Risk from Oral Exposure

An oral slope factor for cadmium is not available at this time (U.S. EPA 2012).

Carcinogenic Risk from Inhalation Exposure

The inhalation unit risk for cadmium from the U.S. EPA IRIS (1.8 per mg/m^3) was used in the assessment (U.S. EPA 2012). The U.S. EPA inhalation unit risk is based on a human study where male smelter workers were exposed to cadmium via inhalation in the workplace.

Health Canada also provides an inhalation unit risk (9.8 per mg/m^3) based on a chronic inhalation study in rats which developed lung cancer following exposure to cadmium chloride aerosols for 23 hours/day, 7 days per week, for 18 months (Health Canada 2009).

Although the unit risk provided by the U.S. EPA (2012) is lower (i.e., less conservative) than that provided by Health Canada (2009), it was considered appropriate to use the U.S. EPA (2012) unit risk as it is based on a human study.

III.3.4.3 Summary of TRVs Used

Oral chronic RfD	1E-3 mg/kg/day (food)	proteinuria	U.S. EPA 2012
Oral chronic RfD	5E-4 mg/kg/day (water)	proteinuria	U.S. EPA 2012
Inhalation RfC	1E-5 mg/m^3		ATSDR 2012
Oral Slope Factor	not available at this time		U.S. EPA 2012
Inhalation Unit Risk	1.8E-3 (μg/m^3)$^{-1}$	lung cancer	U.S. EPA 2012

III.3.5 Cobalt

III.3.5.1 Pharmacokinetics

Oral Exposure

Oral consumption of cobalt results in absorption by the gastrointestinal tract; absorption ranges from 18% to 97% in humans and is dependent upon the dose and form of cobalt as well as the nutritional status of the individuals exposed. Cobalt absorption tends to increase in subjects with iron deficiencies in their diet. Elimination in the feces is the primary excretion method for oral cobalt exposures.

Inhalation Exposure

Inhaled cobalt particles accumulate in the respiratory tract, and particle size determines whether accumulation occurs in the upper or lower respiratory tract. Larger particles (> 2 µm) tend to accumulate in the upper respiratory tract, while smaller particles are transferred to the lower respiratory system. From the lungs, cobalt particles either dissolve into the bloodstream or are transferred to the gastrointestinal tract by actions such as swallowing. Approximately 50% of the cobalt transferred to the gastrointestinal tract is actually absorbed and the rest is eliminated in the feces. About 50% of the portion of the initial lung burden can remain up to 6 months after exposure (Foster et al. 1989 as cited in ATSDR 1992).

Dermal Exposure

Human dermal exposure (hands only) to hard metal dust (~5% to 15% cobalt metal, 95% to 85% tungsten carbide) for 90 minutes showed an increase in urinary cobalt levels by an order of magnitude up to 60 hours after the exposure. The absorption of $2.2x10^{-5}$ mg ^{60}Co/kg as cobalt chloride through the intact or abraded skin of guinea pigs revealed absorption to be very limited through intact skin (less than 1%), while absorption through abraded skin was almost 80%. No studies were available regarding distribution in humans or animals after dermal exposure to cobalt. Excretion of cobalt in hamsters occurs primarily in the urine within 48 hours after a single dermal exposure.

III.3.5.2 Toxicity

Non-Carcinogenic Effects

Oral Exposure

Cobalt is an essential element for humans and is required for the production of vitamin B_{12} (ATSDR 1992). It is found in most body tissues, with the highest concentrations occurring in the liver, kidney and bones. Vitamin B_{12} is a coenzyme in many biological reactions including the production of red blood cells, and cobalt has been used to treat anemia. The Recommended Dietary Allowance (RDA) for vitamin B_{12} for adults is 2.4 µg/day, (this amount contains 0.1 µg cobalt) (ATSDR 1992). Oral exposure to high levels of cobalt has occurred in humans who consumed beer containing cobalt salts; in the 1960s, cobalt salts were added to beer to improve its foaming qualities. This practice has since been discontinued as it led to several deaths among heavy beer drinkers (8 to 30 pints per day) who consumed doses ranging from 3 to 10 mg cobalt/per day. Less serious effects associated with the consumption of beer containing cobalt compounds included nausea, vomiting and diarrhea. Increased production of red blood cells also occurs in humans after oral exposure to cobalt. Decreased uptake of iodine by the thyroid gland has been

observed in humans exposed to short-term doses of 1 mg/kg body weight/day or longer-term doses of 0.54 mg/kg body weight/day.

Developmental effects were not observed in babies born to mothers who were taking cobalt-containing medication to regulate anemia while pregnant (Holly 1955 as cited in ATSDR 1992). Reproductive effects were not observed in the people who died after exposure to high cobalt levels in beer. Some reproductive effects have been observed in animals (adverse effects on the testes and increased length of the estrous cycle), however, the significance of these effects for humans is not clear as the cobalt doses used in these studies were much higher than those to which humans are usually exposed.

Inhalation Exposure

Inhalation of cobalt can affect the respiratory system and if sufficient quantities are inhaled (0.003 mg/m^3), irritation, wheezing, asthma and pneumonia can result. Occupational exposure to cobalt concentrations of 0.038 mg/m^3 for six hours resulted in breathing difficulties, although these levels are approximately 10,000 to 100,000 times the typical outdoor air concentration. Individuals can also develop sensitivity to cobalt through occupational exposure to concentrations ≥0.007 mg/m^3, and subsequent exposures can result in skin rashes or asthma attacks.

Dermal Exposure

Weight loss has been reported in animal studies conducted with various cobalt compounds. Necrosis of the thymus was observed in rats exposed to 19 mg/m^3 as cobalt sulfate, and hyperplasia of the mediastinal lymph nodes was found in mice exposed to 11.4 mg/m^3. Neurological effects such as congestion in the vessels of the brain/meninges was reported in rats and mice exposed to ≥19 mg/m^3 as cobalt sulfate. Several reproductive impacts have been reported including testicular atrophy and decrease in sperm motility in rats and mice exposed to 1.14-19 mg/m^3 for subchronic and acute length exposure times, respectively.

Reference Dose for Chronic Oral Exposure (RfD)

An RfD is not available from Health Canada (2009) or on the U.S. EPA (2012), but the provisional inhalation RfD value from U.S. EPA NCEA is 0.0003 mg/kg/day (U.S. EPA 2008). The non-carcinogenic RfD for cobalt is based on the LOAEL of 1 mg/kg-day for decreased iodine uptake by the thyroid in humans. An uncertainty factor of 3,000 was applied to the LOAEL to derive the RfD; the uncertainty factor considered four separate factors (a factor of 10 for extrapolation from a subchronic to chronic study, a factor of 10 for extrapolation from a LOAEL to a NOAEL, a factor of 10 to account for lack of data on human variability and sensitive populations; and a factor of 3 to account for the lack of multigenerational studies).

Reference Concentration for Chronic Inhalation Exposure (RfC)

An RfC is not available from Health Canada (2009) or on the U.S. EPA (2012), but the provisional inhalation RfD value from U.S. EPA NCEA is 6E-6 mg/kg/day (U.S. EPA 2008). The non-carcinogenic RfC for cobalt is based on decreased pulmonary function in workers (Nemery et al. 1992, as cited in U.S. EPA 2008). The NOAEL of 5.3 µg/m^3 was adjusted for continuous exposure (to 1.9 µg/m^3) and an uncertainty factor of 300 was applied to account for three separate factors (a factor of 3 for extrapolation from an assumed subchronic to chronic study, a factor of 10 for database insufficiencies including lack of developmental inhalation studies and multigenerational studies, and a factor of 10 to account for lack of data on human variability and sensitive populations).

Carcinogenic Effects

Based on animal data, the International Agency for Research on Cancer has classified cobalt and its compounds as possibly carcinogenic for humans (Group 2B). However, a Health Canada and U.S. EPA classification on carcinogenicity are not available at this time (Health Canada 2009, U.S. EPA 2012).

One occupational study reported an increased incidence of lung cancer deaths amongst workers exposed to cobalt in comparison to a control population that had not been exposed to cobalt (ATSDR 1992). The difference between the exposed workers and the control population was not considered to be statistically significant. Additionally, the presence of characteristic lung diseases associated with occupational exposure to cobalt was not documented, although concomitant exposure to arsenic and nickel obscure the observed effects. Tumors have not been observed in humans with prostheses (i.e., artificial knees), which contain cobalt alloys.

Exposure to cobalt oxide dust in hamsters did not lead to an increased incidence of lung tumors in comparison to the control population. Intramuscular injection of cobalt oxide resulted in the production of tumors in rats but not in mice (Gilman 1962 as cited in ATSDR 1992).

Carcinogenic Risk from Oral Exposure

An oral slope factor for cobalt is not available at this time (U.S. EPA 2012).

Carcinogenic Risk from Inhalation Exposure

An inhalation slope factor for cobalt is not available from the Health Canada (2009) or U.S. EPA IRIS website (U.S. EPA 2012).

A provisional peer reviewed inhalation unit risk was available from U.S. EPA NCEA (U.S. EPA 2008). The inhalation unit risk was derived based on a two year carcinogenicity study in rats and mice, based on the dose-response relationship for statistically significant increased incidences of alveolar/bronchiolar neoplasms (adenoma and carcinoma) (U.S. EPA 2008).

III.3.5.3 Summary of TRVs Used

Oral chronic RfD	0.0003 mg/kg/day	thyroid U.S. EPA 2008
Inhalation RfC	6E-6 mg/m^3	respiratory tract U.S. EPA 2008
Oral Slope Factor	not available	Health Canada, 2009, U.S. EPA 2012
Inhalation Unit Risk9	$(mg/m^3)^{-1}$	alveolar/bronchiolar neoplasms U.S. EPA 2008

III.3.6 Iron

III.3.6.1 Pharmacokinetics

Oral Exposure

The US National Academy of Science (US NAS) estimated that the maximum bioavailability of iron in food is 18%, based on calculations of heme and non-heme iron absorption from various types of food (US NAS 2002). The primary mechanism for regulating iron levels in the human body is through changes in the amount of the iron absorbed by the gastrointestinal mucosa. Absorption of dietary iron is influenced by: the amount of iron stored in the body; the amount and chemical type of iron in ingested food; and dietary factors.

Inhalation/Dermal Exposure

No studies could be located on the pharmacokinetics of iron resulting from inhalation or dermal exposure.

III.3.6.2 Toxicity

Non-Carcinogenic Effects

Oral Uptake

Iron is an essential element, and an important component of several proteins including enzymes and hemoglobin. A large portion (approximately 67%) of the iron

in the body is found in the hemoglobin of erythrocytes circulating in the blood system. Another 25% of the iron found in the body is stored in a readily mobilizable form. The remaining iron in the body is found in the myoglobin of muscle tissue and in enzymes necessary for oxidative metabolism (US NAS 2002).

Clinical effects associated with iron deficiency include anemia, developmental delay, cognitive impairment and adverse pregnancy outcomes. Acute iron toxicity effects are well documented, primarily as the result of children who accidentally ingest iron supplements. Symptoms include gastrointestinal distress as well as cardiovascular, metabolic, neurological and hepatic alterations. It is difficult to obtain acute oral toxic doses because they are generally estimated from clinical history in overdose situations. Adverse developmental effects have not been associated with ingestion of supplemental iron intake during pregnancy. There is some controversy over whether individuals with a normal ability to eliminate iron can suffer from a chronic overload due to oral intake; however, the weight-of-evidence indicates that this is possible.

A study (Looker et al. 1998 as cited in U.S. EPA 2001) compared the dietary intake of Americans (6 months to 74 years) to biochemical indices (serum ferritin levels) from the second National Health and Nutritional Examination Survey (NHANES II) database. The results of the comparison showed that the average iron intake levels (0.15 to 0.27 mg/kg-day) consumed by the population were sufficient to protect against iron deficiency and insufficient to cause toxic effects of iron. The study concluded that the range of 0.15 to 0.27 mg/kg/day represents a NOAEL for chronic daily iron intake.

Chronic iron toxicity has been observed in people with disorders that result in excessive iron absorption, hemoglobin synthesis abnormalities, anemia or frequent blood transfusions. The US NAS (2002) indicates that the weight-of-evidence does not support a causal relationship between elevated iron intake and coronary heart disease as five out of seven studies found no association between serum ferritin and coronary heart disease (Aronow and Ahn 1996; Frey and Krider 1994; Magnusson et al. 1994; Manttari et al.; Stampfer et al. 1993 as cited in US NAS 2002). However, US NAS (2002) also indicated that elevated iron cannot be definitively ruled out as a risk factor in coronary heart disease as high serum ferritin concentration and dietary iron intake have been shown to be risk factors for myocardial infarcation in a study of Eastern Finnish men (Salonen et al. as cited in US NAS 2001). The study also showed that high serum low density lipoprotein (LDL) cholesterol levels in conjunction with elevated serum ferritin levels were a strong risk factor for myocardial infarcation. The study concluded that excessive iron concentrations promote the oxidation of LDL, which elevates the risk of myocardial infarcation (US NAS 2001). A reanalysis of the same subjects five years later confirmed these conclusions (Salonen *et al*. 1994, as cited in US NAS 2002).

The American National Academy of Science (U.S. EPA 2002) has developed the following guidelines for iron intake that account for physiological differences during different life stages.

Inhalation and Dermal Exposure

No studies were located regarding toxicity of iron from inhalation or dermal exposures

Provisional Reference Dose for Chronic Oral Exposure (RfD)

Neither a tolerable daily intake nor an RfC for iron are available (Health Canada 2009; U.S. EPA 2012).

The U.S. EPA has not formally assessed iron and therefore has not developed an oral reference dose (RfD). In 1997, it was indicated on HEAST (Health Effects Summary Tables) that there were insufficient data for a quantitative risk assessment. The Superfund Technical Support Center, National Center for Environmental Assessment at the U.S. EPA has derived a provisional references dose for iron to be used at US Superfund sites (U.S. EPA 2006). The provisional reference dose is currently the only toxicological reference dose available for the assessment of iron at contaminated sites. The toxicological properties of iron have not been assessed by ATSDR or the World Health Organization (WHO).

The U.S. EPA (2006) selected a NOAEL of 0.15 to 0.27 mg/kg/day based on study of dietary iron intake and iron status in the American population. This range of iron intake was sufficient to provide protection against iron deficiency, but insufficient to cause toxic effects. A study by Frykman et. al. (1994) was chosen by the U.S. EPA as the critical study for determining a suitable provisional RfD. In this study it was shown that daily treatment with 60 mg elemental iron/day for one month caused a statistically significant increase in gastrointestinal effects compared to placebo. To determine a LOAEL, the LOAEL of 60 mg of elemental iron per day was added to the estimated mean dietary intake of iron of 11 mg of elemental iron/day (NAS 2001) for a total of 71 mg of elemental iron/day. Based on a reference body weight of 70 kg (U.S. EPA 1987), the LOAEL for daily iron intake is 1 mg/kg/day for gastrointestinal effects. An uncertainty factor of 1.5 taking into account the following factors: 1.5 for extrapolation from a LOAEL to a NOAEL, 1 for use of sensitive individuals, 1 for less than lifetime exposure and 1 for an adequate data base. Therefore the provisional RfD (subchronic and chronic) for iron is 0.7 mg/kg/day.

Reference Concentration for Chronic Inhalation Exposure (RfC)

Neither a tolerable inhalation concentration nor an RfC for iron are available (Health Canada 2009; U.S. EPA 2012). U.S. EPA (2005a) in a review of existing data for the

purposes of deriving a provisional chronic inhalation RfC, concluded that no adequate human or animal inhalation data are available for exposure to iron or inorganic iron compounds.

Carcinogenic Effects

No studies assessing the carcinogenic effects of iron were located.

Carcinogenic Risk from Oral Exposure

An oral slope factor has not been developed for iron (Health Canada 2009; U.S. EPA 2012). U.S. EPA (2005b) concluded that the absence of adequate data demonstrating carcinogenicity to humans prohibits derivation of quantitative estimates of cancer risk for ingested iron or iron oxide.

Carcinogenic Risk from Inhalation Exposure

An inhalation slope factor has not been developed for iron (Health Canada 2009; U.S. EPA 2012). U.S. EPA (2005b) concluded that the absence of adequate data demonstrating carcinogenicity to humans prohibits derivation of quantitative estimates of cancer risk for inhaled iron or iron oxide

III.3.6.3 Summary

Oral Chronic RfD	0.7 mg/kg/day NOAEL	U.S. EPA NCEA 2006
Inhalation RfC	not available	U.S. EPA 2012
Oral Slope Factor	not available	U.S. EPA 2012
Inhalation Slope Factor	not available	U.S. EPA 2012

note: the Oral Chronic RfD is a provisional value.

III.3.7 Manganese

III.3.7.1 Pharmacokinetics

Oral Exposure

Manganese is typically found in human tissue, blood, serum and urine. Adult humans generally maintain consistent manganese levels in tissues, irrespective of manganese intake. Manganese absorption and excretion are regulated; absorption

occurs primarily through oral and inhalation exposure routes. The rate of uptake across the gastrointestinal tract is variable, but typically ranges from 3% to 5% in humans (ATSDR 2000). There does not appear to a difference in uptake across the gastrointestinal tract if the manganese is consumed in food or water (ATSDR 2000). Absorption of manganese may be age dependent as studies have shown greater uptake in young children than in adults. Dietary iron levels also influence, manganese uptake, as low iron levels lead to increased manganese uptake. Manganese metabolism is not well understood in human systems, but appears to involve oxidation of manganese from Mn(II) to Mn(III). Excretion of manganese occurs primarily via bile, although other minor routes of elimination include urine, breast milk and sweat. Manganese is removed from the blood by the liver, where it conjugates with bile and is excreted into the intestine and removed with feces.

Inhalation Exposure

Manganese may be absorbed from both the lungs and the gastrointestinal tract following inhalation of manganese dust, however the relative rates of absorption are not known (ATSDR 2000).

Dermal Exposure

Manganese uptake across intact skin is expected to be extremely limited (ATSDR 2000).

III.3.7.2 Toxicity

Non-Carcinogenic Effects

Oral Exposure

Manganese is an essential element for humans and is found widely throughout the body. Adverse health effects can be linked to both manganese deficiency as well as excessive manganese levels. Bone mineralization, protein and energy metabolism, metabolic regulation, cellular protection from free radicals are all functions that require manganese. Manganese is also a component of metalloenzymes and can act as an enzyme activator (ATSDR 2000).

Inhalation Exposure

Chronic exposure to high levels of manganese has caused permanent neurological damage in occupationally exposed miners (ATSDR 2000). Chronic exposures to lower manganese concentrations have resulted in loss of coordination and balance, as well as a decreased ability to perform rapid hand movements (ATSDR 2000). Inhalation of particulate matter containing manganese may also lead to an inflammatory response in the lungs (ATSDR 2000).

Dermal Exposure

Organic forms of manganese (maneb and mancozeb) have been found to cause allergic contact dermatitis reactions.

Reference Dose for Chronic Oral Exposure (RfD)

Health Canada (2009) has developed an oral TDI of 0.1 mg/kg/day for toddlers and children and of 0.2 mg/kg/day for adults based on a weight of evidence approach from human epidemiological and experimental studies. The toxicological endpoint upon which the oral TDI is based is Parkinsonian-like neurotoxicity. No uncertainty factors have been utilized in the derivation of the oral TDI.

Reference Concentration for Chronic Inhalation Exposure (RfC)

The U.S. EPA developed an RfC for manganese of 5E-5 mg/m^3, based on decreased neurobehavioral function found in Belgian males occupationally exposed to manganese dioxide dust in a battery factory (U.S. EPA 2012a). The decreased neurobehavioral function entailed significantly slower visual reaction time and erratic control of hand-forearm movement in the occupationally exposed population in comparison with control subjects. The battery factory workers were exposed to an integrated respirable dust (IRD) concentration, which is calculated by multiplying 8-hour TWA occupational exposures for various job classifications by the number of years that individuals had worked at the factory. A LOAEL of 0.15 mg/m^3 was determined by dividing the geometric metric mean of the IRD by the average duration of the workers' exposure. The LOAEL (HEC) was adjusted for continuous exposure (i.e., rather than 5 work days/ week) and is 0.05 mg/m^3. Several other occupational studies were evaluated and lower LOAEL values were calculated, however, manganese exposure in the other occupational studies was the result of an unknown mixture of manganese compounds and concentrations were variable over time. Therefore, the U.S. EPA decided to use a LOAEL of 0.05 mg/m^3 to derive an RfC for manganese.

An uncertainty factor of 1000 was applied to the LOAEL to derive an RfC of 5E-5 mg/m^3. The uncertainty factor consists of: a factor of 10 to account for sensitive subpopulations; a factor of 10 to account for the use of a LOAEL to set the RfC; and a factor of 10 to account for database limitations reflecting less than chronic exposure, lack of developmental data and unquantified differences in the toxicity of different forms of manganese (U.S. EPA 2012a). Confidence in the RfC is medium, reflecting medium confidence in both the primary study and the database.

Carcinogenic Effects

Health Canada (1996) has not assessed the carcinogenicity of manganese.

The U.S. EPA (2012a) has classified manganese as a Group D carcinogen – not classifiable as to human carcinogenicity, due to a lack of human data and inadequate data from animal studies. Several rodent studies have been conducted, however, none of the animals developed significantly increased number of tumors after administration of manganese in comparison to controls or in one case, evidence for a dose-relationship was marginal. Quantitative estimates of slope factors or unit risk values are not available at this time.

Carcinogenic Risk from Oral Exposure

An oral slope factor is not available for manganese (Health Canada 2009; U.S. EPA 2012a).

Carcinogenic Risk from Inhalation Exposure

An inhalation slope factor is not available for manganese (U.S. EPA 2012a).

III.3.7.3 Summary

Oral TDI (toddler)	0.1 mg/kg/day	Health Canada 2009
Oral TDI (adult)	0.2 mg/kg/day	Health Canada 2009
Inhalation RfC	5E-5 mg/m^3 neurobehavioral impairment	U.S. EPA 2012a
Oral Slope Factor	not available	U.S. EPA 2012a
Inhalation Slope Factor	not available	U.S. EPA 2012a

III.3.8 Nickel

III.3.8.1 Pharmacokinetics

Oral Exposure

Studies examining the absorption of nickel by humans found that nickel sulphate is 40 times more bioavailable if administered in water than in food (ATSDR 1997). The bioavailability of nickel also increases when administered in a soft drink, but not when given in milk, coffee, tea or orange juice (ATSDR 1997). Nickel serum levels were found to be elevated in subjects who had not eaten prior to the administration of nickel in drinking water, but this was not the case for those who were administered nickel in food. Food tends to decrease the bioavailability of nickel. Some nickel sensitive individuals were found to have decreasing nickel serum

concentrations and increasing nickel urinary concentrations with increased administered nickel concentrations (ATSDR 1997). This may be an indication that some nickel sensitive individuals can decrease nickel absorption in response to increased nickel intake. Most ingested nickel is excreted via feces, although the nickel absorbed by the gastrointestinal tract is excreted in the urine. In comparison studies of nickel doses administered with food or water 26% of the dose given in water was eliminated in the urine and 76% in the feces by the fourth day following administration (ATSDR 1997). In contrast 2% of the nickel dose administered in food was eliminated in the urine and 102% was eliminated in the feces during the same time period. Nickel can also be eliminated through hair, sweat, milk and skin.

Inhalation Exposure

Following an inhalation exposure, nickel tends to accumulate in the lungs. Absorption from the respiratory tract is dependent upon solubility of the nickel compound. Occupational exposure to nickel results in higher nickel lung burdens than the general population. Nickel-sensitized individuals had similar nickel levels in blood, urine and hair relative to non-sensitive individuals. Inhaled nickel is excreted through the urine. Studies conducted on nickel workers show that nickel urinary excretion increased towards the end of the shift and also towards the end of the work week, indicating that one fraction is removed quickly, with a second fraction removed slowly. In non-occupationally exposed people, nickel concentrations tend to be highest in lungs, thyroid and adrenal glands, kidney, heart and liver.

Dermal Exposure

No studies were located regarding excretion of nickel in humans or animals after dermal exposure to nickel.

III.3.8.2 Toxicity

Non-Carcinogenic Effects

Oral Exposure

Gastrointestinal effects were reported after workers drank water from a fountain containing nickel sulphate and nickel chloride (ATSDR 1997). Exposure doses ranged from 7.1 to 35.7 mg/kg. Symptoms include nausea, abdominal pain, vomiting and diarrhea. Neurological effects were also observed in the affected workers. Asthma may occur in a small number of sensitized individuals. However, continued oral exposure to nickel has also been shown to desensitize some individuals and prevent sensitization in other cases (ATSR 1997). Based on animal studies, a minimal dietary nickel requirement of 50 µg/kg of diet is recommended (ATSDR 1997). The average dietary nickel intake for the US populations is about 150 to 168

µg/day (70-kg person), so nickel deficiency is not expected to affect the general population (ATSDR 1997).

Inhalation Exposure

The only data available for chronic nickel inhalation exposure for humans are limited to occupational data. One of the limitations associated with the epidemiological data available is that the workers were exposed to several different forms of nickel as well as other metals and irritant gases at the same time, so frequently the observed effects cannot be attributed to a particular type of nickel and in some cases to nickel at all, if other metals were also used in the refining, mining or smelting processes (ATSDR 1997). Other lifestyle factors, such as smoking, which affect disease outcome are also not always available, limiting the conclusions that can be drawn. Respiratory effects found in nickel workers included chronic bronchitis, emphysema, and reduced vital capacity. These workers were also exposed to other metals, so it cannot be concluded that nickel is the sole causative agent of the effects observed. Asthma from primary irritation and as the result of dermal sensitization has also been documented amongst nickel workers. Nickel refinery workers with elevated urinary nickel concentrations also showed a significant increase in urinary β_2-microglobulin levels, which is indicative of tubular dysfunction in the kidneys (ATSDR 1997).

Dermal Exposure

Nickel dermatitis is the most prevalent effect of nickel and occurs in nickel-sensitized individuals (ATSDR 1997). Nickel sensitization results from extensive contact with nickel-containing material such as jewelry, coins, dental braces, stainless steel. Contact dermatitis may also result from occupational exposure. Once an individual has been sensitized to nickel, subsequent exposure (though inhalation, ingestion, or dermal contact) to low levels of nickel may cause a reaction. Populations that are unusually susceptible to nickel are those people already sensitive to nickel due to prolonged contact. Subsequent exposures may result in an allergic reaction. A greater number of women tend to be sensitized to nickel than men and this is believed to be related to the fact that woman tend to wear more metal jewelry than men. Further study is required to determine whether there is indeed a gender difference in nickel sensitivity. Persons with kidney dysfunction are also likely to be more susceptible to nickel as the primary route of nickel elimination is via the urine.

Reference Dose for Chronic Oral Exposure (RfD)

Health Canada has developed a tolerable daily intake (TDI) value of 0.011 mg/kg/day for nickel chloride (Health Canada 2009). The TDI is based on the post-implantation perinatal lethality observed in a two generation reproductive study conducted over two years on rats who received nickel in drinking water. A NOAEL of 1.1 mg/kg/day was identified and an uncertainty factor of 100 was applied to account

for interspecies variability (a factor of 10) and sensitive human subpopulations (a factor of 10).

The RfD for soluble salts of nickel is 2E-2 mg/kg/day (critical effects including decreased body and organ weights) (U.S. EPA 2012).

Reference Concentration for Chronic Inhalation Exposure (RfC)

Health Canada (2009) has published an inhalation RfC of 0.000018 mg/m^3 for metallic nickel. The inhalation RfC is based on subchronic study (13 weeks) of rats and mice in which respiratory tract changes including the presence of alveolar macrophages and hyperplasia were observed at concentrations of 1 mg/m^3 (NOAEL in the mice studies and the LOAEL in the rat studies). An uncertainty factor of 1000 was applied to the NOAEL (mice) to account for (1) interspecies variability, (2) sensitive human subpopulations and (3) a subchronic study duration.

An RfC is not available for soluble nickel salts (U.S. EPA 2012).

Carcinogenic Effects

Health Canada has classified metallic nickel as Group VI (Unclassifiable with respect to carcinogenicity in humans) and soluble nickel as Group 1 (Carcinogenic to humans) (Health Canada 2009).

The U.S. EPA has not assessed soluble nickel salts for carcinogencity.

Nickel refinery dust is classified as Group A – human carcinogen by U.S. EPA (2012) based on human data in which exposure to nickel refinery dust caused lung and nasal tumors in refinery workers in several epidemiologic studies, and on animal data in which carcinomas were produced in rats by inhalation and injection. Nickel carbonyl is classified as a Group B2 – probable human carcinogen based on the observation of pulmonary carcinomas and malignant tumors in rats administered nickel carbonyl by inhalation and intravenous injection, respectively (U.S. EPA 2012). Nickel administered as nickel carbonyl binds to DNA. Nickel subsulfide as Group A – human carcinogen based on increased risks of lung and nasal cancer in humans exposed to nickel refinery dust, most of which was believed to have been nickel subsulfide; increased tumor incidences in animals by several routes of administration in several animal species and strains; and positive results in genotoxicity assays (U.S. EPA 2012).

Carcinogenic Risk from Oral Exposure

An oral slope factor is not available for any nickel compound (U.S. EPA 2012).

Carcinogenic Risk from Inhalation Exposure

Health Canada (2009) has derived an inhalation unit risk (UR) of 0.71 $(mg/m^3)^{-1}$ for soluble nickel (primarily nickel chloride and nickel sulphate). The UR is based on epidemiological studies where workers (cohort of 3250 to 54509) at two nickel refineries were occupationally exposed to nickel by inhalation for at least 12 months. The estimate of the concentration in air associated with a 5% increase in tumour incidence or mortality due to tumours (i.e., TC_{05}) for lung cancer mortality for soluble nickel was 0.07 mg/m^3.

An inhalation slope factor is not available for soluble nickel salts (U.S. EPA 2012). However the inhalation unit risk for nickel refinery dust is 2.4E-4 per $(\mu g/m^3)$ and for nickel subsulphide is 4.8E-4 per $(\mu g/m^3)$. Both unit risks are based on epidemiological studies of lung cancer and animal carcinoma data.

III.3.8.3 Summary

Oral TDI	0.011 mg/kg/day	perinatal lethality	Health Canada 2009
Inhalation RfC	1.8E-5 mg/m^3	respiratory effects	Health Canada 2009
Oral Slope Factor	not available at this time		U.S. EPA 2012
Inhalation Unit Risk	0.71 $(mg/m^3)^{-1}$	lung cancer	Health Canada 2009

III.3.9 Thallium

III.3.9.1 Pharmacokinetics

Oral Exposure

There are limited data that show thallium is absorbed through the gastrointestinal tract in humans, with indirect exposure occurring via mucocilary clearance following inhalation exposure (ATSDR 1992). Absorption is nearly complete in humans and animals, with accumulation in humans occurring in the scalp, renal papilla, renal cortex, heart and spleen, with lower levels found in the brain (ATSDR 1992). A radiolabeling study with a terminally ill patient where thallium was administered over 5 days found 0.4% of the administered dose was excreted in the feces and 11% in the urine during a 72-hour collection period, with a total of 15.3% excreted in the urine after 5.5 days (a half-life of 21.7 days was estimated) (ATSDR 1992).

Inhalation Exposure

No studies were found on the absorption or distribution of thallium in humans or animals following inhalation exposure, although an occupational study in a battery plant found thallium in the urine ranging from ≤ 50 to 236 μg/L (ATSDR 1992).

Dermal Exposure

No studies were found on the absorption, distribution and excretion of thallium in humans or animals following dermal exposure (ATSDR 1992).

III.3.9.2 Toxicity

Non-Carcinogenic Effects

Oral Exposure

Effects in humans following oral exposure to thallium include: death due to nerve damage; respiratory system, cardiovascular system, liver, kidney and muscle damage; and possible hair loss (ATSDR 1992). Lung and nervous system damage was caused following exposure to 54 to 110 mg/kg thallium nitrate. It has also been found that thallium can cross the human placenta, although developmental effects are not well characterized (ATSDR 1992).

Inhalation Exposure

Few studies were found on the effects of thallium on humans and animals following inhalation exposure; one long-term occupational study found effects to the nervous system including parasthesia, numbness of toes and fingers, “burning feet” and muscle cramps) following long-term occupational exposure, unfortunately 50% of patients had concomitant, unrelated diseases such as diabetes, obesity and alcoholism, which limits the value of the study (ATSDR 1992). Limited occupational studies find no effects to the cardiovascular and gastrointestinal systems following inhalation exposure to thallium.

Dermal Exposure

No studies were found on the toxicity of thallium to humans or animals following dermal exposure (ATSDR 1992).

Reference Dose for Chronic Oral Exposure (RfD)

An RfD is not available for thallium on the U.S. EPA IRIS website, however U.S. EPA NCEA provides an RfD as 1E-5 mg/kg/day (U.S. EPA 2010, 2012b) which is based on a study by Midwest Research Institute (1988) which studied the critical effects of thallium (I) sulfate administered to male and female Sprague-Dawley rats.

The rats were administered 0 (untreated and vehicle controls), 0.01, 0.05, or 0.25 mg/kg/day of an aqueous solution of thallium (I) sulfate (approximately 0, 0.008, 0.04, or 0.20 mg/kg/day T1) by gavage over 90 days (U.S. EPA 2010). The endpoint chosen for RfD development was hair follicle atrophy in female rats that also had alopecia (baldness). This endpoint was selected because atrophy of hair follicles is consistent with the atrophic changes observed in cases of human thallium poisoning and may be the best indication for human response to thallium exposure. The mid-dose in the study (0.04 mg/kg/day) was assumed to approximate a NOAEL for skin histopathology. Therefore, an estimated NOAEL of 0.04 mg/kg/day of thallium was used as the point of departure for hair follicle atrophy. An uncertainty factor of 3000 was applied to the NOAEL of 0.04 mg/kg/day (10 for extrapolating from laboratory animals to humans, 10 to account for variation in human susceptibility, 10 for a lack of adequate developmental toxicity and neurotoxicity studies, and 3 for extrapolating from subchronic to chronic exposure duration). The RfD of 1E-5 mg/kg/day is applicable to thallium salts (thallium (I) acetate, thallium (I) carbonate, thallium (I) chloride, thallium (I) nitrate) and thallium (I) sulfate).

Reference Concentration for Chronic Inhalation Exposure (RfC)

An RfC was not available found for thallium (Health Canada 2009; U.S. EPA 2010, 2012a).

Carcinogenic Effects

Carcinogenicity classification is not available for thallium. Health Canada as not classified the potential carcinogenicity of thallium (Health Canada 1996).

Carcinogenic Risk from Oral Exposure

An oral slope factor is not available for thallium (Health Canada 2009; U.S. EPA 2012a).

Carcinogenic Risk from Inhalation Exposure

An inhalation slope factor is not available for thallium (Health Canada 2009; U.S. EPA 2012a).

III.3.9.3 Summary

Oral Chronic RfD	1E-5 mg/kg/day	U.S. EPA 2010, 2012b
Inhalation RfC	not available	U.S. EPA 2010, 2012a
Oral Slope Factor	not available	U.S. EPA 2010, 2012a

Inhalation Slope Factor	not available	U.S. EPA 2010, 2012a

III.3.10 Titanium

III.3.10.1 Pharmacokinetics

Oral, Inhalation and Dermal Exposure

Pharmacokinetic information for titanium is not available.

III.3.10.2 Toxicity

Non-Carcinogenic Exposure

Oral, Inhalation and Dermal Exposure

General non-carcinogenic toxicity information is not available.

Tolerable Daily Intake (TDI) and Reference Dose (RfD) for Chronic Oral Exposure

Oral non-carcinogenic toxicity reference values were not available from the US EPA or Health Canada (US EPA 2012; Health Canada 2009).

Tolerable Concentration (TC) and Reference Concentration (RfC) for Chronic Inhalation Exposure

A tolerable inhalation concentration value for titanium is not available from Health Canada or the US EPA (Health Canada 2009; US EPA 2012).

ATSDR (1997) has developed an inhalation RfC for titanium tetrachloride of 1E-4 mg/m^3 is based upon increased irregular breathing and rhinitis. Vapours of titanium tetrachloride where generated by passing nitrogen over liquid titanium tetrachloride. An uncertainty factor of 100 was applied to the LOAEL of 0.1 mg/m^3 which was adjusted to a human equivalent concentration (LOAEL(HEC) 0.012 mg/m^3). The uncertainty factor was comprised of a factor of 3 for the use of a LOAEL rather than a NOAEL, a factor of 3 for extrapolation from animals to humans and 10 for the protection of sensitive human populations.

Carcinogenic Effects

Health Canada (1996, 2009) and the US EPA (2012) have not classified titanium for human carcinogenicity.

Carcinogenic Risk from Oral Exposure

A tumorigenic dose (TD_{05}) and oral slope factor for titanium is not available from Health Canada (2009) or the US EPA (2012).

Carcinogenic Risk from Inhalation Exposure

A tumorigenic concentration (TC_{05}) and inhalation slope factor for titanium is not available from Health Canada (2010) or the US EPA (2012).

III.3.10.3 Summary of TRVs Used

Oral RfD	not available at this time		Health Canada 2009
Inhalation RfC	1E-4 mg/m^3	respiratory system effects	ATSDR 1997
Oral Slope Factor	not available at this time		Health Canada 2009
Inhalation Slope Factor	not available at this time		Health Canada 2009

III.3.11 Vanadium

III.3.11.1 Toxicity

Non-Carcinogenic Effects

Oral Exposure

Oral exposure from either vanadyl (V^{+4}) and vanadate complexes (V^{+5}), which are most typically used in toxicological studies, result in a mixture of vanadyl and vanadate complexes in the gastrointestinal tract (U.S. EPA, NCEA 2000). As a result, there is currently no toxicological basis for distinguishing dose-response relationships for each form of vanadium compound (U.S. EPA, NCEA 2000). Oral exposure to vanadium in humans is not well characterized, although one study on volunteers has found 0.47 to 1.3 mg/kg vanadium (as ammonium vanadyl tartrate) administered in capsule form for 45 to 68 days causes intestinal cramping and diarrhea, although vehicle and compound controls were not used (limiting the quality of the data) (ATSDR 1992). Rats were also noted to have diarrhea following a 50 ppm dose of vanadium (form unspecified). Rat experiments have also shown mild kidney impairment (exhibited as increased plasma urea and mild histological changes) following 3 months of exposure to sodium metavanadate (at levels up to 10% of the oral LD_{50}), and a slight decrease in body weight.

Inhalation Exposure

Inhalation exposure to vanadium from occupational studies have found minor respiratory irritation as the major form of toxicity; mucus formation, coughing, wheezing, chest pain, runny nose and sore throat are noted as the most common (and reversible) effects (ATSDR 1992). A volunteer study found 0.06 mg/m^3 vanadium (as vanadium pentoxide) caused coughing and mucus formation 7 to 24 hours following exposure. The major target of vanadium toxicity following inhalation exposure in animals is the respiratory system; monkeys breathing 2.8 mg/m^3 vanadium (as vanadium pentoxide) for 6 hours showed increased pulmonary resistance 1 day after exposure, with a dramatic increase in polymorphonuclear leucocytes in bronchioalveolar fluid. Workers chronically exposed to vanadium dust demonstrated moderate eye irritation.

Dermal Exposure

No studies were found on the toxicity of vanadium via dermal exposure (ATSDR 1992).

Reference Dose for Chronic Oral Exposure (RfD)

Health Canada has not developed a tolerable daily intake for vanadium (Health Canada 2009).

U.S. EPA has derived an RfD for vanadium pentoxide (V_2O_5) is 9E-3 mg/kg/day based on a NOAEL of 17.85 ppm (adjusted to 0.89 mg/kg/day) (U.S. EPA 2012a). This value is based on a chronic oral study in rats, with decreased hair cystine content as the critical effect. A total uncertainty factor of 100 is applied; a factor of 10 was applied for interspecies extrapolation and a factor of 10 to protect sensitive populations. Confidence in the oral RfD for vanadium pentoxide is reported as low for the quality of the study, database and RfD value based on the scarcity of data available (U.S. EPA 2012a).

U.S. EPA 2012b has published an RfD of 0.005 mg/kg/day for vanadium, based on the U.S. EPA IRIS RfD for vanadium pentoxide with an adjustment for molecular weight differences.

Reference Concentration for Chronic Inhalation Exposure (RfC)

Health Canada has not developed a tolerable concentration for vanadium (Health Canada 2009).

An RfC for vanadium or vanadium pentoxide is unavailable at this time (U.S. EPA 2012a). ATSDR (2009) has developed a chronic duration inhalation minimum risk

level (MRL) of 1E-4 mg/m^3 based on a lower benchmark concentration ($BMCL_{10}$) from a chronic study of rats and mice. The $BMCL_{10}$ was adjusted to a human equivalent concentration of 3E-3 mg/m^3 and an uncertainty factor of 30 was applied (10 for human variability and 3 for animal to human extrapolation)

Carcinogenic Effects

Health Canada has not classified the potential carcinogenicity of vanadium (Health Canada 2004). No carcinogenicity classification is currently available for vanadium or vanadium pentoxide from the U.S. EPA (U.S. EPA 1988, 1996). Studies of carcinogenicity via chronic oral and dermal exposures have not been located for pentavalent vanadium compounds. Clear evidence of carcinogenicity was found in a study of male and female mice exposed to vanadium pentoxide via inhalation and some evidence of carcinogenicity in male but not female rats based on the presence of alveolar/bronchiolar neoplasms (Ress et al. 2003). These effects were noted at inhaled concentrations that slightly exceed permissible human occupational exposure limits (0.5 mg/m^3; Ress et al. 2003). Chronic studies of rats (Dai et al. 1994; Dai and McNiel 1994 as cited in WHO 2001) and mice (Kanisawa and Schroeder 1967 as cited in UK EGVM) exposed to vanadium sulphate in drinking water did not increase the incidence of spontaneous tumours in mice or rats, but caused reduced body weight gain in rats. The International Agency for Research on Cancer (IARC; 2005) has classified vanadium pentoxide as Group 2B which is possibly carcinogenic to humans.

Carcinogenic Risk from Oral Exposure

There is no oral slope factor available for vanadium or vanadium pentoxide (Health Canada 2009 and U.S. EPA 1988, 1996).

Carcinogenic Risk from Inhalation Exposure

There is no inhalation slope factor available for vanadium or vanadium pentoxide (Health Canada 2009 and U.S. EPA 2012a).

III.3.11.2 Summary

Oral Chronic RfD	5E-3 mg/kg/day	U.S. EPA 2012b
Inhalation RfC	1E-4 mg/m^3	ATSDR 2009
Oral Slope Factor	not available	U.S. EPA 2012a
Inhalation Slope Factor	not available	U.S. EPA 2012a

III.4 REFERENCES

ACGIH (American Conference of Government and Industrial Hygienists). 2001. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices.

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. Toxicological Profile for Antimony. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta, Georgia.

ATSDR. 1992. Toxicological Profile for Cobalt. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta, Georgia.

ATSDR. 1992. Toxicological Profile for Thallium.

ATSDR. 1992. Toxicological Profile for Vanadium.

ATSDR. 1997. Toxicological Profile for Nickel.

ATSDR. 1997. Toxicological Profile for Titanium Tetrachloride.

ATSDR. 1999. Toxicological Profile for Cadmium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta, Georgia.

ATSDR. 2000. Toxicological

ATSDR. 2000. Toxicological Profile for Manganese.

ATSDR. 2008. Toxicological Profile for Aluminum. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta, Georgia.

ATSDR. 2009. Toxicological Profile for Vanadium.

ATSDR. 2012. Minimal Risk Levels (MRLs) for Hazardous Substances. US Department of Health and Human Services, Public Health Service, Agency

for Toxic Substances and Disease Registry. Available on-line at: http://www.atsdr.cdc.gov/mrls/mrllist.asp

ATSDR. 2012. Minimal Risk Levels. http://www.atsdr.cdc.gov/mrls/index.asp

Cal EPA (California Environmental Protection Agency). Air Toxicology and Epidemiology. Office of Environmental Health Hazard Assessment. Available on-line at: http://www.oehha.ca.gov/air/hot_spots/index.html

Cirranni, R., Antonetti, M., Migliore, L. 1995. Vanadium Salts Induce Cytogenic Effects in *in Vivo* Treated Mice. Mutat. Res.343: 53-60. (As cited in WHO, 2001).

Curran, G.L., Arzanoff, D.L., and Bohnger, R.E. 1959. Effects of Cholesterol Synthesis Inhibition in Normochloesteroleremic Young Men. J. Clin. Invest. 38: 1251-1261. (As cited in U.S. EPA NCEA, 2000).

Dimond, E.G., Caravaca, J., Benchimol, A. 1963. Vanadium: Excretion, Toxicity, Lipid Effects in Man. Am. J. Clin. Nutr. 12: 49-53. (As cited in U.S. EPA NCEA, 2000).

Domingo, J.L., Liobet, J.L., Thomas, J.M., Corbella, J. 1985. Short-term Toxicity Studies of Vanadium in Rats. J. Appl. Toxicol. 5:418-421. (As Cited in ATSDR, 1992).

Fawcett, J.P., Farquar, T., Thou, T., Shand, B.I. 1997. Oral Vanadyl Sulfate Does Not Effect Blood Cells, Viscosity or Biochemistry in Humans. Pharmacol. Toxicol. 80:202-206. (As cited in U.S. EPA NCEA, 2000).

Health Canada. 1993. Canadian Environmental Protection Act. Priority Substances List Assessment Report. Arsenic and its Compounds.

Health Canada. 1994. Canadian Environmental Protection Act. Priority Substances List Assessment Report. Nickel and its Compounds.

Health Canada. 1996. Canadian Environmental Protection Act. Priority Substances List Supporting Documentation: Health Based Tolerable Daily Intakes/Concentrations and Tumourigenic Doses/Concentrations for Priority Substances. August 1996.

Health Canada. 1996. Health-Based Tolerable Daily Intakes/Concentrations and Tumorigenic Doses/Concentrations for Priority Substances. Environmental Health Directorate, Health Protection Branch. Minister of Supply and Services Canada. 96-EHD-194.

Health Canada. 2002. Guidelines for Canadian Drinking Water Quality – Supporting Documentation. Ottawa, ON. URL: http://www.hc-sc.gc.ca/hecs-sesc/water/dwgsup.htm

Health Canada. 2009. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs). (January, 2009). Environmental Health Assessment Services. Safe Environments Program. Health Canada, Ottawa, ON.

Health Canada. 2009. Federal Contaminated Site Risk Assessment in Canada. Part II: Health Canada Toxicity Reference Values (TRVs). Version 2.0, May 2009. Environmental Health Assessment Services, Safe Environments Program. Health Canada, Ottawa, ON.

Hrudey, S.E., W. Chen and C.G. Rousseaux. 1996. Bioavailability in Environmental Risk Assessment. CRC Press, Lewis Publishers, Boca Raton.

IARC (International Agency for Research on Cancer). 2005. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 86: Cobalt in Hard-metals and Cobalt Sulfate, Gallium Arsenide, Indium Phosphide and Vanadium Pentoxide. (in preparation).

IARC. 2012. Agents classified by the IARC monographs. Volumes 1 – 100. Available at: http://monographs.iarc.fr/ENG/Classification/

Migliore, L., Bocciardi, R., Macri, C., Lo Jacono, F. 1993. Cytogenetic Damage Induced in Human Lymphocytes by Four Vanadium Compounds and Micronucleus Analysis by Fluorescence *in situ* Hybridization with a Centromeric Probe. Mutat. Res. 319: 205-213. (As cited in WHO, 2001).

OMOE (Ontario Ministry of the Environment). 2011. Rationale for the Development of Generic Soil and Groundwater Standards for Use at Contaminated Sites in Ontario. April 15, 2011. PIBS 7386e01. Available on-line at: http://www.ene.gov.on.ca/environment/en/resources/STDPROD_086519.html

Owusu-Yaw, J., Cohen, M., Fernando, S., Wei, C. 1990. An Assessment of the Genotoxicity of Vanadium. Toxicol. Let. 50: 327-336. (As cited in WHO, 2001).

Profile for Arsenic. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta, Georgia.

Ress, N.B., Chou, B.J., Renne, A., Dill, A. et al. 2003. Carcinogenicity of Inhaled Vanadium Pentoxide in F344/N Rats and B6CF3_1 Mice. Toxicol. Sci. 74: 287-296.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human-toxicological maximum permissible risk levels. RIVM Report 711701 025.

RIVM. 2009. Re-evaluation of some Human Toxicological Maximum Permissible Risk Levels Earlier Evaluated in the Period 1991-2001. Bilthoven, the Netherlands. URL: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

Roldan, R.E., Altamirano, L.M. 1990. Chromosomal Aberrations, Sister Chromatid Exchanges, Cell-Cycle Kinetics and Associations in Human Lymphocyte Cultures Exposed to Vanadium Pentoxide. Mutat. Res. 245: 61-65. (As cited in WHO, 2001).

Sommerville, J., and Davies, B. 1962. Effect of Vanadium on Serum Cholesterol. Am. Heart J. 64:54-56. (As cited in U.S. EPA NCEA, 2000).

United Kingdom Expert Group on Vitamins and Minerals (UK EGVM). 2002. Review of Vanadium.

United States National Academy of Sciences (US NAS), Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and of Interpretation and Use of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board. 2002. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc.

U.S. EPA (United States Environmental Protection Agency). 1989. Risk Assessment Guidance for Superfund (RAGS) Part A.

U.S. EPA, National Center for Environmental Assessment (NCEA). 2001. Risk Assessment Issue Paper - Derivation of the Provisional RfD for Iron and Compounds. SRC SF-021a/11-14-01, dated December 3, 2001.

U.S. EPA, National Center for Environmental Assessment (NCEA). 2005a. Provisional Peer Reviewed Toxicity Values for Iron and Compounds (CASRN 7439-89-6). Devivation of a Chronic Inhalation RfC, dated June 29, 2005.

U.S. EPA, National Center for Environmental Assessment (NCEA). 2005b. Provisional Peer Reviewed Toxicity Values for Iron and Compounds (CASRN 7439-89-6). Devivation of a Carcinogenicity Assessment, dated June 29, 2005.

U.S. EPA, National Center for Environmental Assessment (NCEA). 2006. Provisional Peer Reviewed Toxicity Values for Iron and Compounds (CASRN 7439-89-6). Derivation of a Chronic Inhalation RfC, dated September 11, 2006.

U.S. EPA. 2008. Provisional Peer Reviewed Toxicity Values for Cobalt. Superfund Health Risk Technical Support Center National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency, Cincinnati, OH.

U.S. EPA. 2009. Risk Assessment Guidance for Superfund Volume 1. Human Health Evaluation Manual. Part F. Supplemental Guidance for Inhalation Risk Assessment. January. EPA-540-R-070-002.

U.S. EPA. 2010. Provisional Peer-Reviewed Toxicity Values for Thallium and Compounds. October 2010. National Center for Environmental Assessment. Cincinnati, OH.

U.S. EPA. 2010a. Air Quality: EPA's Integrated Science Assessments. National Centre for Environmental Assessment (CEA). Available on-line at: http://www.epa.gov/ncea/index.htm

U.S. EPA. 2010b. NCEA Provisional Peer-Reviewed Toxicity Values (PPRTV). NCEA. Available upon request.

U.S. EPA. 2012. Integrated Risk Information System (IRIS). URL: http://www.epa.gov/iris/

U.S. EPA. 2012a. Integrated Risk Information System (IRIS) On-Line Database Search. U.S. EPA, Cincinnati, OH. Available on-line at: http://www.epa.gov/iris/index.html

U.S. EPA. 2012a. Integrated Risk Information System (IRIS). URL: http://www.epa.gov/iris/

U.S. EPA. 2012b. Regional Screening Levels (RSL). Last Updated May 2012. Available on-line at: http://www.epa.gov/region9/superfund/prg/

U.S. EPA. 2012b. Regional Screening Levels. Users Guide. Available on-line at: http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/usersguide.htm

U.S. EPA. National Center for Environmental Assessment (NCEA). 2000. Provisional Toxicity Value Assessment for HWIR: RfD for Vanadium (CAS No. 7440-62-2). Document. Prepared by Syracuse Research Corporation. EPA Contract No. 68-C7-0011.

WHO. 2000. Air Quality Guidelines for Europe, Second Edition. WHO Regional Publications, European Series, No. 91. WHO Regional Office for Europe, Copenhagen. Available online at: http://www.euro.who.int/document/e71922.pdf.

WHO. 2001. International Program on Chemical Safety INCHEM. Concise International Chemical Assessment Document No. 29. Vanadium Pentoxide and Other Inorganic Vanadium Compounds.

WHO. 2004. Manganese in Drinking Water. Background Document for Development of WHO Guidelines for Drinking Water Quality. Available on-line at: http://www.who.int/water_sanitation_health/dwq/chemicals/manganese.pdf

WHO. 2005. Nickel in Drinking Water. Background Document for Development of WHO Guidelines for Drinking Water Quality. Available on-line at: http://www.who.int/water_sanitation_health/gdwqrevision/nickel2005.pdf

WHO. 2008. Guidelines for Drinking Water Quality: Third Edition Incorporating the First and Second Addenda, Volume 1, Recommendations. WHO, Geneva. Available on-line at: http://www.who.int/water_sanitation_health/dwq/fulltext.pdf

Zhong, B., Gu, Z., Wallace, W., Whong, W., Ong, T. 1994. Genotoxicity of Vanadium Pentoxide in Chinese Hamster V79 Cells. Mutat. Res. 321: 35-42 (As cited in WHO, 2001).

APPENDIX IV

MULTI-MEDIA EXPOSURE ASSESSMENT

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LIST OF TABLES

IV INTRODUCTION

Exposure assessment is the process of estimating the exposure of a human receptor to a substance under a given exposure scenario. An exposure assessment was conducted for each chemical of concern (COC) identified in the problem formulation. For the multi-media assessment, exposure is determined as a dose. This value is called the Estimated Daily Intake (EDI) and is typically expressed as milligram of a chemical per kilogram of body weight per day (mg/kg BW/day).

The EDI was calculated from site-specific concentrations of substances in each environmental medium (e.g., air, water, sediment, soil and food), the amount of time a receptor spends at a location and receptor-specific parameters, such as body weight, ingestion rates and dietary preferences.

IV.1 EXPOSURE ASSUMPTIONS

Exposure assumptions used in calculating the EDI for human receptors are outlined below.

IV.1.1 Measured Exposure Concentrations

IV.1.1.1 Soil, Sediment, Labrador Tea Leaves, and Berries

Baseline exposure concentrations were based on actual measured concentrations from the various media: soil, sediment, and vegetation. For human health modelling purposes, the 95% upper confidence limit of the mean (UCLM) was calculated if possible (i.e., if there were at least ten discrete detected values), otherwise, the maximum value was used. Baseline exposure concentrations are provided in Tables IV-1 to IV-5.

Table IV-1 Baseline Soil Concentrations

Chemical of Concern	Soil Concentration [mg/kg dry weight]	Statistical endpoint
Aluminum	6,104	95% UCLM
Antimony	0.1	Detection Limit
Arsenic	1.436	95% UCLM
Cadmium	0.191	95% UCLM
Cobalt	4.108	95% UCLM
Iron	8810	95% UCLM
Manganese	66.15	95% UCLM
Nickel	66.01	95% UCLM
Thallium	0.103	Maximum Value
Titanium	301.4	95% UCLM
Vanadium	12.86	95% UCLM

Note: UCLM – upper confidence limit of the mean.

Table IV-2 Baseline Sediment Concentrations

Chemical of Concern	Concentration [mg/kg dry weight]	Statistical endpoint
Aluminum	33,088	95% UCLM
Antimony	0.429	95% UCLM
Arsenic	6.772	95% UCLM
Cadmium	0.478	95% UCLM
Cobalt	21.43	95% UCLM
Iron	47,533	95% UCLM
Manganese	2,712	95% UCLM
Nickel	38.54	95% UCLM
Thallium	0.24	95% UCLM
Titanium	269.5	95% UCLM
Vanadium	50.97	95% UCLM

Note: UCLM – upper confidence limit of the mean.

Table IV-3 Baseline Northern Labrador Tea Leaf Concentrations Used in the Human Health Risk Assessment

Chemical of Concern	Northern Labrador Tea Leaf Concentration[(a)] [mg/kg dry weight]	Statistical endpoint
Aluminum	119	Maximum Value
Antimony	0.06	Maximum Value
Arsenic	0.1	Detection Limit
Cadmium	0.04	Detection Limit
Cobalt	0.23	Maximum Value
Iron	75	Maximum Value
Manganese	722	Maximum Value
Nickel	3.96	Maximum Value
Thallium	0.28	Maximum Value
Titanium	2.16	Maximum Value
Vanadium	0.14	Maximum Value

(a) Humans were assumed to be eating Labrador tea leaves as a surrogate for leafy vegetables.

Table IV-4 Baseline Berry Concentrations Used in the Human Health Risk Assessment

Chemical of Concern	Berry Concentration [mg/kg dry weight]	Statistical Endpoint
Aluminum	689	95% UCLM
Antimony	0.054	95% UCLM
Arsenic	0.2	Maximum Value
Cadmium	0.253	95% UCLM
Cobalt	0.376	95% UCLM
Iron	313	95% UCLM
Manganese	367.2	95% UCLM
Nickel	4.304	95% UCLM
Thallium	0.03 [DL]	Detection Limit
Titanium	29.4	95% UCLM
Vanadium	0.753	95% UCLM

Notes: UCLM – upper confidence limit of the mean; DL – detection limit; all values were less than the DL. The detection limit from the most recent round of sampling was used.

Table IV-5 Baseline vegetation concentrations used in the food web model (to predict Baseline case mammal concentrations for the human health risk assessment)

Chemical of Concern	Berry Concentration [mg/kg dry weight]*	Leaf Concentration [mg/kg dry weight]*	Grass Concentration [mg/kg dry weight]*	Lichen Concentration [mg/kg dry weight]*
Aluminum	689	405	236.5	426.5
Antimony	0.054	0.05 [c]	0.05 [mv]	0.05 [DL]
Arsenic	0.2 [mv]	0.05 [DL]	0.082 [mv]	0.334
Cadmium	0.253	0.252	0.17 [mv]	0.0719
Cobalt	0.376	2.87	0.369	0.489
Iron	313	364	256.1	487.2
Manganese	367.2	356.2	199.6	105.1
Nickel	4.304	7.33	4.28	4.45
Thallium	0.03 [DL]	0.28 [mv]	0.05 [mv]	0.03 [DL]
Titanium	29.4	22.9	10.9	39.4
Vanadium	0.753	0.382	0.95 [mv]	1.10

Notes: Based on measured baseline concentrations – 95% UCLMs were calculated where possible (i.e., if there were at least ten detected values), otherwise 90th percentile concentrations were calculated. Maximum values were used if it was not possible to calculate a 95% UCLM or a 90th percentile concentration based on sample size.
*The 95% UCLM concentration is presented unless otherwise indicated.
C – 90th percentile concentration (detected sample size insufficient for 95% UCLM).
mv – maximum detected value (sample size insufficient for statistics).
DL – detection limit; all values were less than the DL. The detection limit from the most recent round of sampling was used.

IV.1.1.2 Water

Potable Water

For the Baseline case, baseline water quality data (i.e., long term averages) for Kennady Lake (including Area 8) were used to assess exposure of Seasonal Users and Gahcho Kué workers ingesting and coming into contact with potable water. For the Application Case, water concentrations for Area 8 during the Construction and Operations phases of the Project (predicted from the Baseline water quality data) were used to assess ingestion and dermal exposure; the higher of the 95th percentile values for the Operations and Construction phases was used as the exposure concentration. Potable water concentrations used in the human health risk assessment are provided in Table IV-6.

Surface Water

For the Baseline case, water concentrations used to assess exposure of Gahcho Kué workers coming into contact with and incidentally ingesting surface water are predicted long-term averages for Kennady Lake (including Area 6, Area 8 and WMP). For the Application case, maximum annual average concentrations predicted for Kennady Lake (including Area 6, Area 8 and WMP) were used. The Baseline and Application Case water concentrations are provided in Table IV-6. The Seasonal User may come into contact with surface water and sediment while conducting traditional activities such as fishing; dermal contact with water was conservatively evaluated through dermal contact with potable water (which was assumed to be used daily for showering, etc.). Surface water concentrations used in the human health risk assessment are provided in Table IV-6.

Water Used to Predict Fish and Mammal Tissue Concentrations

For fish and mammals, baseline water quality data from Lakes N11 and 410 (the watershed downstream of Kennady Lake) were used to predict Baseline fish and mammal tissue concentrations. Kennady Lake will be dewatered during the construction phase of the Project, and water will be pumped to Area 8 and Lake N11. Mine affected water will flow through Area 8 and continue to downstream through the Interlakes (i.e., the L and M watersheds) into Lake 410 (which is downstream of Lake N11 and the Kennady Lake watershed). For the Application case, maximum predicted concentrations for Lake 410 and Lake N11 during the construction, operations and closure phases of the Project were used to predict Application case fish and mammal tissue concentrations. Water concentrations used to predict fish and mammal tissue concentrations are provided in Table IV-6.

Table IV-6 Water Concentrations Used in the Human Health Risk Assessment

Chemical of Concern	Potable Water Supply		Surface Water Concentrations (used to evaluate incidental ingestion and dermal contact by a Gahcho Kué Worker)		Water Concentrations used to Predict Fish and Mammal Tissue Concentrations	
	Baseline Case Kennady Lake Baseline Water Concentration Long Term Average [mg/L]	Application Case Area 8 Water Concentration (95% UCLM) [mg/L]	Baseline Case Kennady Lake Maximum of Annual Average Concentrations [mg/L]	Application Case Kennady Lake Maximum of Annual Average Concentrations [mg/L]	Baseline Case Lake N11 and Lake 410 Baseline Concentrations [mg/L]	Application Case Lake N11 and Lake 410 Maximum Predicted Concentration during Construction, Operations and Closure [mg/L]
Aluminum	0.0098	0.011	0.0098	0.19	0.019	0.029
Antimony	0.0001	0.00011	0.0001	0.0043	0.000062	0.00035
Arsenic	0.00014	0.00016	0.00014	0.011	0.00012	0.00074
Cadmium	0.00002	0.000026	0.00002	0.000066	0.000019	0.000024
Cobalt	0.00014	0.00015	0.00014	0.0031	0.00019	0.00036
Iron	0.065	0.074	0.065	2.3581	0.059	0.088
Manganese	0.012	0.014	0.012	0.19	0.0057	0.014
Nickel	0.00032	0.00037	0.00032	0.0098	0.00047	0.0012
Thallium	0.000021	0.000023	0.000021	0.00042	0.000014	0.000049
Titanium	n/a	n/a	n/a	n/a	n/a	n/a
Vanadium	0.00024	0.00027	0.00024	0.0059	0.000094	0.00051

n/a = Data not available.

IV.1.2 Modelled Exposure Concentrations

Activities in the two assessment cases (Baseline Case and Application Case) have the potential to increase concentrations of metals in soil and vegetation through deposition of particulate matter. Wildlife (e.g., caribou and hares) that browse vegetation and incidentally ingest soil can take up substances into tissues, which are then consumed by people or carnivorous wildlife. Therefore, concentrations of animal tissues and future concentrations of substances in soil and plants were estimated. The incremental concentrations contributed by activities in the region were calculated using the food chain modelling methods developed by the United States Environmental Protection Agency (U.S. EPA 2006a). The baseline statistics calculated for soil and vegetation (i.e., 95% UCLM and 90th percentile concentrations) were used in the prediction of the modelled concentrations (Section IV.1.1).

Loss mechanisms (i.e., volatilization, degradation, erosion and leaching) were not included in the calculations. Modelling without environmental losses maximizes the exposure assessment. These methods provide a multi-pathway exposure assessment tool based on reasonable, protective assumptions about how substances emitted from combustion sources can be taken up into soil and plants and then by wildlife.

IV.1.2.1 Air Deposition Values

Project-specific depositional rates for the Baseline Case and Application Case are presented in Table IV-7.

Table IV-7 Deposition Values Used in the Human Health Model

Chemical of Concern	Dry Deposition	Wet Deposition	Total Deposition	Dry Deposition	Wet Deposition	Total Deposition
	Baseline [µg/m^2/s]			Application [µg/m^2/s]		
Aluminum	1.60E-07	1.02E-08	1.69E-07	1.73E-02	6.59E-04	1.80E-02
Antimony	4.24E-12	2.88E-13	4.50E-12	6.37E-07	2.41E-08	6.60E-07
Arsenic	2.34E-10	1.21E-11	2.45E-10	1.76E-06	7.36E-08	1.83E-06
Cadmium	3.69E-09	2.96E-10	3.96E-09	4.91E-05	1.37E-06	5.05E-05
Cobalt	8.47E-10	6.39E-11	9.06E-10	3.39E-05	1.23E-06	3.51E-05
Iron	2.99E-07	1.89E-08	3.16E-07	3.25E-02	1.24E-03	3.37E-02
Manganese	5.15E-09	3.30E-10	5.44E-09	4.73E-04	1.80E-05	4.91E-04
Nickel	4.34E-09	2.43E-10	4.55E-09	2.22E-04	9.01E-06	2.31E-04
Thallium	2.08E-12	1.39E-13	2.20E-10	2.91E-07	1.10E-08	3.02E-07
Titanium	1.21E-08	8.00E-10	1.28E-08	1.67E-03	6.31E-05	1.73E-03
Vanadium	4.55E-10	2.92E-11	4.81E-10	5.47E-05	2.08E-06	5.68E-05

A description of the methods used to predict concentrations in soil, plants and meat used in the risk assessment are presented below.

IV.1.2.2 Soil

Deposition onto soil was assumed to occur throughout the construction and operational phases of the Project (i.e., a maximum of 11 years was assumed). All chemicals deposited onto soil were assumed to mix within the top 0.2 m of soil (U.S. EPA 2006b). Soil was assumed to have a bulk density of 1,500 kg/m^3 (U.S. EPA 2006b). The incremental increase in soil concentrations due to the Baseline Case and Application Case was determined using equations presented in Table IV-8.

Table IV-8 Equations for Predicting Incremental Concentrations in Soil

Media	Equation
Soil	SC = (Dep x tD)/(Z_s x BD) SC = soil concentration (mg/kg dry wt) Dep = total wet and dry deposition rate (g/m^2/y); Project-specific (Tables IV-7) Ks = soil loss constant due to all processes (yr^{-1}) tD = deposition time (65 years) Z_s = soil mixing depth; 0.2 m (U.S. EPA 2006b) BD = bulk density; 1,500 kg/m^3 (U.S. EPA 2006b)

Source: Equations from (U.S. EPA 2006b).

The predicted soil concentrations that were used in the risk assessment are provided in Table IV-9 for both the Baseline and Application Case.

Table IV-9 Predicted Baseline and Application Case Soil Concentrations

Chemical of Concern	Project Boundary	
	Baseline [mg/kg dry weight]	Application Case [mg/kg dry weight]
Aluminum	6104	6312
Antimony	0.100	0.108
Arsenic	1.44	1.46
Cadmium	0.191	0.775
Cobalt	4.11	4.51
Iron	8810	9200
Manganese	66.2	71.8
Nickel	66.0	68.7
Thallium	0.103	0.106
Titanium	301	321.4
Vanadium	12.9	13.5

IV.1.2.3 Vegetation

Chemical concentrations in wild plants (berries, leaves, grasses and lichen) were estimated using equations presented in Table IV-10.

Table IV-10 Equations for Predicting Incremental Concentrations in Plants

Parameter	Equation
Total plant concentration	$PC = Pd + Pr$
	PC = incremental concentration (mg/kg dry wt) Pd = incremental concentrations due to air deposition (mg/kg dry wt) Pr = incremental concentration due to root uptake (mg/kg dry wt)
Plant concentration due to air deposition	$Pd = 1{,}000 \times [D_{yd} + (Fw \times D_{wyd})] \times Rp\,[1\text{-}exp(-kp \times Tp)]/(Y_p \times kp)$
	Pd = incremental concentration due to air deposition (mg/kg dry wt) D_{yd} = dry particle deposition rate ($g/m^2/y$); Project-specific Fw = Fraction of COC wet deposition that adheres to plant surface; 0.6 (U.S. EPA 2006b) D_{wyd} = wet deposition rate ($g/m^2/y$); Project-specific Rp = interception fraction; represents portion of chemical deposition intercepted by plants; 0.39 for berry/fruit/vegetable (U.S. EPA 2006b) Y_p = crop yield (kg dry wt/m^2); 0.246 for forage, 0.252 for berry/fruit/vegetable and 0.24 for leaf (U.S. EPA 2006b) Tp = length of plant exposure to deposition per harvest; 0.25 for leaf kp = chemical removal from the plant surface by weathering (yr^{-1}), 18 for all plants (U.S. EPA 2006b)
Plant concentration due to root uptake	$Pr = SC \times BAF$
	Pr = incremental concentration due to root uptake (mg/kg dry wt) SC = predicted incremental soil concentration (mg/kg dry wt) BAF = bioaccumulation factor (unitless); Project-specific presented in Table IV-11

Source: U.S. EPA (2006b).

Plant concentrations were calculated based on the total exposure from direct deposition onto plants (incorporating surface area), absorption from gaseous chemicals in the air and uptake from soil. Site-specific bioaccumulation factors (BAFs) (i.e., soil-to-leaf and soil-to-berry, uptake factors) were used (Table IV-11).

Table IV-11 Soil to Plant BAFs Used in the Human Health Model (for direct ingestion of leaves and berries by humans)

Chemical of Concern	Northern Labrador Tea Leaves Bioaccumulation Factor [kg soil/kg plant]	Berries Bioaccumulation Factor [kg soil/kg plant]
Aluminum	0.0268	0.151
Antimony[(a)]	0.2[(a)]	0.2[(a)]
Arsenic	0.125	0.131
Cadmium	0.824	0.342[(b)]
Cobalt	1.66	0.209
Iron	0.0121	0.0589
Manganese	11.1	10.9
Nickel	1.36	1.56
Thallium	3.11[(b)]	0.440[(b)]
Titanium	0.00865	0.0487
Vanadium	0.0662	0.0854

(a) Antimony was not detected in soil or plant tissues. U.S. EPA (2007) was used to approximate the BAF.

(b) Maximum BAF was used; sample size was too small for use of statistics to determine a BAF.

Note: The 90th percentile BAF values were used unless otherwise noted.

The default values for crop yield (Yp) were used in the prediction of future plant concentrations for the Project (U.S. EPA 2006b). The default values for interception fraction (Rp) (U.S. EPA 2006b) were determined to be sufficiently conservative for wild plants because the surface areas of fruits (e.g., tomatoes, apples) and leafy vegetables (e.g., lettuce, cabbage) are much greater than those of berries, Labrador tea leaves, grasses and lichen. Length of plant exposure (Tp) was estimated to be three months for berries (the length of the growing season in the area) and wild plant leaves because these would either be shed in the fall or they would be covered by snow for most of the winter months.

The predicted leaf and berry concentrations that were used in the human health risk assessment (for direct ingestion by humans) are provided in Tables VI-12 and IV-13 for the Baseline Case and Application Case.

Table IV-12 Predicted Baseline and Application Case Northern Labrador Tea Leaf Concentrations (for direct ingestion of leaves and berries by humans)

chemical of concern	Project Boundary	
	Baseline [mg/kg wet weight]	Application Case [mg/kg wet weight]
Moisture content[(a)] (%)	58.5	
Aluminum	49.4	103.9
Antimony	0.0249	0.0275
Arsenic	0.0415	0.0479
Cadmium	0.0166	0.363
Cobalt	0.095	0.477
Iron	31.1	130.9
Manganese	299.6	327.2
Nickel	1.64	3.82
Thallium	0.116	0.122
Titanium	0.896	5.99
Vanadium	0.0581	0.241

(a) The moisture content for northern Labrador tea was based on the regional average derived for the Oil Sands region of Alberta (n=125; Golder Associates 2010) and was consistent with the range of moisture contents observed in 2011 leaf samples from the LSA (54% to 63%).

Table IV-13 Predicted Baseline and Application Case Berry Concentrations

Chemical of Concern	Project Boundary	
	Baseline Case [mg/kg wet weight]	Application Case [mg/kg wet weight]
Moisture content[(a)] (%)	80	
Aluminum	137.8	168.7
Antimony	0.0109	0.0121
Arsenic	0.0400	0.0431
Cadmium	0.0506	0.160
Cobalt	0.0752	0.140
Iron	62.6	113.2
Manganese	73.4	86.4
Nickel	0.861	2.01
Thallium	0.00600	0.00672
Titanium	5.88	8.44
Vanadium	0.151	0.239

(a) The moisture content for berries was derived from the USDA National Nutrient Database (USDA 2011).

IV.1.2.4 Mammals

Wildlife may ingest soil, plants and water from the Project Area and accumulate substances into their tissues. Therefore, uptake of metals into animals consumed by people was estimated using food chain modelling. Muscle tissue concentrations (i.e., meat) were calculated using the equations presented in Table IV-14. The meat concentrations were calculated based on consumption of plants, soil and water by caribou and hare. Bioaccumulation Factors (BAFs) for soil-to-berries, soil-to-eaves, soil-to-grass and soil-to-lichen (Table IV-15) are used to predict baseline and application case vegetation concentrations (Table IV-16) by using equations presented in Table IV-10. Baseline soil, plant and water concentrations are provided in Tables IV-1, IV-5 and IV-6, respectively.

Equations for Predicting Hare and Caribou Meat Concentrations

Table IV-14 Equations for Predicting Hare and Caribou Meat Concentrations

Media	Equation
Muscle concentration in Caribou or Hare	T_C = $\sum$EDI x BW x BTF
	T_C = incremental chemical concentration in herbivorous mammal (mg/kg dry wt) $\sum$EDI = sum of chemical ingestion from all oral pathways (mg/kg BW/day) BW = animal body weight (kg) BTF = biotransfer factor (day/kg)
Water Ingestion	$EDI_{water} = \frac{IR \times C_{water} \times AF_{GIT}}{BW}$
	EDI_{water} = exposure due to ingestion of water (mg COC/kg body weight/day) IR = ingestion rate (L/day) C_{water} = COC concentration in water (mg/L) AF_{GIT} = absorption factor for the gastrointestinal tract (unitless) (assumed 1) BW = receptor body weight (kg)
Soil Ingestion	$EDI_{soil} = \frac{IR \times C_{soil} \times AF_{GIT}}{BW}$
	EDI_{soil} = exposure due to ingestion of soil (mg COC/kg body weight/day) IR = ingestion rate (kg/day) C_{soil} = COC concentration in soil (mg/kg) AF_{GIT} = absorption factor for the gastrointestinal tract (unitless) (assumed 1) BW = receptor body weight (kg)
Plant ingestion	$EDI_{plant} = \frac{IR \times C_{plant} \times AF_{GIT}}{BW}$
	EDI_{plant} = exposure due to ingestion of plant (mg COC/kg body weight/day) IR = ingestion rate (kg/day) – in dry weight C_{plant} = COC concentration in plant (mg/kg) – dry weight concentration[(a)] AF_{GIT} = absorption factor for the gastrointestinal tract (unitless) (assumed 1) BW = receptor body weight (kg)

(a) See Table IV-10 of equations for calculating predicted plant concentrations.

Table IV-15 Soil to Plant BAFs Used in the Food Web Model (to predict Baseline case mammal concentrations for the human health risk assessment)

Chemical of Concern	Berries Bioaccumulation Factor [kg soil/kg plant]	Leaves Bioaccumulation Factor [kg soil/kg plant]	Grasses Bioaccumulation Factor [kg soil/kg plant]	Lichens Bioaccumulation Factor [kg soil/kg plant]
Aluminum	0.151	0.0268	0.0671	0.157
Antimony[(a)]	0.2[(a)]	0.2[(a)]	0.2[(a)]	0.2[(a)]
Arsenic	0.131	0.125	0.526	0.667
Cadmium	0.342[(b)]	0.824	0.260	1.28
Cobalt	0.209	1.66	0.625	0.625
Iron	0.0589	0.0121	0.0752	0.117
Manganese	10.9	11.1	12.3	4.11
Nickel	1.56	1.36	0.816	0.770
Thallium	0.440[(b)]	3.11[(b)]	0.444	3.75
Titanium	0.0487	0.00865	0.0893	0.248
Vanadium	0.0854	0.0662	0.288	0.157

(a) Antimony was not detected in soil or plant tissues. The U.S. EPA (2007) BAF value was used.

(b) Maximum BAF was used; sample size was too small for use of statistics to determine a BAF.

Notes: The 90th percentile BAF values were used unless otherwise noted.

Table IV-16 Predicted Baseline and Application Case Vegetation Concentrations for Consumption by Wildlife (Project Boundary)

Chemical of Concern	Berries		Leaves		Grasses		Lichens	
	Baseline Case (mg/kg dry weight)	Application Case (mg/kg dry weight)	Baseline Case (mg/kg dry weight)	Application Case (mg/kg dry weight)	Baseline Case (mg/kg dry weight)	Application Case (mg/kg dry weight)	Baseline Case (mg/kg dry weight)	Application Case (mg/kg dry weight)
Aluminum	689	843	405	536	237	376	427	536
Antimony	0.0544	0.0604	0.05	0.0561	0.05	0.0561	0.05	0.0561
Arsenic	0.2	0.215	0.0500	0.0655	0.0820	0.106	0.334	0.0655
Cadmium	0.253	0.798	0.252	1.09	0.170	0.676	0.0719	1.09
Cobalt	0.376	0.701	2.87	3.79	0.369	0.868	0.489	3.79
Iron	313	566	364	604	256	521	487	604
Manganese	367.2	432	356	423	200	273	105	423
Nickel	4.30	10.1	7.33	12.6	4.28	8.08	4.45	12.6
Thallium	0.03	0.0336	0.28	0.293	0.05	0.0537	0.03	0.293
Titanium	29.4	42.2	22.9	35.2	10.9	24.7	39.4	35.2
Vanadium	0.753	1.20	0.382	0.823	0.95	1.54	1.10	0.823

Biotransfer Factors

Biotransfer factors for caribou and bioaccumulation factors for hares that were used in the risk assessment are summarized in Tables IV-17 and IV-18, respectively. Biotransfer factors for beef (from RAIS 2012) were used since wildlife-specific biotransfer factors for metals were not available. Where bioaccumulation factors for hares were not available (uptake factors from U.S. EPA 2007), biotransfer factors for beef (from RAIS 2012) were used.

Table IV-17 Biotransfer Factors for Caribou Used in the Human Health Model

Chemical of Concern	Biotransfer Factor (day/kg BW dry weight)
Aluminum	0.0015
Antimony	0.001
Arsenic	0.002
Cadmium	0.00055
Cobalt	0.02
Iron	0.02
Manganese	0.0004
Nickel	0.006
Thallium	0.04
Titanium	0.03
Vanadium	0.0025

Notes: Biotransfer factors for beef (from RAIS 2012) were used since wildlife-specific biotransfer factors for metals were not available.
BW – body weight.

Table IV-18 Dry weight bioaccumulation factors for soil to small mammals (Snowshoe Hare)

Chemical of Concern	Bioaccumulation Factor Soil to Mammal[a] [mg/kg mammal tissue/mg/kg soil]	Biotransfer Factor [day/kg BW dry weight]
Aluminum	n/a	0.0015[b]
Antimony	n/a	0.05[c]
Arsenic	0.00735[d]	-
Cadmium	0.681[d]	-
Cobalt	0.0182[d]	-
Iron	n/a	0.02[b]
Manganese	0.0205[c]	-
Nickel	0.0834[d]	-
Thallium	n/a	0.04[b]
Titanium	n/a	0.03[b]
Vanadium	n/a	0.0025[b]

[a] BAF used when available, otherwise a BTF was used.

[b] RAIS (2012) cattle BTF.

[c] Eco-SSL uptake factor for soil to small mammals (U.S. EPA 2007).

[d] Eco-SSL BAF (U.S. EPA 2007).

Note: BW = body weight; n/a = Not available; - = Not used in risk assessment.

Mammalian Body Weights, Feeding Preferences and Ingestion Rates

The composition of diet is based on general dietary preferences for receptor organisms; this information has been compiled from life history information for the species. Ingestion rates were approximated using allometric scaling based on body weight. The equations for food (Equation 1) and water (Equation 2) were obtained from Sample et al. (1997):

$$IR_{food} = 0.0687(Bw)^{0.822} \quad (1)$$

Where:

IR_{food} = food ingestion rate (kg dry weight/day)
BW = body weight (kg)

$$IR_{water} = 0.099(Bw)^{0.9} \quad (2)$$

Where:

IR_{water} = water ingestion rate (L/day)
BW = body weight (kg)

Incidental soil ingestion rates were extrapolated from Beyer et al. (1994) for snowshoe hare and from MacDonald and Gunn (2004) for caribou.

Table IV-19 provides a summary of body weights, feeding preferences and ingestion rates for the caribou and snowshoe hare.

Table IV-19 Summary of Body Weights, Diet and Ingestion Rates for Caribou and Snowshoe Hare used in the Food Chain Model

Mammal	Body Weight[(a)] [kg]	Diet	Soil/Sediment Ingestion Rate (kg/day dry weight)	Food Ingestion Rate [kg/day dry weight]	Water Ingestion Rate [Litres per day]
Snowshoe Hare	1.4[(a)]	33% leaves 34% berries 33% grasses	0.0018[(b)]	0.0906	0.134
Caribou	100[(c)]	50% lichen 25% grasses 25% leaves	0.103[(d)]	3.03	6.25

(a) The body weight for an adult snowshoe hare ranges from 1.2 to 1.6 kg (CWS and CWF 2005). The average of the range of body weight was used.

(b) 0.02 x Food Ingestion Rate (Beyer et al. 1994).

(c) Smith 1993.

(d) 0.034 x Food Ingestion Rate (MacDonald and Gunn 2004).

Predicted Meat Concentrations

The predicted caribou and hare meat concentrations that were used in the risk assessment for the Baseline Case and Application Case are provided in Tables IV-20 and IV-21.

Table IV-20 Predicted Caribou Concentrations (mg/kg wet weight) for Baseline and Application Case Scenarios

Chemical of Concern	Project Boundary	
	Baseline Case [mg/kg wet weight]	Application Case [mg/kg wet weight]
Moisture content[(a)] %	71.45	
Aluminum	0.753	0.953
Antimony	0.0000463	0.0000523
Arsenic	0.000430	0.000474
Cadmium	0.0000703	0.000501
Cobalt	0.0206	0.0313
Iron	12.1	16.9
Manganese	0.0670	0.0837
Nickel	0.0382	0.0599
Thallium	0.00349	0.00391
Titanium	0.996	1.40
Vanadium	0.00286	0.00400

(a) The moisture content for caribou was derived from the USDA (2011) National Nutrient Database.

Table IV-21 Predicted Hare Concentrations (mg/kg wet weight) for Baseline and Application Case Scenarios

Chemical of Concern	Project Boundary	
	Baseline Case [mg/kg wet weight]	Application Case [mg/kg wet weight]
Moisture content[(a)] %	74.51	
Aluminum	0.0196	0.0246
Antimony	0.0000618	0.0000696
Arsenic	0.00269	0.00273
Cadmium	0.0332	0.135
Cobalt	0.0191	0.0210
Iron	0.225	0.345
Manganese	0.346	0.375
Nickel	1.40	1.46
Thallium	0.000113	0.000119
Titanium	0.0188	0.0280
Vanadium	0.0000550	0.0000841

(a) The moisture content for caribou was derived from the USDA (2011) National Nutrient Database.

IV.1.2.5 Fish

Fish bioaccumulation factors were derived based on concentrations of substances measured in muscle tissue of lake trout and round whitefish (Aquatic Health Section - Appendix 8.VI of the 2012 EIS Update). Only whole-body concentration data were available for slimy sculpin, and these were not included in BAF derivation (see Aquatic Health Section Appendix 8.VI for more information on the fish BAF derivation). Table IV-22 provides the fish BAFs used for the human health risk assessment. The BAFs were multiplied by the Baseline Case and Application Case water concentrations for Lake N11 and Lake 410 (Table IV-6) to predict fish tissue concentrations. The predicted fish tissue concentrations used in the human health risk assessment are provided in Table IV-23.

Table IV-22 Wet Weight BAFs for the Water to Fish Pathway

Chemical of Concern	Bioaccumulation Factor (wet weight) [L water/kg fish]
Aluminum	278
Antimony	2,729
Arsenic	417
Cadmium	237
Cobalt	157
Iron	150
Manganese	29
Nickel	232
Thallium	800
Titanium	16
Vanadium	95

Notes: Fish tissue BAFs were derived in the Aquatic Health Section (Appendix 8.VI of the 2012 EIS Update).

Table IV-23 Estimated Fish Concentrations for the Baseline Case and Predicted Concentrations for the Application Case

Chemical of Concern	Baseline Case Fish Concentration [mg/kg wet weight]	Application Case Fish Concentration [mg/kg wet weight]
Aluminum	5.14	8.18
Antimony	0.168	0.943
Arsenic	0.0509	0.310
Cadmium	0.00450	0.00558
Cobalt	0.0298	0.05673
Iron	8.85	13.3
Manganese	0.165	0.395
Nickel	0.108	0.283
Thallium	0.0114	0.0393
Titanium	n/d	n/d
Vanadium	0.00893	0.0487

Notes: n/d – not determined as water quality data were not available.
For the Baseline Case, fish concentrations were estimated by multiplying predicted baseline water quality data for Lakes N11 and 410 (Table IV-6) by fish BAFs (Table IV-22).
For the Application Case, fish concentrations were estimated by multiplying predicted water quality data for Lakes N11 and 410 during construction, operations and closure (Table IV-6) by fish BAFs (Table IV-22).

IV.1.3 RECEPTOR ASSUMPTIONS

Based on the results of the problem formulation, the following receptors were retained for the Environmental Impact Assessment:

- Seasonal Users: Includes adults and children of all ages. Seasonal Users are First Nations who live in communities outside the RSA but spend time within the local study area (LSA) and/or RSA throughout the year while pursuing traditional lifestyle activities (hunting, fishing, and gathering of traditional foods) and therefore may be exposed to air, soil, water and food items impacted by the Project.
- Gahcho Kué Worker: Includes adults only.

Exposure pathways applicable to each receptor are presented in Table IV-24. Exposure parameters used in the assessment are presented in Table IV-25.

Table IV-24 Exposure Pathways Evaluated in the Multi-Media Risk Assessment Based on Receptor Type

Exposure Pathway	Seasonal User	Gahcho Kué Worker
Inhalation of air	√	√
Inhalation of dust	√	√
Incidental ingestion of surface water	x	√
Dermal contact with surface water	x[(a)]	√
Ingestion of drinking water (potable water supply)	√	√
Dermal contact with drinking water (potable water supply)	√	√
Ingestion of soil	√	√
Dermal contact with soil	√	√
Ingestion of sediment	√	√
Dermal contact with sediment	√	√
Ingestion of fish	√	x
Ingestion of plants (berries, Labrador tea)	√	x
Ingestion of animals (caribou, hare)	√	x
Background dietary intake (food and water)	√	√

(a) The Seasonal User may come into contact with surface water and sediment while conducting traditional activities such as fishing; dermal contact with water was conservatively evaluated through dermal contact with potable water. Drinking water was assumed to be coming from Area 8 of Kennady Lake, which is a surface water body.

Note: √ = evaluated; x = not evaluated.

Table IV-25 Summary of Exposure Parameters for each Receptor

Parameters	Seasonal User					Gahcho Kué Worker	Source
	Infant	Toddler	Child	Adolescent	Adult	Adult	
	0 to 6 months	7 months to 4 years	5 to 11 years	12 to 19 years	20+ years	20+ years	
Body weight (kg)	8.2	16.5	32.9	59.7	70.7	70.7	1
Exposure duration (yrs) (carcinogens only)	0.5	4.5	7	8	60	60	1
Averaging time for carcinogens (period over which exposure is averaged)	80	80	80	80	80	80	1
Drinking Water Pathways							
Ingestion of chemicals in drinking water							
Ingestion rate (litres / day)	0.3	0.6	0.8	1	1.5	1.5	1
Days per week exposed (days/ 7days)	7/7	7/7	7/7	7/7	7/7	7/7	1
Weeks per year (weeks/ 52 weeks)	26/52	26/52	26/52	26/52	26/52	26/52	4
Dermal contact with drinking water							
Skin surface area available for contact (cm^2)[(a)]	3620	6130	10,140	15,470	17,640	3390	1
Event duration (hr/event)	0.33	0.33	0.33	0.25	0.25	0.25	7
Events per day	1	1	1	1	1	1	1
Days per week exposed (days/ 7days)	7/7	7/7	7/7	7/7	7/7	7/7	1
Weeks per year (weeks/ 52 weeks)	26/52	26/52	26/52	26/52	26/52	26/52	4
Surface Water Pathways							
Ingestion of chemicals in surface water							
Ingestion rate (litres /event)	0	0	0	0	0	0.05	2
Days per week exposed (days/ 7days)	0/7	0/7	0/7	0/7	0/7	7/7	1
Weeks per year (weeks/ 52 weeks)	0/52	0/52	0/52	0/52	0/52	26/52	1
Dermal contact with surface water							
Skin surface area available for contact (cm^2)[(a)]	3620	6130	10,140	15,470	17,640	3390	1
Event duration (hr/event)	0	0	0	0	0	1	1
Events per day	0	0	0	0	0	1	1
Days per week exposed (days/ 7days)	0/7	0/7	0/7	0/7	0/7	7/7	1
Weeks per year (weeks/ 52 weeks)	0/52	0/52	0/52	0/52	0/52	26/52	1
Food Ingestion Pathways							
Days per year (days/year)	365	365	365	365	365	365	1
Days per year conversion	365	365	365	365	365	365	1
Ingestion of chemicals in fish							
High consumer ingestion rate (kg fish / day)	0	0.095	0.17	0.2	0.22	0	1
Low consumer Ingestion rate (kg fish / day)	0	0.01	0.014	0.022	0.022	0	3
Fraction of fish consumed from the Site (unitless)	0.5	0.5	0.5	0.5	0.5	0	4
Ingestion of chemicals in produce (berries)							
Ingestion rate (kg berries / day)	0.004	0.004	0.004	0.004	0.015	0	5
Fraction of berries consumed from the Site (unitless)	0.5	0.5	0.5	0.5	0.5	0	4
Ingestion of chemicals in produce (Labrador tea leaves)							
Ingestion rate (kg produce/ day)	0	0.001	0.001	0.003	0.003	0	6
Fraction of Labrador tea leaves consumed from the Site (unitless)	0.5	0.5	0.5	0.5	0.5	0	4
Ingestion of chemicals in small game (snowshoe hare)							
Ingestion rate (kg snowshoe hare/ day)[(c)]	0	0.0085	0.0125	0.0175	0.027	0	1
Fraction of snowshoe hare consumed from the Site (unitless)	0.5	0.5	0.5	0.5	0.5	0	4
Ingestion of chemicals in large game (caribou)[(d)]							
Ingestion rate (kg game / day)	0	0.0765	0.1125	0.1575	0.243	0	1
Fraction of caribou consumed from the Site (unitless)	0.5	0.5	0.5	0.5	0.5	0	4
Soil Pathways							
Incidental ingestion of chemicals in soil							
Ingestion rate (kg/day)	0.00002	0.00008	0.00002	0.00002	0.00002	0.0001	1
Days per week exposed (days/ 7days)	7/7	7/7	7/7	7/7	7/7	7/7	1
Weeks per year (weeks/ 52 weeks)	26/52	26/52	26/52	26/52	26/52	26/52	4
Dermal contact with chemicals in soil							
Conversion factor (1E-06 kg/mg)	1.00E-06	1.00E-06	1.00E-06	1.00E-06	1.00E-06	1.00E-06	1

Table IV-25 Summary of Exposure Parameters for each Receptor (continued)

Parameters	Seasonal User					Gahcho Kué Worker	Source
	Infant	Toddler	Child	Adolescent	Adult	Adult	
	0 to 6 months	7 months to 4 years	5 to 11 years	12 to 19 years	20+ years	20+ years	
Skin surface area available for contact (hands) (cm^2/event)	320	430	590	800	890	890	1
Soil to skin adherence factor (hands) (mg/cm^2-event)	0.1	0.1	0.1	0.1	0.1	1	1
Skin surface area available for contact (other, i.e., arms + legs) (cm^2/event)	1460	2580	4550	7200	8220	8220	1
Soil to skin adherence factor (other) (mg/cm^2-event)	0.01	0.01	0.01	0.01	0.01	0.1	1
Events per day	1	1	1	1	1	1	1
Days per week exposed (days/ 7days)	7/7	7/7	7/7	7/7	7/7	7/7	1
Weeks per year (weeks/ 52 weeks)	26/52	26/52	26/52	26/52	26/52	26/52	4
Inhalation of chemicals in dust/particulate matter from soil							
Inhalation rate (m^3/day) [5]	2.2	8.3	14.5	15.6	16.6	25.46	1
Event duration (hour / 24 hours)	10/24	10/24	10/24	10/24	10/24	12/24	4
Days per week exposed (days/ 7days)	7/7	7/7	7/7	7/7	7/7	7/7	1
Weeks per year (weeks/ 52 weeks)	26/52	26/52	26/52	26/52	26/52	26/52	4
Portion of dry days (unitless)	0.52	0.52	0.52	0.52	0.52	0.52	4
Particulate emission factor (kg/m^3)	7.60E-10	7.60E-10	7.60E-10	7.60E-10	7.60E-10	7.60E-10	1
Sediment Pathways[(b)]							
Incidental ingestion of chemicals in sediment							
Ingestion rate (kg/day)	0.00002	0.00008	0.00002	0.00002	0.00002	0.0001	1
Days per week exposed (days/ 7days)	7/7	7/7	7/7	7/7	7/7	7/7	1
Weeks per year (weeks/ 52 weeks)	26/52	26/52	26/52	26/52	26/52	26/52	4
Dermal contact with chemicals in sediment							
Conversion factor (1E-06 kg/mg)	1.00E-06	1.00E-06	1.00E-06	1.00E-06	1.00E-06	1.00E-06	1
Skin surface area available for contact (hands) (cm^2/event)	320	430	590	800	890	890	1
Sediment to skin adherence factor (hands) (mg/cm^2-event)	0.66[(e)]	0.66[(e)]	0.66[(e)]	0.66[(e)]	0.66[(e)]	0.66[(e)]	1,8
Skin surface area available for contact (other, i.e., arms + legs) (cm^2/event)	1460	2580	4550	7200	8220	5720[(f)]	1
Sediment to skin adherence factor (other) (mg/cm^2-event)	0.16 [g]	0.16 [g]	0.16 [g]	0.16[(g)]	0.16[(g)]	0.036[(f)]	8
Events per day	1	1	1	1	1	1	1
Days per week exposed (days/ 7days)	7/7	7/7	7/7	7/7	7/7	7/7	1
Weeks per year (weeks/ 52 weeks)	26/52	26/52	26/52	26/52	26/52	26/52	4
Air Pathway							
Hours per day exposed (hour / 24 hours)	10/24	10/24	10/24	10/24	10/24	12/24	4
Days per week exposed (days/ 7days)	7/7	7/7	7/7	7/7	7/7	7/7	1
Weeks per year (weeks/ 52 weeks)	26/52	26/52	26/52	26/52	26/52	26/52	4

(a) The total body was used for the skin surface area for Seasonal User; only hands and arms were used for the worker.

(b) Parameters from soil were applied to the sediment pathway.

(c) The ingestion rate used was 10% of the First Nation wild game ingestion rate.

(d) The ingestion rate used was 90% of the First Nation wild game ingestion rate.

(e) Sediment to skin adherence factors for Seasonal Users are for soil to skin adherence and taken from Health Canada (2009a). The sediment to skin adherence factor for Gahcho Kué Workers is for the hands and is taken from Kissel et al. (1996).

(f) Arms only for the Gahcho Kué worker.

(g) Sediment to skin adherence factor for legs.

Source: Health Canada (2009a); U.S. EPA (1989) RAGS Part A; Health Canada (2007); Site specific assumption; Wein et al. (1991); Alberta Health and Wellness (2007); U.S. EPA (2004) RAGS Part E; Kissel et al. (1996)

IV.1.3.1 Bioavailability

Bioavailability (also referred to as absorption efficiency) is a measure of the amount of a chemical that is absorbed and retained within the body. Consideration of bioavailability may be important under the following circumstances (U.S. EPA 1989):

- if the medium of exposure is different than the medium on which the toxicity reference value is based (e.g., exposure is from soil, but the toxicity reference value is based on exposure from water);
- if the route of exposure is different than the route of exposures in the study used to derive the toxicity reference value (e.g., oral route of exposure, but based on an inhalation study); or
- the toxicity reference value derived by the regulatory agency has been adjusted for bioavailability.

In the human health assessment, exposure estimates were not adjusted for bioavailability because in the majority of cases, TRVs were expressed as the administered dose (i.e., amount taken into the body), rather than the absorbed dose (i.e., amount absorbed and retained in the body) and because bioavailability information was not available.

Special considerations - Arsenic in Fish

The forms of arsenic in fish and shellfish (i.e., arsenobetaine and arsenocholine) have been reported to be essentially non-toxic. However, a small percentage in fish tissue may be the toxic inorganic form. Therefore, an inorganic arsenic fish content of 10% was used in calculations for arsenic exposures via the fish pathway (ATSDR 2007).

In the human health assessment, chemical bioavailability was taken into account by using Relative Absorption Factors (RAFs). Oral and inhalation exposures were assumed to have a relative absorption of 100% (RAF = 1) as pathway specific Toxicity Reference Values (TRVs) were typically available. As TRVs typically do not exist for the dermal exposure pathway, the dermal exposures are estimated from the oral dose taking into account the relative bioavailability and absorption. For dermal bioavailability, the RAFs were obtained from obtained from Health Canada (2009b). In the absence of a Health Canada (2009b) value, relative absorption factors were selected from the Ontario MOE (OMoE 2011). Dermal RAFs used in the assessment are summarized in Table IV-26.

Table IV-26 Chemical Specific Properties Used in the Risk Assessment

Chemical of Concern	Soil Relative Dermal Absorption Factor (unitless)	Gastro-Intestinal Tract Absorption Factor (unitless)	Water Dermal Permeability Coefficient (cm/hour)	Soil/Sediment Relative Dermal Absorption Factor Reference	Gastro-Intestinal Tract Absorption Factor Reference	Water Dermal Permeability Coefficient Reference
Aluminum (NC)	0.01	1	1.00E-03	OMoE, 2011	Default	RAIS 2012
Antimony (NC)	0.1	1	1.00E-03	Health Canada, 2009b	Default	RAIS 2012
Arsenic (C)	0.03	1	1.00E-03	Health Canada, 2009b	Default	RAIS 2012
Cadmium (C)	0.01	1	1.00E-03	Health Canada, 2009b	Default	RAIS 2012
Cobalt (NC)	0.01	1	4.00E-04	Health Canada, 2009b	Default	RAIS 2012
Iron (NC)	0.01	1	1.00E-03	OMoE, 2011	Default	RAIS 2012
Manganese (NC)	0.01	1	1.00E-03	OMoE, 2011	Default	RAIS 2012
Nickel (C)	0.01	1	2.00E-04	Health Canada, 2009b	Default	RAIS 2012
Thallium	0.01	1	1.00E-03	OMoE, 2011	Default	RAIS 2012
Titanium (NC)	0.01	1	N/A	OMoE, 2011	Default	N/A
Vanadium (NC)	0.1	1	1.00E-03	Health Canada, 2009b	Default	RAIS 2012

Notes: NC - non-carcinogen, C – carcinogen; N/A - not applicable. The parameter was not measured in surface water.

IV.1.3.2 Background Dietary Intake

Background exposure from dietary sources (e.g., supermarket food) was included in the human health risk assessment for Seasonal Users and workers. Health Canada carries out Canadian Total Diet Studies (TDS; also referred to as Market Based Surveys Studies, which provide estimate levels of exposure to chemicals that Canadians in different age-sex groups accumulate through the food supply (Health Canada 2011a). The World Health Organization recommends these studies as they provide reliable estimates of dietary intakes of contaminants (Health Canada 2011a). Average dietary intakes were taken from Health Canada's TDS where possible, for the most recent TDS available (i.e., Vancouver in September 2007). Where values were not available for a particular COC from Health Canada, values were taken from the World Health Organization and the Agency of Toxic Substances and Disease Registry (ATSDR). Average dietary intakes used in the human health risk assessment are provided in Table IV-27 below.

Table IV-27 Average Dietary Intakes

Chemical of Concern	Average Dietary Intakes					
	Seasonal User					Gahcho Kué Worker
	Infant	Toddler	Child	Adolescent	Adult	Adult
	max value for age 0-6 months [mg/kg BW/day]	7 mo-4 years [mg/kg BW/day]	5-11 years [mg/kg BW/day]	12-19 years [mg/kg BW/day]	max value for age 20-65+ years [mg/kg BW/day]	max value for age 20-65+ years [mg/kg BW/day]
Metals						
Aluminum (NC)[a]	0.051	0.171	0.203	0.177	0.116	0.116
Antimony (NC)[b]	0.000561	0.000279	0.000140	0.000077	0.000065	0.000065
Arsenic (C)[a]	0.0007	0.00141	0.00177	0.00122	0.00144	0.00144
Cadmium (C)[b]	0.00022	0.00057	0.00048	0.00032	0.00025	0.00025
Cobalt (NC)[a]	0.00063	0.00057	0.00041	0.00029	0.00023	0.00023
Iron (NC)[c]	0.00215	0.000107	0.000535	0.000295	0.000249	0.000249
Manganese (NC)[a]	0.067	0.099	0.083	0.056	0.064	0.064
Nickel (C)[a]	0.0142	0.0098	0.0075	0.0062	0.005	0.005
Thallium (NC)[a]	0.00004	0.000033	0.000021	0.000015	0.000015	0.000015
Titanium (NC)[d]	0.0976	0.0485	0.0243	0.0134	0.0113	0.0113
Vanadium (NC)[e]	0.00082	0.00039	0.00027	0.00018	0.00026	0.00026

(a) Health Canada (2011a).

(b) Dietary intake for antimony taken from Health Canada (2011b). The total antimony intake value (0.0046 mg/day) was divided by the body weights of the receptors: 8.2 kg for infant, 16.5 kg for toddler, 32.9 kg for child, 59.7 kg for teen, 70.7 kg for adult, as per HC PQRA Part I (Health Canada 2009a).

(c) Dietary intake for iron taken from Health Canada (1978, updated 1987). Average daily intake value of iron from typical Canadian diets, estimated as 17.6 mg, was divided by 1000 to convert from mg to g, and divided by body weights of the receptors: 8.2 kg for infant, 16.5 kg for toddler, 32.9 kg for child, 59.7 kg for teen, 70.7 kg for adult, as per HC PQRA Part I (Health Canada 2009a).

(d) Dietary intake for titanium taken from WHO (1982). Daily intake value of 800 µg as estimated by Hamilton and Minski (1972/1973, as cited in WHO 1982) from the United Kingdom. Value was divided by body weights of the receptors: 8.2 kg for infant, 16.5 kg for toddler, 32.9 kg for child, 59.7 kg for teen, 70.7 kg for adult, as per HC PQRA Part I (Health Canada 2009a).

(e) Dietary intake for vanadium taken from ATSDR (2009). Intake values in µg/day were divided by 1000 to convert from µg to mg and were divided by body weights of the receptors: 8.2 kg for infant, 16.5 kg for toddler, 32.9 kg for child, 59.7 kg for teen, 70.7 kg for adult, as per HC PQRA Part I (Health Canada 2009a). Intake values in µg/day were listed as: 6.7 for infant, 6.5 for toddler, no value listed for child (therefore used average between that for teen and that for toddler which is 8.8), 11 for teen (male; higher than value for female of 7.1), 18.3 for adult (highest adult intake listed).

IV.1.4 Exposure Equations

For the multi-media assessment, exposure is determined as a dose. This value is called the EDI and is typically expressed as mg/kg BW/day. Equations used to calculate the estimated daily intake (EDI) for the multi-media risk assessment are provided below (Equation 1 to 11). Example calculations for calculating EDI for each pathway for carcinogens and non-carcinogens are provided in Section IV-1.5.

Ingestion of Contaminated Drinking Water Dose Rate:

$$DR_{DW} = \frac{C_W \times IR_W \times RAF_{oral} \times D_1 \times D_2 \times D_3}{BW \times LE} \quad \text{Equation 1}$$

Where:

DR_{DW}= dose rate from ingestion of COC in drinking water (mg/kg bw-day)

IR_W = water intake rate (L/d)

C_w = COC concentration in groundwater (mg/L)

RAF_{oral} = relative absorption factor from the GI tract (unitless). RAFs were conservatively assumed to be equal to 1.

D_1= days per week exposed /7 days

D_2= weeks per year exposed /52 weeks

D_3= total years exposed to site (for carcinogens only)

BW = body weight (kg)

LE = life expectancy (yr) (for carcinogens only)

Ingestion of Food Items Dose Rate (used for fish, berry, Labrador tea leaves, hare and caribou):

$$DR_F = \frac{C_F \times IR_F \times FF \times D_1 \times D_2 \times D_3}{BW \times LE} \quad \text{Equation 2}$$

Where:

DR_F = estimated dose from ingestion of COC in food item (mg/kg bw/day)

IR_F = ingestion rate for food item (kg/day)

C_F = COC concentration in food item (mg/kg)

FF = fraction of food item in diet obtained from the site (unitless)

D_1= days per week exposed / 7 days

D_2= weeks per year exposed /52 weeks

D_3= total years exposed to site (for carcinogens only)

BW = body weight (kg)

LE = life expectancy (yr) (for carcinogens only)

Inadvertent Sediment Ingestion Dose Rate:

DR_S =	$\frac{C_S \times IR_S \times AF_S \times D_1 \times D_2 \times D_3}{BW \times LE}$	Equation 3

Where:

DR_S = estimated dose from ingestion of COC in sediment (mg/kg bw/day)

IR_S = sediment ingestion rate (kg/day)

C_S = COC concentration in sediment (mg/kg)

AF_S = bioavailability factor via ingestion of sediment (unitless) - conservatively assumed to be 1 for this risk assessment.

D_1= days per week exposed / 7 days

D_2= weeks per year exposed /52 weeks

D_3= total years exposed to site (for carcinogens only)

BW = body weight (kg)

LE = life expectancy (yr) (for carcinogens only)

Dermal Contact with Sediment:

DR_{DC} =	$\frac{C_S/T \times SA \times AD \times AF_{DC} \times UCF \times D_1 \times D_2 \times D_3}{BW \times LE}$	Equation 4

Where:

DR_{DC} = dose rate from dermal contact with COC in sediment (mg/kg bw/day)

SA = skin surface area available for dermal contact (cm^2/event)

AD = adherence factor (soil to skin; mg/cm^2)

C_S = COC concentration in sediment (mg/kg)

AF_{DC} = bioavailability via dermal contact (unitless) – conservatively assumed to be 1 for this risk assessment

UCF=unit conversion factor (1×10^{-6} kg/mg)

D_1= days per week exposed / 7 days

D_2= weeks per year exposed /52 weeks

D_3= total years exposed to site (for carcinogens only)

BW = body weight (kg)

LE = life expectancy (yr) (for carcinogens only)

Inadvertent Ingestion of Surface Water:

DR_{SW} =	$\frac{IR_{SW} \times CD_{SW} \times AFGIT \times D_1 \times D_2 \times D_3}{BW \times LE}$	Equation 5

Where:

DR_{SW} = estimated dose from ingestion of COC in surface water (mg/kg bw/day)

IR_{SW}= surface water ingestion rate (L/day)

C_{SW} = COC concentration in drinking water (mg/L)

AFGIT = Bioavailability factor via gastrointestinal tract

D_1= days per week exposed / 7 days

D_2= weeks per year exposed /52 weeks

D_3= total years exposed to site (for carcinogens only)

BW = body weight (kg)

LE = life expectancy (yr) (for carcinogens only)

Inadvertent Soil Ingestion Dose Rate:

DR_{SI} =	$\frac{C_S \times IR_S \times RAF_{oral} \times D_1 \times D_2 \times D_3}{BW \times LE}$	Equation 6

Where:
DR_{SI} = estimated dose from ingestion of COC in soil (mg/kg bw-day)
IR_S = soil ingestion rate (kg/day)
C_S = COC concentration in soil (mg/kg)
RAF_{oral} = bioavailability via soil ingestion, i.e., relative absorption factor from the gastrointestinal tract (unitless)
D_1= days per week exposed /7 days
D_2= weeks per year exposed /52 weeks
D_3= total years exposed to site (for carcinogens only)
BW = body weight (kg)
LE = life expectancy (yr) (for carcinogens only)

Soil Dermal Contact Dose Rate:

DR_{DC} =	$\frac{[(C_S \times SAH \times SLH) + (C_S \times SAO \times SLO)] \times RAF_{dermal} \times D_1 \times D_2 \times D_3}{BW \times LE}$	Equation 7

Where:
DR_{DC} = dose rate from dermal contact with COC in soil (mg/kg bw-day)
SAH = skin surface area available for dermal contact (hands) (m^2)
SLH = soil loading to exposed skin (hands) (kg/m^2-day)
SAO = skin surface area available for dermal contact (other than hands) (m^2)
SLO = soil loading to exposed skin (other than hands) (kg/m^2-day)
C_S = COC concentration in soil (mg/kg)
RAF_{dermal} = bioavailability via dermal contact (unitless)
D_1= days per week exposed /7 days
D_2= weeks per year exposed /52 weeks
D_3= total years exposed to site (for carcinogens only)
BW = body weight (kg)
LE = life expectancy (yr) (for carcinogens only)

Inhalation of Contaminated Soil Particles Dose Rate:

DR_{IH} =	$\frac{C_s \times P_{air} \times RAF_{inh} \times D_1 \times D_2 \times D_3 \times D_4}{LE}$	Equation 8

Where:
DR_{IH} = dose rate from inhalation of COC on airborne particles (mg/m^3)
C_S = COC concentration in soil (mg/kg)
Pair = particulate concentration in air (kg/m^3)
RAF_{inh} = bioavailability via inhalation (unitless)
D_1= hours per day exposed (hrs/day)
D_2= days per week exposed /7 days
D_3= weeks per year exposed /52 weeks
D_4= total years exposed to site (for carcinogens only)
LE = life expectancy (yr) (for carcinogens only)

Inhalation of Contaminant Air Dose Rate:

DR_V =	$C_A \times AF_{inh} \times D_1 \times D_2 \times D_3 \times D_4$ / LE	Equation 9

Where:

DR_V = dose rate from inhalation of COC in air (mg/m^3)

C_A = COC concentration in air (mg/m^3)

AF_{inh} = bioavailability via inhalation (unitless) – conservatively assumed to be 1.

D_1= hours per day exposed /24hr)

D_2= days per week exposed /7 days

D_3= weeks per year exposed /52 weeks

D_4= total years exposed to site (for carcinogens only)

BW = body weight (kg)

LE = life expectancy (yr) (for carcinogens only)

IV.1.4.1 Calculating the Absorbed Dermal Dose for Surface Water Contact

The approach to calculating the absorbed dermal dose is recommended by U.S. EPA (2004). Dermal absorption is based on a two-compartment model which is composed of two layers, the stratum corneum and the viable epidermis. The stratum corneum acts as the primary barrier to the COCs. The model describes the absorption of COCs from water through the skin as a function of the stratum corneum and the event duration (U.S. EPA 2004). The equations below are used to calculate the absorbed dermal dose for inorganic COCs.

Dermal Absorbed Dose (DA event) for Inorganic Substances in Surface Water

DA_{event} =	$K_p \times C_w \times t_{event}$	Equation 10

Where:

DA_{event} = Absorbed dose per event (mg/cm^2-event)

K_p = Dermal permeability coefficient of the compound in water (cm/hr); chemical specific, obtained from U.S. EPA (2004a)

C_w = Chemical concentration in water (mg/cm^3)

IV.1.5 Example Exposure Calculations

Based on the methods described in the main body of the report and the equations shown in Section 1.4, the EDI was calculated for each COC and pathway. Following calculation of the EDI, the Hazard Quotient (HQ) was calculated. An HQ is the ratio between the exposure likely to be incurred by the person (i.e., EDI) and the amount of exposure that is considered to be safe (i.e., toxicity reference value). The toxicity reference values used in the assessment were described in Appendix III. No health risk is predicted if the HQ is less than one. An example calculation is provided below for arsenic for an adult First Nations Seasonal User receptor for the Baseline Case.

Potable (Drinking) Water Ingestion Pathway

$$EDI_{water} = \frac{C_w \times IR_w \times RAF_{oral} \times D_1 \times D_2}{BW}$$

EDI_{water} = 0.00014 mg/L x 1.5 L/day x 1.0 x 7 days/7 days x 26 weeks/52 weeks / 70.7 kg
= 1.49E-06 mg/kg BW/day

HQ_{water} = EDI_{water} (mg/kg BW/day) / RfDo (mg/kg-BW/day) = 1.49E-06 mg/kg BW/day / 0.0003 mg/kg BW/day
= 4.95E-03

Fish Ingestion Pathway (High Consumption)

$$EDI_{fish} = \frac{C_{fish} \times IR_{fish} \times FF \times RAF_{GIT} \times D_1}{BW \times 365}$$

EDI_{fish} = 0.0509 mg/kg wet wt x 0.1* x 0.22 kg wet wt/day x 0.5 x 1.0 x 365 days / 70.7 kg x 365 days
=7.92E-06 mg/kg BW/day

HQ_{fish} = EDI_{berry} (mg/kg BW/day) / RfDo (mg/kg-BW/day) = 7.92E-06 mg/kg BW/day / 0.0003 mg/kg BW/day
= 2.64E-02

* assumed 10% was inorganic arsenic (ATSDR 2012 internet site)

Berry Ingestion Pathway

$$EDI_{berry} = \frac{C_{berry} \times IR_{berry} \times FF \times RAF_{GIT} \times D_1}{BW \times 365}$$

EDI_{berry} = 0.0400 mg/kg wet wt x 0.015 kg wet wt/day x 0.5 x 1.0 x 365 days / 70.7 kg x 365 days
=4.24E-06 mg/kg BW/day

HQ_{berry} = EDI_{berry} (mg/kg BW/day) / RfDo (mg/kg-BW/day) = 4.24E-06 mg/kg BW/day / 0.0003 mg/kg BW/day
= 1.41E-02

Northern Labrador Tea Leaf Ingestion Pathway

$$EDI_{leaf} = \frac{C_{LT} \times IR_{leaf} \times FF \times RAF_{GIT} \times D_1}{BW \times 365}$$

EDI_{LT} = 0.0415 mg/kg wet wt x 0.003 kg wet wt/day x 0.5 x 1.0 x 365 days / 70.7 kg x 365 days
=8.80E-07 mg/kg BW/day

HQ_{LT} = EDI leaf (mg/kg BW/day) / RfDo (mg/kg-BW/day) = 8.80E-07 mg/kg BW/day / 0.0003 mg/kg BW/day
= 2.93E-03

Snowshoe Hare Ingestion Pathway

$$EDI_{root} = \frac{C_{hare} \times IR_{hare} \times FF \times RAF_{GIT} \times D_{I}}{BW \times 365}$$

EDI_{lroot} = $\frac{2.69E\text{-}03 \text{ mg/kg wet wt} \times 0.027 \text{ kg wet wt/day} \times 0.5 \times 1.0 \times 365 \text{ days}}{70.7 \text{ kg} \times 365 \text{ days}}$
= 5.14E-07 mg/kg BW/day

HQ_{root} = $\frac{EDI_{root} \text{ (mg/kg BW/day)}}{\text{RfDo (mg/kg-BW/day)}} = \frac{5.14E\text{-}07 \text{ mg/kg BW/day}}{0.0003 \text{ mg/kg BW/day}}$
= 1.71E-03

Caribou Ingestion Pathway

$$EDI_{caribou} = \frac{C_{caribou} \times IR_{caribou} \times FF \times RAF_{GIT} \times D_{I}}{BW \times 365}$$

EDI_{moose} = $\frac{0.000430 \text{ mg/kg wet wt} \times 0.243 \text{ kg wet wt/day} \times 0.5 \times 1.0 \times 365 \text{ days/yr}}{70.7 \text{ kg} \times 365 \text{ days}}$
= 7.39E-07 mg/kg BW/day

HQ_{berry} = $\frac{EDI_{berry} \text{ (mg/kg BW/day)}}{\text{RfDo (mg/kg-BW/day)}} = \frac{7.39E\text{-}07 \text{ mg/kg BW/day}}{0.0003 \text{ mg/kg BW/day}}$
= 2.46E-03

Sediment Ingestion Pathway

$$EDI_{sed} = \frac{C_{sed} \times IR_{S} \times AF_{S} \times D_{1} \times D_{2}}{BW}$$

EDI_{sed} = $\frac{6.77 \text{ mg/kg} \times 0.00002 \text{ kg/day} \times 1.0 \times 7 \text{ days} / 7\text{days} \times 26 \text{ weeks} / 52 \text{ weeks}}{70.7 \text{ kg}}$
= 9.58E-07 mg/kg BW/day

HQ_{sed} = $\frac{EDI_{soil} \text{ (mg/kg BW/day)}}{\text{RfDo (mg/kg BW/day)}} = \frac{9.58E\text{-}07 \text{ mg/kg BW/day}}{0.0003 \text{ mg/kg BW/day}}$
= 3.19E-03

Dermal Contact with Sediment Pathway

$$EDI_{dermal\text{-}sed} = \frac{C_{S}/T \times SA \times AD \times AF_{DC} \times UCF \times D1 \times D2 \times D3}{BW}$$

$EDI_{sed\text{-}dermal}$ = $\frac{6.77 \text{mg/kg} \times 1E\text{-}06 \text{ kg/mgx} (890 \text{ cm}^2 \times 0.66 \text{ mg/cm}^2 \text{ -event + 8220 cm2} \times 0.16 \text{ mg/cm2-event}) \times 0.03 \times 1 \text{ event/day} \times 7 \text{ days per week} / 7\text{days} \times 26 \text{ weeks per year} / 52 \text{ weeks}}{70.7 \text{ kg}}$
= 2.73E-06 mg/kg BW/day

$HQ_{sed\text{-}dermal}$ = $\frac{EDI_{dermal} \text{ (mg/kg BW/day)}}{\text{RfDo (mg/kg BW/day)}} = \frac{2.73E\text{-}06 \text{ mg/kg BW/day}}{0.0003 \text{ mg/kg BW/day}}$
= 9.11E-03

Soil Ingestion Pathway

$$EDI_{soil} = \frac{C_{soil} \times IR_S \times RAF_{oral} \times D_1 \times D_2}{BW}$$

EDI_{soil} = $\frac{1.44 \text{ mg/kg} \times 0.00002 \text{ kg/day} \times 1.0 \times 7 \text{ days /7days} \times 26 \text{ weeks /52 weeks}}{70.7 \text{ kg}}$

= 2.04E-07 mg/kg BW/day

HQ_{soil} = $\frac{EDI_{soil} \text{ (mg/kg BW/day)}}{\text{RfDo (mg/kg BW/day)}} = \frac{2.04E\text{-}07 \text{ mg/kg BW/day}}{0.0003 \text{ mg/kg BW/day}}$

= 6.79E-04

Dermal Contact with Soil Pathway

$$EDI_{dermal} = \frac{[(C_S \times SAH \times SLH) + (C_S \times SAO \times SLO)] \times RAF_{dermal} \times EF \times D_1 \times D_2}{BW}$$

EDI_{dermal} = $\frac{1.44 \text{ mg/kg} \times (890 \text{ cm}^2 \times 0.0000001 \text{ kg/cm}^2 \text{ -event} + 8220 \text{ cm2} \times 0.00000001 \text{ kg/cm2-event}) \times 0.03 \times 1 \text{ event/day} \times 7 \text{ days per week/7days} \times 26 \text{ weeks per year/52 weeks}}{70.7 \text{ kg}}$

= 5.23E-08 mg/kg BW/day

HQ_{dermal} = $\frac{EDI_{dermal} \text{ (mg/kg BW/day)}}{\text{RfDo (mg/kg BW/day)}} = \frac{5.23E\text{-}08 \text{ mg/kg BW/day}}{0.0003 \text{ mg/kg BW/day}}$

= 1.74E-04

Inhalation of Soil Particulates (i.e., dust)

$$EDI_{dust} = C_S \times P_{air} \times IR_A \times RAF_{inh} \times D1 \times D2 \times D3 \times D4$$

EDI_{dust} = 1.44 mg/kg x 7.6E-10 kg/m^3 x 1 x 0.52 x 10hr/24hr x 7 days/7days x 26 weeks /52 weeks

= 1.19E-10 mg/m^3

HQ_{dust} = $\frac{EDI_{dust} \text{ (mg/m}^3\text{)}}{\text{RfC(mg/m}^3\text{)}} = \frac{1.19E\text{-}10 \text{ mg/m}^3}{0.001 \text{ mg/m}^3}$

= 1.19E-07

Air Inhalation Pathway

$$EDI_{air} = C_a \times AF_{Inh} \times D_1 \times D_2 \times D_3$$

EDI_{air} = 3.25E-11 mg/m^3 x 1 x 10hr/24hr x 7 days/7days x 26 weeks/52 weeks

= 6.77E-12 mg/m^3

HQ_{air} = $\frac{EDI_{air} \text{ (µg/m}^3\text{)}}{\text{RfC (µg/m}^3\text{)}} = \frac{6.77E\text{-}12 \text{ mg/m}^3}{0.001 \text{ mg/m}^3}$

= 6.77E-09

Dermal Contact with Potable Water Pathway

$$EDI_{water\text{-}dermal} = \frac{C_w \times K_p \times t_{event} \times SA_{skin} \times EF \times D_1 \times D_2}{BW}$$

$$EDI_{water} = \frac{0.00014 \text{ mg/L} \times (1\times10^{-3}) \times 0.001 \text{ cm/h} \times 0.25 \text{ h} \times 17{,}640 \text{ cm2} \times 1.0 \times 7\text{days}/7\text{days} \times 26 \text{ wks}/52 \text{ wks}}{70.7 \text{ kg}}$$

$$= 4.37E\text{-}09 \text{ mg/kg BW/day}$$

$$HQ_{water} = \frac{EDI_{water} \text{ (mg/kg BW/day)}}{\text{RfDo (mg/kg-BW/day)}} = \frac{4.37E\text{-}09 \text{ mg/kg BW/day}}{0.0003 \text{ mg/kg BW/day}}$$

$$= 1.46E\text{-}05$$

Total Hazard Quotient from all Pathways

$$\text{Total HQ} = HQ_{water} + HQ_{fish} + HQ_{berries} + HQ_{LT} + HQ_{hare} + HQ_{caribou} + HQ_{sed\text{-}ing} + HQ_{sed\text{-}derm} + HQ_{soil\text{-}ing} + HQ_{soil\text{-}derm} + HQ_{dust} + HQ_{air} + HQ_{water\text{-}derm} + HQ_{background\ dietary\ intake}$$

$$\text{Total HQ} = 4.95E\text{-}03 + 2.64E\text{-}02 + 1.41E\text{-}02 + 2.93E\text{-}03 + 1.71E\text{-}03 + 2.46E\text{-}03 + 3.19E\text{-}03 + 9.11E\text{-}03 + 6.79E\text{-}04 + 1.74E\text{-}04 + 1.19E\text{-}07 + 6.77E\text{-}09 + 1.46E\text{-}05 + 4.80E+00$$

$$= 4.87E+00$$

IV.2 REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 2009. Toxicological Profile for Vanadium. US Department of Health and Human Services. Public Health Service, ATSDR. September 2009. Available at: http://www.atsdr.cdc.gov/substances/index.asp. Accessed August 2012.

Alberta Health and Wellness. 2007. Assessment of the potential lifetime cancer risk associated with exposure to inorganic arsenic among indigenous people living in the Wood Buffalo Region of Alberta. Prepared by Cantox Environmental Inc., Calgary, Alberta.

ATSDR (Agency for Toxic Substances and Disease Registry). 2007. Toxicological Profile for Arsenic. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Available on-line at: http://www.atsdr.cdc.gov/ToxProfiles/tp2.pdf

Beyer WN, Connor EE and A Gerould. 1994. Estimates of Soil Ingestion by Wildlife. Journal of Wildlife Management 58(2): 375-382.

CWS and CWF (Canadian Wildlife Service and Canadian Wildlife Federation). 2005. Hinterland Who's Who: Snowshoe Hare. Available at http://www.hww.ca/en/species/mammals/snowshoe-hare.html. Accessed August 2012.

Golder (Golder Associates Ltd.). 2010. Christina Lake Thermal Expansion Project, Phases 1E, 1F, and 1G 2010 Update, Appendix 2-VI – Regional Data Analysis. Report submitted to Cenovus FCCL Ltd. August 2010.

Health Canada. 1978 (updated 1987). Guidelines for Drinking Water Quality - Supporting Documents - Iron. Available at: http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/iron-fer/index-eng.php. Accessed August 2012.

Health Canada 2007. Human Health Risk Assessment of Mercury in Fish and Health Benefits of Fish Consumption. Bureau of Chemical safety Food Directorate Health Products and Food Branch.

Health Canada. 2009a. Federal Contaminated Site Risk Assessment in Canada Part 1: Guidance on Human Health Preliminary Quantitative Risk Assessment Version 2.0. . Ottawa, ON.

Health Canada. 2009b. Federal Contaminated Sites Risk Assessment in Canada. Part II. Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors, Version 2.0, May 2009. Health Canada. Ottawa, ON.

Health Canada. 2011a. Dietary Intakes of Contaminants & Other Chemicals for Different Age-Sex Groups of Canadians. Table for Vancouver, 2007: Average dietary intakes (µg/kg bw/day) of trace elements for Canadians in different age/sex groups for Total Diet Study in 2007. Available at: http://www.hc-sc.gc.ca/fn-an/surveill/total-diet/intake-apport/index-eng.php. Accessed August 2012.

Health Canada. 2011b. Water Quality and Health - Antimony. Exposure Section. http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/antimony-antimoine/exposure-expositions-eng.php. Accessed August 2012.

Kissel JC, Richter KY and RA Fenske. 1996. Field Measurement of Dermal Soil Loading Attributable to Various Activities: Implications for Exposure Assessment. Risk Analysis 16(1): 115-125.

MacDonald C and A Gunn. 2004. Analysis of the ash weight and elemental composition in caribou (Rangifer tarandus) faecal pellets collected at Colomac and other sites in the NWT. Manuscript Report No. 159. Department of Resources, Wildlife and Economic Development. Government of Northwest Territories. Yellowknife, NWT.

OMoE (Ontario Ministry of the Environment). 2011. Rationale for the Development of Generic Soil and Groundwater Standards for Use at Contaminated Sites in Ontario. April 15, 2011. PIBS 7386e01. Available at: http://www.ene.gov.on.ca/environment/en/resources/STDPROD_086519.html. Accessed August 2012.

RAIS (Risk Assessment Information System). 2012. Online Database and Web Resource. U.S. Department of Energy (DOE), Office of Environmental Management, Oak Ridge Operations (ORO) Office. Available at: http://rais.ornl.gov/. Accessed August 2012.

Sample, B.E., M.S. Aplin, R.A. Efroymson, G.W. Suter, II., and C.J.E. Welsh. 1997. Methods and tools for estimation of the exposure of terrestrial wildlife to contaminants. Oak Ridge National Laboratory, Oak Ridge TN. ORNL/TM-13391.

Smith HC. 1993. Alberta Mammals: An Atlas and Guide. 239 pp.

USDA (United States Department of Agriculture) National Nutrient Database for Standard Reference. 2011. Available at: http://www.nal.usda.gov/fnic/foodcomp/search/ Accessed August 2012.

U.S. EPA (United States Environmental Protection Agency) 1989. *Risk assessment guidance for Superfund Volume 1. Human health evaluation manual*. (Part A). EPA/540/1-89/002. Available at: http://www.epa.gov/oswer/riskassessment/ragsa/ Accessed August 2012.

U.S. EPA. 2006a. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. U.S. EPA Office of Solid Waste. EPA520-R-05-006. Washington, DC.

U.S. EPA. 2006b. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities Appendix A-1-Contaminants of Potential Concern. U.S. EPA Office of Solid Waste. EPA520-R-05-006. Washington, DC.

U.S. EPA. 2007. Guidance for Developing Ecological Soil Screening Levels (Eco-SSLs). Attachment 4-1: Exposure factors and bioaccumulation models for derivation of wildlife Eco-SSLs. OSWER Directive 9285.7-55.

U.S. EPA. 2004. Risk assessment guidance for Superfund Volume 1. Human health evaluation manual (Part E, Supplemental guidance for dermal risk assessment) EPA/540/R/99/005. Available at: http://www.epa.gov/oswer/riskassessment/ragse/pdf/part_e_final_revision_10-03-07.pdf. Accessed August 2012.

Wein EE, Sabry JH and FT Evers. 1991. Food consumption patterns and use of country foods by Native Canadians near Wood Buffalo National Park, Canada. Arctic 44(3): 196-205.

WHO (World Health Organization). 1982. International Programme on Chemical Safety. Environmental Health Criteria 24. Titanium. World Health Organization, Geneva, Switzerland. Available at: http://www.inchem.org/documents/ehc/ehc/ehc24.htm. Accessed August 2012.

APPENDIX V

MULTI-MEDIA EXPOSURE DOSE AND RISK ESTIMATES

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	1.79E-04	7.14E-07	1.79E-04	7.14E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Antimony (NC)	1.83E-06	7.28E-09	3.05E-04	1.21E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Arsenic (C)	2.56E-06	1.02E-08	8.54E-03	3.40E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium (C)	3.66E-07	1.46E-09	7.32E-04	2.91E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cobalt (C)	2.47E-06	3.93E-09	8.23E-03	1.31E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Iron (NC)	1.19E-03	4.73E-06	1.70E-03	6.76E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Manganese (NC)	2.23E-04	8.89E-07	2.23E-03	8.89E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Nickel (C)	5.85E-06	4.66E-09	5.32E-04	4.24E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Thallium (NC)	3.84E-07	1.53E-09	3.84E-02	1.53E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	4.39E-06	1.75E-08	8.78E-04	3.50E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			2.41E-03	9.60E-06			0.00E+00	0.00E+00		0.00E+00		0.00E+00
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

NA

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m³

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.60E-08	6.37E-11	2.88E-08	1.15E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium (C)	2.29E-09	9.11E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cobalt (C)	1.54E-08	2.46E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Nickel (C)	3.66E-08	2.91E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m³

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	3.36E-02	0.00E+00	0.00E+00	0.00E+00	3.36E-02	0.00E+00	0.00E+00	0.00E+00	5.10E-02	5.10E-02	7.44E-03	1.73E-04	5.03E-07	7.44E-03	1.73E-04	1.01E-04	4.04E-02	8.97E-03	4.04E-02	8.97E-03	4.63E-09	9.26E-07	1.42E-01	1.42E-01
Antimony (NC)	2.65E-06	0.00E+00	0.00E+00	0.00E+00	4.42E-04	0.00E+00	0.00E+00	0.00E+00	5.61E-04	9.35E-02	1.22E-07	2.84E-08	8.23E-12	2.03E-05	4.74E-06	4.12E-08	5.23E-07	1.16E-06	8.72E-05	1.94E-04	1.23E-13	6.13E-10	9.46E-02	9.46E-02
Arsenic (C)	9.76E-06	0.00E+00	0.00E+00	0.00E+00	3.25E-02	0.00E+00	0.00E+00	0.00E+00	7.00E-04	***2.33E+00***	1.75E-06	1.22E-07	1.18E-10	5.84E-03	4.08E-04	1.18E-07	8.26E-06	5.51E-06	2.75E-02	1.84E-02	6.77E-12	6.77E-09	***2.43E+00***	***2.43E+00***
Cadmium (C)	1.23E-05	0.00E+00	0.00E+00	0.00E+00	1.23E-02	0.00E+00	0.00E+00	0.00E+00	2.20E-04	2.20E-01	2.33E-07	5.43E-09	1.57E-11	2.33E-04	5.43E-06	1.57E-06	5.83E-07	1.30E-07	5.83E-04	1.30E-04	1.07E-10	1.07E-05	2.34E-01	2.34E-01
Cobalt (C)	1.83E-05	0.00E+00	0.00E+00	0.00E+00	6.11E-02	0.00E+00	0.00E+00	0.00E+00	6.30E-04	***2.10E+00***	5.01E-06	1.17E-07	3.38E-10	1.67E-02	3.89E-04	5.64E-05	2.61E-05	5.81E-06	8.71E-02	1.94E-02	2.45E-11	4.08E-06	***2.29E+00***	***2.29E+00***
Iron (NC)	1.53E-02	0.00E+00	0.00E+00	0.00E+00	2.18E-02	0.00E+00	0.00E+00	0.00E+00	2.15E-03	3.07E-03	1.07E-02	2.50E-04	7.25E-07	1.53E-02	3.58E-04	NA	5.80E-02	1.29E-02	8.28E-02	1.84E-02	8.65E-09	NA	1.44E-01	1.44E-01
Manganese (NC)	1.79E-02	0.00E+00	0.00E+00	0.00E+00	1.79E-01	0.00E+00	0.00E+00	0.00E+00	6.70E-02	6.70E-01	8.07E-05	1.88E-06	5.45E-09	8.07E-04	1.88E-05	1.09E-04	3.31E-03	7.36E-04	3.31E-02	7.36E-03	1.49E-10	2.97E-06	8.93E-01	8.93E-01
Nickel (C)	2.10E-04	0.00E+00	0.00E+00	0.00E+00	1.91E-02	0.00E+00	0.00E+00	0.00E+00	1.42E-02	***1.29E+00***	8.05E-05	1.88E-06	5.43E-09	7.32E-03	1.71E-04	3.02E-04	4.70E-05	1.05E-05	4.27E-03	9.50E-04	1.25E-10	6.96E-06	***1.32E+00***	***1.32E+00***
Thallium (NC)	1.46E-06	0.00E+00	0.00E+00	0.00E+00	1.46E-01	0.00E+00	0.00E+00	0.00E+00	4.00E-05	***4.00E+00***	1.26E-07	2.93E-09	8.48E-12	1.26E-02	2.93E-04	NA	2.93E-07	6.51E-08	2.93E-02	6.51E-03	6.01E-14	NA	***4.23E+00***	***4.23E+00***
Titanium (NC)	1.43E-03	0.00E+00	0.00E+00	0.00E+00	NA	NA	NA	NA	9.76E-02	NA	3.68E-04	8.56E-06	2.48E-08	NA	NA	2.48E-04	3.29E-04	7.31E-05	NA	NA	3.49E-10	3.49E-06	2.52E-04	2.52E-04
Vanadium (NC)	3.67E-05	0.00E+00	0.00E+00	0.00E+00	7.35E-03	0.00E+00	0.00E+00	0.00E+00	8.20E-04	1.64E-01	1.57E-05	3.65E-06	1.06E-09	3.14E-03	7.31E-04	1.06E-05	6.22E-05	1.38E-04	1.24E-02	2.76E-02	1.31E-11	1.31E-07	2.16E-01	2.16E-01
SUM HQ - chemicals with similar target organs/effects																								
Neurotoxicity (Al, Mn)					2.13E-01	0.00E+00	0.00E+00	0.00E+00		7.21E-01				8.25E-03	1.92E-04	2.09E-04			7.34E-02	1.63E-02		3.90E-06	***1.03E+00***	***1.03E+00***
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)																6.17E-04						1.47E-05	6.32E-04	6.32E-04

NA

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) excep

Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation	Total ILCR	Total ILCR
Metals																								
Arsenic (C)	6.10E-08	0.00E+00	0.00E+00	0.00E+00	1.10E-07	0.00E+00	0.00E+00	0.00E+00	4.38E-06	7.88E-06	1.09E-08	7.65E-10	7.39E-13	1.97E-08	1.38E-09	4.73E-12	5.16E-08	3.44E-08	9.29E-08	6.20E-08	4.23E-14	2.71E-13	8.19E-06	8.19E-06
Cadmium (C)	7.71E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.38E-06	0.00E+00	1.46E-09	3.39E-11	9.83E-14	0.00E+00	0.00E+00	1.77E-13	3.64E-09	8.10E-10	0.00E+00	0.00E+00	6.66E-13	1.20E-12	1.38E-12	1.38E-12
Cobalt (C)	1.15E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.94E-06	0.00E+00	3.13E-08	7.30E-10	2.11E-12	0.00E+00	0.00E+00	3.81E-12	1.63E-07	3.63E-08	0.00E+00	0.00E+00	1.53E-13	2.75E-13	4.08E-12	4.08E-12
Nickel (C)	1.31E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.88E-05	0.00E+00	5.03E-07	1.17E-08	3.40E-11	0.00E+00	0.00E+00	2.41E-11	2.94E-07	6.53E-08	0.00E+00	0.00E+00	7.83E-13	5.56E-13	2.47E-11	2.47E-11
SUM ILCR - chemicals with similar target organs/effects																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																3.28E-11						2.30E-12	3.51E-11	3.51E-11

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assess

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which ex

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	2.01E-04	8.02E-07	2.01E-04	8.02E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Antimony (NC)	1.99E-06	7.91E-09	3.31E-04	1.32E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Arsenic (C)	2.91E-06	1.16E-08	9.70E-03	3.86E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium (C)	4.74E-07	1.89E-09	9.48E-04	3.78E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cobalt (C)	2.79E-06	4.44E-09	9.28E-03	1.48E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Iron (NC)	1.35E-03	5.36E-06	1.92E-03	7.66E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Manganese (NC)	2.50E-04	9.97E-07	2.50E-03	9.97E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Nickel (C)	6.66E-06	5.30E-09	6.05E-04	4.82E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Thallium (NC)	4.24E-07	1.69E-09	4.24E-02	1.69E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	4.90E-06	1.95E-08	9.81E-04	3.91E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			2.70E-03	1.08E-05			0.00E+00	0.00E+00		0.00E+00		0.00E+00
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m³.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.82E-08	7.25E-11	3.28E-08	1.30E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium (C)	2.96E-09	1.18E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cobalt (C)	1.74E-08	2.77E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Nickel (C)	4.16E-08	3.31E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m³.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	4.11E-02	0.00E+00	0.00E+00	0.00E+00	4.11E-02	0.00E+00	0.00E+00	0.00E+00	5.10E-02	5.10E-02	7.70E-03	1.79E-04	5.20E-07	7.70E-03	1.79E-04	1.04E-04	4.04E-02	8.97E-03	4.04E-02	8.97E-03	5.04E-04	1.01E-01	2.50E-01	2.50E-01
Antimony (NC)	2.95E-06	0.00E+00	0.00E+00	0.00E+00	4.91E-04	0.00E+00	0.00E+00	0.00E+00	5.61E-04	9.35E-02	1.31E-07	3.06E-08	8.86E-12	2.19E-05	5.10E-06	4.4312E-08	5.23E-07	1.16E-06	8.72E-05	1.94E-04	1.85E-08	9.26E-05	9.47E-02	9.47E-02
Arsenic (C)	1.05E-05	0.00E+00	0.00E+00	0.00E+00	3.50E-02	0.00E+00	0.00E+00	0.00E+00	7.00E-04	***2.33E+00***	1.78E-06	1.24E-07	1.20E-10	5.92E-03	4.14E-04	1.1998E-07	8.26E-06	5.51E-06	2.75E-02	1.84E-02	5.12E-08	5.12E-05	***2.43E+00***	***2.43E+00***
Cadmium (C)	3.89E-05	0.00E+00	0.00E+00	0.00E+00	3.89E-02	0.00E+00	0.00E+00	0.00E+00	2.20E-04	2.20E-01	9.45E-07	2.20E-08	6.38E-11	9.45E-04	2.20E-05	6.3806E-06	5.83E-07	1.30E-07	5.83E-04	1.30E-04	1.42E-06	1.42E-01	4.04E-01	4.04E-01
Cobalt (C)	3.42E-05	0.00E+00	0.00E+00	0.00E+00	1.14E-01	0.00E+00	0.00E+00	0.00E+00	6.30E-04	***2.10E+00***	5.51E-06	1.28E-07	3.72E-10	1.84E-02	4.28E-04	6.19E-05	2.61E-05	5.81E-06	8.71E-02	1.94E-02	9.85E-07	1.64E-01	***2.51E+00***	***2.51E+00***
Iron (NC)	2.76E-02	0.00E+00	0.00E+00	0.00E+00	3.94E-02	0.00E+00	0.00E+00	0.00E+00	2.15E-03	3.07E-03	1.12E-02	2.61E-04	7.57E-07	1.60E-02	3.73E-04	NA	5.80E-02	1.29E-02	8.28E-02	1.84E-02	9.45E-04	NA	1.62E-01	1.62E-01
Manganese (NC)	2.11E-02	0.00E+00	0.00E+00	0.00E+00	2.11E-01	0.00E+00	0.00E+00	0.00E+00	6.70E-02	6.70E-01	8.76E-05	2.04E-06	5.91E-09	8.76E-04	2.04E-05	1.18E-04	3.31E-03	7.36E-04	3.31E-02	7.36E-03	1.37E-05	2.75E-01	***1.20E+00***	***1.20E+00***
Nickel (C)	4.91E-04	0.00E+00	0.00E+00	0.00E+00	4.46E-02	0.00E+00	0.00E+00	0.00E+00	1.42E-02	***1.29E+00***	8.38E-05	1.95E-06	5.66E-09	7.61E-03	1.77E-04	3.14E-04	4.70E-05	1.05E-05	4.27E-03	9.50E-04	6.46E-06	3.59E-01	***1.71E+00***	***1.71E+00***
Thallium (NC)	1.64E-06	0.00E+00	0.00E+00	0.00E+00	1.64E-01	0.00E+00	0.00E+00	0.00E+00	4.00E-05	***4.00E+00***	1.30E-07	3.03E-09	8.77E-12	1.30E-02	3.03E-04	NA	2.93E-07	6.51E-08	2.93E-02	6.51E-03	8.47E-09	NA	***4.26E+00***	***4.26E+00***
Titanium (NC)	2.06E-03	0.00E+00	0.00E+00	0.00E+00	NA	NA	NA	NA	9.76E-02	NA	3.92E-04	9.13E-06	2.65E-08	NA	NA	2.65E-04	3.29E-04	7.31E-05	NA	NA	4.85E-05	4.85E-01	4.85E-01	4.85E-01
Vanadium (NC)	5.84E-05	0.00E+00	0.00E+00	0.00E+00	1.17E-02	0.00E+00	0.00E+00	0.00E+00	8.20E-04	1.64E-01	1.65E-05	3.84E-06	1.11E-09	3.30E-03	7.68E-04	1.11E-05	6.22E-05	1.38E-04	1.24E-02	2.76E-02	1.59E-06	1.59E-02	2.37E-01	2.37E-01
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					2.52E-01	0.00E+00	0.00E+00	0.00E+00		7.21E-01				8.57E-03	2.00E-04	2.22E-04			7.34E-02	1.63E-02		3.76E-01	***1.45E+00***	***1.45E+00***
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V																6.52E-04						***1.02E+00***	***1.02E+00***	***1.02E+00***

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW =

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation	Total ILCR	Total ILCR
Metals																								
Arsenic (C)	6.56E-08	0.00E+00	0.00E+00	0.00E+00	1.18E-07	0.00E+00	0.00E+00	0.00E+00	4.38E-06	7.88E-06	1.11E-08	7.76E-10	7.50E-13	2.00E-08	1.40E-09	4.80E-12	5.16E-08	3.44E-08	9.29E-08	6.20E-08	3.20E-10	2.05E-09	8.20E-06	8.20E-06
Cadmium (C)	2.43E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.38E-06	0.00E+00	5.91E-09	1.38E-10	3.99E-13	0.00E+00	0.00E+00	7.18E-13	3.64E-09	8.10E-10	0.00E+00	0.00E+00	8.90E-09	1.60E-08	1.60E-08	1.60E-08
Cobalt (C)	2.14E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.94E-06	0.00E+00	3.44E-08	8.02E-10	2.32E-12	0.00E+00	0.00E+00	4.18E-12	1.63E-07	3.63E-08	0.00E+00	0.00E+00	6.16E-09	1.11E-08	1.11E-08	1.11E-08
Nickel (C)	3.07E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.88E-05	0.00E+00	5.24E-07	1.22E-08	3.53E-11	0.00E+00	0.00E+00	2.51E-11	2.94E-07	6.53E-08	0.00E+00	0.00E+00	4.04E-08	2.87E-08	2.87E-08	2.87E-08
SUM ILCR - chemicals with similar target orga																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																3.48E-11						5.78E-08	5.79E-08	5.79E-08

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day except for in

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	1.78E-04	6.01E-07	1.78E-04	6.01E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.48E-02	1.48E-02	1.56E-03	1.56E-03
Antimony (NC)	1.82E-06	6.13E-09	3.03E-04	1.02E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.85E-04	8.08E-02	5.10E-05	8.50E-03
Arsenic (C)	2.55E-06	8.58E-09	8.48E-03	2.86E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.46E-05	4.88E-02	1.54E-06	5.14E-03
Cadmium (C)	3.64E-07	1.23E-09	7.27E-04	2.45E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.30E-05	1.30E-02	1.36E-06	1.36E-03
Cobalt (C)	2.45E-06	3.31E-09	8.18E-03	1.10E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.59E-05	2.86E-01	9.04E-06	3.01E-02
Iron (NC)	1.18E-03	3.98E-06	1.69E-03	5.69E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.55E-02	3.64E-02	2.68E-03	3.83E-03
Manganese (NC)	2.22E-04	7.48E-07	2.22E-03	7.48E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.76E-04	4.76E-03	5.01E-05	5.01E-04
Nickel (C)	5.82E-06	3.92E-09	5.29E-04	3.57E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.11E-04	2.82E-02	3.27E-05	2.97E-03
Thallium (NC)	3.82E-07	1.29E-09	3.82E-02	1.29E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.27E-05	***3.27E+00***	3.44E-06	3.44E-01
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	4.36E-06	1.47E-08	8.73E-04	2.94E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.57E-05	5.14E-03	2.71E-06	5.41E-04
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			2.40E-03	8.08E-06			0.00E+00	0.00E+00		1.96E-02		2.06E-03
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m³.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.43E-07	4.83E-10	2.58E-07	8.69E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.24E-07	1.48E-06	8.67E-08	1.56E-07
Cadmium (C)	2.05E-08	6.90E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.29E-07	0.00E+00	7.68E-08	0.00E+00
Cobalt (C)	1.38E-07	1.86E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.83E-06	0.00E+00	5.08E-07	0.00E+00
Nickel (C)	3.27E-07	2.21E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.75E-05	0.00E+00	1.84E-06	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m³.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	1.67E-02	1.50E-03	5.05E-06	1.75E-03	1.67E-02	1.50E-03	5.05E-06	1.75E-03	1.71E-01	1.71E-01	1.48E-02	1.27E-04	5.03E-07	1.48E-02	1.27E-04	1.01E-04	8.02E-02	6.98E-03	8.02E-02	6.98E-03	4.63E-09	9.26E-07	3.08E-01	2.95E-01
Antimony (NC)	1.32E-06	7.55E-07	1.59E-08	1.07E-07	2.20E-04	1.26E-04	2.65E-06	1.79E-05	2.79E-04	4.65E-02	2.42E-07	2.08E-08	8.23E-12	4.04E-05	3.47E-06	4.1167E-08	1.04E-06	9.06E-07	1.73E-04	1.51E-04	1.23E-13	6.13E-10	1.28E-01	5.60E-02
Arsenic (C)	4.85E-06	1.26E-06	6.93E-07	9.98E-07	1.62E-02	4.19E-03	2.31E-03	3.33E-03	1.41E-03	***4.70E+00***	3.48E-06	8.98E-08	1.18E-10	1.16E-02	2.99E-04	1.1823E-07	1.64E-05	4.29E-06	5.47E-02	1.43E-02	6.77E-12	6.77E-09	***4.86E+00***	***4.82E+00***
Cadmium (C)	6.13E-06	5.04E-07	8.55E-06	1.63E-07	6.13E-03	5.04E-04	8.55E-03	1.63E-04	5.70E-04	5.70E-01	4.63E-07	3.98E-09	1.57E-11	4.63E-04	3.98E-06	1.573E-06	1.16E-06	1.01E-07	1.16E-03	1.01E-04	1.07E-10	1.07E-05	6.01E-01	5.89E-01
Cobalt (C)	9.12E-06	2.89E-06	4.92E-06	4.78E-05	3.04E-02	9.64E-03	1.64E-02	1.59E-01	5.70E-04	***1.90E+00***	9.96E-06	8.56E-08	3.38E-10	3.32E-02	2.85E-04	5.64E-05	5.20E-05	4.52E-06	1.73E-01	1.51E-02	2.45E-11	4.08E-06	***2.63E+00***	***2.38E+00***
Iron (NC)	7.59E-03	9.43E-04	5.80E-05	2.80E-02	1.08E-02	1.35E-03	8.28E-05	4.00E-02	1.07E-04	1.53E-04	2.14E-02	1.84E-04	7.25E-07	3.05E-02	2.62E-04	NA	1.15E-01	1.00E-02	1.65E-01	1.43E-02	8.65E-09	NA	3.00E-01	2.68E-01
Manganese (NC)	8.90E-03	9.08E-03	8.90E-05	1.55E-04	8.90E-02	9.08E-02	8.90E-04	1.55E-03	9.90E-02	9.90E-01	1.60E-04	1.38E-06	5.45E-09	1.60E-03	1.38E-05	1.09E-04	6.57E-03	5.72E-04	6.57E-02	5.72E-03	1.49E-10	2.97E-06	***1.25E+00***	***1.25E+00***
Nickel (C)	1.04E-04	4.98E-05	3.61E-04	8.86E-05	9.49E-03	4.53E-03	3.29E-02	8.05E-03	9.80E-03	8.91E-01	1.60E-04	1.38E-06	5.43E-09	1.45E-02	1.25E-04	3.02E-04	9.34E-05	8.14E-06	8.49E-03	7.40E-04	1.25E-10	6.96E-06	9.99E-01	9.74E-01
Thallium (NC)	7.27E-07	3.52E-06	2.90E-08	8.10E-06	7.27E-02	3.52E-01	2.90E-03	8.10E-01	3.30E-05	***3.30E+00***	2.50E-07	2.15E-09	8.48E-12	2.50E-02	2.15E-04	NA	5.82E-07	5.07E-08	5.82E-02	5.07E-03	6.01E-14	NA	***7.93E+00***	***5.01E+00***
Titanium (NC)	7.13E-04	2.72E-05	4.83E-06	2.31E-03	NA	NA	NA	NA	4.85E-02	NA	7.31E-04	6.28E-06	2.48E-08	NA	NA	2.48E-04	6.53E-04	5.69E-05	NA	NA	3.49E-10	3.49E-06	2.52E-04	2.52E-04
Vanadium (NC)	1.83E-05	1.76E-06	1.42E-08	6.62E-06	3.65E-03	3.52E-04	2.83E-06	1.32E-03	3.90E-04	7.80E-02	3.12E-05	2.68E-06	1.06E-09	6.24E-03	5.36E-04	1.06E-05	1.24E-04	1.08E-04	2.47E-02	2.15E-02	1.31E-11	1.31E-07	1.42E-01	1.38E-01
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					1.06E-01	9.23E-02	8.95E-04	3.30E-03		***1.16E+00***				1.64E-02	1.41E-04	2.09E-04			1.46E-01	1.27E-02		3.90E-06	***1.56E+00***	***1.54E+00***
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V																6.17E-04						1.47E-05	6.32E-04	6.32E-04

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW =

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air		Total ILCR	Total ILCR
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Arsenic (C)	2.73E-07	7.07E-08	3.90E-08	5.61E-08	4.91E-07	1.27E-07	7.02E-08	1.01E-07	7.93E-05	**1.43E-04**	1.96E-07	5.05E-09	6.65E-12	3.52E-07	9.09E-09	4.26E-11	9.23E-07	2.41E-07	1.66E-06	4.34E-07	3.81E-13	2.44E-12	**1.48E-04**	**1.46E-04**
Cadmium (C)	3.45E-07	2.83E-08	4.81E-07	9.17E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.21E-05	0.00E+00	2.61E-08	2.24E-10	8.85E-13	0.00E+00	0.00E+00	1.59E-12	6.52E-08	5.68E-09	0.00E+00	0.00E+00	5.99E-12	1.08E-11	1.24E-11	1.24E-11
Cobalt (C)	5.13E-07	1.63E-07	2.77E-07	2.69E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.21E-05	0.00E+00	5.60E-07	4.82E-09	1.90E-11	0.00E+00	0.00E+00	3.42E-11	2.92E-06	2.54E-07	0.00E+00	0.00E+00	1.38E-12	2.48E-12	3.67E-11	3.67E-11
Nickel (C)	5.87E-06	2.80E-06	2.03E-05	4.98E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.51E-04	0.00E+00	9.00E-06	7.74E-08	3.06E-10	0.00E+00	0.00E+00	2.17E-10	5.26E-06	4.58E-07	0.00E+00	0.00E+00	7.05E-12	5.01E-12	2.22E-10	2.22E-10
SUM ILCR - chemicals with similar target orga																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																2.95E-10						2.07E-11	3.16E-10	3.16E-10

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day except for in

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	2.00E-04	6.75E-07	2.00E-04	6.75E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.35E-02	2.35E-02	2.48E-03	2.48E-03
Antimony (NC)	1.97E-06	6.65E-09	3.29E-04	1.11E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.72E-03	4.53E-01	2.86E-04	4.76E-02
Arsenic (C)	2.89E-06	9.76E-09	9.65E-03	3.25E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.91E-05	2.97E-01	9.38E-06	3.13E-02
Cadmium (C)	4.71E-07	1.59E-09	9.43E-04	3.18E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.61E-05	1.61E-02	1.69E-06	1.69E-03
Cobalt (C)	2.77E-06	3.73E-09	9.23E-03	1.24E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.63E-04	5.44E-01	1.72E-05	5.73E-02
Iron (NC)	1.34E-03	4.51E-06	1.91E-03	6.45E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.82E-02	5.46E-02	4.02E-03	5.74E-03
Manganese (NC)	2.49E-04	8.39E-07	2.49E-03	8.39E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-03	1.14E-02	1.20E-04	1.20E-03
Nickel (C)	6.62E-06	4.46E-09	6.02E-04	4.06E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.16E-04	7.42E-02	8.59E-05	7.81E-03
Thallium (NC)	4.21E-07	1.42E-09	4.21E-02	1.42E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.13E-04	***1.13E+01***	1.19E-05	***1.19E+00***
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	4.87E-06	1.64E-08	9.75E-04	3.29E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.40E-04	2.81E-02	1.48E-05	2.95E-03
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			2.69E-03	9.06E-06			0.00E+00	0.00E+00		3.49E-02		3.68E-03
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m^3.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.63E-07	5.49E-10	2.93E-07	9.88E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.01E-06	9.02E-06	5.28E-07	9.50E-07
Cadmium (C)	2.65E-08	8.94E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.04E-07	0.00E+00	9.52E-08	0.00E+00
Cobalt (C)	1.56E-07	2.10E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.19E-06	0.00E+00	9.67E-07	0.00E+00
Nickel (C)	3.72E-07	2.51E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.59E-05	0.00E+00	4.83E-06	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m^3.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	2.04E-02	3.15E-03	6.35E-06	2.21E-03	2.04E-02	3.15E-03	6.35E-06	2.21E-03	1.71E-01	1.71E-01	1.53E-02	1.32E-04	5.20E-07	1.53E-02	1.32E-04	1.04E-04	8.02E-02	6.98E-03	8.02E-02	6.98E-03	5.04E-04	1.01E-01	4.24E-01	4.03E-01
Antimony (NC)	1.47E-06	8.32E-07	1.79E-08	1.21E-07	2.44E-04	1.39E-04	2.99E-06	2.02E-05	2.79E-04	4.65E-02	2.61E-07	2.24E-08	8.86E-12	4.35E-05	3.74E-06	4.4312E-08	1.04E-06	9.06E-07	1.73E-04	1.51E-04	1.85E-08	9.26E-05	5.00E-01	9.53E-02
Arsenic (C)	5.22E-06	1.45E-06	7.03E-07	1.10E-06	1.74E-02	4.84E-03	2.34E-03	3.66E-03	1.41E-03	***4.70E+00***	3.53E-06	9.11E-08	1.20E-10	1.18E-02	3.04E-04	1.1998E-07	1.64E-05	4.29E-06	5.47E-02	1.43E-02	5.12E-08	5.12E-05	***5.12E+00***	***4.85E+00***
Cadmium (C)	1.94E-05	1.10E-05	3.47E-05	1.16E-06	1.94E-02	1.10E-02	3.47E-02	1.16E-03	5.70E-04	5.70E-01	1.88E-06	1.62E-08	6.38E-11	1.88E-03	1.62E-05	6.3806E-06	1.16E-06	1.01E-07	1.16E-03	1.01E-04	1.42E-06	1.42E-01	7.99E-01	7.84E-01
Cobalt (C)	1.70E-05	1.44E-05	5.41E-06	7.26E-05	5.66E-02	4.81E-02	1.80E-02	2.42E-01	5.70E-04	***1.90E+00***	1.09E-05	9.41E-08	3.72E-10	3.65E-02	3.14E-04	6.19E-05	5.20E-05	4.52E-06	1.73E-01	1.51E-02	9.85E-07	1.64E-01	***3.21E+00***	***2.72E+00***
Iron (NC)	1.37E-02	3.97E-03	8.90E-05	3.92E-02	1.96E-02	5.67E-03	1.27E-04	5.60E-02	1.07E-04	1.53E-04	2.23E-02	1.92E-04	7.57E-07	3.19E-02	2.74E-04	NA	1.15E-01	1.00E-02	1.65E-01	1.43E-02	9.45E-04	NA	3.49E-01	3.00E-01
Manganese (NC)	1.05E-02	9.91E-03	9.67E-05	1.94E-04	1.05E-01	9.91E-02	9.67E-04	1.94E-03	9.90E-02	9.90E-01	1.74E-04	1.50E-06	5.91E-09	1.74E-03	1.50E-05	1.18E-04	6.57E-03	5.72E-04	6.57E-02	5.72E-03	1.37E-05	2.75E-01	***1.56E+00***	***1.55E+00***
Nickel (C)	2.44E-04	1.16E-04	3.76E-04	1.39E-04	2.22E-02	1.05E-02	3.42E-02	1.26E-02	9.80E-03	8.91E-01	1.67E-04	1.43E-06	5.66E-09	1.51E-02	1.30E-04	3.14E-04	9.34E-05	8.14E-06	8.49E-03	7.40E-04	6.46E-06	3.59E-01	***1.43E+00***	***1.36E+00***
Thallium (NC)	8.15E-07	3.68E-06	3.07E-08	9.05E-06	8.15E-02	3.68E-01	3.07E-03	9.05E-01	3.30E-05	***3.30E+00***	2.58E-07	2.22E-09	8.77E-12	2.58E-02	2.22E-04	NA	5.82E-07	5.07E-08	5.82E-02	5.07E-03	8.47E-09	NA	***1.61E+01***	***5.98E+00***
Titanium (NC)	1.02E-03	1.81E-04	7.22E-06	3.25E-03	NA	NA	NA	NA	4.85E-02	NA	7.79E-04	6.70E-06	2.65E-08	NA	NA	2.65E-04	6.53E-04	5.69E-05	NA	NA	4.85E-05	4.85E-01	4.85E-01	4.85E-01
Vanadium (NC)	2.90E-05	7.30E-06	2.17E-08	9.27E-06	5.80E-03	1.46E-03	4.33E-06	1.85E-03	3.90E-04	7.80E-02	3.28E-05	2.82E-06	1.11E-09	6.55E-03	5.64E-04	1.11E-05	1.24E-04	1.08E-04	2.47E-02	2.15E-02	1.59E-06	1.59E-02	1.85E-01	1.60E-01
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					1.25E-01	1.02E-01	9.73E-04	4.15E-03		***1.16E+00***				1.70E-02	1.47E-04	2.22E-04			1.46E-01	1.27E-02		3.76E-01	***1.98E+00***	***1.95E+00***
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V																6.52E-04						***1.02E+00***	***1.02E+00***	***1.02E+00***

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW =

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation	Total ILCR	Total ILCR
Metals																								
Arsenic (C)	2.94E-07	8.17E-08	3.96E-08	6.18E-08	5.28E-07	1.47E-07	7.12E-08	1.11E-07	7.93E-05	**1.43E-04**	1.99E-07	5.13E-09	6.75E-12	3.58E-07	9.23E-09	4.32E-11	9.23E-07	2.41E-07	1.66E-06	4.34E-07	2.88E-09	1.84E-08	**1.55E-04**	**1.47E-04**
Cadmium (C)	1.09E-06	6.19E-07	1.95E-06	6.54E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.21E-05	0.00E+00	1.06E-07	9.09E-10	3.59E-12	0.00E+00	0.00E+00	6.46E-12	6.52E-08	5.68E-09	0.00E+00	0.00E+00	8.01E-08	1.44E-07	1.44E-07	1.44E-07
Cobalt (C)	9.55E-07	8.12E-07	3.04E-07	4.08E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.21E-05	0.00E+00	6.16E-07	5.29E-09	2.09E-11	0.00E+00	0.00E+00	3.76E-11	2.92E-06	2.54E-07	0.00E+00	0.00E+00	5.54E-08	9.97E-08	9.98E-08	9.98E-08
Nickel (C)	1.37E-05	6.51E-06	2.12E-05	7.82E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.51E-04	0.00E+00	9.37E-06	8.05E-08	3.18E-10	0.00E+00	0.00E+00	2.26E-10	5.26E-06	4.58E-07	0.00E+00	0.00E+00	3.64E-07	2.58E-07	2.58E-07	2.58E-07
SUM ILCR - chemicals with similar target orga																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																3.13E-10						5.21E-07	5.21E-07	5.21E-07

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day except for in

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	1.19E-04	4.98E-07	1.19E-04	4.98E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.33E-02	1.33E-02	1.09E-03	1.09E-03
Antimony (NC)	1.22E-06	5.09E-09	2.03E-04	8.48E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.35E-04	7.25E-02	3.58E-05	5.97E-03
Arsenic (C)	1.70E-06	7.12E-09	5.67E-03	2.37E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-05	4.38E-02	1.08E-06	3.61E-03
Cadmium (C)	2.43E-07	1.02E-09	4.86E-04	2.03E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.16E-05	1.16E-02	9.58E-07	9.58E-04
Cobalt (C)	1.64E-06	2.75E-09	5.47E-03	9.15E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.71E-05	2.57E-01	6.35E-06	2.12E-02
Iron (NC)	7.90E-04	3.31E-06	1.13E-03	4.72E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.29E-02	3.27E-02	1.88E-03	2.69E-03
Manganese (NC)	1.48E-04	6.20E-07	1.48E-03	6.20E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.27E-04	4.27E-03	3.52E-05	3.52E-04
Nickel (C)	3.89E-06	3.25E-09	3.54E-04	2.96E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.79E-04	2.53E-02	2.30E-05	2.09E-03
Thallium (NC)	2.55E-07	1.07E-09	2.55E-02	1.07E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.93E-05	***2.93E+00***	2.42E-06	2.42E-01
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	2.92E-06	1.22E-08	5.84E-04	2.44E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.31E-05	4.61E-03	1.90E-06	3.80E-04
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			1.60E-03	6.70E-06			0.00E+00	0.00E+00		1.76E-02		1.45E-03
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/㎥.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.49E-07	6.23E-10	2.68E-07	1.12E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.15E-06	2.07E-06	9.47E-08	1.70E-07
Cadmium (C)	2.13E-08	8.90E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.02E-06	0.00E+00	8.38E-08	0.00E+00
Cobalt (C)	1.44E-07	2.40E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.74E-06	0.00E+00	5.55E-07	0.00E+00
Nickel (C)	3.40E-07	2.85E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.44E-05	0.00E+00	2.01E-06	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/㎥.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	8.38E-03	7.51E-04	3.72E-06	1.29E-03	8.38E-03	7.51E-04	3.72E-06	1.29E-03	2.03E-01	2.03E-01	1.86E-03	9.69E-05	5.03E-07	1.86E-03	9.69E-05	1.01E-04	1.01E-02	5.62E-03	1.01E-02	5.62E-03	4.63E-09	9.26E-07	2.45E-01	2.32E-01
Antimony (NC)	6.61E-07	3.78E-07	1.17E-08	7.91E-08	1.10E-04	6.31E-05	1.96E-06	1.32E-05	1.40E-04	2.33E-02	3.04E-08	1.59E-08	8.23E-12	5.07E-06	2.65E-06	4.1167E-08	1.30E-07	7.29E-07	2.17E-05	1.21E-04	1.23E-13	6.13E-10	9.63E-02	2.98E-02
Arsenic (C)	2.43E-06	6.31E-07	5.11E-07	7.36E-07	8.11E-03	2.10E-03	1.70E-03	2.45E-03	1.77E-03	***5.90E+00***	4.36E-07	6.84E-08	1.18E-10	1.45E-03	2.28E-04	1.1823E-07	2.06E-06	3.45E-06	6.86E-03	1.15E-02	6.77E-12	6.77E-09	***5.98E+00***	***5.94E+00***
Cadmium (C)	3.08E-06	2.53E-07	6.30E-06	1.20E-07	3.08E-03	2.53E-04	6.30E-03	1.20E-04	4.80E-04	4.80E-01	5.81E-08	3.03E-09	1.57E-11	5.81E-05	3.03E-06	1.573E-06	1.45E-07	8.12E-08	1.45E-04	8.12E-05	1.07E-10	1.07E-05	5.02E-01	4.91E-01
Cobalt (C)	4.57E-06	1.45E-06	3.63E-06	3.53E-05	1.52E-02	4.84E-03	1.21E-02	1.18E-01	4.10E-04	***1.37E+00***	1.25E-06	6.52E-08	3.38E-10	4.16E-03	2.17E-04	5.64E-05	6.51E-06	3.64E-06	2.17E-02	1.21E-02	2.45E-11	4.08E-06	***1.82E+00***	***1.58E+00***
Iron (NC)	3.81E-03	4.73E-04	4.28E-05	2.06E-02	5.44E-03	6.76E-04	6.11E-05	2.95E-02	5.35E-04	7.64E-04	2.68E-03	1.40E-04	7.25E-07	3.83E-03	2.00E-04	NA	1.44E-02	8.07E-03	2.06E-02	1.15E-02	8.65E-09	NA	1.06E-01	7.64E-02
Manganese (NC)	4.46E-03	4.55E-03	6.57E-05	1.15E-04	4.46E-02	4.55E-02	6.57E-04	1.15E-03	8.30E-02	8.30E-01	2.01E-05	1.05E-06	5.45E-09	2.01E-04	1.05E-05	1.09E-04	8.24E-04	4.61E-04	8.24E-03	4.61E-03	1.49E-10	2.97E-06	9.41E-01	9.37E-01
Nickel (C)	5.23E-05	2.50E-05	2.67E-04	6.53E-05	4.76E-03	2.27E-03	2.42E-02	5.94E-03	7.50E-03	6.82E-01	2.01E-05	1.05E-06	5.43E-09	1.82E-03	9.53E-05	3.02E-04	1.17E-05	6.54E-06	1.06E-03	5.95E-04	1.25E-10	6.96E-06	7.49E-01	7.25E-01
Thallium (NC)	3.65E-07	1.77E-06	2.14E-08	5.97E-06	3.65E-02	1.77E-01	2.14E-03	5.97E-01	2.10E-05	***2.10E+00***	3.13E-08	1.64E-09	8.48E-12	3.13E-03	1.64E-04	NA	7.29E-08	4.08E-08	7.29E-03	4.08E-03	6.01E-14	NA	***5.89E+00***	***3.19E+00***
Titanium (NC)	3.57E-04	1.36E-05	3.57E-06	1.70E-03	NA	NA	NA	NA	2.43E-02	NA	9.16E-05	4.79E-06	2.48E-08	NA	NA	2.48E-04	8.19E-05	4.58E-05	NA	NA	3.49E-10	3.49E-06	2.52E-04	2.52E-04
Vanadium (NC)	9.16E-06	8.83E-07	1.04E-08	4.88E-06	1.83E-03	1.77E-04	2.09E-06	9.76E-04	2.70E-04	5.40E-02	3.91E-06	2.04E-06	1.06E-09	7.82E-04	4.08E-04	1.06E-05	1.55E-05	8.66E-05	3.10E-03	1.73E-02	1.31E-11	1.31E-07	8.38E-02	7.96E-02
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					5.30E-02	4.63E-02	6.60E-04	2.43E-03		***1.03E+00***				2.06E-03	1.07E-04	2.09E-04			1.83E-02	1.02E-02		3.90E-06	***1.19E+00***	***1.17E+00***
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V																6.17E-04						1.47E-05	6.32E-04	6.32E-04

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW =

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation	Total ILCR	Total ILCR
Metals																								
Arsenic (C)	2.13E-07	5.52E-08	4.47E-08	6.44E-08	3.83E-07	9.93E-08	8.05E-08	1.16E-07	1.55E-04	**2.79E-04**	3.82E-08	5.99E-09	1.03E-11	6.87E-08	1.08E-08	6.62E-11	1.80E-07	3.02E-07	3.24E-07	5.43E-07	5.92E-13	3.79E-12	**2.83E-04**	**2.81E-04**
Cadmium (C)	2.69E-07	2.21E-08	5.52E-07	1.05E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.20E-05	0.00E+00	5.08E-09	2.65E-10	1.38E-12	0.00E+00	0.00E+00	2.48E-12	1.27E-08	7.10E-09	0.00E+00	0.00E+00	9.32E-12	1.68E-11	1.93E-11	1.93E-11
Cobalt (C)	4.00E-07	1.27E-07	3.17E-07	3.09E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.59E-05	0.00E+00	1.09E-07	5.71E-09	2.96E-11	0.00E+00	0.00E+00	5.33E-11	5.70E-07	3.18E-07	0.00E+00	0.00E+00	2.14E-12	3.86E-12	5.71E-11	5.71E-11
Nickel (C)	4.58E-06	2.19E-06	2.33E-05	5.72E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.56E-04	0.00E+00	1.76E-06	9.17E-08	4.76E-10	0.00E+00	0.00E+00	3.38E-10	1.03E-06	5.73E-07	0.00E+00	0.00E+00	1.10E-11	7.79E-12	3.45E-10	3.45E-10
SUM ILCR - chemicals with similar target orga																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																4.60E-10						3.22E-11	4.92E-10	4.92E-10

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day except for in

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	1.34E-04	5.60E-07	1.34E-04	5.60E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.11E-02	2.11E-02	1.74E-03	1.74E-03
Antimony (NC)	1.32E-06	5.52E-09	2.20E-04	9.20E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.44E-03	4.06E-01	2.01E-04	3.34E-02
Arsenic (C)	1.93E-06	8.09E-09	6.45E-03	2.70E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.00E-05	2.67E-01	6.59E-06	2.20E-02
Cadmium (C)	3.15E-07	1.32E-09	6.30E-04	2.64E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.44E-05	1.44E-02	1.19E-06	1.19E-03
Cobalt (C)	1.85E-06	3.10E-09	6.17E-03	1.03E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.47E-04	4.89E-01	1.21E-05	4.02E-02
Iron (NC)	8.95E-04	3.74E-06	1.28E-03	5.35E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.43E-02	4.90E-02	2.82E-03	4.03E-03
Manganese (NC)	1.66E-04	6.96E-07	1.66E-03	6.96E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.02E-03	1.02E-02	8.41E-05	8.41E-04
Nickel (C)	4.43E-06	3.70E-09	4.02E-04	3.37E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.32E-04	6.66E-02	6.03E-05	5.48E-03
Thallium (NC)	2.82E-07	1.18E-09	2.82E-02	1.18E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.02E-04	***1.02E+01***	8.37E-06	8.37E-01
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	3.26E-06	1.36E-08	6.52E-04	2.73E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.26E-04	2.52E-02	1.04E-05	2.07E-03
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			1.80E-03	7.52E-06			0.00E+00	0.00E+00		3.13E-02		2.58E-03
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m ^{3}.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.69E-07	7.08E-10	3.05E-07	1.27E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.00E-06	**1.26E-05**	5.76E-07	1.04E-06
Cadmium (C)	2.76E-08	1.15E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.26E-06	0.00E+00	1.04E-07	0.00E+00
Cobalt (C)	1.62E-07	2.71E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.28E-05	0.00E+00	1.06E-06	0.00E+00
Nickel (C)	3.87E-07	3.24E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.41E-05	0.00E+00	5.28E-06	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m ^{3}.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	1.03E-02	1.58E-03	4.68E-06	1.63E-03	1.03E-02	1.58E-03	4.68E-06	1.63E-03	2.03E-01	2.03E-01	1.92E-03	1.00E-04	5.20E-07	1.92E-03	1.00E-04	1.04E-04	1.01E-02	5.62E-03	1.01E-02	5.62E-03	5.04E-04	1.01E-01	3.56E-01	3.37E-01
Antimony (NC)	7.35E-07	4.17E-07	1.32E-08	8.94E-08	1.22E-04	6.95E-05	2.20E-06	1.49E-05	1.40E-04	2.33E-02	3.27E-08	1.71E-08	8.86E-12	5.45E-06	2.85E-06	4.4312E-08	1.30E-07	7.29E-07	2.17E-05	1.21E-04	1.85E-08	9.26E-05	4.30E-01	5.74E-02
Arsenic (C)	2.62E-06	7.28E-07	5.19E-07	8.11E-07	8.72E-03	2.43E-03	1.73E-03	2.70E-03	1.77E-03	***5.90E+00***	4.43E-07	6.94E-08	1.20E-10	1.48E-03	2.31E-04	1.1998E-07	2.06E-06	3.45E-06	6.86E-03	1.15E-02	5.12E-08	5.12E-05	***6.21E+00***	***5.96E+00***
Cadmium (C)	9.71E-06	5.52E-06	2.56E-05	8.57E-07	9.71E-03	5.52E-03	2.56E-02	8.57E-04	4.80E-04	4.80E-01	2.36E-07	1.23E-08	6.38E-11	2.36E-04	1.23E-05	6.3806E-06	1.45E-07	8.12E-08	1.45E-04	8.12E-05	1.42E-06	1.42E-01	6.80E-01	6.66E-01
Cobalt (C)	8.52E-06	7.24E-06	3.99E-06	5.35E-05	2.84E-02	2.41E-02	1.33E-02	1.78E-01	4.10E-04	***1.37E+00***	1.37E-06	7.17E-08	3.72E-10	4.57E-03	2.39E-04	6.19E-05	6.51E-06	3.64E-06	2.17E-02	1.21E-02	9.85E-07	1.64E-01	***2.31E+00***	***1.86E+00***
Iron (NC)	6.88E-03	1.99E-03	6.56E-05	2.89E-02	9.83E-03	2.84E-03	9.37E-05	4.13E-02	5.35E-04	7.64E-04	2.80E-03	1.46E-04	7.57E-07	3.99E-03	2.09E-04	NA	1.44E-02	8.07E-03	2.06E-02	1.15E-02	9.45E-04	NA	1.41E-01	9.65E-02
Manganese (NC)	5.26E-03	4.97E-03	7.13E-05	1.43E-04	5.26E-02	4.97E-02	7.13E-04	1.43E-03	8.30E-02	8.30E-01	2.18E-05	1.14E-06	5.91E-09	2.18E-04	1.14E-05	1.18E-04	8.24E-04	4.61E-04	8.24E-03	4.61E-03	1.37E-05	2.75E-01	***1.23E+00***	***1.23E+00***
Nickel (C)	1.22E-04	5.81E-05	2.77E-04	1.02E-04	1.11E-02	5.28E-03	2.52E-02	9.32E-03	7.50E-03	6.82E-01	2.09E-05	1.09E-06	5.66E-09	1.90E-03	9.92E-05	3.14E-04	1.17E-05	6.54E-06	1.06E-03	5.95E-04	6.46E-06	3.59E-01	***1.16E+00***	***1.10E+00***
Thallium (NC)	4.09E-07	1.85E-06	2.26E-08	6.68E-06	4.09E-02	1.85E-01	2.26E-03	6.68E-01	2.10E-05	***2.10E+00***	3.24E-08	1.69E-09	8.77E-12	3.24E-03	1.69E-04	NA	7.29E-08	4.08E-08	7.29E-03	4.08E-03	8.47E-09	NA	***1.32E+01***	***3.88E+00***
Titanium (NC)	5.13E-04	9.10E-05	5.33E-06	2.40E-03	NA	NA	NA	NA	2.43E-02	NA	9.77E-05	5.10E-06	2.65E-08	NA	NA	2.65E-04	8.19E-05	4.58E-05	NA	NA	4.85E-05	4.85E-01	4.85E-01	4.85E-01
Vanadium (NC)	1.46E-05	3.66E-06	1.60E-08	6.84E-06	2.91E-03	7.32E-04	3.19E-06	1.37E-03	2.70E-04	5.40E-02	4.11E-06	2.15E-06	1.11E-09	8.22E-04	4.29E-04	1.11E-05	1.55E-05	8.66E-05	3.10E-03	1.73E-02	1.59E-06	1.59E-02	1.22E-01	9.93E-02
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					6.28E-02	5.13E-02	7.18E-04	3.06E-03		***1.03E+00***				2.14E-03	1.12E-04	2.22E-04			1.83E-02	1.02E-02		3.76E-01	***1.59E+00***	***1.56E+00***
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)																6.52E-04						***1.02E+00***	***1.02E+00***	***1.02E+00***

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcino

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR	Total ILCR	Total ILCR
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Arsenic (C)	2.29E-07	6.37E-08	4.54E-08	7.09E-08	4.12E-07	1.15E-07	8.17E-08	1.28E-07	1.55E-04	**2.79E-04**	3.88E-08	6.07E-09	1.05E-11	6.98E-08	1.09E-08	6.72E-11	1.80E-07	3.02E-07	3.24E-07	5.43E-07	4.48E-09	2.87E-08	**2.93E-04**	**2.82E-04**
Cadmium (C)	8.49E-07	4.83E-07	2.24E-06	7.50E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.20E-05	0.00E+00	2.06E-08	1.08E-09	5.58E-12	0.00E+00	0.00E+00	1.00E-11	1.27E-08	7.10E-09	0.00E+00	0.00E+00	1.25E-07	2.24E-07	2.24E-07	2.24E-07
Cobalt (C)	7.45E-07	6.34E-07	3.49E-07	4.68E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.59E-05	0.00E+00	1.20E-07	6.27E-09	3.25E-11	0.00E+00	0.00E+00	5.85E-11	5.70E-07	3.18E-07	0.00E+00	0.00E+00	8.62E-08	1.55E-07	1.55E-07	1.55E-07
Nickel (C)	1.07E-05	5.08E-06	2.43E-05	8.97E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.56E-04	0.00E+00	1.83E-06	9.54E-08	4.95E-10	0.00E+00	0.00E+00	3.51E-10	1.03E-06	5.73E-07	0.00E+00	0.00E+00	5.66E-07	4.02E-07	4.02E-07	4.02E-07
SUM ILCR - chemicals with similar target organ																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																4.87E-10						8.10E-07	8.10E-07	8.10E-07

Notes:
1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcino

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day except for ir

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	8.21E-05	3.17E-07	8.21E-05	3.17E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.61E-03	8.61E-03	9.48E-04	9.48E-04
Antimony (NC)	8.38E-07	3.24E-09	1.40E-04	5.40E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.82E-04	4.70E-02	3.10E-05	5.17E-03
Arsenic (C)	1.17E-06	4.53E-09	3.91E-03	1.51E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.52E-06	2.84E-02	9.37E-07	3.12E-03
Cadmium (C)	1.68E-07	6.48E-10	3.35E-04	1.30E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.54E-06	7.54E-03	8.30E-07	8.30E-04
Cobalt (C)	1.13E-06	1.75E-09	3.77E-03	5.83E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.00E-05	1.67E-01	5.50E-06	1.83E-02
Iron (NC)	5.44E-04	2.11E-06	7.78E-04	3.01E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.48E-02	2.12E-02	1.63E-03	2.33E-03
Manganese (NC)	1.02E-04	3.95E-07	1.02E-03	3.95E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.77E-04	2.77E-03	3.05E-05	3.05E-04
Nickel (C)	2.68E-06	2.07E-09	2.44E-04	1.88E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.81E-04	1.64E-02	1.99E-05	1.81E-03
Thallium (NC)	1.76E-07	6.80E-10	1.76E-02	6.80E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.90E-05	***1.90E+00***	2.09E-06	2.09E-01
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	2.01E-06	7.77E-09	4.02E-04	1.55E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.50E-05	2.99E-03	1.65E-06	3.29E-04
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			1.10E-03	4.27E-06			0.00E+00	0.00E+00		1.14E-02		1.25E-03
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m ^{3}.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.17E-07	4.53E-10	2.11E-07	8.16E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.52E-07	1.53E-06	9.37E-08	1.69E-07
Cadmium (C)	1.68E-08	6.48E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.54E-07	0.00E+00	8.30E-08	0.00E+00
Cobalt (C)	1.13E-07	1.75E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.00E-06	0.00E+00	5.50E-07	0.00E+00
Nickel (C)	2.68E-07	2.07E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.81E-05	0.00E+00	1.99E-06	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m ^{3}.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	4.62E-03	1.24E-03	2.87E-06	9.94E-04	4.62E-03	1.24E-03	2.87E-06	9.94E-04	1.77E-01	1.77E-01	1.02E-03	7.77E-05	5.03E-07	1.02E-03	7.77E-05	1.01E-04	5.54E-03	4.66E-03	5.54E-03	4.66E-03	4.63E-09	9.26E-07	2.04E-01	1.96E-01
Antimony (NC)	3.64E-07	6.26E-07	9.06E-09	6.10E-08	6.07E-05	1.04E-04	1.51E-06	1.02E-05	7.71E-05	1.28E-02	1.68E-08	1.27E-08	8.23E-12	2.79E-06	2.12E-06	4.1167E-08	7.19E-08	6.04E-07	1.20E-05	1.01E-04	1.23E-13	6.13E-10	6.03E-02	1.84E-02
Arsenic (C)	1.34E-06	1.04E-06	3.94E-07	5.68E-07	4.47E-03	3.48E-03	1.31E-03	1.89E-03	1.22E-03	***4.07E+00***	2.41E-07	5.48E-08	1.18E-10	8.02E-04	1.83E-04	1.1823E-07	1.13E-06	2.86E-06	3.78E-03	9.53E-03	6.77E-12	6.77E-09	***4.12E+00***	***4.10E+00***
Cadmium (C)	1.70E-06	4.18E-07	4.86E-06	9.28E-08	1.70E-03	4.18E-04	4.86E-03	9.28E-05	3.20E-04	3.20E-01	3.20E-08	2.43E-09	1.57E-11	3.20E-05	2.43E-06	1.573E-06	8.01E-08	6.73E-08	8.01E-05	6.73E-05	1.07E-10	1.07E-05	3.35E-01	3.28E-01
Cobalt (C)	2.52E-06	2.40E-06	2.80E-06	2.72E-05	8.40E-03	7.99E-03	9.33E-03	9.07E-02	2.90E-04	9.67E-01	6.88E-07	5.23E-08	3.38E-10	2.29E-03	1.74E-04	5.64E-05	3.59E-06	3.02E-06	1.20E-02	1.01E-02	2.45E-11	4.08E-06	***1.28E+00***	***1.13E+00***
Iron (NC)	2.10E-03	7.82E-04	3.30E-05	1.59E-02	3.00E-03	1.12E-03	4.71E-05	2.27E-02	2.95E-04	4.21E-04	1.48E-03	1.12E-04	7.25E-07	2.11E-03	1.60E-04	NA	7.96E-03	6.69E-03	1.14E-02	9.55E-03	8.65E-09	NA	7.25E-02	5.36E-02
Manganese (NC)	2.46E-03	7.53E-03	5.07E-05	8.83E-05	2.46E-02	7.53E-02	5.07E-04	8.83E-04	5.60E-02	5.60E-01	1.11E-05	8.42E-07	5.45E-09	1.11E-04	8.42E-06	1.09E-04	4.54E-04	3.82E-04	4.54E-03	3.82E-03	1.49E-10	2.97E-06	6.74E-01	6.71E-01
Nickel (C)	2.88E-05	4.13E-05	2.06E-04	5.04E-05	2.62E-03	3.75E-03	1.87E-02	4.58E-03	6.20E-03	5.64E-01	1.11E-05	8.40E-07	5.43E-09	1.01E-03	7.64E-05	3.02E-04	6.46E-06	5.42E-06	5.87E-04	4.93E-04	1.25E-10	6.96E-06	6.12E-01	5.98E-01
Thallium (NC)	2.01E-07	2.92E-06	1.65E-08	4.61E-06	2.01E-02	2.92E-01	1.65E-03	4.61E-01	1.50E-05	***1.50E+00***	1.73E-08	1.31E-09	8.48E-12	1.73E-03	1.31E-04	NA	4.02E-08	3.38E-08	4.02E-03	3.38E-03	6.01E-14	NA	***4.20E+00***	***2.51E+00***
Titanium (NC)	1.97E-04	2.25E-05	2.75E-06	1.31E-03	NA	NA	NA	NA	1.34E-02	NA	5.05E-05	3.84E-06	2.48E-08	NA	NA	2.48E-04	4.51E-05	3.79E-05	NA	NA	3.49E-10	3.49E-06	2.52E-04	2.52E-04
Vanadium (NC)	5.05E-06	1.46E-06	8.06E-09	3.77E-06	1.01E-03	2.92E-04	1.61E-06	7.53E-04	1.80E-04	3.60E-02	2.15E-06	1.64E-06	1.06E-09	4.31E-04	3.27E-04	1.06E-05	8.54E-06	7.17E-05	1.71E-03	1.43E-02	1.31E-11	1.31E-07	5.83E-02	5.56E-02
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					2.92E-02	7.65E-02	5.09E-04	1.88E-03		7.37E-01				1.13E-03	8.61E-05	2.09E-04			1.01E-02	8.47E-03		3.90E-06	8.78E-01	8.67E-01
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)																6.17E-04						1.47E-05	6.32E-04	6.32E-04

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcino

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation	Total ILCR	Total ILCR
Metals																								
Arsenic (C)	1.34E-07	1.04E-07	3.94E-08	5.68E-08	2.41E-07	1.88E-07	7.10E-08	1.02E-07	1.22E-04	**2.20E-04**	2.41E-08	5.48E-09	1.18E-11	4.33E-08	9.87E-09	7.57E-11	1.13E-07	2.86E-07	2.04E-07	5.15E-07	6.77E-13	4.33E-12	**2.23E-04**	**2.21E-04**
Cadmium (C)	1.70E-07	4.18E-08	4.86E-07	9.28E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.20E-05	0.00E+00	3.20E-09	2.43E-10	1.57E-12	0.00E+00	0.00E+00	2.83E-12	8.01E-09	6.73E-09	0.00E+00	0.00E+00	1.07E-11	1.92E-11	2.20E-11	2.20E-11
Cobalt (C)	2.52E-07	2.40E-07	2.80E-07	2.72E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.90E-05	0.00E+00	6.88E-08	5.23E-09	3.38E-11	0.00E+00	0.00E+00	6.09E-11	3.59E-07	3.02E-07	0.00E+00	0.00E+00	2.45E-12	4.41E-12	6.53E-11	6.53E-11
Nickel (C)	2.88E-06	4.13E-06	2.06E-05	5.04E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.20E-04	0.00E+00	1.11E-06	8.40E-08	5.43E-10	0.00E+00	0.00E+00	3.86E-10	6.46E-07	5.42E-07	0.00E+00	0.00E+00	1.25E-11	8.90E-12	3.95E-10	3.95E-10
SUM ILCR - chemicals with similar target organ																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																5.25E-10						3.68E-11	5.62E-10	5.62E-10

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcino

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day except for i

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	9.22E-05	3.57E-07	9.22E-05	3.57E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.37E-02	1.37E-02	1.51E-03	1.51E-03
Antimony (NC)	9.09E-07	3.52E-09	1.52E-04	5.86E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.58E-03	2.63E-01	1.74E-04	2.90E-02
Arsenic (C)	1.33E-06	5.16E-09	4.44E-03	1.72E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.18E-05	1.73E-01	5.70E-06	1.90E-02
Cadmium (C)	2.17E-07	8.40E-10	4.34E-04	1.68E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.35E-06	9.35E-03	1.03E-06	1.03E-03
Cobalt (C)	1.28E-06	1.97E-09	4.25E-03	6.58E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.50E-05	3.17E-01	1.05E-05	3.48E-02
Iron (NC)	6.17E-04	2.39E-06	8.81E-04	3.41E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.22E-02	3.18E-02	2.45E-03	3.49E-03
Manganese (NC)	1.15E-04	4.43E-07	1.15E-03	4.43E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.62E-04	6.62E-03	7.28E-05	7.28E-04
Nickel (C)	3.05E-06	2.36E-09	2.77E-04	2.14E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.75E-04	4.32E-02	5.22E-05	4.75E-03
Thallium (NC)	1.94E-07	7.51E-10	1.94E-02	7.51E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.59E-05	***6.59E+00***	7.25E-06	7.25E-01
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	2.25E-06	8.68E-09	4.49E-04	1.74E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.16E-05	1.63E-02	8.98E-06	1.80E-03
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			1.24E-03	4.79E-06			0.00E+00	0.00E+00		2.03E-02		2.24E-03
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m³.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.33E-07	5.16E-10	2.40E-07	9.28E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.18E-06	9.33E-06	5.70E-07	1.03E-06
Cadmium (C)	2.17E-08	8.40E-11	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.35E-07	0.00E+00	1.03E-07	0.00E+00
Cobalt (C)	1.28E-07	1.97E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.50E-06	0.00E+00	1.05E-06	0.00E+00
Nickel (C)	3.05E-07	2.36E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.75E-05	0.00E+00	5.22E-06	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m³.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	5.65E-03	2.61E-03	3.61E-06	1.26E-03	5.65E-03	2.61E-03	3.61E-06	1.26E-03	1.77E-01	1.77E-01	1.06E-03	8.04E-05	5.20E-07	1.06E-03	8.04E-05	1.04E-04	5.54E-03	4.66E-03	5.54E-03	4.66E-03	5.04E-04	1.01E-01	3.13E-01	3.00E-01
Antimony (NC)	4.05E-07	6.90E-07	1.02E-08	6.90E-08	6.75E-05	1.15E-04	1.70E-06	1.15E-05	7.71E-05	1.28E-02	1.80E-08	1.37E-08	8.86E-12	3.01E-06	2.28E-06	4.4312E-08	7.19E-08	6.04E-07	1.20E-05	1.01E-04	1.85E-08	9.26E-05	2.77E-01	4.24E-02
Arsenic (C)	1.44E-06	1.20E-06	4.00E-07	6.26E-07	4.81E-03	4.01E-03	1.33E-03	2.09E-03	1.22E-03	***4.07E+00***	2.44E-07	5.57E-08	1.20E-10	8.14E-04	1.86E-04	1.1998E-07	1.13E-06	2.86E-06	3.78E-03	9.53E-03	5.12E-08	5.12E-05	***4.27E+00***	***4.12E+00***
Cadmium (C)	5.35E-06	9.13E-06	1.97E-05	6.61E-07	5.35E-03	9.13E-03	1.97E-02	6.61E-04	3.20E-04	3.20E-01	1.30E-07	9.87E-09	6.38E-11	1.30E-04	9.87E-06	6.3806E-06	8.01E-08	6.73E-08	8.01E-05	6.73E-05	1.42E-06	1.42E-01	5.07E-01	4.99E-01
Cobalt (C)	4.69E-06	1.20E-05	3.08E-06	4.13E-05	1.56E-02	3.99E-02	1.03E-02	1.38E-01	2.90E-04	9.67E-01	7.56E-07	5.75E-08	3.72E-10	2.52E-03	1.92E-04	6.19E-05	3.59E-06	3.02E-06	1.20E-02	1.01E-02	9.85E-07	1.64E-01	***1.68E+00***	***1.40E+00***
Iron (NC)	3.79E-03	3.29E-03	5.06E-05	2.23E-02	5.42E-03	4.70E-03	7.23E-05	3.19E-02	2.95E-04	4.21E-04	1.54E-03	1.17E-04	7.57E-07	2.20E-03	1.67E-04	NA	7.96E-03	6.69E-03	1.14E-02	9.55E-03	9.45E-04	NA	9.84E-02	7.02E-02
Manganese (NC)	2.90E-03	8.22E-03	5.50E-05	1.10E-04	2.90E-02	8.22E-02	5.50E-04	1.10E-03	5.60E-02	5.60E-01	1.20E-05	9.14E-07	5.91E-09	1.20E-04	9.14E-06	1.18E-04	4.54E-04	3.82E-04	4.54E-03	3.82E-03	1.37E-05	2.75E-01	9.64E-01	9.58E-01
Nickel (C)	6.74E-05	9.60E-05	2.14E-04	7.91E-05	6.13E-03	8.73E-03	1.95E-02	7.19E-03	6.20E-03	5.64E-01	1.15E-05	8.74E-07	5.66E-09	1.05E-03	7.95E-05	3.14E-04	6.46E-06	5.42E-06	5.87E-04	4.93E-04	6.46E-06	3.59E-01	***1.01E+00***	9.72E-01
Thallium (NC)	2.25E-07	3.06E-06	1.75E-08	5.15E-06	2.25E-02	3.06E-01	1.75E-03	5.15E-01	1.50E-05	***1.50E+00***	1.78E-08	1.36E-09	8.77E-12	1.78E-03	1.36E-04	NA	4.02E-08	3.38E-08	4.02E-03	3.38E-03	8.47E-09	NA	***8.96E+00***	***3.10E+00***
Titanium (NC)	2.83E-04	1.50E-04	4.11E-06	1.85E-03	NA	NA	NA	NA	1.34E-02	NA	5.38E-05	4.09E-06	2.65E-08	NA	NA	2.65E-04	4.51E-05	3.79E-05	NA	NA	4.85E-05	4.85E-01	4.85E-01	4.85E-01
Vanadium (NC)	8.02E-06	1.46E-06	1.23E-08	3.77E-06	1.60E-03	2.92E-04	2.46E-06	7.53E-04	1.80E-04	3.60E-02	2.26E-06	1.72E-06	1.11E-09	4.53E-04	3.44E-04	1.11E-05	8.54E-06	7.17E-05	1.71E-03	1.43E-02	1.59E-06	1.59E-02	8.82E-02	7.37E-02
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					3.46E-02	8.48E-02	5.54E-04	2.36E-03		7.37E-01				1.18E-03	8.95E-05	2.22E-04			1.01E-02	8.47E-03		3.76E-01	***1.28E+00***	***1.26E+00***
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V																6.52E-04						***1.02E+00***	***1.02E+00***	***1.02E+00***

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW =

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation	Total ILCR	Total ILCR
Metals																								
Arsenic (C)	1.44E-07	1.20E-07	4.00E-08	6.26E-08	2.60E-07	2.17E-07	7.20E-08	1.13E-07	1.22E-04	**2.20E-04**	2.44E-08	5.57E-09	1.20E-11	4.39E-08	1.00E-08	7.68E-11	1.13E-07	2.86E-07	2.04E-07	5.15E-07	5.12E-09	3.28E-08	**2.31E-04**	**2.22E-04**
Cadmium (C)	5.35E-07	9.13E-07	1.97E-06	6.61E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.20E-05	0.00E+00	1.30E-08	9.87E-10	6.38E-12	0.00E+00	0.00E+00	1.15E-11	8.01E-09	6.73E-09	0.00E+00	0.00E+00	1.42E-07	2.56E-07	2.56E-07	2.56E-07
Cobalt (C)	4.69E-07	1.20E-06	3.08E-07	4.13E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.90E-05	0.00E+00	7.56E-08	5.75E-09	3.72E-11	0.00E+00	0.00E+00	6.69E-11	3.59E-07	3.02E-07	0.00E+00	0.00E+00	9.85E-08	1.77E-07	1.77E-07	1.77E-07
Nickel (C)	6.74E-06	9.60E-06	2.14E-05	7.91E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.20E-04	0.00E+00	1.15E-06	8.74E-08	5.66E-10	0.00E+00	0.00E+00	4.02E-10	6.46E-07	5.42E-07	0.00E+00	0.00E+00	6.46E-07	4.59E-07	4.59E-07	4.59E-07
SUM ILCR - chemicals with similar target orga																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																5.57E-10						9.25E-07	9.26E-07	9.26E-07

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day except for in

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	1.04E-04	3.06E-07	1.04E-04	3.06E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.00E-03	8.00E-03	8.00E-04	8.00E-04
Antimony (NC)	1.06E-06	3.12E-09	1.77E-04	5.20E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.62E-04	4.37E-02	2.62E-05	4.37E-03
Arsenic (C)	1.49E-06	4.37E-09	4.95E-03	1.46E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.92E-06	2.64E-02	7.92E-07	2.64E-03
Cadmium (C)	2.12E-07	6.24E-10	4.24E-04	1.25E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.01E-06	7.01E-03	7.01E-07	7.01E-04
Cobalt (C)	1.43E-06	1.68E-09	4.77E-03	5.61E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.64E-05	1.55E-01	4.64E-06	1.55E-02
Iron (NC)	6.90E-04	2.03E-06	9.85E-04	2.90E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.38E-02	1.97E-02	1.38E-03	1.97E-03
Manganese (NC)	1.29E-04	3.80E-07	6.47E-04	1.90E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.57E-04	1.29E-03	2.57E-05	1.29E-04
Nickel (C)	3.39E-06	2.00E-09	3.09E-04	1.81E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.68E-04	1.53E-02	1.68E-05	1.53E-03
Thallium (NC)	2.23E-07	6.55E-10	2.23E-02	6.55E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.77E-05	***1.77E+00***	1.77E-06	1.77E-01
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	2.55E-06	7.49E-09	5.09E-04	1.50E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.39E-05	2.78E-03	1.39E-06	2.78E-04
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			7.51E-04	2.21E-06			0.00E+00	0.00E+00		9.29E-03		9.29E-04
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m^3.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.11E-06	3.27E-09	2.00E-06	5.89E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.94E-06	**1.07E-05**	5.94E-07	1.07E-06
Cadmium (C)	1.59E-07	4.68E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.25E-06	0.00E+00	5.25E-07	0.00E+00
Cobalt (C)	1.07E-06	1.26E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.48E-05	0.00E+00	3.48E-06	0.00E+00
Nickel (C)	2.55E-06	1.50E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.26E-04	0.00E+00	1.26E-05	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m^3.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	1.46E-02	1.05E-03	3.74E-06	1.29E-03	1.46E-02	1.05E-03	3.74E-06	1.29E-03	1.16E-01	1.16E-01	8.63E-04	7.39E-05	5.03E-07	8.63E-04	7.39E-05	1.01E-04	4.68E-03	4.45E-03	4.68E-03	4.45E-03	4.63E-09	9.26E-07	1.51E-01	1.44E-01
Antimony (NC)	1.15E-06	5.28E-07	1.18E-08	7.95E-08	1.92E-04	8.80E-05	1.97E-06	1.32E-05	6.51E-05	1.08E-02	1.41E-08	1.21E-08	8.23E-12	2.36E-06	2.02E-06	4.1167E-08	6.07E-08	5.77E-07	1.01E-05	9.62E-05	1.23E-13	6.13E-10	5.51E-02	1.58E-02
Arsenic (C)	4.24E-06	8.81E-07	5.14E-07	7.40E-07	1.41E-02	2.94E-03	1.71E-03	2.47E-03	1.44E-03	***4.80E+00***	2.03E-07	5.22E-08	1.18E-10	6.77E-04	1.74E-04	1.1823E-07	9.58E-07	2.73E-06	3.19E-03	9.11E-03	6.77E-12	6.77E-09	***4.87E+00***	***4.84E+00***
Cadmium (C)	5.37E-06	3.53E-07	6.34E-06	1.21E-07	5.37E-03	3.53E-04	6.34E-03	1.21E-04	2.50E-04	2.50E-01	2.70E-08	2.31E-09	1.57E-11	2.70E-05	2.31E-06	1.573E-06	6.76E-08	6.43E-08	6.76E-05	6.43E-05	1.07E-10	1.07E-05	2.70E-01	2.63E-01
Cobalt (C)	7.98E-06	2.03E-06	3.65E-06	3.55E-05	2.66E-02	6.75E-03	1.22E-02	1.18E-01	2.30E-04	7.67E-01	5.81E-07	4.97E-08	3.38E-10	1.94E-03	1.66E-04	5.64E-05	3.03E-06	2.88E-06	1.01E-02	9.61E-03	2.45E-11	4.08E-06	***1.11E+00***	9.72E-01
Iron (NC)	6.64E-03	6.60E-04	4.30E-05	2.07E-02	9.49E-03	9.43E-04	6.14E-05	2.96E-02	2.49E-04	3.56E-04	1.25E-03	1.07E-04	7.25E-07	1.78E-03	1.52E-04	NA	6.72E-03	6.40E-03	9.60E-03	9.14E-03	8.65E-09	NA	8.18E-02	6.41E-02
Manganese (NC)	7.79E-03	6.36E-03	6.60E-05	1.15E-04	3.90E-02	3.18E-02	3.30E-04	5.75E-04	6.40E-02	3.20E-01	9.36E-06	8.01E-07	5.45E-09	4.68E-05	4.00E-06	1.09E-04	3.84E-04	3.65E-04	1.92E-03	1.82E-03	1.49E-10	2.97E-06	3.97E-01	3.96E-01
Nickel (C)	9.13E-05	3.49E-05	2.68E-04	6.57E-05	8.30E-03	3.17E-03	2.44E-02	5.97E-03	5.00E-03	4.55E-01	9.34E-06	7.99E-07	5.43E-09	8.49E-04	7.27E-05	3.02E-04	5.45E-06	5.19E-06	4.96E-04	4.71E-04	1.25E-10	6.96E-06	5.14E-01	5.00E-01
Thallium (NC)	6.36E-07	2.47E-06	2.15E-08	6.00E-06	6.36E-02	2.47E-01	2.15E-03	6.00E-01	1.50E-05	***1.50E+00***	1.46E-08	1.25E-09	8.48E-12	1.46E-03	1.25E-04	NA	3.39E-08	3.23E-08	3.39E-03	3.23E-03	6.01E-14	NA	***4.21E+00***	***2.62E+00***
Titanium (NC)	6.24E-04	1.90E-05	3.58E-06	1.71E-03	NA	NA	NA	NA	1.13E-02	NA	4.26E-05	3.65E-06	2.48E-08	NA	NA	2.48E-04	3.81E-05	3.63E-05	NA	NA	3.49E-10	3.49E-06	2.52E-04	2.52E-04
Vanadium (NC)	1.60E-05	1.23E-06	1.05E-08	4.91E-06	3.20E-03	2.47E-04	2.10E-06	9.82E-04	2.60E-04	5.20E-02	1.82E-06	1.56E-06	1.06E-09	3.64E-04	3.11E-04	1.06E-05	7.21E-06	6.86E-05	1.44E-03	1.37E-02	1.31E-11	1.31E-07	7.56E-02	7.31E-02
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					5.36E-02	3.28E-02	3.34E-04	1.87E-03		4.36E-01				9.10E-04	7.79E-05	2.09E-04			6.60E-03	6.28E-03		3.90E-06	5.49E-01	5.40E-01
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V																6.17E-04						1.47E-05	6.32E-04	6.32E-04

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW =

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation	Total ILCR	Total ILCR
Metals																								
Arsenic (C)	3.18E-06	6.60E-07	3.85E-07	5.55E-07	5.73E-06	1.19E-06	6.94E-07	9.99E-07	1.08E-03	**1.94E-03**	1.52E-07	3.91E-08	8.87E-11	2.74E-07	7.04E-08	5.68E-10	7.18E-07	2.05E-06	1.29E-06	3.69E-06	5.08E-12	3.25E-11	**1.97E-03**	**1.96E-03**
Cadmium (C)	4.03E-06	2.65E-07	4.75E-06	9.07E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.88E-04	0.00E+00	2.03E-08	1.73E-09	1.18E-11	0.00E+00	0.00E+00	2.12E-11	5.07E-08	4.82E-08	0.00E+00	0.00E+00	7.99E-11	1.44E-10	1.65E-10	1.65E-10
Cobalt (C)	5.98E-06	1.52E-06	2.74E-06	2.66E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.73E-04	0.00E+00	4.36E-07	3.73E-08	2.54E-10	0.00E+00	0.00E+00	4.57E-10	2.27E-06	2.16E-06	0.00E+00	0.00E+00	1.84E-11	3.31E-11	4.90E-10	4.90E-10
Nickel (C)	6.85E-05	2.62E-05	2.01E-04	4.93E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.75E-03	0.00E+00	7.00E-06	5.99E-07	4.08E-09	0.00E+00	0.00E+00	2.89E-09	4.09E-06	3.89E-06	0.00E+00	0.00E+00	9.40E-11	6.67E-11	2.96E-09	2.96E-09
SUM ILCR - chemicals with similar target orga																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																3.94E-09						2.76E-10	4.22E-09	4.22E-09

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day except for in

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	1.17E-04	3.43E-07	1.17E-04	3.43E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.27E-02	1.27E-02	1.27E-03	1.27E-03
Antimony (NC)	1.15E-06	3.39E-09	1.92E-04	5.64E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.47E-03	2.45E-01	1.47E-04	2.45E-02
Arsenic (C)	1.69E-06	4.96E-09	5.63E-03	1.65E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.82E-05	1.61E-01	4.82E-06	1.61E-02
Cadmium (C)	2.75E-07	8.08E-10	5.50E-04	1.62E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.69E-06	8.69E-03	8.69E-07	8.69E-04
Cobalt (C)	1.62E-06	1.90E-09	5.38E-03	6.33E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.83E-05	2.94E-01	8.83E-06	2.94E-02
Iron (NC)	7.81E-04	2.30E-06	1.12E-03	3.28E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.06E-02	2.95E-02	2.06E-03	2.95E-03
Manganese (NC)	1.45E-04	4.27E-07	7.26E-04	2.13E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.15E-04	3.08E-03	6.15E-05	3.08E-04
Nickel (C)	3.86E-06	2.27E-09	3.51E-04	2.06E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.41E-04	4.01E-02	4.41E-05	4.01E-03
Thallium (NC)	2.46E-07	7.23E-10	2.46E-02	7.23E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.12E-05	***6.12E+00***	6.12E-06	6.12E-01
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	2.84E-06	8.36E-09	5.69E-04	1.67E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.58E-05	1.52E-02	7.58E-06	1.52E-03
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			8.43E-04	2.48E-06			0.00E+00	0.00E+00		1.58E-02		1.58E-03
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m³.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.27E-06	3.72E-09	2.28E-06	6.70E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.61E-05	**6.50E-05**	3.61E-06	6.50E-06
Cadmium (C)	2.06E-07	6.06E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.51E-06	0.00E+00	6.51E-07	0.00E+00
Cobalt (C)	1.21E-06	1.42E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.62E-05	0.00E+00	6.62E-06	0.00E+00
Nickel (C)	2.90E-06	1.70E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.31E-04	0.00E+00	3.31E-05	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold	Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m³.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	1.79E-02	2.20E-03	4.71E-06	1.64E-03	1.79E-02	2.20E-03	4.71E-06	1.64E-03	1.16E-01	1.16E-01	8.93E-04	7.64E-05	5.20E-07	8.93E-04	7.64E-05	1.04E-04	4.68E-03	4.45E-03	4.68E-03	4.45E-03	5.04E-04	1.01E-01	2.62E-01	2.50E-01
Antimony (NC)	1.28E-06	5.82E-07	1.33E-08	8.99E-08	2.14E-04	9.71E-05	2.21E-06	1.50E-05	6.51E-05	1.08E-02	1.52E-08	1.30E-08	8.86E-12	2.54E-06	2.17E-06	4.4312E-08	6.07E-08	5.77E-07	1.01E-05	9.62E-05	1.85E-08	9.26E-05	2.56E-01	3.60E-02
Arsenic (C)	4.57E-06	1.02E-06	5.21E-07	8.15E-07	1.52E-02	3.39E-03	1.74E-03	2.72E-03	1.44E-03	***4.80E+00***	2.06E-07	5.29E-08	1.20E-10	6.87E-04	1.76E-04	1.1998E-07	9.58E-07	2.73E-06	3.19E-03	9.11E-03	5.12E-08	5.12E-05	***5.00E+00***	***4.86E+00***
Cadmium (C)	1.69E-05	7.71E-06	2.57E-05	8.62E-07	1.69E-02	7.71E-03	2.57E-02	8.62E-04	2.50E-04	2.50E-01	1.10E-07	9.38E-09	6.38E-11	1.10E-04	9.38E-06	6.3806E-06	6.76E-08	6.43E-08	6.76E-05	6.43E-05	1.42E-06	1.42E-01	4.53E-01	4.45E-01
Cobalt (C)	1.49E-05	1.01E-05	4.01E-06	5.38E-05	4.96E-02	3.37E-02	1.34E-02	1.79E-01	2.30E-04	7.67E-01	6.38E-07	5.47E-08	3.72E-10	2.13E-03	1.82E-04	6.19E-05	3.03E-06	2.88E-06	1.01E-02	9.61E-03	9.85E-07	1.64E-01	***1.53E+00***	***1.26E+00***
Iron (NC)	1.20E-02	2.78E-03	6.60E-05	2.91E-02	1.72E-02	3.97E-03	9.42E-05	4.15E-02	2.49E-04	3.56E-04	1.30E-03	1.11E-04	7.57E-07	1.86E-03	1.59E-04	NA	6.72E-03	6.40E-03	9.60E-03	9.14E-03	9.45E-04	NA	1.14E-01	8.79E-02
Manganese (NC)	9.17E-03	6.94E-03	7.17E-05	1.44E-04	4.59E-02	3.47E-02	3.58E-04	7.19E-04	6.40E-02	3.20E-01	1.02E-05	8.70E-07	5.91E-09	5.08E-05	4.35E-06	1.18E-04	3.84E-04	3.65E-04	1.92E-03	1.82E-03	1.37E-05	2.75E-01	6.84E-01	6.82E-01
Nickel (C)	2.13E-04	8.11E-05	2.79E-04	1.03E-04	1.94E-02	7.37E-03	2.53E-02	9.37E-03	5.00E-03	4.55E-01	9.71E-06	8.32E-07	5.66E-09	8.83E-04	7.56E-05	3.14E-04	5.45E-06	5.19E-06	4.96E-04	4.71E-04	6.46E-06	3.59E-01	9.18E-01	8.82E-01
Thallium (NC)	7.13E-07	2.58E-06	2.27E-08	6.71E-06	7.13E-02	2.58E-01	2.27E-03	6.71E-01	1.50E-05	***1.50E+00***	1.51E-08	1.29E-09	8.77E-12	1.51E-03	1.29E-04	NA	3.39E-08	3.23E-08	3.39E-03	3.23E-03	8.47E-09	NA	***8.66E+00***	***3.15E+00***
Titanium (NC)	8.95E-04	1.27E-04	5.35E-06	2.41E-03	NA	NA	NA	NA	1.13E-02	NA	4.55E-05	3.89E-06	2.65E-08	NA	NA	2.65E-04	3.81E-05	3.63E-05	NA	NA	4.85E-05	4.85E-01	4.85E-01	4.85E-01
Vanadium (NC)	2.54E-05	5.11E-06	1.61E-08	6.88E-06	5.08E-03	1.02E-03	3.21E-06	1.38E-03	2.60E-04	5.20E-02	1.91E-06	1.64E-06	1.11E-09	3.82E-04	3.27E-04	1.11E-05	7.21E-06	6.86E-05	1.44E-03	1.37E-02	1.59E-06	1.59E-02	1.07E-01	9.34E-02
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					6.37E-02	3.69E-02	3.63E-04	2.36E-03		4.36E-01				9.44E-04	8.08E-05	2.22E-04			6.60E-03	6.28E-03		3.76E-01	9.46E-01	9.32E-01
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V																6.52E-04						***1.02E+00***	***1.02E+00***	***1.02E+00***

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW =

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation	Total ILCR	Total ILCR
Metals																								
Arsenic (C)	3.43E-06	7.62E-07	3.91E-07	6.11E-07	6.17E-06	1.37E-06	7.04E-07	1.10E-06	1.08E-03	**1.94E-03**	1.55E-07	3.97E-08	9.00E-11	2.78E-07	7.15E-08	5.76E-10	7.18E-07	2.05E-06	1.29E-06	3.69E-06	3.84E-08	2.46E-07	**2.03E-03**	**1.97E-03**
Cadmium (C)	1.27E-05	5.78E-06	1.93E-05	6.46E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.88E-04	0.00E+00	8.22E-08	7.04E-09	4.79E-11	0.00E+00	0.00E+00	8.61E-11	5.07E-08	4.82E-08	0.00E+00	0.00E+00	1.07E-06	1.92E-06	1.92E-06	1.92E-06
Cobalt (C)	1.11E-05	7.58E-06	3.01E-06	4.04E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.73E-04	0.00E+00	4.79E-07	4.10E-08	2.79E-10	0.00E+00	0.00E+00	5.02E-10	2.27E-06	2.16E-06	0.00E+00	0.00E+00	7.39E-07	1.33E-06	1.33E-06	1.33E-06
Nickel (C)	1.60E-04	6.08E-05	2.09E-04	7.73E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.75E-03	0.00E+00	7.29E-06	6.24E-07	4.24E-09	0.00E+00	0.00E+00	3.01E-09	4.09E-06	3.89E-06	0.00E+00	0.00E+00	4.85E-06	3.44E-06	3.44E-06	3.44E-06
SUM ILCR - chemicals with similar target orga																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																4.18E-09						6.94E-06	6.94E-06	6.94E-06

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold
Bold

NA - not applicable. Exposure is in mg/kg BW/day except for in

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion
Metals												
Aluminum (NC)	1.04E-04	5.87E-08	1.04E-04	5.87E-08	3.47E-06	2.35E-07	3.47E-06	2.35E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Antimony (NC)	1.06E-06	5.99E-10	1.77E-04	9.99E-08	3.54E-08	2.40E-09	5.89E-06	4.00E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Arsenic (C)	1.49E-06	8.39E-10	4.95E-03	2.80E-06	4.95E-08	3.36E-09	1.65E-04	1.12E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium (C)	2.12E-07	1.20E-10	4.24E-04	2.40E-07	7.07E-09	4.79E-10	1.41E-05	9.59E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cobalt (C)	1.43E-06	3.24E-10	4.77E-03	1.08E-06	4.77E-08	1.29E-09	1.59E-04	4.32E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Iron (NC)	6.90E-04	3.90E-07	9.85E-04	5.57E-07	2.30E-05	1.56E-06	3.28E-05	2.23E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Manganese (NC)	1.29E-04	7.31E-08	6.47E-04	3.66E-07	4.31E-06	2.92E-07	2.16E-05	1.46E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Nickel (C)	3.39E-06	3.84E-10	3.09E-04	3.49E-08	1.13E-07	1.53E-09	1.03E-05	1.39E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Thallium (NC)	2.23E-07	1.26E-10	2.23E-02	1.26E-05	7.43E-09	5.03E-10	7.43E-04	5.03E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium (NC)	2.55E-06	1.44E-09	5.09E-04	2.88E-07	8.49E-08	5.75E-09	1.70E-05	1.15E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00
SUM HQ - chemicals with similar target organs/effects												
Neurotoxicity (Al, Mn)			7.51E-04	4.24E-07			2.50E-05	1.70E-06		0.00E+00		0.00E+00
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)												

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m³.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish			
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion
Metals												
Arsenic (C)	1.11E-06	6.29E-10	2.00E-06	1.13E-09	3.71E-08	2.52E-09	6.68E-08	4.53E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium (C)	1.59E-07	8.99E-11	0.00E+00	0.00E+00	5.30E-09	3.60E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cobalt (C)	1.07E-06	2.43E-10	0.00E+00	0.00E+00	3.58E-08	9.71E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Nickel (C)	2.55E-06	2.88E-10	0.00E+00	0.00E+00	8.49E-08	1.15E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
SUM ILCR - chemicals with similar target organs/effects												
Lung/respiratory tract tumours (As, Cd, Co, Ni)												

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m³.

Non-Carcinogenic Assessment

Parameter [1]	D. Country Foods								AVERAGE DIETARY INTAKE (BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	HQ - Berries	HQ - Labrador Tea	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ	Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Aluminum (NC)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.16E-01	1.16E-01	4.32E-03	7.39E-04	6.03E-07	4.32E-03	7.39E-04	1.21E-04	2.34E-02	1.86E-03	2.34E-02	1.86E-03	5.56E-09	1.11E-06	1.47E-01	1.47E-01
Antimony (NC)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.51E-05	1.08E-02	7.07E-08	1.21E-07	9.88E-12	1.18E-05	2.02E-05	4.94E-08	3.03E-07	2.41E-07	5.06E-05	4.01E-05	1.47E-13	7.36E-10	1.11E-02	1.11E-02
Arsenic (C)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.44E-03	***4.80E+00***	1.02E-06	5.22E-07	1.42E-10	3.39E-03	1.74E-03	1.4188E-07	4.79E-06	1.14E-06	1.60E-02	3.80E-03	8.12E-12	8.12E-09	***4.83E+00***	***4.83E+00***
Cadmium (C)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.50E-04	2.50E-01	1.35E-07	2.31E-08	1.89E-11	1.35E-04	2.31E-05	1.8876E-06	3.38E-07	2.68E-08	3.38E-04	2.68E-05	1.28E-10	1.28E-05	2.51E-01	2.51E-01
Cobalt (C)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.30E-04	7.67E-01	2.91E-06	4.97E-07	4.06E-10	9.68E-03	1.66E-03	6.76E-05	1.52E-05	1.20E-06	5.05E-02	4.01E-03	2.94E-11	4.90E-06	8.38E-01	8.38E-01
Iron (NC)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.49E-04	3.56E-04	6.23E-03	1.07E-03	8.70E-07	8.90E-03	1.52E-03	NA	3.36E-02	2.67E-03	4.80E-02	3.81E-03	1.04E-08	NA	6.36E-02	6.36E-02
Manganese (NC)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.40E-02	3.20E-01	4.68E-05	8.01E-06	6.54E-09	2.34E-04	4.00E-05	1.31E-04	1.92E-03	1.52E-04	9.59E-03	7.61E-04	1.78E-10	3.57E-06	3.31E-01	3.31E-01
Nickel (C)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.00E-03	4.55E-01	4.67E-05	7.99E-06	6.52E-09	4.24E-03	7.27E-04	3.62E-04	2.73E-05	2.16E-06	2.48E-03	1.97E-04	1.50E-10	8.36E-06	4.63E-01	4.63E-01
Thallium (NC)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.50E-05	***1.50E+00***	7.28E-08	1.25E-08	1.02E-11	7.28E-03	1.25E-03	NA	1.70E-07	1.35E-08	1.70E-02	1.35E-03	7.22E-14	NA	***1.55E+00***	***1.55E+00***
Titanium (NC)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	NA	NA	NA	NA	1.13E-02	NA	2.13E-04	3.65E-05	2.98E-08	NA	NA	2.98E-04	1.91E-04	1.51E-05	NA	NA	4.18E-10	4.18E-06	3.02E-04	3.02E-04
Vanadium (NC)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.60E-04	5.20E-02	9.09E-06	1.56E-05	1.27E-09	1.82E-03	3.11E-03	1.27E-05	3.60E-05	2.86E-05	7.21E-03	5.72E-03	1.58E-11	1.58E-07	7.04E-02	7.04E-02
SUM HQ - chemicals with similar target organs																								
Neurotoxicity (Al, Mn)					0.00E+00	0.00E+00	0.00E+00	0.00E+00		4.36E-01				4.55E-03	7.79E-04	2.51E-04			3.30E-02	2.62E-03		4.68E-06	4.78E-01	4.78E-01
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V																7.41E-04						1.76E-05	7.58E-04	7.58E-04

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BW =

Carcinogenic Assessment

Parameter [1]	D. Country Foods								(BACKGROUND)		E. Soil						F. Sediment				G. Air			
	Exposure - Berries	Exposure - Labrador Tea	Exposure - Snowshoe Hare	Exposure - Caribou	ILCR - Berries	ILCR - Labrador Tea	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR	Total ILCR	Total ILCR
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																								
Arsenic (C)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.08E-03	**1.94E-03**	7.62E-07	3.91E-07	1.06E-10	1.37E-06	7.04E-07	6.81E-10	3.59E-06	8.55E-07	6.47E-06	1.54E-06	6.09E-12	3.90E-11	**1.96E-03**	**1.96E-03**
Cadmium (C)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.88E-04	0.00E+00	1.01E-07	1.73E-08	1.42E-11	0.00E+00	0.00E+00	2.55E-11	2.54E-07	2.01E-08	0.00E+00	0.00E+00	9.59E-11	1.73E-10	1.98E-10	1.98E-10
Cobalt (C)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.73E-04	0.00E+00	2.18E-06	3.73E-07	3.04E-10	0.00E+00	0.00E+00	5.48E-10	1.14E-05	9.02E-07	0.00E+00	0.00E+00	2.20E-11	3.97E-11	5.88E-10	5.88E-10
Nickel (C)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.75E-03	0.00E+00	3.50E-05	5.99E-06	4.89E-09	0.00E+00	0.00E+00	3.47E-09	2.04E-05	1.62E-06	0.00E+00	0.00E+00	1.13E-10	8.01E-11	3.55E-09	3.55E-09
SUM ILCR - chemicals with similar target orga																								
Lung/respiratory tract tumours (As, Cd, Co, Ni)																4.73E-09						3.31E-10	5.06E-09	5.06E-09

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinog

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day except for in

Non-Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish				D. Country Foods					
	Exposure Ingestion	Exposure Dermal	HQ -Drinking Water Ingestion	HQ Dermal	Exposure Ingestion	Exposure Dermal	HQ Ingestion	HQ Dermal	Exposure - High Consumption Ingestion	HQ - Fish Ingestion	Exposure Low Consumption Ingestion	HQ - Fish Ingestion	Exposure - Berries Ingestion	Exposure - Labrador Tea Ingestion	Exposure - Snowshoe Hare Ingestion	Exposure - Caribou Ingestion	HQ - Berries Ingestion	HQ - Labrador Tea Ingestion
Metals																		
Aluminum (NC)	1.17E-04	6.60E-08	1.17E-04	6.60E-08	6.64E-05	4.50E-06	6.64E-05	4.50E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Antimony (NC)	1.15E-06	6.51E-10	1.92E-04	1.08E-07	1.53E-06	1.04E-07	2.56E-04	1.73E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Arsenic (C)	1.69E-06	9.54E-10	5.63E-03	3.18E-06	4.05E-06	2.74E-07	1.35E-02	9.14E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium (C)	2.75E-07	1.55E-10	5.50E-04	3.11E-07	2.33E-08	1.58E-09	4.65E-05	3.15E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cobalt (C)	1.62E-06	3.65E-10	5.38E-03	1.22E-06	1.10E-06	2.97E-08	3.65E-03	9.91E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Iron (NC)	7.81E-04	4.41E-07	1.12E-03	6.30E-07	8.34E-04	5.65E-05	1.19E-03	8.08E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Manganese (NC)	1.45E-04	8.20E-08	7.26E-04	4.10E-07	6.84E-05	4.64E-06	3.42E-04	2.32E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Nickel (C)	3.86E-06	4.36E-10	3.51E-04	3.97E-08	3.46E-06	4.70E-08	3.15E-04	4.27E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Thallium (NC)	2.46E-07	1.39E-10	2.46E-02	1.39E-05	1.47E-07	1.00E-08	1.47E-02	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Titanium (NC)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.00E+00	0.00E+00	0.00E+00	0.00E+00	NA	NA
Vanadium (NC)	2.84E-06	1.61E-09	5.69E-04	3.21E-07	2.07E-06	1.40E-07	4.14E-04	2.81E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
SUM HQ - chemicals with similar target organs/effects																		
Neurotoxicity (Al, Mn)			8.43E-04	4.76E-07			4.08E-04	2.77E-05		0.00E+00		0.00E+00					0.00E+00	0.00E+00
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)																		

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day (where BW = body weight) except for inhalation, for which exposure is in mg/m ^{3}.

Carcinogenic Assessment

Parameter [1]	A. Drinking Water				B. Surface Water				C. Fish				D. Country Foods					
	Exposure Ingestion	Exposure Dermal	ILCR - Drinking Water Ingestion	ILCR Dermal	Exposure Ingestion	Exposure Dermal	ILCR Ingestion	ILCR Dermal	Exposure - High Consumption	ILCR - Fish Ingestion	Exposure Low Consumption	ILCR - Fish Ingestion	Exposure - Berries Ingestion	Exposure - Labrador Tea Ingestion	Exposure - Snowshoe Hare Ingestion	Exposure - Caribou Ingestion	ILCR - Berries Ingestion	ILCR - Labrador Tea Ingestion
Metals																		
Arsenic (C)	1.27E-06	7.15E-10	2.28E-06	1.29E-09	3.03E-06	2.06E-07	5.46E-06	3.70E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cadmium (C)	2.06E-07	1.17E-10	0.00E+00	0.00E+00	1.74E-08	1.18E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Cobalt (C)	1.21E-06	2.74E-10	0.00E+00	0.00E+00	8.22E-07	2.23E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Nickel (C)	2.90E-06	3.27E-10	0.00E+00	0.00E+00	2.60E-06	3.52E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
SUM ILCR - chemicals with similar target organs/effects																		
Lung/respiratory tract tumours (As, Cd, Co, Ni)																		

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcinogen; NA is not assessed because the substance was not a carcinogen or not measured in a particular media

Bold Indicates that risk estimate exceeds target hazard quotient or index (HQ or HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable. Exposure is in mg/kg BW/day except for inhalation, for which exposure is in mg/m ^{3}.

Non-Carcinogenic Assessment

Parameter [1]	AVERAGE DIETARY INTAKE (BACKGROUND)				E. Soil						F. Sediment				G. Air		Total HI - High Fish Consumption	Total HI - Low Fish Consumption
	HQ - Snowshoe Hare	HQ - Caribou	EXPOSURE	HQ	Exposure			HQ			Exposure		HQ		Exposure	HQ		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation		
Metals																		
Aluminum (NC)	0.00E+00	0.00E+00	1.16E-01	1.16E-01	4.46E-03	7.64E-04	6.24E-07	4.46E-03	7.64E-04	1.25E-04	2.34E-02	1.86E-03	2.34E-02	1.86E-03	6.05E-04	1.21E-01	2.68E-01	2.68E-01
Antimony (NC)	0.00E+00	0.00E+00	6.51E-05	1.08E-02	7.61E-08	1.30E-07	1.06E-11	1.27E-05	2.17E-05	5.317E-08	3.03E-07	2.41E-07	5.06E-05	4.01E-05	2.22E-08	1.11E-04	1.15E-02	1.15E-02
Arsenic (C)	0.00E+00	0.00E+00	1.44E-03	***4.80E+00***	1.03E-06	5.29E-07	1.44E-10	3.44E-03	1.76E-03	1.44E-07	4.79E-06	1.14E-06	1.60E-02	3.80E-03	6.14E-08	6.14E-05	***4.85E+00***	***4.85E+00***
Cadmium (C)	0.00E+00	0.00E+00	2.50E-04	2.50E-01	5.48E-07	9.38E-08	7.66E-11	5.48E-04	9.38E-05	7.657E-06	3.38E-07	2.68E-08	3.38E-04	2.68E-05	1.71E-06	1.71E-01	4.23E-01	4.23E-01
Cobalt (C)	0.00E+00	0.00E+00	2.30E-04	7.67E-01	3.19E-06	5.47E-07	4.46E-10	1.06E-02	1.82E-03	7.43E-05	1.52E-05	1.20E-06	5.05E-02	4.01E-03	1.18E-06	1.97E-01	***1.04E+00***	***1.04E+00***
Iron (NC)	0.00E+00	0.00E+00	2.49E-04	3.56E-04	6.51E-03	1.11E-03	9.09E-07	9.29E-03	1.59E-03	NA	3.36E-02	2.67E-03	4.80E-02	3.81E-03	1.13E-03	NA	6.55E-02	6.55E-02
Manganese (NC)	0.00E+00	0.00E+00	6.40E-02	3.20E-01	5.08E-05	8.70E-06	7.10E-09	2.54E-04	4.35E-05	1.42E-04	1.92E-03	1.52E-04	9.59E-03	7.61E-04	1.65E-05	3.30E-01	6.62E-01	6.62E-01
Nickel (C)	0.00E+00	0.00E+00	5.00E-03	4.55E-01	4.86E-05	8.32E-06	6.79E-09	4.42E-03	7.56E-04	3.77E-04	2.73E-05	2.16E-06	2.48E-03	1.97E-04	7.76E-06	4.31E-01	8.94E-01	8.94E-01
Thallium (NC)	0.00E+00	0.00E+00	1.50E-05	***1.50E+00***	7.53E-08	1.29E-08	1.05E-11	7.53E-03	1.29E-03	NA	1.70E-07	1.35E-08	1.70E-02	1.35E-03	1.02E-08	NA	***1.57E+00***	***1.57E+00***
Titanium (NC)	NA	NA	1.13E-02	NA	2.27E-04	3.89E-05	3.18E-08	NA	NA	3.18E-04	1.91E-04	1.51E-05	NA	NA	5.82E-05	5.82E-01	5.82E-01	5.82E-01
Vanadium (NC)	0.00E+00	0.00E+00	2.60E-04	5.20E-02	9.56E-06	1.64E-05	1.34E-09	1.91E-03	3.27E-03	1.34E-05	3.60E-05	2.86E-05	7.21E-03	5.72E-03	1.91E-06	1.91E-02	9.02E-02	9.02E-02
SUM HQ - chemicals with similar target organ																		
Neurotoxicity (Al, Mn)	0.00E+00	0.00E+00		4.36E-01				4.72E-03	8.08E-04	2.67E-04			3.30E-02	2.62E-03		4.51E-01	9.30E-01	9.30E-01
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V										7.82E-04						***1.23E+00***	***1.23E+00***	***1.23E+00***

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcin

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day (where BV

Carcinogenic Assessment

Parameter [1]	AVERAGE DIETARY INTAKE				E. Soil						F. Sediment				G. Air			
	ILCR - Snowshoe Hare	ILCR - Caribou	EXPOSURE	ILCR	Exposure			ILCR			Exposure		ILCR		Exposure	ILCR		
	Ingestion	Ingestion	Ingestion	Ingestion	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Ingestion	Dermal	Inhalation	Inhalation	Total ILCR	Total ILCR
Metals																		
Arsenic (C)	0.00E+00	0.00E+00	1.08E-03	**1.94E-03**	7.73E-07	3.97E-07	1.08E-10	1.39E-06	7.15E-07	6.91E-10	3.59E-06	8.55E-07	6.47E-06	1.54E-06	4.61E-08	2.95E-07	**1.96E-03**	**1.96E-03**
Cadmium (C)	0.00E+00	0.00E+00	1.88E-04	0.00E+00	4.11E-07	7.04E-08	5.74E-11	0.00E+00	0.00E+00	1.03E-10	2.54E-07	2.01E-08	0.00E+00	0.00E+00	1.28E-06	2.31E-06	2.31E-06	2.31E-06
Cobalt (C)	0.00E+00	0.00E+00	1.73E-04	0.00E+00	2.39E-06	4.10E-07	3.34E-10	0.00E+00	0.00E+00	6.02E-10	1.14E-05	9.02E-07	0.00E+00	0.00E+00	8.86E-07	1.60E-06	1.60E-06	1.60E-06
Nickel (C)	0.00E+00	0.00E+00	3.75E-03	0.00E+00	3.64E-05	6.24E-06	5.09E-09	0.00E+00	0.00E+00	3.61E-09	2.04E-05	1.62E-06	0.00E+00	0.00E+00	5.82E-06	4.13E-06	4.13E-06	4.13E-06
SUM ILCR - chemicals with similar target orga																		
Lung/respiratory tract tumours (As, Cd, Co, Ni)										5.01E-09						8.33E-06	8.33E-06	8.33E-06

Notes:

1 - Chemical Qualifier- NC is a non-carcinogen; C is a carcin

Bold

Bold

NA - not applicable. Exposure is in mg/kg BW/day except for

Table V-13
Summary of ILCRs for the High Fish Consumer Seasonal User

	Baseline - Seasonal Users						Application Case - Seasonal Users						Baseline Case	Application Case
	ILCR						ILCR						ILCR	
	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	GK Worker	GK Worker
Metals														
Arsenic (C)	8.19E-06	**1.48E-04**	**2.83E-04**	**2.23E-04**	**1.97E-03**	**2.63E-03**	8.20E-06	**1.55E-04**	**2.93E-04**	**2.31E-04**	**2.03E-03**	**2.71E-03**	**1.96E-03**	**1.96E-03**
Cadmium (C)	1.38E-12	1.24E-11	1.93E-11	2.20E-11	1.65E-10	2.20E-10	1.60E-08	1.44E-07	2.24E-07	2.56E-07	1.92E-06	2.56E-06	1.98E-10	2.31E-06
Cobalt (C)	4.08E-12	3.67E-11	5.71E-11	6.53E-11	4.90E-10	6.53E-10	1.11E-08	9.98E-08	1.55E-07	1.77E-07	1.33E-06	1.77E-06	5.88E-10	1.60E-06
Nickel (C)	2.47E-11	2.22E-10	3.45E-10	3.95E-10	2.96E-09	3.95E-09	2.87E-08	2.58E-07	4.02E-07	4.59E-07	3.44E-06	4.59E-06	3.55E-09	4.13E-06
SUM ILCR - chemicals with similar target organs/effects														
Lung/respiratory tract tumours (As, Cd, Co, Ni)	3.51E-11	3.16E-10	4.92E-10	5.62E-10	4.22E-09	5.62E-09	5.79E-08	5.21E-07	8.10E-07	9.26E-07	6.94E-06	9.26E-06	5.06E-09	8.33E-06

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable

Table V-14
Summary of ILCRs for the Low Fish Consumer Seasonal User

	Baseline - Seasonal Users						Application Case - Seasonal Users						Baseline Case	Application Case
	ILCR						ILCR						ILCR	
	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	GK Worker	GK Worker
Metals														
Arsenic (C)	8.19E-06	**1.46E-04**	**2.81E-04**	**2.21E-04**	**1.96E-03**	**2.62E-03**	8.20E-06	**1.47E-04**	**2.82E-04**	**2.22E-04**	**1.97E-03**	**2.63E-03**	**1.96E-03**	**1.96E-03**
Cadmium (C)	1.38E-12	1.24E-11	1.93E-11	2.20E-11	1.65E-10	2.20E-10	1.60E-08	1.44E-07	2.24E-07	2.56E-07	1.92E-06	2.56E-06	1.98E-10	2.31E-06
Cobalt (C)	4.08E-12	3.67E-11	5.71E-11	6.53E-11	4.90E-10	6.53E-10	1.11E-08	9.98E-08	1.55E-07	1.77E-07	1.33E-06	1.77E-06	5.88E-10	1.60E-06
Nickel (C)	2.47E-11	2.22E-10	3.45E-10	3.95E-10	2.96E-09	3.95E-09	2.87E-08	2.58E-07	4.02E-07	4.59E-07	3.44E-06	4.59E-06	3.55E-09	4.13E-06
SUM ILCR - chemicals with similar target organs/effects														
Lung/respiratory tract tumours (As, Cd, Co, Ni)	3.51E-11	3.16E-10	4.92E-10	5.62E-10	4.22E-09	5.62E-09	5.79E-08	5.21E-07	8.10E-07	9.26E-07	6.94E-06	9.26E-06	5.06E-09	8.33E-06

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable

Table V-15
Summary of Risk Estimates for the Worker and High Fish Consuming Seasonal User

	Baseline Case						Application Case						Baseline Case	Application Case	Baseline Case	Application Case
	Hazard Index						Hazard Index						Hazard Index		ILCR	
	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	GK Worker			
Metals																
Aluminum (NC)	1.4E-01	3.1E-01	2.4E-01	2.0E-01	1.5E-01	N/A	2.5E-01	4.2E-01	3.6E-01	3.1E-01	2.6E-01	N/A	1.5E-01	2.7E-01	N/A	N/A
Antimony (NC)	9.5E-02	1.3E-01	9.6E-02	6.0E-02	5.5E-02	N/A	9.5E-02	5.0E-01	4.3E-01	2.8E-01	2.6E-01	N/A	1.1E-02	1.2E-02	N/A	N/A
Arsenic (C)	***2.4E+00***	***4.9E+00***	***6.0E+00***	***4.1E+00***	***4.9E+00***	**2.6E-03**	***2.4E+00***	***5.1E+00***	***6.2E+00***	***4.3E+00***	***5.0E+00***	**2.7E-03**	***4.8E+00***	***4.8E+00***	**2.0E-03**	**2.0E-03**
Cadmium (C)	2.3E-01	6.0E-01	5.0E-01	3.4E-01	2.7E-01	2.2E-10	4.0E-01	8.0E-01	6.8E-01	5.1E-01	4.5E-01	2.6E-06	2.5E-01	4.2E-01	2.0E-10	2.3E-06
Cobalt (NC)	***2.3E+00***	***2.6E+00***	***1.8E+00***	***1.3E+00***	***1.1E+00***	6.5E-10	***2.5E+00***	***3.2E+00***	***2.3E+00***	***1.7E+00***	***1.5E+00***	1.8E-06	8.4E-01	***1.0E+00***	5.9E-10	1.6E-06
Iron (NC)	1.4E-01	3.0E-01	1.1E-01	7.2E-02	8.2E-02	N/A	1.6E-01	3.5E-01	1.4E-01	9.8E-02	1.1E-01	N/A	6.4E-02	6.5E-02	N/A	N/A
Manganese (NC)	8.9E-01	***1.3E+00***	9.4E-01	6.7E-01	4.0E-01	N/A	***1.2E+00***	***1.6E+00***	***1.2E+00***	9.6E-01	6.8E-01	N/A	3.3E-01	6.6E-01	N/A	N/A
Nickel (C)	***1.3E+00***	1.0E+00	7.5E-01	6.1E-01	5.1E-01	3.9E-09	***1.7E+00***	***1.4E+00***	***1.2E+00***	***1.0E+00***	9.2E-01	4.6E-06	4.6E-01	8.9E-01	3.6E-09	4.1E-06
Thallium (NC)	***4.2E+00***	***7.9E+00***	***5.9E+00***	***4.2E+00***	***4.2E+00***	N/A	***4.3E+00***	***1.6E+01***	***1.3E+01***	***9.0E+00***	***8.7E+00***	N/A	***1.5E+00***	***1.6E+00***	N/A	N/A
Titanium (NC)	2.5E-04	2.5E-04	2.5E-04	2.5E-04	2.5E-04	N/A	4.9E-01	4.9E-01	4.9E-01	4.9E-01	4.9E-01	N/A	3.0E-04	5.8E-01	N/A	N/A
Vanadium (NC)	2.2E-01	1.4E-01	8.4E-02	5.8E-02	7.6E-02	N/A	2.4E-01	1.9E-01	1.2E-01	8.8E-02	1.1E-01	N/A	7.0E-02	9.0E-02	N/A	N/A
SUM HQ - chemicals with similar target organs/effects																
Neurotoxicity (Al, Mn)	***1.0E+00***	***1.6E+00***	***1.2E+00***	8.8E-01	5.5E-01	N/A	***1.5E+00***	***2.0E+00***	***1.6E+00***	***1.3E+00***	9.5E-01	N/A	4.8E-01	9.3E-01	N/A	N/A
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)	6.3E-04	6.3E-04	6.3E-04	6.3E-04	6.3E-04	N/A	***1.0E+00***	***1.0E+00***	***1.0E+00***	***1.0E+00***	***1.0E+00***	N/A	7.6E-04	***1.2E+00***	N/A	N/A
SUM ILCR - chemicals with similar target organs/effects																
Lung/respiratory tract tumours (As, Cd, Co, Ni)	NA	NA	NA	NA	NA	5.6E-09	NA	NA	NA	NA	NA	9.3E-06	***NA***	***NA***	5.1E-09	8.3E-06

Bold	Indicates that risk estimate exceeds target hazard index (HI>1)
Bold	Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

NA - not applicable

Table V-16
Summary of Risk Estimates for the Worker and Low Fish Consuming Seasonal User

Parameter	Baseline Case						Application Case						Baseline Case	Application Case	Baseline Case	Application Case
	Hazard Index						Hazard Index						Hazard Index		ILCR	
	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	GK Worker			
Metals																
Aluminum (NC)	1.4E-01	2.9E-01	2.3E-01	2.0E-01	1.4E-01	N/A	2.5E-01	4.0E-01	3.4E-01	3.0E-01	2.5E-01	N/A	1.5E-01	2.7E-01	N/A	N/A
Antimony (NC)	9.5E-02	5.6E-02	3.0E-02	1.8E-02	1.6E-02	N/A	9.5E-02	9.5E-02	5.7E-02	4.2E-02	3.6E-02	N/A	1.1E-02	1.2E-02	N/A	N/A
Arsenic (C)	***2.4E+00***	***4.8E+00***	***5.9E+00***	***4.1E+00***	***4.8E+00***	**2.6E-03**	***2.4E+00***	***4.9E+00***	***6.0E+00***	***4.1E+00***	***4.9E+00***	**2.6E-03**	***4.8E+00***	***4.8E+00***	**2.0E-03**	**2.0E-03**
Cadmium (C)	2.3E-01	5.9E-01	4.9E-01	3.3E-01	2.6E-01	2.2E-10	4.0E-01	7.8E-01	6.7E-01	5.0E-01	4.5E-01	2.6E-06	2.5E-01	4.2E-01	2.0E-10	2.3E-06
Cobalt (NC)	***2.3E+00***	***2.4E+00***	***1.6E+00***	***1.1E+00***	9.7E-01	6.5E-10	***2.5E+00***	***2.7E+00***	***1.9E+00***	***1.4E+00***	***1.3E+00***	1.8E-06	8.4E-01	***1.0E+00***	5.9E-10	1.6E-06
Iron (NC)	1.4E-01	2.7E-01	7.6E-02	5.4E-02	6.4E-02	N/A	1.6E-01	3.0E-01	9.7E-02	7.0E-02	8.8E-02	N/A	6.4E-02	6.5E-02	N/A	N/A
Manganese (NC)	8.9E-01	***1.2E+00***	9.4E-01	6.7E-01	4.0E-01	N/A	***1.2E+00***	***1.5E+00***	***1.2E+00***	9.6E-01	6.8E-01	N/A	3.3E-01	6.6E-01	N/A	N/A
Nickel (C)	***1.3E+00***	9.7E-01	7.3E-01	6.0E-01	5.0E-01	3.9E-09	***1.7E+00***	***1.4E+00***	***1.1E+00***	9.7E-01	8.8E-01	4.6E-06	4.6E-01	8.9E-01	3.6E-09	4.1E-06
Thallium (NC)	***4.2E+00***	***5.0E+00***	***3.2E+00***	***2.5E+00***	***2.6E+00***	N/A	***4.3E+00***	***6.0E+00***	***3.9E+00***	***3.1E+00***	***3.1E+00***	N/A	***1.5E+00***	***1.6E+00***	N/A	N/A
Titanium (NC)	2.5E-04	2.5E-04	2.5E-04	2.5E-04	2.5E-04	N/A	4.9E-01	4.9E-01	4.9E-01	4.9E-01	4.9E-01	N/A	3.0E-04	5.8E-01	N/A	N/A
Vanadium (NC)	2.2E-01	1.4E-01	8.0E-02	5.6E-02	7.3E-02	N/A	2.4E-01	1.6E-01	9.9E-02	7.4E-02	9.3E-02	N/A	7.0E-02	9.0E-02	N/A	N/A
SUM HQ - chemicals with similar target organs/effects																
Neurotoxicity (Al, Mn)	***1.0E+00***	***1.5E+00***	***1.2E+00***	8.7E-01	5.4E-01	N/A	***1.5E+00***	***2.0E+00***	***1.6E+00***	***1.3E+00***	9.3E-01	N/A	4.8E-01	9.3E-01	N/A	N/A
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)	6.3E-04	6.3E-04	6.3E-04	6.3E-04	6.3E-04	N/A	***1.0E+00***	***1.0E+00***	***1.0E+00***	***1.0E+00***	***1.0E+00***	N/A	7.6E-04	***1.2E+00***	N/A	N/A
SUM ILCR - chemicals with similar target organs/effects																
Lung/respiratory tract tumours (As, Cd, Co, Ni)	NA	NA	NA	NA	NA	5.6E-09	NA	NA	NA	NA	NA	9.3E-06	NA	NA	5.1E-09	8.3E-06

Bold Indicates that risk estimate exceeds target hazard index (HI>1)
Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)
NA - not applicable

Table V-17
Summary of Risk Estimates for the Worker and High Fish Consuming Seasonal User - Excluding Background Dietary Intake

	Baseline Case						Project Case						Baseline Case	Project Case	Baseline Case	Project Case
	Hazard Index					Total	Hazard Index						Hazard Index		ILCR	
	Infant	Toddler	Child	Adolescent	Adult	ILCR	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	GK Worker			
Metals																
Aluminum (NC)	9.08E-02	1.37E-01	4.16E-02	2.70E-02	3.52E-02	N/A	1.99E-01	***2.53E-01***	1.53E-01	1.36E-01	1.46E-01	N/A	3.05E-02	1.52E-01	N/A	N/A
Antimony (NC)	1.05E-03	8.18E-02	7.30E-02	4.74E-02	4.42E-02	N/A	1.22E-03	***4.54E-01***	***4.07E-01***	***2.64E-01***	***2.45E-01***	N/A	3.06E-04	7.01E-04	N/A	N/A
Arsenic (C)	9.32E-02	1.64E-01	8.39E-02	5.78E-02	6.58E-02	**3.90E-05**	9.70E-02	***4.16E-01***	***3.09E-01***	***2.04E-01***	***2.02E-01***	**1.21E-04**	3.00E-02	4.51E-02	**1.22E-05**	**1.85E-05**
Cadmium (C)	1.40E-02	3.08E-02	2.22E-02	1.51E-02	1.98E-02	2.20E-10	1.84E-01	***2.29E-01***	2.00E-01	1.87E-01	***2.03E-01***	2.56E-06	9.77E-04	1.73E-01	1.98E-10	2.31E-06
Cobalt (C)	1.93E-01	***7.32E-01***	***4.50E-01***	***3.11E-01***	***3.45E-01***	6.53E-10	***4.13E-01***	***1.31E+00***	***9.42E-01***	***7.13E-01***	***7.62E-01***	1.77E-06	7.09E-02	***2.73E-01***	5.88E-10	1.60E-06
Iron (NC)	1.40E-01	***3.00E-01***	1.06E-01	7.21E-02	8.15E-02	N/A	1.59E-01	***3.49E-01***	1.41E-01	9.80E-02	1.14E-01	N/A	6.33E-02	6.51E-02	N/A	N/A
Manganese (NC)	***2.23E-01***	***2.62E-01***	1.11E-01	1.14E-01	7.75E-02	N/A	***5.30E-01***	***5.69E-01***	***4.04E-01***	***4.04E-01***	***3.64E-01***	N/A	1.14E-02	***3.42E-01***	N/A	N/A
Nickel (C)	3.26E-02	1.08E-01	6.68E-02	4.88E-02	5.96E-02	3.95E-09	***4.18E-01***	***5.38E-01***	***4.81E-01***	***4.47E-01***	***4.63E-01***	4.59E-06	8.33E-03	***4.40E-01***	3.55E-09	4.13E-06
Thallium (NC)	***2.34E-01***	***4.63E+00***	***3.79E+00***	***2.70E+00***	***2.71E+00***	N/A	***2.56E-01***	***1.28E+01***	***1.11E+01***	***7.46E+00***	***7.16E+00***	N/A	4.99E-02	6.75E-02	N/A	N/A
Titanium (NC)	2.52E-04	2.52E-04	2.52E-04	2.52E-04	2.52E-04	N/A	***4.85E-01***	***4.85E-01***	***4.85E-01***	***4.85E-01***	***4.85E-01***	N/A	3.02E-04	***5.82E-01***	N/A	N/A
Vanadium (NC)	5.22E-02	6.44E-02	2.98E-02	2.23E-02	2.36E-02	N/A	7.27E-02	1.07E-01	6.84E-02	5.22E-02	5.50E-02	N/A	1.84E-02	3.82E-02	N/A	N/A
SUM HQ - chemicals with similar target organs/effects																
Neurotoxicity (Al, Mn)	***3.14E-01***	***4.00E-01***	1.52E-01	1.41E-01	1.13E-01	N/A	***7.29E-01***	***8.22E-01***	***5.58E-01***	***5.40E-01***	***5.10E-01***	N/A	4.20E-02	***4.94E-01***	N/A	N/A
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)	6.32E-04	6.32E-04	6.32E-04	6.32E-04	6.32E-04	N/A	***1.02E+00***	***1.02E+00***	***1.02E+00***	***1.02E+00***	***1.02E+00***	N/A	7.58E-04	***1.23E+00***	N/A	N/A
SUM ILCR - chemicals with similar target organs/effects																
Lung/respiratory tract tumours (As, Cd, Co, Ni)	NA	NA	NA	NA	NA	5.62E-09	NA	NA	NA	NA	NA	9.26E-06	***NA***	***NA***	5.06E-09	8.33E-06

NA - not applicable

Bold Indicates that risk estimate exceeds target hazard index (HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

Table V-18
Summary of Risk Estimates for the Worker and Low Fish Consuming Seasonal User, Excluding Background Dietary Intake

Parameter	Baseline Case						Application Case						Baseline Case	Application Case	Baseline Case	Application Case
	Hazard Index						Hazard Index						Hazard Index		ILCR	
	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	Infant	Toddler	Child	Adolescent	Adult	Total ILCR	GK Worker			
Metals																
Aluminum (NC)	9.08E-02	1.24E-01	2.94E-02	1.93E-02	2.80E-02	N/A	1.99E-01	***2.32E-01***	1.34E-01	1.23E-01	1.34E-01	N/A	3.05E-02	1.52E-01	N/A	N/A
Antimony (NC)	1.05E-03	9.54E-03	6.51E-03	5.61E-03	4.95E-03	N/A	1.22E-03	4.88E-02	3.41E-02	2.95E-02	2.52E-02	N/A	3.06E-04	7.01E-04	N/A	N/A
Arsenic (C)	9.32E-02	1.21E-01	4.37E-02	3.25E-02	4.20E-02	**2.48E-05**	9.70E-02	1.50E-01	6.41E-02	5.01E-02	5.80E-02	**3.44E-05**	3.00E-02	4.51E-02	**1.22E-05**	**1.85E-05**
Cadmium (C)	1.40E-02	1.92E-02	1.15E-02	8.43E-03	1.35E-02	2.20E-10	1.84E-01	***2.14E-01***	1.86E-01	1.79E-01	1.95E-01	2.56E-06	9.77E-04	1.73E-01	1.98E-10	2.31E-06
Cobalt (C)	1.93E-01	***4.76E-01***	***2.15E-01***	1.63E-01	***2.06E-01***	6.53E-10	***4.13E-01***	***8.21E-01***	***4.94E-01***	***4.32E-01***	***4.97E-01***	1.77E-06	7.09E-02	***2.73E-01***	5.88E-10	1.60E-06
Iron (NC)	1.40E-01	***2.67E-01***	7.57E-02	5.32E-02	6.37E-02	N/A	1.59E-01	***3.00E-01***	9.58E-02	6.97E-02	8.76E-02	N/A	6.33E-02	6.51E-02	N/A	N/A
Manganese (NC)	***2.23E-01***	***2.58E-01***	1.07E-01	1.11E-01	7.63E-02	N/A	***5.30E-01***	***5.59E-01***	***3.95E-01***	***3.98E-01***	***3.62E-01***	N/A	1.14E-02	***3.42E-01***	N/A	N/A
Nickel (C)	3.26E-02	8.26E-02	4.35E-02	3.42E-02	4.58E-02	3.95E-09	***4.18E-01***	***4.72E-01***	***4.20E-01***	***4.08E-01***	***4.27E-01***	4.59E-06	8.33E-03	***4.40E-01***	3.55E-09	4.13E-06
Thallium (NC)	***2.34E-01***	***1.71E+00***	***1.09E+00***	***1.01E+00***	***1.12E+00***	N/A	***2.56E-01***	***2.68E+00***	***1.78E+00***	***1.60E+00***	***1.65E+00***	N/A	4.99E-02	6.75E-02	N/A	N/A
Titanium (NC)	2.52E-04	2.52E-04	2.52E-04	2.52E-04	2.52E-04	N/A	***4.85E-01***	***4.85E-01***	***4.85E-01***	***4.85E-01***	***4.85E-01***	N/A	3.02E-04	***5.82E-01***	N/A	N/A
Vanadium (NC)	5.22E-02	5.98E-02	2.56E-02	1.96E-02	2.11E-02	N/A	7.27E-02	8.23E-02	4.53E-02	3.77E-02	4.14E-02	N/A	1.84E-02	3.82E-02	N/A	N/A
SUM HQ - chemicals with similar target organs/effects																
Neurotoxicity (Al, Mn)	***3.14E-01***	***3.82E-01***	1.36E-01	1.30E-01	1.04E-01	N/A	***7.29E-01***	***7.91E-01***	***5.29E-01***	***5.22E-01***	***4.96E-01***	N/A	4.20E-02	***4.94E-01***	N/A	N/A
Respiratory Tract (inhalation; Sb, As, Co, Ni, Ti, V)	6.32E-04	6.32E-04	6.32E-04	6.32E-04	6.32E-04	N/A	***1.02E+00***	***1.02E+00***	***1.02E+00***	***1.02E+00***	***1.02E+00***	N/A	7.58E-04	***1.23E+00***	N/A	N/A
SUM ILCR - chemicals with similar target organs/effects																
Lung/respiratory tract tumours (As, Cd, Co, Ni)	NA	NA	NA	NA	NA	5.62E-09	NA	NA	NA	NA	NA	9.26E-06	NA	NA	5.06E-09	8.33E-06

NA - not applicable

Bold Indicates that risk estimate exceeds target hazard index (HI>1)

Bold Indicates that risk estimate exceeds ILCR value (ILCR >1E-5)

APPENDIX VI

PARTICULATE MATTER LITERATURE REVIEW

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VI.1 POTENTIAL HEALTH EFFECTS ASSOCIATED WITH PARTICULATE MATTER BASED ON TOXICOLOGY STUDIES

The primary toxicological responses to environmental exposure to airborne particulate matter are respiratory and cardiovascular effects. A brief summary of the literature toxicological studies related to health effects associated with exposure to particulate matter is provided below.

The toxicological mechanism associated with particulate matter related health effects is typically inflammation caused by a primary response of alveolar macrophages and pulmonary epithelial cells. The response involves the release of signalling (i.e., cytokines and chemokines) and adherence molecules which mediate a complex interaction between the epithelial cells, the alveolar macrophages and other immune cells such as neutrophils and T-cells (Schwarze et al. 2006). The type of cytokines and chemokines released as a result of exposure determines the type of resulting health effect. For example, allergic asthma caused by attraction of eosinophils involves different mediators than those that attract neutrophils involved in non-allergic inflammatory disease (Schwarze et al. 2006). The recruited immune cells may release secondary cytokines as well as reactive oxygen species, lipid mediators and toxic proteases which can cause epithelial damage leading to the subsequent release of additional cytokines or chemokines thus increasing or prolonging the inflammatory reaction (Schwarze et al. 2006). The increased inflammatory reaction can ultimately cause chronic inflammation resulting in respiratory illness. Particulate matter is also believed to be cytotoxic contributing to cell death which can also cause inflammation, leading to the development of acute and chronic lung disease. Particulate matter that is generated from diesel exhaust has been shown to cause lung cancer. The mechanism by which the particulate matter causes cancer is related to direct interaction of the metabolized particle components and the oxidative stress products with DNA and the subsequent formation of DNA adducts and mutations (Schwarze et al. 2006). The inflammatory process can also contribute to progression of lung cancer but the mechanism (s) by which this occurs are not yet fully understood.

There are also several possible mechanisms by which particulate matter can cause cardiovascular disease. One explanation is that the fine particulate matter and substances that are bound to it such as metals and inflammatory products enter the blood stream and interact with the heart (Schwarze et al. 2006). In addition, some experimental studies have shown that inhalation of particulate matter components or induced substances can alter heart rate, increasing the likelihood of a heart attack (Schwarze et al. 2006). Inflammatory pathways have been identified as being

important in various aspects of the development of cardiac disease but the identification of the critical pathway has not yet been realized.

Particle size and component composition (i.e., metal content) have also been shown to be important drivers of the toxicological mechanism for particulate matter related health effects, although experimental studies have shown that ultrafine particles are particularly toxic due to their high surface area to mass ratio and surface reactivity. However, other experimental studies have also shown larger particles are equally and often more potent than fine fraction particles, and this may be due to the importance of particle composition (i.e., size effects may not override the potential for a higher concentration of toxic or inflammatory components present in larger particles). The available epidemiological studies indicate that there is greater evidence for $PM_{2.5}$ and $PM_{2.5-10}$ to cause mortality than PM_{10}; however both the coarse and fine fractions seem to contribute to morbidity.

Schwarze et al. 2006 conclude that the available data indicate that particle size alone is not the critical determinant of particulate matter induced health effects. The concentration of particulate matter component constituents such as metals, soluble organic compounds and sulphates are also important considerations, but their contribution to toxicological effects are generally not well understood.

There are a limited number of epidemiological studies that indicate an association between metals content and air pollution-related mortality. Metals from particulate matter may also cause allergic reaction but this has not yet been established by the available epidemiological studies (Schwarze et al. 2006). The available data indicate that iron, copper, nickel, vanadium and zinc are the primary metals of concern, but it is also possible that other metals could contribute to health effects. There is also currently insufficient evidence with respect to the role of organic compounds in particulate matter induced disease for the general population based on epidemiological studies. There is some information available from occupational studies for carcinogenic effects associated with organic chemicals in particulate matter. Although experimental studies indicate that soluble organic compounds contribute to the inflammatory response related to exposure of particulate matter from diesel related sources, information is not yet available about the specific organic compounds in particulate matter effects. There is also little information available about the relative contribution associated with soluble organic compounds and other components of particulate matter (i.e., metals). Experimental and epidemiological studies are not currently consistent with respect to whether sulphates contribute to particulate matter induced health effects.

Biological agents such as mold and endotoxin are also known to cause allergic reactions; however, epidemiological studies associated with the biological

components of particulate matter are quite limited (Schwarze et al. 2006). One study in California indicated that pollen present in particulate matter had an effect on people suffering from asthma. Schwarze et al. (2006) indicate that an experimental study has shown a greater cytokine reaction induced by PM_{10} relative to $PM_{2.5}$ and it was thought to be related to the higher endotoxin component of the coarse PM.

Schwarze et al. (2006) indicate that epidemiological studies conducted in areas where crustal particles dominate have caused adverse health effects associated with respiratory morbidity. However, the authors indicate that the majority of the available experimental studies have been conducted with quartz and asbestos and may not be completely representative of coarse particulate matter associated with windblown dust or road abrasion particles. Therefore, although epidemiological studies indicate potential health effects associated with crustal particulates, the experimental studies are not yet available to determine the relative importance of the mineral components of particulate matter.

VI.2 EPIDEMIOLOGICAL STUDIES ASSOCIATED WITH PARTICULATE MATTER FROM CRUSTAL-DERIVED SOURCES

Since the haul road is considered to be a major contributor to the high particulate matter predictions, a literature review of epidemiological studies was completed to determine if a quantitative relationship can be determined relating exposure to particulate matter from crustal sources and human health effects. Epidemiological studies that evaluated crustal sources were most typically associated with dust storms or other natural events such as volcanic eruptions or geographic locations where there was an absence of industrial activities; thereby leading to the assumption that the particulate matter is generally from crustal rather than combustion sources.

Multiple studies suggest that mortality and morbidity are more closely linked to particulate matter from combustion rather than crustal sources (i.e., dust storms, re-suspended dust from road traffic, agriculture, and mining), unless the particulate is derived from geologic sources and contains high concentrations of metals. Dust events containing high coarse matter particulate as the result of high windspeeds tend to have reduced concentrations of fine particulate and other combustion related particulate and measurement of dust storm events allows the authors to unambiguously attribute them to a crustal source (i.e., non-pollution events). As a result dust storms are often studied from the perspective of the effect of crustal sources on health effects (Schwartz et al. 1999; Staniswalis et al. 2005).

Overall there is some evidence that coarse particulate matter has an effect on mortality, most predominately in arid regions where concentrations of coarse particulate matter are high (Ostro 1999; Staniswalis et al. 2005). Of the available studies on the effects of coarse particulate matter on health, few studies have analysed coarse and fine particulate matter jointly. For studies that examined the effects of coarse and fine particulate matter, in association with gaseous pollutants, study results may need to be adjusted to reflect this influence. In studies of morbidity, coarse particulate matter generally had a similar effect on asthma and respiratory admissions to fine particulate matter. The available evidence suggests that coarse particles have the potential to cause respiratory and cardiovascular morbidity. The majority of the studies conducted on the effects of coarse particulate matter derived from crustal sources indicate that the effect of crustal particulate matter on mortality and morbidity is less than that from PM_{10} originating from combustion sources.

VI.2.1 $PM_{2.5}$

VI.2.1.1 Mortality

Table VI-1 presents a summary of information located with respect to mortality as the result of exposure to $PM_{2.5}$ from crustal sources. Epidemiological studies with respect to fine particulate matter from crustal sources are limited. Laden et al. (2000) found that increased mortality was not associated with fine particulate matter from crustal material. Fine particulate matter from a variety of combustion and vehicular sources in addition to crustal material was categorized by determining the elemental composition of the fraction to identify source related factors. Several types of factors were identified including a silicon factor in fine particulate matter from crustal material, a lead factor in fine particulate matter from vehicle exhaust and a selenium factor in particulate matter from coal combustion. Mortality rates were compared to source factor concentrations for six eastern US cities (Watertown, MA, Kingston-Harriman, TN, St. Louis, MO, Portage, WI, and Topeka, KS). The results showed that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ from mobile (i.e., vehicle) sources accounted for a 3.4% increase in daily mortality, an equivalent increase in fine particles from coal combustion sources accounted for a 1.1% increase in mortality and $PM_{2.5}$ crustal particles were not associated with daily mortality.

Table VI-1 Summary of Mortality Studies for $PM_{2.5}$ Originating from Crustal Sources

Location	United States (Various Locations)
$PM_{2.5}$ Concentration during Dust Storm Event (24-hour average) ($\mu g/m^3$)	NA
$PM_{2.5}$ Concentration on Control days (24-hour average) ($\mu g/m^3$)	NA
Change in $PM_{2.5}$ concentration (24-hour average) ($\mu g/m^3$)	10
Daily Increase in Mortality	4.5%[(a)]
All-cause (3-day lag)	NA
All-cause (2-day lag)	NA
Cardio-respiratory	NA
Reference	Fuentes et al. 2006

[(a)] Confidence intervals (2.5% and 97.5% are 4.2% and 4.7%, respectively).

NA = not assessed or not available.

However, Fuentes et al. (2006) found a statistically significant positive association between crustal $PM_{2.5}$ and incidence of mortality from natural causes (i.e., deaths other than from accident, violence or suicide), independent of $PM_{2.5}$ from all other sources at various locations in the United States for data collected in June 2000. The attributable risk was 4.5% per 10 $\mu g/m^3$ increase in crustal $PM_{2.5}$. It should be noted that the other components of the speciated $PM_{2.5}$ such as sulphate, nitrate and ammonium also showed elevated increases in mortality ranging from 6.6 to 7.5% per 10 $\mu g/m^3$ increase in crustal $PM_{2.5}$. Fuentes et al. (2006) indicated that in the Western United States, crustal particulate matter and nitrate had the greatest impact on mortality relative to the other components of $PM_{2.5}$.

Ostro et al. (2000) assessed 10 years (1989 to 1998) of daily data on mortality and PM_{10} concentration for two locations (Palm Springs and Indio) in the Coachella Valley, California. In addition, during the final 2.5 years in this same period, daily data on $PM_{2.5}$ was collected to allow for the assessment of size-specific impacts. Ostro et al. (2000) indicate that they did not find an association between $PM_{2.5}$ concentrations and cardio-vascular mortality. The authors indicate that they found a less robust association between $PM_{2.5}$ and all-cause mortality which was found to be statistically significant only on the fourth day after the dust storm (i.e., four day lag) and suggest that the reduced number of $PM_{2.5}$ samples in comparison to PM_{10} samples and low $PM_{2.5}$ concentrations during monitoring (24-hour $PM_{2.5}$ concentrations ranged from 13 to 17 $\mu g/m^3$ in the Coachella Valley) may have impacted statistical power (Ostro et al. 2000).

VI.2.2 PM_{10}

VI.2.2.1 Mortality

Table VI-2 presents a summary of information located with respect to mortality as the result of exposure to PM_{10} from crustal sources. A study by Schwartz et al. (1999) examined whether coarse particle concentrations are associated with mortality. Coarse particulate matter (PM_{10}) measurements were collected during seventeen dust storm events in Spokane, Washington between 1989 and 1995 and these data were correlated with deaths in the same area. The source of the dust is arid agricultural areas and dust storms occur in the fall after crops have been harvested. Control days were chosen from the same day of the year in a previous year when a dust storm did not occur. The study found that mean 24-hour PM_{10} concentrations were 263 µg/m^3 on exposure days (i.e., dust storm events) compared to concentrations of approximately 42 µg/m^3 on control days. The study found no evidence that mortality was elevated on dust storm days in Spokane as compared to control days and concluded that control of airborne particles should focus on combustion particles not crustal particles if the goal is to reduce human health effects.

Table VI-2 Summary of Mortality Studies for PM_{10} Originating from Crustal Sources

Location	Spokane, Washington	Taipei, Taiwan	Coachella Valley, California	Coachella Valley, California	Taipei, Taiwan	Helsinki, Finland	El Paso, Texas
PM_{10} Concentration during Dust Storm Event (24-hour average) ($\mu g/m^3$)	263	101.1	NA	NA	NA	NA	NA
PM_{10} Concentration on Control days (24-hour average) ($\mu g/m^3$)	42	73.3	NA	NA	NA	NA	NA
Change in PM_{10} concentration (24-hour average) ($\mu g/m^3$)	221	27.8	10	10	64.1	NA	10
Daily Increase in Mortality	NF	NA	1%	4.4%	NA	NA	1.7% to 2.1%
All-cause (3-day lag)	NF	1.7%	NA	NA	NA	NA	NA
All-cause (2-day lag)	NF	3.5%[(a)]; 5.3%[(b)]	NA	NA	7.7%[(c)]	NA	NA
Cardio-respiratory	NF	3.7%[(a)]	1.2%	NA	2.6%[(c)]	NA	NA
Reference	Schwartz et al. 1999	Kwon et al. 2002	Ostro et al. 1999	Ostro et al. 2000	Chen et al. 2004	Penttinen et al. 2004	Staniswalis et al. 2005

(a) All age groups combined.

(b) Over 65 years.

(c) Not statistically significant.

NF = no association found between 24-hour PM_{10} concentrations and mortality, NA = not assessed or not available.

Slaughter et al. (2005) conducted a study to determine whether there is an association between different size fractions of particulate matter and cardiac and respiratory mortality and morbidity. The study examined the association between four fractions of particulate matter (PM_1, $PM_{2.5}$, PM_{10} and $PM_{2.5-10}$) and carbon monoxide and hospital visits/admissions in Spokane, Washington for respiratory and cardiac conditions and mortality between 1995 and 2001. The study did not find any associations with respiratory or cardiac hospital admissions or deaths with any fraction of particulate matter; however, the study did note a greater effect on respiratory health from fine versus coarse particulate matter.

Kwon et al. (2002) studied the effects of wind-blown dust originating from the arid deserts of China and Mongolia on daily mortality for people aged under 65 years as well as in elderly people (greater than or equal to 65 years) in Seoul, Korea from 1995 to 1998. The dust is composed primarily of crustal sources and ranges between 1.35 µm and 10 µm in size, but may also contain chemicals from combustion sources in Eastern China. The association between dust storm events and daily death counts was assessed using regression analysis which was adjusted

for temporal trends and weather variables. The assessment was based on 28 dust storm days observed in Seoul, Korea between 1995 and 1998. The average 24-hour PM_{10} concentration observed during the dust storm was 101.1 µg/m^3 compared to 73.3 µg/m^3 on control days (i.e., non-dust storm days). For all-cause mortality (all ages combined) an increase of 1.7% (95% confidence interval [CI] -2.8–6.5) was observed with the 3-d moving average. The risk was highest 2 days after the event as an increase of 3.5 % (95% CI -0.5–7.4) was observed. The results are based on an increase of 27.8 µg/m^3 in 24-hour PM_{10} concentration. The analysis of subjects older than 65 years indicated a higher risk 5.3% (95% CI 0.3–10.5) 2-days after exposure to the dust storm event which was considered to be significant. For all ages combined, the association with cardio-respiratory mortality was highest on the event day (3.7%; 95% CI -2.7-10.5) and decreased thereafter. For all other non-accidental causes of mortality, negative and non-statistically significant associations were found with exposure to the dust storm events.

Ostro et al. (1999) examined the role of PM_{10} in relation to daily mortality in the Coachella Valley, California, where geological particles comprise a significant percentage of the total particulate mass throughout much of the year, especially during wind storms. Analyses were conducted using daily data on mortality from 1989 to 1992 for several pollutants and meteorological variables. Outcome variables included several measures of daily mortality, including all-cause, cardiovascular and respiratory mortality, and counts of death for those above age 50. The study noted statistically significant associations between PM_{10} (2 or 3 day lags) and each measure of mortality. A 10 µg/m^3 change in daily PM_{10} was associated with an approximately 1% increase in mortality, which is of similar magnitude to particle-associated impacts identified in urban areas.

Ostro et al. (2000) repeated the earlier investigation conducted by Ostro et al. (1999) using 10 years (1989 to 1998) of daily data on mortality and PM_{10} for two locations (Palm Springs and Indio) in the Coachella Valley, California. Outcome variables included several measures of daily mortality, including all case (minus accidents and homicides), cardiovascular and respiratory mortality. The average 24-hour PM_{10} concentrations were 29.8 and 47.4 µg/m^3 for Palm Springs and Indio, respectively. Ostro et al. (2000) found an association between PM_{10} and cardiovascular mortality. Ostro et al. (2000) found a statistically significant positive association between PM_{10} and incidence of death from cardiovascular disease. The attributable risk percent was 3% (95%CI: 1% to 5%) per 24.6 µg/m^3 increase in PM_{10}.

The authors concluded that although this study was carried out in an area in which PM_{10} is strongly correlated with the coarse fraction, the magnitudes of the associations are similar to those observed in numerous areas in which variability in particle concentration is due primarily to changes in combustion-related fine particles.

Staniswalis et al. (2005) performed a study in El Paso, Texas to determine if the mortality that occurred during 1992–95 was associated with the temporal variability of PM_{10} levels within a 24-h period. In addition, the researchers investigated the association of PM_{10} with total mortality in relation to wind speed, assuming that at high speeds wind was composed primarily of coarse PM from re-suspended dust whereas at low wind speeds it was mostly fine PM from urban sources. The 24-hour average PM_{10} concentration ranged from 0.2 and 133.4 $\mu g/m^3$. In this area, $PM_{2.5}$ is about 25% of the total PM_{10} concentrations and hourly PM_{10} concentrations have been noted to peak in the evenings during still-air conditions. Between 1992 and 1995, the daily death rate for the area ranged from 1 to 21 deaths/day with an average 8.5 deaths/day. A principal component analysis (PCA) showed that 40% of the total variation in daily PM_{10} concentration was explained by a peak occurring near 8 pm and that the daily average only accounts for 28% of this variation. Using the results of the PCA (hourly data), an increase of 2.06% total mortality per 10 $\mu g/m^3$ increase in PM_{10} concentrations three days after the event (3-day lag) was found. In contrast, a non-significant increase of 1.7% in total mortality for 10 $\mu g/m^3$ increase in the 24-hour mean PM_{10} concentration (3-day lag), was found using 24-hour average PM_{10} levels. The mortality risk was derived based on high wind speed and low to mid wind speed conditions, and the differences between those wind conditions were examined. A high wind speed at night (greater than 7.6 m/sec) was significantly associated with a 10% lower risk of mortality in the 3 days following high wind speed event as compared to low and mid wind speed conditions. This suggests that crustal particles may have a weaker negative impact on health outcomes. It is important to note that coarse particles in El Paso are believed to contain deposited metals from historic mining and smelting and that the mineral content of the coarse particulate matter may affect the mortality results presented here, when compared to crustal particulate matter from other locations.

Pope et al. (1999) examined the weak association between particulate matter (PM_{10}) concentrations and mortality in Salt Lake City, Utah; however, a reasonably strong association was found in a neighbouring community (Provo, Utah). The study found that Salt Lake City is subject to significantly more episodes of dust storms than Provo. Exclusion of data (24-hour PM_{10} measurements) that were associated with dust storm events and the use of particulate matter measurements from multiple monitors resulted in a revised association that was similar to that for Provo, Utah. The study concluded that particulate matter from combustion sources was more closely associated with increased mortality than wind-blown particulate matter which is high in coarse crustal material.

Chen et al. 2004 also studied the effects of Asian dust storm events from 1995 to 2000 on daily mortality in Taipei, Taiwan. The mean number of deaths due to non-accidental causes was 27, while the mean numbers of deaths due to cardiovascular and respiratory causes were respectively 7.31 and 2.8. Increases of 4.92% and

2.59% were observed for all-cause mortality and cardiovascular mortality, respectively, 2 days after the dust event, which had caused an increase of 68.14 µg/m^3 in PM_{10}. The highest effect was found for respiratory mortality with an increase of 7.66% 1 day after the event. However, all estimates were non-statistically significant.

In Finland, PM_{10} particles originate from re-suspended coarse road dust as well as the spreading of sand on streets in the spring. Penttinen et al. (2004) examined the association between air particulate concentrations in air in the greater Helsinki area for all-cause, respiratory and cardiovascular mortality between 1988 and 1996. A measure of the blackness of TSP was used as a surrogate for fine and combustion-derived particles to evaluate the impact of fine particulate, $PM_{2.5}$, on mortality. The TSP blackness was also highly correlated with carbon monoxide indicating that combustion-derived particles were a major contributor to this measure. The study identified positive but non-significant relationships between PM_{10} and both total and cardiovascular mortality for all age groups. Median 24-hour average concentrations were for TSP, PM_{10} and $PM_{2.5}$ were found to be 57, 28 and 15 µg/m^3, respectively while maximum concentrations ranged from 234, 122 and 55 µg/m^3, respectively. Positive and significant associations were identified for PM_{10} and respiratory mortality. Increases of 3.94% (95% CI 0.01–7.87) on the same day of measurement, 3.96% (95% CI 0.11–7.81) on 1-day after measurement (1-day lag) and 2.13% (95% CI 0.03–4.22) and 4 days after measurement (4-day lag) were noted. Results were not consistent for the association with TSP concentrations and TSP blackness with mortality. Overall, this study provided little evidence of a role for coarse PM from re-suspended road dust in increased mortality; results suggested that combustion-derived particles are more strongly associated with mortality than crustal-derived particles.

VI.2.2.2 Morbidity

Table VI-3 presents a summary of information located with respect to morbidity health outcomes as the result of exposure to PM_{10} from crustal sources. Several authors conducted studies to assess the possible effects of windblown dust storms originating in the deserts of Mongolia and China (Asian Dust Storm [ADS]) on hospital admissions for various health conditions for residents in Taipei, Taiwan, during the period from 1996 – 2001. Mean concentrations of 24-hour PM_{10} during dust storms was 111.68 ± 38.32 µg/m^3 compared to the mean concentration during comparison days (usually the same day of the week, but a week prior and a week after a dust storm) of 55.43 ± 24.66 µg/m^3. All study authors found that there may not have been enough statistical power to detect associations resulting from inadequate sample size of hospital admissions for the various health endpoints on dust storm event days. The studies are summarized below in greater detail.

Chen and Yang (2005) conducted a study to assess the possible effects of [ADS] on hospital cardiovascular disease (CVD) admissions of residents in Taipei, Taiwan, during the period from 1996 – 2001. A 3.65% increase in the risk of CVD admissions during the ADS events (1 day following the day of the ADS) was observed; however, this increase was not statistically significant.

Andersen et al. (2007) found a statistically significant positive association between crustal PM_{10} and the incidence of hospital admission for cardiovascular disease, independent of PM_{10} from all other sources in Copenhagen. The attributable increased risk of cardiovascular disease was 5.1% (95% CI:1.8% to 8.4%) per 25 $\mu g/m^3$ increase in crustal PM_{10}.

Yang et al. (2005a) conducted a study to assess the possible associations of ADS on the hospital asthma admissions of residents in Taipei, Taiwan, during the period from 1996 – 2001. The association between dust storms and asthma admission was prominent 2 days after the ADS event. The estimated relative risk was 1.08 (95 % CI: 0.97 – 8.76); however, this increase was not statistically significant.

Yang et al. (2005b) designed a study to assess the possible associations of ADS on the hospital stroke admissions of residents in Taipei, Taiwan, during the period from 1996 to 2001. The study results indicated a statistically significant association between ADS events and daily primary intracerebral hemorrhagic stroke admissions 3 days after the event (relative risk of 1.15; CI, 1.01-10.10). Yang et al. (2005b) also found a positive but not significant association between ADS events and ischemic stroke admissions 3 days following the dust storms, which was due primarily to PM_{10}.

Yang et al. (2009) conducted a study to assess the possible associations of ADS on the hospital admissions for congestive heart failure for residents in Taipei, Taiwan, during the period from 1996 to 2001. The association between dust storms and congestive heart failure admission was prominent 1 day after the ADS event. The estimated relative risk was 1.11 (95 % CI: 0.99 – 1.25); however, this increase was not statistically significant.

Chang et al. (2006) conducted a study to assess the possible effects of windblown dust storms originating in the deserts of Mongolia and China on the daily clinical visits for allergic rhinitis of residents in Taipei, Taiwan, during the period of 1997-2001. The study found that the mean concentration of PM_{10} during dust storms was 110.37 ± 37.86 $\mu g/m^3$ compared to the mean concentration during comparison days (usually the same day of the week, but a week prior and a week after a dust storm) of 61.73 ± 30.22 $\mu g/m^3$. A 19% increase in the risk of clinical visits for allergic rhinitis

during the dust storm events (2 days following the ADS) was observed; however, this increase was not statistically significant.

Gordian et al. (1996) examined the effects of average 24-hour PM_{10} concentrations and carbon monoxide and temperature on the number of daily outpatient visits for respiratory disease (asthma, bronchitis and upper respiratory tract illnesses) in Anchorage, Alaska between 1992 and 1994. Particulate matter less than 10 µm (PM_{10}) in the Anchorage area is composed primarily of material from crustal sources (unpaved roads, road sanding) and volcanic ash due to the lack of industrial sources of pollution. A volcanic eruption occurred during the study (Mount Spurr on August 18, 1992) and the 24-hour average PM_{10} concentration was 565 µg/m^3 on the day after the eruption. The composition of the volcanic ash was analyzed by electron microscopy and it was determined that the majority of the particle mass (greater than 80%) was composed of particles between 2.5 and 10 µm containing primarily silica and silica-aluminum mixture. The assessment of the volcanic ash composition was consistent with a previous investigation that showed the TSP in the Anchorage area was composed primarily of material from crustal sources. The mean 24-hour PM_{10} concentration measured during the study was 45.54 µg/m^3 and the maximum was the 565 µg/m^3 as the result of a volcanic eruption. Vehicular emissions are a source of benzene and carbon monoxide which have been attributed to incomplete combustion of Alaskan gasoline which is high in benzene content. Carbon monoxide was only measured in the winter in this study. Based on the available information, PM_{10} and carbon monoxide concentrations were not correlated. The study found statistically significant positive associations between PM_{10} and incidence of outpatient medical visits due to respiratory illnesses, independent of the outdoor temperature. For asthma, the attributable risk was 4.2% per 10 µg/m^3 increase in PM_{10}. For bronchitis, the attributable risk was 2.3% per 10 µg/m^3 increase in PM_{10}. For upper respiratory tract infections, the attributable risk was 2.7% per 10 µg/m^3 increase in PM_{10}. Temperature is a marker for season, which could influence both PM_{10} levels and respiratory illness. The association of PM_{10} and increased incidences of outpatient visits was higher and only statistically significant during the period of time following the volcanic eruption. Winter carbon monoxide concentrations were found to be correlated with bronchitis and upper respiratory illnesses but not asthma.

Another study from Washington State (Hefflin et. al. 1991) found a small increase in hospital admissions for respiratory illness following dust storms where maximum concentrations exceeded 1,000 µg/m^3 over a 24-hour period. Hefflin et al. (1991) found positive associations between PM_{10} and incidence of hospital emergency room visits for bronchitis (attributable risk was 0.35% per 10 µg/m^3), and for sinusitis (attributable risk was 0.45% per 10 µg/m^3 PM_{10}). Similarly, a study conducted after the eruption of Mount St. Helens on children attending a camp in the vicinity who were exposed to elevated concentrations of dust (10,000 µg/m^3) in comparison to

the particulate matter standard at that time 260 µg/m^3 did not identify any chronic health effects or differences in health status between the morning and evening following a day of activity in air concentrations exceeding the particulate matter standard (Buist et al. 1983).

A summary of the Concentration Response Factors (Jin 2010, pers comm.) derived from epidemiological studies which demonstrated a significant positive association between exposure to crustal particulate matter and health effects is presented in Table VI-3.

Table VI-3 Summary of Morbidity Studies from PM_{10} Originating from Crustal Sources

Location	Taipei, Taiwan						Anchorage, Alaska
PM_{10} Concentration during Dust Storm Event (24-hour average) (µg/m^3)	111.68	111.68	111.68	111.68	111.68	110.37	NA
PM_{10} Concentration on Control days (24-hour average) (µg/m^3)	55.43	55.43	55.43	55.43	55.43	61.73	NA
Change in PM_{10} concentration (24-hour average) (µg/m^3)	38.32	38.32	38.32	38.32	38.32	48.64	10
Daily Increase in Morbidity							
Cardiovascular Disease Hospital Admissions	3.65% (1-day lag)	NA	NA	NA	NA	NA	NA
Asthma Hospital Admissions	NA	1.1%[(a)]	NA	NA	NA	NA	3 to 6%
Stroke Hospital Admissions	NA	NA	NA	1.2% (2-day lag)[a]	NA	NA	NA
Congestive Heart Failure Hospital Admissions	NA	NA	NA	NA	1.1% (1-day lag)[a]	NA	NA
Allergic Rhinitis Hospital/Clinic Visits	NA	NA	19% (2-day lag)[a]	NA	NA	NA	NA
Clinic Visits for Conjunctivitis	NA	NA	NA	NA	NA	11% (4-day lag)[a]	NA
Upper Respiratory Hospital Admissions	NA	NA	NA	NA	NA	NA	1 to 3%[(b)]
Reference	Chen and Yang 2005	Yang et al. 2005a	Chang et al. 2006	Yang et al. 2005b	Yang et al. 2009	Yang 2006	Gordian et al. 1996

[(a)] Not statistically significant.

[(b)] Correlated with carbon monoxide concentrations from vehicular traffic.

NA = not assessed or not available.

VI.2.3 Other Health Effects

VI.2.3.1 Ophthalmic Effects

Three studies are summarized below on the effects of particulate matter on eyes. The first study by Gupta et al. (2007) completed a case study on the impact of air pollution on eyes in India. The study examined a total of 520 subjects and was designed to investigate the impact of air pollution on eyes from vehicular pollution. The study group commuted daily via highly polluted areas for at least two years. The control group was comprised of people residing on campus and traveling from home to workplace through the campus. The subjects provided a detailed history including history of ophthalmic conditions. The results showed a significantly larger number of subjects in the study group were suffering from ophthalmic symptoms compared to control. Symptoms included redness, watering, irritation, strain, blurring and photophobia. No significant difference was observed in visual acuity in the two groups. The study did not break down air pollutant effects into individual components (e.g., NO_2 or particulate matter).

The second study by Bourcier et al. (2003) examined the effects of air pollution and climatic conditions on the frequency of ophthalmological emergency examinations. The pollutants measured in the study included NO, NO_2, O_3, SO_2 and PM_{10}. Data were collected on the number of daily examinations completed in ophthalmological emergency departments in a Paris hospital over a 1 year period. Based on a linear regression model, the study looked at correlations between air pollution and climatic conditions and the intake of patients for ocular emergencies. The study found a strong relationship between conjunctivitis and related ocular surface problems and air pollution; however the relationship was between NO_2 and the maximum temperature of the day. No relationship was found between the other covariates (e.g., particulate matter in the air) and trauma or surgical emergencies.

The third study by Yang (2006) assessed the possible effects of exposure to windblown dust storms originating in the deserts of Mongolia and China on the clinical visits for conjunctivitis in residents of Taipei, Taiwan during the period from 1997-2001. The study found that the mean concentration of PM_{10} during dust storms was 110.37 ± 37.86 $\mu g/m^3$ compared to the mean concentration during comparison days (usually the same day of the week, but a week prior and a week after a dust storm) of 61.73 ± 30.22 $\mu g/m^3$. An 11% increase in the risk of clinical visits for conjunctivitis during the dust storm events (4 days following the day of the storm event) was observed; however, this increase was not statistically significant. There may not have been enough statistical power to detect associations resulting from inadequate sample size of conjunctivitis visits on dust storm event days.

VI.3 REFERENCES

Andersen, Z.J., P. Wahlin, Raaschou-Nielsen O, Scheike T. and S. Loft. 2007. *Ambient particle source apportionment and daily hospital admissions among children and elderly in Copenhagen*. Journal of Exposure Science and Environmental Epidemiology 17:625-636.

Bourcier, T., F. Thomas, T. Bury, L. Cadiot, V. Borderie and L. Laroche. 2003. *Effects of air pollution and climatic conditions on the frequency of opthalmological emergency examinations*. British Journal of Ophthalmology 87: 809-811.

Buist, A.S., L.R. Johnson, W.M. Vollmer, G.J. Sexton and P.H. Kanarek. 1983. *Acute effects of volcanic ash from Mount St. Helens on lung function in children*. American Review of Respiratory Disease 127:714-719.

Chang, C., I. Lee, S. Tsai and C. Yang. 2006. *Correlation of Asian Storm Events with Daily Clinic Visits for Allergic Rhinitis in Taipei, Taiwan*. Journal of Toxicology and Environmental Health, Part A, 69: 229-235.

Chen, Y. and C. Yang. 2005. Effects of Asian Dust Storm Events on the Daily Hospital Admission for Cardiovascular Disease in Taipei, Taiwan. Journal of Toxicology and Environmental Health, Part A, 68: 1457-1464.

Fuentes, M., H.R. Song, S.K. Ghosh, D.M. Holland and J.M. Davis. 2006. *Spatial association between speciated fine particles and mortality*. Biometrics 62(3):855-63.

Gordian, M.E., H. Ozkaynak, J. Xue, S. Morris and J.D. Spengler. 1996. *Particulate Air Pollution and Respiratory Disease in Anchorage, Alaska*. Environmental Health Perspectives 104 (3): 290-297.

Gupta, S.K., S.C. Gupta, R. Agarwal, S. Sushma, S.S. Agrawal and S. Rohit. 2007. *A multicentric case-control study on the impact of air pollution on eyes in a metropolitan city of India*. Indian Journal of Occupational and Environmental Medicine 11(1): 37-40.

Hefflin, B.J., B. Jalaludin and E. McClure. 1991. *Surveillance for dust storms and respiratory diseases in Washington State*. Archives of Environmental Health 49: 170-174.

Jin, A. 2010. E-mail correspondence between Mike Demarchi of LGL Limited Environmental Research Associates and Andrew Jin, Health Canada. Dated June 15, 2010.

Kwon, H-J., S-H. Cho, Y. Chun, F. Lagarde and G. Pershagen. 2002. *Effects of the Asian dust events on daily mortality in Seoul, Korea*. Environmental Research Section A 90: 1-5.

Laden, F., L.M. Neas, D.W. Dockery and J. Schwartz. 2000. *Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities*. Environmental Health Perspectives 108: 941-947.

Ostro, B.D., R. Broadwin and M.J. Lipsett. 2000. *Coarse and fine particles and daily mortality in the Coachella Valley California: a follow-up study*. Journal of Exposure Analysis and Environmental Epidemiology 10: 412-419.

Ostro, B.D., S. Hurley and M. Lipsett. 1999. Air Pollution and Daily Mortality in the Coachella Valley, California: A Study of PM10 Dominated by Coarse Particles. Environmental Research, Section A, 81:231-238.

Penttinen, P., P. Tiittanen and J. Pekkanen. 2004. *Mortality and air pollution in metropolitan Helsinki, 1988--1996*. Scandinavian Journal of Work, Environmental and Health. 30(Suppl 2):19-27.

Pope, C.A., R.W. Hill and G.M. Villegas. 1999. *Particulate Air Pollution and Daily Mortality on Utah's Wasatch Front*. Environmental Health Perspectives 107: Chen et al. 2004

Schwartz, J., G. Norris, T. Larson, L. Sheppard, C. Claiborne and J. Koenig. 1999. *Episodes of High Coarse Particle Concentrations Are Not Associated with Increased Mortality*. Environmental Health Perspectives 107 (5): 339-342.

Schwarze, P.E., J. Ovrevik, M. Lag, M. Refsnes, P. Nafstad, R.B. Hetland and E. Dybing. 2006. *Particulate matter properties and health effects: consistency of epidemiological and toxicological studies*. Human and Experimental Toxicology 25: 559-579.

Slaughter, J.C., E. Kim, L. Sheppard, J.H. Sullivan, T. Larson and C. Claiborn. 2005. *Association between Particulate Matter and Emergency Room Visits, Hospital Admissions, and Mortality in Spokane, Washington*. Journal of Exposure Analysis and Environmental Epidemiology 15: 153-159.

Staniswalis, J.G., N.J. Parks, J.O. Bader and Y.M. Maldonado. 2005. Temporal analysis of airborne particulate matter reveals a dose-rate effect on mortality in El Paso: indications of differential toxicity for different particle mixtures. Journal of the Air and Waste Management Association 55(7): 893-902.

Yang, C. 2006. Effects of Asian Dust Storm Events on Daily Clinical Visits for Conjunctivitis in Taipei, Taiwan. Journal of Toxicology and Environmental Health, Part A, 69: 1673-1680.

Yang, C., S. Tsai, C. Chang and S. Ho. 2005a. Effects of Asian Dust Storm Events on Daily Admissions for Asthma in Taipei, Taiwan. Inhalation Toxicology 17: 817-821.

Yang, C., Y. Chen, H. Chiu and W. Goggins. 2005b. *Effects of Asian Dust Storm Events on Daily Stroke Admissions in Taipei, Taiwan*. Environmental Research 99: 79-84.